

Diving and Hyperbaric Medicine

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and the European Underwater and Baromedical Society*

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EUBS



Measuring cardiac output in the chamber

Treating decompression sickness in a remote setting

Australian diving-related fatalities in 2008

Epilepsy and scuba diving?

More on the box jellyfish

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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

This issue contains the case reports of diving-related deaths in Australian waters in 2008.¹ Such reports inevitably run some years behind, as these are, in the main, subject to coronial investigation and this sometimes takes several years to complete. The *SPUMS Journal* published the first such report in 1972, and has continued to publish the annual reports ever since.² For three decades, the sole investigator in what was called *Project Stickybeak* was Dr Douglas Walker, a general practitioner in New South Wales, who for many years was also Editor of the *SPUMS Journal*. Since the mid 2000s, John Lippmann and his team at DAN Asia-Pacific have gradually taken over the central role in producing the reports, supported by a diving and hyperbaric medicine specialist, Dr Andrew Fock from Melbourne, and a forensic pathologist, Dr Christopher Lawrence from Hobart, who has a special interest in diving fatalities and other drownings. No other country in the world has such a detailed and chronologically long record of diving-related fatalities. These data represent a major resource for epidemiological studies, despite the uncertainties within them gathered as they so often are from diverse sources of variable reliability.

As editors of *Diving and Hyperbaric Medicine*, my predecessor, Dr John Knight and I have had a considerable input over the years to preparing these reports. The present structure (separating out the facts of the event, autopsy findings and commentary from the various authors) was devised with my encouragement, as earlier reports tended to confuse fact and supposition to some degree, making it difficult to understand just what had happened. Of course, in some cases, such as unwitnessed death, more supposition than fact is inevitable. Some might question the value of these simple reports, lacking as they do any true analysis, and without feedback as to what changes in diving training, practice, equipment, medical assessment, etc, have resulted. Since the same problems appear to arise time and time again when diving accidents occur, this suggests that little might have been achieved. Is this assumption correct?

I think that the answer is no, despite all. A few examples:

- the conduct of autopsies on divers has improved considerably in Australia and New Zealand as a direct result of *Project Stickybeak* and one of the co-author's (Chris Lawrence) efforts;
- the Professional Association of Diving Instructors some years ago looked prospectively at its emergency ascent training data as a result of the *Stickybeak* reports highlighting several deaths in Australia during this training exercise, and radically changed their approach, resulting in a cessation of deaths during this activity;
- improvements have been made in the teaching of buoyancy control (a major risk factor in diving accidents) as a result of these reports and similar reports by the British Sub-Aqua Club and DAN International;
- changes have been instituted recently in the way in

which the police in Australia and New Zealand approach the investigation of diving fatalities, though there is some way to go in implementing these;

- changes are on-going in the approach to 'fitness-to-dive' medical assessments, especially in the middle-aged and elderly and those with a history of cardiac disease;
- Queensland enacted legislation some years ago, based in part on the Queensland fatality reports in *Project Stickybeak*, to regulate the recreational diving industry on the Great Barrier Reef (GBR), resulting in a marked reduction in fatalities and in the incidence of decompression sickness on the GBR.

Recently, an analysis of some aspects of these data has been published.³ Unfortunately, unless one is a member of the Undersea and Hyperbaric Medical Society or has library access to their journal, *Undersea and Hyperbaric Medicine*, many members of SPUMS (and some of EUBS) may not get to see anything more than the abstract of this publication on Medline. If you are interested to follow this up, then contact the principal author at <john.lippmann@danasiapacific.org>. Another interesting publication to track down on this subject would be the proceedings of the 2010 Divers Alert Network workshop on recreational diving fatalities.⁴

Plans for the Tricontinental Meeting on Réunion Island are well advanced. The 01 April deadline for submitting an abstract and for 'early-bird' registration is fast approaching. The organising committee has put together an excellent, user-friendly website. There will be workshops on remote-site decompression sickness, technical diving, whether people with epilepsy should scuba dive, keynote addresses from Guest Speakers, plenary sessions and poster displays, all in a beautiful setting. Places are limited, so book now.

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Michael Davis

The front page photo of mating Mandarinfish or Mandarin dragonet (*Synchiropus splendidus*), was taken by Martin Sayer in the waters off Yap.

The President's page

Costantino Balestra, President EUBS

Creativity in science

All of us have heard boring contributions at congresses. Happily for us, other contributions were often funny or inspiring, or both. Having a glance at the available literature, besides the *British Medical Journal* Christmas issue, some interesting papers are available. I say interesting, as that is the important aspect but, of course, humour is interesting *per se*; however, if some other message is surrogate, so much the better.

To encourage young scientists, and to accept new measuring systems, I will choose the youngest author ever to appear in PUBMED, a five-year-old boy, Antoine-Olivier Cyr.¹ This young Canadian boy, helped by his 8-year-old brother and, of course, his 50-year-old father (a professor of medicine) produced a very creative article showing why Tintin did not age or mature sexually. The authors came to the conclusion that this might be the result of too many blows to the head, with several episodes of loss of consciousness leading to a hypophyseal hormone deficiency together with hypogonadism.

To assess their hypothesis, the authors counted the total amount of head traumas that Tintin endured over his long career as an adventurer, namely counting in every published Tintin album the total number of frames where Tintin was unconscious – consecutive frames were counted as the time spent unconscious – and the number of flying sparrows around the head of the unfortunate as the severity index of the trauma. Of course, all those parameters were then compared to modern trauma scales and led to the conclusion that an acquired hormonal deficiency could be diagnosed.

Scientific journals have a long tradition of publishing formal comment-and-reply (sometimes called discussion-and-reply) in which one group of researchers addresses a recently published paper and the authors of that paper have a chance to respond. This example from a 1963 paper about sediment accumulation rates from *The Journal of Geology* is a bit different. In this case, it seems that those writing the formal comment did not take issue with the results or even the implications of the work. They merely wished to point out a minor error in presentation. Here is the key passage from their comment:

“It is obvious that this error in presenting sedimentation rates has no [effect] whatever on the ages given in the paper. Therefore, the main body of the paper and the conclusions reached by Rosholt et al. require no modification.”

To which the authors reply:

“Oh well, nobody is perfect.”

Another classic journal article is the well-known paper on the unsuccessful self-treatment of writer's block.² This received multicentre confirmation very recently (in 2007).³ The best part of the first paper is the response from one of the reviewers:

“I have studied this manuscript very carefully with lemon juice and X-rays and have not detected a single flaw in either design or writing style. I suggest it be published without revision. Clearly it is the most concise manuscript I have ever seen – yet it contains sufficient detail to allow other investigators to replicate Dr Upper's failure. In comparison with the other manuscripts I get from you containing all that complicated detail, this one was a pleasure to examine. Surely we can find a place for this paper in the Journal – perhaps on the edge of a blank page.”

Coming back to the sometimes-boring presentations at meetings and their opposite, the most inspiring one that I have had the opportunity to hear was at the meeting *The future of diving: 100 years of Haldane and beyond* meeting in honour of the retirement of one of our past Presidents, Alf Brubakk. The paper discussed the patterns and rules that could be derived from an in-depth analysis of jazz.⁴ This interesting approach has been really inspiring and appealing to creative production in science; there are a lot of different contexts that could potentially benefit from the 'rules' of jazz ... you do not even need to be a great musician for it. So, for our young scientists a lot is available, creativity is needed and you do not even need to be a great scientist!

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Key words

Medical society, meetings, research, general interest

Original articles

Monitoring cardiac output during hyperbaric oxygen treatment of haemodynamically unstable patients

Marco Bo Hansen, Frederik Treschow, Martin Skielboe, Ole Hyldegaard, Erik Christian Jansen and Jonas Bille Nielsen

Abstract

(Hansen MB, Treschow F, Skielboe M, Hyldegaard O, Jansen EC, Nielsen JB. Monitoring cardiac output during hyperbaric oxygen treatment of haemodynamically unstable patients. *Diving and Hyperbaric Medicine*. 2013 March;43(1):3-8.)

Introduction: Patients suffering from necrotizing fasciitis (NF) are often haemodynamically unstable and require extended monitoring of cardiovascular parameters; yet this is limited during hyperbaric oxygen treatment (HBOT). We aimed to evaluate the use and safety of transoesophageal Doppler (TED) monitoring of cardiac output (CO) under hyperbaric conditions in haemodynamically unstable patients diagnosed with NF and sepsis or septic shock.

Methods: Cardiac output was measured prior to, during and after HBOT with the use of TED in seven consecutive patients diagnosed with NF and sepsis or septic shock. The HBOT followed our standard protocol for NF patients, consisting of 90 minutes' exposure to 100% oxygen at 284 kPa. The difference between mean CO just prior to HBOT initiation and at near-maximum treatment duration was assessed using the Student's paired *t*-test.

Results: TED was feasible and easy to use under hyperbaric conditions. We experienced no problems with the measurement of CO or with equipment-related safety during HBOT. Five patients had an increase in CO from initiation of HBOT to near-maximum treatment duration, one patient had a stable CO, while one patient experienced a slight decrease in CO. Overall, there was an increase in mean CO of 1.7 L min⁻¹ (95% CI 0.02 to 3.34 L min⁻¹, *P* = 0.048) from initiation of HBOT to near-maximum treatment duration.

Conclusion: This is, to the best of our knowledge, the first study to document that TED can provide a minimally-invasive estimate of CO in haemodynamically unstable patients with NF and sepsis or septic shock during HBOT.

Key words

Doppler, cardiovascular, patient monitoring, hyperbaric oxygen therapy, necrotising infections, physiology

Introduction

Hyperbaric oxygen treatment (HBOT) is used routinely as treatment for several clinical conditions including necrotizing fasciitis (NF).^{1,2} In Denmark (population 5.6 million people), the incidence of NF has increased over the years from three new cases in 1997 to 64 cases in 2011. NF is a rapidly progressive, life-threatening soft tissue infection with a high morbidity and mortality.³ The infections are usually polymicrobial, spread along the subdermal facial planes and are often complicated by sepsis or septic shock.^{4,5} The treatment protocol in our centre is based on prompt and aggressive surgical debridement, intravenous antibiotics with broad-spectrum antibacterial coverage, intravenous immunoglobulin, supportive therapy in an intensive care unit and HBOT.⁶

Patients suffering from NF complicated by sepsis or septic shock are haemodynamically unstable and require extensive haemodynamic monitoring; yet this is limited during HBOT because of technical difficulties including fire safety issues and the physical confinement of the hyperbaric chamber. Standard haemodynamic parameters such as blood pressure, heart rate, diuresis, and peripheral oxygen saturation (SpO₂) are often insufficient and fail to reveal the

true haemodynamic status during septic shock.^{7,8} In these circumstances, additional monitoring parameters, such as cardiac output (CO), are needed to optimize treatment of these patients.⁹

Various technologies are available with which to measure CO in the intensive care and peri-operative settings. Transoesophageal Doppler (TED) is an established, validated, and minimally-invasive case method, which has been described in numerous studies as showing good agreement compared to more invasive procedures (e.g., a Swan-Ganz catheter and thermodilution technique) with respect to relative changes in CO.¹⁰ Whether TED can be used to monitor CO in haemodynamically unstable patients during HBOT is unknown. In addition, previous studies have examined how HBOT affects the normal cardiovascular system, but existing theories regarding CO and the haemodynamic profiles during HBOT in NF patients with sepsis are few and contradictory.^{9,11-14}

We aimed to evaluate the use and safety of TED monitoring of CO in haemodynamically unstable patients diagnosed with NF and sepsis or septic shock under hyperbaric conditions.

Methods

STUDY SUBJECTS

The study was carried out in the Hyperbaric Medicine Unit at Copenhagen University Hospital, Rigshospitalet, a tertiary referral centre, where the HBOT treatment of NF has been centralised in Denmark. Patients diagnosed with NF and who received HBOT were offered participation if they fulfilled the study criteria. The inclusion criteria were:

- diagnosed NF and sepsis or septic shock defined as previously described;¹⁵
- HBOT was indicated and
- if the patient was intubated, receiving intravenous sedation and being mechanically ventilated prior to HBOT.

Exclusion criteria were:

- age < 18 years;
- mean arterial blood pressure (MAP) < 60 mmHg despite optimal intensive treatment and intravenous norepinephrine;
- known or suspected oesophageal cancer or other pathological conditions of the pharynx and oesophagus;
- pregnancy or
- in case of deviation from the standard HBOT protocol.

Each patient was only included once. The patients or, if they were already sedated at the time of arrival at the tertiary referral centre, their relatives gave written informed consent. The study protocol was approved by the institutional ethics committee (KF 01 300992).

HYPERBARIC OXYGEN TREATMENT

Patients were treated with HBOT according to a standard protocol for NF. During the first 48 hours after arrival at the tertiary referral centre, patients typically received three sessions of HBOT. Each treatment consisted of pressurization over 5 minutes to a pressure of 284 kPa. The pressure was applied for 90 min followed by 5 min decompression. The multiplace pressure chamber (Drass Galeazzi Underwater Technology, Italy) was pressurized with air, and the patients were mechanically ventilated with 100% oxygen via an endotracheal tube using a Siaretron 1000 IPER™ (Siare, Bologna, Italy) ventilator. During the study, there were no deviations from the standard HBOT protocol or existing safety procedures. Patients did not undergo myringotomy prior to compression

CARDIAC OUTPUT

Cardiac output was measured with transoesophageal Doppler (CardioQ™, Deltex Medical Inc., UK). As the CardioQ™ is currently not approved for usage in a pressure chamber, the monitor was placed outside the chamber. The connection between the monitor and the Doppler probe inside the chamber was established by a pressure-resistant

power cable made specifically for the purpose (Deltex Medical Inc., UK).

Two trained persons performed the measurements; one inside the chamber handling the probe, while the other controlled the CardioQ™ monitor outside the chamber. They were able to communicate via intercom and visually through a porthole in the pressure chamber. Upon arrival at the hyperbaric unit, the TED probe was inserted via the oral or nasal route to the mid-thoracic level between the fifth and the sixth vertebrae where the aorta and oesophagus run parallel. After the initial placement, the probe was rotated for optimal flow signal. On two occasions, insertion of the TED probe was performed on patients with a gastric tube, which slightly prolonged the time to optimal probe positioning.

Since the oesophageal Doppler probe was not produced specifically to be operated under hyperbaric conditions, we performed a single safety test of a standard 6 mm probe prior to the study to ensure that it did not generate heat under pressure conditions. The probe was placed in a test tube (internal volume 200 ml) filled with water and the water temperature was measured. The probe was pressurized with air to a pressure of 284 kPa and followed the standard HBOT protocol for NF patients.

HAEMODYNAMIC MEASUREMENTS

To validate the use and safety of TED during HBOT, several measurements were performed. CO was measured:

- on arrival at the HBOT unit;
- prior to HBOT initiation and at least 10 minutes after any final ventilator changes were made, assuming a steady cardiopulmonary state;
- after 15 minutes at maximum depth;
- after 80 minutes at maximum depth and
- 15 minutes after the end HBOT.

Secondary observations were cardiac index (CI), heart rate (HR), mean arterial pressure (MAP), and estimated systemic vascular resistance ($SVR = [MAP - CVP] / CO$, assuming a central venous pressure = 0). To address a possible effect of HBOT on haemodynamic parameters including CO, we calculated mean differences of these parameters between baseline just prior to HBOT initiation (measurement no. 2) and at near-maximum treatment duration (measurement no. 4). Each measurement was taken as an average of five cycles to minimize the significance of any beat-to-beat variation. All measurements were repeated three times with search for optimal flow signal between each repetition, and the final result was expressed as mean and 95% confidence interval (95% CI).

STATISTICS

Differences between means were assessed using the Student's paired *t*-test. A *P*-value < 0.05 was considered

Table 1

Clinical characteristics of the patients prior to hyperbaric oxygen therapy ($n = 7$); data are mean (SD) unless otherwise indicated;

* degree of sepsis according to Annane et al.¹⁶

Male/female (no.)	5/2	
Age, y (range)	51.3	(29–74)
Body mass index (kg m ⁻²)	26.6	(7.3)
MAP (mmHg)	86	(10.6)
Heart rate (bpm)	88	(11.9)
Arterial blood gas values		
pO ₂ (kPa)	27.2	(14.6)
pCO ₂ (kPa)	4.9	(0.8)
HCO ₃ (mmol L ⁻¹)	22.0	(1.6)
Base excess (mmol L ⁻¹)	-4.0	(2.5)
pH	7.36	(0.1)
K ⁺ (mmol L ⁻¹)	3.7	(0.3)
Na ⁺ (mmol L ⁻¹)	135.6	(5.1)
Ca ²⁺ (mmol L ⁻¹)	1.1	(0.1)
Glucose (mmol L ⁻¹)	9.3	(2.9)
Haemoglobin (g L ⁻¹)	104.7	(9.6)
Haematocrit	0.32	(0.0)
Degree of sepsis*		
Severe sepsis, no.	1	
Septic shock, no.	6	
Norepinephrine infusion, no.	6	
Ventilator settings:		
IPPV/PRVC (no.)	6/1	
Minute volume (L min ⁻¹)	8.7	(1.2)
Peak insp pressure (kPa)	22.1	(6.0)
PEEP pressure (kPa)	6.1	(2.2)

MAP – mean arterial blood pressure

IPPV – intermittent positive pressure ventilation

PRVC – pressure regulated volume control

PEEP – positive end-expiratory pressure

statistically significant. We used the Stata 12.0 software package (StataCorp LP, Collage Station, Texas, USA) for the analysis.

Results

During the safety test of the oesophageal Doppler probe, there was no increase in temperature during the pressurization or during 90 minutes at 284 kPa.

The clinical characteristics of the seven patients studied are summarized in Table 1. An adequate probe position was achieved quickly in all seven cases and appropriate readings of CO were obtained within a few minutes of positioning the probe. We experienced no equipment-related safety problems during HBOT.

Five out of the seven patients had an increase in CO from initiation of HBOT (baseline) to near-maximum treatment length (80 minutes at maximum depth). CO in one patient

remained stable during HBOT, while one patient experienced a slight decrease in CO (Figure 1). The mean increase in CO was 1.7 L min⁻¹ (95% CI 0.02 to 3.34 L min⁻¹) for the seven patients from baseline to near-maximum HBOT duration ($P = 0.048$). CO continued to increase after the completion of HBOT, such that the mean increase in CO 15 minutes after HBOT was 2.3 L min⁻¹ (95% CI 0.63 to 3.99 L min⁻¹, $P = 0.015$). During HBOT, we observed a decrease in mean MAP and a decrease in mean calculated SVR (Figure 1). Mean cardiac index increased from 2.5 L min⁻¹ BSA m⁻² (95% CI 1.5 to 3.5 L min⁻¹ m⁻²) at baseline to 3.3 L min⁻¹ m⁻² at near-maximum treatment duration (95% CI 1.7 to 4.9 L min⁻¹ m⁻², $P = 0.05$). Mean heart rate did not change significantly: 84 bpm (95% CI 64 to 103 bpm) at baseline, 86 bpm at near-maximum treatment duration (95% CI 66 to 105 bpm, $P = 0.227$). Ventilator settings, intravenous medication and fluid administration were not modified from just prior to the initial measurement of haemodynamic parameters until after the last measurement. No patients deviated from the standard protocol for HBOT. All but one patient had their ventilator settings adjusted just prior to the initial measurement (arrival at the hyperbaric unit), increasing the minute volume by 10%.

Discussion

To the best of our knowledge, this is the first evaluation of TED monitoring of CO in haemodynamically unstable patients during HBOT.

METHODOLOGICAL AND EXPERIMENTAL CONSIDERATIONS

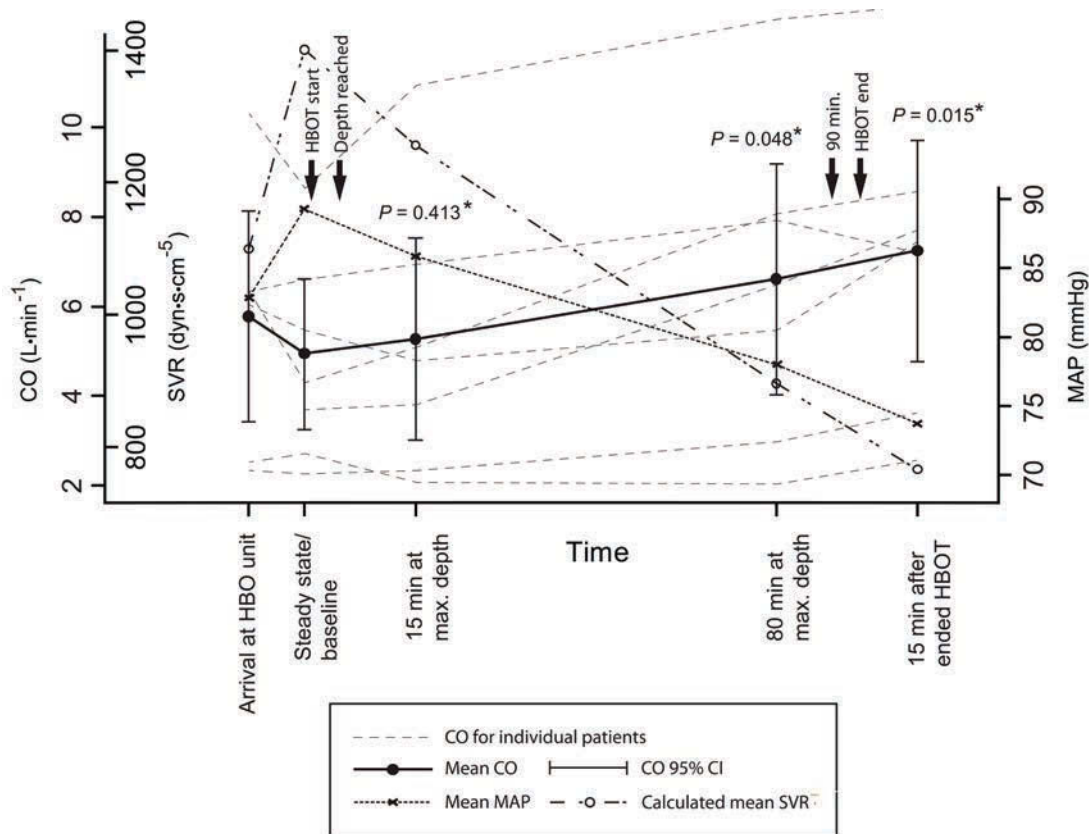
TED has previously been validated as compared to more invasive methods for measurement of CO such as the thermodilution method based on pulmonary artery catheterization (Swan-Ganz technique). The two methods have been found to agree with regard to relative changes in CO (trend monitoring) but less so for absolute values.¹⁰ Accordingly, TED is well suited for estimation of changes in CO and thus the guidance of intravascular volume therapy and inotropic drugs, but not for precise estimation of an exact CO.¹⁰ Neither TED nor the thermodilution method has previously been introduced for routine use in a HBOT setting of haemodynamically unstable patients, partly because it places heavy demands on such a procedure. First, the method should be simple and easy to handle during HBOT. Second, it has to provide valid and reproducible results. Third, it should pose a minimal risk to the patients. Fourth, the method must meet strict safety requirements with respect to electricity and fire precautions. Previous animal experimental studies and clinical trials have used other types of CO measurement during HBOT, with the thermodilution method as the most referenced.^{10–13,16}

In addition to being easy and safe, the use of TED requires only a minimum of technical skills. It has been estimated

Figure 1

Haemodynamic parameters during HBOT ($n = 7$); CO – cardiac output with 95% confidence interval, measured with transoesophageal Doppler; MAP – mean arterial blood pressure; SVR – systemic vascular resistance (calculated from $SVR = (MAP - CVP)/CO$ (assuming central venous pressure = 0);

* difference from 'steady state/baseline' just prior to HBOT



that training in about 10 to 12 patients is needed to achieve adequate positioning of the probe and to obtain reliable CO measurements.¹⁷ In addition, TED has been shown to have a low inter- and intra-observer variability and the risk related to probe positioning is considered as low compared to the risk related to more invasive procedures such as pulmonary artery catheterization.^{10,18} We experienced no problems related to the insertion procedure of the TED probes. However, the time to optimal signal might be slightly prolonged in patients with a gastric tube. During the experiment, we readjusted the probe before each measurement to optimize flow signal. However, the readjustment is probably not necessary under routine clinical settings, which makes the method useful in situations where only limited staff may be available or in situations where pressurization is performed without chamber attendants. The simple technique also minimizes the pressure exposure time of personnel compared to transthoracic echocardiography, where a technician has to be inside the chamber during every measurement.¹⁶

Haemodynamic parameters such as MAP, SpO₂ and heart rate might be insufficient to detect a patient's true haemodynamic status.^{19,20} Studies have shown that TED can

improve early recognition of hypovolaemia and be a guide to intravascular volume replacement and drug-supportive therapy while avoiding the risk of hypervolaemia.^{21,22} This could lead to shorter hospitalization and reduced mortality.²³ Besides providing a continuous estimate of CO during intensive and perioperative care, the fact that we report that TED can be applied under hyperbaric conditions suggests that TED might be used for continuous-trend monitoring of CO in patients with NF from arrival at the hospital until the patient is haemodynamically stable.

TRENDS IN CARDIAC OUTPUT

We observed an initial tendency toward a decrease in CO from arrival at the hyperbaric unit to initiation of HBOT. This decline may well be explained by the fact that the ventilator minute volume was increased by 10% for all but one patient after arrival at the unit, thus increasing positive alveolar pressure and thus decreasing cardiac preload and CO.

