Diving and Hyperbaric Medicine

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EUBS

Getting the sick diver to a recompression chamber

Running low on gas – a surprise to many divers

Hypoxia after HBOT in ventilated ICU patients

New technology for gas monitoring in CCRs

Pulmonary stress from breath-hold diving

2006: Fewer diving deaths in Australia

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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

A fatality documented in the 2006 Australian scuba fatalities (SC 06/03) illustrates graphically how a series of events and factors lead to a tragic outcome.[1] In teaching about diving safety, I have used the Swiss cheese analogy since the mid-1970s. Imagine a block of Swiss cheese with its characteristic holes. For a serious diving accident to occur, one must breach through from one side of the block to the other. The thickness of the block represents the 'flexibility' of the system, i.e., 'safety in depth' – personnel, training and experience, equipment, built-in redundancy in safety procedures, diving environment, support systems, etc. The thinner the block the fewer things need to go wrong for a serious breach of safety to occur. The holes in the cheese are individual breaches of safety procedures or unexpected events. Individual holes may not lead to an accident, but with increasing breaches – either in number (more holes) or severity (larger holes) – the block may be breached through: a life-threatening or fatal incident. Sometimes known sequences of events lead more rapidly to serious accidents; here the holes in the cheese line up from one side of the block to the other.

A more practical approach is 'root cause analysis' (RCA), as used by Lippmann et al.[1] Many different techniques of RCA are described in the literature and summarised usefully in Wikipedia.[2] Without knowing the root cause, the chief goal of this type of analysis – preventing similar accidents by appropriate corrective action(s) – cannot be achieved. One of the simplest structures for conducting this analysis is to consider issues related to people, policies, procedures and equipment. In the case of SC 06/03 deficiencies in all four areas are identifiable, and this tragedy exemplifies that there are often several root causes of a problem or event. In this case, many factors preceded the 'trigger' of entrapment leading to running out of air, as listed in Table 3 (page 82): victim inexperience and lack of familiarity with, failure to check pre-dive and inability to correctly operate his equipment; poor dive planning (failure to identify underwater hazards the lifelines might snag on, and defective equipment – an empty lift-bag tank); bad decision making and poor control of the dive by the supervisor; and poor dive practices, such as poor tethered diving technique to allow entrapment (on the part of both the victim and his surface attendant) and failure to have a standby diver kitted and ready to enter the water. The block of cheese was indeed full of holes.

Another issue raised by the 2006 and by previous Australian reports is the question of the adequacy of supervision of the Great Barrier Reef (GBR) snorkelling experience. Far too often in these reports there appears to be a failure on the part of designated safety lookouts to identify swimmers in trouble (BH 06/05 and BH 06/06) or even to realise that someone is missing (BH 06/03). Often, large numbers of inexperienced and sometimes elderly snorkellers are watched by only a few staff and there is not always a means, such as a small tender deployed in the designated snorkelling area, of rapidly reaching the victim when a problem is identified. This results in delay to effective resuscitation, which inevitably reduces the likelihood of its success. Whether or not some of these cardiac deaths were inevitable, it seems to this writer that fresh ideas are needed to help reduce the incidence of injury on the GBR.

In a preliminary study on the impact of prolonged breath-hold and depth (pressure) on pulmonary diffusing capacity of carbon monoxide (DLCO) and nitrous oxide (DLNO), Garbella et al suggest that they have demonstrated evidence of what they term alveolar-capillary membrane "distress".[3] The lung has only a limited range of responses to hydrostatic or other injury, with initial capillary leak leading to increased extravascular lung water. This, in turn, may be followed by transudation of fluid (with a variable protein content) into the alveoli (pulmonary oedema), loss of surfactant, cellular infiltration and, if unresolved, acute respiratory distress syndrome. Extravascular lung water is currently measured using thermal or osmotic dilution techniques,[4] so the simultaneous measurement of DLCO and DLNO is a surrogate for this. In the healthy lung, lymphatic function is highly dynamic and increased lung water is rapidly mobilised. It may well be that what has been observed here can be explained on a physiological basis of increased pulmonary blood volume and lung compression due to pressure leading to a transient increase in interstitial lung water, which is then rapidly mobilised by the pulmonary lymphatics after the dive. It will be most interesting to see where this line of investigation leads.

References

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2 Root cause analysis. *Wikipedia*, the free encyclopedia. Available from: <http://en.wikipedia.org/wiki/Root_cause_analysis>.

3 Garbella E, Piarulli A, Fornai E, Pingitore A, Prediletto R. Preliminary observations on the effect of hypoxic and hyperbaric stress on pulmonary gas exchange in breath-hold divers. *Diving and Hyperbaric Medicine*. 2011;41(2):97-100.

4 Matthay MA. Measurement of extravascular lung water in patients with pulmonary oedema. *Am J Physiol Lung Mol Physiol*. 2008;294:L1021-2.

Michael Davis

The front page photo shows the helicopter transfer of a diving emergency from the Scottish Outer Hebrides being received at the Dunstaffnage hyperbaric facility, near Oban. Morvern Hills and the Isle of Lismore in the background.

The President's page

Peter Germonpré
President, EUBS

Dear Friends,

Or should I say, dear fellow eco-systems? Whilst we are concentrating on diving physiology and hyperbaric medicine, entire research labs have been studying the human gut microbial population, a most interesting topic to dig into (well, maybe not literally). The latest research published in the field suggests that, worldwide, the human species is characterised by perhaps as few as three distinctive microbial "enterotypes".[1] Independent from nationality, race, age, sex, or dietary culture, each of us seems to be inhabited by one of three mixtures of intestinal bacterial colonies – about 100,000 billion of the little bug(...)s.

Many of these bacteria survive only inside the gut, so it has been virtually impossible to study them. Using a novel detection technique, nicknamed "metagenomics", it is no longer necessary to grow these bacteria in a Petri dish in order to characterise them. Now, it is possible to map out the entire genome of a mixed sample of intestinal contents. Using this technique, German and Belgian scientists have investigated subjects from Japanese, American and European populations. Based on their microbiotic content, all subjects could be stratified into three groups, according to the dominant bacterial species in the mix: *Ruminococcus, Bacteroides* or *Prevotella spp*. Each of these enterotypes has unique properties, in that they seem to employ different techniques to extract nutrients, produce different types and quantities of vitamins and interact differently with their neighbouring bacteria. What exactly determines which enterotype one human possesses is not (yet) known, but there seems to be a genetic predisposition that dates back more than 120,000 years, the era of geographic separation and thus racial divergence of *Homo sapiens*.

To the imaginative observer, the implications of this sort of research may well turn out to be enormous. One must look upon the human body as being one huge (mobile) eco-system, where environmental changes act upon the health and behaviour of our inhabitants, and they, in turn, exert influence on distant body parts. In case of decreased density of some tribes of bacteria, others may start to dominate and cause profound perturbations in our guts (no need to go into more details). When dramatic environmental changes occur, entire populations may be wiped out or decline and only recover with great difficulty.

Maintaining a proper equilibrium of our internal environment seems to be of utmost importance; already there are data indicating that deviations in gut colonisation may be associated with obesity, asthma, intestinal cancer and Crohn's disease. More audacious research even suggests that diseases such as autism may be related to gut flora perturbations. In mice, modification of the intestinal microbiotics during early life is able to induce changes in cerebral development and maturation, resulting in different personality traits and even pathological psycho-syndromes. This goes way beyond the small everyday nuisances provoked by our intestines!

Just as our intestinal microflora can be a source of great concern to its host organism, we humans should be concerned about the influence of our behaviour on our own host organism – planet Earth. Whereas a sigmoid tribe of *Rhodospirillum* bacteria probably has no clue as to its effects on the human brain or liver function, we should be able to conceive that some of our human activity may be noxious to the planet in indirect and less-than-obvious ways. We can no longer pretend that we exert influence on our local environment only – the allegory of the Brazilian butterfly's wings setting off a Texas tornado perhaps holds more truth than we might think.[2]

One environment that suffers dearly from our ignorant pollution and destruction is the ocean, which makes up 71% of the surface of the Earth. An organisation that is making an effort to reduce our patterns of ignorant destruction is the Save Our Seas Foundation, <www.saveourseas.com>. Committed to protecting our oceans by funding research, education, awareness and conservation projects focusing on the major threats to the marine environment, you will see they are probably much closer to your interests than the above story. Their very active blog and article database provides for hours of interesting reading, and fun in places (I have to admit I fell for the Great White Shark Tribute song <http://youtube.com/watch?v=xGhjcz9WFEc>). If you do anything this week, please do yourself a favour and visit the Foundation's website.

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Key words
Medical society, general interest

Original articles

Changes in oxygenation in mechanically ventilated critically ill patients following hyperbaric treatment

Gordon Bingham, Ian Millar, Susan Koch, Eldho Paul, Dinesh Varma and David Pilcher

Key words
Hyperbaric oxygen therapy, ventilators, pulmonary function, physiology, right-to-left shunt, clinical audit

Abstract
(Bingham G, Millar I, Koch S, Paul E, Varma D, Pilcher D. Changes in oxygenation in mechanically ventilated critically ill patients following hyperbaric treatment. *Diving and Hyperbaric Medicine*. 2011;41(2):59-63.)
Background: Some ventilated intensive care unit (ICU) patients may experience reduced oxygenation following hyperbaric oxygen treatment (HBOT).
Methods: In a prospective, single-centre, observational study, we documented changes in oxygenation and the need for associated changes in ventilator settings in 25 consecutive, mechanically ventilated ICU patients immediately post-treatment and 1, 2, 3 and 6 hours following 61 HBOT sessions. The primary outcome measure of oxygenation was the ratio of arterial partial pressure of oxygen (P_aO_2) against the level of inspired oxygen (F_iO_2), P_aO_2/F_iO_2.
Results: Following HBOT, the P_aO_2/F_iO_2 ratio decreased by 27% on return to ICU ($P < 0.001$, 95% confidence intervals (CI) 20.6 to 34.2); 22% at 1 hour post-HBOT ($P < 0.001$, 95% CI 15.1 to 28.6); and 8% at 2 hours post ($P = 0.03$, 95% CI 0.8 to 14.4). The ratio showed no significant differences from pre-HBOT at 3 and 6 hours post-HBOT. P_aO_2/F_iO_2 ratio changes necessitated adjustments to ventilation parameters upon return to ICU following 30 of 61 HBOT sessions in 17 out of the 25 patients. The most common ventilation parameter altered was F_iO_2 ($n = 20$), increased by a mean of +0.17 (95% CI 0.11 to 0.23) above baseline for two hours following HBOT.
Conclusions: Following HBOT, oxygenation is reduced in a majority of mechanically ventilated ICU patients and requires temporary alterations to mechanical ventilation settings. Further study to identify predictive characteristics and to determine causation for those at risk of needing ventilation alterations is required.

Introduction

Hyperbaric oxygen therapy (HBOT) is recognised for the treatment of a variety of conditions for which recipients may require concurrent mechanical ventilation.[1] Subsequent to HBOT, oxygen requirements in some mechanically ventilated patients increase.[2] However, published data on this phenomenon remain limited.[2–4] Possible mechanisms for the transient reduction in arterial oxygenation, which in some patients may result in hypoxaemia following HBOT, are listed in Table 1.[2–6]

Our aim was to document the incidence and magnitude of changes in oxygenation following HBOT utilising the arterial partial pressure of oxygen against the fractional inspired oxygen ratio (P_aO_2/F_iO_2) as the primary outcome measure, and secondarily to look at changes in ventilator settings necessitated post-HBOT to maintain stable oxygenation.[7]

Methods

SUBJECTS

A prospective, single-centre, observational study of 25 consecutive, mechanically ventilated intensive care unit (ICU) patients referred to the Alfred Hospital hyperbaric unit for HBOT from November 2007 to November 2008 was undertaken. This observational study involved only analysis of information routinely collected for clinical care. The institutional ethics committee approval received did not, therefore, stipulate additional informed consent from either the patient or their family.

Although patients received multiple HBOT sessions, data

Table 1
Possible mechanisms for transient hypoxaemia following HBOT

- Increased pulmonary venous admixture (described in both healthy and critically ill patients)
- Blunting of the hypoxic pulmonary vaso-constrictive response
- Hyperoxia-induced atelectasis
- Worsening oxygen efficiency due to changes in patient position
- Changes to vasoactive drug therapies
- Inadequate mechanical ventilation
- Loss of positive end-expiratory pressure or pressure support during changeover of mechanical ventilators for transportation

were gathered from between two to three sessions per patient, typically HBOT session 1, 2 and/or 3. These sessions were chosen based on previous studies, and patient, researcher and recording equipment availability. Data were collected from 61 (36%) of a total of 171 HBOT sessions delivered to the 25 patients.

PROCEDURES

Patients were mechanically ventilated in the ICU by a Puritan Bennett 840 ventilator (Tyco Healthcare, Pleasanton, CA); during transport to and from the hyperbaric unit, either the PB840 ventilator or a Draeger Oxylog 3000 (Draeger Corp, Lübeck, Germany) matched to the ICU ventilator settings was used. Mechanical ventilation during HBOT was achieved with a Servo 900C ventilator (Siemens Corp, Lübeck, Germany). Mechanical ventilation data from the PB840 ventilator pre- and post-HBOT were collected electronically on a computer (IBM Thinkpad™ T30, Ormonk, NY), which queried the ventilator at one-minute intervals using a serial port data management programme (Advanced Serial Data Logger, Version 3.1.0 build 18, AGG Software).

Prior to commencing transport, the transport mechanical ventilation settings were adjusted to match ICU ventilation parameters or adjusted to achieve target end-tidal CO_2 or pulse oximetry (S_pO_2) values. During the brief changeover between ICU and transport ventilators, positive end-expiratory pressure (PEEP) was not maintained or any recruitment manoeuvre applied to the patient. F_iO_2 was increased to 1.0, as per the hospital transport ventilation protocol.[8] During transportation, monitoring of ventilation data was not possible. Therefore, the transportation phase of the treatment was observed by the principal researcher to detect and document any episodes of hypoxaemia and elucidate possible causes.

The same ventilator settings as in ICU were used on commencement of HBOT, then adjusted to compensate for the effect of raised ambient pressure upon the ventilator as described in the literature.[9–11] In most patients, this involved an increase in tidal volume of about 10% and, in some, a slight increase in ventilation rate (mean rate during HBOT 18 breaths per min, compared to 17 per min pre-HBOT). These settings were returned to the baseline ICU levels by the end of HBOT.

Arterial blood gas (ABG) samples were drawn from an in-dwelling arterial catheter using a standard aseptic procedure prior to and during HBOT, on return to ICU and at 1, 2, 3 and 6 hours post-HBOT (range of variation in sampling times was 5 minutes early to 6 minutes late). The F_iO_2 being delivered was recorded, along with other physiological and clinical parameters. The blood gas analyser (RapidLab 1265; Bayer HealthCare AG, Leverkusen, Germany) was automatically single-point calibrated every 4 hours and two-point calibrated every 8 hours as per the manufacturer's guidelines. Of the P_aO_2 values reported here, none were taken during HBOT, when the values were outside the range of the ABG analyser. From the 61 measured HBOT sessions, only one of 244 possible sampling times was missed.

To minimise the chance of alterations in oxygenation incurred as a consequence of patient position changes during transport (with associated changes of sedation, vasoactive drug infusion rates, or mechanical ventilation settings) patients remained on their normal ICU bed throughout. Any changes in ICU ventilator settings required after HBOT to achieve pre-HBOT targets for P_aO_2 and/or S_aO_2 were recorded.

HBOT commenced with an approximate pressurisation rate of 10 kPa min^{-1} and reached a maximum treatment pressure of 284 kPa, held for 60 minutes. A F_iO_2 of 1.0 was delivered throughout, interrupted by two, 5-minute 'air breaks' after 25 and 55 minutes, during which the F_iO_2 was reduced to 0.21. A stepped decompression was then conducted over 35 minutes with a F_iO_2 of 1.0. Three of the 61 HBOT sessions were given at 243 kPa for 99 minutes and one at 203 kPa for 105 minutes, both with a single 5-minute air break.

STATISTICAL ANALYSIS

Power analysis *a priori* determined that 25 patients would provide an 80% power to detect a difference between any two time points equivalent to 80% of one standard deviation (SD), equivalent to an approximate reduction in P_aO_2/F_iO_2 ratio of 19%, with a two-sided *P*-value of 0.05. Changes in the P_aO_2/F_iO_2 ratio from baseline approximated a normal distribution. Analyses were performed using statistical analysis software (SAS) version 9.1 (SAS Institute Inc., Cary, NC, USA). In order to assess the effect of time point on change in P_aO_2/F_iO_2 ratio, a mixed-effects analysis was performed using the PROC Mixed procedure in SAS.

Time was treated as a categorical variable to facilitate specific comparisons and data were analysed using a true intention-to-treat analysis for all patients. Results from the mixed-effects model are presented as percentage change from baseline (95% confidence intervals, CI). A two-sided *P*-value of 0.05 was considered to be statistically significant. Patient demographics were summarised descriptively. Continuous data were reported as mean and standard deviation, whereas categorical data were reported as count and proportions.

Results

The demographic characteristics and indications for HBOT of the 25 patients (15 male, 10 female; mean age 56 (SD 13) years) enrolled in the study are shown in Table 2. On admission to ICU, the mean APACHE II severity of illness score was 16 (6). The median (interquartile range) values for

Table 2
Patient demographics, ICU and HBOT characteristics

Sex	Age (y)	APACHE II score	Length of MV (h)	Length of ICU stay (days)	Total HBOT sessions	ICU mortality	Presenting condition
M	42	10	148	9	11	No	Necrotising infection
F	53	10	271	13	11	No	Anaerobic septicaemia
M	47	8	19	3	2	No	Necrotising infection
F	56	14	390	14	10	No	Necrotising infection
M	78	28	162	7	7	No	Necrotising infection
M	58	10	166	8	3	No	Necrotising infection
M	47	21	127	11	6	No	Necrotising infection
M	76	16	475	8	9	Yes	Clostridial myonecrosis
F	65	12	116	2	11	No	Wound healing
M	65	14	200	7	7	No	Necrotising infection
F	69	17	219	11	7	No	Necrotising infection
F	39	24	741	3	9	No	Acute ischaemia/oedema
M	76	21	130	7	6	No	Necrotising infection
M	66	19	142	11	4	No	Fournier's gangrene
M	37	14	141	6	6	No	Anaerobic septicaemia
M	39	8	13	3	2	No	Necrotising infection
M	38	14	88	4	3	No	Necrotising infection
F	66	10	434	18	8	No	Fournier's gangrene
M	56	14	320	14	9	No	Acute ischaemia/oedema
M	51	28	230	14	10	Yes	Anaerobic septicaemia
M	49	17	268	7	7	No	Necrotising infection
F	40	16	233	11	9	No	Clostridial myonecrosis
F	76	21	119	5	2	No	Arterial gas embolism
F	64	14	200	10	5	No	Clostridial cellulitis
F	56	17	185	11	7	No	Anaerobic septicaemia

Table 3
Characteristics of oxygenation and related mechanical ventilation parameters; data are mean (standard deviation)

Respiratory parameter	Pre-HBOT	Return to ICU	+1 h	+2 h	+3 h	+6 h
F_iO_2	0.47 (0.1)	0.49 (0.2)	0.47 (0.1)	0.46 (0.1)	0.44 (0.1)	0.43 (0.2)
P_aO_2 (mmHg)	141 (85)	101 (54)	104 (28)	118 (35)	115 (32)	116 (43)
P_aO_2/F_iO_2 ratio	301 (99)	221 (87)	234 (75)	274 (86)	274 (16)	278 (78)
S_aO_2 (%)	98 (2)	95 (5)	96 (5)	97 (24)	97 (1)	97 (2)
P_aCO_2 (mmHg)	43 (7)	48 (9)	45 (10)	43 (8)	43 (7)	43 (6)
Respiratory rate min^{-1}	17 (4)	16 (4)	17 (4)	17 (4)	16 (4)	17 (4)
Tidal volume (mL)	551 (170)	553 (170)	549 (130)	540 (150)	558 (130)	549 (150)
PEEP (cmH_20)*	7.0 (3.0)	7.1 (3.2)	7.1 (3.3)	7.1 (3.3)	6.9 (3.1)	6.9 (3.1)
I:E Ratio (1:)	2.4 (0.7)	2.4 (0.6)	2.4 (0.6)	2.4 (0.7)	2.5 (0.7)	2.4 (0.7)
Pressure support (cmH_20)	11 (3)	11 (3)	11 (3)	11 (3)	12 (3)	11.0 (3)

* Positive end expiratory pressure

duration of mechanical ventilation and length of ICU stay were 168 hours (129–263 hours) and 8 days (5.5–11 days) respectively. The presenting indications for HBOT are also shown in Table 2. The modes of ventilation used for the 61 HBOT sessions were synchronized intermittent mandatory ventilation (SIMV)-volume control ($n = 32$), SIMV-pressure control ($n = 25$) and spontaneous-pressure support and PEEP ($n = 3$). Two patients died in ICU during the study.

