Diving and Hyperbaric Medicine

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EUBS

Decompression illness in cave divers

Middle ear barotrauma and language of instruction

Australian diving-related fatalities 2010

Cardiac disease in UK divers

Cone shell envenomation

Underwater blast injury

Oro-facial barotrauma

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PURPOSES OF THE SOCIETIES

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

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The Editor's offering

Barotrauma from pressure changes, decompression sickness related to inert gas kinetics, toxic marine organisms, overhead environments, cardiovascular disease, stupid decision-making – these are just some of the familiar challenges, natural and man-made, of entering the underwater world. Underwater blast injury, however, is a topic that has not been previously addressed in this journal and will be unfamiliar to all but those with military training. Anyone diving a tropical island and having experienced dynamite fishing nearby knows how unpleasant is the sensation of the shock wave even from a relatively distant, small explosion. The review by Lance and Bass[1] highlights the surprising lack of consistency in the data and the understanding of the effects of underwater blast. Peer review of this paper generated controversy amongst 'experts' in this field. It appears that such a review has been long overdue and has highlighted the need for better research data.

In a large cave diving database, Harris et al. have made considerable efforts to establish a reliable denominator for determining the incidence of decompression illness (DCI).[2] The detailed description of their methodologies highlights how difficult this is to achieve. Nevertheless, they have demonstrated that well-controlled recreational diving in an overhead environment does not appear to carry any greater risk for DCI than reported in open-water studies. However, a small subset of deep, technical dives again highlights the increased risk of injury from this type of diving, and suggests that more forethought is needed amongst divers involved in 'pushing the limits'. This is not to say that we should not condone such pursuits, any more than other adventure sports.

Past papers in DHM have reviewed the toxicology of jellyfish, especially the box jellyfish, *Chironex flexeri*, and the clinical management of envenomed victims. Halford et al. now provide a useful review of cone shell envenomation and its management.[3] From a clinical standpoint, the take-home message is that the neurotoxins released are paralytic and that victims die of asphyxia from acute respiratory failure unless respiratory support is provided promptly and efficiently. Medical support providers also need to remember that victims may be paralysed but conscious – a terrifying experience for them.

Of particular value in the Blake et al. paper[4] on the impact of language of instruction on the incidence of middle ear barotrauma (MEBt) are a new set of photographs to illustrate the Edmonds classification of MEBt.[5] These are of superior quality to the original set of otoscopic photos, two of which (grades 1 and 2) on close examination appeared to be the same image, presented with different orientation and colouration but depicting the same tympanic membrane.

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Michael Davis

Editor-in-Chief, *Diving and Hyperbaric Medicine Journal*

Call for expressions of interest – closing date: 31 October 2015

Diving and Hyperbaric Medicine is published jointly by the South Pacific Underwater Medicine Society (SPUMS) and the European Underwater and Baromedical Society (EUBS). The journal is indexed on Medline, SciSearch® and Embase/Scopus.

Expressions of interest are called for the position of Editor-in-Chief from 01 January 2016. This is a contracted position for five (5) years and attracts a modest honorarium.

Applicants must have an established academic track record in medicine and experience in editing and publishing peer reviewed journals. Preference will be given to medically qualified applicants, given the journal's track record as a leading publication in diving medicine.

The Editor of the Journal is a prominent position for both Societies, and the successful applicant is expected to be open minded and adaptable, given the multicultural, multilingual and intercontinental origins of the Societies.

Please forward all enquiries with a curriculum vitae to both the SPUMS President, Assoc. Prof. David Smart, <president@spums.org.au> and EUBS President, Asst. Prof. Jacek Kot, <president@eubs.org>

Front-page photo taken by Richard 'Harry' Harris in Kilsbys Sinkhole, Mount Gambier region. The CCR diver is at about 45 m depth, with light from the entrance silhouetting him and another diver's light in the distance.

The President's page

David Smart, President SPUMS

It is hard to believe a year has passed since my election as President. During the past year we have achieved some milestones, and have multiple projects underway. I am pleased to report that SPUMS has continued to grow as an organisation, in size and influence, and with a sound financial base. Our relationship with the EUBS continues to progress positively. Our website has been migrated to a new host. Nicky McNeish's excellent work is ongoing to upgrade the functionality so it meets our future needs. Now is the time for members to forward ideas to the Executive for improvements and changes they would like to see in the website's functionality. Joel Hissink, SPUMS webmaster, is working with Nicky McNeish to collate and implement our website for the future. Hopefully this can be a multipurpose site to serve our members better; combining subscriptions, administrative processes, education, and other functions.

Following last year's AGM it was necessary to revise our Purposes and Rules to meet new legislative requirements of the Victorian Government through Consumer Affairs Victoria. I am sincerely grateful to Mike Davis and the SPUMS Executive Committee for their input and hard work in redrafting our old Purposes and Rules in a new format to comply with the new regulations. On 01 November 2014, we held a Special General Meeting which accepted the new Purposes and Rules unanimously. I thank the members who took the time to attend or vote by proxy.

Our committee members for 2014–15 are listed below, and the year each member commenced in that role (many members have occupied other executive roles for many years prior to their current positions).
President: David Smart (commenced 2014; previously Education Officer and Chairman ANZHMG)
Immediate Past President: Mike Bennett (commenced 2014; previously President and Chairman ANZHMG)
Secretary: Karen Richardson (commenced 2011)
Treasurer: Peter Smith (commenced 2014)
Webmaster: Joel Hissink (commenced 2013)
Education Officer: David Wilkinson (commenced 2014)
Journal Editor: Mike Davis (commenced 2002; previously Chairman New Zealand Chapter)
Chairman ANZHMG: John Orton (commenced 2014)
Committee Member: Denise Blake (commenced 2012)
Committee Member: Simon Mitchell (commenced 2012)
Committee Member: Janine Gregson (commenced 2014)

This year, Karen Richardson retires from her role as Secretary. Karen has done a terrific job. She kindly continued on for an extra year following an absence of nominees last year. Karen has also overseen the absorption of the Public Officer role into the secretarial position description. Well done and my personal thanks, Karen.

A joint journal Governance Group has been established with EUBS (two members from each Society) in the last 12 months. Although established, it has not yet become fully operational. This operational capability is required urgently to guide the governance of the Journal. It will be a focus of coming months for it to get into its stride.

There is also work being undertaken to create at least one point of parity in diving medicine training for physicians across Europe and the South Pacific. There are considerable differences in the processes of training in diving and hyperbaric medicine, ranging from short courses, clinical attachments, web-based packages, university and college-based courses. All have merits, and there are points of commonality to work with to achieve some form of mutual recognition.

At the time of this AGM, there remains uncertainty about the ANZCA Certificate in Diving and Hyperbaric Medicine. Commenced over a decade ago, this qualification is regarded as the highest level achievable in this field in Australia and New Zealand. Because of multiple factors and small numbers of trainees in the programme, the ANZCA has questioned its viability. A number of SPUMS members, including the Immediate Past President, are working with ANZCA in an effort to maintain the programme. The SPUMS Diploma continues to be a recognised qualification in diving and hyperbaric medicine, and is the only qualification recognised in the Australian Medicare Schedule.

Finally, I had the privilege of attending another SPUMS ASM, this year in Palau. I offer my congratulations and thanks to Cathy Meehan who almost single-handedly convened this meeting. While convening the conference, Cathy has also occupied a role as SPUMS representative on Australian Standards – a huge undertaking. I also thank our keynote speaker, Neal Pollock and guest speaker, Rebecca Johnson. I am sure everyone who attended will agree this was another highly successful and enjoyable meeting. In addition, numbers of delegates this year have again increased. Well done, Cathy. I would also like to sincerely thank Steve Goble for his excellent work as SPUMS Administrator and his support of our ASM.

Planning is underway for the 2016 Annual Scientific Meeting, returning to Fiji now that democratic rule has been re-established. Janine Gregson is convening that meeting and I call upon all members to support Janine's efforts by offering assistance and planning to attend and to speak at the meeting. I mentioned in my March 2015 President's report how much we owe to our volunteers. Again I thank everyone who has contributed to the Society in the last 12 months.

Key words
Medical society, general interest

Original articles

A 10-year estimate of the incidence of decompression illness in a discrete group of recreational cave divers in Australia

Richard JD Harris, Geoff Frawley, Bridget C Devaney, Andrew Fock and Andrea B Jones

Abstract

(Harris RJD, Frawley G, Devaney BC, Fock A, Jones AB. A 10-year estimate of the incidence of decompression illness in a discrete group of recreational cave divers in Australia. *Diving and Hyperbaric Medicine*. 2015 September;45(3):147-153.)

Introduction: The vast majority of freshwater cave diving in Australia occurs within the limestone caves of the Gambier karst in the south-east of South Australia. The incidence of decompression illness (DCI) in cave divers is presumed to be higher than open-water recreational divers because of the greater depths involved, but has not previously been reported. Our aim was to determine the incidence of DCI in cave divers, the patterns of diving and the outcome of hyperbaric treatment.

Methods: This was a retrospective cohort study of cave divers with DCI presenting to the Royal Adelaide Hospital or The Alfred Hospital over a 10-year period between 2002 and 2012. We reviewed case notes of cave divers who were treated for DCI after diving in the Mt Gambier karst. As there are no records of the number of dives performed during the study period we generated a denominator for the incidence of DCI by extrapolating available data and making a number of assumptions about the number of dives per dive permit issued.

Results: Sixteen patients were treated for DCI during the study period. The precipitating dive was a single deep decompression dive in seven cases, multiday repetitive dive sequences in eight and a non-decompression dive in one. Three of the 16 cases of DCI involved dives in excess of 90 metres' fresh water (mfw) using trimix. As the total estimated number of dives in the study period was approximately 57,000 the incidence of DCI in Australian cave divers was estimated to be 2.8:10,000 (0.028%). It is possible that the overall incidence of DCI is as high as 0.05%, and even higher when dives to depths greater than 90 mfw are involved.

Conclusions: The estimated incidence of DCS in this series is lower than expected but consistent with other series describing DCI in cold-water recreational diving.

Key words

Decompression illness; decompression sickness; cave diving; technical diving; first aid; epidemiology; clinical audit

Introduction

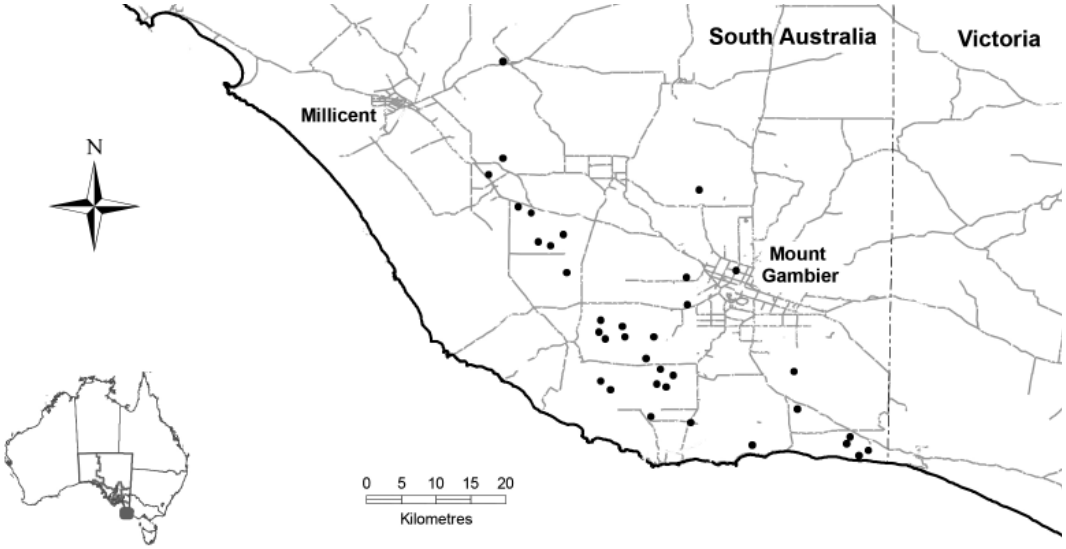
Decompression illness (DCI) describes a range of symptoms caused by bubbles in blood or tissue during or after a reduction in ambient pressure. It encompasses two pathophysiological syndromes, namely arterial gas embolism (AGE) and the more common decompression sickness (DCS). Cave diving involves entering a flooded, overhead environment and is highly equipment and technique intensive. Technical diving can be defined as using equipment and techniques to execute deeper or longer dives than recreational divers,[1] and such techniques are often used by cave divers.

Accurate incidence data for DCI in recreational divers is difficult to obtain, primarily because of the problems involved in establishing the number of participants in the activity (the denominator). It has been estimated at 0.96 per 10,000 dives (0.01%) in a cold-water recreational diving population.[2] The incidence of DCI in technical divers has been described in a number of small series but the incidence in cave divers has not been reported previously. One technical cave diving project in a deep Mount Gambier sinkhole described a DCS probability with a 95% confidence interval of 10–340/10,000 dives (0.1–3.4%).[3]

The vast majority of freshwater cave diving in Australia occurs within the confines of the Gambier karst in the south-east of South Australia (SA). The area contains many hundreds of named limestone features, many of which are a mecca for cave divers from around the country and overseas (Figure 1). Access to the diveable caves is for the most part managed by a single organization; the Cave Divers Association of Australia (CDAA). The diving is highly regulated but there are still several sources of information regarding the number of dives performed per annum. This kind of denominator for dive accident analysis is uncommon in recreational diving data. A discrete population of cave divers who perform multiple similar dives in a limited number of sites offers a unique opportunity to gain insight into the patterns and incidence of DCI in these types of dives.

Mt Gambier lies halfway between the capital cities Melbourne, Victoria, and Adelaide, SA, and the majority of CDAA members come from one of these two states. Divers who recognize that they may be suffering from DCI are likely to either self-treat (especially in mild or resolving forms) or be transferred to one of the two hospital-based recompression chambers in Melbourne and Adelaide. If a diver presents to the Mt Gambier Hospital (MGH), he or she

Figure 1
Sinkholes and cave diving sites in the Mt Gambier region, South Australia (courtesy Ian Lewis)



will usually be transferred to Adelaide regardless of their state of origin. Therefore, it is assumed that most clinically significant cases of DCI will be captured by examining cases treated at these two hospitals. This paper provides a descriptive analysis of the incidence of decompression illness arising in this population of divers.

Methods

Following ethics approval by the Human Research Ethics Committees of The Royal Adelaide Hospital (RAH), Adelaide, (HREC 120812) and The Alfred Hospital (AH), Melbourne, (HREC 365/13) the treatment databases from the two hospitals were examined to identify cases of DCS or CAGE presenting as a result of dives performed in the caves or sinkholes of the Mt Gambier karst. All cases treated at either hospital between 01 June 2002 and 31 May 2012 were identified and the case notes reviewed. Demographic data of the diver, dive profiles, exact location, dive gas and equipment configuration, presenting complaint, pre-hospital treatment, time to recompression, recompression treatment and outcome were recorded. The total numbers of cases in the ten-year period were used to form the numerator for the overall incidence of DCI.

There is no central database that accurately records the number of dives performed in the Mt Gambier cave system. However, diving in this area is highly regulated and requires daily diving permits or landowner permission. Therefore, the denominator was derived from available data recording permits issued and estimates of dives per permit. Where records were incomplete, but the frequency of permit provision was consistent, extrapolation of the available data was used to estimate the permit data for the missing time frame. Any data that were available from 2001 to 2013 (18 months either side of the study period) was used to give a 10-year figure. We gained information on the number of permits issued during the study period from a number of sources.

The CDAA currently has diving access to 24 sites in this region.[4] Through a cooperative relationship with the various landowners, the CDAA controls access and ensures divers have completed approved cave diving training. Forestry South Australia (FSA) issues permits for seven sites and maintains accurate records of permits, whilst the Lady Nelson Visitor Centre releases the access keys for four of the FSA caves. The Department of Environment, Water and Natural Resources (DEWNR) issues permits for several other caves. Eleven caves are on private property or are owned by councils and usage data for these were inconsistent. Different landowners have different access requirements. Some completely entrust the CDAA to manage dive bookings whilst others have a primary role in issuing permits. Some entrust the distribution of access keys to a third party (The Lady Nelson Visitor Centre). Finally, some sites are essentially 'open', requiring only a knock on the farmers' doors. As a consequence of these disparate arrangements, no single data source exists to estimate the number of dives performed within the 10-year study period.

A number of assumptions were made to extrapolate the number of dives from the number of permits issued. This was based on the extensive cave diving experience of two of the authors (RH, AF) at these sites. Diving at sites controlled by FSA requires a permit and each diver is listed on the permit so a record of 'user days' is kept. For example, two divers on the permit for Forestry sites for three days will be recorded as six user days. It was assumed that six dives were performed in this time; however, it is possible that fewer dives occurred (divers not attending, apathy, illness,

Table 1

Dive site depths, (metres' fresh water, mfw) number of dives per dive site (if more than one reporting source for a site, the total shown is the mean), total estimated dives for study period (see text for explanation of how estimates were obtained) and incidents of decompression illness (DCI); DCI was attributed to a site if symptoms appeared during or after a dive in that site, regardless of previous or subsequent dive sites; in two cases, the dive site was not recorded, however, one was a sinkhole dive to 36 mfw, the other a 40 mfw training dive;

*Estimated from four dives per weekend; †Estimated from six dives per weekend; ‡Total is sum of CDAA dives and ASF dives

Sites	Depth (mfw)	FSA	CDAA	Lady Nelson	ASF	DENWR	Total dives	DCI
Deep caverns/sinkholes								
Ela Elap	50						2,080*	
Gouldens Hole	26					6,078	6,078	
Hells Hole	26	350	572	201			374	
Kilsby's Sinkhole	64		6,791				6,791	7
Little Blue Lake	40						3,120†	1
One Tree	50						3,120†	1
Piccaninnie Ponds	110					5,330	5,330	1
The Shaft	120		1,563		116		1,679‡	2
The Sisters	20					400	400	
Caves								
Allendale Sinkhole	27			1,521			1,521	
Baker's Cave	32			18			18	
Mud Hole	18	7,346	3,215				5,280	
Advanced caves								
Iddlebiddy	18	1,126	709	660			832	
Nettlebed	28	855	760	573			729	
Stinging Nettle Cave	35	778	938	400			705	
Tank Cave	18		4,290				4,290	1
The Pines	40	17,654	11,396				14,525	1
Three Sisters Cave	35			27			27	
Unknown site								2
Total							**56,899**	**16**

time constraints) or more dives occurred (divers often perform two or more dives in a day). Some caves readily lend themselves to more than one dive in a day, whereas others would usually be the subject of a single dive due to their small size and tendency for silt disturbance and poor visibility. For some sites controlled by the DEWNR, only one dive per permit is allowed (a specific time slot is booked). For the other sites it is possible that more than one dive may occur per permit. As with the FSA sites, a single dive per permit has been assumed.

Collecting data for the caves on private property or owned by councils was more difficult. For each weekend permit for a site such as Tank Cave (where two to four dives would be commonplace), an assumption was made that three dives were performed. For another site, each user day has been multiplied by 1.5 to best estimate actual dives performed based on the authors' experience. An additional 116 dives performed by the Australian Speleological Federation-Cave Diving Group (ASF-CDG) Shaft Mapping Project during the study period (Payne T, personal communication, 2014) were added to the database. For three other sites, no data only estimates were available. Two sinkholes on private property and one on council land do not require any formal booking for dives, and so no records of diving are kept. An estimate (based on discussions with numerous cave divers) has been made for these sites. Some sites had maximum depths of less than 15 metres fresh water (mfw) and were included in this analysis despite the low likelihood of DCI arising from dives there. A few sites were not included as they are very shallow and seldom dived. Three large deep sinkholes on private property were only open briefly for limited diving during the study period and no data were available.

The CDAA has collected data for many sites but only intermittently. CDAA data relating to FSA sites existed for the study period but were limited to essentially the last 17 months of the 10-year period. Online bookings via the CDAA commenced in November 2010, and dive numbers up to September 2013 were used and extrapolated to a 10-year period. The data for one site, Kilsbys, were of the highest quality, over a period of 8.5 years. However, data were accurate for only or 35 months for The Shaft; and 29 months (from June 2010) for Tank Cave. The Lady Nelson Centre was able to provide seven years' data for five sites. For DEWNR sites, permit data were available for the last 30 months of the study period, and a further 18 months after this. The DEWNR total (four years) was extrapolated to 10 years.

Data from FSA probably overestimates dive numbers as some divers will book multiple sites for multiple days, but are most unlikely to complete all the dives implied by this number. The Lady Nelson numbers reflect the number of times the keys to open certain caves are borrowed and if anything this figure is likely to be more accurate or even underestimate total dive numbers. The CDAA data only commenced in June 2010, so the 10-year totals were extrapolated from a small data set. Since the data are suspected to both over- and underestimate the true values, the authors' felt it reasonable to average it where more than one source existed. These averages were summed to give the total estimated dive number for a 10-year period. For three dive sites, the numbers are truly a best guess. The booking of multiple sites on one permit introduces an error in usage rates that cannot be quantified. For example, in the study period, 7,312 user days were booked for the combination of Pines Cave and Mudhole. If each user dived both of these sites as per the booking, one could ascribe 7,312 dives to each site. However, it is the authors' experience that sometimes the secondary site (Mudhole) is booked because it is close by, but may not be dived (fatigue, time constraints, etc). As it is impossible to know exactly how many dives were performed, one diver day there has been equated with one dive.

Results

The actual and estimated numbers of dives for the caverns, sinkholes and caves in the Mount Gambier region are listed in Table 1. This amounts to a total of 56,899 dives over the 10-year study period between 2002 and 2012.

During this period, 19 divers from the Gambier karst presented to one of the two hyperbaric units for assessment (RAH 9, AH 10). Two of the RAH divers were commercial divers performing training dives in one of the sinkholes. As they were utilizing commercial diving techniques including wetsuits, surface supplied gas, surface directed decompression and DCIEM decompression tables, they were not included in this analysis as the study pertained only to recreational cave divers. One other diver presenting to the Alfred had been treated several weeks earlier at the RAH. His symptoms were attributed to the earlier episode (incomplete resolution) and no further hyperbaric treatment was given. Therefore, this second presentation was not included in the analysis. Thus, 16 divers (all male; mean age 38 +/- 5.7 years old) with DCI (all diagnosed as DCS) were treated with hyperbaric oxygen therapy (RAH 7, Alfred 9). With a total estimated number of dives in the study period of 56,899, this gives a DCI incidence of 2.8:10,000 (0.028%) for the 16 treated cases.

Dive details including gas used, depth attained and decompression plans are summarised in Table 2. The precipitating event was repetitive, multiday dive sequences in eight cases (50%), a single deep decompression dive (> 35 metres' fresh water, mfw) in five cases and a single

Table 2

Maximum depths (mfw – metres' fresh water, median and interquartile range (IQR) or range shown), risk factors, symptoms and subsequent management of 16 divers treated for decompression illness (DCI); 18:60:30 – 18 msw equivalent (284 kPa) for 60 min followed by 30-min ascent; 14:60:30 – 14 msw equivalent (243 kPa) for 60 min followed by 30-min ascent

Diving profiles and clinical data	**Incidence**	**Comments (depth range mfw)**
Maximum depth (IQR)	55 (38–72)	(n = 1) 19 (n = 10) 35–44 (n = 2) 45–60 (n = 3) > 90
Gas mixture		
Air	11	Air 39.9 (33.4–46.4)
Trimix	5	Trimix 87.2 (48.9–120)
Decompression mixture		
Air	9	
Nitrox	5	
Oxygen	2	
Predisposing factors		
Pre-dive fatigue	5	
Alcohol	5	
Post-dive exertion	5	
None	1	
Initial symptoms/signs (more than one in most divers)		
Pain	15	
Motor weakness	5	
Sensory changes	6	
Inner ear	1	
Constitutional	8	
First aid at dive site		
100% oxygen	8	
No first aid at site	8	
In-water recompression	2	
Hyperbaric treatment		
Delay to treatment (h) (IQR)	26.5 (24–48)	1 diver presented at 3 weeks
Initial treatment:		
US Navy Table 6	7	
Royal Navy Table 62	8	
Royal Navy Table 61	1	
Second treatment:		
Royal Navy Table 61	7	
18:60:30 Table	7	
14:60:30 Table	2	
Total treatments (IQR):	3 (2–4)	
Outcomes (at discharge from hospital)		
Full resolution	11	1 diver failed to return
Minor disability	4	
Total cases of treated DCI	16	

Table 3
Incidence of DCS in different dive groups and under different conditions;
AAUS – American Academy of Underwater Sciences; DAN – Divers Alert Network

Dive population	Specific cohort	Incidence per 10,000 dives (%)		Comments
Scientific dives[15]	AAUS divers	0.324	(0.0032)	North America; 1,019,159 dives
Recreational dives	Cruise ships[13]	0.9	(0.009)	Various locations; 77,680 dives
	Cold water[2]	0.957	(0.010)	British Columbia, Canada; 146,291 dives
	Warm water[14]	1.06	(0.011)	Townsville, Australia; 677,767 dives
Project Dive Exploration[12]	All dives 1998–2004	3	(0.03)	DAN members; 80,439 dives
	Cold-water subset[6]	28	(0.28)	Scapa Flow decompression dives
	Warm-water subset[6]	2	(0.02)	
Technical dives[3]	All	10–340	(0.1–3.4)	Small series; wide 95% CIs
	Depth ≥ 90 m subset	1,330–4,550	(13.3–45.5)	

decompression dive in two cases. Three cases of DCI arose from single deep decompression dives (> 90 mfw) using trimix as the bottom gas. Eleven divers in the repetitive and multiday diving series used air as the primary breathing gas with maximum depths ranging from 19–60 mfw (mean 40.2 mfw) during single and repetitive dives. In only two of the air-diving cases did the diver appear to accelerate decompression with enriched air nitrox (EANx) or oxygen. One case involved the use of a closed-circuit rebreather, whilst all other cases presented in this paper arose from traditional open-circuit scuba.

Dives conducted during the Shaft Project and the Piccaninnie Ponds Project[5] were included in this study. The Shaft Project consisted of 116 dives which fell within the study period, (225 project dives total, with 33 dives ≥ 90 mfw). The incidence of DCI for dives deeper than 90 mfw was 6%, which is much higher than the overall incidence of DCI.[3] Similarly the incidence of DCS for dives ≥ 90 mfw during the Piccaninnie Ponds Project (consisting of 51 dives total, 15 ≥ 90 mfw) was 6.7%. This increases to 20% if two self-treated cases of mild DCS are included (Richard Harris, personal communication, 2014).

Factors considered to predispose to DCI are described in Table 2. Pain was the presenting symptom in 15 of the 16 cases and neurological symptoms were present in eleven. Of these 11 neurological presentations, weakness was noted in five and paraesthesiae in six. There was one case of inner ear DCI, which presented with vertigo and nausea. Ascent to altitude > 300 m following diving was listed as a contributor in two cases. All dives were performed in relatively cold water (11–16^{O}C), although the almost exclusive use of dry suits in this population would be expected to prevent significant cooling. A persistent foramen ovale (PFO) was diagnosed in one diver after treatment for DCI.

Appropriate initial management of DCI with 100% oxygen was used in eight of the 16 cases. The four divers who presented to a regional health facility received oxygen and, in some cases, intravenous fluids in a timely manner. Six divers self-administered oxygen in the field and two of these performed some form of in-water recompression (IWR) before presenting to hospital.

The two hyperbaric units are 435 km (RAH) and 441 km (AH) from Mt Gambier. Even allowing for the transport times required there were significant delays to definitive treatment in this series. The mean delay to treatment for divers presenting to the RAH was 48 hours (24–96 h) following the last dive. Additional delays occurred as a result of primary triage at Mount Gambier Hospital and subsequent referral to RAH. Seven divers presented to the Alfred Hospital at a mean average time of 22 hours (12–28 h). Initial review at MGH (one diver) and Hamilton Hospital, Victoria, delayed recompression treatment by 24 h and 2.5 h respectively. Eleven divers made a full recovery and four had only minor symptoms at discharge from hospital (Table 2).

Discussion

The estimated incidence of DCS in this series (2.8:10,000 dives) is consistent with other series describing DCI in recreational divers but potentially may be higher (up to 5:10,000 dives) depending on whether or not some of our assumptions have inflated the estimated dive numbers. Nevertheless, this incidence in cave divers is lower than expected, especially allowing for the year-round cold water in the Gambier karst and the high proportion of divers likely to be performing staged decompression dives. Of all groups, cave and technical divers have been least studied. The reported incidence of DCI varies between 1:10,000 to 9.5:10,000 depending on whether the divers are involved in recreational,[2,6–8] technical,[3] scientific,[9,10] military[11] or commercial activities (Table 3).[12]

This series highlights the difficulty in accurately determining the number of dives performed in any location. The authors are optimistic that most of the significant incidents of DCI have been captured. However, it is possible that some sick divers sought treatment or follow up in other states after diving in Mt Gambier. Approximately 74% of members

are from South Australia and Victoria, which does leave a significant number of interstate visitors.[4] It is also likely that some divers self-treated, ignored or had spontaneously resolving symptoms of DCI. One author (RH) is aware of several such anecdotal cases. Hence, this study almost certainly underestimates the total number of cases of DCI.

A number of areas of concern in the practice of cave diving have been highlighted by this study. The dive plans of some of the patients involved diving on air to depths of 60 mfw and a failure to plan for accelerated decompression with EANx. There are also concerns about the lack of appropriate first aid on site, use of in-water recompression and delays until definitive hyperbaric treatment. The divers in this series who developed DCI may have exacerbated development of injury in a number of ways, including provocative dives and ascent to an altitude of greater than 300 m after onset of DCI symptoms.[13] There were three divers who performed dives in excess of 90 mfw and developed DCS. A high probability of DCS (13.3–45%) was reported in a small group of technical cave divers, especially in dives performed beyond depths of 90 mfw.[3] Whilst the delay to definitive treatment could be considered unacceptably long (average 26.5 h), it does compare favourably with New Zealand recreational diver studies (mean 67 h, SD 113).[14,15] Such delays may adversely impact treatment outcomes,[16–18] although not all studies confirm this.[19]

LIMITATIONS

The greatest uncertainty lies with the accuracy of the number of dives performed. Every effort has been made to correctly determine this figure; however, the authors accept that for some sites the numbers have been extrapolated from limited data, and in other cases there is considerable variation between the different sources. The lack of precision about the number of dives is common in most studies of decompression illness. Other authors have used surrogate measures of dive numbers such as number of tank fills[2] or the results of voluntary surveys to central registries.[20,21] Both formats are likely to underestimate the total number of dives. Greater data precision is possible with scientific or military diving but this precision is unlikely to occur with cave diving until permits are provided by a single authority (such as the CDAA) and a centralised database is established. All the patients with DCI in this series were male. The fact that no female divers presented for treatment of DCI (despite representing 15% the CDAA membership)[4] might reflect different diving patterns or fewer women performing dives over 90 mfw depth.[22]

Conclusions

We found the estimated incidence of DCS in a discrete population of recreational cave divers, diving under similar conditions of depth, temperature and dive profile, to be approximately 2.8:10,000 (or possibly up to 5:10,000 dives). This appears to be well within the expected range for decompression diving in cool water, and suggests that current diving practices and training within this population are effective and appropriate.[21,23] However, in the subset of deep dives beyond 90 mfw, the DCS incidence is much higher, suggesting that current diving practices in this range need further refinement. Only a small proportion of divers self-administered oxygen as first aid and there appears to be a disjoint between diver education and practical application regarding the suggested risk factors for DCI. Despite significant delays to definitive treatment, outcomes for most divers were excellent.