Current data suggest that patients with septic shock who experience adequately volume resuscitation are characterized by a hyperdynamic cardiac state with

pronounced vasodilatation and decreased SVR resulting in a compensatory increase in CO. This may partly mask concomitant underlying cardiac dysfunction.^{24,25} Prior reports on CO measurement in healthy subjects find that HBOT causes a reduction in heart rate leading to a decline in CO, but little is known about the effect of hyperbaric oxygen on CO in haemodynamically unstable patients.^{11,26} Only one study in four critically ill patients has examined CO during their HBOT, but with the use of thermodilution in a monoplace chamber and with intermittent air breaks.²⁷ They observed an increase of CO in one patient, a stable CO in two patients, and a decrease in the fourth, whilst we observed a tendency towards an increasing CO during HBOT in five of seven patients, which may reflect the compensated physiologic response patterns seen in patients with sepsis and systemic vasodilation.

This is also supported by the decrease in SVR and MAP observed in this trial. Normally, peripheral vascular tone increases during HBOT due to the concomitant increased arterial oxygen content, but the effect is probably diminished by the infection itself and its vasodilator effect. In addition, HBOT has been reported to have inhibitory effects on inducible nitric oxide synthase (iNOS) expression during sepsis.²⁸ iNOS has been described as a myocardial depressant during sepsis and its down-regulation may contribute to a less pronounced myocardial depression.²⁹ The increase in CO may, therefore, be explained by a change in myocardial contractility due to a combination of sepsis and HBOT. However, it is not possible to draw any conclusion from this study regarding the causal effect of HBOT on CO.

LIMITATIONS

Patients with NF and sepsis or septic shock represent a complex study population where it can be difficult to isolate individual treatment factors that theoretically can affect the haemodynamic status. Potential confounders might be choice of anaesthesia, level of sedation, intravascular volume therapy, pressor agents, ventilator settings, degree of sepsis, and localization of NF. We tried to minimize these confounding effects by keeping the parameters constant during treatment. Additionally, this study was not designed specifically to elucidate the precise role and mechanisms of HBOT in CO.

Further studies with larger study populations and more standardized experimental conditions are needed. However, this study provides a template for future research on this area.

Conclusions

This is the first study to document that TED can be successfully adapted to hyperbaric conditions to provide a minimally-invasive estimate of CO in haemodynamically unstable patients with NF and sepsis during HBOT. In addition, we observed a rise in CO during HBOT in five

of seven patients. Future studies with more individuals are feasible and needed in order to draw conclusions regarding the precise effects of HBOT on CO in NF patients.

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Long-term analysis of Irukandji stings in Far North Queensland

Teresa J Carrette and Jamie E Seymour

Abstract

(Carrette TJ, Seymour JE. Long-term analysis of Irukandji stings in Far North Queensland. *Diving and Hyperbaric Medicine*. 2013 March;43(1):9-15.)

Introduction: We reviewed the occurrence, trends, definition and severity of the Irukandji syndrome for the Cairns region of North Queensland, Australia.

Methods: A retrospective analysis of patient files from two sources was conducted: historic accounts kept by Dr Jack Barnes for the period 1942 to 1967, and records from the Emergency Unit in Cairns Base Hospital for 1995 to 2007.

Results: There has been a significant increase in the length of the Irukandji season since it was first reliably recorded (15 days in 1961; 151 days in 2002); however, annual numbers of envenomations were highly variable. Traditionally, greater frequencies of Irukandji stings were reported at onshore as opposed to offshore locations. However, in recent years this trend has reversed, potentially because of increased safety protocols for beach regions. Mean Troponin I levels were higher in offshore reef envenomations compared to those from islands or coastal regions. In terms of morphine-equivalent doses, patients given fentanyl received significantly greater opioid doses compared to those given morphine or pethidine. Opioid dosage was indicative of syndrome severity and correlated with other physiological parameters measured. Five major symptoms were associated with Irukandji syndrome: pain, nausea/vomiting, diaphoresis, headache and shortness of breath. Pain was the overwhelming symptom, followed closely by nausea/vomiting.

Conclusions: The duration of the Irukandji season appears to be increasing. Conversely the number of envenomings appears to be decreasing, possibly because of improved beach management in recent years. Offshore envenomings appear to have a higher potential for more severe envenomings with five associated major symptoms.

Key words

Marine animals, jellyfish, envenomation, medical database, pain, treatment, epidemiology

Introduction

Irukandji syndrome is a set of debilitating symptoms, first described from around Cairns, North Queensland, Australia, arising from envenomation by particular species of box jellyfish.¹⁻³ Initial envenomation is typically recorded as insignificant; however, after a delay of generally 20 to 60 minutes, systemic symptoms including headache, backache, nausea, vomiting, abdominal cramps, hypertension, tachycardia and feelings of impending doom develop.³⁻⁵ Although numerous case reports occur annually from this area, great disparity still exists not only in the reporting of the syndrome but also its seasonal occurrence.^{1,3}

The Irukandji season in Australia has previously been reported to start as early as October and run as late as May; however, envenomations occur in all months bar July and August.⁶⁻⁹ The peak time for Irukandji envenomations in the Cairns region has been declared around December/January; however, these observations were made over a single season with no long-term analyses being documented.^{9,10} There is some suggestion of a potential correlation between sting incidence and the ecology of the animals responsible, but this may only reflect conditions in which people opt to utilize the beaches with higher frequency.^{11,12} For example, Christmas Day has one of the greatest recorded incidences of sting occurrence, potentially reflecting increased beach usage.⁶

While the syndrome was originally described as only affecting bathers utilising the sandy coastal beaches and not

on the reef, envenomations from the outer reef and island regions are now commonly reported, with some of the serious cases documented from these offshore locations.^{5,12,13} Anecdotal reports have suggested that there may be a pattern in the timing of the more severe envenomations that present to hospital, with the general consensus being that the more serious Irukandji stings present later in the season.⁴ However, as with the sting-severity hypothesis, no empirical data currently exist to support this premise.

Presently, no detailed studies on the ecology of Irukandji jellyfish exist, so the only avenue available to uncover patterns in Irukandji syndrome envenomations is the retrospective analysis of patient files. To this end, data from Irukandji syndrome envenomations in the Cairns region, covering a period of 65 years, were analysed to investigate trends in sting occurrence in the region. Additionally, trends in sting severity and treatment success were sought for potential insight into improving clinical management. Finally, we investigated the frequency and range of symptoms.

Materials and methods

PATIENT RECORDS

Patients who suffer from Irukandji stings are typically coded as either "marine sting", "Irukandji sting", or "sting from venomous jellyfish or starfish", and these patients were extracted from the Cairns Base Hospital database for potential inclusion into this study. Patients included in

the trial were those who had contact with seawater pre-60 minutes of symptoms developing, a delay in symptoms from an initial sting and at least one of the defined systemic clinical symptoms, which included headache, nausea, anxiety, vomiting, sweating, restlessness, muscle cramps in all four limbs, abdomen and chest or severe lower back pain.¹⁴ Any stings that were deemed to have resulted from contact with a large Chirodroid jellyfish (i.e., *Chironex fleckeri* or *Chiropsella bronzie*) or those from the hydrozoan *Physalia* sp., (noted by the visible and/or substantial welts with an absence of systemic symptoms) were excluded. A total of 347 envenomations covering the years from 1995 to 2007 were included and accessed under Cairns Base Hospital Ethics Committee approval number 287. In several cases, not all categories of data were able to be extracted from the medical records (e.g., geographic location or total amount of opioids administered due to treatment at other locations before transfer to Cairns Base Hospital, admittance into a randomized controlled trial using magnesium), and these cases were excluded from specific analyses if the relevant data were missing.

ADDITIONAL HISTORIC DATA INCLUSION

A historic sting database exists from Dr Jack Barnes' comprehensive records of sting cases from 1942 to 1967.¹⁵ These cases were all from Irukandji syndrome stings from the Cairns region and were all seen and documented by Barnes.¹⁵ The dates of Irukandji syndrome stings from this report were added into this retrospective study for analysis into the occurrence of Irukandji syndrome stings with time.

DATA COLLECTED

Information extracted from patient files covered the three main areas of patient demographics, sting occurrence and symptoms/treatment progression. Some files were incomplete, so not all areas could be comprehensively recorded for all stings. The following factors of envenomed patients were recorded:

- Total opioid requirements in morphine-equivalent doses. Three opioids – pethidine, morphine and fentanyl – were used and dosages were calculated in terms of morphine-equivalent doses to allow for direct comparison; 1 mg morphine = 10 mg pethidine = 10 µg fentanyl.⁵
- The observed peak percentage blood pressure (BP) increase, measured as both the maximum mean arterial pressure (MAP, approximated using the equation: $MAP = (2 \times \text{diastolic pressure} + \text{systolic pressure})/3$), and peak systolic pressure from regular observations. BP just prior to discharge was deemed indicative of the patient's 'normal' level. The peak recorded BP was calculated as a percentage increase above this 'normal' reading.
- The observed peak percentage heart rate (HR) increase: the maximum HR recorded during regular observations while in hospital care was calculated as a percentage increase to the HR recorded at discharge.
- Troponin I level (cTnI, normal < 0.7 g L⁻¹).
- Length of stay (hours) from first admission for each patient was assumed to indicate the level of care needed for the syndrome to subside.

Additional to the physiological information gathered, logistical information on sting events was recorded including, where possible, the geographic location of stings. To distinguish between geographical regions, three categories were selected (these are thought to reflect the different habitats Irukandji jellyfish may inhabit). Any stings occurring from the coastal beaches were defined as 'onshore'; stings originating from water activities around the coastal islands were classified as 'islands'; and stings that occurred from the outer reef regions were classed as 'reef' stings. All the documented symptoms were recorded, with those that appeared in at least 5% of the cases marked into categories. These symptoms recorded throughout all the cases examined were found to fall into five categories: pain (limb pain, back pain, abdominal pain and chest pain), headache, nausea and vomiting, diaphoresis and shortness of breath, so these and troponin levels were used for analysis.

DATA ANALYSIS

For several of the analyses, data were non-normally distributed and residuals were heteroscedastic. In these cases, data were transformed to normalize the distribution and produce homoscedastic residuals to avoid violation of assumptions for a general linear model analysis. A general linear model (ANOVA) was used to investigate the effect of year (1995/6 to 2006/7) and/or geographic location (onshore, reef or island) on the length of the Irukandji season. Post hoc analysis (LSD) was performed on significant effects to determine which treatment means were different. Only stings from 1995/6 onwards were used for this analysis as this information was lacking from many records in Barnes' published database. Similarly an ANOVA was used to elucidate any effects location and/or year had on the level of Troponin I leakage seen in envenomed victims.

Regression analysis was used to determine the relationship between the length of the Irukandji season and time (from 1956 to 2007). Chi squared analysis was used to compare the ratio of stings seen onshore versus offshore locations with each year from 1995 to 2007. Bivariate correlations were performed to determine the significance of correlations between morphine-equivalent dosage and length of stay, percentage MAP and systolic BP changes or Troponin I level.

Finally, to determine if mean morphine-equivalent dosage varied with the number of symptoms recorded (pain, headache, nausea/vomiting, diaphoresis, shortness of breath) or Troponin I leakage, data were analysed using ANOVA and LSD post hoc to determine within-treatment differences.

Figure 1

Scatter plot of total length of stinger season annually in days based on stings recorded (R^2 linear = 0.427)

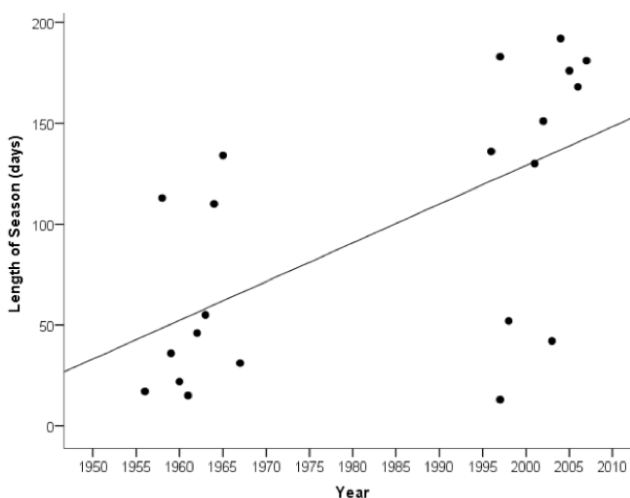
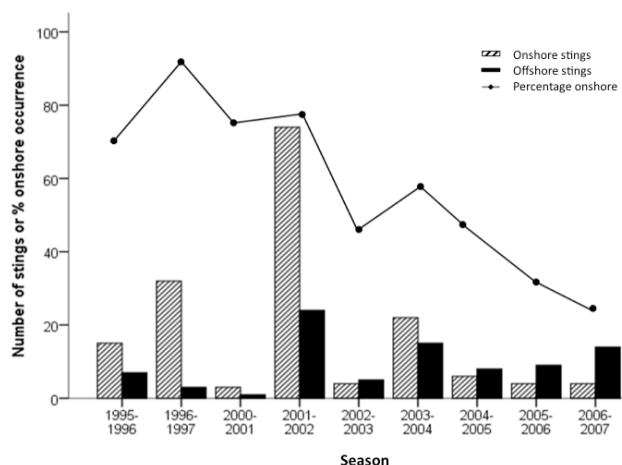


Figure 2

Frequency of recorded Irukandji stings from both offshore and onshore locations in the Cairns region and percentage of those stings that occur onshore



Results

DISTRIBUTION OF ENVENOMINGS

Patients in this study consisted of 55% males and 45% females. Patients ranged in age from 1 to 77 years old with a mean of 24 years. Children (defined in Queensland as age 16 years and under) comprised 25% of the sample population.

There was a significant positive correlation ($F_{(1 \times 19)} = 10.822$, $P < 0.005$) between year and the length of the Irukandji syndrome season, with a minimum of 15 days in 1961 to a maximum of 151 days in 2002 (Figure 1). For stings recorded from 1995 to 2007, significantly more stings occurred onshore than offshore in the earlier years; however, this trend reversed with more stings occurring offshore ($\chi^2_{(8)} = 32.9$, $P < 0.001$) in later years (Figure 2). There was a significant negative correlation ($F_{(9 \times 1)} = 22.03$, $P < 0.05$) between percentage of stings onshore and season with a high of 93% in 1996–1997 and a low of 26% in 2006–2007.

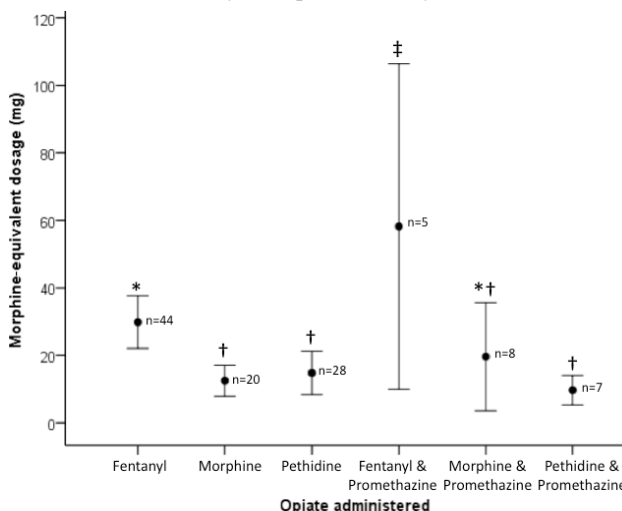
OPIOIDS FOR PAIN RELIEF

The majority of patients given opioids for pain relief received fentanyl (26%) or a mixture of opioids (20.2%). Fewer patients were given pethidine (16.2%) or morphine (11.6%) alone or various combinations of all three opioids. Envenomed patients treated with a fentanyl/promethazine combination required significantly higher ($F_{(106 \times 5)} = 6.230$, $P < 0.001$) mean morphine-equivalent doses (mean = 59 mg) than those treated with any other opioid combination (Figure 3). Those treated with fentanyl alone required significantly higher morphine-equivalent doses than those treated with morphine, pethidine or pethidine/promethazine (Figure 3).

There was a significant positive correlation (Pearson's correlation 0.499) between opioids and symptoms

Figure 3

Mean total morphine-equivalent doses for different opioids (means followed by same symbol are not statistically different at the 0.05 level by LSD post hoc analysis) ($n = 143$)



experienced, with patients receiving significantly higher amounts of opioids as more symptoms developed (Figure 4).

Approximately 31% of patients diagnosed as suffering from Irukandji syndrome showed only one of the five major symptoms, with 43% recording two symptoms (Figure 5). Notably, only one patient showed all five symptoms. For patients experiencing only one symptom, over 80% of these were pain of some description (chest, back, limb pain). The second most predominant symptom was nausea/vomiting with troponin rises being displayed only in patients who showed three or more clinical symptoms (Figure 6).

Morphine-equivalent dosage was shown to have a strong positive correlation with all variables examined but was more

Figure 4

Mean morphine-equivalent dose received (mg) for number of symptoms reported

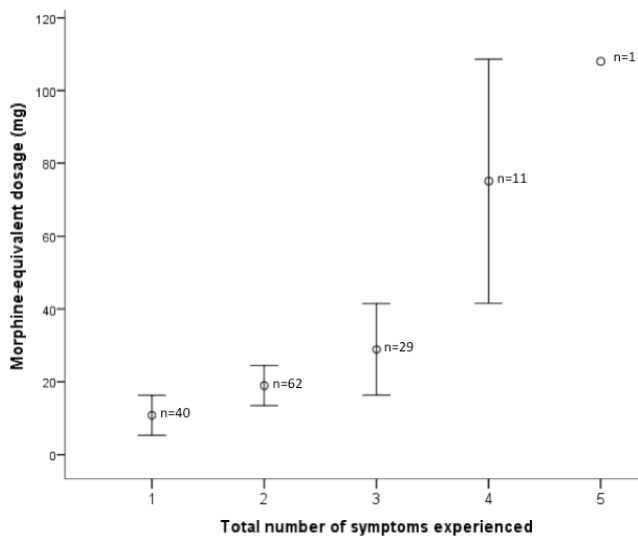
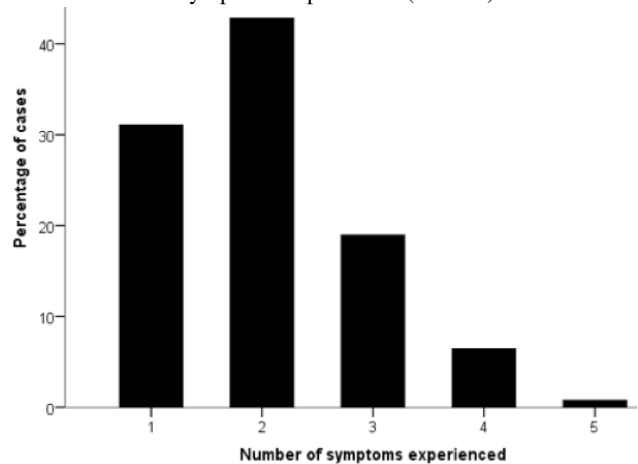


Figure 5

Mean percentage of cases presenting with the number of symptoms experienced (n = 143)



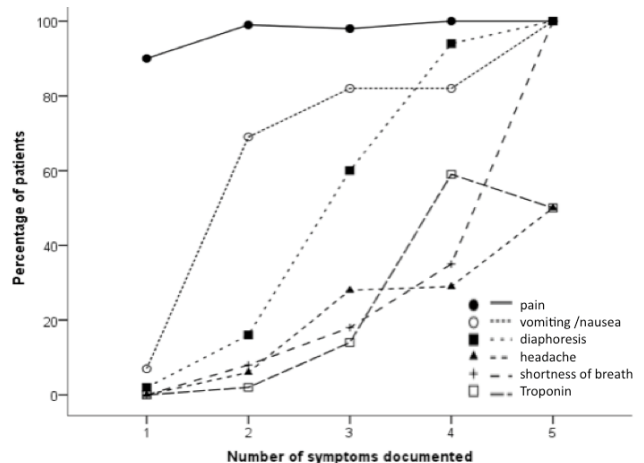
strongly positively correlated with length of hospital stay (Pearson corr. (r) = 0.665), than with cTnI levels (r = 0.509), percentage BP change (r = 0.441), % MAP (r = 0.328) or percentage HR change (r = 0.195). The mean, minimum, maximum and median data for morphine-equivalent dosage, peak BP, peak MAP, peak HR, cTnI level and length of stay are presented in Table 1.

TROPONIN I

Envenomed patients from reef locations had significantly higher levels of cTnI (mean = 3.78 µg L⁻¹) than those from either the island (mean = 0.99 µg L⁻¹) or onshore locations (mean = 1.06 µg L⁻¹) (F_(2x152) = 7.577, P = 0.001; Figure 7).

Figure 6

Percentage of patients presenting with the number and type of symptoms reported (n = 143)



Discussion

Irukandji syndrome, while difficult to determine, has most recently been defined as requiring at least three systemic clinical symptoms, including nausea, vomiting, headache, sweating, anxiety, restlessness, muscle cramps in all four limbs, the abdomen and chest or severe low back pain.¹⁴ Investigation of the Cairns records shows that the use of this definition would in fact exclude 74% of Irukandji sting

cases, with 31% of patients examined displaying only one symptom. As such, we suggest that a new variation of the definition for the syndrome be:

- recent contact with seawater;
- a delay of 5 to 60 minutes between a relatively mild sting and the onset of constitutional symptoms and
- one or more of the following five symptoms: pain,

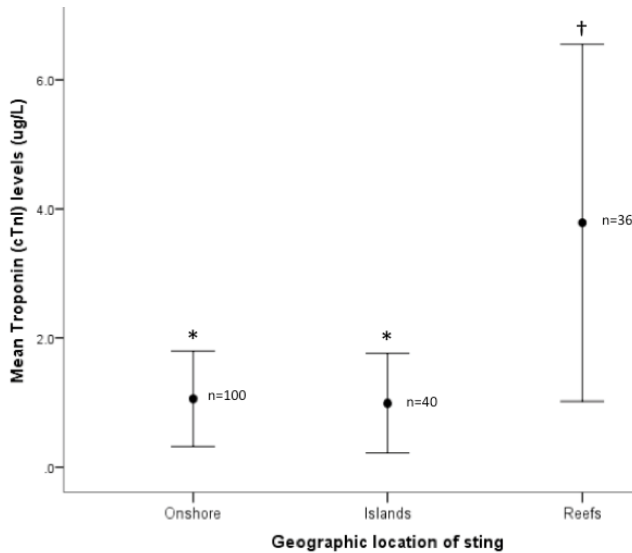
Table 1

Range of physiological parameters recorded for envenomed patients documented

	n	Mean	Median	Mode	Range	Minimum	Maximum	25th percentile	75th percentile
Morphine-equivalent dose (mg)	264	25	13	0	255	0	255	1.2	35
Peak blood pressure (mmHg)	251	141	140	130	140	90	230	125	155
Peak MAP (mmHg)	249	105	105	100	115	68	183	93	116
Peak heart rate (bpm)	204	95	93	80	156	47	203	80	107
Troponin score (ug L ⁻¹)	189	1.5	0	0	34	0	34	0	0.1
Length of stay (h)	265	16	12	3	167	1	167	3.9	21

Figure 7

Mean detected Troponin I (cTnI) levels in patients stung from different geographical locations (means followed by same symbol are not statistically different at the 0.05 level by LSD post hoc analysis) ($n = 176$)



headache, nausea and vomiting, diaphoresis and shortness of breath, +/- cTnI leak.

To date, this is the largest data set of Irukandji envenomings analysed, spanning 65 years, with the majority of previous studies focusing on only a single season's records. These findings give insight into the seasonal occurrence of the syndrome, symptoms that are displayed and the treatment and management of this disease.

These data suggest the length of the Irukandji season in the Cairns area appears to be increasing with time. With the global rise in seawater temperatures, a prolonged state of optimal temperature conditions may exist, which in turn may allow medusae to survive until later in the year than they have in the past.¹⁶ Certainly there has been a trend in other cnidarian species to increase in numbers due to sea temperature increases, eutrophication and possible over-fishing.¹⁷ The northern Queensland data, while not conclusive, may support this hypothesis. This has direct consequences to physicians who may be under the impression that this syndrome only occurs over a few months of the year.

These data also show that recently there has been a change in the ratio of onshore to offshore stings, with onshore sting numbers decreasing. Although impossible to test, a potential for this reversal in trend is the increase in beach safety protocols that have been instigated in recent years. In 2001, there was a change in the Irukandji sting protocols of Surf Lifesaving Queensland for onshore beach locations, to include a 24-hr mandatory closure if a positive Irukandji sting occurred, and the addition of daily drags conducted by surf lifesavers to check for the presence of carybdeids

on a routine basis (SLSQ Stinger protocol). The sting data that coincide with this new management strategy are supportive of this increased safety approach for beachgoers in decreasing the sting incidence. No such strategy has been initiated for offshore reef and island regions and, with increasing numbers visiting these regions, there is great potential for sting numbers to increase. A preventative strategy and risk management system is essential in these regions if occurrence of this syndrome is to be controlled.

Of additional concern for the reef envenomings is the higher levels of cTnI measured in patients from this region. While cTnI levels are used as an indicator of myocardial damage and the link to Irukandji syndrome patients experiencing myocardial dysfunction has been reported previously, there is uncertainty as to the relationship of these levels and their association with cardiac dysfunction.⁵ Patients with elevated cTnI levels have been flagged as potentially developing cardiac complications and, as patients originating from reef regions show higher mean levels, origin of sting would seem a potential tool for severity diagnosis in Irukandji syndrome presentation. Further evidence for this comes from the only recorded Irukandji syndrome-related death in this region that occurred from an offshore reef location, with this patient also displaying vastly elevated cTnI levels.^{5,18}

Additional to geographical origin for sting severity prediction would be the number of symptoms experienced, with patients who display three or more of the defined symptoms, increasing their propensity for cTnI presence. This suggests that a combination of sting origin, opioid requirements and number of symptoms displayed could be used as a risk assessment technique for patient severity prediction, assuming that those with an elevated cTnI are at higher risk.