Table 3 presents the mean (SD) oxygenation values for each time point. Following HBOT, the mean P_aO_2/F_iO_2 ratio decreased compared to the pre-HBOT ratio by 27% on return to ICU ($P < 0.001$, 95% CI 21 to 34), by 22% at 1 hour post-HBOT ($P < 0.001$, 95% CI 15 to, 29) and by 8% at 2 hours ($P = 0.03$, 95% CI 0.8 to 14). At 3 and 6 hours there were no significant differences from baseline.

Table 4
Characteristics of alterations to F_iO_2 and pressure-assist levels following HBOT

Ventilation parameter altered	*n*	Pre-HBOT	Return to ICU	+1 h	+2 h	+3 h	+6 h
F_iO_2	20	0.41 (0.10)	0.58 (0.24)	0.58 (0.17)	0.53 (0.15)	0.50 (0.16)	0.47 (0.16)
PEEP* (cmH_20)	5	8.5 (3.3)	10.2 (4.2)	11.3 (3.5)	11.5 (3.7)	11.5 (3.7)	11.3 (3.5)
PS† (cmH_20)	5	9.4 (3.6)	12.0 (2.5)	12.4 (3.3)	12.4 (3.3)	13.4 (3.1)	13.4 (3.1)

* Positive end-expiratory pressure; † Pressure support; data are mean (standard deviation)

Changes in F_iO_2 or positive pressure settings to compensate for reduction in oxygenation such as lowered P_aO_2 or S_pO_2 were needed in 17 patients (68%) after at least one of their monitored HBOT sessions. These 17 patients required such changes to be made after 30 of the 61 sessions (Table 4). The most common change made (n = 20) was an increase in F_iO_2 above baseline values (mean change +0.17, 95% CI 0.11 to 0.23), followed by a return towards baseline values by 2–6 hours post-HBOT. A number of alterations in PEEP (n = 5) and pressure support (n = 5) too small for inferential statistical analysis also occurred (Table 4).

Discussion

This study confirms previous observations that post-HBOT oxygenation reductions of approximately 20–24% at 1 hour and 8–18% at 2 hours post-HBOT occur in many mechanically ventilated ICU patients.[3,4] This deterioration in oxygenation is sufficient to require compensatory alterations in ventilation settings, typically by raising the F_iO_2 or less frequently by increasing PEEP or pressure support. At the time of writing, the authors were unaware of other reports describing the incidence or magnitude of mechanical ventilation changes necessitated by impaired oxygenation in the post-HBOT period.

Because of the observational and non-invasive nature of this study, it is not possible to determine whether the reductions in oxygenation post-HBOT are related to blunting of the hypoxic pulmonary vasoconstrictive response, hyperoxia-induced atelectasis or other causes (Table 1). However, inadequate mechanical ventilation during HBOT compared to ICU ventilator settings is unlikely to have contributed, since ventilation was adjusted during HBOT to compensate for the hyperbaric environment. The placing of patients on F_iO_2 1.0 prior to transport may have resulted in absorption atelectasis leading to alterations in the P_aO_2/F_iO_2 ratio.[12] The deleterious effect on oxygenation of not maintaining PEEP during changeover to transportation ventilators is reported in the literature.[5] However, neither this nor the use of prophylactic recruitment manoeuvres were assessed in this study.

There are a number of limitations to this study. Firstly, although data were collected over one year from a consecutive set of patients, further, possibly larger studies will be required to characterise the differences between those subjects who require adjustments in ventilation versus those who do not. Secondly, this study gathered data at 1-hour intervals only, whereas more frequent observations within the first 2 hours post-HBOT would better characterise the time course of this phenomenon. Thirdly, indices of oxygenation such as the P_aO_2/F_iO_2 ratio have been reported to have varying reliability in describing the severity of alterations in oxygenation.[13–15] Fourthly, data were obtained from only about a third of the HBOT sessions these patients underwent, and no effort was made to look at whether these effects on oxygenation were progressive over time.

Conclusions

Significant reductions in oxygenation as measured by the P_aO_2/F_iO_2 ratio were observed among 25 mechanically ventilated ICU patients following HBOT. A majority of these reductions necessitated increases in F_iO_2 or pressure-assist levels. From the data gathered it is not possible to identify specific characteristics to predict those patients at risk, to determine causation or to infer any impact upon medium- and long-term outcomes. Prospective interventional studies could usefully investigate the impact of recruitment manoeuvres or maintenance of PEEP levels during changeover of transportation ventilators to minimise falls in oxygenation in mechanically ventilated patients post-HBOT.

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Transportation of divers with decompression illness on the west coast of Scotland

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Key words
Decompression illness, decompression sickness, transport, recompression, clinical audit

Abstract
(Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. *Diving and Hyperbaric Medicine*. 2011;41(2):64-9.)
Introduction: There is a time line for divers who develop decompression illnesses (DCI) from the completion of their dive to the initiation of recompression. The time to treatment is influenced by many factors; two being the time before acknowledgement that the diver has a pressure-related illness and the time taken for transfer from that point to commencment of recompression.
Method: Time to onset of symptoms, and time from onset of symptoms to treatment were analysed for 233 divers, 202 recreational and 31 professional, presenting within 24 h of onset of symptoms to the Dunstaffnage Hyperbaric Unit between 1990 and 2009, who were transported by air, sea or road.
Results: Divers with severe DCI had significantly shorter times for onset of symptoms (95% confidence intervals 0.9 to 2.3 h longer for mild/moderate compared to severe DCI) and were transferred for treatment approximately twice as fast as those with mild/moderate symptoms (inter-quartile ranges: recreational divers, 2.25–5.63 h for mild/moderate DCI versus 1.54–3.25 h for severe DCI; professional divers, 2.63–11.13 h for mild/moderate DCI versus 2.25–2.92 h for severe DCI). Although choice of transport was most likely influenced both by location and disease severity, transfer modality did not significantly affect time to treatment for divers with severe DCI. In addition, no differences in time to treatment were observed between professional and recreational divers irrespective of disease severity.
Conclusions: The data suggest that transport was optimised to fit the particular circumstances of the patient and that divers treated for DCI in Scotland may benefit from there being a single, integrated, co-ordinated clinical service.

Introduction

A number of challenges exist when considering any temporal element during the triage of divers with decompression illness (DCI). General opinion is that decompression illness should be treated as rapidly as possible, although the basis for determining the most favourable transfer time to a compression chamber is not apparent.[1] Severe decompression illness can be life-threatening because of neurological and cardio-pulmonary injury and is associated with hypovolaemic shock.[2–5] While some studies indicate a relationship between time to treatment and clinical outcome in decompression illness, others relate outcome to severity of the initial condition rather than delayed recompression.[6–13] In making assessments of any effects and/or benefits of the method of patient transport and the concomitant delay in treatment, there is a raft of factors that could influence decision making, such as the location of the most appropriate treatment facility, severity of symptoms, transport costs, transport safety, transfer time and infrastructure costs.[1,14,15]

The vast majority of diving worldwide probably relies on recompression facilities that are located some distance from the diving operations. Each location will differ markedly both in the type and the quality of the available transport infrastructure at the regional level but also in the capability of the emergency framework that is available to support patient transport. Invariably some of the transfer decisions will be dictated by cost, which, in turn, will be influenced by the severity of the diver's condition. Patient transfer is a significant component of the whole treatment pathway but is rarely studied.[15,16] The present account reports on an examination of transport data collated by one treatment centre in relation to severity of symptoms.

The catchment area for the Dunstaffnage Hyperbaric Unit, near Oban, covers most of the west coast of Scotland. The west coast is predominantly fjordic in nature, with numerous isolated peninsulas, isthmuses and inshore and offshore islands.[17] Road and rail infrastructure is limited or discontinuous in much of the region making land transportation difficult in many cases. The diving population on the west coast is predominantly recreational and boat-based. There are also a significant number of occupational divers employed in mariculture and shellfish industries. Many of the better recreational diving locations, the epicentres of the fish farming industry in Scotland and the most productive shellfish sites are in relatively remote locations.

Treatment of divers presenting with DCI in Scotland is provided by a single, co-ordinated clinical service.[18] The vast majority of cases are initially processed through a single telephone helpline provided at the consultant level by the hyperbaric facility at Aberdeen. Through discussions with the divers, local medics and/or the emergency services,

the Aberdeen doctor will advise on the most appropriate treatment pathway in terms both of which recompression chamber the patient should be taken to and the best form of transport to use. An exception to this is a relatively small proportion of divers that self-present, either at a local hospital or at the recompression facility itself.

The present study investigates the transfer modality in three distinct groups of presenting divers. Following the onset of recognisable symptoms of DCI, divers who have made it ashore tended to travel to the unit by road, either by private vehicle or ambulance. Divers who were still at sea, and within a reasonable distance from the unit, were transferred by lifeboat (operated by the Royal National Lifeboat Institute[19]), the vessel they had been diving from, or a combination of the two. Cases from further afield were usually transported by air when it was the only practical method dictated by the geographical position of the diver. Air transportation was either from the search and rescue organisations (military or coastguard) or air ambulance services (helicopter or fixed-wing).

Methods

The present study adheres to the procedures of implied consent operated by the UK National Health Service (NHS) for clinical audit. A total of 233 cases presenting within 24 hours (h) of onset of symptoms to the Dunstaffnage Hyperbaric Unit within the 20-year period 1990 to 2009 inclusive were analysed. All data were based on the information contained within the clinical record; all but three were first examined by one of a total of six physicians from a single practice employing a standard *pro-forma* record. Twenty-two other patients were omitted from the analyses if no information on transportation modality had been entered into the patient record (n = 5) or if the time from onset of symptoms to treatment exceeded 24 hours (n = 17).

The patient population was initially examined for basic demographics (sex and age) and the number of recreational divers. Cases were then assessed for symptom latency (time from surfacing to the onset of symptoms) and transfer time (time from onset of symptoms to start of treatment). Transfer time did not allow for differences between patients transported directly to the hyperbaric unit against those presenting via the hospital Accident and Emergency Department. Cases were analysed in two groups depending on the severity of their condition, based on previously published criteria: mild/moderate was no symptoms, pain only or sensory; severe was ataxia, motor involvement, nausea or vertigo, or cerebral symptoms and signs.[18] The severity groups were subdivided into their transport modality: land, sea or air. Time from onset of symptoms to treatment and number of patients were analysed by transfer modality, regional location and severity of presentation; professional status was analysed against transfer time for both of the DCI severity criteria. Statistical analysis followed preliminary examination for normality using modified (Lilliefors) Kolmogorov-Smirnov tests with transformation where necessary.[20–22]

Results

The proportion of recreational divers in the two main subsets was 85–89% (Table 1). Divers classified as having

Table 1
Transport type, demographics, diving type, symptom latency and time to treatment in 233 cases of decompression illness presenting within 24 h of onset of symptoms to the Dunstaffnage Hyperbaric Unit (1990-2009)

	Severe DCI				Mild/moderate DCI			
	Overall	**Land**	**Sea**	**Air**	**Overall**	**Land**	**Sea**	**Air**
Number of divers	65	19	29	17	167–168	67–68	54	46
Males (%)	86	89	86	83	80	81	80	80
Age (y)								
Mean	40.4	41.4	40.1	39.8	34.9	33.9	35.2	36.0
Median	39	39	39	36	33	32	34	34
Range	20–62	22–62	20–61	27–58	16–77	18–59	16–77	20–61
Recreational (%)	89	95	89	83	85	82	85	78
Time to onset of symptoms								
Mean	0.36	0.22	0.38	0.49	1.99	3.24	1.29	0.99
Median	0.17	0.08	0.17	0.17	0.33	0.50	0.29	0.25
IQ range	0–0.33	0–0.21	0.05–0.33	0.08–0.41	0.17–1.34	0.25–2.0	0.17–1.34	0.10–1.0
Range	0–3.5	0–2.0	0–2.5	0–3.5	0–24	0–24	0–19	0–13
Time from onset to treatment								
Mean	2.96	2.86	2.97	3.04	5.65	6.44	4.78	5.54
Median	2.0	2.0	2.0	2.92	3.08	3.88	3.08	4.0
IQ range	1.75–3.25	1.5–2.63	1.75–3.25	2–3.25	2.25–6	2.25–6.48	2.25–6	2.75–5.88
Range	0.67–12.75	0.83–12.75	0.67–12.0	1.75–7.0	0.25–22.5	0.25–22.3	0.58–20.0	0.83–22.5

severe DCI were significantly older than those in the mild/moderate group (*Z*-test following log transformations, $P < 0.05$; 95% confidence intervals (CI) +1.1 to +1.3 y); there was no significant difference for sex ratio between the two groups (Z-test, $P > 0.05$) (Table 1).

In total, air transfer was used in 27% of the cases examined, transfer by sea occurred in 36% and land transport in 37% of cases. Sea transport was the most common method of transfer for severe cases; land transport was the most common for mild/moderate cases. The proportion of transfers conducted by each of the three transport methods examined did not differ significantly with the two severities of condition (*Z*-test, $P > 0.05$ in all cases).

Time from the onset of symptoms to treatment was analysed by professional status for both DCI severity groups (Table 2). In general, professional divers took longer than recreational divers to receive treatment for mild to moderate presentations, and shorter for severe cases; however, both relationships were not significant (t-test, $P > 0.05$ in both cases).

A map of Scotland laying out the main diving areas, transport infrastructure and recompression facilities is shown in Figure 1. Comparing transport modality with location illustrates some of the drivers of the decision process (Table 3). With reasonable road and sea (lifeboat plus divers' own boats) links, and the relative distance from helicopter support, the main methods for patient transfer in the Oban area are land and sea for all types of DCI (Table 3). There was no clear transport of choice for the Inner Hebrides, irrespective of severity of presentation, and this probably reflects the variation in proximity to short ferry times, boat rides and lifeboat transfers for these islands. Not surprisingly, where the only non-emergency transport available from the Outer Isles to the mainland is by scheduled ferries, all Outer Hebrides transfers were by air (helicopter in all but one which was a fixed-wing transfer). This may also have been influenced by the availability of an emergency helicopter based at Stornoway (St; Figure 1). Road and rail links between Skye and Oban are generally poor, and the location of the Skye area midway between the helicopter based at Stornoway and the Oban facility makes air transfer a more logical choice of transport (Table 3). The transfer modality and transport times from the southern sea lochs reflect a large variation in the quality of communication links (but not necessarily straight-line distances) to Oban in addition to the closer proximity to the Prestwick helicopter base (Pr; Figure 1).

Table 2
Time from onset of symptoms to treatment (minutes) in relation to professional status and severity of presentation for 233 cases of decompression illness presenting within 24 h of onset of symptoms to the Dunstaffnage Hyperbaric Unit (1990-2009)

	Recreational		Professional	
	Mild/mod	Severe	Mild/mod	Severe
Number	144	58	24	7
Mean	5.34	3.01	7.56	2.54
Median	3.42	2.00	4.59	2.67
I-Q range	2.25–5.63	1.54–3.25	2.63–11.13	2.25–2.92

Figure 1
Diagrammatic representation of the west coast of Scotland (shaded light grey; darker shading represents greater population density – west coast population density is 8–15 persons km^{-2}). Trunk roads are solid lines; railways are dashed lines. The Dunstaffnage Hyperbaric Unit is located just north of Oban (solid circular dot; Ob); Oban has three helicopter and one fixed-wing landing facilities. Two emergency helicopter bases exist, one at Stornoway (solid circular dot; St) and one at Prestwick (solid circular dot; Pr); one hour flight time radii for both bases are marked. Royal National Lifeboat Institute stations are marked as open squares. The Dunstaffnage facility provides an emergency recompression service for the whole of the west coast from the Mull of Kintyre (MK) in the south, to Cape Wrath (CW) in the north. Fish farm and shellfish diving occurs throughout the whole of the region but is concentrated in the Outer and Inner Hebrides (OH and IH, respectively). Centres of recreational diving activity are around Oban (Ob), the Sound of Mull (M), on and around the Isle of Skye (S) and the offshore islands of St Kilda (SK). Transport between the islands and the mainland is by ferry; the two main ferry hubs are at Oban (Ob) and Ullapool (U). Railway lines run inland from Oban and Fort William (FW); between Fort William and Mallaig (Ma); and between Inverness (IN) and Kyle of Lochalsh. (KL). Scale bar = 50 miles; inset is the Shetland Isles.

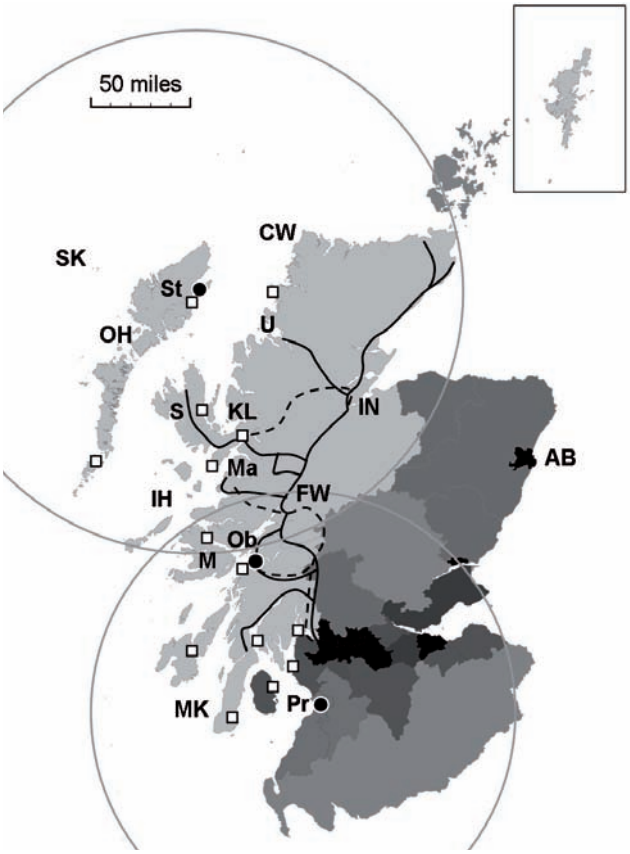

Table 3
Median time from onset of symptoms to treatment (h), transport modality (*n*, in parentheses) in relation to location (refer to Figure 1) and symptom severity for 233 cases of decompression illness presenting to the Dunstaffnage Hyperbaric Unit (1990–2009); spaces = no data

Location	Severe DCI			Mild/moderate DCI		
	Land	Sea	Air	Land	Sea	Air
Oban / Sound of Mull	1.63 (14)	2.00 (27)	2.46 (6)	3.50 (57)	3.00 (53)	2.46 (6)
Inner Hebrides	2.00 (1)	6.88 (2)	2.00 (3)	20.00 (3)	8.75 (1)	4.13 (4)
Outer Hebrides			3.25 (4)			4.33 (12)
Isle of Skye *	7.75 (2)		2.67 (3)	6.84 (2)		4.00 (20)
Southern sea lochs †	1.75 (2)		5.00 (1)	3.75 (7)		6.00 (3)
Other ‡				21.00 (1)		

* Includes mainland sea lochs to the north (Sutherland) and the south (Knoydart) of Skye
† Southern sea lochs refers to Lochs Fyne, Long, and the Firth of Clyde
‡ Case treated at Dunstaffnage but originating out of the area

Onset of symptoms was significantly more rapid in the severe group compared with mild/moderate cases (Mann-Whitney $P < 0.05$; 95% CI 0.9 to 2.3 h longer for mild/moderate; Table 1). The longest time for onset of symptoms was 3.5 h in the severe group and 24 h for the mild/moderate cases (although there were longer times than 24 h recorded for the mild/moderate group, these were outside the time limit considered in this study).