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Conflict of interest

Richard Harris is the Search and Rescue Officer for the CDAA.

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Provisional report on diving-related fatalities in Australian waters 2010

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Abstract

(Lippmann J, Lawrence CL, Wodak T, Fock A, Jamieson S, Walker D, Harris R. Provisional report on diving-related fatalities in Australian waters 2010. *Diving and Hyperbaric Medicine*. 2015 September;45(3):154-175.)

Introduction: An individual case review was conducted of known diving-related deaths that occurred in Australia in 2010.

Method: The case studies were compiled using statements from witnesses and reports of the police and coroners. In each case, the particular circumstances of the accident and details from the post-mortem examination, where available, are provided. A root cause analysis was made for each case.

Results: There were 20 reported fatalities, one less than the previous year. Five of the victims were female (four scuba divers) and 15 were males. Twelve deaths occurred while snorkelling and/or breath-hold diving, seven while scuba diving (one of whom was using a rebreather), and one diver died while using surface supplied breathing apparatus. At least two breath-hold divers likely drowned as a result of apnoeic hypoxia. Cardiac-related issues were thought to have contributed to the deaths of at least three and possibly five snorkellers, and of at least one, possibly two compressed gas divers.

Conclusions: Snorkelling or diving alone, poor supervision, apnoeic hypoxia, pre-existing medical conditions, lack of recent experience and unfamiliar and/or poorly-functioning equipment were features in several deaths in this series. Reducing delays to CT-scanning and autopsy and coroners' reports documenting that the victim of a drowning was snorkelling or scuba diving at the time are aspects of the investigation of these fatalities that could be improved.

Key words

Diving deaths; scuba; breath-hold diving; surface-supply breathing apparatus (SSBA); diving accidents; case reports

Introduction

Scuba diving and snorkelling are popular recreational activities in Australia in which, during or as a result of their participation, some die each year. Given that diving takes place in a relatively inhospitable environment, some of these deaths are unavoidable. However, analysis of diving-related fatalities indicates that many might have been avoided through appropriate preventative measures such as more extensive education and training, greater experience, better planning and decision-making, appropriate medical screening, improved supervision, or better equipment choice, familiarity and maintenance.

The aims of the Divers Alert Network (DAN) Dive Fatality Reporting Project are to:

- educate divers and the diving industry about good, safe diving and snorkelling practices;
- inform physicians on the causes of fatal dive accidents

in the hope of reducing the incidence of similar accidents in the future and of detecting, in advance, those who may be at risk. This report includes the diving-related fatalities between 01 January and 31 December 2010 that are recorded on the DAN Asia-Pacific (AP) database. When an accident was unwitnessed, it is difficult to determine accurately what had occurred. We have sometimes included considered speculation within the comments to provoke thought about the possible sequence of events.

Methods

As part of its on-going research into, and reporting of diving fatalities in Australia and elsewhere in the Asia-Pacific region, DAN AP has obtained ethics approval from the Victorian Department of Justice Human Research Ethics Committee to access and report on data included in the Australian National Coronial Information System (NCIS); the Royal Prince Alfred Hospital Human Research Ethics Committee; the Coronial Ethics Committee of the Coroner's Court of Western Australia; and the Queensland Office of the State Coroner. The methodology used for this report was identical to that described previously for the 2004 Australian diving-related fatalities.[1]

Breath-hold and snorkelling fatalities

BH 10/01

This victim was a 39-year-old (y.o.) male who ran for exercise and, other than being obese (BMI 31.7 $kg \cdot m^{-2}$) appeared to have been relatively healthy. His medical history revealed a compound fracture of the right elbow with subsequent osteomyelitis, renal colic and recent ureteroscopy and laser lithotripsy to remove a kidney stone, after which he had been cleared to dive by his doctor. He was an experienced snorkeller and scuba diver who was certified seven years earlier but had been diving unqualified for many years prior to that. He regularly dived alone catching crabs. The victim was snorkelling alone at a familiar site on a warm, still night. In addition to a reef, there was a large wreck scuttled at this site as a breakwater at a depth of 4–6 metres' sea water (msw). He wore a mask, snorkel, fins, full wetsuit with attached hood, booties and gloves and carried a torch and a catch bag.

Table 1
Summary of snorkelling and breath-hold diving-related fatalities 2010; BMI – body mass index; BNS – buddy not separated; BSB – buddy separated before problem; GNS – group not separated; GSB – group separated before; mw – metres' water; n/a – not applicable; n/s – not stated; jkt – wearing lifejacket

BH	Age (yr)	Sex	Height (cm)	Weight (kg)	BMI ($kg \cdot m^{-2}$)	Experience	Dive group	Dive purpose	Depth (mw)	Incident (mw)	Weight belt, weights (kg)	Disabling injury
10/01	39	M	181	104	31.7	Yes	Solo	Hunting	4–6	n/s	Yes, n/s	Unknown, asphyxia?
10/02	77	M	184	111	32.8	n/s	BNS	Recreation	n/s	surface	No, jkt	Cardiac incident
10/03	27	M	185	83	24.3	Yes	Solo	Training	1–1.5	n/s	n/s	Asphyxia
10/04	24	M	—	—	—	n/s	Solo	Recreation	n/s	n/s	n/s	Unknown, asphyxia
10/05	55	M	173	130	43.4	n/s	GNS	Recreation	n/s	surface	No, n/a	Cardiac incident
10/06	27	M	173	64	21.4	n/s	GSB	Recreation	5–6	n/s	n/s	Unknown, asphyxia?
10/07	73	M	197	120	30.9	Some	GNS	Recreation	n/s	surface	n/s	Cardiac incident
10/08	60	M	175	83	27.1	Yes	BSB	Spearfishing	10–15	surface	Yes, n/s	Cardiac incident?
10/09	30	M	180	78	24.1	Yes	GSB	Spearfishing	10	n/s	Yes, n/s	Cardiac incident? asphyxia?
10/10	64	F	164	58	21.6	nil	GSB	Recreation	15–20	surface	n/s	Unknown, asphyxia
10/11	31	M	185	85	24.8	Yes	BNS	Spearfishing	23–30	n/s	Yes, 4.5	Asphyxia
10/12	28	M	178	78	24.6	Yes	GSB	Recreation	16	n/s	n/s	Asphyxia? cardiac incident?

His wife alerted family members early the next morning as she became concerned that he had not returned home. His car was soon located near where his wife believed he would have been diving. The police were notified and a large air, sea and underwater search was conducted, without success. Police divers searched inside the wreck but found no trace of the victim. Three months later, the victim's badly decomposed body was found within a compartment inside the wreck; his weight belt was still in place but his mask, snorkel and one fin had been displaced.

Autopsy: At autopsy four months post incident, the body showed decompositional change including adipocere (decompositional breakdown of fatty acids in moist conditions) which made any interpretation of autopsy findings difficult. The cause of death was reported as unascertained. Possibilities include drowning due to entrapment or disorientation and a sudden natural event such as cardiac arrhythmia.
Toxicology: nil

Comments: How this victim died is unknown. The wreck is in an unstable state and prone to substantial silting, creating a high risk of entrapment, and is a prohibited site for divers and snorkellers. The victim likely became disoriented or trapped inside the wreck and drowned. It is impossible to diagnose drowning in the presence of significant decomposition.

Summary: Male, 39 y.o.; experienced snorkeller and scuba diver; snorkelling alone at night, most likely inside a wreck; body found four months later; unknown cause of death

BH 10/02

This 77 y.o. male overseas tourist was obese (BMI 32.8 $kg \cdot m^{-2}$), with a history of coronary bypass surgery eight years prior, hypertension, hypercholesterolaemia and prostatic hyperplasia. He was taking terazosin, simvastatin, furosemide, metoprolol, telmisartan and aspirin. He had visited a general practitioner one month earlier before travelling but it is unknown what, if any, advice was provided about snorkelling. His swimming and snorkelling ability and experience were unreported.

He and a friend were on a day trip to the Great Barrier Reef (GBR) on a large tourist vessel with 291 guests. The group was taken to a large pontoon anchored on the GBR, from which organised snorkelling was conducted. In addition to public announcements of the risks posed by various health conditions when snorkelling, there was a pre-snorkel briefing and guests were asked to declare relevant medical details from which a 'risk register' was created. The victim was not recorded on this register.

The victim was provided with a mask, snorkel, fins, stinger suit and a life vest. There was a moderate wind (15 knots), a surface chop and waves 0.5–1 m high, visibility was described as "*good*" and the water temperature was 28°C.

The friend reported that there was a strong current. On entering the water from the pontoon, the victim initially seemed to be calm and swam a short distance along a rope before being swamped by some swells and banging his head on a buoy. He appeared to panic and tried to climb onto his friend whilst signalling for help. The lookout saw this and a nearby tender was sent to assist. The tender driver asked the victim if he was okay, to which he replied "*No*". The driver reached out and held the victim's hand shortly before the victim became limp, unconscious and cyanotic. The tender driver and two assistants could not lift the victim into the tender due to his size, so they towed him the 15 metres to the pontoon. Basic life support (BLS) was soon commenced by trained staff, and oxygen (O_2) gear and an AED were requested. The AED battery was flat and needed to be replaced, delaying its use by several minutes. Oxygen supplementation was provided to ventilations. When the functional AED was attached, no shock was advised. BLS continued. When a guest, who was a doctor was found, he re-assessed the victim and provided adrenaline orally. BLS was continued for a total of about 50 minutes before the doctor pronounced the victim dead.

Autopsy: The autopsy was performed two days after death. External examination revealed a small bruise on the right forehead and a midline thoracotomy scar and vein harvesting scars consistent with coronary artery bypass surgery. There were no bites or stings. The heart was large, weighing 900 g (normal range (NR) 400 ± 69 g) and the pericardial sac was obliterated by fibrous adhesions from the previous surgery. The left ventricle was hypertrophied. The native coronary arteries showed severe occlusive atheroma, including distally, although the grafts were patent. The myocardium showed extensive fibrous scarring but no acute ischaemia. There was a stent in the left renal artery. The upper airways showed no pulmonary oedema and the right (R) and left (L) lungs weighed 600 g (NR 663 ± 217 g) and 569 g (NR 569 ± 221 g) respectively and appeared slightly over-expanded.
Toxicology: nil

Comments: It is likely that this victim's cardiac-related death was precipitated by the combination of immersion, exertion, aspiration and anxiety in a person predisposed to sudden cardiac death in a variety of circumstances, not specifically diving-related. This man had significant enlargement of the heart with progressive coronary atheroma despite bypass grafting. He was probably unfit for snorkelling. Relatively minor trauma can precipitate drowning especially in an unfit snorkeller in a current. The snorkelling was well-organised and well-supervised, and the tour operator's staff acted swiftly and appropriately. However, he was not on the risk register and the AED had a flat battery despite purported regular checks. It is fortunate that a spare, charged battery was available. This should serve as a warning to those who keep an AED in their workplace or at home to ensure it is operational at all times.

Summary: Male, 77 y.o.; history of open heart surgery and hypertension; swimming and snorkelling ability unknown; conditions choppy with current; panicked when swamped by waves; prompt rescue; BLS unsuccessful; cardiac death in a predisposed person

BH 10/03

This fit, active, 27 y.o. man swam five or six days per week and had no known medical conditions. He was a qualified scuba diver and keen spearfisherman. Dressed in board shorts and wearing a mask, snorkel and fins, he was apparently practicing extending his breath-hold time in the swimming pool of the residential complex where he lived. The pool was 20 m long and 1 to 1.5 m deep.

Another tenant entered the pool area and noticed the victim lying motionless and apparently unconscious on the pool bottom. He called for help and for an ambulance. Another tenant entered the water and, with difficulty, lifted the victim out of the pool, unconscious, apnoeic and cyanotic. When another person arrived, two-operator BLS was commenced and continued until paramedics arrived approximately 10 minutes later. There were stomach contents and frothy sputum in the victim's mouth. Advanced life support (ALS) was implemented and spontaneous circulation was restored after defibrillation. The victim was transported to hospital where a CT scan revealed diffuse cerebral oedema consistent with severe hypoxic brain injury. He died the next day.

Autopsy: The trachea and bronchi contained pink frothy fluid. The lungs were heavy (R = 1045 g, NR 651 ± 241 g; L = 959 g, NR 579 ± 201 g) and oedematous. The heart weighed 403 g (NR 370 ± 75 g) and was normal. The cause of death was drowning.
Toxicology: nil

Comments: This young man likely became unconscious from apnoeic hypoxia, with or without hyperventilation. Loss of consciousness in water often ends tragically, especially if there is no rescuer immediately available. Although there was a surveillance camera in the area, the monitor was in an unmanned security room, so was useless in this incident. It is important for the community (diving and general) to understand that apnoeic hypoxia can occur after extended breath holding even in shallow water. Drowning after loss of consciousness due to a cardiac arrhythmia such as long QT remains a possibility, although specific enquiries disclosed no family history of sudden death or syncope.

Summary: Male, 27 y.o.; fit and healthy; regular swimmer, qualified diver and keen spearfisherman; likely practising breath holding in pool alone; found on pool bottom; defibrillation restored spontaneous circulation; died next day; drowning

BH 10/04

This 24 y.o., male overseas tourist was in Australia on a working holiday. His medical history is unknown although his friend believed him to be healthy, but "*not a strong swimmer*". The victim and his friend were on a guided tour of a semi-tropical island. The group walked about 2.5 km to an inland freshwater lake. Wearing shorts, swim goggles and a snorkel, the victim snorkelled for a short time before returning to shore. He then re-entered the water and the friend noticed him snorkelling about five metres from the shore. The friend looked away for possibly 30 seconds and, when he turned back, his friend was nowhere in sight. Despite a short search by the group and others, the victim was not found. The group then walked back to the waiting tour guide, alerted him, and the police were contacted. Police divers found the victim's body the next day, after a presumed submersion time of 16 hours.

Autopsy: Autopsy, performed five days after death, revealed early decompositional changes and some post-mortem skin damage to the right thigh. Post-mortem X-rays revealed no bony damage. There was no pulmonary oedema in the upper airways (this feature may be lost with decomposition) and the R and L lungs, which weighted 720 g (NR 663 ± 239 g) and 600 g (NR 583 ± 216 g) respectively, appeared overexpanded and contained pulmonary oedema fluid. The heart weighed 395 g (NR 365 ±71 g) and was normal. The cause of death was given as drowning.
Toxicology: nil

Comments: The victim's disappearance was unwitnessed and apparently silent. Precisely how and why he died is unknown. He was reportedly not a good swimmer and likely an inexperienced snorkeller. He may have aspirated water through the snorkel or his nose and subsequently drowned.

Summary: Male, 24 y.o.; apparently healthy; poor swimmer; using goggles and snorkel; submerged silently; drowning

BH 10/05

This 55 y.o. man was severely obese with a BMI of 43.4 $kg \cdot m^{-2}$. He had a history of coronary artery bypass surgery (six years prior), diabetes, hypertension and hypercholesterolemia. His prescription medications included felodipine, irbesarton, hydrochlorothiazide, spironolactone, metformin hydrochloride, glimepiride, isosorbide mononitrate, atorvastatin, carbamazepine, aspirin and tadalafil. His swimming ability and snorkelling experience were not reported.

The victim went on a snorkel safari on the GBR. At the dive shop, he and others were briefed on medical issues and snorkelling and were asked to write any personal medical conditions on the relevant form. The victim declared hypertension but no other medical conditions. Because of his size, the tour operator assessed him as a potential risk and allocated him to a small group with a snorkel guide. The victim entered the water wearing a mask, snorkel, fins and a two-piece wetsuit without a weight belt. He also took a 'noodle' buoyancy aid. The water was described as calm and visibility was 3–6 metres. There was no current.

Shortly after entering the water, the victim rolled onto his back, holding onto the 'noodle'. The dive guide was soon with him. The victim complained that his wetsuit was too tight. The guide handed him a life ring to help support him while she removed his wetsuit top. He became distressed, and asked to return to the boat. The guide signalled to the tender driver and they began to swim towards the tender. When the tender arrived, the victim could not lift himself into it and became less responsive. The tender driver was unable to drag him aboard owing to his size. The guide used the 'noodle' to support the victim as they were towed about 50 m to the boat by the tender. Two staff dragged the now unconscious victim onto the boat and rolled him into the recovery position. Frothy sputum emerged from his mouth. He was soon apnoeic and cyanotic and the guide and captain began BLS, promptly adding supplemental O_2 via a resuscitation mask with oxygen inlet. Contrary to local regulations, there was no defibrillator available on the boat. The victim was taken to a nearby island, arriving about 55 minutes post incident. Two nurses attached a defibrillator but no shock was indicated. Adrenalin was administered but the victim failed to respond, so resuscitation efforts were soon abandoned.

Autopsy: This was conducted two days after death. The heart was significantly enlarged weighing 990 g (NR 400 ± 69 g). The pericardial sac was densely adherent to the heart due to previous coronary artery grafting. The native coronary arteries showed severe occlusive atheroma and there was severe stenosis distal to the vein grafts although the graft anastomoses were patent. There was left ventricular hypertrophy. Histology showed ischaemic fibrosis without acute infarction. The upper airways showed no pulmonary oedema. The R and L lungs weighed 760 g (NR 663 g +/- 217 g) and 800 g (NR 658 g +/- 257 g) respectively, and showed pulmonary oedema. The cause of death was given as ischaemic heart disease.
Toxicology: nil

Comments: Despite snorkelling on a tropical reef being on many people's 'bucket list', not everyone is compatible with snorkelling, whether they realise it or not. With his severe obesity, cardiac disease, diabetes and extensive list of medications, this man was at very high risk. Added to the effects of immersion and exertion, the tight wetsuit top may have compromised his breathing and increased his anxiety. Had he fully declared his medical conditions he may likely not have been permitted to snorkel. Although it may not have been a factor in this incident, there is sometimes tension between commercial interests, the fear of

upsetting a customer, and provision of sufficient information to deter those most at risk, by spelling out clearly what those risks may lead to. The snorkelling staff appear to have done well under difficult circumstances. However, it is important to have a pre-determined and well-practiced protocol for dealing with such eventualities, such as lifting an unconscious and/or large person into a tender.

Summary: Male, 55 y.o.; history of coronary artery bypass surgery, diabetes, hypertension in a very obese man; swimming and snorkelling ability unknown; calm conditions, no current; complained wetsuit too tight; became anxious; delayed rescue; BLS unsuccessful; cardiac death

BH 10/06

This 27 y.o., male tourist was on a working holiday in Australia. There was no information about his medical history or whether he was taking any medications. He was described by his friends as a "*weak swimmer at best*". He and four friends went swimming from a surf-prone beach with a coral reef nearby. At the time, waves were reported to be less than one metre, there was a light wind and the weather was warm but cloudy. The visibility was not reported, but a local diver later stated that it typically became poor in the afternoons, owing to a freshening wind and choppy surface conditions. The victim was wearing a mask, snorkel and board shorts.

After swimming together for a while, the friends headed further from shore while the victim remained closer to the beach. When the friends returned around 20 minutes later, there was no sign of him. They notified locals who contacted the police. The others did not return to the water to look for the friend as they considered themselves poor swimmers. There was little information provided due to language difficulties and the absence of a proper interpreter. Eventually the victim's body was found the next morning lying on the seabed about 10 m from shore, at a depth of 5–6 msw. He was still wearing his mask and snorkel.

Autopsy: Autopsy was limited to external examination. There was foamy fluid in the mouth consistent with drowning.
Toxicology: nil

Comments: It is inappropriate, although not uncommon, that this likely inexperienced snorkeller, a weak swimmer, was left to snorkel alone. An alert buddy could have raised the alarm earlier when the victim got into difficulties or disappeared. The depth of the snorkelling site was not stated, however, given that the body was found only 10 metres from shore at a depth of 5–6 msw, it is likely that the victim would have been unable to stand to rectify any problems even a very short distance from shore.

Summary: Male, 27 y.o.; medical history unknown; weak swimmer; not wearing fins; buddy separation; unwitnessed submersion; body found next day; drowning

BH 10/07

This 73 y.o. man, an interstate tourist holidaying on the GBR, had a history of atrial fibrillation and bilateral hip arthroplasty. His regular medications were candesartan, clexetil and low-dose aspirin. He was also taking cephalexin for a toe infection and had taken two hyoscine hydrobromide tablets that morning to prevent sea-sickness. His swimming experience was not reported. He had snorkelled before, but it seems he was relatively inexperienced as two days earlier he was reported to have "*swallowed a lot of water*".

He was on a vessel with six friends when they decided to snorkel. He was wearing a mask, snorkel and 'rashie' but no fins. The water temperature was 24^{o}C, surface conditions were not reported, though one friend stated that there was not much current. After about 20 minutes, the victim signalled to the tender driver that he wanted to be picked up. He said that he was OK but did not want to snorkel any longer. While trying to board the tender, made more difficult due to his large size, he became exhausted, short of breath and began coughing. He was unable to climb the ladder despite assistance from others. With his leg straddled over the tender, he was slowly towed 50 m to the main vessel.

After being helped aboard, he was sitting in a deck chair near the stern, looking very ill, and wheezing, exhausted and dyspnoeic. He fell out of the chair and was unable to get back into it. He became unconscious and was rolled onto his side so that "*muck*" could be cleared from his mouth. BLS was commenced and continued during the 15-minute boat ride to a nearby island, where two nurses from the island attached a defibrillator (it is not clear whether or not any shock was delivered) and continued resuscitative efforts, including administration of adrenaline, unsuccessfully.

Autopsy: The heart weighed 630 g (NR 400 ± 69 g) and showed left and right ventricular hypertrophy and some mitral valve prolapse. The left coronary artery and its branches were 60% occluded by atherosclerosis. Histology revealed mild fibrosis and some early ischaemia (eosinophilia of the myocytes). The R and L lungs weighed 1210 g, (NR 663 ± 217 g) and 990 g (NR 569 ± 217 g) respectively and were oedematous. The cause of death was given as drowning due to cardiac arrhythmia due to cardiomegaly.
Toxicology: nil

Comments: Given his reportedly poor snorkelling skills, he likely aspirated water. This, combined with the effects of immersion and exertion, could have triggered a fatal arrhythmia in a man with a history of atrial fibrillation. While generally neither 75% stenosis nor unstable plaques are regarded as significant lesions, the combination of 60% stenosis with left and right ventricular hypertrophy in the presence of atrial fibrillation is probably sufficient to account for an arrhythmia sufficient to cause drowning. However, immersion pulmonary oedema (IPO) cannot be ruled out as a possible differential diagnosis.

Summary: Male, 73 y.o.; history of atrial fibrillation; poor snorkel technique; possible aspiration; exertion trying to board tender; collapsed on board vessel; resuscitation unsuccessful; drowning? (cardiac related?, IPE related?)

BH 10/08

This 60 y.o. man was reported as "*fit for his age*", a highly experienced freediver and spearfisherman (being the recipient of several freediving awards and accolades), as well as an active and experienced recreational and commercial scuba diver. He was being treated for well-controlled bipolar affective disorder, depression, hypothyroidism and insomnia. Medications included olanzapine, lithium carbonate, thyroxine sodium and temazepam.

The victim went spearfishing with a friend, also an experienced spearfisherman, at a site familiar to both of them. Dressed in a wetsuit, weight belt and carrying mask, snorkel, fins and a line with float, the pair walked about 300–400 m from the car park down a rocky hill to reach the shoreline. The victim then returned to the car to retrieve a forgotten item. They entered the water from the rocky shore. The weather was warm, there was a light wind and the swell was around 1.5 metres high. The buddy later described the conditions as "*challenging, but not beyond* [their] *capabilities*". After swimming through a channel in the rocks they began spearfishing in 10–15 msw. After several dives, the victim reported that he was "*having trouble catching my breath and am going in*". The buddy said that he would follow soon and, after several more dives, he also swam towards the agreed exit point against a current. When nearby, the buddy noticed the victim's fins on a rock and saw the victim floating face-up at the surface near the rocky shoreline. When the buddy reached him, the victim was unconscious, cyanotic and apparently apnoeic and was not wearing his weight belt.

The buddy dragged his friend out of the water, rolled him onto his side and noticed some bile and water draining from his mouth. He began BLS, assisted by bystanders. After every few cycles, the airway needed to be cleared of water and stomach contents. An ambulance was called and volunteer paramedics arrived 35 minutes later, continuing resuscitation efforts for another 30 minutes before ceasing. A defibrillator was attached but it is unclear if any shock was delivered.

The friend later reported that on their last dive outing approximately five weeks earlier, which involved strenuous breath-hold dives over an extended period, the victim "*ran out of steam*" while swimming back to shore. The buddy noted that his friend looked unwell and, on palpating his pulse believed it to be very fast. He advised the victim to see a doctor but this advice went unheeded. Apparently he had been scuba diving in the interim.

Autopsy: This was conducted two days after death and there were early decompositional changes. The heart weighed 456 g (NR 400 g ± 69 g) and the left and right ventricles were slightly dilated. The coronary arteries were between 50 and 70% narrowed by atherosclerosis. The upper airways were clear the R and L lungs weighed 424 g (NR 663 g ± 217 g) and 340 g (NR 569 g ± 221g) respectively. The cause of death given was consistent with coronary atherosclerosis.
Toxicology: citalopram, temazepam and olanzapine detected; measurement of lithium is usually performed on serum rather than whole blood and obtaining serum at post mortem is difficult if there has been any post-mortem delay.

Comments: This victim apparently ignored warning signs of increasing dyspnoea while diving and consequently the opportunity to investigate his cardiac function. Although his coronary atherosclerosis was marginal, combined with exercise, breath holding and possible drug effects it likely sufficed to cause the cardiac event. This was possibly further exacerbated by the need to return to shore against a current. It is always good practice to accompany a buddy out of the water, especially if unwell. It is unlikely that this affected the outcome in this instance, given the remote location and the delay in availability of a defibrillator.

Summary: Male, 60 y.o.; history of bipolar affective disorder, hypothyroidism, insomnia and depression; highly experienced breath-hold and scuba diver; challenging conditions; previous episode of breathlessness while spearfishing; became unwell and swam to shore alone; found unconscious by buddy; BLS unsuccessful; moderate coronary atherosclerosis; cardiac-related death; ?immersion pulmonary oedema

BH 10/09

This 30 y.o. man was an experienced and apparently competent breath-hold diver and a member of a spearfishing club. His family reported that he appeared to be healthy and on no medical treatment. He went spearfishing with two others, one of whom had dived with him previously. They dived from a small boat. The victim was wearing mask, snorkel, fins, full-length wetsuit, weight belt; and carried a speargun, float and a Shark Shield.

The weather was warm, the water temperature 22^{O}C, the current was described as "*light – less than 1 knot*" and visibility was at least 15 metres. The surface conditions were not reported. They initially anchored the boat in a depth of 14–17 msw and dived there for about an hour. When they reboarded the boat, the victim seemed to be fine but mentioned that it had been a bit deep for him. They subsequently moved and anchored in water about 10 msw deep. After snorkelling with the others for about 10 to 15 minutes, the victim swam off by himself and snorkelled nearer to the boat. When one of the divers returned briefly to

the boat to offload a fish, he passed by the victim and called out to him. The victim was swimming steadily, appearing to be concentrating on something below and failed to respond but appeared to be fine.

When his companions returned to the boat about 30 to 40 minutes later, the victim was found nearby, floating vertically just below the surface, unconscious, with his weight belt in place. His speargun was missing. His belt was ditched and he was dragged aboard the boat, unconscious and apnoeic with a grey appearance. When checking, one companion initially thought that he felt a faint pulse. He was placed on his side and a large amount of water flowed from his mouth and nose. One companion commenced BLS while the other made a Mayday call on the boat radio. It was necessary to place the victim onto his side periodically to drain large amounts of water and blood-stained froth. A large vessel came to assist, the victim was transferred aboard and BLS was continued en route to the harbour. On arrival, they were met by an ambulance crew who continued resuscitation efforts. An AED was attached and, although initially no shock was advised (i.e., the victim was likely in asystole), after a period one shock was advised and given, albeit unsuccessfully. The victim was declared dead shortly afterwards.

Autopsy: The autopsy was performed three days after death. The heart weighed 322 g (NR 370 ± 75 g). The coronary arteries showed a 90% narrowing at the midpoint of the left anterior descending (LAD) coronary artery with 40% occlusion of the right coronary artery and 50% stenosis of the left main and left circumflex coronary arteries. The R and L lungs weighed 764 g (NR 651 ± 214 g) and 700 g (NR 579 ± 201 g) respectively. There was frothy fluid in the oropharynx, trachea and bronchi and the lungs were congested and oedematous. The cause of death was given as secondary drowning due to ischaemic heart disease.
Toxicology: nil

Comments: The police report suggested that this victim might have drowned as a result of 'shallow water blackout'. There was no mention in any of the reports whether the victim practiced pre-dive hyperventilation. Given the evidence of significant coronary atherosclerosis, the victim may have suffered an arrhythmia, become unconscious and subsequently drowned. The absence of a nearby and vigilant buddy made survival highly unlikely.

Summary: Male, 30 y.o.; apparently healthy; experienced spearfisherman; separation; found unconscious in water; BLS unsuccessful; significant coronary atherosclerosis; drowning (likely cardiac-related)

BH 10/10

This 64 y.o. woman was an overseas tourist with a history of dyslipidaemia. She was reported to be a competent swimmer but she had no prior snorkelling experience. The victim, her husband and daughter were among 10 tourists on a commercial snorkel tour on a charter boat. Prior to departure, she signed a liability waiver that confirmed that she could swim and was aware of the risks on the planned activity. Although she spoke no English, her daughter, a fluent English-speaker, translated it for her. She was issued with a mask, snorkel, fins and wetsuit which were dry-tested for correct fit.

Once at the site, the victim entered the water with nine other snorkellers and a guide to snorkel with some manta rays. The depth and visibility were about 15 to 20 m, and the water was described as calm with no current or surge and a temperature of 24^{o}C. After a while, her husband had problems with his mask. Their daughter accompanied him to the boat and was told to come aboard as it was time for another group to enter the water. The first group was then recalled. As the rest of her group was re-boarding, the victim was seen snorkelling without obvious distress, with her arms by her side and finning some 10 m from the boat. A crew member entered the water to help her but before he reached her she went limp. He rolled her over and noticed that she was unconscious with froth flowing from her mouth. Another crew member jumped in and helped to tow the victim back to the vessel.