Effective pain relief is the key to managing Irukandji syndrome. Opioid requirements varied and, with its substantially shorter half-life than that of either morphine or pethidine, higher total doses of fentanyl were required for pain relief; the shorter duration of action requiring more frequent doses to maintain effective blood concentrations.^{19,20} Morphine has been reported to be more likely to induce vomiting in Irukandji syndrome patients, whilst pethidine in large doses may possibly cause seizures and cardiac depression, worsening myocardial function of patients with Irukandji syndrome.^{3,9} Therefore, pethidine is not recommended in Irukandji syndrome.⁹ Fentanyl would appear to be the opioid of choice because of its relatively low toxicity and good tolerance profile reported in other studies.^{9,21} However, individual patient responses vary and each case must be assessed with patient safety considerations foremost.²¹ There is no reported experience with the newer fast-acting opioids such as remifentanyl.

Promethazine has previously been suggested as improving the outcome of patients with this syndrome by lowering the amount of opioid required and because of its antiemetic,

antihistaminic and sedative effects.⁹ However, our data appear contrary to this view, with no significant decrease in morphine or pethidine dosages and a higher average dosage of fentanyl in patients receiving promethazine. While promethazine may provide some favourable effects for the patient, there is no evidence in this series to suggest it affects opioid dosage advantageously.

Ecological data on the types of carybdeids giving rise to Irukandji syndrome are still lacking and such fundamental questions as which species are involved and how these animals vary both geographically and seasonally are needed urgently. Control measures for onshore coastal locations appear to have reduced the number of envenomings in the short term; however, a broader, proactive warning system for all regions would be highly valuable, and this cannot be accomplished without further widespread investigations. Irukandji syndrome represents a significant health problem in the North Australian region, but there is also evidence for its increased reporting from other global locations.^{12,22-24} With evidence for the season increasing in length, medical practitioners are now facing exposure to patients presenting with this syndrome for approximately six months of the year so further clarification of protocols and treatment strategies is paramount to ensure optimal patient treatment.

Conclusions

This retrospective study indicates that the season for Irukandji stings is increasing. The percentage of these stings originating from the offshore reef areas is also increasing and these stings have shown an increased potential for cardiac complications as indicated by higher levels of cTnI in these patients. Opioid dosage appears to correlate with the severity of the syndrome. Although total dosage (in morphine equivalents) is greater with fentanyl, it is probably the opioid of choice, as it appears to have fewer adverse reactions than, for instance, pethidine, which may increase complications in Irukandji syndrome. Finally, we believe that a new definition for the syndrome should be made, mainly marine envenomation with a delay in the onset of one or more of five major systemic symptoms.

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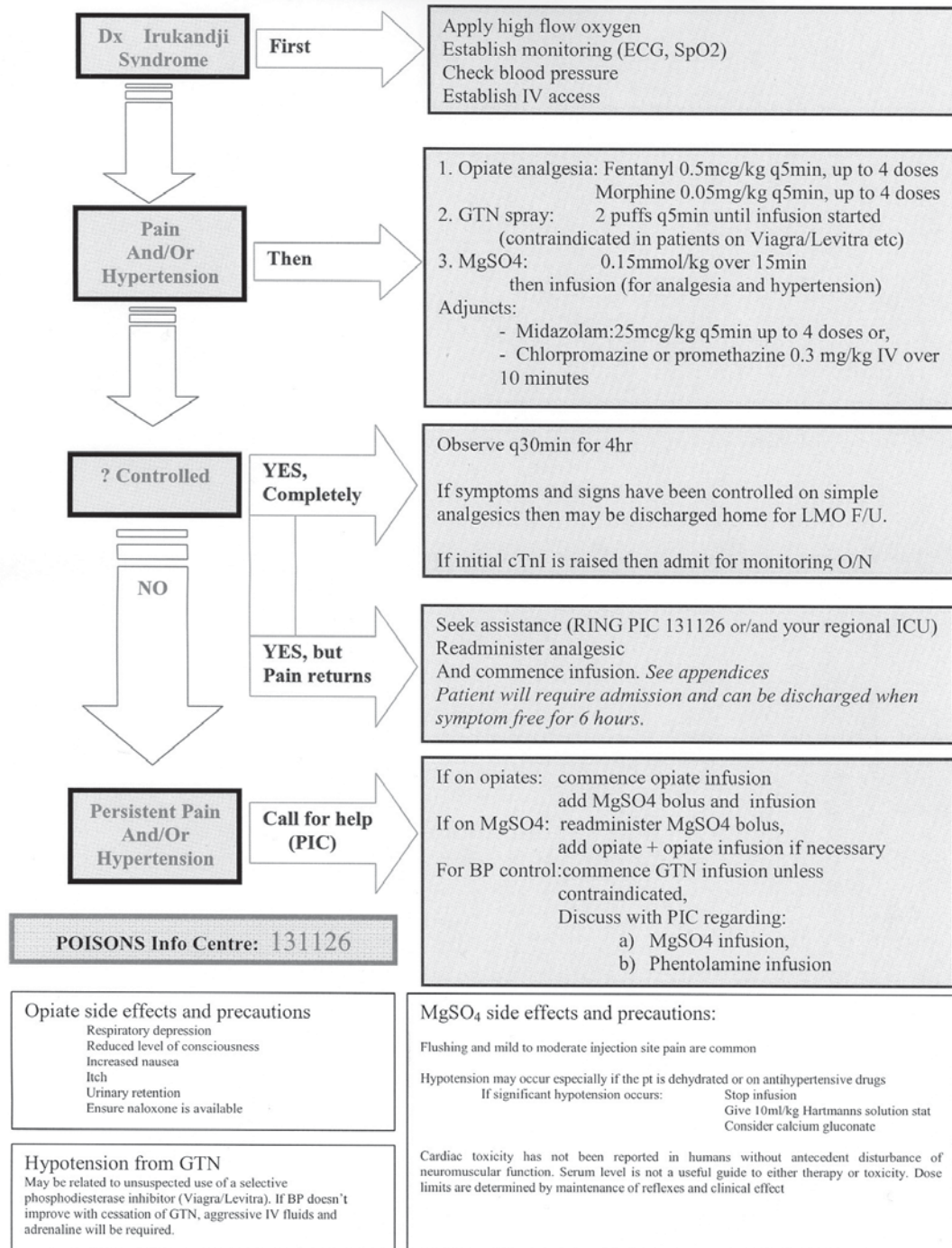
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Guidelines for the Management of Irukandji syndrome



Provisional report on diving-related fatalities in Australian waters 2008

John Lippmann, Christopher Lawrence, Thomas Wodak, Andrew Fock, Scott Jamieson, Douglas Walker and Richard Harris

Abstract

(Lippmann J, Lawrence C, Wodak T, Fock A, Jamieson S, Walker D, Harris R. Provisional report on diving-related fatalities in Australian waters 2008. *Diving and Hyperbaric Medicine*. 2013 March;43(1):16-34.)

Introduction: An individual case review of diving-related deaths, reported as occurring in Australia in 2008, was conducted as part of the DAN Asia-Pacific dive fatality reporting project.

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

Results: In total, there were 19 reported fatalities (the same as for 2007), 17 involving males. Twelve deaths occurred while snorkelling and/or breath-hold diving, and six while scuba diving. One diver died while using surface-supply breathing apparatus. Two breath-hold divers appear to have died as a result of apnoeic hypoxia, at least one case likely associated with hyperventilation. Two deaths resulted from trauma: one from impact with a boat and the other from an encounter with a great white shark. Cardiac-related issues were thought to have contributed to the deaths of five snorkellers and at least two, possibly three, scuba divers.

Conclusions: Trauma from a marine creature, snorkelling or diving alone, apnoeic hypoxia and pre-existing medical conditions were once again features in several deaths in this series.

Key words

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

Introduction

Each year in Australia there are a number of deaths associated with snorkelling and diving using compressed gas (i.e., scuba or surface-supply breathing apparatus, SSBA). Although some incidents are unavoidable, many might have been avoided through more extensive education and training, greater experience, better planning and decision-making, appropriate medical screening, improved supervision, and/or better equipment choice and maintenance. The aim of the Diver Alert Network (DAN) Dive Fatality Reporting Project is to educate divers and the diving industry and to inform physicians on the causes of fatal dive incidents in the hope of reducing their incidence in the future and of detecting, in advance, those who may be at risk. This report discusses the diving-related fatalities between 01 January and 31 December 2008 that are recorded on the DAN Asia-Pacific (AP) database. When an incident is unwitnessed, it is difficult to determine exactly what has occurred. We have sometimes included considered speculation within the comments to provoke thought about the possible sequence of events leading to death.

Methods

As part of its ongoing 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 08/01

This 21-year-old, fit, healthy male was an experienced and frequent spear fisherman. On this day, he went spearfishing from a boat while a friend remained on board. There was a light wind and the sea was dead calm. He wore mask, snorkel, fins, a full wetsuit with hood, a knife and a weight belt with 4 kg of weight and was carrying a speargun. He did multiple breath-hold dives at between 15 and 20 metres of seawater (msw) at a variety of locations and caught many fish. On two occasions, he encountered large sharks which harassed him and tried to take his speared fish.

He was often observed to take several deep breaths prior to diving. On one dive, his friend noticed him rising towards the boat before "conking out" just below the surface beside the boat. The friend reached over and managed to grab the diver and lift him to the surface. The diver began to cough and, when asked if he had become unconscious, he denied this and said that he had just taken in some water. The friend was concerned that the victim was slightly disoriented but could not dissuade him from retrieving his spear, which had become embedded in a rock at a depth of about 20 metres' sea water (msw) after he had fired at a fish. The victim

re-entered the water and, after some hesitation, took some deep breaths and descended. When he failed to surface after an extended period, the friend became concerned. Several minutes later he saw a dark shape below the surface which he thought might possibly have been the victim but could not identify this or reach it before it seemed to drift away in the current. Some of the victim's equipment was found at the dive site three days later. The weight belt and wetsuit remnants indicated shark bites. His body was never found.

Comments: This diver practised hyperventilation prior to breath-holding, a well-known precipitant to apnoeic hypoxia of ascent. It is likely that he became unconscious, or very nearly so, briefly on the previous dive and he took a great risk diving again in his apparently disoriented condition. It is possible, given his friend's statement of events, that he suffered an underwater hypoxic blackout, subsequently drowned and that the sharks later attacked his body. It is also possible that shark attack was the primary event.

Summary: Fit and healthy; experienced spear fisherman; known to practise hyperventilation; apnoeic hypoxia on previous dive; appeared disoriented but dived again to retrieve spear; failed to surface; equipment found showed evidence of shark contact; body never found; probable drowning following post-hyperventilation apnoeic hypoxia; evidence of shark attack

BH 08/02

This 82-year-old male, an overseas tourist, was a passenger on a cruise ship visiting the Great Barrier Reef (GBR). He had a history of several heart attacks and was currently taking aspirin, a statin (type unknown) and nitrazepam but reportedly looked fit for his age. He had snorkelled about five times before, most recently one month earlier. Although he failed to disclose to the dive instructor any health conditions or that he was taking medications, the instructor was concerned about him because of his age and apparent anxiety. He alerted his supervisor and they agreed to observe him closely, along with some other elderly participants. There were nine snorkellers in the victim's group.

The victim entered the water wearing mask, snorkel, fins and a Lycra suit, and with a 'noodle' to provide additional floatation. Some current was running along the reef, and there was a moderate chop. The conditions were later described by the instructor as "*not perfect*", and that it would have been "*tough*" for some participants, including the victim. After a short time, the victim approached the instructor and was out of breath but said he was "*OK*", and, after catching his breath, returned to snorkelling. The instructor was then asked to assist another snorkeller to the boat and when he returned, an estimated two minutes later, he noticed that the victim was away from the group but appeared to be snorkelling normally. However, when he approached the victim, he found him to be unconscious, cyanotic and apparently apnoeic so he began in-water rescue

breathing and signalled for help. The tender arrived and the victim was dragged on board. There was insufficient space to perform cardiopulmonary resuscitation (CPR) so the instructor continued providing rescue breaths on the way to the main boat. Once on board, CPR was begun with the rescue breaths enhanced with supplemental oxygen. The delay from determining unconsciousness to commencing CPR was thought to have been about 10 minutes. After several cycles of CPR, gurgling sounds were heard while ventilating and the victim was briefly rolled onto his side to drain water from his mouth. His dentures also became displaced and were removed to facilitate a better mask seal. CPR was continued throughout the 35-minute boat trip back to the island base, but was unsuccessful.

Autopsy: The heart was heavy (481 g; normal range 331–469 g) but the ventricles were of normal thickness. The coronary arteries showed severe atherosclerosis with severe narrowing of the right coronary artery (~1 mm diameter) by atheromatous plaque 40 mm from its origin. The left coronary artery had significant narrowing 45 mm from its origin. There was subendocardial fibrosis of the myocardium on microscopic examination. The left and right lungs weighed 673 g and 579 g, respectively and there was evidence of moderate congestion and oedema. However, there did not appear to be excessive pulmonary oedema fluid. Toxicology screening showed traces of nitrazepam and aminonitrazepam. The cause of death was given as ischaemic heart disease due to severe double-vessel atherosclerosis.

Comments: This elderly victim withheld important information about his cardiac history. Had he been honest, he may not have been permitted to snorkel and this incident could have been averted. The dive instructor appeared to have been reasonably vigilant but was responsible for a group and was required to divert his attention from a person who required his attention to assist someone else.

Summary: Elderly; history of myocardial infarctions; limited experience; current and surface chop; became breathless (although claimed to have recovered); silent unconsciousness; cardiac-related death

BH 08/03

This victim was a 66-year-old, obese (BMI = 33.5 kg m⁻²) woman with a history of breast cancer and was under treatment for hypertension. She was taking doxazosin, spironolactone and celecoxib. She was reported to have been a good swimmer but had little snorkelling experience. She was an overseas tourist on a day trip to the GBR, accompanied by her daughter.

The weather was sunny with a light wind and the sea was calm. The victim was wearing bathers and t-shirt, mask, snorkel and fins. She and her daughter entered the water and, when the victim had trouble clearing water from her snorkel, her daughter got her a 'noodle' for additional

Table 1
Summary of snorkelling and breath-hold diving-related
BNS – buddy not separated; BSB – buddy separated before problem;

ID	Age	Sex	Height (cm)	Weight (kg)	BMI (kg m ⁻²)	Training	Experience	Dive group
BH08/01	21	M	183	n/s	–	n/s	yes	solo
BH08/02	82	M	175	80	26.1	n/s	some	GSB
BH08/03	66	F	164	90	33.5	n/s	some	BNS
BH08/04	52	M	176	77	24.9	n/s	n/s	GSB
BH08/05	53	M	178	85.5	27.0	nil	some	solo
BH08/06	47	M	n/s	n/s	–	nil	nil	BSB
BH08/07	21	M	181	74	22.6	n/s	yes	BSB
BH08/08	25	F	155	52	21.6	n/s	some	GNS
BH08/09	42	M	179	95	29.6	n/s	yes	solo
BH08/10	46	M	181	98	29.9	trained	yes	GSB
BH08/11	51	M	n/s	n/s	–	n/s	yes	BNS
BH08/12	25	M	170	78	27.0	n/s	yes	BSB

buoyancy. About a minute after they resumed snorkelling the daughter found her mother lying motionless, face-down and unresponsive. The daughter called for help and some of the charter boat staff retrieved the victim and towed her back to the boat, providing rescue breaths on the way. She was brought aboard and four doctors, who were passengers on the vessel, came to assist. The victim was cyanotic, apnoeic and pulseless and CPR was begun by the doctors, assisted by some crew members. An oropharyngeal airway was inserted and she was ventilated via a bag-valve-mask with supplemental oxygen. The victim regurgitated a large amount of stomach contents. An automated external defibrillator (AED) was attached after 10–15 minutes of CPR and one shock was advised and delivered but the victim failed to respond. No further shocks were indicated and the doctors pronounced the victim to be deceased after a total of 44 minutes of CPR.

Autopsy: There was over 400 ml of blood in the pericardial sac. The source of the haemorrhage was a rupture of the anterior wall of the left ventricle in an area of myocardial discolouration due to recent myocardial infarction. The heart weight (283 g) was within normal limits for body weight (213–457 g). The coronary arteries showed severe focal calcific atheroma with near-complete occlusion of the left main coronary artery and adjacent proximal left anterior descending (LAD) artery and a small left circumflex artery. The right coronary artery showed less than 60% luminal

narrowing. Histology of the heart showed necrosis and haemorrhage consistent with days-old recent infarction. The lungs (Lt 456 g; Rt 489 g) showed congestion and mild pulmonary oedema. Toxicology screening was not done. The cause of death was given as cardiac tamponade due to cardiac rupture resulting from recent myocardial infarction.

Comments: The dive operator reportedly mentioned the potential risks associated with certain medical, including cardiac, conditions, but the daughter stated that she and her mother did not take much notice, believing that it did not apply to them. The autopsy indicated that the victim had suffered a myocardial infarction in the previous few days but there was no mention in the witness reports that the victim had complained of any symptoms, or appeared to have been unwell. Typically rupture occurs three to five days post infarct owing to to weakening of the wall by tissue necrosis. Among forensic cases with rupture, the symptoms of the acute infarct are not infrequently mistaken for indigestion. The victim may well have died undertaking a variety of other activities, although water is an unforgiving medium in which to become unconscious.

Summary: Hypertension; 3–5 days post myocardial infarction (not disclosed, and may have been undiagnosed antemortum); good swimmer; little snorkelling experience; calm conditions; silent death; cardiac related

Table 1 (cont.)

fatalities in Australian waters in 2008

GNS – group not separated; GSB – group separated before problem; n/s – not stated

Dive purpose	Depth (msw)	Incident (msw)	Weight belt	Weights (kg)	BCD	Disabling injury
spearfishing	20	n/s	yes	4	n/s	asphyxia
recreation	n/s	surface	n/s	n/s	n/s	cardiac
recreation	n/s	surface	n/s	n/s	n/s	cardiac
recreation	n/s	surface	n/s	n/s	n/s	cardiac?
spearfishing	n/s	surface	n/s	n/s	n/s	asphyxia
recreation	3	surface	n/s	n/s	n/s	asphyxia
spearfishing	20	n/s	yes	n/s	n/s	unknown
recreation	n/s	surface	n/s	n/s	n/s	cardiac?
abalone	3–4	n/s	yes	12.25	n/s	asphyxia
crayfish	n/s	surface	n/s	n/s	n/s	cardiac
crabs	n/s	surface	yes	1.5	n/s	trauma
spearfishing	2.5	surface	n/s	n/s	n/s	trauma

BH 08/04

This 52-year-old male had a history of heart valve replacements in 1990 and 2006. A pacemaker was fitted in 2003 and he was currently taking warfarin and perindopril for hypertension. His wife later reported that his pacemaker was overdue for new batteries and that, two weeks earlier, he had seen a doctor as “*the top of his heart was not beating properly*”. She also stated that he had not taken his medications for one or two days prior to this incident. His previous snorkelling experience is unknown.

The victim, his wife and two friends had chartered a yacht and were on a sailing holiday on the GBR. He, his wife and one friend decided to snorkel from the boat while the other friend was on a nearby beach. There was no information about the sea conditions or what equipment he was using other than a mask, snorkel and wetsuit. After a short time, the victim swam ahead of the others and out of sight. After being in the water for about 15 minutes, his companions returned to the yacht some 50 metres distant. They could not see the victim but were soon alerted by the friend on the beach who pointed to the victim about 70 metres from the yacht. They eventually started the tender, picked up the friend on the beach and went to the victim who was found to be floating face-down, unconscious, apnoeic and cyanotic. He was dragged aboard the tender where CPR was commenced. It was maintained despite continual

regurgitation while they made their way to a nearby vessel where it was continued by two medical students. The emergency services were activated and a medical rescue crew, who were coincidentally on a nearby island, were brought to the vessel and implemented advanced life support (ALS). Although initially there was no shockable rhythm, after administering adrenaline and sodium bicarbonate the doctor delivered several defibrillation shocks unfortunately without success.

Autopsy: The pharynx and larynx contained a moderate amount and the trachea and bronchi a large amount of watery fluid. The lungs were heavy (Lt 597 g; Rt 604 g), with evidence of severe congestion and oedema. The CT scan revealed fluid in the maxillary sinuses. All these findings suggest drowning. The heart weighed 721 g with severe hypertrophy of the wall of the left ventricle and the septum. These both measured at least 25 mm in diameter (normal < 14 mm). In the anterior wall of the left ventricle there was an area of almost full-thickness scarring. The wall of the right ventricle was thicker than normal (5–6 mm, normal < 4 mm) and the ventricle was dilated. The left atrium was extremely dilated and thick-walled. A dual-chamber pacemaker had been inserted into the right side of the heart, with one electrode attached to the wall of the right atrium and the other to the wall of the right ventricle, near its tip. There was a prosthetic aortic valve, which appeared to have been functioning normally. The ostia of the coronary arteries

were easily identified and were widely patent. Dissection of coronary arteries showed only mild atherosclerosis. Toxicology showed warfarin 0.6 mg kg⁻¹. The cause of death was reported as drowning following cardiac arrhythmia due to valvular heart disease and cardiomegaly.

Comments: This death was likely a result of a cardiac arrhythmia in a man with significant pre-existing aortic valvular heart disease and significant enlargement of the heart. He had recent symptoms of arrhythmia, and was due to have his pacemaker battery replaced, so it is unsurprising that this would be exacerbated by the known cardiac triggers associated with snorkelling.

Summary: Cardiac valve replacement and pacemaker; recent arrhythmia; unknown general fitness and snorkelling experience; unknown conditions; silent death; cardiac related

BH 08/05

This victim was a 53-year-old, apparently healthy male who was not known to be taking any medications. He went to the beach with a group of friends who planned to go spearfishing. He had said that he had done so before when he was young but was not known to have had any recent experience. Two of the friends entered the water and began snorkelling. A short time later, the victim decided to go spearfishing alone as his other friend didn't want to go in. He entered the water wearing a t-shirt, long pants, sandals and mask and carrying the hand spear. He had no snorkel or fins. The sea was choppy and there was a two-metre swell crashing onto the nearby rocks. After about 10 minutes, he was seen to remove his mask and drop his spear, and appeared to be struggling to stay afloat. He was drifting in the current towards the rocks and was 'dog paddling'. At one point he raised his hands and called "Hey", but then collapsed face-down and drifted in the water. A bystander helped to drag the victim onto the rocks, assessed him to be unconscious and apnoeic and began CPR. He continued this with the help of one of the victim's friends until an ambulance arrived about 25 minutes later. The paramedics continued resuscitation attempts but soon pronounced the victim to be deceased.

Autopsy: The lungs were heavy (Lt 668 g; Rt 812 g) and overexpanded, with the anterior margins of the lungs overlapping on the anterior surface of the heart, and they appeared to pit on pressure, with a dough-like consistency. The trachea and main bronchi contained small amounts of light-brown-stained fluid. The heart weighted 458 g and the left ventricle showed marginal hypertrophy (15 mm). The coronary arteries showed minimal atherosclerotic narrowing. There were superficial abrasions on the face and fractures of the right anterior third to seventh ribs. However, based on the description of the incident, these injuries probably occurred during recovery and resuscitation. Toxicology was negative. The cause of death was given as drowning.

Comments: An inexperienced spear fisherman wearing street clothing and swimming alone without snorkel or fins in choppy seas with a large swell was 'a tragedy waiting to happen'. He was likely to have become exhausted, struggling in water-logged clothing; an ounce of common sense could have prevented this (alcohol-free) calamity.

Summary: Apparently healthy; inexperienced; spearfishing alone with others nearby; wearing street clothes and no fins or snorkel; rough seas and current; drowning

BH 08/06

This 47-year-old man was an overseas tourist. His medical history is unknown and he and his wife had never snorkelled before. During the boat trip to an island, a snorkelling brief was provided by means of a video in the victim's language but it was later reported that he and his wife paid little attention to this. They were also provided with a snorkelling safety brochure in their own language and advised (in their own language) where to swim in a patrolled area.

After hiring mask, snorkel and fins from the local dive store (and refraining from also taking a Lycra suit and snorkelling vest), they entered the water off a patrolled beach. The conditions were described as "average", with a wind of 20–25 knots and a choppy sea. There was a slight current and the visibility was poor, being less than one metre. After about 15 minutes and in water with a depth of around 3 msw, they decided it was too choppy and that they would return to shore with the wife swimming in front of the victim. At one point she heard him call out but, failing to realise that he was likely to have been in distress, she continued to shore, struggling against the current. She became concerned when he failed to arrive after 5–10 minutes and began to look for him in the general vicinity, without success. After a further 30 minutes she notified the lifeguard who initiated a missing person alert. The beach was closed and swimmers were recalled from the water. After a search of more than an hour, the victim's body was found approximately 200 metres from shore on the sandy seabed at a depth of 2–3 msw. He was recovered but CPR was not attempted because of the submersion time of more than an hour. A rescue helicopter and medical crew arrived at the island but, after discussion with the ground staff, did not land.

Autopsy: Details are unavailable at the time of publication as the case has not yet been closed.

Comments: Much is unknown about this case as the coronial documents are currently unavailable. However, it does appear to highlight the difficulty often faced by lifeguards in identifying a snorkeller in distress among other swimmers and in relatively poor conditions. It also serves to highlight the problems that can more easily occur after buddy separation. Given the reported conditions and the victim's reported lack of previous snorkelling experience, it may

have been wise for him to have refrained from snorkelling in such conditions, or at least worn a snorkelling vest. It is important that operators involved in such activities provide considered advice to those of unknown or limited experience.