In severe DCI cases, transport type was not related significantly to symptom latency (Kruskal-Wallis test, $P > 0.05$); in mild/moderate cases, divers transferred by land had significantly longer symptom latency compared with air or sea transfers (Kruskal-Wallis test, $P < 0.05$).

In these data, it was not possible to distinguish the time following onset of symptoms from when there was acceptance of an issue, nor the time at which medical advice/help was requested during the total transportation process to a hyperbaric facility. However, overall time from symptom onset to treatment was significantly shorter for severe cases compared with mild/moderate ones (Mann-Whitney test, $P < 0.05$; 95% CI 1.7 – 3.7 h longer for mild/moderate; Table 1). There were no significant differences in transfer time between land, air and sea within both the severity groups (Kruskal-Wallis test, $P > 0.05$ in both cases).

Discussion

In the present study, once a diving-related problem was recognised, patients with severe symptoms were transported to the Oban recompression facility faster than those with mild-to-moderate symptoms, irrespective of the form of transport used. In the geographical region studied, there was a single NHS-registered treatment facility and rarely was there an even or open choice between the three transport modalities; transport type was more likely to be affected by the location of incident. For example, divers entering the emergency system on outer islands or peninsulas with no existing road infrastructure were more likely to be transported by air. The alternative solution would have been to rely on sea transfers that may have been beyond the operational range of some vessels or would have been affected by timetabled schedules, if relying on commercial ferries. Therefore, slower sea transfers were unlikely to be considered irrespective of issues related to perceived severity of condition, costs or risks.[15]

It may be the case that divers presenting with severe symptoms were more prevalent in the areas where there was actually a transport choice (e.g., the use of faster lifeboats or ambulances compared with the divers' own boat or car). Although the basis for the decisions about transport type was not recorded, the lack of any significant differences in transfer time for severe cases tends to suggest that an informed triage was adopted in order to optimise transfer time. However, the lack of difference is probably influenced greatly by there being a lack of any major choice in terms of the most appropriate chamber for treatment and/or the most appropriate modality of transfer. It is also unlikely that a diver with deteriorating severe DCI would be transferred any differently and/or more quickly than one with stable and/or improving severe DCI.

The integrated, single clinical service for emergency recompression in Scotland permits an initial transfer immediately to the more advanced clinical facility in Aberdeen. However, this has never happened for divers in the region described in the present study; critically ill patients have either received primary treatment at Oban before transfer to Aberdeen for additional treatment or have obtained initial stabilisation at the Oban Hospital, without recompression, before transfer to Aberdeen for their initial recompression treatment.

In the present study, cases of severe DCI had a more rapid

onset of symptoms. Indications of severe DCI in divers will usually be more distinctive than those of mild/moderate disease and so it is not surprising that there was a shorter time in recognising the problem in these cases. Consequently, there was a more rapid call for advice and transportation for treatment. This meant that 75% of the severe cases began their treatment in less than four hours from surfacing compared with approximately 7.3 hours for 75% of mild-to-moderate cases. Again, this indicates that a form of triage had been adopted whereby transport type and transfer time may have been considered to be less important for the mild to moderate cases. Even though the transfer modality may have been the same, mild/moderate cases were probably ranked in their importance for transfer: for example, air transfer times for mild-to-moderate cases were significantly longer than those for severe even though transport times, because of relatively short direct flight distances and fixed maximum speeds, would be expected not to differ (Table 1).

Professional status did not affect time to treatment. Delayed treatment could have been expected from the professional diving group because of a potential negative perception to how their future income may be affected. Time to treatment for the professional group presenting with mild-to-moderate symptoms was longer than that of recreational divers, but this was not statistically significant. While any delay could reflect a reluctance to seek treatment, it could also be influenced by location, with more professional divers being transferred from the Outer Isles.

There is a tendency to categorise the west coast of Scotland as the most remote location in the UK for transporting divers with DCI. However, median transfer times for west coast cases for both severe and mild-to-moderate DCI (2.00 and 3.88 h, respectively) are considerably shorter than those found in, for example, typical DAN data (20.25 h) or in the north-east coast of Australia (53.2 h).[15,23] The present study ignored cases where transfer was over 24 h (although this only occurred in mild-to-moderate cases) because the transfer infrastructure and distances involved would place all cases well within a 24 h transfer period. Therefore, transfers taking longer than 24 h were obvious outliers most often associated with denial or continued diving with DCI symptoms. Inclusion of these outliers would not have affected the overall trend that divers with DCI on the west coast of Scotland tend to be transferred relatively quickly to treatment facilities. This is mainly because transfer modalities are available to reduce the problems of a relatively remote and maritime-dominated coastline. As such, the issues related to the management of mild or marginal DCI in remote locations are largely irrelevant to incidents that occur in Scotland.[24]

Previous studies have shown that divers treated for DCI in Scottish facilities have a high rate of good clinical outcome.[25] Part of the reason for this may be that Scotland has an integrated transfer system that prioritises and optimises patient transport.

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Provisional report on diving-related fatalities in Australian waters 2006

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Key words
Diving deaths, scuba, breath-hold diving, closed-circuit rebreathers, diving accidents, case reports

Abstract
(Lippmann J, Walker D, Lawrence CL, Fock A, Wodak T, Jamieson S. Provisional report on diving-related fatalities in Australian waters 2006. *Diving and Hyperbaric Medicine*. 2011;41(2):70-84.)
Introduction: An individual case review of diving-related deaths reported as occurring in Australia in 2006 was conducted as part of the Divers Alert Network Asia-Pacific (DAN AP) 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 16 reported fatalities (eight fewer than 2005), all involving males. Ten deaths occurred while snorkelling and/or breath-hold diving and six while scuba diving, one of which involved the use of a closed-circuit rebreather. One death resulted from an encounter with a stingray and two involved scuba divers diving alone after an extended absence from diving. Cardiac-related issues were thought likely to have contributed to the deaths of six snorkel divers and one scuba diver.
Conclusions: Trauma from a marine creature, snorkelling or diving alone and pre-existing medical conditions once again featured in several deaths in this series.

Introduction

Each year in Australia, there are deaths associated with snorkelling and diving using compressed gas (i.e., scuba or surface-supplied breathing apparatus). Although some accidents are unavoidable, many might be avoided through better education, appropriate medical screening, greater experience, common sense, improved supervision or better equipment maintenance and design. The aim of the Divers Alert Network Asia-Pacific (DAN AP) Dive Fatality Reporting Project (incorporating Project Stickybeak) is to educate divers and the diving industry and to inform diving physicians on the causes of fatal dive accidents in the hope of reducing the incidence of similar accidents in the future and of detecting, in advance, those diving candidates who may be at risk. This report includes the diving-related fatalities between 01 January and 31 December 2006 that are recorded on the DAN AP database. When an accident is unwitnessed, it is often 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.

Methodology

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 Human Research Ethics Committee, Department of Justice, Government of Victoria, Australia to access and report on data included in the Australian National Coronial Information System (NCIS). In addition, ethics approvals have been sought and obtained from various coronial offices in certain Australian States and Territories. The methodology used for this report was identical to that described previously for the 2004 Australian diving-related fatalities.[1]

Snorkelling and breath-hold diving fatalities

BH 06/01

Although the coronial papers about this incident are unavailable (at the request of the victim's family), some of the media reports combined with other enquiries provided useful details.

This victim was a 44-year-old male who was apparently fit and healthy and an experienced snorkeller. His death occurred while he was making a documentary film about marine life. He was snorkelling and, at the time of the incident, was reportedly standing on, or floating above the reef in water of a depth of about 1.5 metres' of seawater (msw). He was very near a large stingray (bull ray), reportedly with a wingspan of approximately one metre. He was positioned to enable a nearby cameraman to film both the stingray and himself in the same frame. The cameraman was in front of the stingray and the victim was behind and above the ray when the ray turned around, rose and flicked its barbed tail, hitting the victim forcefully to the left of his sternum. He was seen to pull the barb out but then collapsed. Resuscitation was attempted on the boat and continued on shore, but was unsuccessful.

Autopsy: There is no autopsy report available but other sources report that death was found to have resulted from cardiac tamponade.

Comment: A tragic death resulting from the victim positioning himself too closely over and above a stingray and likely causing it to feel threatened and trapped. The shallow depth placed the victim within range of the stingray's barbed tail. Removal of an embedded stingray spine is generally discouraged in the first-aid setting, as it is likely to further increase the trauma caused by the rear-facing serrations, and 'unplug' the wound. However, it is unknown whether this act made any difference to the outcome of this incident.

Summary: 44-year-old male; experienced; snorkelling in shallow water close to large stingray; penetrating injury of the chest caused by a stingray barb; trauma causing death from cardiac tamponade

BH 06/02

This 55-year-old male overseas visitor went snorkelling with a group off an island resort on the Great Barrier Reef (GBR). No details are available relating to his health, swimming and snorkelling experience, the water conditions, whether he or his group received any instruction or advice prior to snorkelling, or whether any buddy system was supposed to be in place.

The victim was noticed floating motionless by lifesavers and was quickly reached, found to be unconscious, and brought ashore. It is unknown how long he had been unconscious. He was unresponsive, apnoeic and there was no palpable pulse, so basic life support (BLS) was commenced while awaiting the arrival of oxygen (O_2) equipment and an automated external defibrillator (AED). Oxygen-supplemented ventilations were soon provided using a bag-valve-mask. When the AED was attached, no shock was advised, indicating the likelihood of asystole. Resuscitation was ultimately unsuccessful.

Autopsy: A pre-autopsy CT was performed after the absence of trauma or evidence of marine stings was noted, and it revealed no significant pathology. At the autopsy, the heart (296 g; normal range 150–400 g) had a normal appearance and weight. The right coronary artery was unusually small (maximal diameter 1 mm, with 40% narrowing by atheroma) and the left anterior descending (LAD) coronary artery was up to 60% narrowed by atheroma. There was no evidence of any old or recent myocardial infarction and the histology showed patchy ischaemic-type fibrosis and some fat infiltration in the myocardium of the left ventricle. There was severe fatty change in the liver. His BMI was 25 kg cm^{-2}. The lungs (right (Rt) 558 g, left (Lt) 508 g; normal range for combined weight 600–1000 g) showed gastric contents in the upper airways. The stomach was full of food. There were several rib fractures due to vigorous resuscitation efforts. The cause of death was given as coronary atherosclerosis.

Comment: It is unknown how long the victim was unconscious before being noticed and, therefore, how long the delay prior to BLS and attempted defibrillation. It is pleasing to note the availability of high-concentration O_2 equipment and an AED, which are becoming increasingly available at dive sites on the GBR, a valuable model for other diving destinations.

A narrowing of the LAD of only 60% would normally be considered to be borderline as a cause of clinical symptoms (i.e., less than 75%). However, the pathologist commented that the right coronary was unusually small, which may represent congenital hypoplasia of the right coronary artery. The combination of an LAD stenosis and a small right coronary artery with atheroma may have produced apical ischaemia in this case.

Summary: 55-year-old male; solo or separated snorkeller of unknown experience; found unconscious on surface; atherosclerosis; cardiac-related death

BH 06/03

This 70-year-old male had no history of medical problems and was taking no medications. He and his wife went snorkelling from a commercial vessel on the GBR. While he had stated that he could swim, there is no record of whether he had snorkelling experience. All passengers intending to snorkel were offered a 'noodle' flotation aid, which the victim took. He used his own mask. The boat was anchored to windward of the chosen reef and there was a strong current running off the reef towards the boat and the conditions were described as "*quite rough*". The victim and his wife took an opportunity for a short practice snorkel before his wife left on a supervised tour. His wife reported that he managed to use the fins after an initial period of uncertainty.

When the guided tour started, the victim remained in the designated snorkelling area between the boat and the reef. A lookout was on duty to watch those in this area. When his wife returned from the guided tour and enquired about her husband's whereabouts, it was eventually realised he was missing. Another boat in the area was alerted and its captain found the victim about 300 metres further from the reef, floating face down and unconscious, with his mask and fins in place and supported by the 'noodle'. He was brought back to the boat where BLS was commenced. When an AED from the other vessel was attached, no shock was advised, indicating the likelihood of asystole. The victim failed to respond to BLS, which was terminated after about 35 minutes on medical advice via radio.

Autopsy: The autopsy revealed his heart was healthy (395 g), with only mild atheromatous changes and widely patent coronary arteries. His lungs (Rt 710 g, Lt 624 g) were

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

ID BH	Age	Sex	Height (cm)	Weight (kg)	BMI	Training	Experience
06/01	44	M	n/s	n/s	n/s	trained	yes
06/02	55	M	165	68	25.0	n/s	n/s
06/03	70	M	177	70	22.3	nil	nil
06/04	74	M	176	81	26.1	nil	nil
06/05	61	M	196	95	24.7	n/s	n/s
06/06	69	M	175	71.5	23.3	n/s	n/s
06/07	26	M	182	78	23.5	nil	nil
06/08	23	M	187	66	18.9	n/s	n/s
06/09	76	M	173	74	24.7	n/s	n/s
06/10	41	M	177	75	23.9	n/s	n/s

oedematous and congested. There was a recent haemorrhage in the mediastinum adjacent to the Lt hilum, and fractures of the Rt ribs 2–3 and Lt 2–6, in the mid-clavicular line, consistent with resuscitation attempts. Histology showed mild interstitial fibrosis of the heart, and atheromatous narrowing of up to 50% was noted in the epicardial arteries. The cause of death was given as drowning.

Comment: Despite the presence of a 'safety watch' the victim drifted, possibly unconscious, some 300 m from the designated snorkelling area. As he was wearing a bright yellow shirt which was highly visible, the company received an 'infringement notice' for having failed to adequately watch over those in the designated swimming area. As this death was unwitnessed, it is unknown what led to his drowning. However, inexperienced snorkellers often have trouble clearing a snorkel and aspiration of salt water can cause drowning through a variety of mechanisms. The cardiac fibrosis seen on microscopic examination of the heart raises the possibility of some cardiac precipitant in the drowning. Since the flotation noodle was still under his arms death is unlikely to have been caused by asphyxia of ascent. It is not known how close the other boat was anchored or whether persons snorkelling or swimming from it were close to the designated swim area of the victim's boat. This factor could have compromised the efficiency of supervision.

Summary: 70-year-old male; no history of medical problems and not on medication; snorkelling alone but supposedly under supervision; discovered unconscious; drowning

BH 06/04

This 74-year-old male overseas tourist joined a day trip to visit a pontoon moored on the GBR. There were 428 passengers and a crew of 40. He was reported to have been active and apparently healthy, although taking ramipril daily for mild hypertension. He had been a lifeguard in the past so was presumably a competent swimmer, although it is unknown what previous snorkelling experience he had.

A safety talk was given and a video on snorkelling was shown during the outward trip. An information leaflet in multiple languages, including the victim's primary language was also available. When they arrived at the pontoon, the tour leader gave instructions, at which the victim was present, on how to fit and use the snorkelling equipment. Sea conditions were reported to be rough due to a moderate wind, with a slight current. Water temperature was 27°C. The victim was wearing mask, snorkel, fins and a Lycra suit, without any additional buoyancy aid.

On this day, there were three designated lookouts/lifesavers with responsibility for those in the buoy-defined, designated swimming area. At the time, there were approximately 25 swimmers in the water, although the victim was snorkelling alone. Minutes after the first passengers had entered the water, one of the lookouts saw the victim floating slightly submerged just outside the buoyed area. When she went to him in the safety dinghy, she found him unresponsive; however, being unable to lift him into the dinghy unaided, she returned to the pontoon to get another lifeguard. They returned to the victim, who was now 70 metres from the pontoon. His eyes were open but he remained unresponsive. Although his mask was in place, it was full of white froth and his snorkel was out of his mouth. He was brought aboard the dinghy where it was noted that he was cyanotic, apnoeic and pulseless. He was taken to the pontoon where BLS was commenced with supplemental oxygen provided via a resuscitation mask with O_2 inlet. When an AED was attached, no shock was advised, indicating that he was likely to have been in asystole. BLS was continued for 70 minutes until paramedics arrived and declared the victim dead.

Autopsy: The heart weighed 477 g and showed a dilated left ventricle. The coronary arteries showed generalised atherosclerosis with significant stenosis of the LAD (70%). Sectioning of the myocardium revealed a degree of circular

Table 1 (cont.)
fatalities in Australian waters 2006
n/a – not applicable; n/i – not inflated; n/s – not stated

Dive group	Dive purpose	Depth (msw)	Incident (msw)	Weight belt	Wts (kg)	BCD	Disabling injury
GNS	filming	2	n/s	n/s	n/s	n/s	Trauma (Stingray)
Solo	recreation	n/s	surface	n/s	n/s	n/s	Cardiac
BSB	recreation	n/s	surface	nil	n/a	nil	Asphyxia
GSB	recreation	n/s	surface	nil	n/a	nil	Cardiac
GSB	recreation	n/s	surface	nil	n/a	nil	Cardiac
GSB	recreation	15	surface	nil	n/a	nil	Cardiac
BSB	recreation	2	surface	nil	n/a	nil	Asphyxia
Solo	spearfishing	n/s	surface	n/s	n/s	nil	Asphyxia
Solo	recreation	n/s	surface	nil	n/a	nil	Cardiac
BSB	recreation	n/s	surface	n/s	n/s	n/s	Cardiac

hypertrophy of the left ventricle and a well-defined extensive area of old infarction (maximally 20 x 45 mm) at the apex of the left ventricle, which histology showed was nearly full-thickness scarring of the myocardium. The lungs were heavy (Rt 1,100 g, Lt 970 g), markedly congested and clear haemorrhagic fluid oozed from the cut surfaces. The formal finding was death from cardiomegaly, coronary artery atherosclerosis with stenosis of major vessels and pulmonary congestion changes.

Comment: It appears that the previous myocardial infarction may have been silent as there was no indication that the victim had been treated for this. The reviewing pathologist would give the cause of death as ischaemic heart disease due to generalised atherosclerosis. The mechanism of death could have been a cardiac arrhythmia due to the atherosclerosis or ventricular dilatation, with terminal drowning due to loss of consciousness. The inadequate (single lifesaver in safety boat) response delayed retrieval but was not thought to have influenced the outcome. The operator's revised response rules now require the safety boat response involve two crew members.

Summary: 74-year-old male; unknown experience; history of hypertension; snorkelling with others but without buddy in patrolled area; silent death; atherosclerosis and cardiomegaly; cardiac-related

BH 06/05

This 61-year-old male was visiting from overseas and touring the GBR on a large cruiser. He had a history of polycystic renal disease and was on multiple medications: minoxidil 10 mg, frusemide 40 mg, ibesartan 15 mg, omeprazole 20 mg, gaviscon suspension, cephalexin 250 mg and ecsitolopram 15 mg.

On the previous day, there had been a presentation to passengers on safety procedures when snorkelling. It is unknown whether the victim had reported his health status to the cruise operator. He was reported to have been a competent swimmer, but it is unknown whether or not he had snorkelled before. On this day, the vessel was moored off an island resort and the group were ferried ashore, given a mask, snorkel, fins, and a life vest to wear, the last of which the victim appears to have declined. Then they entered the water from the beach, whilst a safety boat with lookouts was present. The weather was reported to be clear, with a moderate wind and waves of less than one metre high, but it is unclear whether there was a significant current. The snorkelling area was apparently not clearly marked.