She was brought aboard and placed in the recovery position as she was vomiting. Her airway was cleared and she was assessed as apnoeic. Shortly afterwards, when another passenger, a nurse, was recalled to the boat, he again rolled the victim into the recovery position to drain water and froth from her airway before beginning BLS, assisted by another passenger, a doctor. Oxygen equipment was provided but proved useless as the only delivery device was a non-rebreather mask which is unsuitable for use with a non-breathing victim. BLS was continued en route to the wharf and maintained for a short time by an attending ambulance crew. They attached a defibrillator and no shock was advised (asystole). Resuscitation efforts were abandoned about one hour after being commenced because of the lack of response.

Autopsy: At the request of the family, only an external autopsy was conducted and, as a result, the cause of death was recorded as "*unascertainable*".
Toxicology: nil

Comments: Given the lack of an internal autopsy, it is impossible to ascertain whether a cardiac or other medical condition played any part in this incident. However, given her lack of snorkelling experience, and her insignificant medical history, it is quite possible that drowning was the primary event. The available O_2 equipment, while being suitable for use with many spontaneously breathing victims, was unsuitable for oxygen-supplemented ventilation. The investigating coroner recommended that dive charter vessels carry a positive pressure O_2 system.

Summary: Female; 64 y.o.; history of dyslipidaemia; competent swimmer; first snorkel experience; brief

separation from group; sudden unconsciousness; O_2 equipment unusable; BLS unsuccessful; disabling agent and cause of death unknown

BH 10/11

This fit, 31 y.o. man was an experienced breath-hold diver and spearfisherman. He had recently attended an extended apnoea training programme to enable him to dive deeper and longer. He and six friends, also experienced, set out to spearfish from two boats. The weather and sea conditions were not reported. After some 'warm-up' diving at depths of 11–13 msw, they moved to a new site, anchoring the boats on a wreck. The depth ranged from 23 msw at the top of the wreck to 27–30 msw to the sand. The water temperature was 29^{O}C and visibility varied from around 7 m at the surface to 2–4 m on the wreck.

The victim and five of his friends entered the water while one of the group remained on board as a lookout. The victim was wearing a mask, snorkel, fins, a 1.5 mm thick wetsuit with hood, weight belt with 4.5 kg and was carrying a speargun. When they entered the water there was no current. They dived for a while using a 'one-down-one-up' protocol for greater safety. After a while one of the group's spear became stuck in the wreck and several divers tried unsuccessfully to retrieve it. The victim offered to get it and was reported to be seen "*breathing up*" on the surface before descending, carrying a friend's speargun. After about 30 seconds, the owner of the stuck spear felt the tension on its attached cord release, indicating that the victim had freed it. However, he became concerned after about another 30 seconds when the victim failed to surface. The buddies then performed many dives in an unsuccessful attempt to find their friend, hampered by the depth, increasing current and poor visibility. His speargun was found floating on the surface 100–200 m from the wreck about 45 minutes after he disappeared. The spear had been discharged and was later found under the wreck. The two-metre cord that had attached it to the speargun had been sheared, which, according to the police had likely resulted from rubbing against the wreck.

Almost four hours later, about five minutes into their search, police divers located his body lying on his back about three metres from the wreck at a depth of 30 msw. He was brought to the surface and declared dead by a doctor who had arrived with one of the search teams.

Autopsy: The autopsy was done three days after death. There were petechiae on the orbital conjunctiva and on the eyelids (possibly from mask squeeze). There was white frothy fluid in the mouth. The R and L lungs weighed 800 g (NR 651 ± 241 g) and 740 g (NR 579 g ± 201 g) respectively and were unremarkable apart from some congestion. The heart weighed 270 g (NR 370 g ± 75 g) and was normal without significant coronary atheroma. The cause of death was given as drowning.
Toxicology: nil

Comments: This drowning resulted from apnoeic hypoxia either from pre-dive hyperventilation, or possibly, after freeing the initial spear from the wreck, the victim may have speared a fish using the gun he was carrying and the cord from his spear snagged on the wreck delaying the ascent and causing unconsciousness. Being negatively-buoyant he sank to the bottom. Whatever the actual sequence of events leading to this death, the practice of pre-dive hyperventilation is known to be dangerous. The combination of pre-dive hyperventilation, depth, extended breath holding and exertion was a potentially lethal mixture.

Summary: Male, 31 y.o; healthy and fit; experienced breath-hold diver and spearfisherman; deep dive to retrieve friend's spear; breath-hold search made difficult by depth, poor visibility and current; BLS not attempted; drowning (apnoeic hypoxia post hyperventilation or entrapment?)

BH 10/12

This 28 y.o., male overseas tourist was an experienced spearfisherman who had qualified as an Open Water Diver in his home country some four months earlier. He was on a large live-aboard vessel on the GBR and had arranged to do some general work on the boat for part of the four-day trip in exchange for a discount on accommodation and diving. The victim did not declare any medical conditions on a pre-dive/snorkel medical questionnaire. Another casual worker/tourist who had snorkelled with him earlier on during the trip reported that he could hold his breath underwater for 60–90 seconds and tended to snorkel alone.

On this day, the victim had done an early-morning scuba dive from another vessel. No details of this are available but nothing untoward was reported. He was said to have been of "*normal disposition*" afterwards and had not consumed any alcohol. He then transferred back to the large vessel which was carrying approximately 80 passengers and 11 crew. After a briefing, the victim entered the water with around 30 others. He was wearing mask, snorkel and fins, a stinger vest and bathers. He was not wearing a wetsuit despite this being specified as a requirement by the operator. The weather and sea conditions were described as "*good*" with a slight breeze and current, and visibility of 10 metres. The skipper of the vessel was reported to have been acting as the sole look-out.

When a pre-departure head-count was taken about two hours later, the victim was missing. After an on-board search, some tenders and snorkellers entered the water and, after about 20 minutes, found the victim's body close to where he had last been seen by a witness, and reportedly by the skipper, outside the designated snorkelling area. He was lying on the seabed. His dive watch indicated that his last submersion was to a maximum depth of 16 msw for 90 minutes.

The victim was brought on board, where some crew who were qualified in first aid and O_2 provision began BLS. They were soon assisted by a passenger who was a doctor

who noted that the victim was apnoeic and cyanotic with fixed dilated pupils. There was bloodstained, frothy sputum coming from his mouth and nose. It was necessary to roll the victim onto his side periodically to clear his airway. The doctor reported that the boat's bag-valve-mask O_2 unit was not functional owing to a missing part. BLS was continued until a rescue helicopter arrived, when he was found to be in asystole and ALS was commenced, including endotracheal intubation, intravenous cannulation and administration of adrenalin. This was abandoned after 20 minutes when the victim failed to respond.

The skipper was generally uncooperative with the investigation conducted by the workplace authority and with the subsequent coronial enquiry. He also discouraged his crew from assisting. As a result, important information may not have become available.

Autopsy: The heart weighed 365 g (NR 400 g ± 69 g). The LAD showed a greater than 75% stenosis proximally with focal scarring and there was equivocal left ventricular hypertrophy (14–16 mm). The R and L lungs were heavy, weighing 1027 g (NR 663 g ± 217 g) and 973 g (NR 569 g ± 221 g) respectively and were oedematous. The cause of death was given as secondary drowning due to a cardiac arrhythmia and ischaemic heart disease.
Toxicology: nil

Comments: It is likely that the victim drowned as a result of a cardiac arrhythmia, although blackout subsequent to apnoeic hypoxia is also possible. It was reported that the skipper was acting as the sole lookout most of the time. It is difficult, often impossible, for a single lookout to adequately monitor such a large group of snorkellers. One guest reported that she developed a cramp and raised her arm for assistance, as advised to do, but received no response or assistance from the staff of the vessel. The skipper stated that he and another crew member had seen the victim swimming alone outside the designated snorkelling area, but had not followed up on this until the victim was found to be missing, possibly 90 minutes later.

It is generally accepted that appropriate O_2 equipment and at least one trained provider should be available where diving or snorkelling activities are conducted. This is an industry standard of care, especially in Queensland where it is required by regulation. Although it would have made no difference to the outcome in this case, it can be invaluable. It is unacceptable for O_2 equipment to not be fully functional. If the skipper and crew were aware of the state of the O_2 equipment before setting out, then this is also unacceptable.

Although the operator had written instructions that all divers were required to wear wetsuits, this was not adhered to. Wearing a wetsuit provides some added buoyancy (as well as some protection from stingers, if present). In practice, many snorkellers are unwilling to wear a wetsuit in warmer waters, and it can sometimes be unreasonable to try to force the issue. In a case such as this, where a person becomes unconscious in the water, the additional buoyancy from a wetsuit may cause the body to float to the surface where it can be more quickly and easily seen. This is obviously dependent on whether or not the diver is wearing weights and, if so, how much, as well as on their natural buoyancy.

It is very concerning to note that the operator obstructed the investigation, and encouraged his crew to do so as well. This behaviour was displayed again by the same operator after a subsequent death of another snorkeller from the same vessel. Learning as much as possible from each such tragedy allows trends and deficiencies to be identified and appropriate management and preventive strategies to be established, or reinforced. In this case, there appear to have been breaches of guidelines and regulations, adherence to which might possibly have altered the outcome of this incident.

Summary: Male, 28 y.o; significant coronary stenosis; experienced breath-hold diver; snorkelling alone away from large group; ineffective lookout; submerged for approximately 90 minutes; BLS unsuccessful; likely cardiac-related; operator unco-operative with investigations

Scuba diving fatalities

SC 10/01

Although still obese (BMI 32.5 $kg \cdot m^{-2}$), this 46 y.o. woman had lost 40 kg since having gastric banding surgery five years earlier. She was described as being in good health since losing weight and led a reasonably active lifestyle. She was taking perindopril for hypertension but the dosage had been reduced and her hypertension had become better controlled. Her medical history also included past glomerulonephritis (non-IgA mesangio-profilerative type) and a cholecystectomy. She had begun diver training 17 months earlier but withdrew shortly into it. At that time it is thought that she had undergone a diving medical examination although there is no evidence of this in the coronial documents. One year later, she recommenced training and successfully completed this several months before this incident. She had completed several post-certification dives.

On this day, the victim and her buddy, a considerably more experienced diver, set out on a shore dive in a small harbour, largely sheltered although exposed to the ocean near the breakwater. This was their fifth dive together and the buddy stated that victim appeared anxious. The weather was described as cool but sunny, there was a slight chop on the surface of the sheltered waters although it was rougher beyond the shelter of the rocks, where there was also some surge. The water temperature was 21^{O}C. The victim was wearing a mask, snorkel and fins, rented 5-mm wetsuit without hood, weight belt with about 8 kg of weights, buoyancy compensation device (BCD) and a regulator with 'octopus' attached to a hired 10.5 L steel cylinder, filled to over 200 bar.

Table 2

Summary of scuba and surface-supply diving-related fatalities in Australian waters in 2010; BNS – buddy not separated; BSB – buddy separated before problem; BSD – buddy separated during problem; GNS – group not separated; + sufficient air (to surface safely); ++ 1/4–1/2 full tank; +++ > 50% full; nad – nothing abnormal discovered; n/a – not applicable; n/i – not inflated; n/s – not stated; CAGE – cerebral arterial gas embolism; IPE – immersion pulmonary oedema; mw – metres' water

SC	Age	Sex	Height (cm)	Weight (kg)	BMI ($kg \cdot m^{-2}$)	Training	Experience	Dive group	Dive purpose	Depth (mw)	Incident (mw)	Weight belt	Wts (kg)	BCD	Residual air	Equipment test	Disabling injury
10/01	46	F	156	79	32.5	Yes	Some	BNS	Recreation	14	surface	Off	8	n/s	+	Some issues	Asphyxia
10/02	46	F	170	63	21.8	Yes	Some	solo	Recreation	1–5	n/s	On	5.5	n/s	nil	Poor condition	Asphyxia
10/03	51	M	–	–	–	Yes	Experienced	BSD	Recreation	45	35	n/s	n/s	n/s	nil	nad	Asphyxia
10/04	48	M	173	102	34.1	Yes	Experienced	BNS	Hunting	6	surface	n/s	n/s	inflated	+++	nad	Cardiac incident
10/05	31	F	167	64	22.9	Yes	Some	BSB	Recreation	4	n/s	On	9	n/s	+++	Some issues	Asphyxia
10/06	49	F	160	105	41.0	Training	nil	GNS	Training	2.5	surface	n/s	17	inflated	+++	n/s	IPE
RB																	
10/01	49	M	181	111	33.9	Yes	Experienced	BSD	Recreation	36	36	n/s	11	n/s	+++	Some issues	Asphyxia
SS																	
10/01	48	M	174	112	36.9	n/s	Experienced	BSB	Hunting	12	surface	On	n/s	not worn	+++	Poor condition	CAGE

They entered the water from the boat ramp and swam underwater towards a rock wall. Visibility was good initially but deteriorated nearer to the wall. The victim indicated that her dive computer was not working although at this time she seemed to be fine, swimming in sheltered water and stopping to look at marine life. On reaching the rock wall, the buddy checked and noted that the victim had 140 bar of remaining air (compared to her own 170 bar). She seemed to be fine so the buddy lead her around the rock wall after which they descended to their maximum depth of 14 msw and then swam along the outside of the rock wall. About 40 minutes into the dive, the victim grabbed her buddy's arm and showed her gauge, which now read 30 bar. After checking her own gauge (which read 120 bar), the buddy handed her 'octopus' to the victim and they swam along together for about five minutes at a depth of approximately 7 msw before the victim grabbed the buddy and indicated, insistently, that she wanted to surface.

On reaching the surface after what was described as a slow, controlled ascent, the victim was very anxious, gasping for air and unable to speak, only communicating by nodding or shaking her head. They were now about 100 m from shore and, as they were being swamped by waves, the buddy suggested they re-descend but the victim was unable to use either her regulator or snorkel, even with her buddy's assistance. When the victim continued to shake her head and struggle, the buddy told her to roll onto her back and began to tow her towards a moored boat. While towing, the buddy turned to check on the victim a couple of times, and saw her continuing to gasp for air. After about five minutes she observed that the victim was unconscious and then waved her arm and called for help.

Within about two minutes, a pair of swimmers arrived at the scene. One (a nurse) described the victim as unconscious and cyanotic with fixed dilated pupils. There was froth coming from her mouth and nose. The nurse gave three rescue breaths while her companion supported the victim. A short time later, a boat arrived and, after the victim's gear was removed, she was pulled into it. The swimmers had boarded as well and performed BLS while the boat motored towards shore, a trip estimated to have taken eight minutes. Waiting paramedics boarded the boat on arrival and continued resuscitative efforts. When attached, a defibrillator indicated fine VF/asystole. Given that this was a non-shockable rhythm according to ambulance protocol, and that the victim was lying on the wet floor of an aluminium boat, no shock was given. Another ambulance with intensive care paramedics arrived. Intravenous cannulation was unsuccessful but the victim was intubated, transferred into the ambulance and ALS (asystole protocol) was performed en route to the hospital. The victim failed to respond.

When later tested by the police, the remaining air conformed to relevant purity standards, the regulator was functional, but there was a substantial leak where the scuba-feed hose attached to the BCD inflator/deflator mechanism. This could explain the victim's high air usage (the BCD still held air so buoyancy was not affected), although the buddy did not notice a leak at the time and stated that the mechanism might well have been damaged while she was towing the victim.

Autopsy: Post-mortem radiology revealed no obvious gas and there was no surgical emphysema. The actual dive profile is not known because the dive computer had malfunctioned. The heart weighed 290 g (NR 362 g ± 77 g), with normal left and right ventricular wall thicknesses (13 mm and 3 mm respectively). There was no coronary atherosclerosis. Some myocyte hypertrophy was noted on histology of the heart. The R and L lungs weighed 420 g (NR 561 g ± 256 g) and 370 g (NR 491 g ± 204 g) respectively, there was moderate oedema and histology showed changes of *emphysema aquosum*. The cause of death was given as drowning.
Toxicology: 2% carboxyhaemoglobin (non-toxic level, consistent with smoking).

Comments: It appears that the victim became anxious during the latter part of the dive, in deeper water, poorer visibility, some surge and her air was getting low. The buddy reported that on previous dives the victim had used much more air than she did, unsurprising given the differing experience. The buddy believed that she would have noticed a significant leak from the victim's equipment. There are at least two possible explanations for the victim becoming so distressed and dyspnoeic on surfacing. Firstly, it may have been anxiety from the dive, concern at being so low on air and distant from the shore. Secondly, as mentioned by the pathologist, she may have suffered from a cardiac arrhythmia and subsequently became dyspnoeic and unconscious. There is no compelling pathological evidence to support this (the evidence of left ventricular hypertrophy is minimal with normal heart weight and normal left ventricular thickness). In any case, an inexperienced, panicking and breathless diver surfacing into rough conditions with waves washing over her created a potent scenario for drowning. The efforts of the buddy and other rescuers were impressive and appropriate, but unfortunately in vain.

Summary: Female, 46 y.o.; history of hypertension, gastric band surgery, glomerulonephritis and cholecystectomy; swimming ability unreported; recently certified; high air consumption (possible faulty equipment); octopus breathing; anxiety; rough conditions; drowning

SC 10/02

The victim, a 46 y.o. woman with an unremarkable medical history, led an active and healthy lifestyle. She was a strong swimmer and participated in a variety of aquatic activities including surfing, windsurfing and kite surfing. She certified as a diver 27 years earlier and had done more than 86 dives, although had not dived for the past 11 years. She had two sets of her own regulators – one was old and familiar, the other newer and yet unused. A friend had lent the victim and her husband a full cylinder which her husband had tried out some three months earlier, leaving it with a residual pressure thought to be about 150 bar.

The victim rode a bicycle to the beach towing a trailer carrying her dive equipment. The dive site was off a sandy beach in a protected bay with surrounding reef and a small island about 300 m offshore. On arrival, she had dressed into a 3-mm wetsuit, weight belt with 5.5 kg of weights, mask, snorkel, fins, BCD and used her old scuba regulator and a 10 L steel tank. She entered the water alone. Conditions at the time were reported to have been a light wind, "*quite choppy*", a swell of less than one metre inside the reef, a depth of 1–5 msw, a slight current, a water temperature of 18^{o}C and visibility likely to be less than 2 m. She was reported missing approximately 4 to 6 hours later when her bicycle was again noticed where it was left.

Police divers located her body two days later at a depth of less than 1 msw, 20 m from shore and about 150 m from where she had entered. Most of her equipment was still in place, including her weight belt, although her regulator was out of her mouth and her mask was slightly displaced (although marks on her forehead indicated that this was recent) and it contained some "*pink fluid*". The cylinder was empty.

When later tested by police, the equipment was found to be in poor condition. The cylinder contained some seawater which was tested and believed likely to have been introduced post mortem. All components of the regulator were in poor condition with sediment deposits, corrosion and distorted o-rings, among other defects. However, despite this, the first stage was mainly functional. The low pressure hose had some obvious weaknesses and was easily bent and, when this occurred, the air supply to the demand valve was completely cut off. The demand valve which, although reported to have a slight 'free-flow', was found to be difficult to breathe from (in the flow setting found) and allowed water ingress in inverted positions. The BCD inflator/deflator mechanism was also faulty, leaking air into the BCD indicating that the wearer would need to dump air regularly to maintain their position in the water. Her contents gauge was found to be reasonably accurate.

Autopsy: The autopsy was performed four days after death. A post-mortem CT scan showed a fluid column in the upper airway and fluid in the lungs. There was no significant intravascular gas. There were no significant injuries apart from some minor and irrelevant abrasions on the face, and evidence of mask squeeze and haemorrhage in the middle ear which probably occurred on descent. Some gas was noted in the mediastinum but this may be due to early

decompositional change (the body was not found for two days and there were early decompositional changes to the skin). The heart weighed 246 g (NR 308 g ± 68 g) and was normal with no coronary atherosclerosis. The R and L lungs weighed 604 g (NR 547 g ± 256 g) and 612 g (NR 491 ± 204 g) respectively and appeared over-distended and covered most of the mediastinum. There was sand in the trachea and frothy fluid on the cut surface of the lung. The stomach contained 382 g of partly digested food. The cause of death was given as drowning.
Toxicology: nil

Comments: This diver, who was considered to be a "*risk-taker*", had not dived for 11 years and entered the water alone with poorly-functioning equipment, a wetsuit that would not have provided adequate thermal protection for an extended period in water of that temperature, and the amount of weight that she had previously used with a thicker wetsuit. It is likely that the dive was difficult given the stressors of poor visibility, poorly functioning regulator, probable buoyancy control issues due to overweighting and poor BCD function and becoming cold, among other possible factors.

The presence of bilateral middle ear barotrauma at autopsy can be an indicator of an unconscious descent as the victim doesn't have the ability to equalise. This may indicate that the victim reached the surface before becoming unconscious and sank owing to being overweighted. This is consistent with the presence of water in the empty cylinder, a situation that could occur if the cylinder was empty on the surface and then returned to depth. There are several possible scenarios which could have led to her demise, all of them somewhat speculative. However, it seems likely that the victim had ascended to the surface and run out of air before subsequently drowning and sinking back to the seafloor.

Summary: Female, 46 y.o.; no significant medical history; apparently fit and healthy; strong swimmer and experienced diver but not for a decade; overweighted; solo dive; tank empty; poorly maintained equipment; drowning

SC 10/03

The victim was a 51 y.o., experienced, male cave diver. Although the coronial documents for this fatality were not made available, reliable information was obtained through a variety of other sources including police reports and witness statements. The victim had no known medical problems and appeared healthy. He had been diving for approximately 20 years and had performed many freshwater cave dives with a regular cave diving buddy over the past nine years. This buddy described him as a calm and safe diver. The victim normally dived with twin back-mounted cylinders of air. On this particular weekend he was trying a side mount diving system for the first time. He was also using new regulators and a new drysuit, although he was an experienced drysuit diver. He and his regular cave diving buddy completed two cave dives the previous day in relatively restrictive sites without incident, aside from the victim falling and injuring his toe. That night the victim had an early night after a pleasant dinner, with no alcohol being consumed.

The next day the pair prepared to dive in a deeper, less restrictive cave. The visibility was very clear and fixed lines were already present in the site to orientate divers. His buddy stated that the victim was not himself. In fact, he had been somewhat withdrawn and unhappy all weekend. He was distracted, disorganised and required several reminders about usually routine aspects of dive preparation. They placed a cylinder with a decompression mixture of nitrox80 with two second stage regulators on a decompression shotline at 9 metres' fresh water (mfw) depth prior to the dive. The divers both utilised twin cylinders of air (the victim in his new side-mount configuration) and each diver also carried an additional 'travel' gas cylinder containing nitrox (33% oxygen in the victim's case and 44% with the buddy).

The descent proceeded uneventfully, except that the buddy stated that the victim looked "*clumsy*" in the water. After approximately seven to eight minutes, the pair dropped their travel gas cylinders at 35 mfw depth (a dive computer, set for air, was attached to the victim's stage cylinder). The victim needed some assistance with this task. From this point, the victim did not respond swiftly to buddy signals and was already possibly suffering the effects of narcosis. A degree of buddy separation then followed with the buddy dropping down to 45 mfw before being joined by the victim. The buddy descended to 52.3 mfw for three minutes before noting the victim back at 45 mfw, inverted in his drysuit. The buddy assisted righting the victim who indicated he wanted to ascend. The buddy led the pair back to the travel cylinders but again the victim fell behind. He had stopped and was motionless and facing back into the cave. A light signal attracted his attention, and the victim swam out past the buddy but failed to stop and collect his travel cylinder. He became inverted again but, on this occasion, when the buddy tried to assist, the victim appeared to panic and pulled the buddy's mask off. The buddy performed a barely controlled ascent along the steep roof of the cave, closely avoiding drowning himself. After recomposing himself on the surface, he descended to the nitrox 80% decompression cylinder to do his decompression. He looked down to see the victim swimming along the cave floor at around 35 mfw before he became inverted for the last time and stopped breathing.

The buddy completed his decompression obligation and surfaced to alert the authorities of his friend's demise. When police divers recovered the victim's body the next day, they found him significantly entangled in the guideline and his side-mount cylinders were completely empty. When tested, all other equipment was found to be in good working order.

Autopsy: All findings were consistent with drowning. There were no other contributory findings and the toxicology screen was clear.

Comments: The story as presented contains several elements that are difficult to reconcile. These include the breaking of several basic cave diving rules concerning depth and gas usage. In particular, the rule of thirds (i.e., use one third of breathing gas going in, one third coming out and leaving one third in reserve), the placement of a dive computer on a stage cylinder, and the depositing of the stage cylinders at a depth beyond the maximum safe breathable depth of the gases within them. Given that this is a cavern where direct access to the surface is almost possible from the site of the victim's demise, it seems strange that, if he was critically low on gas, he would not have simply ascended as did his buddy. Indeed, the analysis of the victim's dive computer indicates several ascents including one of up to 14 mfw before he finally died. Although the buddy did not report any signs of the victim struggling or trying to free himself from the guideline in the final moments before he stopped moving, entanglement was clearly documented during the recovery. Unfortunately, further investigation of these events was not pursued at the request of the family so no further understanding or lessons can be gleaned from this event. It was suggested that the victim had not dived (at least in caves) for many months and he was using new equipment, some being unfamiliar to him. Although he had dived without incident the day before, this was in a more confined, shallower area where buoyancy was more easily controlled and he would have been less affected by narcosis. It is likely that the interaction of his unexplained poor mental state pre-dive and profound narcosis contributed to the sequence of events that led to this tragedy.

Summary: Male, 51 y.o.; apparently healthy; experienced; unfamiliar equipment; unexplained poor mental state pre-dive; narcosis; inversion and entanglement; out of air; drowning

SC 10/04

This 48 y.o. man was described as overweight with a history of diabetes and cardiac disease and had undergone angioplasty approximately 11 months earlier. He had been prescribed aspirin and clopidogrel but was non-compliant with his medication. He was a keen and regular diver although he had not dived since his angioplasty. It is unknown whether he sought advice about his fitness to dive post surgery. He had spent a few days fishing with friends and had complained of chest pain and dyspnoea several days before going diving.

On this day, the victim and his buddy, with whom he dived six or seven times a year, went diving from a small boat. One friend remained on board and other friends were on another boat nearby. Earlier that day, the victim had complained of breathing difficulty and chest pain. He appeared to be stressed while gearing up. He was wearing mask, snorkel and fins, a 3-mm wetsuit with hood, gloves and bootees; BCD and scuba gear; and was carrying a knife; he was also presumably wearing weights but this was not mentioned. The plan was to catch some crayfish and abalone. There was no description of the conditions.

The victim and his buddy entered the water and descended 5–6 msw to the seabed. They collected some abalone. After about 10 minutes, the victim indicated that he was having difficulty breathing. Initially he signalled that he did not wish to ascend but soon changed his mind and signalled to his buddy to ascend, before inflating his own BCD in doing so. On the surface, he was distressed and told his buddy that his chest hurt and he could not breathe. The boats came alongside, the buddy removed the victim's BCD and tank and the victim was dragged onto one of the boats. He was described as "*in and out of consciousness*" and there was froth coming from his mouth. He was placed in the recovery position to try to assist his breathing. The emergency services were called during the 20-minute boat ride to land, by which time the victim was unresponsive and apnoeic. One of his companions began BLS for a short time before paramedics arrived, who found him to be in asystole and implemented ALS for about 25 minutes before pronouncing him dead.

His equipment was found to be functional. The 'cracking pressure' of his primary second stage was found to be relatively high and the subsequent flow was also high. The secondary demand valve worked effectively and was believed to have been likely what the victim had been breathing from. There was 140 bar of air remaining in his tank and the air met relevant purity standards.

Autopsy: The autopsy was performed four days after death. X-rays showed air in the great vessels and heart, some or all of which could be due to decomposition and or post-mortem decompression artefact. The heart weighed 499 g (NR 400 ± 69 g) and was heavy. The right coronary artery was dominant, and showed a proximal 50–70% stenosis. The left main coronary artery had a 40% stenosis, whilst the LAD had an 85% stenosis with a stent which contained thrombus. The left circumflex coronary artery had a 75% stenosis, with a distal stent. The R and L lungs weighed 732 g (NR 663 ± 217 g) and 644 g (NR 569 g ± 221 g) respectively and were congested with marked oedema. Vitreous glucose was 7.6 $mmol \cdot L^{-1}$ which is high (normal upper limit < 5.7 $mmol \cdot L^{-1}$) and his HbA1c was 14% which also is high and suggests his diabetic control was poor. The cause of death was given as ischaemic heart disease.
Toxicology: nil

Comments: This man had obvious cardiac-related symptoms, was non-compliant with medication and grossly unfit for diving. This death could well have occurred during terrestrial activities.

Summary: Male, 48 y.o.; history of diabetes and angioplasty; non-compliant with medication; chest pain and dyspnoea before dive; unfit for diving; cardiac death

SC 10/05

This 31 y.o. woman had no known significant medical history and appeared to be healthy. She had suffered a non-fatal drowning incident as a child. She and her partner had been certified as Open Water Divers in Thailand two years earlier in an attempt to help her overcome her fear of the water. Both were inexperienced, having done only seven dives, all under supervision, the last being nine months prior, and all in Thailand's tropical waters.

She and her partner/buddy entered the water from the shore for their first unsupervised dive. The aim of the dive was to view cuttlefish. She was wearing mask, snorkel and fins, 5-mm semi-drysuit, separate hood, bootees and gloves, BCD, weight belt with 9 kg of weights and a regulator on a 7.7 L steel tank. The police reported that there was a light wind and surface conditions were likely to have been calm, with visibility of around 8 metres. The water temperature was 13–14^{o}C; there was no mention of any current.

After about 20 minutes diving at a depth of 3–4 msw, the buddy could not see the victim and surfaced to look for her or her bubbles. Unable to see either, he re-submerged for another five minutes before surfacing and returning to shore. He then phoned a local dive shop to ask for advice. The owner immediately contacted other divers whom he knew were nearby and asked them to help with a search.

A boat with three men soon arrived and soon sighted the victim lying face-up on the bottom at a depth of 4 msw. She was visible from the surface. Her rescuer could not recall if she was wearing her mask, snorkel and hood but noted that her regulator was out of her mouth. Using a 'bail-out' tank one of the men dived down to the victim, released her weight belt and brought her to the surface. She was dragged onto the boat, unconscious and apnoeic. BLS was begun and continued en route to shore until paramedics arrived and continued resuscitative efforts before abandoning these as there was no response.

The victim's weight belt, mask and snorkel were recovered the next day. On examination, her equipment was all found to be serviceable. There was 180 bar of air remaining and this met relevant purity standards. There was a slight tear in one of the second stage regulators which would likely have "*breathed wet*", although this was not believed to have been substantial. The other second stage was set into pre-dive mode and would have been more difficult to breathe from in this setting. However, it was unclear which one she had used as her primary demand valve.