Summary: Unknown medical history; no previous snorkelling experience; poor conditions; separation; body submerged more than an hour; case not yet closed; disabling agent/cause of death unknown

BH 08/07

This 21-year-old, male student was fit and healthy and an experienced spear fisherman who would often breath-hold dive to 20–25 msw. He went spearfishing with two friends from a small private boat wearing mask, snorkel, fins, full wetsuit with hood, weight belt and knife and carrying a speargun, all of which were his own. The victim and one friend, who had dived together many times before, were diving while the other friend remained on the boat. They had multiple, uneventful dives at a first location before moving to a second site, which was a beacon where the depth of water was 20 msw with a predominantly sandy bottom. This was one of their regular spearfishing sites. The sea was calm and visibility initially 6–8 metres although this later deteriorated. There was a slight current.

After several dives at this site, the victim's buddy suggested that they return to the boat as there were few fish. After agreeing to this and while the buddy was returning to the boat, the victim dived again and surfaced briefly before disappearing again underwater and failing to surface. The concerned buddy made several unsuccessful dives to try to find the victim in the poor visibility, but only retrieved the victim's float and spear, to which a large fish was attached. He gave up after about five minutes as he was exhausted and believed that it was by now too late to help his friend. The boat driver sent up a flare and called for assistance on his mobile phone and they were joined by several recreational vessels and a helicopter, which performed an unsuccessful surface search. The victim's body was found by police divers several hours later very close to where he had been diving.

Autopsy: The mouth contained frothy fluid and upper airways a small quantity of frothy mucus. The lungs were heavy (Rt 820 g; Lt 720 g) and slightly over-inflated. The cut surface of the lungs showed intense basal congestion and oedema, suggestive of drowning. The heart weighed 290 g and was of normal size for the body weight with no coronary artery disease and no obvious abnormality. Toxicology was negative. The cause of death was given as drowning.

Comments: The precise mechanism by which this young, healthy and experienced breath-hold diver died is unknown. It is likely that he became unconscious from apnoeic hypoxia after an extended breath-hold dive. There was no evidence provided that he practiced hyperventilation. There was no

report of the amount of weight he was wearing, but it is likely that he was negatively buoyant as his body failed to rise to the surface.

Summary: Healthy; experienced spear fisherman; others nearby; speared fish; failed to surface; likely apnoeic hypoxia; drowning

BH 08/08

This 25-year-old woman was an overseas tourist on a working holiday. She and a friend went on a day trip to snorkel on the GBR. There were 14 passengers and two crew on board the charter vessel. The skipper later reported that the victim said that she had snorkelled before in Thailand and Cairns but this was not confirmed. There is no other information about her swimming or snorkelling experience. When completing the pre-snorkel medical questionnaire, she told her friend (who was translating for her) that her "*heart sometimes beats quickly*". She also mentioned that after she had had a tooth extracted it would not stop bleeding. After initially marking "*Yes*" to the heart condition question, this was changed to "*No*" after the receptionist said that the question only applied to "*serious heart conditions*". She also changed her answer to a question relating to nose bleeds after advice from the receptionist.

The group went snorkelling at a site about 30 metres from where the vessel was anchored close to a beach. The victim was wearing a stinger suit. The sea conditions were not reported. The skipper remained on the boat as a secondary lookout while the supervisor was watching the group from a tender. After going to the vessel to replace a snorkel for one of the group, the supervisor returned to the snorkelling area and found the victim face-down and motionless, with the end of her snorkel submerged. She had been in the water for about 20 minutes. When she was pulled into the tender she was unconscious and apnoeic and froth was coming from her mouth and nose. The supervisor drove the tender to the main vessel and the skipper came aboard and began CPR. He was later assisted by other passengers. Efforts were complicated by regurgitation of stomach contents and water. After about 30 minutes medical staff from a nearby resort arrived by boat and continued resuscitation efforts. A defibrillator was attached indicating pulseless electrical activity, adrenaline was administered and spontaneous cardiac output eventually returned. When the medical team from the rescue helicopter arrived and intubated the victim, they noted that her vocal cords were oedematous and that there was "*pink frothy sputum in the endotracheal tube, consistent with pulmonary oedema*". She was then placed on a ventilator and transferred to hospital where she died later that night.

Autopsy: The upper airways contained a large amount of frothy fluid. The lungs were heavy (Lt 755 g; Rt 808 g) and showed severe pulmonary oedema. The heart weighed 265 g and appeared normal with normal coronary arteries.

The brain was mildly oedematous and showed hypoxic ischaemic encephalopathy on CT scan, consistent with drowning. There was a thyroidectomy scar on the neck and no thyroid gland was detected at autopsy. Toxicology showed only lignocaine. The cause of death was given as drowning.

Comments: It is unknown whether this victim showed any signs of distress before becoming unconscious, as the primary lookout was temporarily elsewhere. Likely diagnoses include drowning and a cardiac dysrhythmia with immersion pulmonary oedema a remote possibility. Whilst there was a history of palpitations, ECG and cardiac echocardiogram performed in hospital after resuscitation were reported as normal and there was no other evidence to suggest a cardiac cause for a loss of consciousness. Although the rescue efforts of the dive crew and various medical crews were gallant, the delay in the initial recognition of a problem made ultimate success unlikely.

Summary: Possible history of tachycardia; some snorkelling; amongst group; silent unconsciousness; CPR successful; likely drowning/cardiac event

BH 08/09

This victim was an apparently fit 42-year-old male, an experienced snorkeller who often dived for abalone. He had told a friend that he had once gone to the doctor for an asthma attack but there was no report of the frequency or severity of attacks or if he had been taking medication for asthma. He went with a friend to a familiar beach to look for abalone. The sea conditions were calm and clear with no swell and only a small wind chop. The victim wore a mask, snorkel and fins, and a two-piece wetsuit and weight belt with 12.25 kg of weights. He had a knife strapped to his leg and carried a catch bag.

The victim entered the water alone. His friend remained on shore and tried to keep a lookout from their car parked on a cliff above. The victim snorkelled for about an hour before the friend lost sight of him. He became worried and mentioned this to an acquaintance who arrived and who then offered to conduct a search. The acquaintance entered the water approximately 25 minutes after the diver was last seen and, after searching for about 20 minutes, found the victim sitting on the seabed at a depth of 3–4 msw and about 30 metres from shore. He was unconscious and apnoeic, and still wearing his mask and weight belt, but the snorkel was out of his mouth, which was closed. He was not entangled. The rescuer dragged the victim to the surface and managed to support him there, despite being unable to remove the weight belt. He towed the victim to shore, where he found no palpable pulse. His hands were “*curled up and stiff*” and the rescuer decided that the victim was dead and did not attempt CPR. When ambulance officers arrived they pronounced the victim to be deceased. The victim’s weight belt had been threaded through under part of the wetsuit and was therefore difficult to remove. His knife was missing.

Autopsy: The autopsy was performed six days after death and the body showed decompositional changes. There was no fluid in the upper airways. The lungs together weighed 1095 g (normal range: 600–1000 g) and were congested. There was 50 ml of red putrefactive effusion in both pleural cavities. Decomposition can obscure some of the changes of salt water drowning. The heart weighed 363 g (normal range: 150–400 grams) and was structurally normal with no scarring or abnormalities identified on sectioning. The coronary arteries were unremarkable except for the distal portion of the LAD coronary artery, which demonstrated 30% narrowing from atherosclerosis. Coronary artery ostia were normal. Toxicology showed alcohol 0.025 g 100 ml⁻¹. The cause of death was given as drowning.

Comments: The victim was an experienced snorkeller so it is reasonable to assume that the threading of the weight belt under part of his suit was likely to have been intentional rather than accidental. Although it is unknown what triggered this accident, if the victim did attempt to access or remain on the surface in a distressed state, it would have been extremely difficult for him to release his weight belt to attain positive buoyancy. Any diver wearing weights must ensure that they can be readily and easily discarded if necessary. It is obvious that the distant lookout on land was ineffective.

Summary: Experienced; snorkelling alone with distant lookout on land; disappeared from sight; found unconscious underwater; weight belt threaded under suit; drowning

BH 08/10

This 46-year-old male had a medical history that included osteoarthritis (knees), sinusitis, gout, gastro-oesophageal reflux, hyperlipidaemia, cervical spinal disease requiring surgical fusion and disc arthroplasty. He also had anxiety neurosis for which he was receiving counselling and was taking mirtazapine. His family reported that he was a strong swimmer and a competent snorkeller who became a certified open-water diver 11 years earlier, although he had not snorkelled or scuba dived for the previous three years.

On this day, he went snorkelling in temperate waters with three other family members off a popular island with the intent to put out a crayfish pot. The weather was described as hot and very windy, with choppy seas, a slight current and poor underwater visibility. He was wearing mask, snorkel, fins and a short wetsuit. The group took it in turns supporting the pot while they swam out against the current. The victim duck-dived several times looking for a place to drop the pot, which was eventually dropped 80 metres from shore. However, while on the way, the victim indicated to one of the others that he would return to shore. Another family member on the beach saw the victim leaving the others and snorkelling towards shore; however, several minutes later she noticed that he was motionless about 30 metres from shore. She waded out and found him to be unconscious and apnoeic and began giving him some rescue breaths. With help, she

dragged him to shore and commenced CPR. Sometime later, a doctor and two nurses from the island arrived and took over resuscitation efforts but resuscitation was abandoned after the arrival of the local Flying Doctor medical staff.

Autopsy: The right and left lungs weighed 627 g and 598 g respectively and both lungs appeared hyper-expanded, protruding from the chest cavity on removal of the breast plate. The cut surfaces showed diffuse, patchy purple-coloured congestion, with free-flowing watery fluid. There was frothy mucus in the trachea. The heart weighed 394 g and appeared normal. There was a narrowing of at least 75% in the LAD coronary artery. There was no obvious scarring of the myocardium. Toxicology showed mirtazapine 0.2 mg L⁻¹. The cause of death was given as drowning with coronary atherosclerosis as a contributing factor.

Comments: It is likely that the exertion involved in dragging the cray pot triggered a cardiac event causing the victim to become unconscious and subsequently drown.

Summary: History of osteoarthritis, cervical spinal surgery, gout, GORD, hyperlipidaemia and anxiety; experienced snorkeller; choppy seas and dragging cray pot with others against slight current; abandoned group to return to shore prematurely; found unconscious close to shore; CPR unsuccessful; drowning (likely cardiac-related)

BH 08/11

This victim was a 51-year-old male who had over 30 years of snorkelling experience. His medical history was unreported. He went snorkelling with his son to look for crabs, wearing mask, snorkel, fins, sleeveless short wetsuit and weight belt with 1.5 kg. The conditions were calm with a slight swell. They swam out to about 50 metres from shore and then swam along the weed line. The visibility was about eight metres. After swimming about 300–500 metres and seeing very few crabs, they decided to turn back. The victim was swimming close behind his son when the son heard his father call out. When he turned around, he noticed a very large dark shape swimming rapidly underwater towards his father. He then saw his father pulled underwater. Witnesses on shore noticed a large dorsal fin, estimated to be one metre, and thrashing in the water and heard the victim's son's calls for help. Sometime later the crew of a rescue helicopter saw a diver's float and wetsuit top floating on the surface, which were identified to have belonged to the victim. The wetsuit was torn, consistent with bite marks from a large shark. Another helicopter filmed a large shark in the vicinity and another piece of the wetsuit was later found. A shark expert concluded from the bite marks and footage of the shark filmed that the attacking shark was a 4 to 4.5 metre great white shark (*Carcharodon carcharias*).

Autopsy: Body not recovered.

Comments: This diver was the unfortunate victim of a shark

attack. It was not reported whether or not the victim was carrying any crabs. If so, this could have been a precipitant to the attack. There was some fishing activity in the vicinity, another potential precipitant.

Summary: Experienced; looking for crabs; attacked by large shark; parts of wetsuit recovered but not body; presumed death by trauma

BH 08/12

This 25-year-old male was an experienced spear fisherman who went spearfishing with a friend from the shore in a marine park well known for its abundant marine life. The weather was sunny, there was little wind and the sea was calm and clear. The victim wore a black mask, snorkel and fins; army camouflage-coloured wetsuit with a black hood; and black and white gloves. He carried a speargun and Shark Shield. He and his buddy snorkelled for a while approximately 30–50 metres from shore in water about 3 msw deep. They were not using a float with a 'Diver Below' flag. The site is covered with reef and coral bommies interspersed on a sandy bottom.

After about 60–75 minutes, the buddy tired and returned to the beach, while the victim continued to spearfish. Shortly afterwards, a high-powered 8.5 metre tourist boat drove past at high speed and collided with the victim. Some of the passengers saw the victim surface briefly just in front of the boat and then heard thuds from an impact. The boat driver had not noticed the diver as he was driving from a rear console, the 12 passengers were seated in front of him and there was no designated lookout to warn him. After impact, passengers started yelling at the driver but he could not hear them as he was wearing ear-muffs to protect his ears from the engine noise and wind. However, he eventually stopped when he saw passengers waving to him to do so and they directed him back to where there was blood in the water and a mask, fins and speargun floating on the surface. They could see a dark shape below but abandoned attempts to retrieve the victim owing to concern about sharks. The depth of water was approximately 2.5 msw. The friend on shore heard the boat rush past and then saw it turn back and circle. He swam to the boat and was taken to the site where he could see the victim's body on the bottom, motionless and bleeding. However, he was unable to recover his friend, which was later done by crew from another vessel.

The coronial investigation concluded that the vessel was likely travelling in excess of 30 knots approximately 45 metres from shore in water which was a little deeper than three metres. Although this was believed to be unsafe, there was no regulation prohibiting this. The coroner made several recommendations which included imposing an 8 knot speed limit within 150 m of the low tide mark in that area.

Autopsy: There was head injury with large deep incised wounds in the left forehead and right face with skull

Table 2

Summary of scuba and surface-supply diving-related
BSB – buddy separated before problem; BSD – buddy separated during problem; GSB – group separated before problem;

ID	Age (yr)	Sex	Height (m)	Weight (kg)	BMI (kg m ⁻²)	Training	Experience	Dive group
SC08/01	70	M	177	76.5	24.4	trained	experienced	BSD
SC08/02	41	M	179	98	30.6	trained	some	BSB
SC08/03	48	M	184	119	35.1	trained	some	GSB
SC08/04	52	M	188	107	30.3	trained	n/s	GNS
SC08/05	66	M	170	76.6	26.5	trained	experienced	solo
SC08/06	35	M	170	91	31.5	trained	inexperienced	BSD
SSBA 08/01	30	M	178	78	24.6	trained	experienced	solo

fractures and laceration of the right frontal, temporal and parietal lobes of the brain. There was traumatic amputation of the right upper arm at the mid-humerus, and deep incised wounds on the right shoulder and on the right knee. The heart weighed 266 g and was normal apart from a 30–40% narrowing of the LAD coronary artery. The upper airways contained a small amount of blood-stained frothy fluid. The right and left lungs weighed 442 g and 388 g respectively and were mildly congested. Toxicology revealed traces of mirtazepine, cannabis and alcohol. The cause of death was given as head injury.

Comments: The injuries are typical for a diver struck by a boat propeller. Previously in this State, the use of a dive flag when snorkelling was mandatory but this regulation was repealed several years earlier and the use of the flag became optional but generally encouraged. Had the victim been towing a float and dive flag it would have increased the likelihood of his being seen. However, this would have been no guarantee under these particular circumstances where the boat was driven so fast, from the rear and without a proper lookout. The boat operator was lucky to have avoided prosecution. Despite a regulation that a vessel should not travel at a speed greater than 8 knots in water less than 3 m deep, he was afforded the benefit of doubt of water depth at that time, and whether he had exceeded the speed for which his vessel was licensed. The ‘culpability’ of the boat driver was owing to a combination of speed and not having a lookout, especially given the location of the driving controls.

Summary: Experienced; not displaying dive flag; hit by high-speed, stern-driven boat with inadequate lookout; trauma

Scuba diving fatalities (Table 2)

SC 08/01

This 70-year-old male had a history of ischaemic heart disease, stroke, mitral valve surgery (10 years prior), hypertension and hyperlipidaemia. He was a non-smoker and walked and cycled regularly to keep fit. He was on a variety of medications, including candesartan, cilexetil, atorvastatin calcium, clopidogrel, celecoxib and finasteride. He was an experienced diver who owned his own equipment and dived regularly, reportedly having done over 100 dives. His buddy reported that, two months earlier, the victim became very breathless when walking back from a dive wearing his equipment, this apparently being the first time this had occurred in the six years they had dived together.

They dived from shore near the entrance to a bay in relatively poor conditions, although nothing unusual for them. The weather was overcast and raining with a 15–20 knot wind, a surface chop, a one-metre swell and strong current. They dived to a maximum depth of around 24 msw, swimming with the current with visibility of 10–15 m. When they surfaced after 67 minutes to get their bearings, they were in the open ocean about 50–60 metres from the bay’s entrance and being pushed further away by the swell and current. The buddy encouraged the victim to swim towards shore but the victim said to his buddy something to the effect of ‘we have had it here’. Look, the swell has got us and we can’t swim back to the bay’. He then started to unzip his wetsuit top as he was breathing heavily. He did not respond to the buddy and did not swim. The buddy exited the water some 600 m from where they surfaced, ditched his equipment and re-entered the water to assist the victim who was now about

Table 2 (cont.)

fatalities in Australian water in 2008

+ sufficient air (to surface safely); ++ 1/4–1/2 full tank; +++ >50% full; nad – nothing abnormal discovered;

n/i – not inflated; n/s – not stated; CAGE – cerebral arterial gas embolism

Dive purpose	Depth (msw)	Incident (msw)	Weight belt	Wts (kg)	BCD	Gas supply	Equip test	Disabling injury
recreation	24	surface	off	n/s	inflated	n/s	n/s	cardiac
hunting crayfish	11	surface	on	10.9	n/i	+	nad	cardiac?
recreation	34	n/s	n/s	17	inflated	nil	nad	CAGE
recreation	2	surface	n/s	n/s	inflated	n/s	n/s	cardiac
commercial	3	1	on	9.7	nil	++	faults	asphyxia
crayfish	10.7	surface	off	12	n/i	++	nad	asphyxia? CAGE?
collecting for aquarium	8.1	8.1	on	n/s	nil	+++	nad	cardiac? asphyxia? epilepsy?

30 m from the rocks. On arrival he found the victim floating on his back supported by his BCD, unconscious, apnoeic with foam coming from his mouth. Despite difficulty in the sea conditions, he began rescue breathing, without a response. Unable to drag the victim to shore, the buddy again exited the water and went for help, believing his friend to be dead. A rescue helicopter arrived around 90 minutes later, found the victim to be unconscious and cyanotic and floating on his back without mask and weight belt. The victim was retrieved and brought to shore where attending paramedics did not attempt resuscitation.

Autopsy: Erect and supine x-rays showed no obvious gas/fluid levels. The heart was heavy (548 g; 100 g heavier than one would expect) with left ventricular hypertrophy (16 mm, normal < 14 mm). There was evidence of previous surgery on the mitral valve, which was thickened, and there was calcification of the mitral valve ring and aortic valve. There was near-complete occlusion of the LAD coronary arteries, up to 75% occlusion of the right and left circumflex coronary artery, and scarring of the left ventricular wall on histology. Both lungs appeared hyperinflated and heavy (Lt 662 g; Rt 856 g) and showed prominent oedema fluid on the cut surfaces. The trachea and larger airways showed copious amounts of frothy fluid. Toxicology showed 3% carboxyhaemoglobin and traces of hydrochlorothiazide. The cause of death was given as drowning due to valvular and ischaemic heart disease.

Comments: Although his buddy reported that the victim became breathless on exertion, this was not reported to the victim’s cardiologist at his last review. If breathlessness on exertion had been reported, investigated and confirmed, it is likely he would have been advised not to dive, and this

fatality averted. Loss of consciousness in the water often results in drowning. Distress leading to unconsciousness is a clear risk, especially in such physically strenuous and adverse circumstances as those encountered on the day of the dive.

Summary: Experienced; history of mitral valve repair, stroke, hypertension, hyperlipidaemia; history of breathlessness with exertion; surfaced in rough seas and strong current; breathless and unable to swim to shore; buddy attempted rescue breathing; drowning (cardiac-related)

SC 08/02

This 41-year-old male was described as energetic and fit with no known adverse health conditions, despite being obese (BMI 30.6 kg m⁻²). He was a non-smoker and only a light drinker who had passed a diving medical examination eight months earlier and had been certified as an open-water diver for seven months. He had completed over 20 dives, always with the same buddy, and was described as a careful diver who owned his own equipment, reported to be in good working order.

On this occasion, the victim and his buddy dived for crayfish from a 5.2 m boat anchored in a channel off a small island. Conditions were described as good, with a 10-knot wind, minimal chop and a 1 m swell. The depth at the site was around 11 msw, water temperature was 19°C, underwater visibility was about 4 m and there was a slight surface current. The victim was wearing a wetsuit and BCD with 8.1 kg of integrated weights, as well as an additional 2.8 kg in the pockets. He and his usual buddy dived while another friend remained on the boat fishing.

At the start of the dive the buddy's contents gauge read 240 bar, but it is unknown how much air was in the victim's cylinder. The plan was to surface when their air reached 120 bar, as was their usual practice. After swimming towards the reef, the pair reportedly turned back when their gauges read 120 bar, and swam along to the edge of the reef towards the boat before surfacing 20–50 m from the boat. At this point, the victim said that he would swim to the boat on the surface as he was low on air. The buddy descended to look at some rocks and return to the boat underwater. The friend on the boat then saw the victim swimming strongly towards him and he called out that he was out of air. By the time the friend had organised enough rope to throw to the victim, he was now on his back and finning but not making headway and then did not respond to his calls. The friend jumped into the water to assist but, before he reached him, the victim sank and despite several duck-dives the friend could not see him.

The buddy surfaced some time later and learned of the problem. He was almost out of air but had a quick and unsuccessful look underwater before re-surfacing and changing his tank. He dived again but failed to locate his buddy, finding only his 'cray loop'. In response to an emergency call made from another vessel, a police diver arrived about 30 minutes later and located the victim's body 20 m from where he was last seen. His mask was full of blood and water, his regulator was out of his mouth, his face was cyanotic, his BCD was not inflated and no weights had been ditched. His contents gauge read 10 bar and both second stage regulators purged effectively. His computer displayed a maximum depth of 11.3 msw and a dive time of 98 minutes. When tested by the police, his equipment was found to be in good working order.

Autopsy: Watery pink-coloured fluid readily extruded from the nostrils and there was frothy fluid in the trachea. The right and left lungs were over-expanded and heavy (Rt 934 g; Lt 916 g). The heart weighed 518 g (slightly heavy) and there was up to 20% narrowing of the coronary arteries by atheroma. Microscopic examination of one of the coronary arteries showed focal atherosclerotic narrowing (degree not specified). Toxicology was negative. The cause of death was given as drowning.

Comments: This diver failed to make himself positively buoyant on the surface, which he could have done by inflating his BCD or ditching some weights. Even if he had become unconscious, his friend on the boat could have retrieved him quickly and given him a better chance of survival. He was also not wearing a snorkel, with which he could have managed better on the surface if he was tired. Additionally, had the diver and his buddy returned to the boat close together, the buddy may well have been able to assist him and increase the likelihood of survival. Normally a 20% narrowing of the coronary arteries would not be regarded as significant. The listing of focal coronary atherosclerosis as a contributing factor in the cause of death in the final autopsy report suggests that the narrowing seen by later histology

may have been greater than 20%.

Summary: Experienced; intentionally separated from buddy; low on air; no snorkel; became unconscious whilst swimming on surface to boat; drowning (cardiac related?)

SC 08/03

This diver was a 48-year-old, severely obese (BMI 35.1 kg m⁻²) male. He had passed a diving medical examination a year earlier (six months after his initial certification) and had stopped smoking a year before that. He had recently lost 8 kg and was not known to be under medical treatment. Said to be a strong swimmer, he had gained his open-water scuba certification 19 months earlier and had subsequently done additional training courses including night, navigation, nitrox, deep and wreck diver certificates. He had also participated in a drysuit diving course a week or two earlier and had recently bought a new drysuit. He was not certified for this course as he failed to provide the required dive medical. His logbook indicated that he had done a total of 34 dives, with a lifetime maximum depth of 30 msw. Most of these dives were done in local temperate waters. On several of these dives he indicated that he had "*blown the ascent*" for various reasons generally related to poor buoyancy control. On one occasion he had surfaced without any remaining air and was not impressed when the boat operator pointed this out to him. He was described as a very enthusiastic diver who used to work on and configure his own equipment.

He was participating on a boat dive with six other divers on a wreck lying at a depth of 34 msw. Weather conditions were reported good, a slight swell, not too choppy, with underwater visibility of 5–10 m and a slight surface current, but none on the bottom. The victim was wearing a drysuit and a home-made horse-collar-type vest with twin 9l cu ft cylinders joined by a manifold which allowed a diver to switch between cylinders as the air in one became low, but did not enable decanting between the cylinders. The equipment was described by others as poorly configured, overly complicated, and very heavy, weighing 56 kg, including 8 kg of removable weights.

On the boat before the dive, the victim was noticed to be unusually quiet, looked pale and appeared unusually clumsy. He was buddied with two other divers. When he entered the water, other divers noted that he appeared to be heavy, floating very low, despite his BCD and drysuit being inflated. He initially had trouble dumping air to descend and managed to do so when one of his buddies helped him to find an inflator hose. After seeing him descending with his right side down in what was described as "*a bizarre manoeuvre*", the other two divers lost sight of the victim. A short time after reaching the wreck they noticed he was missing and backtracked to see if they could find him, without success. They then assumed he had joined another group and so continued diving.

After “*about 10 minutes*” others on the boat saw the victim break surface feet first, preceded by a burst of bubbles, and roll face-down in the water, seemingly unconscious. His BCD and drysuit were both inflated and the BCD was floating next to him, attached only by the inflator hose. An off-duty dive instructor who was on the boat jumped into the water, grabbed the victim and, with the help of others, brought him aboard. His mask was full of froth, which was also coming from his mouth. He was apnoeic, cyanotic and had no palpable pulse. The instructor and a friend began two-person CPR and continued while the boat sped towards shore, a 20-minute trip. Oxygen was available and ready but was not used as they believed they could not manage it in the circumstances. Within a few minutes of arrival at the jetty, paramedics commenced ALS. The victim was found to be asystolic and was intubated; IV access was gained and adrenaline was administered. ALS was discontinued after 30 minutes.