After about 45 minutes, two of the group noticed the victim floating at the surface, face down and motionless, with the tip of his snorkel bobbing below the surface. On approaching him, they found him unresponsive and cyanotic. Despite believing him to be dead, one rescuer attempted in-water rescue breathing but was hampered by his own safety vest. The tender soon arrived and took the victim aboard, and BLS was initiated as they returned to the cruise boat. Resuscitation continued with the assistance of the resort nurse until the arrival of a doctor who declared the victim to be dead.

Autopsy: The heart was large, weighing 570 g, with dilatation and concentric hypertrophy of the left ventricle. The coronary arteries showed some atheroma but no significant atherosclerotic stenoses. The Rt and Lt lungs weighed 780 g and 800 g respectively. Cystic hepatic disease was present with cysts of various sizes scattered in a random manner. The typical changes of polycystic kidney disease were confirmed. The Lt kidney weighed 3,000 g, the Rt 2,760 g and each was 35 cm long with no normal renal architecture. The pathologist considered the deceased was to be regarded as being only a short time from developing full renal failure, though histology showed residual intact glomeruli and tubules throughout. There was some patchy myocardial fibrosis in the heart. The coroner found cause

of death to be acute cardiac failure, cardiomegaly, and polycystic renal disease.

Comment: The reviewing pathologist would give the cause of death as hypertensive heart disease due to polycystic kidney disease while snorkelling. As the pathologist described this 61-year-old man as 'elderly' it is probable his ill health was apparent to others. His disease was first identified when he was aged 25. He was under the care of a kidney specialist whom he had last attended about two months previously. This was a seriously ill man who died silently not far from others while gently snorkelling.

Summary: 61-year-old male; long history of kidney disease; snorkelling with group but without buddy; found by other snorkellers but unnoticed by safety lookout; cardiomegaly; cardiac-related

BH 06/06

This 69-year-old male was an overseas tourist of unknown prior snorkelling experience who was on a sailing vessel visiting several GBR resort islands. He was described as appearing healthy and had not reported having any ill health on the pre-snorkelling questionnaire. No medications were later found in his luggage.

The victim and other passengers had been provided with snorkelling equipment and were swimming or snorkelling between the vessel and the beach, some 20 metres away. The water depth was about 15 msw and the conditions described as good, Those in the water were monitored by crew on the vessel. The alarm was raised when two of the passengers saw a pool of blood under the water. The safety tender quickly found the victim floating and unresponsive with blood in his mask. He was quickly returned to the vessel where BLS was attempted without success.

Autopsy: The heart weighed 383 g, and appeared normal macroscopically. There was generalised severe atherosclerosis of all the coronary arteries, with a pinhole lumen in the LAD artery, 10 mm from its origin. Histology showed patchy subendocardial fibrosis in the left ventricle. The trachea and bronchi contained a large amount of watery, blood-stained, frothy fluid. The lungs showed congestion and pulmonary oedema (Rt 825 g, Lt 696 g). There was approximately 700 ml of blood-stained fluid in the left pleural cavity. The findings were of myocardial ischaemia, coronary artery stenosis and atherosclerosis.

Comment: An apparently fit man who was gently snorkelling and died silently. The lookouts reported that only 5–6 minutes earlier the victim had been seen to look up, smile, and give a 'thumbs-up' signal indicating that all was well. The reviewing pathologist would give the cause of death as ischaemic heart disease due to triple vessel atherosclerosis. It is possible this caused him some symptoms but his medical history is unknown. This is the first occasion in the long history of these reviews where a cardiac failure has involved blood staining the water. This was an unpredictable 'natural death' event.

Summary: 69-year-old male; no known medical history; apparently healthy; experience unknown; silent death while snorkelling in calm conditions; cardiac failure; cardiac-related

BH 06/07

The victim was a 26-year-old male overseas visitor with no known health problems. He and his girlfriend hired mask, snorkel and fins to go snorkelling from a popular, although unpatrolled, beach. Both were weak swimmers and the victim had not snorkelled before.

The water was calm and clear with only a slight current. The sea bed dropped steeply and the depth reportedly reached 2 msw only 10 metres from shore. The couple entered the water off a beach, and the victim appeared to be managing well and enjoying it. He chose to remain in the shallow water when his buddy went further out into the deeper water. When she looked back she saw him standing up with the upper half of his body out of the water and thought he may have waved to her. She decided to swim back to him and, alarmed not to find him, she alerted others, and a search was made. The victim was found on the sea bed about 45 minutes later at a depth of 2 msw and approximately 10 metres from shore. He was unconscious, cyanotic and apnoeic. BLS was commenced by a bystander and continued by a nurse. Airway management was complicated by the presence of copious amounts of blood-stained sputum and water. There was no response and efforts were ceased after 10–15 minutes.

Autopsy: There was frothy blood-stained fluid in the trachea and bronchi and 300 ml of blood-stained fluid in each pleural cavity. The lungs (Rt 920g, Lt 890g) showed pulmonary congestion and oedema. The heart and the cardiovascular system were normal. The cause of death was given as drowning (immersion).

Comment: The buddy had described how, when in shallow water, they both went towards shore to stand and clear their snorkels on previous occasions. It is possible that the victim inadvertently stepped into deeper water and was unable to clear his snorkel. Aspiration from a snorkel can trigger laryngospasm and subsequent silent drowning.

Summary: 26-year-old male; poor swimmer; first use of a snorkel; separated; drowning

BH 06/08

This 23-year-old male had a history of epilepsy and was described as 'poorly compliant' with his medication. He was

an experienced snorkeller and spear fisherman.

On this occasion, he donned a mask, snorkel, fins and a wetsuit and was spearfishing beside a long pier while his friend motored quietly in a boat nearby. The victim was observed to be snorkelling gently alongside the jetty for around an hour and was about 150 metres from the shore when his friend in the boat noticed that he was motionless. When the friend came alongside, he found the victim to be unconscious and, with difficulty, pulled him into the boat. He was apnoeic and pulseless. BLS was initiated and a faint pulse was detected after the victim vomited some water. A witness on the pier was alerted and called an ambulance. Paramedics arrived and provided advanced life support (ALS). He was taken to hospital where he died two days later.

Autopsy: The cause of death was listed as post-immersion cerebral anoxic changes with terminal bronchopneumonia. His heart (weight 400 g) was healthy on both macroscopic and histological examination. Brain and lung changes were consistent with the diagnosis. Toxicology tests showed the presence of phenytoin within the therapeutic range (approximately 16 mg L^{-1}), methamphetamine (0.1mg L^{-1}), and lignocaine and propofol, consistent with his hospital management. The cause of death was found to be hypoxic brain damage due to drowning in a person with a history of epilepsy.

Comment: It would appear likely that this man lost consciousness and drowned. Possible mechanisms include:

- Epileptic seizure followed by drowning.
- Cardiac arrhythmia followed by drowning. A link between sudden cardiac death and epilepsy has been described previously.[2] This may be related to the presence of long QT syndrome during swimming in susceptible epileptics.[3]
- The presence of methamphetamine at autopsy is difficult to attribute to his hospital management. Methamphetamine has been associated with cardiac arrhythmias and may also alter the seizure threshold in epilepsy.

Summary: 23-year-old male; history of poorly-managed epilepsy; experienced snorkeller; spearfishing alone with observer in boat nearby; drowning (possibly as a result of a seizure)

BH 06/09

This 76-year-old male was reported to have been "*in generally good health but had recently been complaining of a tightness in his chest*". It is unknown whether he had consulted a doctor about this.

He had first been seen walking up and down the beach with a metal detector, wearing bathers and a vest and with a mask and snorkel. Some 15 minutes later, a lifesaver taking part in training activity saw him floating, apparently unconscious, on the surface of the water, near to the beach. He was brought ashore unconscious and apnoeic and BLS was commenced. Paramedics arrived several minutes later and ALS was administered without success.

Autopsy: The heart weighed 460 g. There was 476 ml of blood in the pericardial sac and a rupture of the lateral wall of the left ventricle due to a recent (3–5 days) myocardial infarct. The coronary arteries showed generalised, severe atherosclerosis, with at least 90% stenosis in the first 1 cm and 50% to 70% more distally, but no thrombotic occlusions were seen. The kidneys showed granular changes. No other pathology was noted. The cause of death was given as cardiac rupture causing cardiac tamponade, secondary to a recent, unreported myocardial infarction.

Comment: This death could have occurred anywhere and at any time and just happened to occur while the victim was snorkelling. It seems surprising that three to five days after an unrecognised myocardial infarct this man felt well enough to go snorkelling.

Summary: 76-year-old male; recent myocardial infarction; recent chest symptoms; snorkelling alone; cardiac-related (sudden death from cardiac rupture).

BH 06/10

This victim was a 41-year-old male with a history of alcoholism and unreported snorkelling experience. He and his wife were snorkelling from a friend's boat off a popular island in temperate waters. The sea conditions were not reported.

The victim had snorkelled earlier that day without apparent problems. However, during this second snorkel excursion, the victim told his wife that he was "*not feeling well*" and swam back to the boat. He was seen to hold on to the side before his head slumped forward and he floated away face down. He was quickly dragged into the boat and rescue breathing was commenced en route to the jetty where the island nurse was waiting. BLS was unsuccessful.

Autopsy: His heart (462 g), showed concentric hypertrophy of the left ventricle. The LAD artery was noted to be almost completely occluded in the first 1 cm from its origin but thereafter had minimal atheroma, while the other coronary arteries had minimal atheroma. No thrombotic occlusion was noted. There was pallor of the posterior basal myocardium. There was nicotine staining of the fingers. The lungs (Rt 650 g, Lt 642 g) were plum-coloured, oedematous and congested. The formal finding of the coroner was "*coronary atherosclerosis in a man engaged in a marine activity*".

Table 2
Summary of scuba (SC) and rebreather (RB) diving-related
BSB – buddy separated before problem; BSD – buddy separated during problem;
GSB – group separated before; trng (com) – commercial diving training;

ID	Age	Sex	Height	Weight	BMI	Training	Experience	Dive group	Purpose
SC			(cm)	(kg)					
06/01	52	M	180	76	23.5	trained	experienced	solo	work
06/02	72	M	172	97.5	33.0	trained	experienced	GSB	recreation
06/03	35	M	167	73.5	26.4	trained	some	BSB	trng (com)
06/04	64	M	176	107	34.5	trained	some	BSD	recreation
06/05	40	M	175	89	29.1	trained	some	solo	work
RB									
06/01	31	M	181	104	31.7	trained	experienced	BSD	recreation

Comment: Only the police summary of witness statements is available and there is no information concerning his recent health.

Summary: 41-year-old male; history of alcoholism; unknown experience; felt unwell while snorkelling; cardiac-related

Scuba diving fatalities

SC 06/01

This victim was a 52-year-old male with a medical history of hypertension, although he was not taking any medication for this. He was an experienced diver who had been scuba diving since his early twenties, but he had dived rarely over recent years.

On this occasion, he decided to clean the hull of his boat, which was moored in a sheltered inlet, and a friend remained on board to assist with various ropes. He was wearing a wetsuit but no weight belt; however, he placed four weights (a total of 9.8 kg) in his BCD pockets. The depth was 3 msw and visibility less than one metre.

He was seen to surface very shortly after his water entry. He then redescended, before surfacing again about 5 metres from the boat, this time without his mask and with the regulator out of his mouth. He called out for help and his friend threw him a rope, but he had already submerged again. The friend entered the water and attempted to locate him, but he failed to see him in the muddy water. Police divers were called and they located him two hours later about 20 metres from where he had last been seen. His mask and snorkel were nearby and one fin was missing.

The equipment was tested and described as being in poor condition. The contents gauge showed the tank (which was out of test) was filled to 230 bar, slightly over its correct pressure, indicating the victim had taken few breaths from it after entering the water. The weights in the BCD pockets were excessive and could not be ditched in an emergency. The lost fin was recovered and its buckle strap noted to be undone, while the other was capable of easily dislodging. The fins were described as too floppy to provide effective thrust. The low-pressure corrugated hose had snapped off the BCD but it is unknown if this occurred while the victim was alive or during the retrieval of his body.

Autopsy: The heart weighed 400 g, and there was a focal 60% narrowing of the proximal LAD artery. The left ventricular wall thickness was 16 mm, possibly associated with the victim's hypertension, which was of unknown severity. The lungs were overinflated and heavy (Rt 1,080 g, Lt 1,040 g) and the upper airways contained frothy fluid consistent with drowning. No CT-scan imaging was done, though occasional bubbles were detected in the aorta but not in the pulmonary or coronary arteries. An area of blotchy red discolouration was noted over the left side of the forehead, and irregular areas of loss of skin surface on the dorsum of the left hand. A carboxyhaemoglobin level of 10% was detected in his blood. The cause of death was given as drowning.

Comment: The sudden loss of mask, regulator and fin may have led to drowning and there were injuries on the left forehead and left hand that may have been the result of contact with the hull of the boat. Carboxyhaemoglobin levels of 10% can occasionally be observed in heavy smokers. Gas analysis of the cylinder was normal for carbon monoxide (< 0.0002%), indicating that there was no contamination of the compressed air in the tank. Of the possible explanations, the most likely cause of death was drowning due to loss of equipment. This was possibly exaccerbated by an impact with the boat and an inability to ditch weights. This victim appears to have had little or no recent experience, was using poorly maintained equipment and was grossly overweighted according to police. Although the sequence of events is uncertain, it appears likely that his regulator became dislodged and he was unable to achieve positive buoyancy due to weights in the BCD pockets, the loss of a fin and/or possible BCD malfunction.

Table 2 (cont.)
fatalities in Australian waters in 2006
BCD – bouyancy compensation device; + sufficient air (to surface safely); ++ 1/4–1/2 full tank; +++ >50% full; nad – nothing abnormal discovered; CAGE – cerebral arterial gas embolism

Depth (msw)	Incident (msw)	Weight belt	Weights (kg)	BCD	Remaining air	Equip test	Disabling injury
3.5	surface	nil	9.8 (in BCD pocket)	defective	+++	defective	Asphyxia
17	ascent/surface	off	not stated	removed	not stated	nad	Asphyxia
12	12	nil	7.25 (integrated)	not inflated	nil	nad	Asphyxia
12	5	on	7.5	inflated	++	nad	CAGE
11.5	surface	on	11	not inflated	++	nad	Asphyxia
76	surface	not stated	not stated	not stated	+	nad	Asphyxia

Summary: 52-year-old male; experienced but likely little or no recent experience; poorly maintained equipment; weights in BCD pocket and broken fin; overweighted and unable to achieve buoyancy on surface; drowning

SC 06/02

This very experienced 72-year-old male had conducted over 1,000 dives in 30 years and continued to dive regularly. He was obese (BMI 33), had a residual limp from polio as a child, and history of six idiopathic nocturnal seizures eight years earlier. These were eventually ascribed to work stress and he subsequently retired. He was prescribed carbamazepine but it is uncertain whether he was still taking this at the time of the incident. No further seizures were recorded. Mild hypertension had been noted at the time of the seizures.

The victim made a boat dive with six of his usual dive group. They initially remained roughly together while inspecting the rocky area at a depth of about 17 msw. After about 30 minutes, they ascended to a depth of 9.5 msw, where there was a passage under a large boulder. A strong surge passed through this archway and then upwards, and four of the group, including the victim, decided to allow the surge to take them through it. The victim was last seen alive as he entered the arch. A witness on another boat later told of seeing the victim surface and look around as if to get his bearings. He was showing no signs of distress and the witness left the location – which was close to where the victim's body was later found floating – unaware of any problem.

When the victim failed to rejoin his friends, they searched for him and soon found two packets of his integrated weights and then one fin at a depth of 5 msw. However, when they could not locate the victim they notified the police. About an hour later his backpack and cylinder were found floating by a lifesaver on a surf ski, and the body was then seen floating unconscious and apnoeic about 50 metres further away. Subsequent BLS was unsuccessful.

Autopsy: An X-ray was taken before commencing the autopsy, and there was no evidence of any significant air in the heart or any other injuries. The heart weighed 440 g and appeared normal. There was atherosclerotic narrowing of the LAD and right coronary arteries of about 30%, and the left circumflex had about 50%. The trachea and bronchi contained frothy fluid and the lungs showed congestion and oedema. The histology showed moderate liver steatosis, the stomach had evidence of a gastrointestinal stromal tumour, and there was mild perivascular fibrosis and thickening of the walls of the intramural vessels in the myocardium. There was diverticular disease in the lower colon. There were scars anteriorly on both shoulders and a vertical scar on the lower abdomen of unknown origin. The cause of death was given as drowning.

Comment: There is insufficient information to determine why this man drowned. Whether the loss of a fin had any influence on the course of events is uncertain, and it is unknown when and why he ditched his weights and BCD. Seizures can lead to drowning but there is no evidence to support it in this case and the clinical history is vague. There was no evidence of any trauma to his head other than a small (2 mm) laceration over his right temple.

Summary: 72-year-old male; experienced; history of polio, seizures, hypertension; dived through underwater passage with strong surge; ditched weights; later found floating on surface; drowning (from unknown cause)

SC 06/03

This 35-year-old male had undergone rescue diver training four years earlier and logged 18 dives during the previous year, although it is not known how much overall diving experience he had. He was apparently in good health, with no known history of medical problems, and appeared to be very fit.

He was undertaking a commercial diving training course in a fresh-water quarry in which there was very low visibility

and a muddy bottom. The victim and another student were practising locating and raising a concrete-filled drum at a depth of about 12 metres' fresh water. He and his buddy were each wearing a full-face mask, suit, harness and lifeline with voice communications. His air supply was from a 2,500 L cylinder, with a 700 L pony bottle attached valve-down as a reserve supply. The victim's reserve was configured so that his primary air supply could be almost exhausted prior to accessing the reserve. However, the buddy's configuration was such that he would need to access his reserve supply when his primary supply fell to around 60 bar.

The victim and his buddy commenced the dive and located the drum. However, when they tried to inflate the lift bag they found that there was no air in the dedicated inflation cylinder and returned to the shore to report the situation. At that point, the victim's contents gauge read 110 bar and the buddy's 120 bar.

The instructor advised them to abort the lift, but to tie a marker buoy to the drum and return. They set out to do this, but after a short time and a 30-metre surface swim the buddy needed to activate his reserve. He reported this to the instructor and was told to return to shore, which he did. The victim was also called back as his buddy was returning and he agreed to do this. However, when the handler of his lifeline was pulling in the 'slack' as the victim returned, he felt a resistance. When he again pulled, the victim asked him to stop pulling and the handler fed back additional slack on the line. Within 20 seconds there was an urgent, but barely intelligible call for help and the sound of water entering the mask.

The instructor dived straight into the water with a mask and knife, but was unable to locate the victim. Eventually, another person located the victim, who was not wearing his mask, and managed to lift him to the surface after some initial resistance from the attached and entangled lines.

It was estimated that a period of 7 to 12 minutes elapsed from the cry for help until the diver was brought to shore. BLS was promptly commenced by trained staff and continued until the ambulance arrived shortly afterwards. Paramedics provided ALS, although defibrillation was not attempted as the victim was in asystole. The paramedics noted palpable gas within the subcutaneous vessels. He was transferred to hospital where he was soon pronounced dead.

Police divers later checked the equipment, which was found to be functional. While the primary tank was empty, the pony bottle was almost full (190 bar) but was turned off. It was difficult to reach and open the valve of the reserve, particularly if it had been firmly turned off. the victim was wearing a borrowed BCD with 7.25 kg of integrated weights.

Autopsy: A CT scan was performed at the local hospital within 4 hours of death. This was reported to show gas filling the arterial system. Review of this CT scan showed a large amount of gas in the left ventricle and arch of the aorta, and in the carotid, cerebral and subclavian arteries. There was less gas in the right ventricle. There was gas in the vessels of the liver. There was some gas in the chest wall and arms but this was probably in blood vessels. The preponderance of gas in the arterial system is not typical for post-mortem off-gassing and suggests cerebral arterial gas embolism (CAGE). X-rays taken prior to autopsy the next day also showed significant intra-arterial gas, but also more gas in the right ventricle.