Autopsy: A chest X-ray performed on the day of death did not identify air in the great vessels or the heart and no pneumothorax was evident. The autopsy was performed three days after death**.** The heart weighed 270 g (NR 308 ± 77 g). The origin of the left coronary artery was high (just above the coronary sinus) and there was focal 30% narrowing of the LAD. There was no gas in the heart or major vessels. Examination of the conduction system revealed no abnormality. There was frothy fluid in the main bronchi. The R and L lungs weighed 675 g (NR 547 g ± 203 g) and 652 g (NR 472 g ± 181 g) respectively, and felt heavy and airless with congestion and marked oedema. The cause of death was given as undetermined but the pathologist commented that the frothy fluid in the bronchi was supportive of drowning. Toxicology: nil

Comments: The victim was an inexperienced diver who had only ever dived in the tropics and under supervision. This was her first dive for nine months and, very significantly, the first in colder water, wearing a full wetsuit and a substantial amount of weight; quite possibly too much. This was also the first time that she and her buddy had dived unsupervised. Given her inherent fear of the water she was likely to have been very anxious. She might have got into difficulties unnoticed by her buddy or after separating from him. In either case, there was a breakdown in the buddy system in these inexperienced divers – not an uncommon event. If she had reached the surface, she would have found it very difficult to remain there without inflating her BCD and/or dropping her weight belt. She likely sank and drowned.

Summary: Female, 31 y.o.; apparently healthy; diving to help overcome fear of drowning; very inexperienced with inexperienced buddy; first dive in colder water; separation; drowning

SC 10/06

This 49 y.o. woman was severely obese (BMI 41 kg·m^{-2}), with a history of mild hypertension, hypercholesterolaemia, anxiety, depression and laparoscopic cholecystectomy. She appears to have been taking paroxetine for depression, alprazolam for anxiety and levonorgestrel at the time, and had been treated previously with diuretics for ankle oedema. Over the previous year she had been hospitalised several times for acute chest pain which settled after the administration of glycerol trinitrate. Standard cardiac investigations at that time showed no evidence of myocardial infarction and the pain was thought to be of biliary origin. She subsequently underwent a laparoscopic cholecystectomy. On-going symptoms resulted in a thallium exercise cardiogram which showed ECG changes during maximal exercise and scan abnormalities suggestive of reduced blood flow to the anterior wall of the left ventricle. It was unclear whether this was artefact owing to the overlying breast tissue. These changes were asymptomatic and normalised post exercise. She also suffered episodes of dyspnoea requiring hospital admission via ambulance. Chest X-ray showed non-specific changes and a CT pulmonary angiogram showed no evidence of pulmonary embolism or focal lung or pleural abnormality. She was subsequently prescribed salbutamol, although there was no definitive diagnosis of asthma.

In an effort to improve her fitness, the victim enrolled in a scuba diving course. She underwent a diving medical with a doctor with training in the assessment of fitness to dive but it appears that she failed to reveal her previous cardiac and respiratory problems. The doctor noted her obesity and hypertension (160/83 mmHg) and issued a fitness-to-dive certificate. Some problems during her initial pool training were largely attributed to the victim's ill-fitting wetsuit and she subsequently obtained a custom-made semi-drysuit.

On the day of the first open-water dive, the victim and four other students were under the supervision of two instructors and a trainee divemaster. The dive was from the shore and along a jetty, a relatively shallow site with sandbanks en route to deeper water. The conditions were described as windy with a slight surface current and the water "*looked clear*". The group geared up on the beach. The victim was wearing a mask, snorkel and fins; 6.5-mm semi-drysuit and hood, BCD, 14 kg of weights distributed between a weight belt, integrated pockets and ankle weights and a scuba unit. She was buddied with the trainee divemaster. The divers waded about 50 m into the water parallel to the pier until they reached chest-deep water. They then put on their fins and snorkelled for a few minutes to the dive buoy to descend. The depth here was 2.5 msw. However, the victim was too buoyant, so her buddy put an additional 3 kg of weights into her BCD pockets before she was able to descend.

Almost immediately, after possibly a metre of descent, the victim signalled that she wanted to ascend. When she and her buddy reached the surface, the buddy inflated the victim's BCD. The victim discarded her regulator, complained of dyspnoea and of "*feeling sick*" and was noted to be breathing rapidly and deeply with a faint wheeze. Her buddy began to tow her to shallower water but the victim began to panic when a wave washed over her. The buddy continued to alternately tow the victim and support her as they walked slowly towards shore. After another small wave splashed over the victim's face, she began to cough and became flushed. She asked a bystander to fetch her ventolin (salbutamol) from her bag. Once in shallower water the victim was helped to remove her hood and scuba unit and to unzip her wetsuit. She self-administered a total of four puffs of salbutamol and an ambulance was called. However, she soon deteriorated and became unresponsive and cyanotic with yellow, frothy sputum coming from her mouth. She was dragged to shore and placed in the recovery position as the rescuers believed that she was still breathing spontaneously, albeit with frothy sputum still oozing from her mouth and nose. Paramedics arrived soon afterwards and found her to be unconscious, cyanotic, with agonal respirations and no palpable pulse. A defibrillator was attached and indicated Pulseless Electrical Activity (36 beats per minute) decreasing to asystole within seconds. ALS was implemented between the rescuers and the paramedics. Suction was required frequently. Resuscitation was continued for 30 minutes but the victim failed to respond.

When examined later, her equipment was found to be functioning correctly although there was a small perforation in the mouthpiece of the primary demand valve. There was 200 bar of remaining air which was found to meet acceptable purity standards. The total weight of equipment carried by the victim was estimated to have been 37 kg.

Autopsy: The autopsy was four days after death. A post-mortem CT scan was taken but the results are not reported. The heart was enlarged 513 g (NR 285–439 g). The ventricles appeared of normal dimensions and there was only mild atherosclerosis of the coronary arteries. There was some fatty infiltration of the right ventricle but no other features suggestive of arrhythmogenic cardiomyopathy and the mitral valve showed thickening of the anterior leaflet with shortening and thickening of the papillary muscle (possibly mild mitral valve prolapse). Histology confirmed fatty infiltration of the heart which may be a feature in obesity). The AV node showed mild muscular hypertrophy and myxoid changes in some vessels as well as in the mitral valve. The kidneys showed occasional sclerosed glomeruli and a patchy cortical lymphocytic infiltrate but no features diagnostic of hypertension. The R and L lungs weighed 895 g (NR 561 g ± 256 g) and 700 g (NR 491 g ± 204 g) respectively and appeared slightly hyper-inflated and firm. There was 100 ml of fluid in the right pleural cavity, a small amount of frothy fluid in the airways and moderate oedema of the lungs. Histology of the lungs showed no evidence of asthma. The cause of death was unascertained. The mild cardiomegaly and mild mitral valve changes were discussed but the degree of disease was felt to be insufficient to account for death. The possibility of sudden death due to a cardiac arrhythmia due to a cardiac channelopathy was raised.
Toxicology: paroxetine 0.1mg·L^{-1}

Comments: This morbidly obese woman with a history of cardiac-like pain and dyspnoea requiring repeated hospital admissions was an unsuitable candidate for scuba training and had she declared her past medical history would almost certainly not have been passed as fit. Spirometry was performed in the dive medical and was not suggestive of the presence of asthma. It is likely that the combination of the physical, physiological and psychological stresses on her first open-water dive and the effects of immersion precipitated acute pulmonary oedema in a predisposed individual. It is also possible that the administration of salbutamol in this situation may have precipitated or worsened an arrhythmia, a situation not unlikely given the already existing cardiac changes.

Summary: Female, 49 y.o.; severe obesity, mild hypertension, hypercholesterolaemia, anxiety, depression, episodes of cardiac-like chest pain and dyspnoea; first open water dive; likely carrying 37 kg (including scuba unit); brief submersion; severe dyspnoea; collapse; acute pulmonary oedema with probable terminal arrhythmia

Rebreather fatality

RB 10/01

This 49 y.o. man was obese (BMI 33.9 kg·m^{-2}) and had a history of depression and migraine. Past history also included an episode of pleuritic-type chest pain in 2001. A ventilation-perfusion scan at that time showed a large unmatched perfusion defect in the left lung base suggestive of pulmonary embolism. His latest ECG, taken seven months prior to the incident, was reported to have been normal, as had a stress test in 1999. He was taking paroxetine hydrochloride and pizotifen malate. He was a qualified divemaster and had been an active and experienced open-circuit diver. He had recently purchased a 10-year-old Dräger Dolphin rebreather which had been converted from a semi-closed to a fully-closed unit by the friend who had sold it to him. That friend had also certified him to use a Dolphin rebreather two months earlier. There is some debate as to the configuration of the unit during this training. The diver's logs indicated that he had possibly done about 10 dives using this unit. The log also indicated that all of these dives were relatively shallow.

On the day of the incident, the victim's buddy, with whom he had dived about 10 times before, stated that the victim appeared to be quieter than usual and complained of having a headache. He said that he would take some medication for his headache and was sure that he would be able to dive. His buddy offered him a seasickness medication (hyoscine hydrobromide) which he took. The pair then set out with 15 other divers on a charter boat which took them to a wreck sitting in the ocean at a maximum depth of about 39 msw.

The victim and his buddy were the second pair to enter the water. Conditions were described as quite good, with some surface chop. The victim was wearing a drysuit with undergarments, a hood, leg gaiters, boots and fins, mask, BCD, his rebreather (with one cylinder of air and one of oxygen), a 5-L bail-out cylinder (containing nitrox29.6) which was connected to his drysuit inflator. He carried 11 kg of weight, distributed around the shoulders of his rebreather (1.5 kg each) and in two ditchable mesh bags (4 kg each). He was also carrying his camera. The buddy was diving on open-circuit breathing nitrox30.

They began descending the shotline together and did a mutual bubble check under the surface. On reaching a depth of about 23 msw, the buddy noticed that the victim was well above him and ascending so he swam towards him, meeting at the surface. When asked if he was okay, the victim replied "*just me*", stated that he wished to continue the dive and they re-descended to the wreck at 36 msw. The buddy reported that there was a slight surge and current on the bottom, visibility was 10 m and water temperature 13^{O}C.

The pair swam around the wreck while the victim took photographs. Wishing to stay within his no-decompression limits, the buddy indicated that he wanted to surface, the victim signalled agreement and they began to ascend. However, after rising about 7–8 msw, the buddy looked down and noticed that the victim was still near the wreck, sinking despite efforts to ascend. On reaching him and concerned that he did not understand rebreathers and was unsure of what to do, the buddy offered the victim his 'octopus' in case he needed it. However, the now wide-eyed and anxious-looking victim pushed the 'octopus' away. The buddy gestured towards the victim's bail-out bottle but again his hand was brushed aside by the victim who didn't respond to his signals. In light of the rejection of both alternate breathing supplies, the buddy, believing the victim to be negatively buoyant, reached for the victim's BCD inflator but again the victim, behaving erratically, pushed him away. The buddy then looked for the victim's weight belt but could not see it.

Finally, the buddy decided to hold onto the victim and use his own BCD to lift them both to the surface. When he grabbed the victim the latter did not push away, so the buddy inflated his own BCD but this was insufficient to lift them as the victim was so heavy. When the buddy let go of the victim his own positive buoyancy caused him to rise rapidly until he could dump some gas. After descending a few metres and being unable to see the victim, he decided to do a controlled ascent (of about four minutes) to get help. He believed that the victim was conscious and breathing when he last saw him.

Shortly afterwards, a trio of divers found the victim lying on the deck of the wreck. He was unresponsive. His eyes were closed and his mouthpiece was hanging loosely in one corner of his mouth. One of the group tried unsuccessfully to replace his mouthpiece. Two of the trio grabbed the victim; one inflated her BCD and the other finned hard and they began to rise. Their ascent rate became rapid nearer to the surface from the expanding air in their BCDs.

On reaching the surface the rescuer called for help. She struggled to keep the victim's head above the water as he was so heavy. He was unconscious and frothy sputum was oozing from his mouth and nose. Assisted by one of her buddies, the rescuer managed to remove the victim's rebreather which sank quickly. The victim was soon dragged aboard the dive boat, rolled onto his side to drain his airway and an oropharyngeal airway was inserted. BLS was commenced by some of the crew and continued as the boat sped back to the jetty. Ventilations were provided via a manually-triggered oxygen-powered resuscitator. The rescuers, one of whom was a nurse, needed to turn the victim onto his side regularly to clear bloody, frothy sputum and water from his mouth. On reaching the jetty, paramedics implemented ALS, without success.

Two weight pouches belonging to the victim were recovered the next day. Neither the buddy nor subsequent rescuers had reported ditching these so it is likely that they had been

ditched by the victim in an attempt to ascend. The rebreather unit was received by police four days later. When examined it had been modified to work as a mechanically operated closed circuit unit. Both the oxygen and diluent tanks were empty and the diluent tank contained seawater. The mouthpiece was partially bitten through. The bailout cylinder was turned off and contained 190 bar pressure.

Autopsy: Post-mortem CT scan showed widespread gas within the arterial and venous circulation. Since the victim's body was brought up rapidly from 40 msw, it is highly likely this was post-mortem decompression artefact. The heart weighed 400 g (NR 400 ± 69 g) and appeared normal with no coronary atherosclerosis. The R and L lungs weighed 690 g (NR 663 g ± 217 g) and 670 g (NR 569 ± 221 g) respectively and appeared mildly expanded. Fluid exuded from the cut surface. On initial presentation, there was a plume of pulmonary oedema fluid coming from his mouth. No pathological abnormality was detected in the brain, which weighed 1515 g, and there was no evidence of pulmonary embolism (see history). The pathologist commented on the possibility of carbon dioxide narcosis; however, it is not possible to determine post-mortem carbon dioxide levels. The cause of death was given as unascertained.
Toxicology: nil

Comments: This case raises a number of issues, many of them of a moral nature. The instructor stated that he had converted the unit back to semi-closed-circuit rebreather (SCR) configuration during training and that he had only certified the victim as a Dolphin SCR diver, but that he had supplied all the parts to convert it back to CCR configuration. One might then question why, as an instructor, he allowed the victim to dive with him without insisting that he get appropriate training and qualification in CCR mode if he was going to dive with the unit in that configuration, especially as such training is readily available. Furthermore, the dive operator was unaware that the victim was untrained in the use of the unit as a CCR, and stated that he would not have allowed him to dive from the boat had he been aware. He was assured by the instructor that training had been completed although the certification had not yet been received. At the time, this instructor was not qualified to teach CCR diving.

The use of air as a diluent at 39 msw reflects to some extent this lack of training. Not only would this have provided significant narcosis at this depth to a diver relatively unfamiliar and untrained with his CCR, but also would create a significant work of breathing in a unit not designed as a CCR or for such depth. It is quite probable that narcosis contributed to his inability to solve his problem underwater.

From the evidence of the witnesses and the state of the equipment, it would appear that the victim was considerably over-weighted. Analysis of his dive computer implies that this was the deepest dive that he had conducted on the CCR unit. It would seem that, for some reason, the victim exhausted his diluent gas before the end of the dive. This would not normally be an emergency situation as diluent is not required when on the bottom or at a stable depth. However, as this cylinder also inflated his BCD and the bailout cylinder which was attached to his drysuit was turned off, he was unable to get sufficient, positive buoyancy to ascend. He seems not to have recognised that his bailout cylinder was turned off (a practice that may have been carried over from his open-circuit diving). When his buddy attempted to rescue him and dragged him up by inflating his BCD, his CCR would have vented gas from the breathing loop. When he was subsequently released, the descent would have required the addition of gas to prevent the loop from collapsing. The only gas remaining to provide this would have been oxygen. It is possible that the victim realised this and attempted to remove his ditchable weights before being overcome by oxygen toxicity. While a convulsion was not observed, the indication that the mouth-piece was bitten through is highly suggestive of convulsion.

Summary: Male, 49 y.o.; history of depression and migraine; divemaster open-circuit scuba diver; untrained closed-circuit diver; modified rebreather; overweighted; loss of diluent; likely narcosis and subsequent oxygen seizure; drowning

Surface-supplied breathing apparatus diving fatality

SS 10/01

This 48 y.o. man had a history of single shoulder and hip arthroplasties, palpitations and dizziness, angina, paroxysmal atrial tachycardia, ventricular tachycardia, and tight stenosis of a small LAD-origin septal vessel. He had had a positive stress ECG two years earlier and an associated technesium scan indicated areas of ischaemia. He had undergone Holter monitoring three months prior to the accident to investigate the recurrence of palpitations. Although he had previously been on a variety of medications, there was no record of any currently prescribed medications. Despite severe obesity (BMI 36.9 $kg \cdot m^{-2}$), his wife described him as "*quite healthy for his age … had extra weight but was reasonably fit and he played underwater hockey*". There is no record of his having any training, certification or medical examination for scuba diving. He was said to have been a keen and active fisherman and scallop diver of many years.

He went diving for scallops with two friends from a 7-m boat. The weather was reported to have been sunny and calm with a light wind. The water temperature was around 12°C. After an uneventful first dive to 6 msw, the group moved to a new site, anchoring their boat in about 8 msw depth. The victim was wearing a 5-mm wetsuit with an additional 3-mm vest with attached hood, weight belt (weights not reported), mask (snorkel unknown), boots, fins and gloves, and he was carrying a catch bag. He was not wearing a BCD.

After a surface interval of 30 minutes, he and one of his friends dived together using a home-made 'Hookah' while their friend remained on the boat to watch the compressor.

The victim's 'hookah' hose was threaded under his weight belt from behind, between his legs and under his weight belt at the front, around his left shoulder and, finally, around his neck to the demand valve. He did not carry a bail-out bottle. After swimming together for about 5–10 minutes at a depth of 12 msw, the pair separated when the bottom became stirred-up and visibility deteriorated. The buddy surfaced an estimated 15 minutes later as he had filled his bag and swam back to the boat. After a short time, the buddy looked back and saw the victim on the surface about 40 m away, apparently struggling, with his head and shoulders just above the water. He was wearing his mask and his regulator was out his mouth. He sank briefly before surfacing again and calling for help. The buddy jumped back in and swam to where the victim had been but he had submerged and could not be seen. The hookah line was vertical and there were no visible bubbles.

The friend in the boat began to haul in the line while the buddy swam back to the boat. The victim was brought to the surface unconscious, cyanotic and apparently apnoeic about one to two minutes from when he was last seen. The buddy ditched the victim's weight belt (which possibly had the catch bag attached to it) and supported him from behind in the water while the friend on the boat removed his mask (which contained a small amount of blood) and tried to give a rescue breath. Unable to lift the victim, the friend on the boat went to the radio to call the emergency services while the buddy supported his friend and heard what are likely to have been agonal respirations.

In response to a flare, a large boat with divers arrived 10 minutes later and one of them helped to lift the victim into this boat and roll him onto his side to drain water and mucus from his mouth. He and the buddy began BLS and continued on the way to the jetty. The buddy described a regular "*liquid*" sound when they gave rescue breaths and rolled the victim onto his side periodically, although little water came from his mouth. On arrival, they were met by a police rescue vessel and its crew took over resuscitation, adding supplemental oxygen via a manually-triggered ventilator. After about five minutes, they were relieved by ambulance crew who found the victim to be in asystole with fixed, dilated pupils and signs of post-mortem lividity. Resuscitation was abandoned shortly afterwards, approximately 70 minutes after the victim had been found unconscious.

When later tested the compressor unit was found to be in poor condition with multiple faults. These included a fuel leak, the absence of a suitable air filter or water trap, the absence of non-return valves, an incorrect supply pressure setting, minimal distance between the inlet and exhaust as well as other faults, such as a small hose that kinked easily, reducing or stopping the air flow to the divers. The tests revealed that if a diver on the surface purged his demand valve it would greatly reduce the airflow to the other diver at depth. His primary demand valve was functional. His secondary demand valve, if used, could have caused some water aspiration. The air test results indicated that both the carbon monoxide (CO, 70 ppm) and moisture content (> 160 $mg \cdot m^{-3}$) of the air in the compressor reservoir greatly exceeded the relevant Australian Standard (10 ppm and 160 $mg \cdot m^{-3}$ respectively).

Autopsy: A whole-body CT scan was carried out five hours after death. This showed gas in both ventricles of the heart, in the aorta and in the liver with relatively small amounts of gas in the portal venous system. Large amounts of gas were seen in the vessels of the brain. At post mortem, there was 70 ml of gas in the right ventricle and 20 ml of gas in the left ventricle. There was a 70-mm-long, deep laceration on the scalp which probably occurred during recovery of the body (supported by comment from police). The heart weighed 446 g (NR 400 g ± 69 g) with left dominant circulation and a 30% narrowing of the LAD. The proximal stenosis of the small septal branch of the LAD, reported on angiography, was not seen. Histology of the heart showed mild hypertrophy but no scarring. The R and L lungs weighed 470 g (NR 663 g ± 217 g) and 450 g (NR 569 g ± 221 g) respectively, and appeared slightly over-expanded. There were a few small apical bullae, a small quantity of oedema fluid in the upper airways but little in the lungs. The cause of death was given as cerebral arterial gas embolism (CAGE) due to pulmonary barotrauma while surface supply diving for scallops. There was also a history of ventricular tachycardia and tight stenosis of a small LAD-origin septal vessel which may have contributed to death.
Toxicology: carboxyhaemoglobin negative

Comments: Although when tested, the compressor was found to produce a high level of CO, there was no evidence that this was a factor in the victim's demise as his toxicology was negative for carboxyhaemoglobin and his buddy had no problems. It is likely that this diver had an interrupted gas supply resulting from a drop in pressure from surface purging or the kinking of a vulnerable narrow hose. Without a 'bail-out' cylinder and/or BCD he would have to swim to the surface (possibly from about 12 msw), likely overweighted by scallops. Despite his experience, such circumstances created a high risk of inadvertent breath-holding which can lead to pulmonary barotrauma and consequent CAGE. It is possible given the strong clinical history of cardiac arrhythmia that the rapid ascent causing the CAGE could have been precipitated by a cardiac arrhythmia. The other interpretation is that death was caused by a cardiac arrhythmia and that gas seen at post mortem represents post-mortem decompression artefact. However, it was the examining pathologist's (CL) impression at the time that the gas represented CAGE. Post-mortem examination will usually identify structural heart disease but is poor for diagnosis of functional cardiac arrhythmia.

Summary: Male, 48 y.o.; severely obese; history of palpitations, angina, paroxysmal atrial tachycardia, ventricular tachycardia, and tight stenosis of a small septal coronary vessel; active lifestyle and played underwater

Table 3
Root cause analysis of diving-related fatalities in Australian waters in 2010

Case	Trigger	Disabling agent	Disabling injury	Cause of death
BH10/01	Unknown; silting? Disorientation?	Unknown; entrapment?	Asphyxia?	Drowning?
BH10/02	Immersion, exertion, aspiration, anxiety	Cardiovascular disease	Cardiac incident	Cardiac-related
BH10/03	Prolonged breath holding	Apneic hypoxia	Asphyxia	Drowning
BH10/04	Unknown; inexperience? Panic?	Unknown; aspiration? Sudden unconsciousness?	Unknown; asphyxia?	Unknown; drowning
BH10/05	Immersion, tight wetsuit, anxiety.	Cardiovascular disease	Cardiac incident	Cardiac-related
BH10/06	Unknown, inexperience? Panic?	Unknown, aspiration? Sudden unconsciousness?	Unknown; asphyxia?	Unknown; drowning?
BH10/07	Inexperience, exertion, aspiration	Atrial fibrillation	Cardiac incident	Cardiac-related
BH10/08	Exertion	Cardiovascular disease	Cardiac incident?	Cardiac-related?
BH10/09	Immersion? Exertion? Prolonged breath holding?	Unknown; cardiovascular disease? Apnoeic hypoxia?	Cardiac incident? Asphyxia?	Drowning
BH10/10	Unknown; inexperience? Panic?	Unknown; aspiration? Sudden unconscious?	Unknown; asphyxia?	Unknown; drowning?
BH10/11	Prolonged breath holding	Apnoeic hypoxia	Asphyxia	Drowning
BH10/12	Prolonged breath holding	Unknown; apneic hypoxia? Cardiovascular disease?	Asphyxia? Cardiac incident?	Drowning
SC10/01	Anxiety, low air, rough conditions	Aspiration	Asphyxia	Drowning
SC10/02	Unknown; buoyancy-, equipment-, gas supply-related?	Unknown; buoyancy-related?	Asphyxia	Drowning
SC10/03	Poor mental state, narcosis, unfamiliar equipment, gas supply-related	Buoyancy-related, gas supply-related, entanglement	Asphyxia	Drowning
SC10/04	Exertion	Ischaemic heart disease	Cardiac incident	Cardiac-related
SC10/05	Unknown; anxiety?	Buoyancy-related?	Asphyxia	Drowning
SC10/06	Exertion, immersion	Morbid obesity, poor cardiopulmonary fitness	Immersion pulmonary oedema	Cardiac-related
RB10/01	No formal CCR training, narcosis	Oxygen toxicity	Asphyxia	Drowning
SS10/01	Unknown; gas supply-related?	Ascent-related	CAGE	CAGE

hockey regularly; training unknown; experienced; using faulty 'hookah' compressor; surfaced, called for help and sank; BLS unsuccessful; CAGE/pulmonary barotrauma

Discussion

A summary of the possible sequence of events (root cause analysis) in each of these incidents is shown in Table 3.

APNOEIC HYPOXIA

In this series, it is likely that at least two BH divers died as a result of apnoeic hypoxia. As in the 2009 report,[2] one victim (BH10/03) was alone and doing underwater laps in a pool. In another (BH10/11), the dive may have been complicated by entrapment. It is possible that apnoeic hypoxia led to the death of another victim (BH10/12); however, the reviewers believe this death was more likely the result of a cardiac arrhythmia triggered by breathholding.

SOLO DIVING OR SEPARATION AND/OR SUPERVISION PROBLEMS

Solo diving or separation has contributed to many diving deaths.[2–5] It is a recurring theme in dive accident reports and this series is no exception, likely being implicated in five of the breath-hold (BH10/01, BH 10/04, BH10/06, BH10/08 and BH 10/12), one scuba (SC10/03) and the SSBA fatalities. Having a buddy nearby does not guarantee rescue but it generally increases the likelihood of support and assistance. However, as highlighted in SC 10/03 and RB 10/01, a dive buddy can sometimes be at risk when trying to assist a stricken companion. In both incidents, the buddy was finally forced to make the unenviable decision to abandon his companion for the sake of his own survival.

Poor supervision appears to have been a factor in at least two incidents. In BH 10/04, the victim was on a guided tour and went snorkelling alone from the shore of a lake. It is not clear what the arrangements were with the tour guide or what assessment and briefing was done, but this "*poor swimm*er" went snorkelling alone, under what turned out to be inadequate supervision from friends. In BH 10/12, the captain chose to be the single observer for up to 30 snorkellers; clearly inadequate since one disappeared and another reported signalling for help and not receiving any. Commercial operators need to have and adhere to realistic ratios for supervision and do so diligently.

EQUIPMENT

Equipment problems were implicated as a likely or possible contributor to at least four incidents. Equipment-related problems are commonly reported to be associated with diving incidents, whether fatal or non-fatal.[3–7] In SC 10/03 and RB10/01, the victims were using relatively unfamiliar equipment. Faults were found in the equipment used by the victims in SC 10/02, RB 10/01 and SS 10/01 and these may have precipitated or exacerbated the incident.

In addition to issues with diving equipment, problems with first-aid-related equipment were also obvious in three cases. In one (BH 10/02), although an AED was available, the battery was flat. In another two, the O_2 equipment was unusable either because the delivery device was unsuitable (BH 10/10) or because an integral part was missing (BH 10/12). In all cases, the problems could have been readily averted by having the appropriate equipment in the first place, including an adequate O_2 supply to enable delivery of near-100% O_2 until medical assistance (with more O_2) was available.[9] Dive operators should have and adhere to appropriate protocols for checking and maintenance of first-aid equipment and supplies and the performance of pre- and post-excursion function testing. This might have been beneficial for BH 10/10. It cannot be emphasised enough that any operator catering for diving activities must ensure that they have appropriate and functional O_2 equipment and trained personnel readily available at the dive site.

DIVE PREPARATION

There are several lessons to learn from SC 10/04. Experience is important but dive currency perhaps more so. If new equipment is being used, divers should revert to simple open-water dives until the equipment is mastered and only then return to their previous level/complexity of diving. Furthermore, divers need to recognise when they are not feeling up to a dive on the day, whether because of feeling unwell, the presence of poor diving conditions or perhaps due to problems with equipment, and either abandon the dive altogether or refine their dive plan to something shallower or less challenging. This is not only for their own sake, but in the interests of their buddy and other divers. Finally, the risks of deep air diving are well described and seem to have been ignored in this case.

CARDIAC-RELATED FATALITIES AND OBESITY

Once again, cardiac-related deaths were well-represented in this series and are thought to have been contributory in at least a quarter and possibly nearly half of these fatalities. The effects of immersion are known to precipitate cardiac arrhythmias in both breath-hold and scuba divers, especially in cold or deep water.[10–13] Of note, nine of these 20 divers were obese, with BMIs ranging from 30.9 to 43.4 $kg \cdot m^{-2}$. At least five of these obese divers are believed to have been disabled by a cardiac-related event. Obesity is incompatible with safe diving. The effects of what is often a restrictive wetsuit, excessive weighting to overcome positive buoyancy, impairment of respiratory function, especially when immersed and increased cardiac demands to overcome these can present a serious hazard. Even if a cardiac event did not underlie the death in some obese divers, obesity per se can be a contributory factor.[14,15] As indicated in BH 10/02, BH

10/07 and SS 10/01, it can be more difficult to lift an obese person onto a boat or platform and this should be considered in advance when dealing with such divers.

IMMERSION PULMONARY OEDEMA (IPE)

The topic of IPE is currently of great interest to researchers as there have been an increasing number of reported cases, both fatal and otherwise.[16,17] IPE was discussed by the authors as a possible contributing factor or differential diagnosis in several of the above cases. However, a definitive diagnosis can be elusive in the absence of a clear clinical history, as autopsy findings can readily be attributed to cardiac disease or drowning.

DELAY TO AUTOPSY

In a number of these cases, there was an interval of three or more days between death and the autopsy. A study of drowning fatalities demonstrated a time-dependent fall in the combined lung weights in drowning, thought to be due to post-mortem transudation from the lungs and an increase in fluid in the pleural cavity especially after three days.[18] Given this and the problem with post-mortem decompositional gas formation, the sooner these autopsies are carried out the more likely the pathologist is to be able to identify features of drowning. Given the large number of CT scanners in Australia now, all scuba diving fatalities should have a CT scan as soon as possible after death and preferably within eight (8) hours of death. This does not entirely solve the problem of post-mortem decompression artefact but it is helpful if there is a delay in autopsy examination.