When tested, most of the equipment appeared to be functional, except that one of the three demand valves had been incorrectly assembled (with the diaphragm poorly seated) so that it would have delivered water, rather than air, if used. The equipment configuration was described as inappropriate and overly complicated. No air remained in either cylinder. The dive computer indicated that the victim had descended to a maximum depth of 32 msw over 1.5 minutes, and remained at that depth for a further 11 minutes before ascending rapidly to the surface over one minute.

Autopsy: An examination of the pre-autopsy CT scan imaging showed extensive dissemination of gas within the vascular system predominantly on the arterial side. There was marked gas formation within the cerebral arteries and within both cardiac chambers. At autopsy, gas bubbles were noted in the vertebral and basilar arteries and circle of Willis. Subcutaneous emphysema was not described. The heart appeared moderately enlarged (476 g) and the left ventricular myocardium uniformly measured 20 mm in thickness. The LAD coronary artery was focally 80–90% occluded by atherosclerosis. There was right renal artery stenosis with a hypoplastic right kidney (Rt kidney 46 g; Lt kidney 257 g). The trachea contained a small amount of blood-stained fluid. The lungs were well inflated and the right and left lungs weighed 698 g and 920 g respectively. Toxicology was negative. The cause of death was given as cerebral artery gas embolism (CAGE) in a man with significant single-vessel ischaemic heart disease.

Comments: This is a well-documented case of CAGE. It appears that the victim tried to ditch his equipment as it was found unclipped and was attached only by an inflator hose. This is likely in response to an out-of-air emergency, given that his cylinders were empty on retrieval. Some witnesses reported that the victim had appeared unwell prior to the dive and, given the autopsy finding of severe coronary atherosclerotic disease, it is possible that he suffered a cardiac event prior to or during the dive. The combination

of his obesity, inexperience and inappropriate equipment configuration was a recipe for disaster.

Despite reports from friends and the police that he was a capable and experienced diver, this victim had logged only 34 previous dives to a maximum depth of 30 msw. It is unusual for a diver of this level of experience to adopt a twin-cylinder configuration with all the associated complications of multiple regulators, hoses and clips. Such configurations are usually part of specialist training for advanced diving techniques. He seems to have been considerably over-weighted with equipment and this would have caused buoyancy and swimming problems during the dive, and would be expected to substantially increase gas consumption. Given his inexperience, especially at depth, the victim would have likely been suffering from nitrogen narcosis, which could have created or compounded problems that arose. The feet-first ascent likely occurred as a result of the equipment weighing him down, and the air in his drysuit rising to his feet, causing him to invert prior to or during the ascent.

Summary: Morbidly obese but no other known medical history; 90% occlusion of LAD coronary artery; trained; some experience; complicated and very heavy gear configuration; separated from buddies on descent; uncontrolled buoyant and out-of-air ascent to surface; CAGE

SC 08/04

The victim was a 52-year-old, obese (BMI 30.3 kg m⁻²) male with a long-standing history of hypertension, which was reportedly difficult to control and for which he was prescribed amlodipine. He had also had an abnormal glucose tolerance test 10 months earlier. He was certified as an open water diver 15 years earlier but his diving experience is unknown.

He was in a party of nine recreational divers, including an instructor, on a club shore dive. The group walked across rocks to enter the water, which was described as a little choppy. Shortly after entering the water, the victim was floating in water about 2 msw deep and about 20–30 m from the shore. His BCD was inflated but his fins were not on and the water was just too deep for him to stand. He appeared to be panicking although he told the instructor he was ‘OK’. The instructor descended to put on the victim’s fins, but when she surfaced after about 30 seconds, she found him slumped back, unconscious, grey and apnoeic. After giving two rescue breaths, she towed the victim to shore and commenced CPR, which she continued until relieved by paramedics about 45 minutes later. The victim was transferred to hospital, where he was found to be in asystolic cardiac arrest and failed to respond to further ALS.

Autopsy: The heart weighed 512 g (normal range: 142–451 g) and appeared dilated and “*floppy*”, but of normal configuration. The internal diameter of the right and left

ventricles measured 65 mm and 50 mm respectively. The right ventricular wall measured approximately 7–8 mm in thickness and the left ventricular wall was approximately 15 mm thick. The mid-portion of the LAD coronary artery showed up to 50% stenosis. The lungs were heavy (Rt 976 g; Lt 712 g). There was diffuse congestion and pulmonary oedema and the mouth contained some blood-stained fluid. Toxicology showed amlodipine 0.09 mg L⁻¹ and traces of atropine. The cause of death was given as atherosclerotic and hypertensive heart disease.

Comments: This death likely resulted from a cardiac dysrhythmia secondary to dilatational cardiomyopathy. Although 50% stenosis is not usually regarded as sufficient to cause sudden death, one author (CL) has seen cases where stenosis of less than 75% appeared to have been responsible for sudden death. It is likely that this cardiac event could have occurred in a variety of non-diving-related scenarios, although the physical demands of diving, combined with anxiety can readily trigger an event in a diver so predisposed. The efforts of the instructor were commendable.

Summary: History of poorly control hypertension and abnormal glucose tolerance test; qualified but with unknown experience; became unconscious on surface on entry and had not descended; CPR commenced rapidly and maintained until ambulance arrived; cardiac death

SC 08/05

The victim in this incident was a 66-year-old man who was a highly experienced diver who had dived regularly over the previous 40 years. He was in good health, other than for some arthritis, a non-smoker and social drinker. He was not taking any medications. He agreed to do a dive to free a rope tangled around the propeller of a yacht in a mooring area in a tidal river with a fast-flowing current. He was familiar with the dive site and was experienced in fixing moorings in the area. The weather was fine with a light to medium breeze; however, there was a 5–6 knot current. He was wearing mask, snorkel, fins, wetsuit, a weight belt with 9.7 kg and scuba gear. He was not wearing a BCD and did not appear to have a dive knife, although he was carrying a hacksaw.

The yacht owner towed the diver near to the bow of the yacht from where he let go of the rope and drifted underwater towards the stern, where the yacht was tangled in mooring lines. The water depth was 1–3 msw. By the time the owner had tethered the dinghy and walked to the stern, he saw the victim about a metre below the surface, motionless, with his regulator out of his mouth and being held under by mooring ropes. After jumping into the water to try unsuccessfully to free the victim, the owner climbed back aboard his yacht and called for assistance. A policeman arrived, entered the water and was eventually able to duck-dive down and cut the victim free from the two or three ropes in which he was entangled. He dragged the diver to shore where an ambulance officer attempted resuscitation, without success.

When the equipment was tested it was noted that the mouthpiece had been torn from the second stage and could not be purged. There was 80 bar of air remaining in the cylinder. When tested, the air contained high levels of water vapour and the carbon dioxide concentration was 120% of the allowable limit.

Autopsy: Careful subcutaneous dissection with the chest in the highest position revealed no obvious gas in the heart. The lungs appeared over-expanded, consistent with the diagnosis of drowning. The right and left lungs weighed 808 g and 624 g respectively and showed severe pulmonary oedema. The upper airways contained blood-stained fluid. The heart weighed 456 g (normal range 262–560 g) and there was a 50% stenosis of the LAD coronary artery. Toxicology showed alcohol 0.024 g 100 ml⁻¹. The cause of death was given as drowning due to entrapment by mooring rope and tidal flow and loss of facemask and regulator mouthpiece while scuba diving.

Comments: Diving in an area with a strong current and entanglement hazards is a recipe for potential problems. It is likely that the diver was pushed into the ropes by the strong current and wedged under them. His regulator appears to have been torn from his mouth and, if he was able to replace it, he would have been unable to use the purge button to clear it. Without a nearby buddy or surface watch with an alternative air supply, drowning was inevitable. His old-style weight belt would have been difficult to release quickly, although this is unlikely to have been a factor here as he was firmly trapped by the ropes. Although his air supply did not meet purity standards, it is unlikely that this was a factor in his demise. Given that he was carrying a hacksaw, it is reasonable to conclude that even had he been carrying a dive knife, he would not have been able to cut himself free in time.

Summary: Healthy; experienced; diving alone; strong current; entangled in mooring ropes; demand valve displaced and purge faulty; drowning

SC 08/06

This diver, a 35-year-old male, was obese (BMI 31.5 kg m⁻²) but otherwise reportedly fit and healthy. He had been certified as an open-water diver about six weeks earlier and had done a total of six dives, including his four shallow training dives. On this day, he and his buddy, another novice with whom he had done all of his diving, planned to dive for crayfish from the shore of a small island. They had been warned previously by their instructor that this site was subject to swell and strong surge. They were taken to the island by another friend with a boat, who was going to fish elsewhere and then return to pick up the divers later in the day. They brought three cylinders each as they were planning to do three dives. The weather was described as mild to warm with a light breeze, a small swell and slight surface chop. Visibility was 5–6 m and there was a strong current. The victim had recently bought new dive equipment.

In addition to mask, snorkel and open-heel fins (which he had borrowed), he was wearing a full wetsuit with hood but no boots, a BCD with tank and regulator (with 'octopus'), and a weight belt (possibly with 12 kg of weights, which is what he used during training).

He and his buddy entered the water and swam underwater away from shore. After a while, they checked each other's air and decided to return towards shore. While the buddy began to swim away from the island and work his way towards the surface slowly, he saw the victim ascend directly and rapidly. They were separated for 2 to 3 minutes and when the buddy surfaced, he could see the victim about 25 metres away being pushed toward rocks by the current and surge. He wasn't wearing his mask or snorkel and did not have his regulator in his mouth. The victim tried to climb onto some rocks but kept slipping and getting thrown around by the surge and smashed against the rocks. After calling out his buddy's name, the victim became quiet and floated face-down. The buddy swam to him, turned him over and tried to drag him out of the water but had trouble as the victim kept sinking. After inflating the victim's BCD, the buddy managed to drag him away from the rocks. He was unconscious and apnoeic, with froth coming from his mouth. The buddy then saw a nearby boat and called for help. The divers were brought aboard and the buddy commenced CPR, assisted by one of the men on the boat. This was continued en route to the jetty where an ambulance was awaiting their arrival. The paramedics found the victim to be cyanotic and asystolic and he failed to respond to ALS.

When later checked, his equipment was found to be in good working order and there was 50 bar of air remaining in his tank. His dive computer indicated that he had been to a maximum depth of 10.7 msw (average depth 7.6 msw) for a dive time of 25 minutes. His mask, snorkel, fins, weight belt, knife and cray noose were missing.

Autopsy: At the scene, the facemask had been lost and there was pulmonary oedema fluid coming from the mouth, features suggestive of drowning. A radiograph, taken approximately six hours post mortem, showed gas in the thoracic aorta extending into the carotid arteries. Gas collections were observed in the vascular system (predominantly arterial) and tissues. No other evidence of barotrauma was detected. The heart weighed 362 g and was normal with only mild coronary atherosclerosis. The right and left lungs weighed 656 g and 694 g respectively. The lungs were well inflated; there was no pulmonary oedema fluid in the upper airway (the pathologist noted that resuscitation had been attempted). The gas detected on X-ray was attributed to decompression, possibly post-mortem decompression. Toxicology showed traces of cannabis and 2% carboxyhaemoglobin. The cause of death was given as drowning, based particularly on the scene findings.

Comments: This inexperienced diver appeared to ascend too closely to rocks where there was a strong surge and current.

The interpretation of the gas seen at autopsy is difficult. It could represent:

- CAGE/pulmonary barotrauma due to a rapid ascent in an inexperienced diver low on air, or caught in an upward surge;
- post-mortem off-gassing;
- gas due to vigorous cardiopulmonary resuscitation.

The pathologist amongst the authors (CL) favours drowning due to strong surge and loss of facemask as a cause of death, mainly because of the plume of pulmonary oedema and the loss of the mask. He believes the gas seen on X-ray was likely due to CAGE or resuscitation.

Summary: Obese but otherwise healthy; recently trained and inexperienced; rapid ascent; smashed against rocks by strong current and surge; drowning

Surface-supply fatality

SS 08/01

This 30-year-old male was described as fit and strong. He had a history of epilepsy which began in his teens, however, although he had apparently been seizure- and medication-free for the previous five years. He had been certified as an occupational diver and had several years' experience collecting aquarium fish for sale. He was certified fit to dive by an experienced diving medical examiner 10 months earlier, but he had failed to disclose his history of epilepsy.

Together with his brother and another companion, the victim dived from his 12.5 m boat. His equipment included mask, fins, two wetsuits, a weight belt ("*with three or four weights*"), gloves and a dive computer. He did not wear a BCD or carry his bail-out cylinder (the regulator was missing). They dived using the victim's surface-supply breathing apparatus (SSBA). The victim did not wear a harness to which to attach the air hose. Instead, he secured the hose by passing it under his weight belt and through his legs. His first dive was to a maximum depth of 24.1 msw for 192 minutes, with all decompression indicated by the dive computer being completed. Although they had all dived separately, the victim and his brother completed their decompression/safety stop together. After a surface interval of 70 minutes, the three dived again. The victim's computer indicated a maximum depth of 17.3 msw for a total dive time of 120 minutes. The computer log showed that he had ascended faster than 9 m min⁻¹ at some stage, but there was no decompression requirement.

After that dive, the group moved the boat to another site and set anchor for the night. They drank a can of beer and the victim decided to dive again, which he did after a surface interval of 76 minutes. His brother watched him enter the water alone. The others then showered and logged their catch for the day. However, after about an hour, they realised that they had not noticed the air compressor activate for a while

and, on checking, confirmed that no-one was breathing from it. After tugging on the hose without a response from the victim, the companions began to haul in his hose, which was extended out to almost its full length of 100 m. Initially the hose appeared to be snagged, but after tugging hard it came free and the victim was hauled to the surface, backwards and upwards at an angle from depth, over about a minute. They dragged him aboard and found he was unresponsive, apnoeic and cyanotic and there was water, mucus and blood in his mask and froth and water coming from his mouth. Other than one missing fin, his equipment was in place, although they did not notice whether or not the regulator was in his mouth. They cut off the victim's wetsuit and began CPR, which they continued for over 2.5 hours. Although there was oxygen delivery equipment on board it was not used (the cylinder was missing anyway). They were relieved by a doctor from another vessel who, after peer consultation, declared the victim to be dead.

When tested, the compressor and regulator were functional and the air supply was free from contamination. The regulator mouthpiece was torn. The victim's dive computer indicated that the fatal dive was to a maximum depth of 8.1 msw for a total of three minutes.

Autopsy: The heart weighed 390 g and appeared normal. There was a focal 60% atheromatous stenosis at the junction of the left main and LAD coronary arteries with some inflammation in the plaque. Foam was noted in the mouth and the larynx, trachea and bronchi contained pulmonary oedema fluid. The right and left lungs weighed 910 g and 750 g, respectively. The lungs on section showed oedema and peripheral displacement of air. Overall, these lungs showed the features of wet drowning. Toxicology showed 5% carboxyhaemoglobin and a urinary alcohol of 0.014 mg 100 ml⁻¹. The cause of death was given as ischaemic heart disease.

Comments: There are a number of possible scenarios which could explain these events, although, given that it was unwitnessed, all are speculative. Such scenarios would include:

- A primary cardiac event related to the observed coronary lesions. While the critical stenosis for cardiac sudden death is usually taken to be 75% during exertion, CL has seen deaths believed to have been a result of 60% stenosis, particularly if the stenosis was recent and collateral circulation had not developed.
- An underwater epileptic event. Epileptic events have been reported to have occurred despite prolonged seizure-free periods post cessation of anti-epileptic medication.^{2,3}
- A primary drowning event resulting from possible hose entanglement and loss of regulator; it is important for such a regulator to be anchored to a harness so that it is not pulled from the mouth unexpectedly.

The dive computer record of a three-minute dive time was interesting, given that the witnesses were adamant that he had been underwater for at least an hour. This, together with the companions' statements that the victim was dragged in at an angle from depth, led the investigating coroner to conclude that there was most likely an error in the computer's record, a conclusion that seems reasonable, albeit apparently uncommon. The damage to the mouthpiece of the regulator could have occurred during recovery of the body, or could have indeed been the primary event. It is possible that the diver became entangled and the regulator was forced from his mouth.

Summary: Previous epilepsy not declared on dive medical; severe atherosclerosis; trained and experienced commercial diver; dived alone without proper surface watch; drowning

Discussion

APNOEIC HYPOXIA

This series includes the tragic deaths of two fit and healthy young breath-hold divers (BH 08/01 and BH 08/07) who likely died as a result of apnoeic hypoxia. It appears likely that at least one of these divers had practised hyperventilation, a well-reported precipitant for loss of consciousness during or after ascent.^{4,5} It is important for the diving medical and training community, dive clubs and spearfishing groups to better inform breath-hold divers of the risks of extended apnoea, with or without hyperventilation, and the potential benefits of a vigilant and efficient buddy system to facilitate a rapid rescue in the event of unconsciousness. Although this problem has been recognised for a long time, deaths related to apnoeic hypoxia are still common. For example, 19 of 130 snorkelling-related deaths in Australia between 1994–2006 appear to have resulted from prolonged breath-hold diving, largely in experienced divers.⁶

It is also important for such divers to adjust their buoyancy in order to be positively buoyant in the last few metres to the surface. In that way they will be more likely to rise to the surface if unconscious and so be easier to locate and rescue. Unfortunately this is less likely to occur if unconsciousness occurs at depth. Also of note, there is a relatively new product available for those keen apnoea divers who are willing to go to the effort and expense. The auto-inflatable *Freediver Recovery Vest* can be pre-programmed to a trigger depth or time and will auto-inflate and bring the wearer to the surface when either of these parameters is reached.⁷

CARDIAC-RELATED DEATHS

Coronary artery disease is a common cause for sudden, unexpected death, especially during exertion.⁸ Of the 130 snorkelling-related deaths reported in Australia between 1994 and 2006, 60 (46%) appeared to have been attributed to cardiac causes.⁶ Twenty-six of these victims (43%)

had no cardiac or other significant medical history. Once again, cardiac factors appear to be the disabling injury in many of the deaths in the present series, in both snorkellers (BH 08/02, BH 08/03, BH 08/04, BH 08/10 and possibly BH 08/08) and compressed gas divers (SC 08/01 and SC 08/04, and possibly SC 08/02, SC 08/03 and SS 08/01). This on-going and increasingly recognised issue highlights the importance of potential or active snorkellers and divers, as well as dive operators and doctors, appreciating the various cardiac stressors associated with these activities and carefully considering the suitability of the participant (and, sometimes, his/her equipment). This is often easier to ascertain in an individual with a relevant medical history if this person is forthcoming with the pertinent information, which is not always the case. Increasing age is a general indicator of an increased risk.⁹ In this series, the ages of victims of suspected cardiac-related deaths ranged from 41 to 82 years.

Of note, three of the nine likely cardiac-related deaths were in victims who were obese, with BMIs ranging from 30.3 to 35.1 kg m⁻². Even if a cardiac event did not underlie the death in some obese divers, obesity per se can be a contributory factor.^{10,11} The adverse effect of obesity on respiratory mechanics when immersed, the often excessively tight suit or equipment in obese divers, and the need for excessive weights are common in such divers and act to compromise safety. Undiagnosed obstructive sleep apnoea, diabetes, as well as hypertension and high cholesterol are more common in obese individuals, and are all risk factors for coronary artery disease.

The relevance of cardiac stress testing in asymptomatic individuals remains controversial. Current recommendations from the American College of Cardiology/American Heart Association are not to conduct tests in such individuals due to the high rate of false positives. However, it may be worth screening individuals who have multiple risk factors.^{12,13}

MEDICAL QUESTIONNAIRE

In Australia, as in many other countries, snorkellers and divers are often required to complete a short medical questionnaire prior to diving with a commercial operator – an important safety measure. Not uncommonly, participants seek clarification of some of the questions from staff of the dive operation, who usually have only a basic, if any, understanding of various medical conditions and their implications for diving. This problem may be exacerbated by a language barrier. Case BH 08/08 highlights this problem. It appears that, on the advice of the receptionist for the tour operator, the victim altered her declaration of a possible pre-existing medical condition(s), possibly contributing to her demise. Relevant training of everyone who is involved with taking bookings as well as those on board the vessels is imperative. Ideally, prospective divers and snorkellers should be vetted onshore, and at the time of enquiry about,

or making a booking for, a proposed diving or snorkelling outing. The questions should cover their medical history and condition and relevant experience prior to them committing to the proposed activity. In reality, this is not always practicable. However, this may assist in identifying, at a time when there is less pressure to proceed, those who should not snorkel or dive. Some of the case histories also show that the two-way communication of information between dive operator and client remains a challenge, especially when there are language barriers.

SUPERVISION OF IN-WATER ACTIVITY

The right balance between the number of snorkel/dive guide and that of participants is another ongoing challenge. Cases BH 08/02 and BH 08/08 again illustrates the difficulty in focusing on the entire group when one or more of the participants require attention. This problem is exacerbated when the group includes inexperienced and/or potentially medically challenged participants.

USE OF DEFIBRILLATORS ON DIVE BOATS

Although details are often not included or well-described in the reports provided to the coroners, it appears that in at least in one case (BH 08/03) an AED from the dive vessel (as opposed to defibrillators brought by medical attendants) was used, albeit unsuccessfully. In this case, the AED was attached after 10–15 minutes of CPR and a single shock advised and delivered. To provide the greatest potential benefit, these devices need to be available and utilised immediately. BLS plus defibrillation within 3–5 minutes of cardiac arrest can produce survival rates as high as 49–75%.¹⁴

BUDDY SYSTEM

The buddy system is generally taught and reinforced as an important part of diving and snorkelling safety, although diving with a buddy certainly does not guarantee safety. In fact, some commentators have argued that a buddy can sometimes endanger a diver, either directly through lack of competence or by causing an unsafe reliance to the detriment of increasing self-sufficiency. There is little doubt that training in self-sufficiency including equipment redundancy can be beneficial. Historically, a large proportion of dive accident victims die alone. A 34-year review of 351 compressed-gas diving accidents in Australia revealed that only 18% of victims were with their buddy when the fatal event occurred. Sixteen per cent had been diving solo, 49% had separated prior to the event and 17% separated during the event.¹⁵

Lack of a buddy or separation of a diver or snorkeller from their buddy is once again a feature in this series. Although the victims of BH 08/01, BH 08/05, BH 08/09 and SC 08/05 had supposed above-water look-outs (one being in a car high above!), it was unsurprising that these were

Table 3
Root cause analysis of diving-related fatalities in Australian waters in 2008

Case	Trigger	Disabling agent	Disabling injury	Cause of death
BH08/01	Apnoeic hypoxia (hyperventilation)? Presence of speared fish?	Sudden loss of consciousness? Shark attack?	Asphyxia? Trauma?	Unknown
BH08/02	Immersion; exertion?	Cardiovascular disease	Cardiac incident	Cardiac-related
BH08/03	Immersion; exertion?	Acute myocardial infarct	Cardiac incident (rupture)	Cardiac-related (cardiac tamponade)
BH08/04	Immersion; exertion?	Aortic valve replacement; enlarged heart	Cardiac incident (arrhythmia)	Drowning
BH08/05	Lack of buoyancy in poor sea conditions	Buoyancy-related	Asphyxia	Drowning
BH08/06	Poor sea conditions; inexperience?	Unknown	Unknown	Unknown
BH08/07	Prolonged breath-holding	Apnoeic hypoxia	Asphyxia	Drowning
BH08/08	Immersion; exertion; aspiration?	Unknown	Cardiac incident, arrhythmia? Asphyxia	Cardiac-related?
BH08/09	Unknown	Buoyancy-related?	Asphyxia	Drowning
BH08/10	Exertion	Cardiovascular disease	Cardiac incident	Drowning
BH08/11	Unknown	Shark attack	Trauma	Trauma?
BH08/12	Diving in presence of speeding boat	Contact with boat	Trauma	Head injury
SC08/01	Exertion	Ischaemic and valvular heart disease	Cardiac incident	Drowning
SC08/02	Exertion	Buoyancy-related	Asphyxia? Cardiac?	Drowning
SC08/03	Exertion.	Gas supply-related? Cardiovascular disease?	CAGE	CAGE
SC08/04	Exertion; immersion; anxiety?	Cardiomyopathy	Cardiac incident	Cardiac-related
SC08/05	Strong current; presence of mooring ropes	Entanglement; gas supply-related	Asphyxia	Drowning
SC08/06	Strong current and surge; rapid ascent?	Smashed against rocks. Ascent-related?	Asphyxia	Drowning
SS08/01	Unknown	Cardiovascular disease? Epilepsy? Gas supply-related?	Cardiac incident? Asphyxia?	Drowning

ineffective when needed. The buddies of the victims of BH 08/06, SC 08/02 and SC 08/03 lost contact with them, and the victim in SSBA 08/01 was diving solo and without a surface watch. A well-trained and vigilant buddy can be an important asset in an emergency. The time saved in searching for and recovering a missing diver may well prove an important factor in increasing their likelihood of survival, depending on the mechanism of their problem. The buddy in SC 08/01 was faced with a difficult decision; whether or not

to temporarily abandon his friend. His efforts were valiant, although sadly in vain.