The pathologist noted an absence of interstitial emphysema. When he opened the pericardial sac the distended heart bulged out and when a needle was inserted, clear gas was aspirated from both ventricles. The heart weighed 420 g. There was minimal coronary atheroma. There was a small amount of frothy, lightly blood-tinged fluid in the trachea and bronchi and more blood-stained fluid in the lower bronchial tree. The lungs (Rt 960 g, Lt 740 g) were well aerated and moderately oedematous. The pathologist attributed the gas seen mostly to peri-mortem "*extra-alveolar air syndrome*" caused by rapid ascent of the unconscious diver with a closed upper airway during recovery, possibly with some post-mortem off-gassing. However, the cause of death was attributed to drowning in an out-of-air situation when the mask had been lost.

Comment: The finding of large amounts of almost entirely intra-arterial gas so soon after death is not typical of post-mortem off-gassing and is difficult to explain with the history as given. Whilst laryngospasm secondary to aspiration has been reported, this ceases with increasing hypercapnia and hypoxia, leaving the airway open.[4] This diver was recovered after approximately 7 to 12 minutes, so he would have been deeply unconscious and hypoxic at the time of the recovery attempts. One would have to postulate positional airway obstruction during the ascent rescue efforts to explain the arterial gas if it was generated during the recovery. Whilst the finding of arterial gas is suggestive of barotrauma, the history of entrapment and the difficulty experienced by the rescuer in releasing the victim would tend to militate against this hypothesis. The presence of large amounts of arterial gas at autopsy could represent a peri-mortem CAGE after drowning due to an out-of-air situation. In the end, whether the death was the result of drowning or gas embolism is a moot point compared to the larger issues surrounding his inability to deploy his emergency gas supply.

It is likely that the victim exhausted the supply in his main cylinder and was insufficiently experienced to manage the reserve air unit he was wearing while dealing with the added stress of his lifeline being snagged. Zero visibility and entanglement would likely have exacerbated the out-of-air situation. It is also possible that his mask might

have become dislodged while he was attempting to reach and open his pony bottle. Practice in managing a situation such as this was an important element in the course being conducted, but being trapped may have led to panic and subsequent drowning. The instructor's decision to tell the divers to make a second dive on the available air supply proved to be costly.

Summary: 35-year-old male; healthy and fit; some experience; undertaking commercial diver training; entanglement; out of air; apparently unable to open reserve; mask dislodged; drowning

SC 06/04

This 64-year-old male from overseas, was a guest on a liveaboard dive vessel on the GBR. Although he had completed more than 100 dives since being certified 20 years earlier, it appears that he had dived infrequently, if at all, during the past three years. He declared that he had no medical conditions and was described as "*looking fit*", reportedly doing vigorous exercise for 1½ hours daily. However, he was obese and his luggage was later found to contain several medications which included doxazosin 2 mg, naproxen sodium 250 mg, finasteride 5 mg, cetirizine hydrochloride and montelukast sodium 10 mg.

This was the first dive of the trip. There were to be four divers in the group; the instructor, two inexperienced divers, and the victim, who was wearing full scuba gear, including a wetsuit and weight belt with 7.5 kg. There was a slight swell and current. Early in the dive, one of the inexperienced divers had a problem and the instructor assisted her, while the victim and the other diver continued for a short time together. After reaching a maximum depth of approximately 12 msw, the victim signalled to turn back and they slowly ascended to 5 msw where the victim knelt down and grasped the coral for a couple of minutes. He appeared to be mildly confused or distressed and possibly also clutched at his chest before inflating his BCD and ascending rapidly to the surface.

When his buddy surfaced 30 seconds later, the victim's eyes appeared glazed and he soon fell face down in the water. The buddy rolled him onto his back, found him to be unresponsive and alerted the dive boat some 60 metres away. A tender was quickly sent, but the crew were unable to drag the victim on board immediately because of the inherent difficulties bringing an unconscious diver on board a vessel. After another few minutes, and with additional assistance, he was successfully brought onto the tender and was assessed to be unconscious and apnoeic, with a faint, erratic pulse. Rescue breathing was commenced and this was replaced with CPR with supplemental O_2 once on board the main vessel. BLS was continued by crew and two guest doctors for 40 minutes, prior to ALS being provided by a paramedic who was air lifted to the vessel. However, resuscitation attempts were unsuccessful.

Autopsy: A total body CT scan was done within 24 hours of retrieval, which was reported to indicate gas embolism in the heart and major vessels. At autopsy, some gas was found in the right atrium, less in the right ventricle, but no surgical emphysema or any other intravascular air or pulmonary pathology was noted. The heart showed cardiomegaly, weighing 533 g with concentric hypertrophy of the left ventricular wall. There was generalised coronary arterial atherosclerosis but no occlusions, the maximal narrowing of the lumen being 40% in the LAD branch. Histology of the heart showed widespread ischaemic fibrosis. He was obese, BMI 34.5. Several ribs had been fractured during the resuscitation efforts. The cause of death was given as cardiac air embolism due to barotrauma. The pathologist considered that a cardiac event related to the enlargement of the heart and ischaemia could have precipitated the uncontrolled ascent.

Comment: The police summary includes an unsourced note of the victim possibly clutching his chest before inflating his BCD, his buddy's statement only mentioning his apparent confusion or distress. The clinical picture is consistent with his experiencing anginal pain and responding with a panic ascent, resulting in CAGE. The pathology would fit with this, despite the lack of a critical coronary occlusion. It is important to note that it is often difficult to bring an unconscious diver aboard a vessel. Divers and boat operators should have a pre-planned and, ideally, well-practised system to do this, taking into consideration the number of people likely to be able to assist, as well as the size of and access to the vessel.

Summary: 64-year-old male; experienced although few or no dives for past three years; taking medications for hypertension, asthma, allergy; cardiomegaly and atherosclerosis; aborted dive early and made a rapid ascent to surface; CAGE (possibly precipitated by cardiac event)

SC 06/05

The victim was a 40-year-old, apparently healthy male who was the skipper of a crayfishing boat. He had certified as a recreational diver 28 years earlier but his subsequent diving experience is unknown.

He was planning to dive to retrieve his boat's anchor, which had been lost at sea several days earlier at a depth of approximately 10–12 msw. After hiring scuba equipment at a local dive store, he set out to the site. The conditions were described as calm. A shot-line was dropped where he believed the anchor to be. Prior to entering the water, the victim shackled a rope to his BCD. The plan was to attach this rope to the anchor and use it to drag it up when back on the boat. He appeared to be very anxious prior to entering the water. His crew member remained on board to manoeuvre the boat as required. The victim submerged and the crew member watched the rope feed out as the victim descended.

The boat drifted away from the shot-line several times and the crew member repositioned it.

After what he estimated was 10–20 minutes, the crew member noticed that the victim had surfaced and appeared to be "*in serious trouble*". He drove the boat to the victim who appeared to be unconscious and had partly submerged by then. He managed to hook the victim's BCD with a gaff and dragged him to the side of the boat although he was unable to bring him fully on board. He then dragged the victim to the rear of the boat and eventually, with the use of a winch, managed to lift the victim onto the transom where he attempted BLS, without success.

The equipment report indicated that the equipment was functioning adequately and had no major faults. The remaining air was 120 bar, the maximum depth reached was 11.5 msw, and the recommended ascent rate had been exceeded (this indicator is activated at any rate faster than 9 m min^{-1}, so the actual rate is not known). The victim had worn a sleeveless full-length wetsuit, a weight belt with 8.2 kg, and carried an additional 2.7 kg of weights in the pockets of his BCD.

Autopsy: The management of this death was unusual in that, after the victim was brought ashore and declared to have died, the body was taken to a hospital and CT scan of the chest was performed. This did not show the presence of intravascular gas. A further radiographic check later, pre-autopsy, confirmed this. The heart weighed 390 g and coronary arteries showed no significant narrowing. The lungs were over-expanded and heavy (combined weight 1,589 g) and oedematous, and there was oedema in the larynx, trachea and bronchi. The cause of death was given as drowning.

Comment: The clinical history in this case is suggestive of CAGE, but this appears to be excluded by the negative CT scan. This incident was investigated by the local workplace authority as a workplace-related death. The management of the investigation by the local police was criticised for the failure to follow a protocol, which required involvement of a diving medicine expert at the autopsy. Neither health nor equipment factors can be faulted but inexperience may have led to a panic ascent.

Summary: 40-year-old-male; no known medical history; certified diver for 28 years but unknown experience; hired equipment; diving alone to retrieve anchor; surfaced and became unconscious; drowning

Rebreather fatality

RB 06/01

This report lacks vital details because the victim's family refused permission for access to the full coronial documentation. However, some key witnesses provided important information. The victim, a 31-year-old male, was an experienced technical diver with extensive experience in open-circuit trimix diving. He had 20 years' diving experience with 75 dives in the past year. He cycled regularly, went to the gym twice-weekly for weight training, and was described as "*fit and healthy*", without any known medical conditions.

He had trained in the previous year on a 'PRISM' electronic closed-circuit rebreather (CCR) using air diluent. However, due to the unreliability of this unit he had recently retrained in the use of an 'Inspiration' CCR. This rebreather differs from the former in a number of key areas, including the direction in which the gas circulates around the breathing loop and the placement of the O_2 and diluent cylinders and their manual injection buttons on opposite sides. The victim had reputedly had these differences highlighted to him during the retraining and had been encouraged to gain experience with the unit conducting air dives only. He had apparently conducted several dives in the 40–50 msw range on the unit after the course.

The victim and two friends were planning to dive from his boat, with another friend remaining on board as boat operator. The dive site was a wreck at a depth of 75 msw located 5 km offshore. Conditions were choppy and, as the victim was suffering from seasickness, it was decided that he would enter the water first in an attempt to alleviate his symptoms. A shot-line with an attached 'mermaid' line was dropped at the site and the dive boat was anchored. The victim was assisted in gearing up and entered the water with one of his buddies. The victim soon complained that he felt nauseous and decided to abort the dive. As the boat had now drifted some distance away, the pair decided to wait at the shot-line buoy until it returned to drop off the third diver.

The buddy turned to watch the approaching boat and then turned again to check on the victim, only to find him underwater at about 5 msw, apparently unconscious and sinking rapidly. He immediately tried to descend but had to return to the surface as, in his haste, he had failed to allow sufficient air out of his drysuit. He called to the other diver on the boat and when he entered the water, the two descended to try to reach the victim. After descending to 55 msw without sighting the victim and with visibility rapidly deteriorating, they decided to ascend as the second diver was now experiencing problems with his CCR. The buddies ascended, boarded the boat and notified the emergency services.

A subsequent search using boats and aircraft failed to locate the victim. His body was eventually found by a diver eight days later at 75 msw depth with all equipment in place.

Autopsy: The autopsy reported the cause of death as drowning following loss of consciousness from an unknown cause.

Comment: The use of hypoxic diluents with CCRs makes the possibility of becoming unconscious particularly likely at the surface. Because of this, CCR divers are usually encouraged to purge the unit with oxygen if they are on the surface while waiting to return to the boat, exerting themselves or feeling unwell. The fact that the unit that the victim was using on this occasion had the diluent and oxygen manual addition buttons on opposite sides to that of the unit on which he had originally trained may well have contributed to his death as, if he had flushed with diluent (150 L min^{-1}), it is unlikely that the electronic controlled injection of oxygen would have kept up. Why no alarms were heard by his buddy remains a mystery, although one possible explanation is that the 'buzzer' on the CCR was underwater and hence the sound was not transmitted across the water/air interface.

Another theory that has been put forward to explain this death is that the unit, an early production 'Inspiration' units, may have suffered from battery 'bounce' as the victim rolled backwards into the water from the boat, resulting in the electronics turning off and back on into 'no-dive mode', in which case the CCR would no longer automatically add O_2. While this was a known problem with early 'Inspirations', a factory fix had been available for several years. There is no mention whether this 'fix' had been applied to this unit. In any case, correct procedure and vigilance by the victim should have avoided either of these scenarios.

The combination of stress, motion sickness and task loading would appear to have distracted the victim and resulted in him failing to adequately monitor his O_2 partial pressure; hence he became hypoxic and lost consciousness. Once the mouthpiece had fallen from his mouth, the subsequent loss of buoyancy as the CCR flooded resulted in him sinking rapidly. It is interesting that the buddy was shocked by how quickly these events unfolded. The issue of vigilance when using a CCR and the importance of correctly retraining experienced open-circuit divers about the differences between open-circuit scuba and a CCR cannot be understated.

Summary: 31-year-old male; apparently fit with no known health conditions; experienced technical rebreather diver; recent change to different rebreather type; probable seasickness; possibly breathed hypoxic mix by mistake; became unconscious and sank; drowning

A summary of the possible sequence of events in these 16 fatalities is shown in Table 3.

Discussion

The main purpose of these ongoing fatality reports, as stated in the introduction, is to highlight problems so that similar events can be minimised in the future. This report again details potentially avoidable diving accidents, but also some cardiac-related deaths that were likely to have been unavoidable and could have occurred in non-diving circumstances.

BH 06/01 was one of the deaths that was likely to have been avoidable. Stingrays are generally relatively placid and attacks on divers, snorkellers and swimmers are rare and usually the result of the creature trying to escape or defend itself, rather than attacking without provocation. There are only 17 well-documented deaths due to stingray injuries up till 1996, although there are likely to have been many more that were not adequately documented.[1,5] Such fatalities are usually due to trauma, severed arteries, or infection and rarely from the direct effects of the venom. This was the third recorded stingray-related fatality in Australia, the others being in 1945 and 1989.[6–8] All three deaths resulted from chest penetration by the barb.

Unlike the two previous annual reports, there were no breath-hold fatalities in 2006 thought to have been associated with apneic blackout. Among the snorkelling group, chiefly identified from the GBR area, five of the ten victims were overseas visitors. Once again, there were a number of cardiac-related deaths, mainly in snorkellers, 55 years or older, with pre-existing cardiovascular disease, whether previously identified or not. These included BH 06/02, BH 06/04, BH 06/05, BH 06/06, BH 06/09 and BH 06/10. In only one (BH 06/05) was there any reason to have suspected ill health and in none was strenuous exertion a factor. One scuba diver (SC 06/04) possibly suffered cardiac pain prior to a rapid ascent during which he sustained a CAGE. Of interest, in three of these cases that occurred on the GBR, AEDs were available (BH 06/02, BH 06/ 03 and BH 06/04). The availability of AEDs on dive boats in Queensland is encouraged by the local Workplace Health and Safety Regulations and has the potential to save lives in the right circumstances. However, in all these cases, the AEDs indicated that no shock was required, so it was likely that ventricular fibrillation had not occurred or, more likely, had already ceased by the time the AEDs were attached.

The main factor determining the chances of survival after many cases of cardiac arrest is how quickly sinus rhythm can be restored. CPR plus defibrillation within 3–5 minutes of cardiac arrest can produce survival rates as high as 49–75%.[9] Therefore, the inevitable delays in recognising that a victim has become unconscious in the water, bringing them to boat or land, commencing CPR and retrieving and attaching an AED all reduce the likelihood of success. Such delays occurred in each of these cases, so the likelihood of a successful outcome was low.

In three cases (BH 06/04, SC 06/04, SC 06/05), there were great difficulties and delays in bringing the victim aboard the boats. This can often be a problem, depending on the size of the victim, the amount and type of equipment worn, the number of rescuers available and the type of boat. It is important to have a pre-planned, time-efficient, effective and adequately practised system in place to deal with such a situation. There are commercially made devices specially designed to facilitate the easy removal of a diver from the

Table 3
Root cause analysis of diving-related fatalities in Australian waters in 2006

Case	Trigger	Disabling agent	Disabling injury	Cause of death
BH06/01	Startling stingray	Stingray barb injury	Trauma	Cardiac tamponade
BH06/02	Unknown (exertion?)	Cardiovascular disease	Cardiac incident	Cardiac-related
BH06/03	Unknown	Unknown (cardiac?)	Asphyxia	Drowning
BH06/04	Unknown	Cardiovascular disease	Cardiac incident	Cardiac-related
BH06/05	Unknown (exertion?)	Cardiovascular disease	Cardiac incident	Cardiac-related
BH06/06	Unknown	Cardiovascular disease	Cardiac incident	Cardiac-related
BH06/07	Unknown (water inhalation from snorkel?)	Sudden loss of consciousness (laryngospasm?)	Asphyxia	Drowning
BH06/08	Unknown (epilepsy?, methamphetamine?)	Sudden loss of consciousness	Asphyxia	Drowning
BH06/09	Previous myocardial infarction	Cardiovascular disease	Cardiac incident (cardiac rupture fr. recent infarct)	Cardiac-related
BH06/10	Exertion	Cardiovascular disease	Cardiac incident	Cardiac-related
SC06/01	Unknown (loss of equipment/panic?)	Loss of regulator/mask due to mild trauma?	Asphyxia, ascent-related?	Drowning
SC06/02	Unknown (epilepsy?)	Unknown	Asphyxia	Drowning
SC06/03	Entrapment, gas-supply-related	Gas-supply-related	Asphyxia	Drowning
SC06/04	Unknown (exertion?)	Ascent-related, cardiovascular disease	CAGE	CAGE
SC06/05	Unknown (anxiety/panic?)	Ascent-related	Asphyxia	Drowning
RB06/01	Seasickness	Equipment-related	Asphyxia	Drowning

water by a single rescuer, ideally in the horizontal position (e.g., Jason's Cradle™).[10]

Two drowning deaths occurred in individuals who had a history of epilepsy (BH 06/08 and SC 06/02). It is unknown whether or not epilepsy contributed to these deaths, but this is always a consideration in an aquatic environment. Epilepsy is regarded as a contra-indication to breath-hold and scuba diving.[11]

Failure to gain adequate buoyancy quickly in an emergency is often identified as an issue in dive fatality reports, and this year was no exception. SC 06/01 was excessively overweighted and carried all his weight in his BCD pockets, from where it could not be readily ditched. It is also possible that his BCD was not functioning adequately and that his fins provided little thrust. Had he been able to attain and maintain positive buoyancy when he initially surfaced, there should have been a greater likelihood of survival. It has been claimed that divers who manage to reach the surface promptly in an emergency, whether conscious or unconscious, have a greater chance of survival than those who need to be sought out and retrieved from underwater.[12]

Once again, solo diving, coupled with lack of recent experience, may well have contributed to the deaths of two scuba divers (SC 06/01 and SC 06/05). It is important that divers have ready assistance available throughout the dive. It is also important that divers without recent experience return to diving in appropriate circumstances, with adequate supervision and support.

Commercial diver training inherently involves teaching participants how to prevent, as well as how to deal with, potential problems while using commercial diving equipment and performing various tasks underwater. Case SC 06/03 likely highlights the importance of not only having a redundant gas supply, but being able to quickly and easily

access it. Course participants vary greatly in diving training and experience and close supervision is always important, especially early in the training while they are familiarising themselves with their equipment. This case also highlights the importance of having a standby diver appropriately equipped and ready to enter the water with minimal delay in the event of an emergency.

The problem of whether to make a diagnosis of CAGE in the absence of physical evidence is noted in one case. (SC 06/05). Many dive sites are remote, and it may be difficult to get a CT scan of the body within 8 hours of death, although this remains the most useful test for CAGE. Problems with decomposition and post-mortem off-gassing make scan results unreliable after this time. The history of a rapid ascent followed by loss of consciousness is a very important factor in the diagnosis of CAGE, but if a CT scan has been done and is negative it is hard to justify the diagnosis of CAGE.

Technical diving and, in particular, rebreather diving are rapidly growing areas of recreational diving. CCRs are complex pieces of equipment with a number of potential failure modes that are not necessarily familiar to even the experienced open-circuit scuba diver. An open-circuit scuba failure will usually result in the diver being unable to breathe. In contrast, a diver using closed or semi-closed rebreather scuba will often still be able to breathe despite a failure in the unit, which may result in a non-life-sustaining gas mixture being delivered. The subtle onset of such failures can result in a loss of consciousness with little to no warning. As such, these divers must be ever vigilant as to the partial pressure of O_2 they are breathing.