REBREATHERS

Rebreather deaths have been rare in Australia up to 2010. In a review conducted in 2013, CCR divers were estimated to be ten times more likely to be involved in a fatal diving accident than were recreational open-circuit scuba divers.[8] The perception of increased risk with CCR diving is well known in the dive industry and amongst divers and, therefore, it seems inexplicable that this diver would be supported to dive a CCR unit without appropriate training by a senior diving instructor (himself a CCR diver but not a CCR instructor). While CCR units can be dived safely, there is no place for home-made units dived by untrained individuals in what is already a risky undertaking. Training on semi-closed rebreathers does not substitute for CCR training as, apart from the basic breathing loop, the two types have little in common.

DISCLOSURE OF MEDICAL CONDITIONS AND THE NEED FOR PROPER MEDICAL ASSESSMENT

Once again, some of these cases (e.g., BH 10/02 and BH 10/05) highlight the importance of prospective or active snorkellers and divers disclosing medical conditions to the dive physician or dive operator to enable a more appropriate decision to be made about their fitness to dive or snorkel, and/or indicate the need for closer supervision. Some (such as SC 10/04 and SC 10/06) also showcase the need for divers or potential divers with significant medical conditions, not only to undergo a fitness-to-dive assessment (preferably by a doctor with relevant training and experience), but also for them to provide an honest and complete medical history to facilitate a more accurate assessment.

CORONERS' FINDINGS

Several of the coroners' findings do not mention that the victim was scuba diving or snorkelling at the time of their death; it is simply stated, for example, that death was due to drowning or ischaemic heart disease. The addition of several words to indicate the circumstances of death provide context for the death. It would also enable easier tracking by researchers who do not have access to the complete file and are trying to track diving-related (or other) deaths. This need will be communicated to the Coronial Service.

LIMITATIONS OF THE STUDY

As with any uncontrolled case series, there were inevitable limitations and uncertainties associated with our investigations:

- Incomplete case data: fatalities were sometimes unwitnessed, and reports provided by any witnesses and by police varied in their likely reliability, as well as the content and expertise of the investigators.
- Autopsy reports may be unreliable as a result of the difficulty of determining the presence of CAGE in the absence of relatively prompt post-mortem CT scans, and the inability to detect evidence of cardiac arrhythmias, among other factors. Care must be taken to critically examine the available evidence and minimise speculation when determining the likely disabling injuries.
- Classification of cases into a sequence of four events (trigger, disabling agent, disabling injury, cause of death) using root cause analysis (Table 3) requires a single choice for each component event, which may omit important factors in some cases because, at each level, multiple factors rather than a single one may be at play.
- Limited annual case data: 20 deaths are too few to reliably determine trends.

Conclusions

- There were 20 reported diving-related fatalities during 2010 including 12 deaths while snorkelling and/or breath-hold diving, seven while scuba diving (one of these while using a closed-circuit rebreather) and one while using surface-supply breathing apparatus.
- Snorkelling or diving alone, poor supervision, apnoeic

hypoxia, pre-existing medical conditions, lack of recent experience and unfamiliar and/or poorly-functioning equipment were features in several deaths in this series.
- Other contributory or causal factors were entanglement and diver error.
- With snorkellers, the likely disabling injuries were asphyxia and cardiac causes.
- In scuba divers, the disabling injuries appear to have been asphyxia, CAGE and cardiac-related causes.
- Factors that may reduce mortality in the future include the avoidance of solo diving and snorkelling and improved buddy oversight; better supervision of organised activities; improved medical screening of older divers; operational integrity of equipment and ensuring familiarity with new equipment in a controlled environment.
- Reducing delays to CT-scanning and autopsy and coroners' reports documenting that the victim of a drowning was snorkelling or scuba diving at the time are aspects of the investigation of these fatalities that could be improved.

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Middle ear barotrauma in a tourist-oriented, condensed open-water diver certification course: incidence and effect of language of instruction

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Abstract

(Blake DF, Gibbs CR, Commons KH, Brown LH. Middle ear barotrauma in a tourist-oriented, condensed open water diver certification course: incidence and effect of language of instruction. *Diving and Hyperbaric Medicine*. 2015 September;45(3):176-180.)

Introduction: In Professional Association of Diving Instructors (PADI) Open Water Diver certification courses that cater to tourists, instruction is often condensed and potentially delivered in a language that is not the candidate's native language.

Objective: To assess the incidence of middle ear barotrauma (MEBt) in open-water diver candidates during a condensed four-day certification course, and to determine if language of instruction affects the incidence of MEBt in these divers.

Method: The ears of participating diving candidates were assessed prior to commencing any in-water compression. Tympanic membranes (TM) were assessed and graded for MEBt after the confined and open-water training sessions. Tympanometry was performed if the candidate had no movement of their TM during Valsalva. Photographs were taken with a digital otoscope.

Results: Sixty-seven candidates participated in the study. Forty-eight had MEBt at some time during their course. MEBt was not associated with instruction in non-native language (adjusted odds ratio = 0.82; 95% confidence intervals 0.21–3.91). There was also no significant association between the severity of MEBt and language of instruction.

Conclusion: Open-water diver candidates have a high incidence of MEBt. Education in non-native language does not affect the overall incidence of MEBt.

Key words

Barotrauma; middle ear; risk factors; instruction – diving; training; education

Introduction

The Professional Association of Diving Instructors (PADI) Open Water Diver course typically consists of classroom instruction, five confined-water sessions, and four open-water dives often spread over weeks. Candidates must learn the laws of physics that are important for divers, including Boyle's law. One practical implication of this law is the need for divers to 'equalize' the middle-ear air space as they descend, using various methods.

Ear pain and middle ear barotrauma (MEBt) are common in divers.[1,2] Open-water diver candidates may be at a greater risk of this as they are simultaneously learning how to equalize their ears, breathe through a regulator, adjust their buoyancy, clear their mask and perform other essential tasks required of a diver. This multi-tasking may overwhelm the diving candidate so that they equalize their ears late or not at all. It has been reported that the inability to equalize the middle ear is the most common reason for diving candidates to quit their training.[3]

Some PADI open-water diver certification courses cater specifically to tourists. In these circumstances, instruction is often condensed and potentially delivered in a language that is not the candidate's native language. Few studies have looked at the incidence of MEBt in diving candidates completing open water diver courses,[4] and no study has examined the role of language of instruction. This prospective, observational study was performed to assess the incidence of MEBt in open-water diver candidates during a condensed, four-day Open Water Diver course and to determine whether language of instruction affects the incidence of MEBt in these divers.

Methods

Ethical approval for this study was granted by the Human Research Ethics Committee of the Townsville Hospital and Health Service (HREC/12/QTHS/7). The study was conducted at a training centre in Cairns, Queensland, Australia, certifying approximately 3,600 divers each year. Cairns is the most common departure point for diving along Australia's Great Barrier Reef (GBR). The GBR is a chain of reefs, islands and coral cays, extending 2,300 kilometre along Australian's north-east coast. Instruction is offered in English, German and Japanese; however, students may be tourists who primarily speak other languages, and some German and Japanese tourists choose to attend English language classes due to their greater availability. Approximately half of the 21,000 open-water diver certificates issued each year by PADI Australia are issued in Queensland (Nimb H, personal communication, 2014).

Open-water diver candidates were approached to participate in the study. All subjects were determined fit to dive by either completing a diver's medical questionnaire or by passing a dive medical (Australian Standard 4005.1) prior to their first confined-water session. The exclusion criteria were children, if no parent was available to give consent, and non-English speaking candidates with no interpreter available. All participants were given a study information sheet and informed consent was obtained.

Table 1
Incidence of middle ear barotrauma after each water session by language of instruction; there were no statistically significant differences at any stage; * one subject withdrew before the final open-water dive

Instruction in:	1st pool	2nd pool	1st open-water	Last open-water	Any
Native language (n = 42)	12	20	21	25*	31
Non-native language (n = 25)	4	8	10	17	17

The courses evaluated in this study were completed over four days. All of the instruction was delivered by certified instructors. Classroom instruction occurred at the training centre. The five confined-water dives were performed during two pool sessions in 4-metre-deep, heated training pools. Open-water dives were performed from live-aboard dive boats in warm, tropical waters with candidates wearing thin wetsuits for warmth or stinger suits for protection; no gloves or hoods were worn.

Investigators accompanied the candidates and instructors during the confined and open-water dives. Baseline data, including age, gender and BMI, were collected prospectively using pre-formatted data forms. Candidates were asked if they were smokers, had any medical or previous ear, nose or throat (ENT) problems, were using any medications or had any allergies, including environmental. Time since their last flight was recorded along with previous scuba experience. Instructor-to-student ratio, candidate's native language and language of diving instruction were also documented.

The candidates were assessed for MEBt prior to the first pool session, after each of the two pool sessions and after the first and last open-water certification dives. Candidates were questioned about any difficulties equalizing or ear pain. Examination of the tympanic membrane (TM) was conducted using a Welch Allyn Digital MacroView® otoscope immediately after completion of the pool sessions and within one hour of completion of the open-water dives. Digital photos were taken of those TMs that were abnormal. Movement of the TM with Valsalva was documented and any candidate whose TM was not seen to move had tympanometry performed (MicroTymp3®, Welch Allyn Inc, Skaneateles Falls, NY, USA). When necessary, cerumen was gently removed with a disposable Jobson-Horne probe to allow for visualization of the TM. Grading of MEBt was done using the Edmonds grading scale of 0 to 5.[5] Figure 1 provides recent example photographs of each grade. The digital images of the abnormal TMs were reviewed by a senior physician (DFB) for accuracy of grading. When a student experienced symptoms but the view of the TM was obscured, the student was considered to have MEBt of unknown grade. All collected data were de-identified and entered into a pre-formatted Excel spreadsheet. These data were subsequently exported into Stata Statistical Software: Release 11 (StataCorp. 2009. College Station, Tx: StataCorp LP) for analysis.

ANALYSIS

The objectives of this analysis were to determine the incidence of MEBt in open-water diving candidates completing a condensed course, and to assess the influence of language of instruction on the incidence of MEBt in these candidates. Using Fisher's Exact Test (FET), the incidence of MEBt was compared at each stage of the course among candidates instructed in either their native or non-native language. To account for potential covariates and confounders, we also conducted logistic regression for MEBt including language of instruction, participant age and gender, previous scuba experience, time since last flight, and instructor-to-student ratio in the model. We compared the grade of MEBt among subjects instructed in their native and non-native language using Wilcoxon Rank Sum Test. Finally, to differentiate clinically significant from clinically insignificant MEBt, we evaluated the incidence of grade 2 or greater MEBt (the level at which diving and hyperbaric physicians would advise divers to refrain from further dives) at each stage of the course, again using FET. In all analyses, $P < 0.05$ was considered statistically significant.

Results

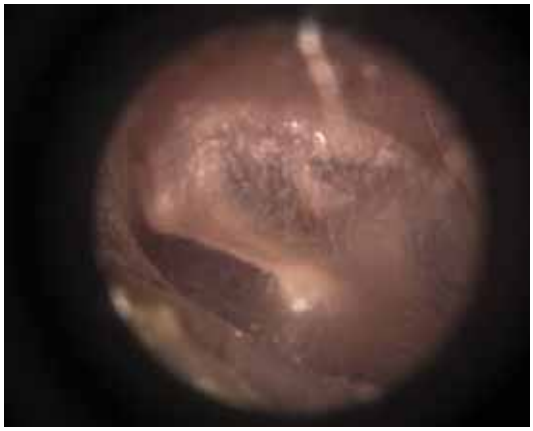
Sixty-seven dive candidates participated in the study, 37 male and 30 female. Mean age was 26.7 (standard deviation, SD 8.6) years; 29.4 (9.6) years in the native language group (n = 42) and 22.2 (3.4) years in the non-native language group (n = 25; $P < 0.001$). One candidate withdrew (for unrelated reasons) prior to the final open-water dive. Twenty-seven had previous scuba experience, including *Discover Scuba Diving*; 21 in the native language group and six in the non-native language group (P = 0.043). English was not the native language of 38 of the students, and 25 of the students received instruction in a language other than their native language. There were 10 candidates whose TM did not move with Valsalva; all 10 had a normal tympanogram.

Forty-eight of the 67 candidates had MEBt at some time during the course. There were no associations between MEBt and gender (P = 0.296), scuba experience (P = 0.599), inability to Valsalva (P = 0.260), previous ENT problems (P = 0.357), allergies (P = 0.551) or instructor-to-student ratio (P = 0.064). Table 1 shows the incidence of MEBt for each water session by language of instruction. There was no significant association between language of instruction and incidence of

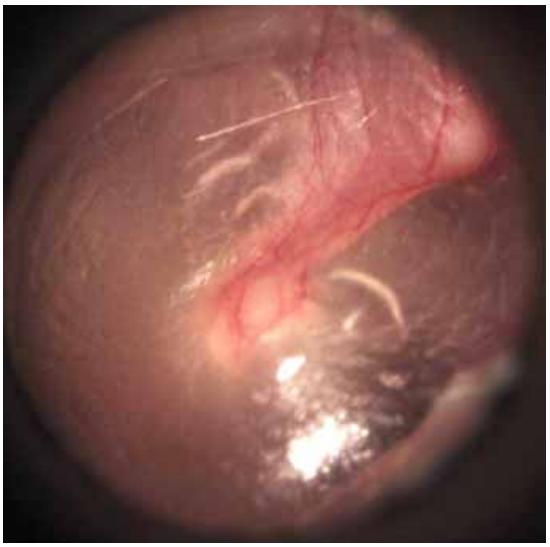
Figure 1

Grades of tympanic membrane barotrauma seen in this study (except for the grade 5 photo) as defined by Edmonds[5]

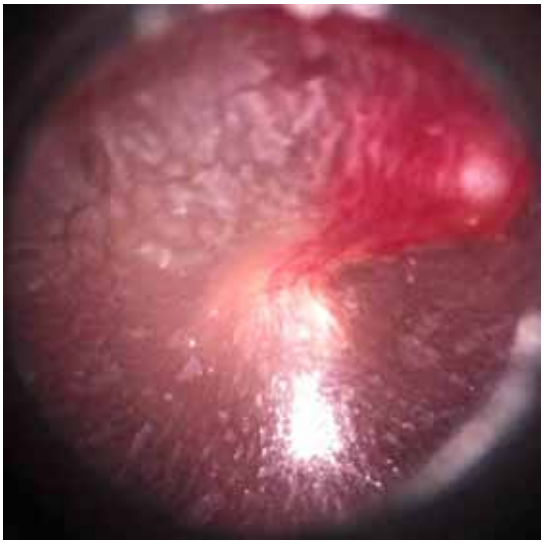
Grade 0: symptoms with no signs



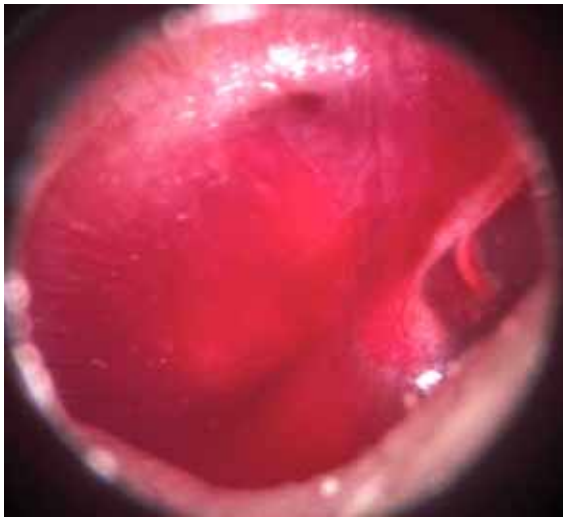
Grade 1: injection of the tympanic membrane (TM)



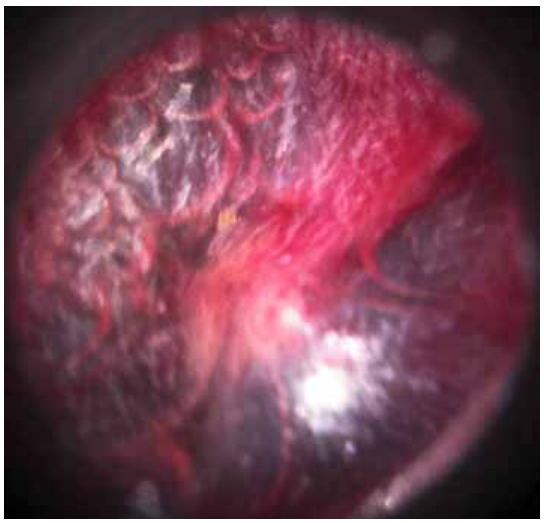
Grade 2: injection of the TM plus slight haemorrhage within the substance of the TM



Grade 3: gross haemorrhage within the substance of the TM



Grade 4: free blood in the middle ear, as evidenced by blueness and bulging



Grade 5: perforation of the TM

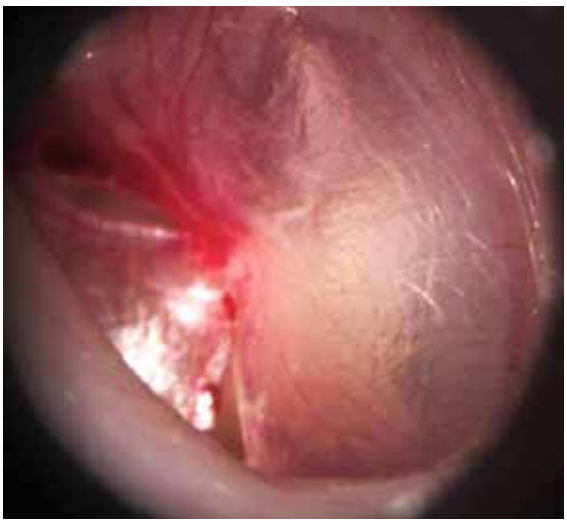


Table 2
Grade of MEBt in relation to language of instruction, median (inter-quartile range); there were no statistically significant differences between the two groups

	Grade of MEBt	
	Native language	Non-native language
1st pool session	1 (1–1)	1.5 (1–2)
2nd pool session	1 (1–1.5)	1.5 (1–3)
1st open-water dive	1 (1–2)	1 (1–3)
Last open-water dive	1 (1–2)	1 (1–2)

MEBt. Indeed, subjects instructed in a language other than their native language had lower rates of MEBt than students who were instructed in their native language ($P = 0.780$).

In logistic regression modelling for MEBt, including language of instruction and various potential covariates and confounders, there remained no significant association between language of instruction and MEBt (adjusted odds ratio = 0.82; 95% confidence intervals: 0.21–3.91). There was also no association between any of the potential explanatory and/or confounding variables and MEBt. Neither were there any statistically significant associations between the severity (grade) of MEBt and language of instruction (Table 2). When dichotomizing MEBt severity as grade < 2 or grade ≥ 2 there remained no statistically significant association. Two subjects had MEBt of unknown grade in one ear, one after the first and one after the second open-water dive.

Discussion

Ear problems are common in divers.[1–3,7] Otoscopic changes have been reported in 71.5%[7] to 100%[3] of ears in experienced divers after repetitive diving. In one survey, 52% of divers stated they had experienced ear 'squeeze' on at least one occasion.[7] Divers often seek medical advice for equalization difficulties encountered during confined water training,[2] and these difficulties can lead to open-water dive candidates not completing their diving certification.[9]

While the rates of MEBt at various stages of this four-day course are seemingly high, they are consistent with the previous literature, in which the reported incidence of MEBt in diving candidates ranged between 41% and 48% after confined-water sessions,[4,10,11] and up to 66%[12] after the first open-water session. While we do not have an extended-course control group for direct comparison, it does not appear that a condensed course increases the risk of MEBt.

About a third of the divers studied were instructed in a language other than their native language. The PADI open-water dive manual is an essential component of the candidate's education. Whilst this manual is available in 23 languages, most dive centres realistically cannot have instructors speaking all 23 languages. Completing the Open Water Diver course in their non-native language did not increase the incidence or severity of MEBt in the divers we studied, which is consistent with research from traditional education settings that has explored such things as students learning a second language[13] or the effect of non-native, English-speaking university teaching assistants on student mastery of content.[14,15] In those studies, minimal[14] to no decrease[15] in student performance was found when the teaching assistant did not speak the students' native language.

There were some associations between demographic characteristics and language of instruction that might have confounding effects in our data. Candidates instructed in a language other than their native language were younger, less likely to be female, and less likely to have prior scuba experience. However, in the logistic regression for MEBt that included these variables, there remained no significant association between language of instruction and MEBt.

Although this was not a focus of our study, we do note that despite the high incidence of MEBt, this did not prevent any candidate in this cohort from successfully becoming a certified Open Water Diver.

Limitations

Most Open Water Diver candidates in our study were tourists and, therefore, we were unable to follow the divers after the three-day, live-aboard dive trip. However, some candidates developed signs or symptoms of MEBt in the day following the course, their first day of diving as a certified diver. Documentation of these later equalizing difficulties was not done, though there appeared to be no increased incidence in candidates instructed in a language other than their native language.

Although we know that 25 candidates were instructed in a language other than their native language, we do not know whether these students accessed the PADI manual or additional educational materials (e.g., via the internet) in their native language. This may have influenced the incidence of MEBt in the non-native language group. We also did not assess English proficiency in the non-native language group; it is common in many countries for people to have a good working knowledge of English. These potentially mitigating factors, however, are not unique to our study population and would likely be equally present and equally effective at minimizing the effects on non-native language instruction in other settings.

Other than language of instruction, this study was not designed to elicit the predictors of or contributors to MEBt. As smoking, ENT pathology, medications and allergies were not incidentally associated with language of instruction in our sample, they were not included in our logistic regression modelling. That does not suggest that those variables

are not associated with MEBt; rather, only that, in this sample, those variables did not confound the relationship between language of instruction and MEBt. Previous scuba experience was more common among students instructed in their native language and, therefore, was included in our logistic regression. Although there was no association between MEBt and scuba experience, the small sample size likely limited the ability to detect such an association.

Finally, this analysis is a sub-analysis of a larger on-going trial exploring MEBt in open-water diver candidates. That larger study was not designed to specifically explore the effects of language of instruction, so there might be other explanatory or confounding variables that were not collected or evaluated. This study has a small sample size which does limit its statistical power. However, the raw rate of MEBt was lower in candidates who were instructed in a language other than their native language; a larger study would have to both strengthen and reverse the observed association between language of instruction and MEBt to achieve clinically meaningful significance.

Conclusion

Open Water Diver candidates instructed in a condensed, four-day course had a high incidence of MEBt, but it did not appear to be higher than the incidence of MEBt reported in previous studies. Training in a candidate's non-native language did not appear to increase the overall incidence or severity of MEBt.

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The prevalence of oro-facial barotrauma among scuba divers

Mohammed K Yousef, Maria Ibrahim, Abeer Assiri and Abdulaziz Hakeem

Abstract

(Yousef MK, Ibrahim M, Assiri A, Hakeem A. The prevalence of oro-facial barotrauma among scuba divers. *Diving and Hyperbaric Medicine*. 2015 September;45(3):181-183.)

Introduction: Barotrauma is a physical injury that results from ambient pressure changes during flying, diving or hyperbaric oxygen therapy. The aim of this study was to assess the prevalence of oro-facial barotrauma among a sample of scuba divers in Jeddah, Saudi Arabia.

Materials and methods: Data for the study were collected through a self-reported questionnaire that was distributed to 166 divers. The questionnaire was divided into two parts, in which the first part contained demographic data and the second part consists of multiple choices questions and a few open-ended questions discussing the different signs and symptoms of orofacial barotraumas.

Results: One-hundred-and-sixty-three divers responded. The most frequent symptoms during diving were dry mouth (51.9%), followed by clenching (32.5%) and temporomandibular joint (TMJ) pain (19.5%), while the most frequent symptoms after diving were dry mouth (22.7%) followed by clenching and facial pain (16.9%).

Conclusion: Clenching and dry mouth were common findings but are temporary in nature and do not warrant any dental intervention. TMJ and facial pain were also reported but were temporary. The use of commercial mouthpieces during diving may be related to more symptoms when compared with customized types.

Key words

Barotrauma; dental; scuba diving; pain; underwater medicine

Introduction

Scuba diving continues to be a popular sport. However, research conducted in the field of barotrauma and oro-facial problems associated with diving is relatively scarce.[1] Barotrauma, which is defined as a physical injury resulting from ambient pressure changes during flying, diving or hyperbaric oxygen therapy,[1,2] may be associated with different oro-facial complications, including barodontalgia (barotraumatic toothache),[3–6] sinus, myofacial and temporomandibular joint (TMJ) pain[5,7,8] and odontocrexis,[5] which is the loosening or fracture of restorations. The aim of this study was to assess the prevalence of oro-facial barotrauma among a sample of scuba divers in Saudi Arabia.

Materials and methods

The study is a descriptive, non-experimental, retrospective survey that was conducted in Jeddah, Saudi Arabia. Ethical approval was obtained from the Research and Ethical Committee of the Dental College at King Abdulaziz University, Jeddah.

Data for the study were collected using a self-reported questionnaire* that was distributed to 166 divers. Recruitment of participants was through the local scuba diving association. Also, divers who agreed to participate were encouraged to invite their fellow divers to participate. Before distribution, the questionnaire was initially surveyed as a pilot study to ensure clarity of questions asked. Before participation, each participant signed a consent form. The questionnaire was divided into two sections, the first contained demographic data and the second consisted of multiple choice questions and a few open-ended questions discussing the different symptoms and signs of orofacial barotrauma. The survey included questions about the presence of dental pain during or after dives, its location (upper or lower jaw), any facial pain or limited mouth opening, TMJ pain or clicking, dry mouth, and any problems with dental restorations or appliances, such as aspiration, loss or fracture. Participants reporting previous head and neck surgery or symptoms were excluded. Two authors surveyed the questionnaires and one entered the data for statistical analysis.

STATISTICAL ANALYSIS

Data were tabulated and analyzed using the Statistical Package for Social Science (IBM SPSS Statistics for Windows, Version 20, Armonk, NY: IBM Corp, USA). Frequencies for each answer to the questionnaires were calculated or the number of subjects responding to each question. The statistical differences between the prevalence of different oral barotrauma symptoms and signs were determined using chi-square tests for nominal data. The level of statistical significance was considered at $P < 0.05$.

Results

Questionnaires were distributed to 166 scuba divers of whom 163 responded (98%). Among the 163 divers, nine were excluded from the study because of previous injury

* **Footnote:** A copy of the questionnaire (in English) is available from the authors on request.

Table 1
The prevalence of odontocrexis *n* (%) among 151 scuba divers

	Divers	Fracture	Loss
Dental filling	107 (70)	19 (12)	23 (15)
Fixed prosthesis	34 (22)	27 (18)	5 (3)
Removable prosthesis	2 (1.3)	0	0

or surgery in the head and neck area. Responders were 15 females and 139 males. Mean age was 38.5 years (40.5 years for men and 32 years for women, range 14–63 years); the majority were in their twenties and thirties (70%).

The prevalence of oro-facial problems the divers had faced at least once during or after their diving activities is shown Figure 1. The most frequent symptom during diving was dry mouth (80 of the 154 scuba divers investigated, 52%), while the least was limited mouth opening (six divers, 4%) during dives. Jaw clenching occurred in 50 divers (33%) during diving.

Table 1 reports the prevalence of odontocrexis in divers who had dental fillings or fixed or removable prosthesis. Twenty-nine divers (19%) of the sample reported dental pain: six in the upper jaw; 11 in the lower jaw and 12 reported dental pain in both upper and lower jaws at the same time.

There was a highly significant association between TMJ pain and limited mouth opening ($P < 0.001$) and clicking ($P = 0.001$) (Table 2). When evaluating the relationship between the type of mouthpiece used and reported symptoms, TMJ and facial pain occurred more in divers using commercial mouthpieces compared to customized ones (Table 3).

Table 2
The association between temporomandibular joint (TMJ) pain and limited mouth opening and TMJ clicking ($P \leq 0.001$; $n = 154$)

	Limited mouth opening		Clicking	
TMJ Pain	Yes	No	Yes	No
Yes	4	18	7	15
No	1	131	10	122

Table 3
The type of mouthpiece and TMJ and facial pain ($n = 154$)

	TMJ pain (during diving)		TMJ pain (after diving)		Facial pain	
Mouthpiece	Yes	No	Yes	No	Yes	No
Customized (*n*)	1	10	1	10	2	9
Commercial (*n*)	26	116	21	121	15	127

Discussion

Barotrauma may lead to various effects on facial, oral or dental structures. Most symptoms in the present study occurred more often during than after diving. The high percentage of clenching and mouth dryness may be related to emotional stress or the cold environment during diving,[7] whilst breathing dry, compressed gases may may contribute to mouth dryness. Other symptoms, such as TMJ clicking, pain and limited mouth opening, may be the result of the downwards and backwards displacement of the mandible to varying degrees depending on the type of mouthpiece. Using commercial-type mouthpieces showed the largest difference in the position of the mandible from normal, while the customized-type displaces the mandible the least.[7]

Barodontolgia has been reported at a rate (21%) similar to that of this study.[5–7] It is related to various causes, such as to trapped gases, low temperature, pulpal embolism, prolonged vasoconstriction, dentinal tubule permeability, impacted teeth, recent extraction, recent restoration, recurrent caries or periodontal disease.[5–7] In contrast to our findings, previous studies of barodontolgia during diving have reported pain more commonly in the upper jaw.[5–7] This difference however, could be explained by the differences in existing dental restorations or dental appliances in upper and lower jaws which were not recorded in previous studies. One study reported that some divers may experience headaches related to TMJ stress following their dives,[8] but this was not supported in the present study.