SEA CONDITIONS

Even mild to moderate sea conditions can provide challenges for inexperienced snorkellers and divers. However, a choppy surface, or large swell, surge, strong current and/or poor visibility, or a combination of these factors, can

prove dangerous even for experienced veterans, who can sometimes become complacent. Poor conditions appeared to have been a factor in the deaths of at least seven of the victims (BH08/02, BH08/05, BH08/06, BH08/10, SC08/01, SC08/05, SC08/06). Dive sites need to be carefully assessed for suitability under the prevailing conditions, the likely problems that a diver might encounter and ways to avoid these. The diver needs to rationally consider his/her suitability to deal with the conditions. Divers sometimes adopt overly optimistic views about their ability to handle the adverse environment in which they dive. This is especially true for inexperienced divers, who can easily underestimate the challenges of diving in poor conditions, which include current, surge, surface chop and reduced underwater visibility. This should be reinforced in basic training, as well as in advanced courses.

DIVE FLAGS

Divers and snorkellers should continue to be strongly encouraged to clearly display a 'Diver Below' flag when on or under the water. Some of the authors believe that this should be supported by appropriate regulation. It cannot guarantee protection from careless boat drivers, as evidenced in BH 08/12, but it can certainly increase the likelihood of being seen and avoiding injury.

LIMITATIONS

As with any uncontrolled case series, there are 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 did the expertise of the investigators. Autopsy reports can sometimes be unreliable owing to 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 dysrhythmias, among other factors. Care must be taken to critically examine the available evidence and minimise speculation when determining the likely disabling injuries (Table 3).

Classification of cases

Classification of cases into a sequence of four events (trigger, disabling agent, disabling injury, cause of death) requires a single choice for each event, which may omit important factors in some cases.

Limited annual case data

Nineteen deaths are too few to determine reliable trends.

Conclusions

There were 19 reported diving-related fatalities during 2008 including twelve deaths while snorkelling and/or breath-hold diving, six while scuba diving and one while using a surface-supply breathing apparatus.

Causal factors associated with these deaths included inexperience, extended breath-hold dives, non-existent or poor buddy system, diving in poor conditions, poor equipment configuration, pre-existing medical conditions, shark attack and boat impact.

The likely disabling injuries in snorkellers were trauma, asphyxia and cardiac causes. In scuba divers, the disabling injuries appear to have been asphyxia, CAGE and cardiac causes.

Factors that may reduce mortality in the future include better supervision of inexperienced and older snorkellers; improved medical screening of older divers; better education of prospective and active divers about health risks; careful buddy monitoring; appropriate equipment configuration and use; the use of dive flags and care on the part of divers and boat operators in areas where both co-exist.

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Conflict of interest

John Lippmann is the Executive Director of Divers Alert Network (DAN) Asia-Pacific. DAN is involved in the collection and reporting of dive accident data and provides evacuation cover and dive injury insurance to recreational divers.

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Case report

Livedoid vasculopathy successfully treated with hyperbaric oxygen

Neil DG Banham

Abstract

(Banham NDG. Livedoid vasculopathy successfully treated with hyperbaric oxygen. *Diving and Hyperbaric Medicine*. 2013;43(1):35-36.)

Livedoid vasculopathy is a painful, ulcerating condition of the lower legs, ankles and feet with the typical histological feature of hyalinising vascular change of dermal blood vessels with minimal inflammation. Therapeutic interventions have been diverse and varyingly successful. We report a biopsy-proven case in a 27-year-old male, which responded rapidly and completely to hyperbaric oxygen therapy. A few such cases have been reported previously, but only in dermatological journals, not in the hyperbaric medicine literature.

Key words

Wounds, hyperbaric oxygen, hyperbaric oxygen therapy, case reports

Introduction

Livedoid vasculopathy (LV) is a painful, ulcerating condition of the legs, especially the ankles and feet. It has had many names, including livedoid vasculitis, segmental hyalinising vasculitis, *atrophie blanche* and PURPLE (painful purpuric ulcers with reticular pattern of the lower extremities).¹⁻³ It is most common between 15 and 50 years of age and affects females two to three times more frequently than males.²

The pathophysiological mechanism is considered to be a vaso-occlusive phenomenon due to intraluminal thrombosis of dermal venules.² Typical histological features are of hyalinising vascular changes of the subintimal layer of dermal blood vessels with minimal inflammation.⁴ Inherited defects of coagulation, thrombophilias and auto-immune connective tissue diseases have been associated with LV.² The typical clinical presentation is the development of painful, purpuric, erythematous, papular plaques and papules which may then become vesicles and ulcerate. These heal slowly over weeks or months leading to white atrophic scars (*atrophie blanche*).²

Pain is a constant characteristic and may be debilitating. This pain is perhaps ischaemic in nature as reduced transcutaneous oximetry measurements (TCOMs) have been documented in 20 of 27 patients (74.1%) in one case series.⁴ Many therapies have been promulgated, including various regimens of anticoagulation (aspirin, heparin, low molecular weight heparins and warfarin), low-dose tissue plasminogen activator (t-PA), and hyperbaric oxygen therapy (HBOT) has been used in a few patients.^{2,5-8} As the literature is very limited, a further case given HBOT is reported here. The patient provided written consent for this report.

Case report

A 27-year-old, previously fit, male, non-smoking

geophysicist contacted our hyperbaric medicine unit with a view to HBOT for his ongoing painful foot ulcers from biopsy-proven livedoid vasculitis. The patient had found a report on the internet of the successful treatment of two patients with LV in a dermatology journal and requested a trial of HBOT prior to considering methotrexate, which his dermatologist had advised.⁵

The patient reported developing a “bruising” effect on his feet a year earlier, which had spread over a few weeks and become quite painful with some open wounds. This was initially diagnosed as eczema by his general practitioner but as it was unresponsive to topical steroids, he was referred to a dermatologist who diagnosed LV, confirmed on punch biopsies of both feet. He was commenced on prednisolone 25 mg daily for a week and then tapered over two weeks, with a good but incomplete response, with most of the ulcers healing. However, upon steroid cessation the condition returned. Many subsequent courses of steroids over the next year had the same transient effect, with the condition becoming progressively worse such that most of both feet and ankles were covered in ulcers.

Apart from aspirin, 50 mg daily, there had been no trial of other anti-thrombolytic therapy or anticoagulants. Immunological testing for an auto-immune cause was negative as were a thrombophilia screen and serum protein electrophoresis. A full blood count, CRP and ESR were all normal. Bilateral leg arterial Doppler studies were normal with an ankle brachial index (ABI) of 1.1. There was no clinical evidence of varicose veins.

He was assessed as to his fitness for HBOT and no contraindications were present (he had been an enthusiastic scuba diver). TCOMs were not performed. He commenced HBOT (90 min at 243 kPa in a monoplace chamber) six weeks after presentation. Within the first week of his planned six-week course of HBOT (30 sessions) he reported

a dramatic lessening in pain and a reduction in the extent of ulceration. By the end of the course, all of his ulcers had healed for the first time since their original onset.

Upon review six months later, he reported: “*I have absolutely no pain, no ulceration, my scarring has reduced and I am living my life entirely as I was prior to becoming ill*”. At recent further contact, over a year after completing HBOT, the patient remained well and without recurrence.

Discussion

There are limited reports of HBOT for treating LV, all of which have been published in dermatology journals. Some of these report success, others failure.^{2,5-8} A recent review of LV reported four patients treated with HBOT for 20–25 sessions with complete healing of the lesions. Pain was completely relieved between the seventh and twelfth sessions.²

This early relief of pain with subsequent complete healing without relapse, as occurred in our case, was also reported in a series of twelve patients.^{5,6} Eight patients completed the planned course of HBOT with reduction in analgesic usage after an average of five HBOT and complete healing at a mean of 3.4 weeks (range 2–5 weeks).^{5,6} Six of these patients subsequently relapsed but responded to further HBOT. A transient increase in pain (not evident in our case) was noted in some after their first HBOT, which soon resolved in those that chose to continue. Two patients withdrew from further HBOT after the first session because of this increased pain. The outcome of the four patients that did not commence or complete HBOT in this series is not described. The authors commented that another mechanism of response to HBOT in LV, in addition to that usually attributed to HBOT in wound healing, could be the effect of HBOT on increasing the release of various fibrinolytic (including t-PA) molecules from endothelial cells. This effect of HBO₂ had been reported previously *in vitro* and *in vivo*.^{9,10}

A therapeutic ‘ladder’ for treatment of LV with references (which included HBOT) was published in 2006.¹ HBOT was tenth on the list of 12 considered therapies. There are no randomised controlled trials of any therapy for LV, and all forms of treatment proposed for LV are based on reports of isolated cases or case series.²

Conclusion

HBOT should be considered as a therapy in patients with livedoid vasculopathy, particularly where other treatments have failed and where pain is a significant factor and in whom there are no significant contraindications to its use.

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Editor’s note:

Colour photos of the patient’s feet and ankles just prior to and at the end of the HBOT course may be viewed until 01 June 2013 on the journal website <<http://dhmjournal.com>>; select “LV case photos”.

Diving doctor's diary

Epilepsy, scuba diving and risk assessment. Near misses and the need for ongoing vigilance

David Smart and John Lippmann

Abstract

(Smart D, Lippmann J. Epilepsy, scuba diving and risk assessment. Near misses and the need for ongoing vigilance. *Diving and Hyperbaric Medicine*. 2013 March;43(1):37-41.)

There is ongoing debate about the safety of scuba diving for individuals with a history of epilepsy. An in-water seizure is highly likely to be fatal. Recommendations for fitness to dive vary with some regarding epilepsy as an absolute contraindication to diving (South Pacific Underwater Medicine Society) and other permitting diving under strict criteria (United Kingdom Sport Diving Medical Committee) with diving to be postponed for a period of three to five years without seizures. Long-term follow up of people with epilepsy shows that at least one-third will have a recurrence and that the risk remains elevated for many years. We present three cases where individuals with a history of epilepsy (or likely epilepsy) almost fell through the cracks of health risk assessment, two with near-fatal consequences. These cases inform the on-going debate about fitness to dive for those with current or past epilepsy, and highlight the importance of education for doctors, dive professionals and divers about the risks associated with epilepsy and diving.

Key words

Epilepsy, scuba diving, scuba accidents, fitness to dive, recreational divers

Introduction

The increase in knowledge in diving medicine, coupled with the changing legal environment in which fitness-to-dive assessments are conducted, have led to changes in the criteria for determining whether a candidate is considered fit to dive. Previously there was a variety of absolute contraindications, which included asthma, diabetes, previous myocardial infarction and epilepsy, among others. Current fitness-to-dive assessments, certainly in Australia, often involve the physician identifying the risks and thoroughly explaining these to the candidate. The candidate then, to a large extent, makes his or her own decision about risk acceptance, based on information and advice provided.

These changes have enabled many individuals with a variety of chronic or previous medical conditions that would have formerly excluded them from diving to take up or continue the activity. Divers Alert Network Asia-Pacific (DAN AP) membership records indicate that in excess of 10% of its members are diving despite admitted histories of asthma, diabetes, cardiac disease and a broad range of other conditions. However, a history of epilepsy is still generally considered an absolute contraindication to diving. This raises potential problems relating to medical education, dive industry education and compliance, and diver/prospective diver education, disclosure and compliance. The following three cases are presented to inform this ongoing debate.

Case 1

A 24-year-old male presented to a dive shop to enroll in an

open-water dive course. He explained that he had a history of epilepsy but was now seizure-free and handed the operator a copy of a recent fitness-to-dive assessment. This indicated positive responses to the question about 'migraines; fainting or blackouts; convulsions, fits or epilepsy' and to the question about 'concussion or head injury'. The medical form was accompanied by a letter from a neurologist stating that the patient "*has well-treated epilepsy and has been seizure-free for a period greater than 18 months and is tolerant of and compliant with medication. He is at a very low risk of further seizures and there should be no impediment to him obtaining his certification for scuba diving ...*".

Still apprehensive about enrolling the person in a dive course, the operator contacted DAN AP for advice. One of the authors (JL) subsequently called the certifying doctor, who was himself a diver, although untrained in diving medicine. He was aware of the reported risks of epilepsy and diving but had been swayed by the letter from the neurologist. The same author then called the neurologist and informed him of the various guidelines for epilepsy and diving. The neurologist admitted that he had equated the risks of diving with those of driving a motor vehicle. Now concerned about the advice he had given the patient, the neurologist decided that he would contact him to inform him of his change of advice. It is not known whether the person has proceeded with diving with another operator.

Case 2

This 27-year-old male overseas tourist had a history of "*absence seizures*" throughout childhood, for which he took

medication (sodium valproate and topiramate). However, he had ceased taking medication some 10 years earlier and his last 'absence' was when he was 19 years old. He stated that he had never become unresponsive during any of these events and described them as him "*losing focus but remaining able to speak*". He had become certified as a diver at college some six years earlier and had done an estimated seven open-water dives in total prior to the accident described below.

On this occasion, the diver was holidaying on the Great Barrier Reef. For two days prior to the dive, he had been 'partying', drinking more alcohol than usual and had had relatively little sleep. On the dive charter vessel, he was provided with a standard dive medical questionnaire which, among other conditions, specifically asked if the prospective diver has or was suffering from 'epilepsy', and separately 'fainting, seizures or blackouts'. The diver answered "No" to these questions. He later stated that, given he had never become unconscious or had convulsions he did not consider that he suffered from epilepsy and was not withholding or providing false information. He did not believe that he was at risk. He also stated that he was rushing to get ready and was keen to go diving. His travelling companion was reportedly aware that he had declared that he had suffered from epilepsy when applying for a gymnasium membership.

He entered the water in a group of six divers, submerged to a depth of about 6 metres of seawater with his buddy and was swimming along with another pair of divers behind. After about a minute, his buddy noticed that his body position was "*odd*" and, when she checked, she found he was unconscious. His regulator was out of his mouth and his mask was filled with "*frothy white bubbles*". She signalled nearby divers to help her and they inflated his BCD, brought him to the surface and signalled to the dive boat crew for help.

When brought aboard the tender, he was found to be unconscious, cyanotic and apneic and there was froth coming from his mouth. CPR was begun immediately and fortunately he responded following the second group of chest compressions. He was evacuated to hospital and discharged without sequelae after two days. A follow-up MRI showed no abnormalities. An EEG, conducted after 24 hours without sleep, indicated some abnormal brain activity thought to be consistent with 'absence seizures'. He was then prescribed medication (topiramate) again.

Case 3

A 20-year-old student nurse presented to an experienced diving physician for a recreational diving medical. The only positive history that she gave was having "*fainted a couple of times*" on night duty on a busy surgical ward. On closer questioning, she insisted that these were brief, minor events brought on by lack of sleep and a heavy, stressful workload, that she had carried on to complete the shifts and that she

had had no other similar events. Physical examination was entirely normal and, despite this history, she was passed 'fit' to participate in an open-water dive course.

About two weeks later, the diving physician was rung by the diving instructor (whom he knew well) to say that a week previously, the student had had a "*funny turn*" in the swimming pool during training and he thought that she had either panicked or "*passed out briefly*", he was not sure which. Given his (misplaced) confidence in the physician, he had chosen not to refer her back for reassessment, but to continue her training. The first open-water experience was on the day before he phoned – a shore-entry dive off a sandy beach with a slight swell. Whilst wading out in about thigh-deep water she was seen to have a tonic-clonic fit and was promptly recovered without harm from the water by the instructor, assisted by other trainee divers in the class.

Further medical assessment was arranged, but she did not attend several attempted appointments. Soon after, she quit her nursing training programme and disappeared from follow up. The instructor was convinced that this was a full-blown epileptic fit as he had witnessed such convulsions in a family member on several occasions in the past.

Commentary

IN-WATER SEIZURE

There appears to be little argument that a seizure underwater while using conventional scuba equipment is usually fatal, most often due to drowning. While there may be some conjecture as to the cause of loss of consciousness in Case 2, the previous history of absence seizures, a positive follow-up EEG and re-introduction of active treatment suggest that epilepsy was a likely contributor. Absence seizures have a typical EEG pattern. Clinically, the individual is rendered 'incapacitated' during an episode when they sustain an abrupt impairment or loss of consciousness which is not remembered. During this period of incapacity, loss of a regulator from the diver's mouth could lead to drowning. This diver was very lucky to have survived, and did so only as a direct result of close buddy scrutiny and rapid and effective rescue and first aid. Not all divers are so fortunate.¹

Between 1997 and 1999, DAN America received 29,239 calls to its Medical Information Line of which 212 were regarding seizures, and seven of these involved new onset seizures post diving.² Project Stickybeak and DAN Asia-Pacific fatality records indicate that epilepsy may have been a contributing factor in the deaths of at least 11 snorkellers and two scuba divers in Australia since 1972.³⁻⁶ In New Zealand, there were 229 diving-related fatalities derived from Water Safety NZ's *DrownBase* between 1980 and 2006.^{7,8} Epilepsy was noted in 10, six snorkellers and four scuba divers, and was suspected of having been contributory to the deaths in all 10 cases. However, it is often difficult to determine with

certainly the role epilepsy played, if any, in the absence of a reliable history, witnesses and/or autopsy, as occurs with some fatality investigations.

Diving and/or snorkelling may involve a variety of factors that can reduce the seizure threshold. These include stress, exercise, sensory deprivation, hypercapnoea, hyperventilation and hypothermia. It has been suggested that the elevated oxygen partial pressures associated with diving, especially using enriched air mixtures, may increase the likelihood of a seizure in a diver with epilepsy.⁹ However, there are no reliable data to support this belief.¹⁰ The preceding night's activities combined with diving, may have increased the risk of an event leading to impaired consciousness for Case 2.

GUIDELINES FOR EPILEPSY AND DIVING

Guidelines for epilepsy and diving from diving medical advisory bodies differ. The South Pacific Underwater Medical Society (SPUMS) recommends that *"a candidate with a history of fits (apart from childhood febrile convulsions), or unexplained blackouts should be strongly advised against diving. Any condition associated with fits or blackouts will be a grave risk to life during diving."*¹¹

The United Kingdom Sport Diving Medical Committee (UKSDMC) is less stringent. It advises that it is unsafe for any epileptic to dive while taking anti-epileptic medication because of the likely sedative effects of the drugs. It states that:

"The relapse rate in epileptics who are taken off medication decreases exponentially, with the majority of those relapsing doing so within the first eighteen months of ceasing treatment and the rate of relapse becoming insignificant after three years. The suggested requirements for an epileptic to be permitted to dive are therefore set at five years free from fits and off medication. Where the fits were exclusively nocturnal, this can be reduced to three years."

According to the UKSDMC,

*"A past history of petit mal should not disqualify, provided that no attacks have occurred for five years and that the condition has not progressed to epilepsy. Pyrexial convulsions in childhood may be disregarded if not followed by epilepsy."*¹²

There is on-going debate about the suitability of these guidelines, which some argue are too stringent. For example, a review by Almeida and colleagues, after considering available evidence, suggested that

"Those who have been entirely seizure-free on stable antiepileptic drug therapy for at least four years, who are not taking sedative antiepileptic drugs and who are able to understand the risks, should then be able to consider

*diving to shallow depths, provided both they and their diving buddy have fully understood the risks."*¹⁰

The prospective diver in Case 1 would have been judged unfit to dive according to any of the three above-mentioned guidelines. Both doctors involved placed this person at risk and themselves in a potentially precarious medicolegal position by suggesting it would be safe for him to dive.

The diver in Case 3 probably would have been passed for recreational diving by almost all physicians. This is an example of how reliant the examiner is on the honesty of the candidate. However, without witnesses, the physician involved, whilst slightly suspicious about the nature of the 'faints', elected (misguidedly) to trust the word of the patient.

LONG-TERM RISKS OF RECURRENT SEIZURES

The diver in Case 2 would have been advised not to dive following the SPUMS recommendation as he had a history of epilepsy. However, he would have been determined as fit to dive in accordance with both the UKSDMC guidelines and those suggested by Almeida et al, as he had been seizure-free without medication for almost eight years, considerably longer than required under the UKSDMC guidelines. It is also relevant to acknowledge that although absence seizures usually do not cause loss of consciousness, they do cause impairment of function that, in the aquatic environment, may place the diver at great risk. There was no evidence of an equipment fault or any other cause to explain his loss of consciousness. The victim has no recollection of having any problem underwater and only recalls taking some photographs and then regaining consciousness on the boat.

A recent long-term follow up of 148 individuals with epilepsy is less reassuring.¹³ In this study, 90 individuals who stopped antiepileptic medication because they had achieved five years seizure free, were followed up for an average of 32 years. Overall, 37% of these individuals suffered a relapse of their epilepsy, with two-thirds occurring within three years of treatment cessation. In the remaining population, relapse occurred between three and 28 years, suggesting that it is difficult to predict which individuals are at risk in apparently stable epilepsy. A Canadian study had similar results, with 30% of 260 children experiencing recurrence within five years of discontinuing antiepileptic drugs.¹⁴ These two studies do create uncertainty when applying the UKSDMC guidelines.

An argument advanced by Almeida et al was that there was a lack of data regarding diving and epilepsy, and that

*"objections to diving by people who have been seizure-free for a long time are largely theoretical."*¹⁰

They also stated

"current data do not allow precise assessment of the magnitude of any risk assessment."

Table 1Risk assessment matrix;¹⁵

L – low; M – moderate; H – high; E – extreme

Likelihood	Consequences				
	Insignificant	Minor	Moderate	Major	Severe
Almost certain	M	H	H	E	E
Likely	M	M	H	H	E
Possible	L	M	M	H	E
Unlikely	L	M	M	M	H
Rare	L	L	M	M	H

It is true that these events are infrequent, providing challenges for any meaningful interpretation.

RISK MATRICES FOR ASSESSING RISK

When assessing risk in the absence of precise data, risk matrices derived from Australian/New Zealand and International Standards Organisation Standard 31000:2009 (formerly AS 4360), provide pragmatic guidance (Table 1).¹⁵ These matrices have been validated across many industries from manufacturing to finance and even the Federal Government. Although not formally validated in the health-care setting, risk management is progressively gaining acceptance and a higher profile. For example, the World Health Organisation is in the final stages of producing general quality risk management guidelines, which also utilize a matrix.¹⁶ Risk is calculated by assessing *likelihood* and *consequence*. From this matrix, risks can be classified as low (L), moderate (M), high (H), and extreme (E).¹⁷

One of the authors (DS) has used the risk matrix since 2004 when discussing health risks with individual divers. It provides a useful semi-quantitative format for risk evaluation in the absence of precise epidemiological data. For a condition such as epilepsy, even though the frequency may be rare, the consequence is severe; hence the risk is evaluated as high. On that basis, we assert that evidence of safety must be provided before any form of diving with epilepsy is considered. Recent data from a large epidemiological study of epilepsy after traumatic brain injury (a different but related issue) suggested that there was increased risk even at 10 years after injury. This paper provided ‘decay curves’ for risk from their population of over 1.6 million.¹⁸

GENERAL COMMENTS

Case 1 as presented raises a number of issues regarding the current system of health risk assessment of divers. In Australia, as in most other countries, there is no mandate for recreational divers to receive assessment by doctors trained in diving medicine. Knowledge of the potential risks of impairment in the non-respirable aquatic environment among even expert health professionals is variable, particularly if they have not received training in diving medicine. Guidance material is available, even if it is

challenged by some authors.^{10–12} This case also illustrates the important role of the dive instructor in the overall system of evaluation of recreational diving candidates. Some dive centres find that establishing long-term links with specific physicians helps in informing a two-way exchange about fitness problems.

Education of dive professionals, divers and health professionals is the key, informed by quality studies or, in their absence, a careful risk assessment applied to the individual circumstances of the potential diver. Equally important is the establishment of a quality database for reporting near misses and non-fatal diving accidents, such as the DAN AP Non-Fatal Diving Incident Reporting Project¹⁹ (the successor to Acott’s *Diver Incident Monitoring Survey*),²⁰ which will permit meaningful analysis of risk, and better inform health professionals who are assessing that risk.

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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 now at:

<<http://hboevidence.unsw.wikispaces.net/>>

Assistance from interested physicians in preparing critical appraisals is welcomed.
Contact Associate Professor Michael Bennett: <M.Bennett@unsw.edu.au>

The
Diving and Hyperbaric Medicine
journal website is at

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World as it is

How delay to recompression influences treatment and outcome in recreational divers with mild to moderate neurological decompression sickness in a remote setting

Till S Mutzbauer and Enrico Staps

Abstract

(Mutzbauer TS, Staps E. How delay to recompression influences treatment and outcome in recreational divers with mild to moderate neurological decompression sickness in a remote setting. *Diving and Hyperbaric Medicine*. 2013;43(1):42-45.)

Introduction: This retrospective review examined the influence of delay to recompression on mild/moderate neurological decompression sickness (DCS) in divers, as a pilot for an abandoned prospective study.

Methods: The medical histories of 28 divers treated at a hyperbaric facility in the Maldivian Islands in the Indian Ocean were evaluated. The term 'oxygen unit' (OU; 1 OU = 1 bar (ambient pressure) x 1 min x 1.0 (inspiratory oxygen fraction)) was used to enable a quantification of administered hyperbaric oxygen. Visual analog symptom scale (VASS) scores of the worst symptom at presentation (used routinely at the clinic to quantify treatment response) were analysed.

Results: Divers presenting later than 17 hours after surfacing (the median time to treatment after surfacing for the whole group) were likely to have more intense symptoms on VASS (median 100%) than those who presented earlier for treatment (median 30%, $P = 0.02$). Total OU needed to treat divers presenting within 17 hours did not differ from those treated later ($P = 0.11$). Divers with $\geq 70\%$ symptom reduction with the first hyperbaric oxygen treatment (HBOT) needed between 260 and 1,463 OU in total, whereas those with less than 70% reduction in VASS needed between 263 and 2,126 OU ($P = 0.04$).

Conclusion: Neither more HBOT nor a worse outcome of DCS could be related to delay to treatment longer than 17 hours. The amount of oxygen that had to be administered in total during the whole HBOT course was lower in cases that responded better to the initial HBOT.