Case RB 06/01 highlights the importance of divers being thoroughly familiar with the CCR that they are using, including the gas mixes utilised on a particular dive. It also highlights that CCR divers should inflate other buoyancy devices when on the surface and not rely on their counterlung as a primary buoyancy device.

It was noted during the preparation of these case reports that the practice of police reporting a summary of the evidence, rather than including copies of the witness statements significantly degrades the value of the reports as a complete resource for research and the subsequent discussion of critical factors in fatal dives, even though there may be valid reasons for doing this.

Conclusions

In 2006, there were 16 reported diving-related fatalities, which included 10 deaths while snorkelling, five while diving on open-circuit scuba and one while using a CCR. Causal or contributory factors associated with these deaths included cardiac disease or other co-existing illnesses, inexperience or lack of recent experience, and injury from a stingray.

The main disabling injury with snorkellers was cardiac-related episodes (six of ten cases), followed by asphyxia leading to drowning (three cases). With scuba divers, the main disabling injury appears to have been asphyxia, leading to death by drowning in five of the six cases. CAGE was identified as the likely disabling injury in the other death, although it is thought that there may have been a cardiac-related disabling agent to this incident. A summary of the possible sequence of events in these 16 fatalities is shown in Table 3.

Factors that may reduce mortality in the future include improved medical screening of older divers, improved supervision of snorkelling activities, avoidance of close contact with potentially dangerous marine creatures, such as stingrays, increased adherence to the buddy-diving system and attention to the integrity of and familiarity with the equipment being used.

Conflict of interest

John Lippmann is the Executive Director of DAN AP. 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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Risk factors for running low on gas in recreational divers in Western Australia

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Key words
Low air, risk factors, recreational diving, scuba diving, epidemiology, research

Abstract
(Buzzacott P, Rosenberg M, Heyworth J, Pikora T. Risk factors for running low on gas in recreational divers in Western Australia. *Diving and Hyperbaric Medicine*. 2011;41(2):85-9.)
Introduction: The aim of this study was to investigate risk factors associated with running low on gas among certified recreational divers.
Methods: Dive and diver information were collected from divers joining organised recreational dives in Western Australia and depth/time loggers were attached to each diver. Case dives ending with < 50 bar in the cylinder were compared with control dives made at the same dive site and time by divers with ≥ 50 bar remaining. A conditional logistic regression model identified factors significantly associated with running low on gas.
Results: In total, 1,032 dive profiles were collected. Case dives (n = 183) returning with < 50 bar were compared with 510 control dives ending with ≥ 50 bar. Perceived workload was associated with rate of consumption of gas. Factors associated with a dive ending low on gas included: younger age; males; lower number of lifetime dives; a longer period since last dive; deeper maximum depth and breathing at a heavier rate. Eleven per cent of case divers, compared with 1% of control dives, reported surprise at the low remaining level of gas.
Conclusions: Dive organisers are recommended to select dive sites based on the recent experience of the group and to encourage divers to monitor their remaining gas frequently, relative to the depth of the site. Divers are reminded that, if they perceive a strenuous workload, they should pay even closer attention to monitoring their gas reserves. That 89% of low-on-gas dives were reported to be no surprise to the divers making them warrants further investigation.

Introduction

Running low on gas has been implicated in diving morbidity and mortality.[1,2] An analysis of 859 reported diving incidents in Australia found that 168 (19.5%) involved an out-of-gas problem, 57 (35%) of which resulted in diver harm.[3] A survey completed by 515 Western Australian divers in 2000 found that 19% divers reported running low on gas.[4] A 2005 survey of Western Australian recreational divers reported running out of gas and making for the surface among 7% of respondents, sharing gas with a buddy among 9% and making an emergency ascent among 9%.[5]

To reduce the likelihood of running out of gas, the Western Australian Code of Practice for Recreational Divers specifies "*Certified divers should be briefed on:... the need to regularly monitor gas levels in gas cylinders and note minimum gas content requirements for a safe return to the surface*".[6] Dive leaders in Western Australia (WA) commonly set the minimum reserve in the cylinder with which to return at 50 bar, as many submersible pressure gauges (SPG) have the lowest 50 bar colour coded, usually red (Figure 1).

Excepting cases where entrapment is implicated, information on the reasons why divers run low on gas is limited. A Delphi survey of diving experts suggested the most likely reasons for recreational divers to run out of gas were, in order:

- failing to monitor the gauge;
- inexperience;
- overexertion/strong current;
- inadequate training;
- poor dive planning;
- panic/anxiety/stress;
- diving deeper than usual.[7]

A recent cross-sectional analysis of 52,582 open-circuit scuba dives made by 5,046 adult recreational divers found that divers who reported running out of gas (n = 86) were more likely to be older and to be female.[8] Controlling for age and sex, and comparing the 86 out-of-gas dives to 1,207 normal dives made by the same group of divers, the study found that out-of-gas dives were more likely to have been deeper, shorter, made from a live-aboard or day boat and to

Figure 1
Submersible pressure gauge with lowest 50 bar coloured coded (usually red)

have involved a higher perceived workload.[8]

By controlling for the factors of depth, length and type of dive, the aim of this study was to further investigate other dive- and diver-related factors associated with finishing a dive low on gas among certified recreational divers. As perceived workload has been found to be significantly associated with the likelihood of reporting running out of air, we also hypothesized that a higher perceived workload would manifest as higher rates of air consumption.[8]

Methods

Adult, certified divers attending organised recreational group dives were recruited as previously described.[9] Briefly, dive businesses and dive clubs in WA were invited to participate. A researcher (PB) then met organised groups of recreational divers at popular dive sites around the coast of WA. The study was approved by the Human Research Ethics Committee of the University of Western Australia.

Dive and diver information were collected using a modified Divers Alert Network (DAN) Project Dive Exploration (PDE) survey questionnaire and Sensus Ultra data loggers (ReefNet, Ontario) were attached to each diver. Data collected included sex, age, weight, dive experience, certification level and problems experienced during the dive. Self-reported starting and returning gas pressures and stamped cylinder volumes were recorded on the dive record. Consumed gas volume was calculated by multiplying cylinder volume by the difference between starting and ending cylinder pressures, expressed as surface-equivalent air consumption (SAC, L min^{-1} kg body $weight^{-1}$).

ANALYSIS

Mean depth was calculated by dividing the total of recorded depths from each dive by the number of samples recorded between the time the diver left the surface (depth >1 metres' sea water, msw) and the time of returning to the surface (depth = 0 msw). This included divers swimming back to the boat underwater, but excluded time spent at the surface. For example, when taking a bearing back to the boat near the end of a dive, it is assumed that divers at the surface would have temporarily discontinued using scuba and breathed air from the atmosphere. SAC was calculated by dividing the gas volume used by the number of minutes spent underwater and by the mean ambient pressure in bar at the mean depth (excluding time at the surface, as described above). Trends in decreasing gas pressures and increasing depth were tested for significance using a general linear model.

To control for environmental conditions, dives in which a diver exited with < 50 bar in the cylinder were classed as 'case' dives, and dives made at the same dive site and at the same time by another diver that ended with ≥ 50 bar remaining were classed as 'control' dives. The data were imported into SAS version 9.2 (Cary, North Carolina) and the distribution of variables tested for normality. Bivariate analyses were conducted for each factor. Four variables with cell counts of less than five were excluded from further analysis. These were regulator malfunction (two cases), reported panic (four cases), reported severe workload (one case), and losing the weight system (one case). Twelve remaining factors were fitted to a conditional logistic regression model, which was achieved by numbering each organised dive consecutively and stratifying the regression by dive number. Non-significant associations ($P > 0.05$) were removed by backwards elimination.

Results

A description of the participants and range of diving conditions has been reported previously.[9] A total of 1,032 dives were recorded, of which 339 were made by groups where no diver returned with < 50 bar remaining. Case dives returning with < 50 bar ($n = 183$) were compared with 510 simultaneous control dives ending with ≥ 50 bar.

Dives made by females ($n = 199$, 29%) tended to be longer than those by males (55 versus vs. 49 mins, $P < 0.01$), although they reached similar maximum depths (mean 19.8 msw for females vs. 20.3 msw for males, $P = 0.36$). When divided by reported body weight, there was no difference between the sexes in mean SAC rates (males 0.22 vs. females 0.23 L min^{-1} kg^{-1}, $P = 0.70$). Females ascended 10% slower than males (10.7 vs. 11.8 m min^{-1}, $P < 0.01$), used smaller dive cylinders than males (11.5 vs. 11.9 L, $P < 0.01$) but returned with more gas remaining than males (80.0 vs. 68.8 bar, $P < 0.01$).

There were three methods of supervision employed by divemasters leading recreational dive groups: dives made from live-aboard vessels were supervised from the deck; dives made from the shore or a day boat were either supervised from the surface or personally guided in the water. The method of supervision had no effect on the likelihood of running low on gas ($P = 0.63$).

RUNNING LOW ON GAS

Case dives ($n = 183$) ended with a mean of 36.3 bar of gas remaining and control dives ($n = 510$) ended with twice as much, a mean of 75.9 bar remaining. Table 1 presents univariate comparisons between case and control dives. SAC was associated with running low on gas ($P < 0.01$, Table 1) and with perceived workload ($P < 0.01$, Table 2).

Divers reported keeping watch on their remaining gas pressure more often during the last half of the dive for case dives than for control dives (95% vs 91%, $P = 0.09$). Case divers were also more likely to report being surprised at the end of the dive by their remaining gas pressure (11% vs 1%, $P < 0.01$).

Table 1
Univariate associations with case dives (< 50 bar) and control dives (≥ 50 bar)
* Each risk factor modelled as a continuous variable per units indicated in parentheses

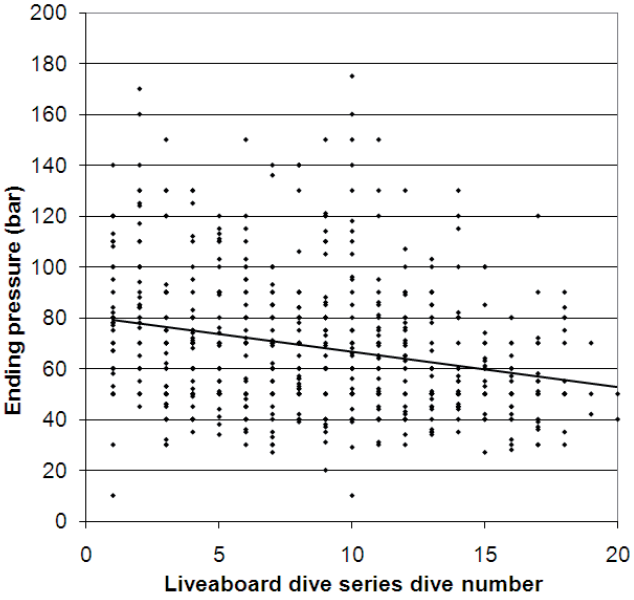
Risk factor	Cases	Controls	Univariate OR	(95% CI)	*P*-value
Younger age* (mean) (per 10 years)	39.0	41.8	1.37	1.07 to 1.74	<0.01
Male/female ratio	83:17	67:33	3.30	1.99 to 5.49	<0.01
Fewer dives last 5 yrs* (median; per 100 dives)	100 (range 0–1,500)	150 (range 5–1,500)	1.22	1.11 to 1.35	<0.01
Time since last dive* (wks; per year)	10.4	3.4	1.23	0.95 to 1.51	<0.01
Deeper average depth* (per 5 msw)	12.2	11.3	1.81	1.11 to 2.93	<0.01
Higher SAC* ($L\ min^{-1}\ kg^{-1}$)	0.25	0.21	1.07	1.04 to 1.10	<0.01
Smaller cylinder* (L)	11.5	11.7	1.45	1.11 to 1.91	<0.01
Surprised by low gas pressure (%)	11.0	1.0	10.42	3.58 to 30.30	<0.01
Low vs high certification (%)	73:17	58:35	2.35	1.46 to 3.76	<0.01
Fewer years of diving* (median)	6.0 (range 0–35)	10.0 (range 0–39)	1.60	1.27 to 2.02	<0.01
Fewer dives with BCD* (median; per 10 dives)	67.5 (range 0–1,100)	100.0 (range 0–1,100)	1.02	1.01 to 1.03	<0.01
Buoyancy problem (%)	8.0	3.0	2.33	0.99 to 5.52	<0.01

LIVE-ABOARD DIVERS

Among live-aboard dives (n = 656/1,032 dives by 44 divers), the median number of dives in each dive series was 15 (range 10–20). Returning gas pressures by dive number in live-aboard dive series are shown in Figure 2. The trend line indicates that returning pressures fell on average, over the course of each trip, and this trend was significant ($P < 0.01$). Maximum depth also increased significantly over the dive series ($P < 0.01$) as shown in Figure 3.

Mean maximum depth during dives one to three was 17.8 msw. By dives 17–20, after six days of diving, mean maximum depth was 25.3 msw. Maximum depths reached by divers who had dived within the previous 12 weeks and by divers who had not are shown separately in Figure 4. Divers who had dived more recently (n = 449) increased their maximum depth over the dive series whereas divers who had not dived during the previous 12 weeks did not (n = 207).

Figure 2
Decreasing remaining air over 656 dives in 44 dive series ($P < 0.01$)



MULTIVARIATE ANALYSIS

Twenty-six dives (4%) were not considered because of missing data, leaving 667 dives for multivariate analysis. Variables significantly associated with dives ending with < 50 bar are shown in Table 3. The four most significant risk factors for running low on gas were dives made by younger divers, being male, deeper average depth, and reporting surprise at how low the remaining gas pressure was at the end of the dive. However this last factor had a broad confidence interval, suggesting an imprecise estimate.

Table 2
Surface-equivalent air consumption (SAC) by perceived workload (L.min^{-1}.kg^{-1}) in the low-on-gas subset (n = 677)

	Perceived workload		
	Resting/light	Moderate	Severe
SAC	0.22 (0.07)	0.24 (0.08)	0.28 (0.06)

Discussion

Whilst a recent study found that dives running out of gas were associated with older females, this study found that dives ending with < 50 bar were more commonly made by younger males.[8] There are a number of plausible explanations for this disparity. The two studies were conducted in different geographic locations using different methods and this may have had an impact on their findings.

That divers returning low on gas were surprised by their remaining pressures supports the consensus of diving experts who suggested the most likely cause of running out of gas was failure to monitor the gauge.[7] The experts suggested the second most likely reason might be inexperience and, in this study, case dives were made by divers with 50% fewer dives during the previous five years (100 vs. 150), a longer period since previously diving (10.4 vs. 3.4 weeks) and lower median years of diving overall (6.0 vs. 10.0 y). An increasing SAC rate was significantly associated with increasing perceived workload in the low-on-gas case-control subset. A higher SAC rate was also significantly associated with case dives, thus supporting the expert opinion that overexertion is a likely cause of running out of gas.

Failure to discuss a returning gas pressure during the pre-dive plan was not included in the final model as there was little difference between case dives and control dives (71% vs. 74%). However, there is more to dive planning than simply agreeing a turn-around pressure. In this study, panic was not common enough to determine if it was significantly associated with running low on gas, but diving deeper than usual was found to be significant. Of the top seven reasons suggested why divers run out of gas, this study suggests the predictive tally of the expert panel to be four significant associations, one further association significant at the univariate level only, and two as yet unproven.[7] It should be acknowledged, however, that the expert panel considered running out of gas whereas this study investigated running low on gas. How non-participants may have differed to participants could not be investigated, nor was how self-organised dives might differ from professionally organised dives. Caution is needed in generalising these findings beyond the population sampled.

Another limitation of this study was the large proportion of dives made from live-aboard dive platforms, suggesting a sampling bias. However, there was no significant difference between live-aboard and day-trip divers in age, sex, height, weight, dive certification level, years of diving, number of dives, dive computer use or likelihood of being a 'case'. Furthermore, dive platform was controlled for by the study design. That divers on live-aboard vessels returned with decreasing gas pressures over the duration of their dive series was an unexpected finding, as was the fact that maximum depths concurrently increased among divers with more recent diving experience. If this pattern of increasing depth and decreasing gas reserves is observed on other live-aboard dive vessels then dive organisers should consider this likely trend when selecting each day's dive sites and in reiterating the need for divers to monitor their gas.

While 95% of divers running low on gas stated that they

Figure 3
Increasing maximum depth over 656 dives in 44 dive series ($P < 0.01$)

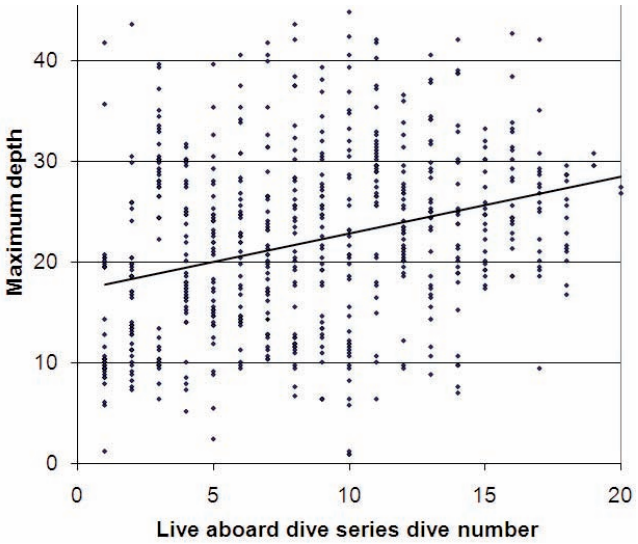


Figure 4
Maximum depth over dive series by recent experience status; dotted line – recently dived; solid line – not dived recently

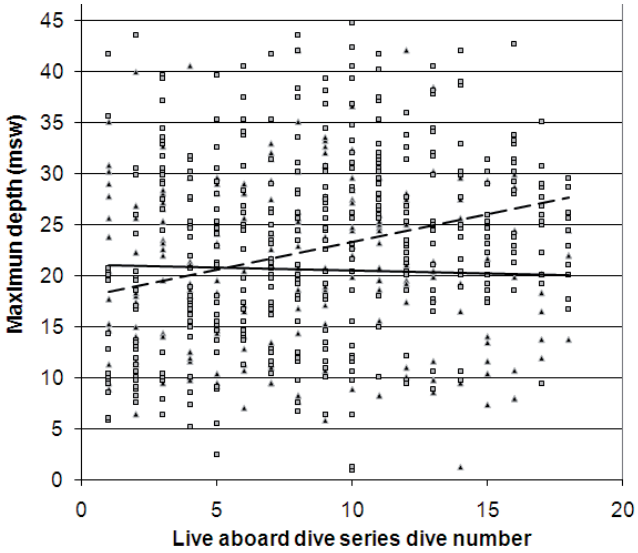


Table 3
Multivariate risk factors for running low on gas

Risk factor	Adjusted OR	95% CI	*P*
Male vs. female	13.51	6.41 to 28.57	<0.01
Deeper average depth* (per 5 msw)	3.46	1.85 to 6.48	<0.01
Younger age* (per 10 years)	2.02	1.47 to 2.77	<0.01
Surprised by low remaining gas	21.74	5.00 to 90.91	<0.01
Time since last dive* (per year)	1.51	1.11 to 2.06	<0.01
Fewer dives last 5 yrs* (per 100 dives)	1.22	1.00 to 1.49	0.01
Higher SAC* (per L min^{-1} kg^{-1})	1.14	1.09 to 1.19	<0.01
Warmth (warm vs. cold)	4.25	1.28 to 14.13	0.02
Cylinder volume* (per L)	1.01	1.03 to 1.90	0.03

* Variables modelled as per units indicated in parentheses

had kept a close eye on their remaining gas during the last half of the dive, 11% of them reported being surprised by how low their gas was at the end of the dive. Due to the limitations of this study it is difficult to interpret this finding. Did the 11% who were surprised not actually monitor their gauge, or were they surprised at how rapidly their reserve was depleted, and/or did the 89% who did not report being surprised consciously return with < 50 bar?