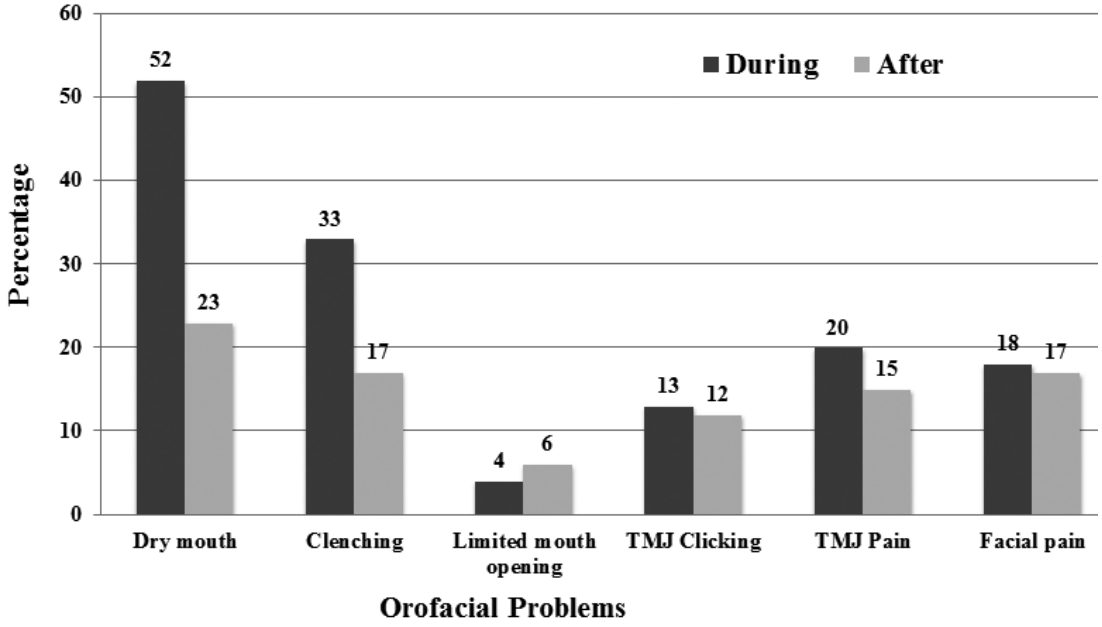
Dental barotrauma may result in restoration fractures or displacement by reducing the retention of the restoration.[7] It has been hypothesized that, with the pressure changes during diving, changes in the volume of air bubbles in the cement layer underneath prostheses can reduce the retention, and this may lead to displacement of the fixed prosthesis or of restorations.[7] Also micro-leakage may increase and retention may decrease in fixed prostheses that are cemented with zinc phosphate and glass ionomer cements.

Because of the method of recruitment of subjects, the study may have suffered from selection bias. Therefore, firm conclusions regarding prevalence cannot be drawn. Further studies are needed in this field since the literature is scarce in reporting oral and facial problems amongst divers. A similar study among professional divers is also encouraged.

Conclusion

Scuba diving can be considered as a safe sport with regard to orofacial barotrauma, but divers should undergo regular dental checkups and inform their dentists of their diving activities. Clenching and dry mouth were common findings but were temporary in nature and did not warrant dental intervention. Reported TMJ and facial pain was also temporary in nature. The use of commercial mouthpieces during diving may be associated with more symptoms when compared with customized types.

Figure 1
The prevalence of some oro-facial problems among scuba divers during and after dives; number of subjects shown



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Does self-certification reflect the cardiac health of UK sport divers?

Marguerite St Leger Dowse, Matthew K Waterman, Christine EL Penny and Gary R Smerdon

Abstract

(St Leger Dowse M, Waterman MK, Penny CEL, Smerdon GR. Does self certification reflect the cardiac health of UK sport divers? *Diving and Hyperbaric Medicine*. 2015 September;45(3):184-189.)

Background: Since 2009, the United Kingdom diving incident data show an increasing number of fatalities in the over-50s age group. Previous studies also suggest some divers take cardiac medications. Since 2001, diving medicals have not been mandatory for UK sport divers. Instead, an annual medical self-certification form, submitted to their club/school or training establishment, is required. We documented in a survey of UK sport divers the prevalence of cardiac events and medications and the frequency of medical certifications.

Methods: An anonymous on-line questionnaire was publicised. Measures included diver and diving demographics, prescribed medications, diagnosed hypertension, cardiac issues, events and procedures, other health issues, year of last diving medical, diagnosed persistent foramen ovale (PFO), smoking and alcohol habits, exercise and body mass index.

Results: Of 672 completed surveys, hypertension was reported by 119 (18%) with 25 of these (21%) having not had a diving medical. Myocardial infarction 6 (1%), coronary artery bypass grafting 3 (< 1%), atrial fibrillation 19 (3%) and angina 12 (2%) were also reported. PFOs were reported by 28 (4%), with 20 of these opting for a closure procedure. From 83 treated incidences of decompression illness (DCI), 19 divers reported that a PFO was diagnosed.

Conclusions: Divers inevitably develop health problems. Some continue to dive with cardiac issues, failing to seek specialised diving advice or fully understand the role of the diving medical. Physicians without appropriate training in diving medicine may inform a diver they are safe to continue diving with their condition without appreciating the potential risks. The current procedure for medical screening for fitness to dive may not be adequate for all divers.

Key words

Health surveys; recreational divers; cardiovascular; medicals – diving; fitness to dive

Introduction

Undertaking a diving medical when starting recreational scuba diving and thereafter at intervals determined by age (every five years to age 40, every three years to age 50, and thereafter annually) was mandatory in the United Kingdom (UK) until the year 2000. From 2001, recreational dive agencies in the UK required club divers to annually self-certify the state of their health by submitting a UK Sport Diving Medical Committee (UKSDMC) questionnaire to their club, and for dive school participants to complete a Recreational Scuba Training Council (RSTC) medical statement at each level of training.[1–4] Answering "*yes*" to any question requires a diver to seek advice from a physician. The UKSDMC form requires this to be from a diving physician, whilst this is unspecified on the RSTC form. The system is not nationally regulated or uniform, and the data are not collated centrally. Some divers may conceal health conditions which they perceive may threaten the acceptance of their ability to dive. Lack of knowledge concerning the physiology of diving by a diver, or a physician untrained in diving medicine, may place a diver at risk, with the potential interaction of a medical condition and the diving environment inadvertently going unrecognised.

The average age and the socioeconomic status of sport divers in the UK have changed over time.[5,6] There is now an older diving population who have access to technical diving equipment, allowing deeper, longer and more remote dives.[6] Since 2009, the British Sub Aqua Club's (BSAC) annual diving incident data show that the proportion of divers over 50 years of age is increasingly represented in the mortality data. Over the last five years, between approximately half and three-quarters of annual fatalities have been from this age group, though this may be a reflection of the average age of the diving population.[6] In contrast, UK mortality rates from coronary heart disease (CHD) have fallen in recent years, potentially attributable to improved treatment and risk factor modification.[7–9]

Two separate studies of UK sport divers regarding drug and alcohol usage showed 9% and 10% of the study participants were taking cardiac medications for either primary or secondary disease prevention.[10,11] Data concerning the national usage of primary and secondary disease prevention medications for cardiovascular disease (CVD) and CHD in the general population is less clear, and is not directly comparable owing to differing methods of data collection.[12–14]

The recent, apparent increase in mortality rates in the older diver age group and the consistent reports of cardiac medication usage in divers challenge the evaluation by specialists in diving medicine and the efficacy of self-declaration. The aim of this study was to gain an insight into the general cardiac health of UK sport divers, along with the manner and frequency of fitness-to-dive assessments. The study did not attempt to evaluate the risk associated with cardiac health and diving incidents but, rather, to question whether the UK self-certification and diving medical statement are reliable indicators of diver health over time.

Methods

An anonymous, observational, on-line questionnaire* was compiled using a combination of demographic questions designed, validated and used in previous field data studies.[5,10,11] The survey was available for completion for five months from August 2013 and was publicised through the DDRC Healthcare website, diving exhibitions and social media. Divers were free to participate at will and were not actively recruited.

Fixed-option questions included basic diving demographics (affiliation, year of first dive, year of most recent dive, total dives since learning, dives in the last twelve months, maximum depth ever dived), physician-prescribed medications, diagnosed hypertension, other health issues, year of last diving medical, first degree relative under 60 years of age with a history of cardiovascular issues, events and procedures, diagnosed persistent foramen ovale (PFO), PFO closure, smoking and alcohol consumption, exercise and body mass index (BMI; $\geq$ 30 kg·m^{-2} defined as overweight). Free-text answers provided the opportunity for divers to list current medications. Information was also gathered regarding situations leading to the diagnosis of PFO, and free text for other cardiovascular issues. The divers were also asked how they perceived their health condition and/or medication affected their ability to dive safely. In addition, divers were asked if they had ever had physician-diagnosed and treated decompression illness (DCI), or if they had experienced signs and symptoms they considered to have been DCI but had not sought advice.

The survey was successfully piloted for comprehension and data integrity. All data were anonymous, and checks for possible duplicate entries by scrutinizing and comparing dates of birth, gender, and diving demographics were carried out. Descriptive statistical analysis was used where appropriate. Data, where appropriate, are reported as median. Ethical opinion was sought from the National Health Service (NHS), Health Research Authority, NRES Committee South West, Cornwall and Plymouth, and written confirmation received that ethical review was not required.

Results

A total of 685 responses were received of which 13 were discarded owing to incomplete data, leaving 672 records (males 76%, females 24%; aged 12 to 78 years, median 46) to be analysed. Diving experience was from < 1 to 60 years (median 12). The approximate number of dives since learning to dive was from 5 to 15,000 (median 400) with a collective total of 609,000 dives. The number of dives in the last twelve months ranged from 0 to 980 (median 45). Maximum depth ever dived was from 4 to 207 metres of water (mw, median 50).

GENERAL HEALTH

Fifty (7%) of respondents were current cigarette smokers (1–30 per day), with 228 (34%) ex-smokers, having smoked between six months and 45 years ago. Within that group, 69 (30%) had ceased smoking within the last five years. Alcohol was regularly consumed by 462 (69%) of respondents (1–70 units per week). Of the 672 respondents, 175 (26%) exercised most days, 266 (40%) said they exercised three to four times a week, with the remaining 231 (34%) taking little or no exercise at all. Only 218 (34%) had a normal BMI, with 426 (66%) overweight or obese; two females were underweight and 26 respondents did not record their data. Of the 672 respondents 226 (34%) reported two or more of the four health risk factors: current cigarette smoking; exceeding the recommended upper weekly limit for alcohol consumption; exercising less than three to four times a week and a BMI $\geq$ 30 kg·m^{-2}.

Asked if respondents' blood pressure, cholesterol, and blood glucose had been checked in the last 12 months, 240 (36%) said all three had been checked and 280 (42%) reported having one or two checked. No checks at all in the last 12 months (or no record) were reported by 155 (23%).

HYPERTENSION

Physician-diagnosed hypertension was reported by 119 (18%), with 41 (34%) of this group either having no diving medical for more than 10 years or none at all. A broad range of cardiac medications had been prescribed to 60 of these 119 (50%, Table 1) whilst exercise, weight-loss, and dietary changes had been recommended by their physician for the remaining 50%. Thirty-four (29%) belonged to technical diving organisations.

CARDIAC MEDICATIONS

Categories of cardiac medications reported by 60 respondents are shown in Table 1, with some respondents using more than one category. Not all respondents who reported cardiac issues, events and procedures (n = 64) gave detailed information regarding their medications. Of the 19 respondents reporting atrial fibrillation, none specified whether they were anticoagulated with a coumarin (warfarin/acenocoumarol) versus a novel oral anticoagulant (dabigatran/rivaroxaban/apixaban).

CARDIOVASCULAR ISSUES, EVENTS, AND PROCEDURES

There was a total of 64 (10%) in this group, with 14 of the 64 reporting more than one issue, event or procedure; five of this sub-group had either no medical or none for more than 10 years. Details are provided in Table 2. Four of the

* **Footnote:** A copy of the questionnaire is available from the authors on request.

Table 1
Respondents reporting use of cardiac medications (n = 60); some respondents reported more than one category

Categories	
Angiotensin converting enzyme inhibitors/ Angiotensin-II receptor antagonists	47
Lipid lowering agents	17
Unspecified anticoagulant	14
Antiplatelet drugs: aspirin (11) clopidogrel (3)	14
Diuretics	4
Calcium-channel blockers	2
Beta-adrenoceptor blockers	3
Alpha-adrenoceptor blockers	3
Anti-anginals	1

six respondents reporting myocardial infarction had been treated with stents; one who was a technical diver reported having suffered three episodes of infarction and had logged 120 dives in the last 12 months. All three coronary artery by-pass grafting respondents were males, aged 55, 68 and 70 years. All were experienced divers with ≥ 1,200 dives, two being technical divers. Two had been cleared for diving by their cardiologist and the third had sought advice from outside his home area. A respondent aged 65, who reported having 8 stents, had logged 20 dives in the last 12 months and 1,500 dives in 26 years. He stated "*I am an Advanced Instructor and do about three trimix dives per annum to extreme depths between 60 to 80 metres*". One respondent, aged 54, reported an implanted pacemaker which had been fitted in 2010 (manufacturer and model undisclosed). He had 22 years' experience, averaging approximately 29 dives a year and had logged 35 dives in the last 12 months. His last diving medical was in 2011.

PERSISTENT FORAMEN OVALE

PFOs were reported by 28 of the 672 respondents (4%; age 32 to 63 years, median 47) with a diving experience of 1 to 41 years (median 14). Twenty of these proceeded to PFO closure and 16 returned to diving. Seven of the eight who did not undergo closure returned to diving. PFO after an episode of DCI was diagnosed in 22 of the 28 PFO respondents. Of the remaining six, three had been tested due to migraine and three did not specify. Of the 11 technical divers, eight opted for closure.

The majority of divers who had a procedure to close their PFO did so for one of the following reasons: in order to continue diving; to avoid a possible stroke; to avoid another DCI and to avoid making major changes to dive profiles. The 16 divers who returned to diving after PFO closure had logged a collective total of 2,683 dives post closure, (range of 15–400, median 90). The maximum depths dived ranged from 25 to 65 mw (median 43.5 mw). Six respondents changed their diving practices and continued to dive without PFO closure and reported more conservative profiles, greater care in ascent rates, extra stops and self-imposed depth limits; three were technical divers.

DECOMPRESSION ILLNESS

There were 84 (12%) respondents who reported physician-treated and/or diagnosed DCI, whilst 56 (8%) respondents reported self-diagnosed symptoms and signs of DCI without obtaining medical advice; 18 respondents out of these two groups reported both self-diagnosed and physician-diagnosed DCI.

Discussion

The divers in this study were active and dived more regularly than might be expected from some sport diving groups in other countries. In the UK, there is a well-entrenched culture of diving year round, both within club and regular dive centre groups. Additionally, not all divers log their dives in the same format, with some UK divers recording every training dive in all circumstances. The diver and diving demographics in this data set were similar to previous diving studies and were from across all active diving organisations in the UK.[5,10,11]

The study design did not allow for follow up, a source of potential bias in anonymous surveys which may exist in this study. The self-selecting nature of the survey introduces bias such that some divers respond because they feel they

Table 2
Cardiovascular issues, events and procedures reported by 672 diver respondents
Age (years) – median (range); PFO – persistent foramen ovale

Cardiovascular issue	n	Age	Years diving	Technical diver	Diving medical (none/>10 years)	Comment
PFO	28	47 (32–63)	1–42	11	9	20 closures and 16 returned to diving
Atrial fibrillation	19	53 (26–66)	2–44	3	5	7 also with hypertension
Angina	12	60 (39–70)	6–51	5	3	2 type 2 diabetes
Coronary stent	11	63 (47–78)	15–60	4	3	3 respondents reported 3, 4 and 8 stents
Myocardial infarction	6	62 (47–78)	15–60	3	3	2 family history of cardiac disease
Coronary bypass	3	68 (55–70)	13–49	2	3	1 "*suffers from cold induced angina*"

have something to report; conversely others may not respond due to reluctance to admit they are diving with a condition which may negatively impact on their diving safety. Additionally, only those who are still active divers will respond, thus precluding the participation of those who may have ceased diving for health reasons, and those who have died. However, the strength of anonymous methodology is that it allows the covert respondent to contribute data in the knowledge that they will not be challenged in any way, enabling the researcher to gather data that might otherwise remain unreported.

In this study, divers reporting cardiac health problems had not sought diving physician advice when recommended or had undergone a diving medical.[3,4] These data are consistent with other studies where 9% of divers were shown to be taking some type of cardiac medication.[10,11] Some respondents were diving with medical conditions and medications that potentially placed them or their buddy at risk whilst diving. This suggests the need for further diver health education during training regarding such risks as immersion pulmonary oedema. The data also imply that some non-diving medical practitioners may not account for the interactions between a patient's health, their drug regime, and the physiology of diving. Two respondents with coronary artery bypass grafting stated they had been cleared to dive by their cardiologist. It was unclear whether these practitioners had any training in diving medicine. One respondent sought advice from outside his home area, perhaps suggesting the medical practitioner consulted may not have had full knowledge of the diver's health issues. The risk of in-water incapacitation, exacerbation of an existing condition, risk to fellow divers or the increased risk of a diving-related injury may not be appreciated.

Few studies have specifically addressed the efficacy of self-certification regarding fitness to dive. In a report on the first three years of self-declaration in Scotland, in which records were processed centrally, the number of forms referred to a diving physician for review increased from 1.2% the year self-declaration commenced to 7.7% after three years.[1,15] Analysis of diving incidents over the three years showed no incident was caused by an unknown medical condition. It was concluded that the system was identifying divers who should not be diving, but it was also noted that there was an increase in the number of divers who refused medical assessment when it had been recommended. The study did not take into account divers who did not complete a self-certification form, or the remainder of the UK where there is no central collation of the data. In a group of 1,000 consecutive entry-level divers in Australia, one in 70 divers indicated they had no relevant medical problems on a self-certification medical form, but then were failed during a face to face medical consultation with a single physician trained and experienced in diving medicine.[16] It was concluded that self-certification forms may not necessarily identify individuals who are at risk whilst diving.[16]

The number of divers in our study with significant medical problems who had no diving medical or one that was more than 10 years old is of concern. The data showed a number of respondents to be diving who had not taken diving physician advice for their condition or medications, a small number of whom would likely be deemed unfit to dive. Within the UK diving fraternity, doubts have long been expressed with regard to the reliability or accuracy of some divers when self-certifying their health, aware that divers can be reluctant to acknowledge health problems, particularly if it might prevent them diving.

The data in Table 1 are from divers who were physician-diagnosed with hypertension. A further 27 respondents did not record an answer to this question, but subsequently listed medications prescribed for hypertension or vascular protection in another section of the questionnaire. These respondents may lack an understanding of their medical condition, or are perhaps unable to recognise the limitations of their cardiovascular health on diving safety. Whether or not there was a formal diagnosis of hypertension in this group, the reported medications suggested these divers were deemed to have sufficient hypertension risk score to warrant medication. Of additional note was the number of technical divers who were in these sub-groups.

Although the proportion of the adult UK population taking cardiac medications is not known with any accuracy, the data in this study reflect other published literature with 9% of the respondents on cardiac medication, and 10% reporting a cardiac event or procedure.[7–11,17] In an Australian survey, 10% of responding divers reported hypertension or coronary heart disease. The reliability of divers to disclose their health conditions prompts the question as to whether medical screening should take place at regular intervals.[18] Other investigators have also expressed concern regarding the cardiovascular health of the older diver.[19–21]

PFO and the associated risk of DCI have been discussed previously.[22–24] Undiagnosed PFO in the general population is estimated to be approximately 20–30% suggesting a similar percentage of divers would be expected to have a PFO. The incidence of DCI is estimated to occur in 0.005–0.08% of dives, with the estimated risk of a DCI incident in divers with PFO between 0.002 and 0.03% of dives.[25] As a result, it is generally agreed that PFO screening of all scuba divers would not meet the criteria for successful screening programmes and is not recommended.[22–25] There is less agreement with regard to when it becomes desirable to screen an individual diver who may be considered at risk; and funding from the UK NHS to undertake PFO closure is not currently forthcoming. In our study it was not possible to establish from the respondents, who reported a diagnosed and/or treated DCI how many had been screened for a PFO prior to DCI. Very recently a joint statement on PFO and diving has been published by the UK Sport Diving Medical Committee and the South Pacific Underwater Medicine Society.[26]

This survey questions the effectiveness of self-certification and opens the debate for what changes to the system could be made to identify those with high-risk health issues. It is debatable as to whether fitness to dive medicals would improve the current situation and a national central data collection would have to be implemented for any effective result. Although the UK NHS is free at the point of care, assessment for fitness to dive is not so. Diving medical advice generally results in a fee and many divers do not feel disposed to pay for such a service. UK sport divers are also not required to purchase diving health insurance. These facts, together with the self-certification health questionnaire requirement, may contribute to the lack of rigorous health surveillance and/or accurate self-certification by UK divers.

Conclusion

A range of cardiovascular issues were reported by divers of all levels, including technical divers. Divers were also taking a range of cardiac-related medications. Not all divers who reported cardiovascular issues had sought appropriate medical advice. As divers progress through their diving career, some inevitably develop health problems and continue to dive. The recreational diving population appears to be aging and may be less fit. Divers with many years' experience have also grown into their medical conditions over the years.

The scrutiny and requirement to self-certify at given time points varies, and there is no mandatory central point for collection of self-certification data in the UK and the system is not universal, regulated or coordinated. The current system is reliant on honesty and an assumed level of knowledge by the diver. Some forms of self-certification encourage reliance on the opinion of non-diving medical practitioners or specialists who may not understand the pathophysiology of diving. These data raise the question as to whether the current system is fit for purpose.

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

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

Back articles from DHM

After a one-year embargo, articles from *Diving and Hyperbaric Medicine* are placed on the Rubicon Foundation website <http://www.rubicon-foundation.org/>, an open-access database, available free of charge and containing many other publications, some otherwise unobtainable. At present, this task is not fully up to date for DHM but articles to the end of 2012 are now available.

Rubicon seeks donations to continue its work to document the hyperbaric scientific literature.

More recent articles or other enquiries about articles should be sent to: <editorialassist@dhmjournal.com>
Embargoed articles will be charged for; details on application.

Review articles

Underwater blast injury: a review of standards

Rachel M Lance and Cameron R Bass

Abstract

(Lance RM, Bass CR. Underwater blast injury: a review of standards. *Diving and Hyperbaric Medicine*. 2015 September;45(3):190-199.)

The first cases of underwater blast injury appeared in the scientific literature in 1917, and thousands of service members and civilians were injured or killed by underwater blast during WWII. The prevalence of underwater blast injuries and occupational blasting needs led to the development of many safety standards to prevent injury or death. Most of these standards were not supported by experimental data or testing. In this review, we describe existing standards, discuss their origins, and we comprehensively compare their prescriptions across standards. Surprisingly, we found that most safety standards had little or no scientific basis, and prescriptions across standards often varied by at least an order of magnitude. Many published standards traced back to a US Navy 500 psi guideline, which was intended to provide a peak pressure at which injuries were likely to occur. This standard itself seems to have been based upon a completely unfounded assertion that has propagated throughout the literature in subsequent years. Based on the limitations of the standards discussed, we outline future directions for underwater blast injury research, such as the compilation of epidemiological data to examine actual injury risk by human beings subjected to underwater blasts.

Key words

Underwater hazards; blast; injuries; diving incidents; pathology; risk assessment; review article

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Introduction

This article summarizes the development of the current major guidelines used for underwater blast, illustrates how insufficiently and inconsistently they attempt to predict injury, and offers a direction for the establishment of validated human underwater blast injury criteria. While injuries from blast in air have been extensively studied and quantified, the level of blast exposure that produces injuries from underwater blast still remains mysterious.[1–6]

The first documented cases of underwater blast injury occurred in 1916 during WWI, but even as recently as 2001 underwater blast researchers have acknowledged that there are still no scientifically based criteria for predicting injury or death.[7,8] Over 1,500 underwater blast-related casualties were identified in case studies during WWII alone; presumably a far larger number occurred but were not identified in medical publications.[9] This lack of criteria is certainly not due to lack of scientific effort. Hundreds of papers have been published on the subject, but the resulting injury guidelines have been grossly inconsistent, and often poorly scientifically founded.[8,10]

Background

Explosions in air typically injure through any of four general categories: primary blast from direct effects of the shock wave; secondary blast from energized projectiles, tertiary blast from whole body translation and quaternary blast from effects of inhaled gases and other sources.[11] However, the increased density and viscosity of water relative to air mean that underwater blast injuries occur almost exclusively as the direct result of overpressure, or primary blast. This type of injury is the result of the energy of the shock wave interacting with the tissues of the human body.

Shock waves from blasts travel faster than the speed of sound in a given material. The speed of sound and, therefore, the speed of the shock wave depend on the density of the medium that the wave is travelling through. Therefore, shock waves transiting material interfaces, especially in the transition from denser to less dense interfaces, may deposit energy near those interfaces. The most vulnerable, easily injured tissues in the human body are those that contain air, such as the lungs, intestinal tract, and the airspaces of the ears.[11] Since the speed of sound in and material density in lung or intestinal tract is much less than in the surrounding tissue, shock wave transit may damage sensitive lung or gut tissues. Typically pulmonary injuries occur through spalling, when the alveolar surfaces rupture and bleeding into the lungs occurs. Symptoms of pulmonary injuries can include coughing, difficulty breathing, haemoptysis, and apnoea.[12,13] Abdominal injuries generally include perforation or tearing as well as haematoma and ecchymosis. These injuries can occur at any point along the large and small intestine, but have a tendency to cluster near the ileocaecal junction.[14–17] The pulmonary injuries are very similar to those seen in air blast.[1,2] However, abdominal injuries typically manifest in air blast only if the lungs are shielded in some way,

e.g., by the presence of a bulletproof vest.[18,19] Injuries generally increase in severity with exposure to a 'stronger' blast.

There are many factors that determine the 'strength' of a blast exposure. These include peak positive overpressure, duration of overpressure, total energy of the blast, and maximum area under the pressure-time curve. This area, called the impulse, is often equal to the area under the positive phase of the pressure-time curve, but may be further augmented by bottom reflections or bubble action following the initial shock wave. While it is likely that multiple variables are necessary to accurately predict injury risk across the range of reasonable exposures, methods of predicting injury risk from underwater blast have historically used one of three physical criteria to describe exposure: 1) explosive charge weight and range, 2) blast (explosion) impulse, or 3) peak positive overpressure.

Methods

HOMOGENIZATION OF GUIDELINE TYPES

As discussed above, there have historically been three types of guidelines used to describe risk from underwater blast. These guidelines are difficult to compare directly because they measure different physical quantities. To compare the guidelines together, a representative underwater explosion was simulated using the US Navy's Gemini Solver.[20–24] As well as simulating the underwater detonation, the Gemini Solver simulates the propagation of the resulting shock wave. The programme receives as inputs the variables describing the blast scenario, and computes the complex waveforms produced by the underwater explosions. The resulting pressure and impulse values were used to assign theoretical distances from the explosive charge to the values given by those guidelines. Gemini has been extensively experimentally validated.[20, 25]

To consistently estimate the blast parameters, a representative case simulated the detonation of 136 kg TNT with pressure curves sampled at distances of 5 to 200 m from the centre of the charge. Both the explosive charge and the sampled curves were 40 m below the surface of the water, with a free lower boundary condition to simulate deep open water. This case was used to establish example mathematical relationships between range, the distance from the charge centre, and peak pressure. Similarly, the relationship was also established between range and impulse. These functions are shown as equations (1) and (2), and describe predicted range (R) as a function of peak pressure (P_m) and impulse (I) ($R^2 = 0.997$ and 0.995, respectively). Equations (1) and (2) are not the direct output of the Gemini Solver, but rather empirical curve fits to the outputs that the Solver calculated at the sampled locations. These equations are valid only for this specific underwater blast scenario with 136 kg TNT charge mass. They were used only as tools for direct comparison of guideline types and are not valid as generalized descriptions of all underwater blast scenarios.

$$R(P_m) = 332.8e^{(-1.62x10^{-3})P_m} + 64.6e^{(-1.41x10^{-4})P_m} \quad (1)$$

$$R(I) = 258.7e^{(-8.68x10^{-4})I} + 60.5e^{(-7.58x10^{-5})I} \quad (2)$$

Using these equations, guidelines that provided peak pressure or impulse values were converted to range values so that they could be directly compared. The guideline types were assigned numerical values of 3 for 'Safe/Deterrent', 2 for 'Danger/Injury', or 1 for 'Lethal' for analysis. Linear regression analyses were performed to assess the consistency of the guidelines. For the regression analyses, the guidelines from Richardson (1991) were omitted as they were never intended to be applied to humans.[26]

GUIDELINES BASED ON PEAK PRESSURE

The most common type of guideline for underwater blast injury provides a recommended maximum overpressure. Researchers' attempts to apply a peak pressure-based guideline originate from the successful use of peak pressure and overpressure duration to predict injury risk from air blast.[1,2] However, in air blast, the entire blast waveform can often be described using an ideal overpressure (Friedlander) wave and an easily-identified duration, while in water there is no such simple formula for prediction of the pressure trace.[27,28] The waveform resulting from an underwater blast is substantially affected by variables such as depth of explosion, depth at point of measurement, bottom depth, and bottom composition/topography.

There is no single equation that accurately describes a generalized waveform for underwater blasts. The wide variation in the shape of underwater waveforms often makes it difficult to identify the duration of the blast exposure, which is important in the wounding process and in estimating the injury risk and severity. The guidelines based on peak pressure are presented in Table 1, with each recommended maximum peak pressure given in both psi and kPa for consistency. The ranges at which these recommended maximum peak pressures will occur for the representative 136 kg TNT explosion, as calculated by equation (1), are also shown in Table 1. The same guidelines are shown graphically in Figure 1 as a function of the year they were published.