Key words

Decompression sickness, recreational diving, scuba diving, resort diving, hyperbaric oxygen therapy, outcome

Introduction

Data on the management of decompression sickness (DCS) in third-world and remote-area diving resorts are largely absent from the literature. Treatment of DCS may be delayed considerably, and this could result in the need for more intense treatment and/or a poorer clinical outcome.¹ It was thought that the grade of symptom intensity reduction during initial hyperbaric oxygen treatment (HBOT) of patients with DCS may be an indicator of the further course of the disease. Another hypothesis to be considered was that the total amount of oxygen administered would be lower in cases that respond better to the initial HBOT.

Bandos Medical and Hyperbaric Treatment Centre (BMC) is located near the capital island, Male, the Republic of The Maldives. Divers are transferred for treatment from other atolls as well as from the vicinity of the centre. Quality standards for treatment of diving injuries at BMC are controlled by the European Divers Alert Network.

Methods

A small, retrospective review of divers with mild to moderate

peripheral neurological DCS (e.g., numbness, tingling or paraesthesia) treated at BMC in 2000 and 2001 was carried out. As a quality assurance review, the Maldivian authorities advised that ethical approval was not required. However, formal ethics approval for a proposed prospective randomised study based on these preliminary results was provided by the Ministry of Health, but that study never eventuated.

All divers presenting to BMC with severe type II (cardiopulmonary or neurological) or type I DCS with musculoskeletal symptoms only and cases with incomplete data were excluded. Also excluded were divers with mild/moderate DCS whose symptoms developed later than the 90% quantile for all divers, and those with a treatment delay exceeding the 90% quantile for all divers. This left two partly identical subgroups of 28 or 26 divers for analysis from a larger data pool of 81 divers presenting over the time period of the study.²

Data were entered anonymously into a Microsoft Excel® sheet. A visual analog symptom scale (VASS) allowed divers to rate their symptom intensity, based on the maximum (100%) perceived after onset. The most intense

Table 1

Analyses conducted on 28 or 26 divers (some data for two divers missing) with mild/moderate peripheral neurological decompression sickness; OU – ‘oxygen unit’; VASS – visual analogue symptom severity expressed as a % based on the maximum (100%) at the time of admission

Variable	Group 1	Group 2	Examined parameters
Symptom intensity (VASS) change with initial hyperbaric therapy (Red _{init})	Red _{init} < 70% n = 8	Red _{init} ≥ 70% n = 18	Total oxygen administered (OU); time from surfacing to onset of the first symptom (t _s); time from surfacing to beginning of hyperbaric treatment (t _b)
Time from surfacing to beginning of hyperbaric treatment (t _b)	t _b ≤ 17 h n = 15	t _b > 17 h n = 13	OU; time from surfacing to onset of the first symptom (t _s); VASS change with initial hyperbaric therapy (Red _{init})

symptom was chosen for evaluation. The first VASS value (t₁) represents the symptom intensity before initial HBOT, t₂ the intensity after the initial HBOT and t₃ the intensity at discharge. Additionally, the reduction in symptom intensity following the initial HBOT was calculated for each diver:

$$Red_{init} = (1 - t_2/t_1) \times 100\% \quad (1)$$

The term ‘oxygen unit’ (OU) was generated to enable a comparison between different treatments/individuals, where:

$$1 \text{ OU} = 1 \text{ bar (ambient pressure)} \times 1 \text{ min} \times 1.0 \text{ (inspired oxygen fraction)} \quad (2)$$

The total oxygen dose in OU was calculated for each patient.

Having collected half of the available data, a provisional analysis was made. The median symptom-intensity reduction of all divers after the initial HBOT served as a cut-off value to form two groups for comparison with regard to the OU administered. Patients were also divided into two groups according to the delay between surfacing from the last dive to commencing HBOT (t_b ≤ 17 h and > 17 h). The total OU administered and VASS reduction were compared between these two groups (Table 1).

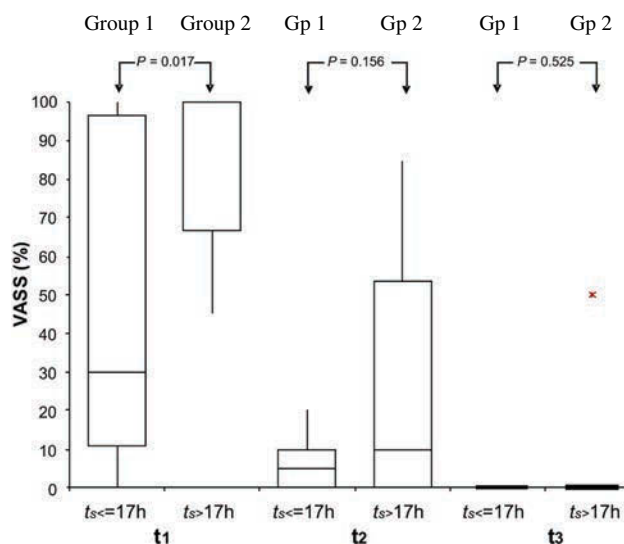
Differences between groups were compared using the Mann-Whitney U-Test. Statistical significance was taken at a P-value < 0.05.

Results

Half of the divers examined reported onset of the first symptom within one hour after surfacing; three-quarters had developed symptoms within two hours. The median time to start of HBOT was 17 hours after surfacing, in two thirds within 26 hours and in 90% within 54 hours. At the time of admission (t₁) VASS in

Figure 1

Box plots for intensity of symptoms VASS (%) of two groups of divers with decompression sickness divided according to the delay to treatment and the times of measuring VASS: t₁ (on admission); t₂ (end of initial hyperbaric oxygen therapy, HBOT) and t₃ (at discharge); Group 1 ≤ 17 h, n = 15; Group 2 > 17 h, n = 13; “+x” maximum (column 3); “—” median; box: interval between 25% and 75% quantiles; t_s – time from surfacing to commencing HBOT

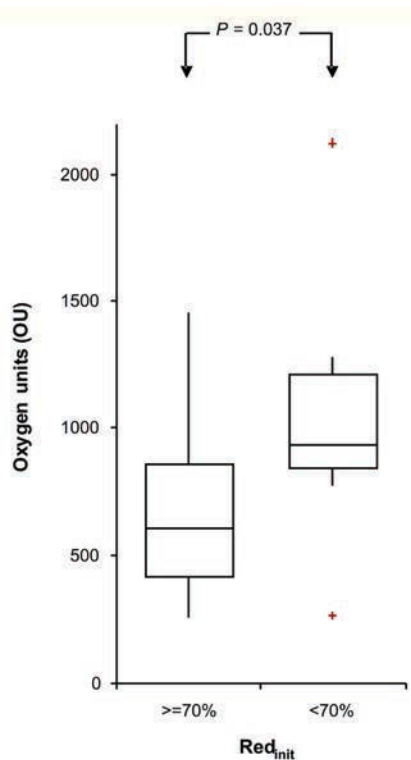


Group 1 ranged from 0% to 100% (median 30%) and in Group 2 from 45% to 100% (median 100%) (P = 0.017); i.e., a higher VASS was found if the initial treatment was started later than 17 hours after surfacing (Figure 1).

The median VASS reduction after the initial HBOT was 70%. There was no difference between the two VASS groups (≤ 70% and > 70% VASS improvement) with respect to the time to onset of the first symptoms (P = 0.48) or the time interval until the beginning of treatment (P = 0.82). Total

Figure 2

Box plots for oxygen dose in two groups of divers with decompression sickness divided according to the percentage reduction in intensity of the predominant symptom during the first hyperbaric treatment (Red_{init}); Group 1 $\geq 70\%$, $n = 18$; Group 2 $< 70\%$, $n = 8$; “+” maximum and minimum; “—” median; box: interval between 25% and 75% quantiles



OU required was less in patients with $\geq 70\%$ improvement in VASS with the initial HBOT (median 604 OU, range 260 to 1,463) than in those with $< 70\%$ improvement (median 942 OU, range 263 to 2,126, $P = 0.037$; Figure 2). No differences were seen between the time-delay groups in terms of clinical outcome ($P = 0.9$) or in total OU ($P = 0.11$).

Discussion

Times to symptom onset are similar to those reported previously, with about half of all divers presenting at BMC noticing onset within one hour and 90% within six hours after their last dive.^{3,4}

In cases of poor initial response, repeated HBOT may achieve a better outcome.⁵ The present study, in which patients whose most severe symptom was reduced by at least 70% with the initial HBOT, needed less HBOT, is consistent with this view. It has been recommended that a patient be treated as long as no further improvement of symptoms can be observed.⁶ In another study, divers who were free of symptoms after the initial HBOT were treated an average of 10 hours from onset, whereas in divers who needed more than one recompression therapy the average

delay was 18 hours.⁷ Increasing severity at presentation and delay to the initial treatment are reported in some studies to have a negative effect on treatment and residual symptoms, whereas in other studies neither the time to recompression nor the choice of initial hyperbaric procedure appeared to influence recovery.⁸⁻¹¹ In this small BMC series, those divers with a delay of more than 17 hours to treatment tended to have more intense symptoms measured on a VASS than those treated in under 17 hours, which may be an indicator of more severe tissue damage induced by the delay. However, this did not influence outcome.

Use of a simple parameter – the oxygen unit (OU) – to measure total oxygen exposure, especially as the HBOT schedules were sometimes not identical, suggested that delay also resulted in more treatment being required to achieve a satisfactory clinical outcome.

This study has several limitations. Firstly, only a small number of divers were studied. Secondly, it is difficult to determine in all divers the exact time when symptoms occur, as this may happen when the diver is asleep. The reported symptom onset may, therefore, be an unreliable parameter for studies, in contrast to the exact time of surfacing from the last dive. Thirdly, although most of the divers had received first-aid normobaric oxygen, there were no data regarding the actual duration and inspired oxygen fraction. Further, not all initial HBOT schedules were identical. Finally, dehydration was almost always an issue; however, haematocrit was not monitored. Despite these limitations, it was considered useful to report these findings from a remote area environment as such data are very limited.

Conclusion

This small study from a medical centre in the Maldivian Islands does provide encouragement to remotely situated hyperbaric chambers as well as in third-world resort areas. With attention to good standards of care, satisfactory outcomes can be achieved in mild to moderate peripheral neurological DCS.

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Accepted: 04 February 2013

Conflict of interest: Nil

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Enrico Staps, MD, is a medical officer in the Federal Armed Forces, presently at the Armed Forces Medical Centre, Ulm, Germany. He was a medical student at the time of the study.

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European Committee for Hyperbaric Medicine Workshop 2013 Diagnosis and treatment of mild DCS in remote diving destinations

Date: 22 September 2013

Timing: 1400–1900 hr

Venue: Tamarun, St Gilles les Bains, Réunion Island

Organizing Committee:

Alessandro Marroni, Ramiro Cali-Corleo, Jacek Kot

Theme:

Diagnosis and initial treatment of mild decompression sickness occurring in remote diving destinations and its eventual management at the hyperbaric facility

Programme:

- Definition of mild DCS, clinical manifestations, differential diagnosis and threshold between mild and serious DCS
- Natural history of DCS – case histories with special emphasis on delayed versus early treatment and final outcome
- Telemedicine triage and decision making for ‘remote locations’
- Immediate care and in-water recompression
- Non-hyperbaric treatment: pros and cons
- Cost-benefit evaluation; liability implications of local non-hyperbaric treatment vs. standard Medevac
- (Panel Discussion and Workshop Conclusions)

For further details:

Websites: <www.ECHM.org> or <www.reunion2013.org>

EUBS Workshop

Summary of EUBS Workshop 2011: Validation of dive computers

Lesley Blogg, Michael Lang and Andreas Møllerlækken

The results of the *Validation of Dive Computers Workshop*, convened for the European Underwater and Baromedical Society by the Baromedical and Environmental Physiology Group of the Norwegian University of Science and Technology on 24 August 2011 in Gdansk, Poland are reported in Blogg et al. (2012).¹ The workshop objectives were to discuss the validation of dive computers (DC) for use by working (commercial inshore) divers under the control of the Norwegian Labour Inspection Authority.

A review of validation procedures of dive tables and dive computers set the stage, followed by consideration of the applicability of dive computers to commercial diving operations. The need for standardization of DC technology and their classification as European Union personal protective equipment was discussed. The case was made for well-documented decompression algorithm testing via man-dives, calibrated against a measurable risk of decompression sickness. The relative conservatism of dive computers was evaluated via test chamber profiles, which could be used to identify a test plan for human trials. The applicability of venous gas emboli as an endpoint in the validation process was debated. The experiences of the military, scientific and recreational dive communities with dive computer use and management were reported.

It is worth noting that none of the dive computer manufacturers provides any details as to the inner workings of their models and none has ever performed any substantial human validation. However, in recreational diving, dive computers have been used effectively for over 25 years. The workshop advocated that a validated dive computer would be a useful tool for providing real-time decompression guidance for working divers. It was recommended that a Configuration Control Board be formed to assess conformance with validation requirements, monitor dive computer operational performance, and specify diver education and training.

The following findings and recommendations from the workshop were advocated to the Norwegian Labour Directorate:

General community specific requirements:

- accept that at present decompression sickness (DCS) is the measurable negative outcome;
- specify acceptable level of DCS risk and how it is measured;

- define window of applicability for the DC;
- a dive planner to support the DC is required;
- equipment functionality/functional safety must be documented and verified.

Findings applicable to commercial diving:

- a DC is a risk management tool. The operational risk of DCS in the recreational and scientific diving communities is no worse than previous experience with sub-no-decompression diving compared to table use, primarily as the DCs are not pushed to their model or algorithm limits. There is no evidence that multi-level dives with DCs are more risky than square dives following the same algorithm;
- documentation of theory (i.e., logic and equations) is required – what is in the box;
- this documentation must include methods to test the implementation of the theory in the DC;
- use a DCS-risk indicator model to validate the algorithm, or manufacturers may produce a DC with a validated and documented algorithm;
- specify platform technical requirements;
- develop and implement a configuration control plan.

Recommendations:

- 1 The workshop advocates that a validated dive computer would be a useful tool for providing real-time decompression guidance for working divers.
- 2 A mechanism for making judgment should be part of the system.
- 3 Institute a Configuration Control Board to assess conformance with validation requirements, monitor DC operational performance, and specify diver education and training.

The Proceedings of the EUBS Workshop on Validation of Dive Computers were dedicated to Dr RW 'Bill' Hamilton (1930–2011) whose intellect, compassion, and love of life will not soon be forgotten. The proceedings are available for download from: <www.ntnu.no/diving>.

Reference

- 4 Blogg SL, Lang MA, Møllerlækken A, editors. *Proceedings of the Validation of Dive Computers Workshop*. 24 August 2011, European Underwater and Baromedical Society Symposium, Gdansk. Trondheim: Norwegian University of Science and Technology; 2012.

Key words

Medical society, meetings, decompression, computers – diving, diving at work

Lesley Blogg, PhD, SLB Consulting, UK

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Continuing professional development

The 'normobaric oxygen paradox': another potential way to use oxygen. CME activity 2013/1

Costantino Balestra

Accreditation statement

Intended audience

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

Objectives

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

Faculty disclosure

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

Do I have to pay?

All activities are free to subscribers.

Background reading

Practitioners are referred to the following background references and reading.

- 1 Lafere P, Schubert T, De Bels D, Germonpre P, Balestra C. Can the normobaric oxygen paradox (NOP) increase reticulocyte count after traumatic hip surgery? *J Clin Anesth.* 2013 Jan 16. pii: S0952-8180(12)00417-5. doi: 10.1016/j.jclinane.2012.06.021. [Epub ahead of print].
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the normobaric oxygen paradox in cardiac surgery patients. *Br J Anaesth.* 2011;106:752-3.

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Some useful links:

<https://dipot.ulb.ac.be/dspace/bitstream/2013/114522/1/Edito%20EJAP2010.pdf>

http://www.aekkt.at/documents/10745/1501501/Balestra-Normobaric_Oxygen_Paradox.pdf

How to answer the questions

Please answer all responses (A to E) as True or False.

Answers should be posted by e-mail to the nominated CPD co-ordinator.

For EUBS members for this CPD issue this will be Costantino Balestra, e-mail: <daneuben@skynet.be>

For ANZCA DHM SIG and other SPUMS members, this will be Suzy.Szekely, e-mail: <Suzy.Szekely@health.sa.gov.au>

On submission of your answers, you will receive a set of correct answers with comprehensive material.

A correct response rate of 80% or more is required to successfully undertaking the activity. Each task will expire within 24 months of its publication to ensure that additional, more recent data have not superceded the activity.

Key words

Oxygen, haematology, treatment, physiology, case reports, research

Question 1. Increasing erythropoietin can be achieved by:

- A. Hyperbaric hyperoxia;
- B. Repeated apneas;
- C. Administering a prolonged supplement of N-acetylcysteine (NAC);
- D. A sustained exposure to decreased oxygen partial pressure;
- E. Intermittent exposure to increased oxygen partial pressure (PO₂).

Question 2. The normobaric oxygen paradox (NOP) is explained as:

- A. A rebound reaction of the ‘oxygen sensing’ system of the cell, reading the return to normal levels of oxygen as a hypoxic stimulus;
- B. Scavenging reactive oxygen species reaching hypoxic levels without hypoxia;
- C. A rebound increased amount of hypoxia-Inducible Factor-1alpha after oxygen exposure;
- D. Increased glutathione activity after oxygen exposure scavenging more reactive oxygen species;
- E. Decreased glutathione activity.

Question 3. The NOP has been reported to be unsuccessful:

- A. In some reports on healthy subjects;
- B. In some patients without NAC supplementation;
- C. In patients after cardiac surgery;
- D. When tried with too large a change in PO₂, e.g., from hypoxia to normobaric hyperoxia or from normoxia to hyperbaric hyperoxia;
- E. When applied with too small a change in PO₂

Question 4. The NOP is believed to be useful:

- A. As an adjuvant for cancer treatment;
- B. In preconditioning patients for surgery;
- C. In adding value to post-operative care;
- D. For sepsis patients;
- E. To cure dermatitis.

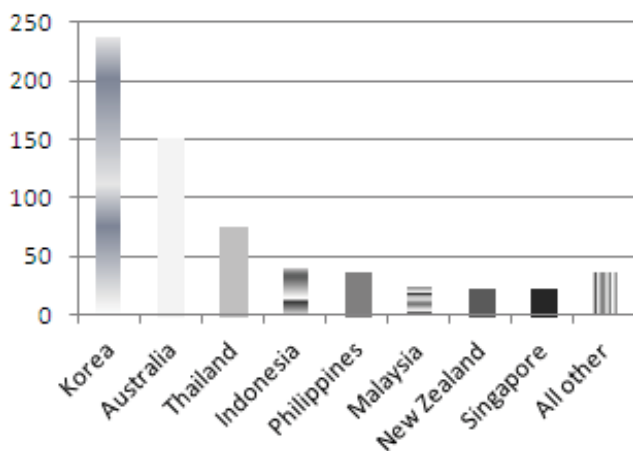
Question 5. The exact posology and administration pattern for the NOP is not fully understood. Only some limited clinical studies are available with:

- A. A patient with cancer undergoing chemotherapy;
- B. A patient with myelofibrosis;
- C. Traumatic orthopaedic patients (hip surgery);
- D. Cardiac surgery patients;
- E. Transplant surgery patients.

Provisional DAN Asia-Pacific decompression illness and diving-related fatality data for 2012

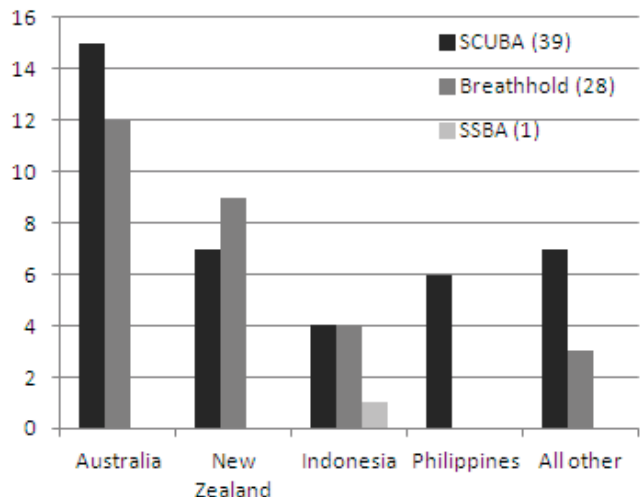
Decompression illness (DCI) in the Asia-Pacific 2011

Below are the provisional data as reported to DAN AP for the number of recreational divers treated for DCI in this region during 2011. Data from The Philippines are estimated as numbers were not provided by all chambers. Once again, the data from Korea appear to be high, the explanation for which remains unclear.



Diving-related fatalities in the Asia-Pacific in 2011

Below are the provisional data as reported to DAN AP for the number of diving-related deaths in this region during 2011. The numbers from Australia and New Zealand are probably reasonably accurate. Numbers for most other countries are probably understated because of poor reporting. Divers should notify DAN if they hear about a diving fatality so that we can better understand where and how these problems occur.





For the first time ever, the European Underwater and Baromedical Society, the South Pacific Underwater Medicine Society and the Southern African Underwater and Hyperbaric Medical Association will hold a joint 'Tricontinental' scientific meeting.

We invite you to attend a full week of science, scuba and social interaction on the exotic French island of Réunion in the Indian Ocean. The conference will be hosted by the Association Réunionnaise de Médecine Subaquatique et Hyperbare in the picturesque coastal village of St Gilles les Bains, where a range of hotel packages will be available to suit all styles and budgets. There will be possibilities for diving, whale watching, island excursions; we invite you to extend your stay before or after the meeting and to bring your family.

The meeting format will be a meld from all three societies, including discussion and workshop sessions, keynote lectures, free papers, a scientific poster session/display and industry exhibition.

Programme:

22 September	ECHM Workshop: <i>Diagnosis and treatment of mild decompression sickness</i>
23 September	ARESUB meeting
23–28 September	Tricontinental Scientific Meeting on Diving and Hyperbaric Medicine
28 September	SPUMS, EUBS, SAUHMA General Assemblies
29 September	International DAN Diver's Day

Call for Abstracts:

Abstracts for oral and poster presentations should be submitted electronically to: <www.reunion2013.org>
The Organizing Committee intends to publish all accepted abstracts in a conference proceedings and encourages speakers to submit full papers for consideration in *Diving and Hyperbaric Medicine*.

Timetable:

01 April 2013:	Deadline for submission of abstracts
01 April 2013:	End of early-bird registration period
12 July 2013:	Notification of accepted abstracts

Language: The official language for the Tri-continental Scientific Meeting, the ECHM Workshop and the International DAN Diver's Day will be English. The language for the ARESUB meeting will be French.

Full information on registration and abstract submission format may be found on the website:

<www.reunion2013.org>

All enquiries: <info@reunion2013.org>



SPUMS Notices and news

SPUMS Diploma in Diving and Hyperbaric Medicine (updated March 2013)

Requirements for candidates

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

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

In the absence of documentation otherwise, it will be assumed that the paper is submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions to Authors' – full version, July 2011, 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 (e-mail is acceptable) to advise of their intended candidacy, and to discuss the proposed subject matter of their research. A written research proposal must be submitted before commencing the research project.

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

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: <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 documented evidence of approval by an appropriate research ethics committee. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author, where there are more than one.

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

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2012, the SPUMS Academic Board consists of:

Associate Professor David Smart, Education Officer;
Associate Professor Simon Mitchell;
Associate Professor (retired) Mike Davis.

All enquiries and applications should be sent to:

Associate Professor David Smart
GPO Box 463, Hobart, Tasmania 7001
E-mail: <david.smart@dhhs.tas.gov.au>

Key words

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

Minutes of the SPUMS Executive Committee teleconference 13 August 2012

Opened: 1804 h

Present: M Bennett (MB), K Richardson (KR), D Blake (DB), S Bowen (SB), G Hawkins (GH), D Smart (DS), M Davis (MD), S Mitchell (SM), A Fock (AF)

Apologies C Acott(CA) and P Smith (PS)

1. Minutes of previous meeting

1.1 Minutes of the Executive teleconference April 2012 were approved via e-mail.

1.2 Minutes Annual General Meeting, May 2012, will be published in *Diving and Hyperbaric Medicine* (DHM) and approved at AGM 2013.

2. Matters arising from previous minutes

2.1 Diabetes dive training: MB is working with a diving instructor who wishes to challenge the Australian Diabetic Association to modify their 1992 stance on diabetes and diving. If successful, this person plans to establish a diabetes and diving training programme in Sydney area.

2.2 Epilepsy and diving: MB reports that authors of the UK position paper received censure from endocrinology journal reviewers for failing to provide an evidence base to support the stance that epilepsy was a contra-indication to diving. Fresh dialogue has ensued between members of SPUMS and EUBS and this topic suggested for formal review at a special workshop at Réunion 2013.

2.3 Trademarking of SPUMS and DHM name and logo: GH reports this would require initial outlay around AUD10,000 plus ongoing maintenance fees. Risk of not establishing trademark were noted to include malevolent takeover or deliberate misuse of our branding as per recent examples in the USA. SPUMS ExCom could not justify this expense for a perceived small risk with the SPUMS brand. The cost of AUD270 for trademarking SPUMS within Australasia was deemed a worthwhile investment.

Actions: GH to organise trademarking of SPUMS in Australia and New Zealand.

KR and MB to convey above decision to EUBS ExCom.

3. Annual Scientific Meetings

3.1 ASM 2011: S Lockley has submitted a final and comprehensive report on ASM 2011 and she is thanked for her exhaustive efforts.

3.2 ASM 2012: C Meehan has submitted a preliminary report on ASM 2012 via e-mail and notes 70 registrations with 69 attending; 60 divers and 56 full delegates with 13 associates. Overall profit will exceed AUD10,000. A formal report will be submitted in future along with complete balance sheet.

3.3 ASM 2013: KR reports the website has been launched at <http://reunion2013.org> with preliminary content. Cvent is unlikely to be used because of increased expense. Format of meeting now likely to be a blend

of full academic days and traditional SPUMS days to ease transport logistics on location. Flight arrangements remain unclear with recent airline route changes but will be addressed in coming months. Several workshop topics have been suggested. Themes and keynote speakers are being considered.