Conclusions

Dive organisers are recommended to select sites based on the recent experience of the group and to encourage divers to monitor their remaining gas frequently, relative to the depth of the site. This may be especially important over the course of live-aboard dive trips, when some divers appear to increase their maximum depth as the trip progresses. Perceived workload is correlated with SAC rate, and divers should be reminded that, if they feel as though they are working harder, it is likely they are consuming their gas faster than if they were drifting along relaxed. In that circumstance, even closer than normal attention should be paid to the SPG. Lastly, divers are reminded to heed the advice of dive organisers and to turn their dives before the SPG needle enters the red zone.

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Technical report

Solid-state electrolyte sensors for rebreather applications: a preliminary investigation

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Key words
Rebreathers/closed circuit, oxygen, carbon dioxide, transducer, capnography, helium, trimix

Abstract
(Sieber A, Baumann R, Fasoulas S, Krozer A. Solid-state electrolyte sensors for rebreather applications: a preliminary investigation. *Diving and Hyperbaric Medicine*. 2011;41(2):90-6.)
Introduction: Recently developed prototypes of zirconium dioxide and NASICON-based micro solid-state electrolyte oxygen (O_2) and carbon dioxide (CO_2) sensors were tested for their potential suitability in rebreathers. The O_2 sensor has a quasi-indefinite lifetime, whilst that of the CO_2 sensor is approximately 700 h. This is a preliminary report of a new technological application.
Methods: The O_2 sensor was tested in a small pressure chamber to a partial pressure of oxygen (PO_2) of 405 kPa (4 bar). The CO_2 sensor was tested up to 10 kPa CO_2. The response times to a step change of pressure were measured, and cross-sensitivity for helium tested using trimix. A rebreather mouthpiece was modified so that breath-by-breath gas recordings could be observed. Power consumption to heat the sensors was measured.
Results: The O_2 sensor demonstrated non-linearity, particularly above 101.3 kPa (1 bar) PO_2, whereas the output of the CO_2 sensor showed an inverse logarithmic relationship. Cross-sensitivity to helium was observed. The mean t90 response times were 90 (SD 10) ms for the O_2 sensor, and 100 (SD 10) ms for the CO_2 sensor. Breath-by-breath recordings showed slight damping of the CO_2 trace due to electronic filtering. Power consumption was 1.5–2 W per sensor.
Conclusions: The fast response times would allow accurate breath-by-breath measurement. Even though the O_2 sensor has a non-linear response, measurement is possible using multi-point calibration. Further design is necessary to allow trimix to be used as the diluent. A major disadvantage is the high power consumption needed to heat the sensors to high temperatures.

Introduction

GAS MONITORING IN CLOSED-CIRCUIT REBREATHERS

While in open-circuit scuba diving, exhaled gas is usually vented into the water, in a rebreather it is returned to a counterlung, carbon dioxide (CO_2) is removed in a 'scrubber' and metabolised oxygen (O_2) is added from the supply tank. Information about the various types of rebreathers available can be found elsewhere.[1,2] In a closed-circuit rebreather (CCR), the partial pressure of O_2 (PO_2) is usually kept at a constant level and only metabolised O_2 is replaced with fresh O_2 from the supply tank.[3,4] Oxygen is diluted in the breathing circuit with nitrogen or helium, or a combination of these gases. To maintain the PO_2 at a constant level, a control loop is needed. Therefore, electrochemical oxygen transducers, whose output signals are proportional to the PO_2, are used as sensing elements. In a manually controlled rebreather, the diver reads the PO_2 from a display, then, if necessary, adds O_2 manually and/or, as in one model, adjusts the O_2 injection needle valve. In an electronically controlled rebreather, this control task is usually performed by a micro-controller and a solenoid valve.

CCRs have many advantages, such as:

- high gas efficiency, close to 100%;
- stealth (silent, bubble-free diving);
- warm, humid breathing gas;
- extended diving time;
- reduced decompression obligations due to optimised gas mixtures and decompression mixtures.

Risks and accidents associated with rebreathers have been discussed in detail elsewhere.[5] The most commonly identified systems failures that cause fatalities are:

- PO_2 outside of life-sustaining limits;
- high CO_2 levels.

In current rebreathers, O_2 is measured using galvanic sensors. The core element of this transducer is an electrochemical cell (fuel cell) consisting of two electrodes of dissimilar metals (cathode – a noble metal behind a diffusion barrier, made usually of Teflon; anode – lead) in contact with a liquid or semi-solid basic electrolyte, usually potassium hydroxide. The transducer output is linear in the range up to 203 kPa (2 bar) PO_2 with a slope of 40–70 mV bar^{-1}. The most common and, for the diver, most dangerous failure mode of a PO_2 transducer is an incorrect electrical output for a given PO_2.

In order to achieve maximum safety in the case of a PO_2 transducer failure, two main strategies are used:

- redundancy, using several transducers together with a voting logic;
- transducer validation.[6,7]

As well as having several failure modes, current PO_2 transducers also face disadvantages such as:

- short lifetime (< 18 months);
- relatively slow and temperature-dependent response (t90 typically approximately 6 s at room temperature).

High CO_2 levels may be due to scrubber failure from various causes or check valve/direction valve failures that lead to 'pendulum breathing', in which the breathing gas no longer passes through the scrubber. Another important physiological rather than technical cause of hypercapnia is hypoventilation, especially at depth.[8] When CO_2 is chemically absorbed in the scrubber, heat and water are produced as by-products. Temperature at several points in the scrubber can thus be used for monitoring the scrubber and predicting its life-time.[9]

Although these technologies have been successfully implemented already, they provide feedback on the activity of the scrubber only, not on the inspired CO_2. CO_2 measurement is usually performed using optical sensors. CO_2 shows strong (infrared) light absorption at 4.26 μm and 15 μm wavelengths. Most commercial optical CO_2 transducers are based on Beer-Lambert light absorption at 4.26 μm. They have fast response times, a quasi-indefinite lifetime and are inexpensive, and until recently have not been used in rebreathers. In a rebreather, where the humidity in the loop is approximately 100%, condensation may lead to the failure of optical transducers, as any additional absorption in the optical measurement path leads to unreliable readings.

A proof of concept of a gaseous CO_2 monitor for rebreathers based on an optical CO_2 transducer (Gas Sensing Solutions, UK) was first presented by Amphilogic Ltd (UK) in 2008 at the Birmingham dive show. Recently, a CO_2 transducer for rebreather applications, based on the same transducer module has been launched.[10] In order to overcome the problem with high humidity, hydrophobic membranes and a sponge are mounted in front of the transducer. However, such measures are likely to lead to increased response times. Another approach is based on an optical CO_2 transducer at the end of the exhale hose. A preliminary study of the performance of this CO_2 transducer, particularly in relationship to its positioning in the rebreather circuit was reported recently in this journal.[11]

SOLID-STATE TRANSDUCERS FOR GAS MONITORING

An alternative to liquid electrolyte transducers is solid-state technology, based mainly on the ionic conductivity of ceramic materials.[12,13] This technology has been used for many years in cars for combustion control (Lambda probe). At present, only yttrium oxide-doped zirconium dioxide (Zirconia, YDZ) is used in commercial sensors as a conducting solid-state electrolyte. Conductivity in YDZ requires high temperatures. Therefore, the sensor is heated by an electrical resistance to reach an operational temperature of about 650^{O}C. The O_2 transducers typically used in cars are not applicable for a rebreather, mainly because of their power consumption and size; they also require a reference chamber. In automotive applications, ambient air is taken as a reference, which is, of course, impossible for an underwater breathing apparatus. Micro manufacturing allows miniaturisation of such transducers. An overview of micro solid-state gas sensors can be found elsewhere.[14] A suitable ionic conductor for a CO_2 sensor is sodium super-ionic conductor (NASICON).

Recently, micro solid-state sensors have been developed in Germany for O_2 measurement in low earth orbit and tested in the International Space Station (ISS). These are based on the amperometric principle, where the electrical current is proportional to PO_2 flow through the sensor membrane when the O_2 is 'pumped' from one electrode to the other by an applied voltage. The reaction proceeds in several steps. Firstly, molecular O_2 is transformed to oxygen ions at the cathode. Then these ions migrate through the solid electrolyte towards the anode, where they recombine again into O_2 molecules. In addition, a diffusion barrier limits the O_2 flux to the cathode. If the flux limitation is high enough, the PO_2 at the cathode is very low. In this particular case, the measured current is limited by the diffusion of O_2 to the cathode and, therefore, a linear dependence on the ambient PO_2 is achieved as long as 'crowding' of O_2 at the anode is avoided. Unlike the typical Lambda probe in a car, these types of sensors do not require a reference but instead require an efficient O_2 clean up from the anode.

Based on the test results from the ISS, a new project was started to develop micro solid-state O_2 and CO_2 transducers for respiratory analysis using this sensor technology. This is supported by the European Space Agency within the framework of the Microgravity Application Promotion Program (MAP Project No. AO-99-058; ESTEC Contract No. 14350/01/NL/SH). The focus for these investigations is to design and test a new miniaturised transducer system that enables simultaneous in-situ measurement of O_2, CO_2 and volume/mass gas flow rates. This allows for direct in-situ measurements inside the mask during cardio-respiratory studies of astronauts, athletes and medical patients. Since the in-situ measurements are performed with the transducer inside the mask, one measures these parameters in the main gas stream, avoiding any of the problems of side-stream measurement. The main characteristics of the transducers are listed in Table 1.

The aim of the present investigation was to validate the

Table 1
Specifications of the micro solid-state O_2 and CO_2 sensors

Oxygen
- Yttria-doped Zirconia (YDZ)
- Amperometric principle
- Operational temperature approx. 650°C (high temperature is required for linearity)
- 1.8 W power supply (1 W supply in principle possible, but more difficult to fabricate)
- Accuracy of < 2% volume at ambient pressure should be possible after 3 s heating
- Accuracy of +/- 0.1% at ambient pressure after 3–4 min
- Quasi-indefinite lifetime

Carbon dioxide
- NASICON-based
- Potentiometric transducer principle (with a solid reference), thus the electromotive force (EMF) is measured
- Operational temperature approximately 550°C
- 1.8 W power supply
- Operational lifetime approx. 700 h

performance of the micro solid-state sensors in conditions similar to those existing in a rebreather (PO_2 15–203 kPa, PCO_2 0–6 kPa). The response times of the transducers were of considerable interest as, due to their small size, they can be integrated into a mouthpiece, which theoretically allows breath-by-breath analysis of the inhaled and exhaled gases. Of particular interest was performance of the present transducer design when used in conjunction with the gas mixtures characteristic for the diving environment, i.e., O_2 in nitrogen (N_2) and helium (He)-N_2 admixtures.

Methods

A test bench, which included a small pressure chamber (approximately 10 cm³ volume) that can be pressurised up to 10 bar (Figure 1), was designed for measuring the performance of the solid-state sensors. Gas was supplied to the chamber via an adjustable needle valve from a 4-L cylinder. The supply pressure was reduced to 8 bar over ambient with a commercially available first stage regulator [DS4, Apeks]. A mechanical gauge and a calibrated absolute pressure transducer [MPX5700AP, Motorola] were used to measure the pressure inside the chamber. A pressure regulator [Porter USA] kept the pressure at a manually selectable level (0–4 bar above ambient). Alternatively, a quarter-turn shut-off valve could be mounted instead of the pressure regulator. In the closed position, the pressure inside the chamber will rise to the output pressure of the first stage regulator; opening the valve leads to a sudden loss of pressure, allowing measurement of the response time of the sensors to a step change in pressure. All components are either oil/grease free or are assembled using oxygen-compatible grease.

The O_2 sensor output was loaded with a 1 kΩ resistor. The voltage was then amplified with an operational amplifier (AD8630, Analog Devices). The CO_2 sensor signal was buffered with a precision operational amplifier (AD8601, Analog Devices) with a very low input bias current, typically 0.2 pA, making it the ideal choice for signal conditioning of a CO_2 sensor with a high output impedance. The conditioned sensor signals and the output signal of the pressure transducer were sampled with a 12-bit data acquisition card (USB-6008, National Instruments).

The solid-state sensors were heated by a printed platinum resistor (approximately 3 Ω at room temperature and 9 Ω at 700°C). For temperature control and transducer readout, custom electronics were developed, based on an 8-bit microprocessor (ATXmega16A4, Atmel). The temperature control programme essentially measures the heating current with the internal AD converter of the microprocessor, calculates the resistance and then sets the pulse-width modulation based on a proportional integral algorithm (PI control). Another two analog inputs of the microprocessor are used to sample the signal from the O_2 and the CO_2 sensors. These data, together with the heater currents and the calculated resistances, are transmitted via a serial interface to a PC at a sampling rate of 50 Hz. Data acquisition, conditioning and visualisation were performed using the software package Lab View™ 8.5 (National Instruments, Austin, Texas, USA).

EXPERIMENTAL PROCEDURES

Four separate tests were conducted. First, the O_2 sensor was installed in the test chamber and the gas flow was set to 0.5 L min^{-1} STPD. The chamber was gradually pressurised with O_2 in 0.1 bar steps from 1 to 4 bar absolute pressure. For O_2 values below 1 bar (101.3 kPa) the chamber was pressurised with air. To assess He cross interference, the O_2 sensor was exposed to trimix 21/50 from 1 bar to 5 bar absolute pressure.

Figure 1
Test bench for hyperbaric characterisation of micro solid-state O_2 and CO_2 sensors

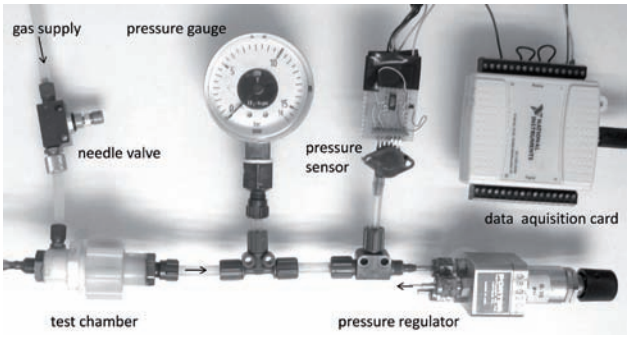
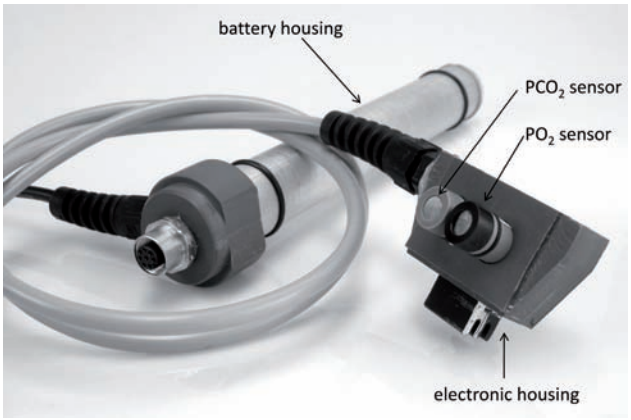


Figure 2
PO_2 and PCO_2 solid-state transducer module, electronics and rechargeable battery pack



The heating power was about 2.5–2.6 W.

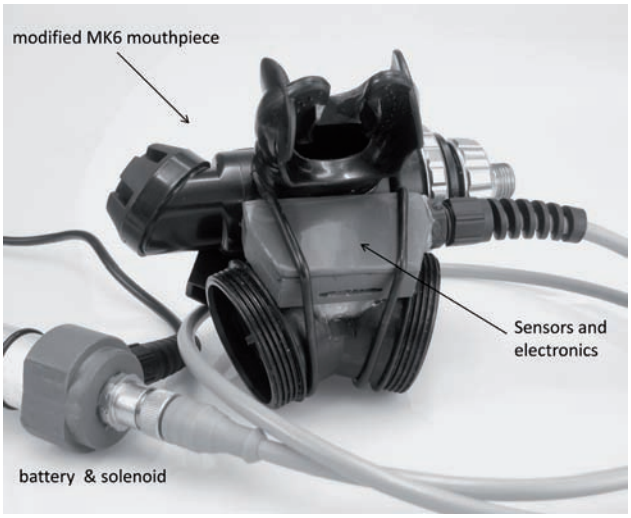
For response-time measurement, the pressure regulator was substituted with the quarter-turn shut-off valve and the chamber was pressurised with air at a gas flow of 0.1 L min^{-1} STPD. After reaching 7 bar, the shut-off valve was opened leading to a rapid decrease in pressure. Pressure signals and transducer signals were recorded and t90 was estimated using Microsoft Excel®.

Assessment of the signal response of the CO_2 sensor to CO_2 partial pressures (PCO_2) from 0–10 kPa was performed by gradually increasing the pressure inside the chamber using an air/CO_2 mixture with an FCO_2 of 1%. The CO_2 sensor is a potentiometric transducer, thus the output is logarithmic (Nernst equation). The response time of the sensor was measured in the same manner as for the O_2 sensor but using the 1% CO_2 gas mixture.

For the third test, a small module, measuring 22x10x5 mm and containing CO_2 and O_2 sensors and the required electronics, was built into a custom-made PVC housing filled with silicone gel. As well as transducer control and readout, it was also equipped with a solenoid controller, a display interface and a serial output for real-time data transmission to a PC (Figure 2).

A commercially available rebreather mouthpiece (Mk6, Poseidon, Sweden) was modified with a support that allowed fitting of the transducer module onto the mouthpiece such that the sensors sat between the mushroom valves (Figure 3). The module was connected via a water- and pressure-proof cable to the battery housing. It was also possible to install into the head of the battery housing a micro-solenoid for O_2 partial pressure (PO_2) control. As reference, a commercially available main-stream infrared CO_2 analyser (IRMA, Phasein, Sweden) was fitted between the bite and the rebreather mouthpiece.

Figure 3
Transducer module fitted onto a modified rebreather mouthpiece



In a final study to measure breath-by-breath PO_2 and PCO_2, a subject (35-year-old male) breathed through the rebreather mouthpiece. In all studies, repeat observations were made. The CO_2 transducer was two-point calibrated with air (assuming 350 ppm CO_2) and with air containing 1% CO_2.