Most of these references were based on unscaled, summarized animal data,[30,37,38] or referenced no data at all.[39,40] The current US Navy 'injury' standard of 500 psi appears to be derived from a paper by several prominent Naval Medical Corps researchers, Greaves, Draeger, Brines, Shaver, and Corey.[32] They allege in their 1943 paper that when a compression wave of 500 psi or greater reaches the surface of the water "*it breaks through into the air with a shredding effect and literally 'blows off' the surface*". This logic was then extended to the human body, claiming that when a compression wave greater than 500 psi is transmitted

Table 1
Guidelines based on recommended peak pressure of exposure; * calculated ranges, based on test case;
† reference describes use of experimental data in guideline development

	Reference	P_{max} (psi)	P_{max} (kPa)	Range* (m)	Notes
SAFE/DETERRENT	Ellis, 1944[29]	25	172	315	„Safe" level used in unpublished Chesapeake Bay trials, 1942; no disclosure of original basis
	Christian, 1974[30]	125	862	139	Based on unscaled, summarised sheep data (no reference provided)
	Navy Dive Manual, Rev. 6[31]	50	345	252	No reference provided; seems to trace back to assertion in Corey[34]
	Fothergill, 2002[32]	0.03	0.2	--	High-frequency sound, not blast (omitted from figures, provided for reference only)
	Ainslie, 2008:[33] NATO DMAC (safe) DMAC (deterrent)	 0.1 0.9 11.5	 0.7 6.3 79	--	High-frequency sound, not blast (omitted from figures, provided for reference only)
DANGER/INJURY	Corey, 1946[34] †	300	2,068	60	Based on experiments with strips of intestine inside diving dress; attempted to model the human torso surrounding the intestine, but ignored that majority of perforations occur near ileocaecal junction, suggesting a contribution by surrounding support structures (which would lower the pressure required).
	Draeger, 1946[35]	250	1,724	71	Quotation of guideline presented by Corey;[34] approximated number based on experiments with beef intestine as well as unreferenced experiments at Fort Pierce
	Greaves, 1943[36]	500	3,447	41	Based on plumes of water "*shredding*" at the surface during a ~500 psi blast wave; no data referenced or presented
	Navy Dive Manual, Rev. 6[32]	500	3,447	41	No reference provided; seems to trace back to Greaves, 1943[36]
	Williams, 1944[37]	800	5,516	30	Based on unscaled summarised animal data; no reference provided
LETHAL	Bebb, 1951[38]	650 1,800	4,482 12,411	35 11	Alleged fatal exposure from charge W = 136 kg at unspecified range, based on unscaled animal data and possibly clinical experience Alleged fatal exposure from charge W = 4.5 kg at unspecified range, based on unscaled animal data
	Committee on Amphibious Operations, 1952[39]	300	2,068	60	No data provided; adds caveat that most of the data to date is invalid because of lack of experimental pressure measurements and experimentation conducted in shallow water
	Draeger, 1946[35]	500	3,447	41	Apparent quotation from Greaves[36] (reference reads only "*Draeger and others*")
	Navy Dive Manual, Rev. 6[32]	2,000	13,790	9	No reference provided
	Rawlins, 1953[40]	500 1,200	3,447 8,274	41 20	Purported LD50 for a rat; no scaling for humans; no data provided Purported LD90 for a rat; no scaling for humans; no data provided
	Williams, 1944[37]	1,300	8,963	18	Unscaled; summarised animal data (no reference provided)

Table 2
Blast injury guidelines based on charge weight and range; all equations converted to metric units (kg TNT, m);
† calculated ranges based on 136-kg TNT charge weight

	Reference	Weight (kg TNT)	Given Range (m)	Range† (m)	Notes
SAFE/DETERRENT	Committee on Amphibious Operations, 1952[39]	$R = 25.4*W^{1/3}$		131	Application of scaling law to P_{max} = 200 psi (1,379 kPa); basis for 200 psi guideline not given
	Ellis, 1944[29]	2.3	10.1	--	Cites experiments by Williams, but no published data was found
	Greaves, 1943[36]	136 272	50 67	50 –	Based on unscaled animal data, pressure approximated using scaling laws
	Wright, 1950[42]	$R = 18.1*W^{0.5}$		211	As cited by Cudahy[8]; reference describes experimental data
DANGER/INJURY	Cameron, 1947[43]	136	5–640	5–640	Anecdotal range based on undocumented medical case reports
	Committee on Amphibious Operations, 1952[39]	0.45 45	20 91	--	Based on anecdotal information; no data source or reference provided
	Cudahy, 2001[8]	$R = 10.8*W^{1/3}$		56	Application of scaling laws to Navy Dive Manual guideline of 500 psi (3,447 kPa)
	Ellis, 1944[29]	136	229	229	Cites experiments by Williams, but no published data was found
	Nedwell, 1988[44]	$R = 22.5*W^{0.2}*d^{0.33}*h^{0.33}$		686	(d = diver depth, h = charge depth) As cited by Cudahy[8]
	Rawlins, 1953[40]	100	700	--	Given weight and range values intended to match P_{max} = 25 psi (172 kPa); air blast injury criteria are adapted from White, 1961[45] and grouped with water blast injuries
LETHAL	Auster, 1943[46]	136	< 91	91	Charge weight assumed as standard US depth charge, given range refers to authors' experience with survival rates of depth-charged sailors
	Bebb, 1951[47] Zuckerman, 1969[48]	$R = 3.17*W^{1/2}$		37	Equation first appears in Bebb,[47] then is cited in English units in Zuckerman[48]; square root scaling of 500 psi guideline for 300-lb depth charge
	Committee on Amphibious Operations, 1952[39]	$R = 17.5*W^{1/2}$		204	Application of scaling law to P_{max} = 300 psi (2,068 kPa)
	Ellis, 1944[29]	136		30	Based on clinical experience

through the torso and reaches the airspace within the lungs, it tends to "*blow off the surface of the tissues exactly as it blows off the surface of the water*", damaging the alveolar walls.[36] This physically and physiologically unlikely assertion has never been tested, neither at the time of publication nor subsequently, yet it has propagated through the decades as the definitive guideline for underwater blast injury. Even as early as 1944 some experimenters tried to cast doubt on the guideline using clinical experience[29] but, without a scientifically substantiated replacement, the 500 psi guideline continues to be published.[8,32,35] The US Navy 'safety' standard of 50 psi seems to be a simple reduction of this 500 psi value by an order of magnitude; no justification, testing, or validation of the 50 psi value could be found.

GUIDELINES BASED ON CHARGE WEIGHT AND RANGE

One medically and operationally useful type of guideline is a severity assessment by standoff range (distance of

Figure 1
Peak pressure guidelines shown vs. the year they were published; no visual trends can be seen with year of publication; injury severity levels overlap and show little separation, even when published within the same decade

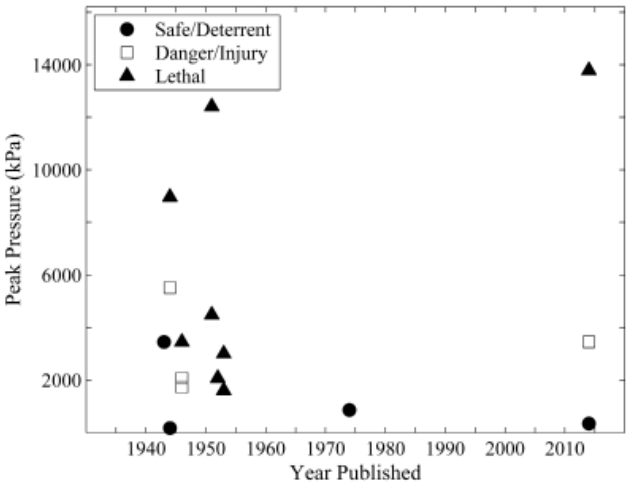
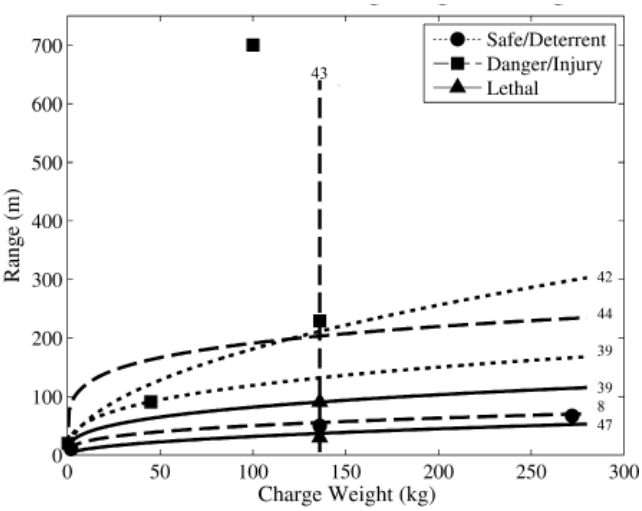


Figure 2
Guidelines based on charge weight and range; guidelines that were given in the form of ranges or equations are cited in the figure so that they may be related to Table 2; guidelines show no visual trend, as several 'safe' exposures lie within the ranges predicted by some 'lethal' guidelines



person from charge) based on charge weight. This type of guideline would be useful because it could be implemented without complex calculations and straightforwardly used in initial clinical severity assessments with estimated ranges. Many researchers have attempted to create such a guideline, usually based on the application of blast scaling laws such as equation (3) to peak pressure guidelines.[41] Table 2 outlines the blast injury guidelines that prescribe a standoff range based on charge weight. These guidelines are also plotted in Figure 2.

$$P_m = 21{,}600\left(\frac{W^{1/3}}{R}\right)^{1.13} \quad \text{(lb TNT·ft·psi)} \qquad (3)$$

$$P_m = 52{,}390\left(\frac{W^{1/3}}{R}\right)^{1.13} \quad \text{(converted to kg TNT·m·kPa)} \qquad (4)$$

Many of these guidelines provide equations for range based on charge weight, but several researchers also gave exact ranges from specific charge weights without extrapolating to a generalized equation. These specific guidelines are typically based on the clinical experiences of the authors, who published statements summarizing their experiences treating blast victims from WWII.[17,29,36,39,40,46]

While these guidelines are based on human data, the cases involve sailors at the surface of the water; proximity to the surface alters the pressure waveform sufficiently that these data points cannot be used to create standards for any degree of immersion. The surface of the water reflects a negative pressure (rarefaction) wave that mitigates the positive pressure of the blast wave, so a sailor at the surface will receive a significantly lower exposure level than a fully immersed diver, even at the same distance from the charge.[27] Therefore, to create standards for immersion, the exposures for sailors at the surface would need to be carefully computed or measured, taking into account the effects of the surface.

With the exception of the guidelines from Nedwell (1988), all of the range equations are derived from the application of different scaling laws to peak pressure guidelines.[44] The US Navy 500 psi guideline is by far the most frequent source for the development of these range equations.[8,38,43,48] Scaling laws can similarly be applied to determine standoff ranges in air blast, but are less complicated by effects specific to the in-water environment.[49]

GUIDELINES BASED ON IMPULSE

The blast impulse is a measure of intensity based on the maximum of the cumulative area under the pressure-time curve. It is related to the amount of blast momentum delivered to the person. Underwater, there are many factors that can influence the impulse, leading to high variability in impulse depending on circumstance. Additionally, some historical research groups prior to the advent of modern computers used differing methods for the calculation of impulse, potentially adding to the variability even further. Ideal explosives like TNT and C-4 show a tight coupling between peak pressure and impulse, but for non-ideal explosives such as thermobaric, aluminized explosives and for blasts near reflecting surfaces, peak pressure and impulse may be essentially independent.[41] Some coupling of peak pressure and impulse variables is present both in air and in water for ideal explosives.[49] Both sets of data were fit with second-order exponential decay functions.

It has long been postulated that the true destructive force of an underwater blast is linked more closely to the impulse than to the peak pressure.[27] This assertion has yet to be

Table 3
Blast injury guidelines based on impulse † calculated ranges based on 136-kg TNT test case; ‡ reference describes experimental data

	Reference	Impulse (kPa*msec)	Range (m) †	Notes
SAFE/DETERRENT	Christian, 1974[30] British Standard 5607[57]	14	316	No reference provided for limits; guidelines seem to be based on Richmond;[53] this guideline is sometimes referred to as the Gaspin Criteria and presented in English units as 2 psi*ms
	Richardson, 1991[26] ‡	0.212	319	Equation in reference: LN(Impulse) = 3.68 + 0.3857*LN(weight) solved using 77 kg; designed to predict injury risk for aquatic mammals
	Richmond, 1973[54] ‡	14–21	314–316	Injury threshold of 10 psi*msec (69 kPa*msec) determined using unscaled animal experiments; this threshold was then lowered to 2-3 psi*msec (14-21 kPa*msec) to provide a factor of safety
	Richmond, 1977[53] ‡	6.9	318	Experiments based on preliminary guidelines set by Richmond[54]
	Wright, 1950[42] ‡	138–276	263–289	Based on experiments listed in reference
	Yelverton, 1981[55] ‡	1.4×10^{-5}	319	Animal data with maximum animal mass 45 kg; mostly fish (have a different injury mechanism)
DANGER /INJURY	Zuckerman, 1969[48]	690	200	Stated with an accompanying P_{max}= 1800 psi (12.41 MPa); however, states higher impulses prove fatal even at lower P_{max}
LETHAL	Richardson, 1991[26] ‡	0.801	319	Equation in reference: LN(Impulse) = 5.01 + 0.3857*LN(weight) solved using 77 kg; designed to predict fatality risk for underwater mammals, not explicitly stated as relevant for humans
	Stuhmiller, 1991[58]	600	212	LD_{50} value stated but unreferenced; all other underwater blast information referenced to Richmond papers;[53,54] no data referenced
	Zuckerman, 1969[48]	827	183	Impulse value ascribed to guidelines from Bebb[47]

proved physiologically, but has been reiterated consistently through medical case reports and underwater blast analyses since World War II.[30,40,42,50–55] Impulse has become the standard for predicting destruction of buildings and other structures, but little experimental data was found in the literature to either support or refute conclusively the same assertion for physiological damage.[56] It seems unlikely that the contributions of peak pressure and impulse can be statistically separated for ideal explosives in underwater blasts, as the two variables are very well correlated.

The complexity of underwater blast waveforms means that impulse-based guidelines were of limited utility before validated computational models were developed to predict underwater blast. Even if the guidelines were solidly based in experimentation, they could only be used in environments that were similar to previously obtained test data. Table 3 outlines the blast injury guidelines based on impulse, along with their calculated ranges for the test case. The guidelines are shown plotted in Figure 3. The purpose of this figure is to show that there is no discernible trend in the guidelines over time (i.e., becoming more or less conservative) and to maintain consistency with the other guideline types.

Results

The distances prescribed by the different injury and lethality guidelines are plotted in Figure 4. The guidelines themselves are listed in detail in Tables 1–3, and discussed in detail in the previous sections. A total of 42 different guidelines were evaluated. When possible, these guidelines were traced back to their original sources. Of these 42 guidelines, only six were found to have associated experimental data; however, even these six still showed gross inconsistencies. The publications with these six guidelines are described in more detail in the Discussion section.

Figure 3
Guidelines based on impulse shown vs. the year they were published; guidelines span several orders of magnitude, the only discernible relationship being the prescription of extremely low impulse values as 'safe' following the 1970s

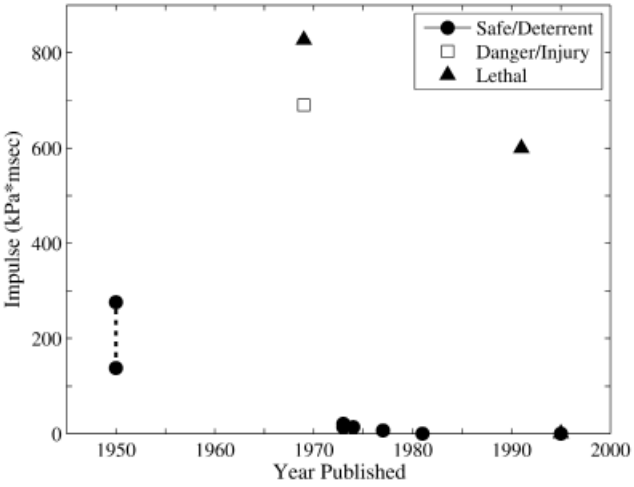
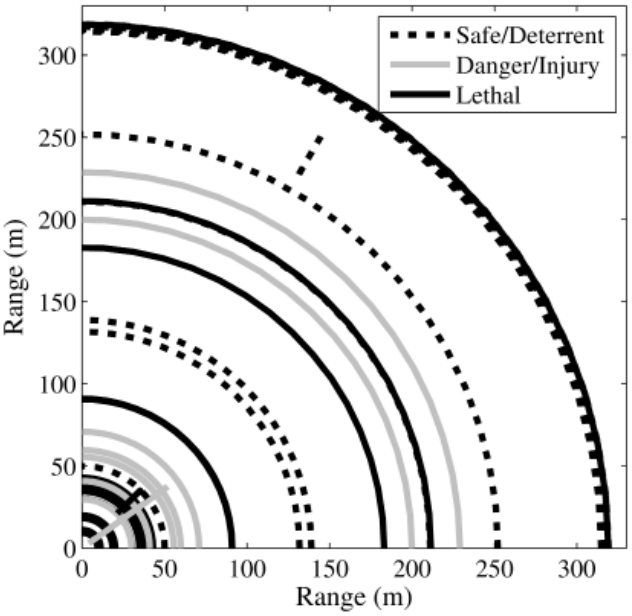


Figure 4
Published ranges for underwater blast injury from 42 separate references, 1943 to present; each has been converted to a range from the centre of an example explosion; 'Safe', 'Injury', and 'Lethal' ranges between various sources vary by an order of magnitude



As is evident from the figure, different range guidelines for the same degree of injury vary by orders of magnitude. When the injury types were assigned numerical values of 3 for Safe/Deterrent, 2 for Danger/Injury, or 1 for Lethal and plotted as a function of range, the resulting R^2 value was 0.21 for a linear regression fit curve. In other words, given the range value for a randomly selected guideline, there is no reliable way to determine whether that range value prescribes a 'safe' guideline or a 'lethal' guideline.

This wide variability could not be explained as guidelines evolving over time. When the guidelines were divided into categories by injury severity, no statistically significant trends in range could be found over time for any category ($P > 0.69$ for Safe/Deterrent, $P > 0.48$ for Danger/Injury, $P > 0.48$ for Lethal).

Discussion

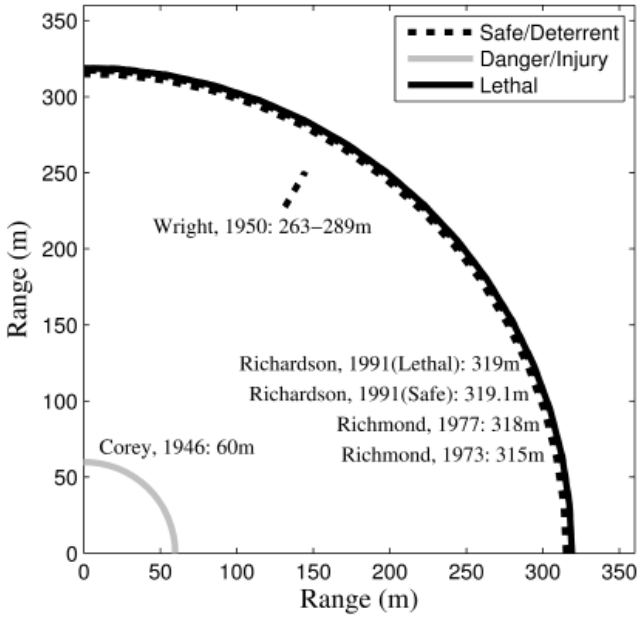
Forty-two guidelines for underwater blast exposure were found, only six of which were linked to documented experimental data. However, even these six guidelines are based on non-ideal experimental designs. Corey et al. used strips of beef intestine inside a model of a human torso to try to determine the exposure levels required to create abdominal perforations.[34] The experimenters ignored the hundreds of available medical reports that document the overwhelming location of abdominal perforations near the ileocaecal junction.[17] It is probable that the structure of the junction and attachment to the abdominal wall contribute stresses that lead to perforations at much lower blast exposure levels, as they do in blunt force trauma.[58]

Wright et al. conducted extensive human experiments to find a deterrent blast level, but operated under the assumption that peak pressure was the crucial factor in determining exposure levels and therefore only occasionally reported impulse values.[42] This omission was despite the researchers' own admission in the paper that impulse was a critical predictive factor. Also, it was found later that many of the pressure gauges used by this research group at that time were possibly inaccurate by 25% or more, meaning that while the 'safe/deterrent' levels established are probably approximately accurate, higher-exposure values may not be correct.[60]

Richmond et al. conducted meticulous animal and human experiments that are, to date, the most complete references for exposure found.[53,54,61] However, even these meticulous analyses were performed mainly using animal data that was not scaled to approximate humans and, within the seminal 1973 paper itself, they call for caution and further testing before applying these standards for human safety.[54] In spite of this warning, these experiments, with a factor of safety applied, are used to determine the current British standards for underwater blast exposure as found in British Standard 5607.[57] These standards, in their English-unit form of 2 psi*ms, are also published and used by the US Navy as the Gaspin Criteria.[30] While no universal standard for underwater blast safety currently exists, the guideline developed by Richmond et al. seems to be the most commonly applied safety standard today.[54]

Richardson et al. and Yelverton et al. also created standards for blast safety, but largely using animal data.[26,55] These two groups attempted inter-species scaling, but had very limited data points for animals with body masses in the same

Figure 5
Ranges prescribed by guidelines that have a documented experimental basis; only six guidelines were found that had a documented experimental basis, and these were still grossly inconsistent with each other



range as humans. Yelverton et al. had no data for animals above 45 kg and most data were from fish. Fish are injured through a different physiological process, rupture of the swim bladder, to that in mammals and are more sensitive to blast than humans. Therefore, they are not a valid test model for humans.[62] Richardson et al. studied the effects of blast on large aquatic mammals and made no attempt to extend their model to humans. It is uncertain whether lung and gut blast pathophysiology of marine mammals and terrestrial mammals is similar.

Figure 5 illustrates the ranges predicted for the six guidelines based on experimental data. Even though they are all based on test data, they are still grossly inconsistent because of the listed limitations of those experiments. The guidelines from Corey et al.[34] for injury risk is an order of magnitude less than the Richardson et al. lethal guideline,and the available safe/deterrent guidelines lie in between the two.[26] There is an obvious need for additional data and modelling to create a consistent, reliable set of guidelines similar to those available for air blasts. The impulse-based guidelines have a maximum range of 319.2 m because of the nature of the regression-fit curves; however, this model is still sufficient to show the gross inconsistencies between the guidelines. Properly constructed guidelines should, at the very least, predict fatal ranges as closer than dangerous ranges, and farthest from the blast should be the safety guidelines. Currently available guidelines appear in random order from the blast, highlighting the lack of physiological foundation.

The injury risk curves in air blast were initially computed using meticulous, extensive human and animal testing performed by the Lovelace Foundation as well as several researchers from the UK.[2,11,42,63] While hundreds of humans and animals have been exposed to underwater blast, the exposure data have never been reconstructed and evaluated as a whole. In addition, all of the underwater animal data available suffered from experimental shortcomings.

Conclusions

The current guidelines for injuries from underwater blast are grossly inconsistent, poorly experimentally founded, and vary by orders of magnitude. Researchers have been declaring the need for valid standards since the original cases of underwater blast in 1916, and the need still exists. It is impossible to predict safe operational distances or to design protective equipment without valid standards for the risk of injury or fatality from underwater blasts.

The field of underwater blast injury needs solid, data-based guidelines that can be used by operators and medical personnel while in the field. An important next step should be the compilation of available injury data and a realistic, quantitatively validated evaluation of the blast exposure levels that cause human injuries, similar to the injury risk curves available for air blast. This evaluation could be performed using either human historical data and epidemiological exposure data or may be developed using animal data in experimental series designed to provide realistic underwater blast exposures scaled to human values. The underwater blast community would benefit from a standard that has been developed using experimental methods that are comparably systematic to those used in the air blast community.

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Cone shell envenomation: epidemiology, pharmacology and medical care

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Abstract

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The marine environment presents much danger, specifically in regards to the numerous venomous inhabitants within tropical and subtropical waters. The toxins from one such group of venomous marine snails, commonly referred to as 'cone snails', have been well documented in causing human fatalities. Yet information regarding medical treatment for cone snail envenomation is limited and poorly accessible. To correct this, medical and scientific expertise and literary review on *Conus* provide a basic and comprehensive directive focused on the medical treatment and post-mortem investigative analysis of cone snail envenomation. We emphasize what we expect to be the most lethal feeding group of *Conus* and provide a brief background to the epidemiology of their stings. We describe the venom apparatus of *Conus* and its utility of rapid venom delivery. We have compiled the documented incidences of *Conus* envenomation to offer thorough reference of known signs and symptoms – this too drawing on personal experiences in the field. We have also made available a brief background to the biochemistry and pharmacology of *Conus* venoms to highlight their complex nature.

Key words
First aid; envenomation; toxins; pharmacology – marine; deaths; symptoms; treatment; review article

Introduction

In a recent paper, we illustrated the molecular composition of the milked venom obtained from *Conus geographus* (Table 1).[1] We believe this particular species, given the documented human fatalities, provides a 'worst case scenario' to use for revising medical treatment protocols in treating cone snail envenomation. The potential need to access this information is warranted by public interactions with cone snails and more so with increased acti vities in field collection and venom milking for scientific and medical research.

The photo of *Conus geographus* in Table 1 is of the actual specimen, collected on 27 June 1935 at Hayman Island, Northern Queensland, Australia, that caused a well-documented human fatality in 1935.[3] Accessioned on 19 July 1935 (Albert H Longman, Director of the Queensland Museum, Reg. # QMMO 1689), its length is 84 mm and the dried animal is inside shell (Photo: J Healy, Queensland Museum)

Here, we summarise the epidemiology of *Conus* envenomation, review the symptoms and signs of envenomation and provide revised recommendations for first aid and medical treatment. These details are based on personal, medical, laboratory and field experiences with cone snails and their toxins, together with literary research. A concise background into venom biochemistry and pharmacology is provided to deepen clinical awareness and assist in emergency treatment.

Epidemiology of *Conus* envenomation

Cone snails, representing the genus *Conus*, have been a source of interest and injury, with cases cited as early as 1706.[2] Fifty-five reported stings from the Indo-West Pacific, the Atlantic shores of Brazil, and the islands of the Indian and Pacific Oceans have been reviewed and reported.[3–7] Shell collectors, scuba divers and beachcombers are the typical cohort of cone snail victims as the collecting and handling of live specimens risk envenomation.

The most potentially dangerous cone shell species belong to piscivores (fish eaters; ~10% of genus; Table 1). Envenomation by *C. geographus* has the greatest mortality rate at 67% and has been responsible for nearly 85% of all lethal cases reported.[24] The principal conotoxins discussed that contribute to the mortality rate in humans, have been mathematically modelled to estimate a human lethal dose, based on the correlations found between dry weight per volume of injected venom and the size of the shell; the human lethal dose (0.029–0.038 $mg \cdot kg^{-1}$),[25] was extrapolated based on the historic fatal case of *C. geographus*.[3] Revised estimates indicate the mortality rate due to piscivorous envenomations to be 15–25%.[4,26] Molluscivore species (mollusk eaters; approx. 25% of the genus) have been inadvertently implicated in several fatal cases. This may be attributed to specimen misidentification, as the likelihood of this occurring is high considering there are 600 species in the genus. Moreover, molluscivores are known to be aggressive when removed from their natural environment. The remaining *Conus* species, the vermivores (worm eaters, approx. 65% of the genus), have no reported human deaths

Table 1
Known and suspected highly venomous cone snails; * the information was not explicitly indicated in the article
GBR – Great Barrier Reef

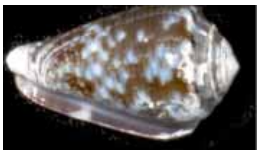
	Conus species	Place of collection	Foraging behaviour
	*C. geographus*	Marinduque, Philippines	Only feed on specific species including a puffer fish and small eels; also feed on frozen and thawed anchovies after spending some time in aquarium; length 70–153mm.[8]
		Boult Reef, GBR, Australia	Feed guppies and milked for venom.[1]
	*C. californicus*	Western coast of Baja, California	Fed on variety of fresh and saltwater fish and/or Canadian night crawlers; attack lasted for ~30 min; display organized/ cooperative attacks upon shrimps and snails.[9]
		Monterey Bay, California	Starved for a week before feeding with live juvenile specimens of three species of prickleback; length 15–35 mm.[10] Fed as above, with milked venom collected by arousing with squid skin (internal layer) stretched over a 0.5 ml microfuge tube and enticing a venom injection into the vial.[11]
	*C. striatus*	American Samoa	Fed on commercially procured goldfish;[12]
		Hawaii*	Fed on several fish species;[13]
		Various locations; GBR, Australia; Oahu, Hawaii	Fed on goldfish and milked for venom;[14] Fed on swordtail once a week after milking;[15] length 60–130 mm.
	*C. catus*	Hawaii	Feed on small marine fish, generally sculpins;[12]
		Kauai, Hawaii	Fed on freshly killed killifish and arrow goby; uses a high-speed hydraulic prey capture mechanism similar to the fish-hunting *C. pennaceus*.[13]
		Hawaii*	Feed on goby;[16] length 25–50 mm.
	*C. purpurascens*	Gulf of California; Near Smithsonian Tropical Research Institute, Panama	Fed twice weekly on goldfish after milking.[17] Fed weekly on goldfish and milked for venom; length 30–84 mm.[18]
	*C. ermineus*	Palm Beach County, FL, USA	Fed on goldfish; locally acquired for bait/feeding and milking purposes; length 30–70 mm.[19]
	*C. consors*	Chesterfield Islands, New Caledonia	Most fed on live fish (not specified) for milking purposes; one snail displayed scavenging feeding behavior on dead fish; length 50–80 mm.[20,21]
	*C. obscurus*	Oahu, Hawaii	Fed on goldfish and milked for venom; length 20–41 mm.[22]
	*C. magus*	Night Island, GBR	Specimens were fed weekly on juvenile goldfish and milked for venom for 12 months.[23]

Table 2
Symptoms and signs of cone snail envenomation

Local manifestations
- Mild to sharp burning sensation at the site of the sting;
- Sensations of tingling, burning, pricking (paraesthesiae) or numbness;
- Pruritus at site of penetration;
- Oedema at site of penetration; actual puncture wound may not be evident; possible localized discoloration;
- Oedema may show effects within the entire limb.

Systemic manifestations
- Spreading paraesthesiae and numbness, especially about lips and mouth;
- Blurred vision or diplopia;
- Fatigue and malaise;
- Faintness or altered mentation;
- Nausea, prolonged stomach cramps;
- Facial muscle paralysis;
- Ptyalism (drooling/hypersalivation);
- Slurred speech and potentially aphonia;
- Ptosis;
- Progressive muscle paralysis and numbness;
- Absence of limb reflexes;
- Dyspnoea;
- Unconsciousness;
- Respiratory arrest 40 min to 5 h after sting;
- Cardiac impairment, leading to cardiac arrest;
- Death (typically from acute respiratory failure).

Table 3
First aid and advanced care for cone shell envenomation; there is no antivenom for cone shell toxin

- General ***DRSABC*** (***D***anger, ***R***esponse, ***S***end for help), ***A***irway, ***B***reathing and ***C***irculation);
- Administer Basic Life Support (BSL) as indicated;
- Activate emergency medical services;
- Seek medical evacuation;
- Advanced Life Support (ASL) as indicated;
- Pressure immobilization (see below);
- If possible, with caution, retain specimen(s) for identification;
- If first aid measures of BSL and ASL are effective, remember that the victim may be paralyzed but fully conscious. Thus, reassurance and talking to the victim is important.
- Prolonged cardio-respiratory support may be required, including mechanical ventilation, iv fluids and inotropes;

Pressure immobilization
- Apply a broad pressure bandage directly over the sting area about as tight as elastic wrap to a sprained ankle.
- Ensure that arterial circulation is not cut off and fingers or toes stay pink and warm.
- In cases that involve swelling of the affected area, the compression bandage may need to be more proximally positioned to wrap ahead of the swollen area.
- Bind splint or any rigid object to support limb/affected area; focus on immobilization to limit toxin circulation.
- Reassure patient and prevented patient from walking or physically moving.

DO NOT:
- cut or excise the stung area;
- attempt to suck out the venom;
- submerge limb in hot water or pour hot water, vinegar, denatured alcohol, ethanol or other home remedies on sting area;
- apply an arterial tourniquet;
- elevate sting site;
- operate vehicle if envenomed.

associated with stings, mostly owing to their timid nature and phyla-selective toxins.