3.4 ASM 2014 and beyond: N Banham and SB have been proposed as conveners for ASM 2014 and have begun preliminary investigations in Bali. DS has visited the Krabi region of Thailand and finds it unsuitable. DS will investigate Marshall Islands noting new flight accessibility. DS and SB both support further investigation of Heron Island.

3.5 Update to Convener's Handbook not yet received from S Lockley and C Meehan.

3.6 SPUMS has been unable to establish a suitable, cost-effective mechanism for accrediting the ASM for CPD with the RACGP despite numerous negotiations. No further action will be taken on this issue. General practitioners will need to apply to their college individually for recognition of learning.

3.7 The SPUMS oxygen and first aid kits will now be stored with MB at POW Hospital and serviced or updated as necessary. Provision is made for potential future costs for this service. Each ASM convener is charged with determining if the kit is required at their event by virtue of local medical infrastructure and availability of oxygen at the dive sites. Contents of the kit will be updated as per perceived needs and in consultation with DS who holds a suggested list. The AED will not be replaced at this time.

4. Journal matters

4.1 A repeat offer from EBSCO to participate in their search engine service has been discussed between members of SPUMS, EUBS and the Journal Board and again rejected.

4.2 The budget for DHM 2012 has been presented to EUBS and formally approved by them.

4.3 MB has presented the reconciled 2011 DHM budget to EUBS and awaits a response. MD is congratulated for producing final figures in keeping with predicted budget.

4.4 EUBS agree in principal with nomination of one individual from within SPUMS to undertake oversight of the management of journal finances. PS is identified as the most likely candidate to be coopted into this position.

In future, this process will aim towards a democratic selection involving both EUBS and SPUMS members.

4.5 MD is keeping itemised spreadsheet of incidental journal expenditure and will coordinate with new Treasurer SB.

4.6 Further discussion on the future of the Journal, including appointment of the Editor for the next three years, will be held at December meeting.

5. Website update

5.1 The fully-formatted November 2011 edition of the SPUMS Purposes and Rules is on the website.

5.2 All SPUMS committee members now have a spums.

org.au e-mail address for publication in DHM.

5.3 Action: GH to add clear labels in the dropdown menus for the SPUMS Diving Medical Examination so it is easy to find and can be printed in convenient sections.

5.4 Action: GH to add the updated diving and hyperbaric medicine course information to the website as dropdown menu item under Education.

5.5 Out-of-date news and ASM items have been removed from the website, ASM 2013 information has been added.
Action: GH to update SPUMS Executive contact list and ensure all membership prices are displayed correctly.

5.6 A disclaimer has been added to the website's forum to ensure users are aware opinions are those of the individual not the Society.

5.7 A draft "*Frequently Asked Questions*" (FAQs) page has been compiled by KR.

Action: MB will distribute questions to ExCom members to provide answers for posting on the website by GH.

5.8 Action: GH will establish a presence for the future ASM Steering Committee on the website in forum style with access areas for both members and non-members.

5.9 Motion to reciprocate website links between SPUMS and the *Whatsthatfish* website as per 2012 AGM Minutes.
Action: GH to attend.

5.10 Action: GH to place ODEX logo and link on website with highest priority as part of agreement to provide SPUMS with booth space at the convention in Sydney.

5.11 Proposal: to include Diver Medical Technician course details on website to encourage website use by potential SPUMS associate members. Passed unanimously.

Action: KR to gather information and forward to GH for posting on website.

5.12 Multiple further upgrades to website content and appearance have been collated and forwarded to GH in e-mail correspondence in June 2012.

Action: GH to continue to update website.

5.13 Proposal: to use part of Webmaster honorarium to employ Nicky McNeish to effect changes as suggested is supported by the ExCom. Passed unanimously.

5.14 GH is thanked for providing SPUMS ExCom with a group e-mail address for ease of communications.

Action: GH to provide Secretary with a full list of personal e-mails included in the group mail out for inclusion in the ExCom contacts list.

5.15 Further suggestions for future additions to website will be discussed once the current list has been completed.

6. Education Officer's report

6.1 Discussion paper on international parallels and how these relate to the SPUMS diploma is still in production.

6.2 Correspondence from Professor D Gorman has highlighted inconsistencies between SPUMS Diploma requirements as published on the website and in the Journal; this has been corrected. Diplomats are no longer required to be SPUMS members of two years' standing.

6.3 SPUMS Diploma accredited two-week courses in diving and hyperbaric medicine are the Royal Adelaide Hospital course, the ANZHMG course hosted by Prince

of Wales and the Royal Australian Navy Medical Officer Underwater Medicine Course.

Action: GH to ensure these are posted clearly on the website as per the Diploma requirement documents

6.4 Consideration is being given to a system of cross recognition for training and experience between diving and hyperbaric medicine organisations internationally; however, at this time, SPUMS does not have the capability to accredit training outside of Australia and New Zealand.

6.5 KPI: SPUMS Diploma awarded to Kate Commons.

7. ANZHMG Representative's report

7.1 RCC facility accreditation will be discussed at HTNA.

7.2 Despite further meetings with representatives of MSAC and submission of further evidence, the decision to cut funding to non-diabetic wounds remains unchanged. DS is drafting a complaint to the Ombudsman and will consider media and lobby group campaigns. Loss of funding in three months' time has implications to the financial viability of hyperbaric units.

7.3 Action: DS to create journal and website announcement aiming at gathering data on negative outcomes subsequent to Safework legislation changes.

7.4 MD has corresponded with NZ authorities re standardising 4774.2 for use for New Zealand RCC accreditation process, but received no responses.

7.5 DS has been nominated as SPUMS representative to sit on the occupational diving section of Australian Standards Committee SF-017.

8. Treasurer's report

8.1 Handover of Treasurer role is nearly completed.

8.2 Proposal: To appoint Mediq Financial Services to take over book-keeping and auditing roles from Barrett Baxter Bye. Proposed M Bennett, seconded D Smart, passed unanimously.

8.3 KPI satisfied: timely response to counter-signing.

9. Public Officer's report

9.1 A handover guide is being produced for the next Public Officer.

9.2 Need for notification of change of date for 2013 AGM to Public Office is noted.

9.3 KPI satisfied: relevant documents lodged with authorities before due date.

10. Secretary's report

10.1 SPUMS Administrator is attending database clean as time allows and reports no new issues.

10.2 The Asset List continues to be updated.

10.3 Action: GH to provide direct access to SPUMS database for Secretary.

10.4 KPI satisfied: timely generation of Minutes.

11. Membership report

11.1 Current membership numbers are Full 449, Associate 46, Student 0, Retired 14, Corporate 6. DB is congratulated for recruiting so many enthusiastic new

members from Townsville Hospital.

11.2 Further initiatives to combat falling membership numbers will be discussed at meeting in December. These include targeting specific groups: corporate, associate, student and active website members plus provision of new category for institutional subscribers to DHM.

11.3 KR has been granted access to the EUBS members-only webpages.

Action: GH will give reciprocal access to SPUMS members-only web pages to P Germonpré and C Balastera with a view to exploring a combined membership category to allow access to each other's websites.

11.4 SPUMS notes that due to the nature of our website, we do not have the capability currently to administer global membership for EUBS members. This will be discussed further at next ExCom.

11.5 SPUMS will be represented at ODEX in Sydney, 08–09 September; it will not have an official presence at the ACEM in Hobart in November because of prohibitive cost. Space is being sought at ASA in late 2012 and ANZCA in 2013.

11.6 Proposal: for SPUMS to join the Diving Industry Association of Australia as an Associate Member with KR as representative. Proposed M Bennett, seconded K Richardson, passed unanimously.

12. Other business

12.1 MB will liaise with HTNA representative in Christchurch about increased relationship with SPUMS.

12.2 MB formally welcomes D Blake, S Mitchell and S Bowen to the Executive Committee.

13. Correspondence

D Gorman to D Smart as above, nil further.

14. Next meeting

Dept of Diving and Hyperbaric Medicine, Prince of Wales Hospital, 01–02 December 2012

Closed: 2030 h

Karen Richardson
Honorary Secretary

Key words

Medical society, meetings

SPUMS website news

Glen Hawkins resigned in December as Webmaster. The Committee wishes to express its warmest appreciation to Glen for all his hard work in establishing and maintaining the site; it has not been an easy task.

Joel Hissink has been coopted onto committee as the new Webmaster.

Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) Chairman's Report November 2012

1. Medical Services Advisory Committee (MSAC)

1.1 In April, the Federal Minister for Health and Aging released with the budget papers a statement that Medicare funding would be withdrawn from hyperbaric oxygen treatment of non-diabetic problem wounds and ulcers. This represents a low point in a 13-year fight by ANZHMG, with assistance from the AHHA and ASA, to continue the funding. The last six months has seen a hectic period of lobbying; unfortunately on all occasions, the DOHA has retreated into their comfort zone, stating that the science was not of sufficient quality. They did not address three fundamental issues:

1. that MSAC has withdrawn funding for a funded technology without appropriate process (i.e., the evidence for other treatments of non-diabetic problem wounds was not evaluated);
2. MSAC has no independent appeals process (because it was set up to evaluate new technology, not existing technology);
3. the ANZHMG wound study which was requested as a result of the 2004 MSAC 1054 report was ignored and dismissed by MSAC, when MSAC actually had requested the data be collected.

1.2 Details regarding MSAC 1054.1 and the dissenting report from Associate Professors David Smart and Michael Bennett may be found at: <<http://www.msac.gov.au/internet/msac/publishing.nsf/Content/1054.1>>.

1.3 As a result of the concerns regarding the MSAC process, a senate inquiry was held on 12 November 2012, which provided opportunity to submit concerns regarding the MSAC process and HBOT.

The submissions to that inquiry can be found at: <http://www.aph.gov.au/Parliamentary_Business/Committees/Senate/Committees?url=fapa_ctte/hbot/submissions.htm>.

The ANZHMG submission by Professors Smart and Bennett may be obtained from the SPUMS Secretary at: <secretary@spums.org.au>.

2. Australian and New Zealand Standards

2.1 Committee SF017 – ANZ Standards 2815 series and 2299.1

Standards Australia has suddenly become busy with respect to Australian and New Zealand Standards. Committee SF017 has convened to review the Occupational Diver Training Standards 2815.1–6, and also the Operational Diving Standard 2299.1, which form the platform for most professional diving. The reviews were hosted by ADAS. The Committee met on 13–14 November 2012, and commenced review of AS 2815.2, 5 and 6. Parts 2 and 6 were approved for public comment and were released in December. Unfortunately I was unable to attend that meeting. A further meeting is planned in February to

cover AS2299.1. The proposed timetables for public comment, review and approval are such that AS 2815 series will be released in September 2013 and AS 2299.1 in December 2013.

2.2 ANZ Standard 4774.2

This is hosted by the Hyperbaric Technicians and Nurses Association, and is due to commence review in 2013. It is desperately needed because there is no reference to hyperbaric chamber staff in the Federal Government 2011 Work Health and Safety Legislation. The standard will also be used as a template for facility accreditation.

Associate Professor David Smart

Hon. Chairman, Australian and New Zealand Hyperbaric Medicine Group

E-mail: <david.smart@dhhs.tas.gov.au>

Key words

Medical society, meetings, hyperbaric medicine, policy, standards, occupational diving

Education Officer's Report, November 2012

SPUMS Diplomas

I offer congratulations to Dr Kate Commons, who completed her project entitled:

A prospective analysis of independent patient risk factors for middle ear barotrauma (MEBT) in a multiplace hyperbaric chamber

There has been a resurgence of interest in the SPUMS Diploma with nine doctors registering projects in 2012. Topics are many and varied and it is exciting to see a new generation of diving and hyperbaric medicine physicians emerging.

Associate Professor David Smart

Education Officer

E-mail: <david.smart@dhhs.tas.gov.au>

Key words

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

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

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

Eligibility criteria are:

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

2 Completion of training courses in Diving Medicine and in Hyperbaric Medicine of at least four weeks' total duration.

For example, one of:

a ANZHMG course at Prince of Wales Hospital Sydney, **and** Royal Adelaide Hospital or HMAS Penguin diving medical officers course **OR**

b Auckland University Postgraduate Diploma in Medical Science: Diving and Hyperbaric Medicine.

3 **EITHER:**

a Completion of the Diploma of the South Pacific Underwater Medicine Society, including six months' fulltime equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS **AND**

b Completion of a further 6 months' full-time equivalent clinical experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.

OR:

c Completion of 12 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA **AND**

d Completion of a formal project in accordance with ANZCA Professional Document TE11 "Formal Project Guidelines". The formal project must be constructed around a topic which is relevant to the practice of diving and hyperbaric medicine, and must be approved by the ANZCA Assessor prior to commencement.

4 Completion of a workbook documenting the details of clinical exposure attained during the training period.

5 Candidates who do not hold an Australian or New Zealand specialist qualification in Anaesthesia, Intensive Care or Emergency Medicine are required to demonstrate airway skills competency as specified by ANZCA in the document "Airway skills requirement for training in Diving and Hyperbaric Medicine".

All details are available on the ANZCA website at:

<<http://anzca.edu.au/edutaining/DHM/index.htm>>

Dr Suzy Szekely, FANZCA

Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine, Australia

E-mail: <Suzy.Szekely@health.sa.gov.au>

John Pennefather: 40 years' service at the Submarine and Underwater Medicine Unit

The Submarine and Underwater Medicine Unit (SUMU) Scientific Officer, Mr John Pennefather, BSc (Hons), has recently completed 40 years' service to the Royal Australian Navy (RAN). Mr Pennefather began working at SUMU (then known as the School of Underwater Medicine) in 1972. As a young physiologist with an interest in animal climate physiology, and having studied the oxygen consumption of grazing sheep, he sent a letter addressed to the "The RAN, Canberra" outlining his design idea for a closed-circuit rebreather diving set. The letter reached SUMU and John was invited to the unit to discuss his ideas. The discussion evidently went well as John was subsequently offered a job as the SUMU Scientific Officer.

Over his 40 years' service, John has been involved in countless projects and areas of research in an effort to improve safety within the diving and submariner communities. One of his first projects was working on a new oxygen diving set design in the mid-1970s. He also conducted research that found that clearance diver trainees were burning more energy than they received, leading to increased food rations and more sleep while on the training course. John is co-author of a highly regarded diving medicine textbook, *Diving and Subaquatic Medicine*, which is now in its fourth edition and regularly consulted by diving physicians across the globe.¹ He has helped to study the effect of bubbles on the brains of rabbits (which may explain the current rabbit plague at *HMAS Penguin*), worked tirelessly on the complex problems of submarine escape and rescue, as well as the maintenance of a healthy submarine atmosphere. John has investigated hundreds of military and civilian diving accidents and has provided design advice on diving- and hyperbaric-related equipment ranging from portable two-man recompression chambers to methods to keep live fish fresh during transit.

John has established a unique and extensive collection of scientific papers at SUMU. He has also represented SUMU and the RAN on many occasions while travelling overseas to diving and submarine medicine scientific meetings, where he often presented his research. Every RAN diver, medical officer and underwater medic has been fortunate to have crossed paths with John at some point during their careers. He has taught divers about physiology, subjected them to experiments in his laboratory and has educated every medical officer and underwater medic in various aspects of underwater medicine.

Perhaps it is John's enjoyment of his work combined with his habit of frequent walks along Balmoral Beach or lunchtime swims in the *Penguin* pool over summer that have kept him in good health over the years. John intends retiring in 2013 but is eager to continue to provide expert advice to the RAN whilst in retirement. It is likely that such advice will be

John Pennefather, BSc (Hons), retires

The Scientific Officer, SUMU, being presented a commemorative plaque and certificate by Rear Admiral Robyn Walker, Commander Joint Health / Surgeon-General Australian Defence Force, to recognise his 40 years' service for the Royal Australian Navy (photo taken by Warrant Officer J O'Brien)



sought frequently, as his replacement will be difficult to find. John is held in very high regard among diving and submarine medical communities all over the world and continues to be asked for his opinion on a diverse range of underwater medicine topics. John's 40 years' service and dedication to the continuing improvement of diver and submariner safety is unparalleled and is an admirable achievement.

Reference

- 1 Edmonds C, Lowry C, Pennefather J, Walker R. *Diving and subaquatic medicine*, 4th ed. London: Hodder Arnold; 2002.

Joel Hissink, Medical Officer-in-Charge, SUMU

Key words

Military diving, diving research, research, general interest

This is an edited version of an article in *Navy News*

The

SPUMS

website is at

<www.spums.org.au>

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



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EUBS website news, January 2013

Dear EUBS member,

Please find below some important messages from the EUBS Executive Committee.

Reunion 2013

Registration and Abstract submission are now open, so please visit the REUNION 2013 website <<http://reunion2013.org>> to make sure you will be part of it!

Publication news

We have an official Facebook page that will help keep you keep instantly updated with news and events related to diving and hyperbaric medicine – please have a look: <<https://www.facebook.com/pages/European-Underwater-and-Baromedical-Society/283981285037017>>.

The Discussion Forum on the website is accessible to all members of EUBS directly from the homepage: <<http://www.eubs.org/members/forum>>. Please participate!

Membership news

Those members who had not renewed their membership by 15 Sept 2012 may experience problems accessing their membership pages. If so, please send us an e-mail, and we will be happy to restore your account!

Visit your EUBS website <<http://eubs.org>> for all society news and updates.

Sincerely yours,

EUBS ExCom



website is at
<www.eubs.org>

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

EUBS Annual Scientific Meeting 2014 Preliminary Announcement

Dates: 24–27 September 2014

Venue: Wiesbaden, Germany

The 40th EUBS Annual Scientific Meeting will be held in conjunction with the 2014 Congress of the German Society for Diving and Hyperbaric Medicine (GTUeM) in Wiesbaden, Germany (near Frankfurt/Main). Dr Peter Müller has been appointed by both societies to serve as the Secretary General for the EUBS ASM 2014.

For further information at this early stage see:

<www.eubs2014.org>

Enquiries: <peter.mueller@eubs.org>

Belgian Medical Accreditation Committee

It is a pleasure to announce that the Belgian Medical Accreditation Committee has recognised participation in the 2012 EUBS ASM in Belgrade as a CME activity for 9.5 credit points.

This CME is valid for all specialties in Belgium and may be useful for other countries as well. The necessary accreditation information will be posted on the EUBS website.

German Society for Diving and Hyperbaric Medicine (GTUeM)

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

Royal Adelaide Hospital Hyperbaric Medicine Unit Courses 2013

July/August

Wk 1: 29 July–02 August

Wk 2: 05–09 August (lecture week)

Wk 3: 12–16 August

Refresher Course – September/October

Wk 1: 23–27 September

Wk 2: 30 September–04 October

Medical Officers Course

Wk 1: 02–06 December

Wk 2: 09–13 December

All enquiries to:

Lorna Mirabelli

Senior Administrative Asst/Course Administrator

Hyperbaric Medicine Unit

Level 2, Theatre Block

Royal Adelaide Hospital

North Terrace, Adelaide, SA 5000

Phone: +61-(0)8-08-8222-5116

Fax: +61-(0)8-8232-4207

E-mail: <Lorna.Mirabelli@health.sa.gov.au>

Royal Australian Navy Medical Officers' Underwater Medicine Course 2013

Dates: 11–22 November 2013

Venue: HMAS PENGUIN, Sydney

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

Cost: AUD705 without accommodation
(AUD1,600 with accommodation at HMAS Penguin)

For information and application forms contact:

Rajeev Karekar, for Officer in Charge,
Submarine and Underwater Medicine Unit

HMAS PENGUIN

Middle Head Rd, Mosman

NSW 2088, Australia

Phone: +61-(0)2-9647 5572

Fax: +61-(0)2-9960 4435

E-mail: <Rajeev.Karekar@defence.gov.au>

Australian and New Zealand College of Anaesthetists 2013 Annual Scientific Meeting

Workshop (W35)

An introduction to diving and hyperbaric
medicine

Date/Time: Tuesday 07 May 2013, 1530–1700 h

Location: The Alfred Hospital

Cost: \$25

Maximum participants: 30

This workshop will provide an insight into aspects of diving and hyperbaric medicine of interest to anaesthetists. It will include a display and discussion of commercial diving equipment and a tour of the new Therapeutic Goods Administration (TGA)-approved Alfred hyperbaric chamber.

Facilitator: Dr Andrew Fock

Andrew Fock is head of Diving and Hyperbaric Medicine at The Alfred Hospital, Melbourne. He also works as a cardiac anaesthetist in private practice as well as with the Royal Australian Navy as a naval reserve officer with the Submarine and Underwater Medicine Unit.

If you have any queries, please contact the SIG Coordinator, Lana Lachyani at:

<llachyani@anzca.edu.au>

Advertising in *Diving and Hyperbaric Medicine*

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

Details of advertising rates and formatting requirements are available on request from:

E-mail: <editor@dhmjournal.com>

Fax: +64-(0)3-329-6810



**DIVING HISTORICAL
SOCIETY
AUSTRALIA, SE ASIA**

P O Box 347, Dingley Village
Victoria, 3172, Australia

E-mail:
<deswill@dingley.net>

Website:
<www.classicdiver.org>

Hyperbaric Oxygen, Karolinska

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

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

We offer video lectures from:

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

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

For further information contact:

Folke Lind, MD PhD,

E-mail: <folke.lind@karolinska.se>

Website: <www.hyperbaricoxygen.se>

Asian Hyperbaric and Diving Medical Association (AHDMA) IXth Annual Meeting 2013

Dates: 02–06 July 2013

Venue: Singapore

Pre-conference workshop: 02–04 July

Medical support of diving operations

Main Conference: 05–06 July

Guest Speaker: Professor Alf Brubakk, Professor of Environmental Physiology, Norwegian University of Science and Technology is the guest speaker and will conduct the workshop.

The event promises rich learning opportunities and a sharing platform with a wide spectrum of physicians from all over the world on all aspects of scientific information on diving and hyperbaric medicine.

For further information:

E-mail: <tarun.sahni@adventhcg.com>

Website: <www.ahdma.org>

Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organized more than 100 courses over the past few years, both in the Netherlands and abroad. Below is a list of courses planned for 2013.



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

Course details for 2013

06 and 12 April: Basic course in diving medicine Part 1 (Loosdrecht NL)

13, 19 and 20 April: Basic course in diving medicine Part 2 (AMC Amsterdam NL)

25 May–01 June: Basic course in diving medicine Part 2 (Oman)

June (dates TBA): 21st in-depth course “*Challenges in diving medicine*” (NL)

09–16 November: Basic course in diving medicine Part 1 (to be decided)

16–23 November: 21st in-depth course in diving medicine (to be decided)

23–30 November: 21st in-depth course in diving medicine (to be decided)

For further information: <www.scotthaldane.nl>

Undersea and Hyperbaric Medical Society 46th Annual Scientific Meeting

Dates: 13–15 June 2013

Venue: Lowes Royal Pacific Resort at Universal Studios, Orlando, Florida, USA

Phone: +1-(0)877-533-UHMS (8467)

E-mail: <lisa@uhms.org>

Website: <www.uhms.org>

DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.

It can be accessed at: <<http://www.daneurope.org/web/guest/>>.

Keeping the whole DAN Europe family updated with what is going on...Enjoy!

Instructions to authors

(Short version, updated December 2012)

Diving and Hyperbaric Medicine (DHM) welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *DHM*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing. An accompanying letter signed by all authors should be sent.

Contributions should be sent to:

E-mail: <submissions@dhmjournal.com>

Individual correspondence should be addressed to:

E-mail: <editor@dhmjournal.com>

Requirements for manuscripts

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

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

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

Illustrations, figures and tables must NOT be embedded in the word processor document, only their position in the text indicated, and each should be submitted as a separate file. **Tables** should be presented either with tab-separated columns (preferred) or in table format. No gridlines, borders

or shading are to be used.

Illustrations and figures should be submitted in TIFF, high resolution JPG or BMP format. If figures are created in Excel, submit the complete Excel file.

Photographs should be glossy, black-and-white or colour. Colour is available only if essential and will be at the authors' expense. Indicate magnification for photomicrographs.

References

The *DHM* Journal reference style is based closely on the *International Committee of Medical Journal Editors (ICMJE) Uniform Requirements for Manuscripts*. To ensure the correct formatting of all types of citation go to:

<http://www.nlm.nih.gov/bsd/uniform_requirements.html>

(last updated July 2011). References must appear in the text as superscript numbers at the end of the sentence after the full stop.^{1,2} Number them in order of quoting (including in tables). Use Index Medicus abbreviations for journal names: <<http://www.nlm.nih.gov/tsd/serials/lji.html>>

Examples of the exact format for a standard paper and a book are given below:

- 1 Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol.* 1972;95:556-63.
- 2 Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 431-59.

Accuracy of references is the responsibility of the authors.

Manuscripts not complying with the above requirements will be returned to the author(s) before consideration.

Consent and ethics

Studies on human subjects must state that they comply with the Declaration of Helsinki (1964, revised 2008) and those using animals must comply with health and medical research council guidelines or their national equivalent. A statement affirming ethics committee (institutional review board) approval should be included in the text. A copy of that approval (and consent forms) should be provided.

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Full instructions to authors (revised July 2011) may be found on the DHM Journal, EUBS and SPUMS websites and must be consulted in preparing a submission.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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+61-8-8212-9242 (International)

SOUTHERN AFRICA

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

NEW ZEALAND

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

EUROPE

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

ASIA

+852-3611-7326 (China)
+10-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

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. Information, all of which is treated as being confidential in regard to identifying details, is utilised in reports on fatal and non-fatal cases. Such reports can be used by interested people or organisations to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence unless otherwise agreed) to:

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

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>

DISCLAIMER

All opinions expressed in this publication are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policies or views of SPUMS or EUBS or the Editor.

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