Human cone snail envenomations are uncommon due to the animal's nocturnal nature and the lack of public knowledge regarding their habitats, although chance discoveries can lead to their handling and removal for their attractive shell patterns. This dangerous practice is frequently dismissed with the delusion that a snail cannot kill. South Pacific islanders are aware of the dangers associated with these marine gastropods, and typically group all cone snails, called "*intrag*" or "*nunus*", as dangerous and revered. Undocumented envenomations have occurred, with local inhabitants stating they were told or know of such occurrences. In these locations, a potential indicator of a [surviving] cone snail victim can be the presence of multiple laceration scars to the affected limb caused by bloodletting; a treatment islanders wrongly believe limits the circulation of injected venom.[7]

Scientific researchers use cone snail toxins to dissect ion channel functions.[27] Due to their toxic nature, the US Centers for Disease Control and Prevention classify these as 'select agents'.[28] Thus, governmental regulated safety measures are employed within the research laboratory. Regardless of toxin source (synthetic or native), incident locations (laboratory or field sites) or post-exposure symptoms and signs, the principles and practices for medical treatment are identical.

Venom delivery

The route of venom delivery is unique to this family (toxoglossa, meaning 'poisonous tongue,' includes cone snails, turrids, and terebra). These predatory snails are armed with a quiver of single-use, hollow, barbed and serrated hypodermic-like harpoons or radula. The structures are commonly used as a taxonomical tool in species identification, since each species has a distinct form and

structure.[29] Typically in piscivorous *Conus*, these eyelash-sized chitinous impalers can penetrate woven layers and even 5 mm neoprene wetsuit materials at high velocity, indicating that a wetsuit provides little protection (Gilly, personal communication, 2014).[30–32] The volume of venom delivered ranges from 1 to 50 μL. The effective harpoon trajectory range is increased by the proboscis, an extendable and flexible tongue-like structure that can rapidly extend one-to-two shell body lengths. Thus, holding a snail at the rear (the broadest end of its shell) offers little protection. In rare occurrences with antagonism, such as scraping the shell with a knife or dropping it, cone snails have been known to 'shoot' harpoons.[6] Therefore, avoiding handling these live shells represents the best prevention.

Envenomation

Reported envenomations[30,32,33] have provided various symptoms and signs, summarised in Table 2. These reports concur with personal experience and observations.[32] The puncture and envenomation sensations vary. Most victims feel an immediate stinging sensation and later local numbness. Localized swelling may occur, accompanied by redness or discoloration from ischaemia/cyanosis. Local numbness and paraesthesia spread quickly from the affected area about 10–30 min after the sting. Many victims report these sensations intensely around the lips and mouth. Less common manifestations are nausea, muscle cramping, headache, and itching. The venom is a neurotoxin, therefore, after about 30 min, systemic abnormalities such as muscle weakness (including respiratory), diplopia, dysarthria, the inability to swallow, and an absent gag reflex start to develop.[4] Within an hour, generalized paralysis and respiratory failure can occur and, without medical support, coma and death may follow.[3,4,6,33]

Medical treatment

FIRST AID

Because envenomation may cause paralysis, coma and death, it is essential to first remove the victim from water in order to prevent drowning and subsequently transport the victim to a medical facility as quickly as possible. Owing to the chemical complexity, rarity of stings, and geographic diversity of cone snail venom, no attempts to produce cone snail antivenom have been successful.[21,23] To slow venom distribution, a pressure immobilization technique, as recommended for snake envenomations, should be used (Table 2). There have been documented cases in which medical attention was not possible, and the course of action was vigorous rubbing and squeezing to expel contaminated serum. More drastically, some cases have involved wound lancing/bloodletting and sucking fluids from the site.[33] There is no evidence to support these harmful measures and so they are not recommended. Prioritize the patient's airway, watch for signs of respiratory insufficiency, and administer oxygen as indicated (10–15 $L \cdot min^{-1}$). Gag reflex may be absent and suction may be required. Management should focus on airway protection and ventilation with Basic and Advanced Life Support (ALS) as indicated. The victim may be paralyzed but remain conscious, thus reassurance and talking to the victim are important.

ADVANCED CARE

Provide on-going ALS, including mechanical ventilation, anti-arrhythmic agents, inotropes and cardioversion as indicated. No coagulopathy has been observed in cone snail envenomation.[35] The onset of respiratory paralysis can require mechanical ventilation for more than 24 hours. Paralysis is not permanent and typically resolves within 12–36 hours. Intravenous fluid and inotropes may be required for hypotension. Administer fluids cautiously since some patients have exhibited pulmonary oedema. Continue monitoring respiratory and cardiac function until autonomic and motor function are fully regained. Animal studies suggest that seizures are unlikely as, owing to the peripheral peptidic neurotoxic nature of the venom, it does not penetrate the blood brain barrier. If seizures occur, these are likely secondary to hypoxia.

POST-ENVENOMATION CARE

The wound should be regarded as potentially contaminated, and treatment may be required for secondary bacterial infection.[4,33,36] Treatment for secondary soft tissue infections should be directed at usual flora and additionally, those unique to the marine environment. For this circumstance, consideration of empiric therapy with ceftriaxone and doxycycline is recommended.[37]

Ulceration may occur and may require long-term wound care with the potential of recurring infection (Jackson, personal communication, 2014).[26] In such cases, it is likely that foreign material is still present within the dermis. Thus, to avoid infection, the wound must be examined and debrided. Nerve damage at or around the sight of envenomation is possible, leading to temperature perception reversal – a similar permanent localized effect to that seen with ciguatera poisoning (East, personal communication, 2005). Most recovering patients seem no worse for wear, with some having been medically discharged within 48 hours of envenomation.

Post-mortem examination

In the event of post-mortem examination, external inspection of the extremities is paramount. It is possible to locate the area of radula harpoon penetration, indicated by a small area of dermal pigment differentiation and/or the presence of the imbedded radula harpoon(s). It is possible to see < 50% of the exposed harpoon length. The imbedded harpoon(s) may still retain a posterior thread-like ligament, which can be

equal in length to the chitinous harpoon itself. The naturally barbed structure of the harpoon may be damaged if forcibly removed from the dermis. Typical points of entry are hands, fingers and waist region if the animal was collected and then stowed or 'pocketed'.

Animal studies of the toxic effects of cone snail venom have demonstrated decreased red blood cell count, increased immunoglobulins, and elevated serum enzyme levels, including glutamic-oxaloacetic transaminase, glutamic-pyruvic transaminase, lactate dehydrogenase, and both alkaline and acid phosphatases.[35] Unlike snake-bite envenomation, no forensic use of immunoassay approaches have been conducted for cone snail venoms, and very limited data are available in regards to attributed blood changes in humans.

Respiratory failure is typically stated as the cause of death, often with minimal pathological findings. In the rare instance of post-mortem examination of a human *C. geographus* victim, the victim "*showed that all the organs, heart, lungs, et cetera were quite healthy*".[3] Such lack of findings fits with the known pharmacological diversity of toxic peptides that primarily illustrate neuromuscular targeting (Table 4).[1] The speed of lethality, venom volume, and toxin concentration are elements for consideration, as these may cause different findings at autopsy.

Biochemistry of venom

Potential for death from a piscivorous cone snail envenomation comes from the mixture of high-affinity peptide toxins that target different ion channels and receptors.[1,14,38] There are an estimated half-million bioactive peptides, commonly referred to as conotoxins or conopeptides within the genus (see Table 4 for examples). Recent evidence indicates that *Conus* has the ability to deploy different venom profiles for prey capture and for defense, resulting in changes within the injected venom.[39] These features highlight the unparalleled biochemical nature of these venomous marine snails.

Biochemically these small peptide neurotoxins (5–100 different peptides per milked venom; 10–40 amino acids in length) commonly contain stabilizing chemical modifications such as disulfide bonds.[40–42] Venom extracts collected in 1962 by the late Dr.Robert Endean exemplify venom constituent stability; even today, these extracts still demonstrate potency and high molecular mass composition compared to freshly dissected venom from the same species.[14] This stability is reflected in the inability to minimize venom toxicity by heat and renders hot water submersion as an ineffective first-aid procedure.[43]

Proteinaceous material (> 6,000 Da) is also expressed within the milked venom of *Conus*. These proteins potentially include phospholipases and proteases.[44,45] The expression of phospholipases within *Conus* is neither as predominant nor as lytic as those found in some snake venoms.

Pharmacological properties

Isolated conotoxins from *C. geographus* venom, our hallmark for human lethality, have been pharmacologically characterized, and their elicited and complex symptoms have been individually observed (Table 4). The pharmacological predation/defensive strategy for all piscivorous *Conus* species is the same – to rapidly paralyze. In laboratory animals, a wide range of behaviours of head swinging, kicking on back, scratching, uncoordinated jumping, trembling, back leg dragging, depressed activity, sleeping, convulsing and bleeding, and finally paralysis and coma are revealed upon intracranial injection.[54] In nature, the venom's pharmacological effectiveness is maximized by a synergistic binding strategy that achieves paresis more rapidly than other observed venomous groups (snakes, scorpions, spiders, anemones).[42,55]

As in *C. geographus* venom, a single venom may contain both pre- and post-synaptic inhibitors that specifically target voltage-gated calcium channels and acetylcholine receptors, respectively. The pre-synaptic inhibitors act upon the neuronal calcium channels located in the axonal terminal; when these receptors are blocked, incoming action potentials that would allow for the influx of Ca^{2+} ions through the external vestibule are obstructed. Otherwise, the influx of Ca^{2+} would have triggered the release of neurotransmitter (acetylcholine) into the synaptic cleft to stimulate the propagation of another neuronal action potential or a muscular contraction event. Thus, the post-synaptic neuronal inhibitors which have high binding affinity for neuronal acetylcholine receptors block the downstream propagation of the action potential by inhibiting the ability of the acetylcholine receptors to respond to increased concentrations of their associated ligand, acetylcholine. This synergistic blocking prevents synaptic nerve action potential propagation completely, a common trend in underlining its neurotoxicity. Those peptide toxins responsible are identified as the ω-conotoxins: ω-GVIA and ω-GVIIA or 'shaker peptides' and members of the ω-conotoxins: ω-GI and ω-GIA (Table 4).[42,56] This represents a highly conserved strategic pharmacological targeting process within all piscivorous *Conus*.[57]

The venom may also contain μ-conotoxins: GIIIA, GIIIB and GIIIC, which act by blocking the voltage-activated sodium channels in muscle membranes (Table 4). These conotoxins are also lethal in mice whether by intracranial or interparietal injections and potentiate the mechanism for rapid onset of paralysis.[42] Other ω- and μ-conotoxin variants have been reported in other piscivores. While within the genus and encompassing all feeding groups, many phyla-selective ω-conotoxins are reported which demonstrate a preferential molecular selectivity.[58]

Conantokin-G represents a non-paralytic toxin in *C. geographus* venom that subdues young mice to a sleeping state upon intracranial injections (Table 4).[42] This peptide

Table 4

The dominant and consequential conotoxins within milked venom of *Conus geographus*, tested for activity in mice; LD50 = lethal dose; ND = Structure not determined; * C-terminal amidation; O = 4 trans-hydroxyproline; γ = gamma-carboxyglutamic acid; W = bromotryptophan

Conotoxin		Amino acid sequence	Target	Intracerebral injection	intraperitoneal injection	Symptoms	Reference
α-GI		E**CC**NPA**C**GRHYS**C***	Neuronal nAChR	–	LD_{50} 12 µg/kg BW	Paralysis, death	8,46
ω-GVIA (SNX-124)		**C**KSOGSS**C**SOTSYN**CC**R**SC**NOYTKR**C**Y*	$Ca_v2.2$	3 µg·kg^{-1} BW	–	Persistent tremors	47–49
µ-GIIIA		RD**CC**TOOKK**C**KDRQ**C**KOQR**CC**A*	$Na_v1.4$	–	LD_{50} 340 µg/kg BW	Paralysis, death	50,51
Conantokin G (CGX-1007)		GEγγLQγNQγLIRγKSN*	NMDA receptor	11 µg·kg^{-1} BW	–	Drowsiness/ hyperactive (age dependent)	52,53
ω-GVIIA (SNX-178)	ND	**C**KSOGTO**C**SRGMRD**CC**T**SC**LLYSNK**C**RRY	Ca_v	<2 nmol per mouse	–	Persistent tremors	42

targets the NMDA receptor. It is not consequently deadly in mice, but its presence may act on the peripheral circuits of fish.[54] To date, the numerous conantokins that have been isolated and their expression appears to be exclusive to the piscivores of *Conus*.[59] With the potential of hundreds of conotoxins in a single envenomation and many still being unknown, the pharmacological correlation to specific symptoms and signs in humans can be complex and variable. Therefore, *Conus* deserves both respect and caution as a highly venomous marine invertebrate.

Conclusions

Although considered rare, cone snail envenomation can be lethal in humans. Given the diversity, potency and specific neurotoxicity of conotoxins and the lack of antivenom, the emergency care of an envenomation must focus on maintaining airway protection and ventilation. The best prevention is education. All cone snails should be considered venomous and thus should be avoided.

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Disclaimer

The views expressed in this abstract/manuscript are those of the author(s) and do not reflect the official policy or position of the Department of the Army, US Department of Defense, or the US Government and that of the Australian Department of Defense.

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Letters to the Editor

On diver thermal status and susceptibility to decompression sickness

In a recent Letter to the Editor,[1] Clarke, et al, indicated that divers who deliberately chill themselves on a dive to reduce risk of decompression sickness (DCS) may be misinterpreting our 2007 Navy Experimental Diving Unit (NEDU) report.[2] Indeed, we did not advocate that divers should risk hypothermia on bottom to reduce risk of DCS, nor do we dispute the authors' overall admonition to avoid diving cold unnecessarily. However, Clarke, et al, imply more generally that results of our study are not applicable to recreational or technical divers because the dives we tested were atypical of dives undertaken by such divers. We wish to clarify that our study does have implications for recreational and technical divers, implications that should not be ignored.

The dives we tested were not intended to be typical of dives undertaken in any actual operational context. Instead, we chose to expose divers to temperatures at the extremes of their thermal tolerance in order to ensure that effects of diver thermal status on DCS susceptibility would be found if such effects existed. Our initial test dive profile provided appreciable time both on bottom and during decompression to allow any differential thermal effects during these two dive phases to manifest, while affording a baseline risk of DCS that could be altered by thermal effects without exposing subjects to inordinately high risks of DCS.

Our results strongly indicate that the optimal diver thermal conditions for mitigation of DCS risk or minimization of decompression time entail remaining cool during gas uptake phases of a dive and warm during off-gassing phases. While the dose-response characteristics of our observed thermal effects are almost certainly non-linear in both exposure temperature and duration, it is only reasonable to presume that the effects vary monotonically with these factors. We have no reason to presume that such responses and effects under less extreme conditions would be in directions opposite those found under the conditions we tested. Similarly, responses to thermal exposures even more extreme than we tested might not be larger than the responses we observed, but it would be unwise to ignore the trends in our results under some unfounded presumption that the effects reverse with changes in thermal conditions beyond those tested. Finally, thermal effects on bottom and during decompression in dives to depths other than the 120 feet of sea water (fsw) or 150 fsw depths of the dives we tested are unlikely to be qualitatively different from those observed in our tested dives. The original question has therefore been answered: Chill on bottom decreases DCS susceptibility while chill during decompression increases DCS susceptibility. Under conditions encountered by recreational or technical divers, the only open issue is arguably magnitudes of effects, not directions. Neither does lack of technology to control thermal status during a dive render our study results inapplicable. It only renders the diver unable to actively optimize his or her thermal exposure to minimize DCS risk or decompression obligation.

Effects of diver thermal status on bottom hold regardless of whether the dive has a decompression long enough for a thermal effect to manifest in the decompression phase of the dive. We pointed out that US Navy decompression tables have historically been developed and validated with test dives in which divers were cold and working during bottom phases and cold and resting during decompression phases. Thus, our results indicate that it is not prudent for very warm divers to challenge the US Navy no-stop limits. However, becoming deliberately chilled on bottom only to remain cold during any ensuing decompression stops is similarly ill-advised. We agree with Clarke et al. that relative conservatism of some dive computer algorithms or alternative decompression tables, or the depth and time roundups necessary to determine table-based prescriptions, work in the diver's favour, but note that diving any profile to a shorter bottom time is a ready means to reduce the risk of DCS – i.e., enhance safety – without compromising comfort. Any active diver heating is best limited while on bottom to a minimal level required to safely complete on-bottom tasks, and dialed up only during decompression. Diver warming during decompression should not be so aggressive as to risk heat stress, and care should be taken to ensure that divers remain hydrated.

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Key words
Decompression sickness; hypothermia; risk factors; letters (to the Editor)

Re: Don't dive cold when you don't have to

The letter by Clarke et al[1] unfortunately misrepresents the work at the US Navy Experimental Diving Unit (NEDU) to which it refers,[2] and delivers a confused picture of the physiological impact of thermal status on decompression stress. A series of earlier reports outline the importance of thermal status. Being warm during a dive results in higher post-dive Doppler bubble scores.[3] Hot water suits are associated with a higher rate of decompression sickness (DCS) than passively insulated drysuits.[4] Post-dive cooling can prolong the risk window for developing symptoms of skin bends.[5]

The NEDU chamber study provided an elegant design to further assess the impact of thermal stress. Dives to 37 msw (120 fsw) were divided into descent/bottom and ascent/stop phases, prolonging the latter so that bottom times could be increased if results allowed without compromising the experimental structure. The water temperature was held at either 36^{O}C (97^{O}F; 'warm') or 27^{O}C (80^{O}F; 'cold'). The 'warm/cold' exposure, with a bottom time of 30 minutes, yielded a DCS rate of 22% (7/32 subject-exposures). The 'cold/warm' bottom time was increased to 70 minutes and still yielded a DCS rate of only 1.3% (2/158). Even if the effects are exaggerated by the prolonged ascent/stop phase, the dramatic results demand serious attention.

Contrary to the claim made by Clarke et al in their letter, the high temperature employed in the NEDU study could almost certainly be maintained at the skin by a number of active heating garments available to the diving public. Hot water suits are not required for the effect; and the 'cold' study temperature (better described as 'cool') is clearly well within the range experienced by divers.

The statement by Clarke et al that "*the Navy uses their extensive mathematical expertise to select the one dive profile that, in their estimation, is the most likely to identify a difference in decompression risk...*" is frankly baffling. Use of a single dive depth in no way invalidates the relevance to other dive profiles. Similarly, it is not reasonable to characterize skin temperatures lower than those produced in the study as "*venturing into the unknown*" and thereby invalidating the results.

Scientific method does encourage the confirmation of findings. This goal, however, does not diminish the value of singular, well-designed studies. The NEDU study is certainly one of these, most valuable in reminding divers that factors beyond the pressure-time profile will affect decompression risk.

Divers must have adequate thermal protection to function effectively (physically and cognitively) throughout a dive. However, excessive warming during the descent/bottom phase increases inert gas uptake and can compromise decompression safety. Practically, while it may be optimal for divers to be cool or cold during the descent/bottom phase, it is prudent to recommend a thermoneutral range and avoidance of any excessive warming. Being cool during the ascent/stop phase inhibits inert gas elimination and can compromise safety but sudden warming must be constrained to avoid reducing the gas solubility of superficial tissues that could promote localized bubble formation and symptoms of skin bends.

Active heating systems are attractive, but they have the potential to create the worst decompression stress condition; excessive heating during the descent/bottom phase and cooling during the ascent/stop phase if they fail part way through a dive. The risk is still elevated, though, if the systems work throughout a dive.[2,4] Gerth et al were able to increase the bottom time to 70 minutes for both the 'cold-warm' and 'warm-warm' conditions, but the rate of DCS was significantly lower for the 'cold-warm' condition (see above).[2] This lesson is relevant to any diving exposure.

Ultimately, divers need to be aware of the potential impact of thermal status. Thermal protection should preserve clear thinking and physical performance, but excessive manipulation should be avoided. For many, passive systems will provide adequate and appropriate protection. For those who need or choose active warming systems, thoughtful use is vital. Further research is required to quantify the hazards and be able to incorporate thermal status into decompression algorithms in a meaningful way.

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Key words
Decompression sickness; hypothermia; risk factors; letters (to the Editor)

Management of severe spinal cord injury following hyperbaric exposure

There is an increasing body of evidence that drainage of lumbar cerebrospinal fluid (CSF) improves functional neurological outcome after reperfusion injury to the spinal cord that occasionally follows aortic reconstructive surgery.[1,2] This beneficial effect is considered owing to lowering of the CSF pressure thereby normalising spinal cord blood flow and reducing the 'secondary' cord injury caused by vascular congestion and cord swelling in the relatively confined spinal canal. Whilst lacking definitive proof, there are convincing randomised controlled trials (RCTs), cohort data and systematic reviews supporting this intervention. The therapeutic window for lumbar CSF drainage requires further elucidation; however, it appears to be days rather than hours post insult.[3,4] We contend that the same benefit is likely to be achieved following other primary spinal cord injuries that cause cord swelling and elicit the 'secondary' injury.

Traditionally the concept of CSF drainage has been considered more applicable to the brain as contained in a 'closed box' by lowering intracranial pressure (ICP) to improve cerebral perfusion pressure (CPP). The control of CPP is intended to limit 'secondary' brain injury and is a key concept of brain injury management. Using microdialysis in the spinal cords of trauma patients, it has been shown that intraspinal pressure (ISP) needs to be kept below 20 mmHg and spinal cord perfusion pressure (SCPP) above 70 mmHg to avoid biochemical evidence of secondary cord damage.[5] Vasopressor have also been used in spinal cord injury to improve perfusion, however complications are common, typically cardiac in nature, and require very careful monitoring; the evidence supporting this approach is notably less convincing.

Decompression illness (DCI) of the spinal cord is treated with recompression, hyperbaric oxygen, various medications designed to reduce the inflammatory response and fluid administration to normalise blood pressure and haematocrit.[6] These management protocols are based largely on anecdote and transferred evidence from conventional cord trauma, as the low numbers and sporadic nature of DCI in divers makes RCTs nigh on impossible. Unfortunately even with best management, some patients are left with significant neurological deficit.

The 'iceberg phenomenon', occurs when patients with DCI of the cord make a good neurological recovery but actually have profound cord damage as revealed in one case some four years later at post mortem and another example in a diver who developed late functional deterioration due to loss of neuronal reserve.[7,8] This clinical evidence, together with animal study data, support the notion that even a modest preservation of spinal cord axons is associated with significant improvement in neurological outcome.[9]

In the light of the positive level two evidence in the vascular literature that CSF drainage limits 'secondary' injury thereby improving neurological outcome, we propose that centres with appropriate clinical experience consider using lumbar CSF drainage to normalise SCPP, as an adjunct to the conventional treatment of severe spinal cord DCI. Divers with severe spinal cord DCI are generally in the most productive years of their lives and, given the potentially devastating impact of this condition, should be given the benefit of any possible adjuvant treatment that may serve to improve long-term outcome.

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Key words
Decompression illness; central nervous system; cerebral blood flow; neuroprotection; letters (to the Editor)

Continuing professional development

Marine envenomation

Neil Banham

Accreditation statement

INTENDED AUDIENCE

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

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

OBJECTIVES

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

FACULTY DISCLOSURE

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

DO I HAVE TO PAY?

All activities are free to subscribers.

Key words
Marine animals; envenomation; jellyfish; clinical toxicology; toxins; MOPS (maintenance of professional standards)

Recommended background reading

Practitioners are referred to the following background references and reading.

1 Fenner PJ. Venomous marine animals. *SPUMS Journal*. 2004;34:196-202.
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How to answer the questions

Please answer all responses (A to E) as True or False.
Answers should be posted by email to the nominated CPD coordinator.

EUBS members should send their answers to Lesley Blogg.
E-mail: <lesley.blogg@eubs.org>

ANZCA DHM SIG and other SPUMS members should send their answers to Neil Banham.
E-mail: <Neil.Banham@health.wa.gov.au>

If you would like to discuss any aspects with the author, contact him at: <Neil.Banham@health.wa.gov.au>.

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

Question 1. With regard to cone shell envenomation

A. there have been numerous Australian fatalities;
B. *Conus geographus* is responsible for most fatalities;
C. the venom is primarily cardiotoxic;
D. an antivenom is available;
E. prolonged cardio-respiratory support may be required.

Question 2. In box jellyfish (Chironex fleckeri) envenomation

A. vinegar is recommended for first aid;
B. stings are typically not initially painful;
C. death may occur within minutes;
D. an antivenom is available;
E. IV magnesium is indicated in cardiac arrest.

Question 3. In Irukandji syndrome

A. it may be caused by several species of jellyfish;
B. symptoms typically occur within a few minutes of being stung;
C. large amounts of opiate analgesia may be required;
D. IV magnesium is indicated for significant envenomation;
E. systemic manifestations are via venom-induced catecholamine release.

Question 4. In blue-ringed octopus bites

A. the octopus is found in all Australian coastal waters;
B. pressure immobilization first aid is indicated;
C. the venom is identical to that found in puffer fish;
D. an antivenom is available;
E. flaccid paralysis may occur.

Question 5. Pressure immobilization first aid is indicated for

A. sea snake bite;
B. cone shell sting;
C. box jellyfish sting;
D. stonefish spine injury;
E. Irukandji jellyfish sting.

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Course Convener: Clinical Associate Professor David Smart
E-mail: <dsmart@iinet.net.au>
Phone (mobile): +61-(0)4-3722-1422

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- Assessment of fitness to dive
- Accepted indications for hyperbaric oxygen treatment
- Wound management and transcutaneous oximetry
- In water rescue and simulated management of a seriously ill diver
- Visit to HMAS Penguin
- Practical workshops
- Marine Envenomation

Approved as a CPD learning project by ANZCA: (knowledge and skills category): 56 hours for attendance at lectures and presentations for one credit per hour; 24 hours for workshops/PBLDs/small group discussions for two credits per hour

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

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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

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

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

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

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

Additional information – prospective approval of projects is required

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

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one.

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

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

As of 01 June 2014, projects will be deemed to have lapsed if

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

With respect to 1 above, for unforeseen delays where the project will exceed three years, candidates must advise the Education Officer in writing if they wish their diploma project to remain active, and an additional three-year extension will be granted.
With respect to 2 above, if there are extenuating circumstances that a candidate is unable to maintain financial membership, then these must be advised in writing to the Education Officer for consideration by the SPUMS Executive.

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

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

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

Notices and news

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

42nd EUBS Annual Scientific Meeting 2016 Preliminary notice

Dates: 14–17 September 2016
Venue: Geneva International Congress Centre, Switzerland

A traditional meeting place for 2000 years, an international metropolis, Geneva offers its visitors different faces from its humanitarian commitment and cultural activities to numerous conferences and renowned gastronomy. Its international airport and privileged location on the major routes make it one of the leading European centres of business and leisure tourism.

A dedicated conference website will be operational as from September. In the meantime, check your EUBS website <www.eubs.org> for updates.

Welcome to Geneva from the convening committee:
Rodrigue Pignel, Jean-Yves Berney, Marco Gelsomino, Marie-Anne Magnan and Michel Pellegrini

2nd International conference on hyperbaric oxygen therapy and the brain

Dates: 19–21 Nov 2015
Venue: Hotel Ganim, Dead Sea, Israel

- Cerebral palsy
- Anoxic brain damage: traumatic brain injury, stroke
- sudden sensorineural hearing loss; central retinal artery occlusion
- Post traumatic stress disorder
- Chronic pain syndromes
- Neurodegenerative diseases
- Diving injuries

Abstract presentations on these topics are most welcome.

Israeli Society for Hyperbaric and Diving Medicine
E-mail: <hbo.diving.israel@gmail.com>
Website: <www.reg.co.il/ISHDM2015>

Amsterdam Capita Selecta Diving Medicine

Diving pathophysiology and cases
Date: 21 November 2015
Venue: Academic Medical Centre, Amsterdam
Lecturers: Jacques Regnard and Adel Taher
For more information: <www.diveresearch.org>

Important message: EUBS membership

Membership renewals are due by the end of the year 2015. As decided by the EUBS General Assembly in 2014, the EUBS membership year will now run from 01 January to 31 December. Members will receive renewal notices by e-mail in October, and again in November and December; alternatively, you can renew by logging in to your personal membership page on the EUBS website and choose 'renew my membership'.

The Science of Diving

Support EUBS by buying the PHYPODE book "The science of diving". PHYPODE research fellows, <www.phypode.org>, have written a book for anyone with a keen interest in the latest research trends and results about diving physiology and pathology. Edited by Tino Balestra and Peter Germonpré, the royalties from this book are being donated to the EUBS. Need more reason to buy? We don't think so! Available on Amazon at: <http://goo.gl/DAEn6R> and at Morebooks: <http://goo.gl/0VFMq7>

UHMS award for European Editor of DHM

At the annual scientific meeting of the UHMS, which took place in Montreal in June, Lesley Blogg was awarded the President's Award for the Best Overall Oral Presentation. Her talk was entitled:
Observed incidence of decompression sickness and venous gas bubbles following 18 m dives on RN Table 11 / Norwegian air diving table

Co-authors: Andreas Møllerløkken, Karen Jurd and Mikael Gennser

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

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

Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, organizing more than 180 courses over the past 20 years. In 2015 SHF have organized more courses then ever, targeting an international audience.

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

SHF courses for 2015
07–14 November: Basic course diving medicine (level 1 part 1); Kubu, Bali
14–21 November: 23rd SHF In-depth course "*A life long diving*" (full); Kubu, Bali
21–28 November: 23rd SHF In-depth course "*A life long diving*"; Kubu, Bali

For further information: <www.scotthaldane.org>

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 – EUBS and SPUMS – appears on the journal website: <www.dhmjournal.com>

Details of advertising rates and formatting requirements are available on request from:
E-mail: <editorialassist@dhmjournal.com>

DAN Europe

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

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
E-mail: <hdsaustraliapacific@hotmail.com.au>
Website:
<www.classicdiver.org>

British Hyperbaric Association ASM 2015

Dates: 22–24 October
Venue: National Exhibition Centre, Birmingham

Jointly with the UK Sport Diving Medical Committee (UKSDMC) and chosen to coincide with DIVE 2015.
The content of 22–23 October will be aligned to the requirements of refresher training for Health and Safety Executive-approved Medical Examiners of Divers.

Further information in next issue or contact:
<http://www.hyperbaric.org.uk/>

Hyperbaric Oxygen, Karolinska

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

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

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

German Society for Diving and Hyperbaric Medicine

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

Instructions to authors

A downloadable pdf of the 'Instructions to Authors' (revised August 2015) can be found on the *Diving and Hyperbaric Medicine* (DHM) website: <www.dhmjournal.com>. Authors must read and follow these instructions carefully.

All submissions to DHM should be made using the portal at <http://www.manuscriptmanager.com/dhm>. Before submitting, authors are advised to view video 5 on how to prepare a submission on the main Manuscript Manager web site <http://www.manscriptmanager.com>.

In case of difficulty, please contact the Editorial Assistant by e-mail at <editorialassist@dhmjournal.com>.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

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

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

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

UNITED KINGDOM
+44-7740-251-635

USA
+1-919-684-9111

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

DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

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

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

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

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

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

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