Results

Figure 4 illustrates the results of the O_2 sensor characterisation with 100% O_2, air and Trimix 21/50. As the sensor elements studied were prototypes only mean values are shown at this

Figure 4
Illustrative characterisation of the O_2 sensor with O_2, air and trimix 21/50

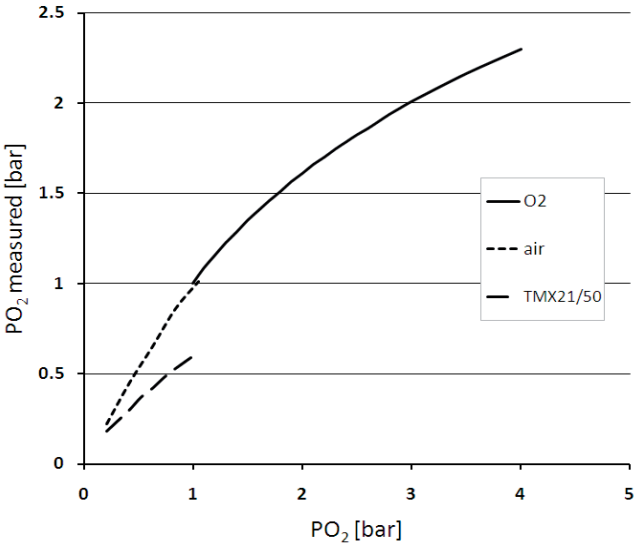


Figure 5
Characterisation of the CO_2 sensor from 0.01 to 0.1 bar PCO_2 with a 1% CO_2/air mixture

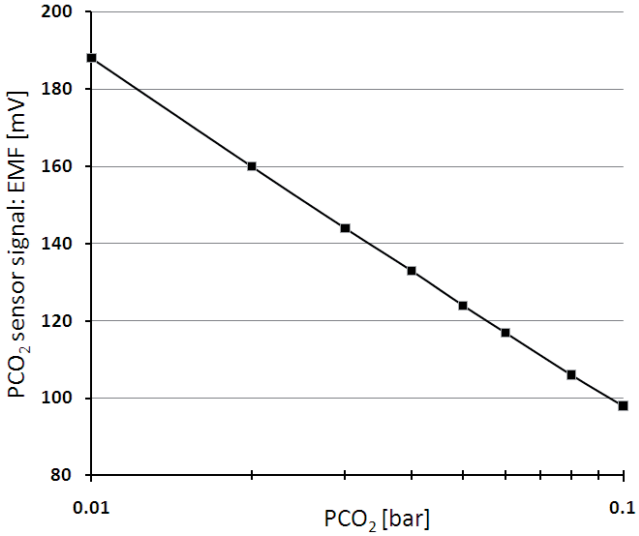
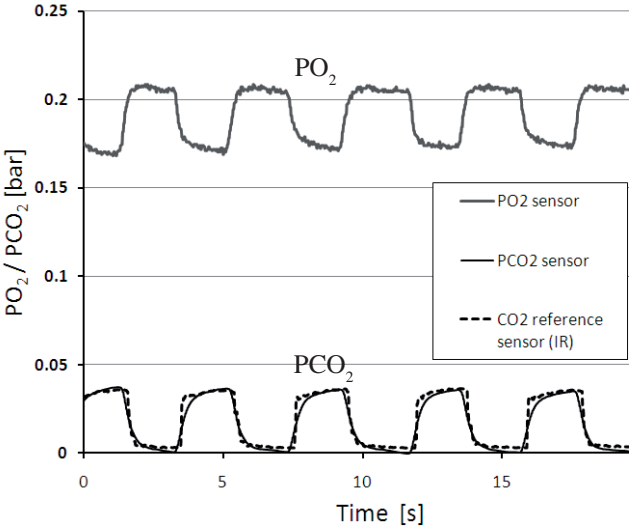


Figure 6
Sample breath-by-breath O_2 and CO_2 recording from the transducer module in the rebreather mouthpiece; measurements from mainstream CO_2 monitor also shown



stage. The sensor signal is non-linear, particularly at a PO_2 above 1 bar. The heating power was fairly constant with 1.65 W at 1 bar and 1.75 W at 4 bar chamber pressure. Using the same calibration factor as in the previous test, the sensor output using trimix was lower than expected. Instead of 0.21 bar O_2 at a chamber pressure of 1 bar, the observed PO_2 was 0.18 bar. At 5 bar chamber pressure, equal to a PO_2 of 1.05 bar, the observed PO_2 was only 0.65 bar. In comparison to the previous tests with 100% O_2, the required heating power was increased by approximately 40%.

Because the PCO_2 transducer is based on a potentiometric principle, the EMF produced is inversely related to PCO_2 in a logarithmic fashion, shown as a semi-log plot in Figure 5. The required heating power was 1.7 W and was fairly constant from 1 to 10 bar absolute pressure. The sensitivity was found to be -90 mV per decade (EMF @ 0.1 bar – EMF @ 0.01 bar PCO_2).

Using the quarter-turn valve, the pressure inside the chamber fell from 7 to 1 bar within approximately 25 ms. The mean t90 response time of the O_2 transducer was 90 (SD 10) ms, and that of the CO_2 transducer was 100 (SD 10) ms (n = 10).

Figure 6 shows an example recording of the breath-by-breath PO_2 and PCO_2 outputs and that of the reference CO_2 monitor. The average end-tidal CO_2 difference between the solid-state PCO_2 transducer and the infrared reference transducer over a 3 min recording period was found to be 0.26 (0.06) kPa.

Discussion

The recently developed prototypes of solid-state O_2 and CO_2 sensors seem to be suitable for applications in an O_2/N_2 rebreather. The main advantages are:

- very long lifetime;
- high accuracy;
- mechanically robust;
- insensitive to moisture/humidity (heated up to 600^{o}C);
- very fast response time, that allows assessment of PO_2 and PCO_2 values in inhaled and exhaled gas.

The main disadvantages are a high power consumption and non-linearity.

The current sensor prototype was developed for O_2 measurements below 1 bar PO_2. Above 1 bar PO_2, the sensor is non-linear, and so requires multi-point calibration to enable accurate measurements. In comparison to traditional wet-electrochemistry-based O_2 transducers containing a consumable lead anode, the micro solid-state technology is non-consuming and theoretically the signal response of the sensor does not change over time. Thus, a one-time factory calibration is conceivable. Even though this would obviate the need for pre-dive calibration, we believe that a pre-dive calibration with normobaric O_2 is a necessary safety measure to validate transducer function.

Further development will include optimisation of the diffusion layer in terms of thickness and density to improve linearity up to a PO_2 of 2 bar. An alternative approach to improving linearity might be three-electrode configuration together with a potentiostat, which is an electronic circuit used to maintain the voltage on one electrode at a defined level by using a third electrode, usually referred to as the reference electrode. Long-term aging effects caused by thermal stress (because of fast heating and cooling

of the transducer elements) were not investigated in this preliminary study.

The typical heating power required for a sensor is 1.7 W. Even with He in the diluent, no higher values than 2.5 W heating power were recorded. A standard 3.3 V, 2000 mA h^{-1}, rechargeable battery would allow operation of a single transducer for approximately 3 h with air as the diluent, which is sufficient for a rebreather for recreational purposes. Promising approaches to reduce the required heating power are based on further miniaturisation of the sensor element, the use of different ionic conductors that work at lower temperatures and smart system design with an emphasis on isolation and packaging of several transducers in one device.[15,16]

The PO_2 transducer in its current version does not appear suitable for use in a rebreather with He gas mixtures as the addition of He to the diluent decreased the sensitivity of the sensor. We believe this is due to an increased temperature gradient that is created by the presence of He. He has a much higher heat conductivity than air (or O_2 or N_2), which causes an increased heat loss at the sensor surface. To compensate for this, more power is necessary. The heating resistor, which is also used for sensing the sensor temperature, is located in the first layer above the sensor substrate. The increased heating power required results in a larger temperature gradient across the sensor. The diffusion layer, which is the current-limiting component, is distant from the resistor layer and, because of the temperature gradient, is most likely colder when He is used compared to air, which then leads to a reduced output signal in the presence of He. Possible solutions could be the integration of a temperature-sensing element in front of the sensor surface or measures for calibration of the transducers for different He fractions.

Figure 6 shows the results of the comparison of the PCO_2 transducer and the reference infrared-based CO_2 transducer. Even though the O_2 and CO_2 sensors have a short t90 response time and can be used for breath-to-breath gas analysis (Figure 6), the signal response of the solid-state PCO_2 transducer is slower than that of the reference monitor. This is because of an internal low-pass filter in the electronics. The filter frequency will be changed in the next hardware revision to allow full exploitation of the sensor's capabilities. It should be noted, that a CO_2 calibration using a precision gas with a higher CO_2 content might lead to more accurate values. Whether such a calibration can be carried out as a one-time factory calibration, or has to be performed at regular intervals by the user or a service centre, has yet to be assessed by long-term studies.

Conclusions

This paper presents preliminary data describing the characteristics of micro solid-state sensors for O_2 and CO_2 monitoring being developed for gas monitoring in diving rebreathers. These data confirm the potential successful application of this technology. Issues of non-linearity and helium cross-sensitivity require further investigation. Future testing will also address vulnerability of the heated transducers to heat shock and immersion in water, aging effects (especially from thermal stress) and how different breathing patterns influence transducer output on a breath-by-breath basis.

Acknowledgements

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Continuing professional development

Important notice: New Continuing Professional Development Coordinator needed

Associate Professor Michael Bennett is now the Chief Examiner for the ANZCA Certificate in Diving and Hyperbaric Medicine. He has been advised by the College that he will not be able to continue to produce the CPD exercises in future issues of *Diving and Hyperbaric Medicine* because of a perceived potential conflict of interest. Therefore, we are seeking a volunteer to continue the coordination of these.

ANZCA SIG members: time to front up and support your College, this journal and your colleagues!

Those interested should contact the SIG Chairperson, Dr Margaret Walker, E-mail: <margaret.walker@dhhs.tas.gov.au>, or Associate Professor Michael Bennett, E-mail: < M.Bennett@unsw.edu.au>.

This notice has now been in the last three issues of the Journal. Regrettably, nobody from SPUMS, and more particularly from the ANZCA SIG, working in hyperbaric medicine, has put themselves forward. As a result, there is no CPD item in this issue. Fortunately, we have several EUBS members who have volunteered their services for the next several issues. However, this does not resolve the problem of a coordinator for ANZCA SIG members. Without a College moderator, future CPD exercises regrettably will not be available as MOPS points on the ANZCA programme.

Michael Davis, Editor

Short communication

Preliminary observations on the effect of hypoxic and hyperbaric stress on pulmonary gas exchange in breath-hold divers

Erika Garbella, Andrea Piarulli, Edo Fornai, Alessandro Pingitore and Renato Prediletto

Key words
Breath-hold diving, physiology, pulmonary function, pulmonary oedema, carbon monoxide, nitric oxide

Abstract
(Garbella E, Piarulli A, Fornai E, Pingitore A, Prediletto R. Preliminary observations on the effect of hypoxic and hyperbaric stress on pulmonary gas exchange in breath-hold divers. *Diving and Hyperbaric Medicine*. 2011;41(2):97-100.)
Aim: To evaluate pulmonary alveolar-capillary membrane integrity and ventilation/perfusion mismatch after breath-hold diving.
Methods: Pulmonary diffusing capacity to carbon monoxide (DLCO) and nitric oxide (DLNO), haemoglobin (Hb) and haematocrit (Hct) were measured in six elite divers before and at 2, 10 and 25 minutes after a maximal breath-hold dive to a depth of 10 metres' sea water.
Results: Compared to pre-dive, DLCO showed a slight increase at 2 minutes in five subjects and a tendency to decrease at 25 minutes ($P < 0.001$) in all subjects. DLNO showed an increase at 10 minutes in three divers and a slight decrease at 25 minutes in five subjects. There was a small but significant ($P < 0.001$) increase in Hb and Hct at 2 minutes, possibly affecting the DLCO measurements.
Conclusions: An early but transient increase in DLCO in five divers may reflect the central shift in blood volume during a breath-hold dive. The late parallel decrease in DLCO and DLNO likely reflects alveolar-capillary distress (interstitial oedema). The DLNO increase in three subjects at 10 minutes may suggest ventilation/perfusion mismatch.

Introduction

Breath-hold diving leads to significant changes in the pulmonary system, such as lung-volume reduction by depth, increased blood flow and central pooling of blood, as well as local hypoxic vasoconstriction and heterogeneous blood redistribution.[1,2] Whereas heterogeneous redistribution of pulmonary capillary blood may lead to ventilation/perfusion mismatch, thus affecting pulmonary gas exchange, the acute increase in trans-capillary pressure may stretch the alveolar wall and weaken its integrity, possibly leading to interstitial-alveolar oedema or even haemorrhage.[3–5]

Pulmonary blood shift, early subclinical interstitial odema and alveolar haemorrhage can be functionally distinguished by different modifications of diffusing lung capacity to carbon monoxide (DLCO): respectively, early and transient increase, late and consistent decrease, early and persisent increase.[5,6] DLCO is affected by both alveolar surface characteristics (area, thickness and integrity) and pulmonary blood flow and volume.[7] It has been shown that the presence of ventilation/perfusion mismatch can be inferred in the single-breath diffusing capacity to DLCO when coupled with nitric oxide (DLNO) in the same breath (since DLNO measurements are independent of pulmonary capillary blood volume and flow), thus representing the true membrane diffusing capacity.[8] Therefore, we decided to measure, in a preliminary study, the DLCO and DLNO before and after a maximum-duration apneic dive to a depth of 10 meters' sea water (msw) in a small group of volunteer divers in order to elicit the effects of both hydrostatic pressure and hypoxia on early post-dive pulmonary function.

Methods

SUBJECTS

Six healthy, non-smoking, elite breath-hold divers (five male and one female), with no known medical problems and at least five years of practice and personal depths exceeding 30 msw, were studied. None reported any history of barotraumatic lung injury or decompression sickness, and physical examinations were normal. No drugs or alcohol were taken within the five days before the study, and subjects were not allowed to dive prior to the test dives. The study was conducted in accordance with the principles of the declaration of Helsinki and the protocol was approved by the Institutional Review Board of the Institute of Clinical Physiology (Pisa), National Research Council of Italy. The designed pre-and post-diving protocols were considered not to affect health risks related to diving activity. Written informed consent was obtained from all participants in the study.

EXPERIMENTAL PROCEDURES AND MEASUREMENTS

The study was performed in June at Asinara Island (Italy). A dive site with the bottom at 10 msw close to a small pier was chosen. Water temperature was about 24°C. The

test dive consisted of a maximum-duration, immersed breath-hold dive to 10 msw depth. This was preceded by two warm-up dynamic breath-hold dives also to 10 msw. Baseline measurements were preceeded by 10 minutes of acclimatisation on the water surface. All measurements pre- and post-dive were made with the subjects in the sitting position, wearing their diving suits.

The following measurements were taken: venous blood samples from an antecubital fossa vein for haemoglobin (Hb) and haematocrit (Hct); trans-jugular echo Doppler for cardiac output estimation (MY LAB 30, Esaote);[9] (both via removable Velcro patches in the suit at the elbow and neck); spirometric indices and lung volumes; DLCO, DLNO, alveolar volume (V_A) single breath measurement, the last by helium dilution technique, using a portable computerised spirometer (Sensor Medics). The spirometer was calibrated prior to the study for barometric pressure, ambient temperature, humidity, lung flow and volume. Duplicate measurements of DLCO, DLNO and V_A were repeated within 5 minutes. Following these baseline measurements, the divers prepared for diving, the time to get ready being 80 ± 20 seconds. All measurements, excluding spirometry, were repeated at 2, 10 and 25 minutes post-dive.

Spirometry and lung volumes were performed according to ERS-ATS guidelines and reference values were derived from Quanjer.[10,11] The single-breath diffusion tests were also performed according to ERS-ATS guidelines, and European reference equations for CO and NO lung transfer were used.[8,12–14] DLCO values were adjusted for the measured haemoglobin level and CO back-pressure in order to distinguish genuine changes in gas transfer over time from measurement variability.[12] Blood samples were stored in a portable fridge and analysed within 4 hours of sampling at a nearby hospital laboratory.

STATISTICAL ANALYSIS

Temporal trends of DLCO, DLNO, Hb and Hct were estimated performing a Friedman test with a 4-level within-subject factor (TIME: basal, 2, 10, 25 minutes).[15] Variations (d) of DLCO, Hb and Hct between pre- and post-dive values were estimated (dDLCO, dHb and dHct respectively) and correlations between dDLCO on the one hand and dHb or dHct on the other were performed using Spearman's rank correlation. Differences with a P-value < 0.05 were considered significant.

Results

Pre-dive, all subjects had normal respiratory function and lung-diffusion indices, and these and demographics of the subjects are shown in Table 1. The test dive lasted for a mean time of 270 (SD 53) s. Friedman test on DLCO showed a significant time effect ($P < 0.01$), whereas no significant effect was found for DLNO. Compared to pre-dive, DLCO showed

Table 1
Descriptive characteristics of subjects; functional values reported as single determinations as well as % predicted. FEV_1 – forced expiratory volume in one second; V_A – alveolar volume; DLCO – diffusing lung capacity for carbon monoxide; DLNO – diffusing lung capacity for nitric oxide

Parameter	Mean	SD
Age (years)	34	(8)
Body mass index (kg m^{-2})	22	(2)
Vital capacity (VC) (L)	6.26	(1.06)
FEV_1/VC (% pred)	98	(5)
Total lung capacity (L)	7.13	(1)
DLCO (mL min^{-1} $mmHg^{-1}$)	12.5	(2.25)
DLCO/V_A (% pred)	103	(21)
DLNO (mL min^{-1} $mmHg^{-1}$)	49.8	(9)
DLNO (% pred)	141	(25)
Haemoglobin (g L^{-1})	133	(16)
Haematocrit (%)	41	(4)
Cardiac output (L min^{-1})	3.8	(1.5)

Figure 1
Changes in DLCO over time following a maximal duration breath-hold dive to 10 msw (n = 6); a significant decrease was seen at 25 minutes post-dive ($P < 0.001$)

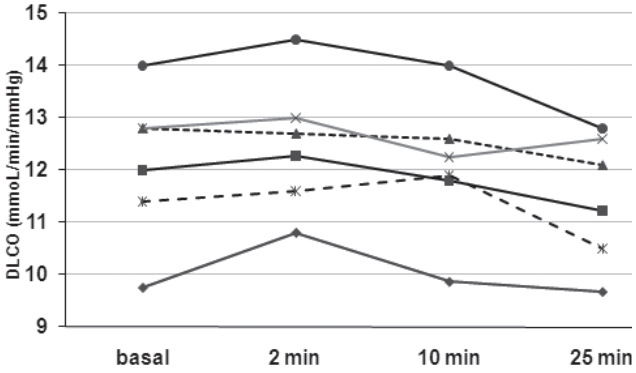
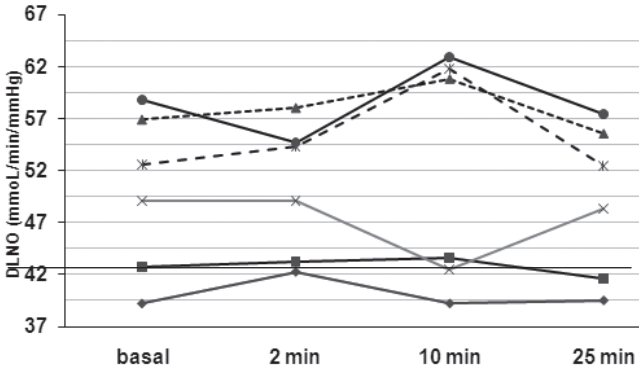


Figure 2
Changes in DLNO over time following a maximal duration breath-hold dive to 10 msw (n = 6); not significant



an increase at 2 minutes in five subjects and a tendency to decrease at 25 minutes ($P < 0.001$), with a decrease in all six subjects (Figure 1). In contrast, DLNO showed no clear trend at 2 minutes, an increase in three subjects at 10 minutes and a slight decrease at 25 minutes in five subjects (Figure 2). Hb and Hct showed a significant time effect ($P < 0.03$ and $P < 0.04$ respectively), both increasing at 2 minutes ($P < 0.001$; mean (SD) % change in Hct = 1.77 (0.8)). Cardiac output showed a slight but non-significant decrease after the dive. Although correlations between dDLCO and dHb or dHct did not reach statistical significance ($r = 0.78$, $P < 0.08$ and $r = 0.82$, $P < 0.07$, respectively), probably because of the small sample size, a trend was evident.

Discussion

These are the first observations of the mechanical and hypoxic effects of maximum-duration, immersed apnea at depth on alveolar-capillary membrane integrity, as assessed by the DLNO and DLCO tests. Our observations appear to be consistent with the initial hypothesis from a previous study, based on DLCO only, that breath-hold diving may elicit damage to the alveolar-capillary membrane.[5] An early, transient DLCO increase after a dive may reflect the central shift of blood into the pulmonary circulation, and this is further supported by the separation of the DLNO and DLCO trends at that point. On the other hand, both the slight DLNO and significant DLCO tendencies to decrease at 25 minutes likely reflect alveolar-capillary distress, such as interstitial pulmonary oedema.

In addition, the increase in DLNO at 10 minutes in three subjects may suggest ventilation/perfusion mismatching, secondary to pulmonary capillary blood flow redistribution. Finally in this study, DLCO measurement appeared likely to be affected by Hb and Hct but not by cardiac output. Furthermore, the increases in Hb and Hct early post-dive are consistent with the diving response (splenic contraction).[1]

Limitations of our study were primarily the small size of the data sample (six subjects observed at four time points) and large variability between subjects. Also, due to technical constraints (length of the challenge), only one dive was studied per subject, so that the reproducibility of observations within subjects could not be investigated. Yet, in order to support the observed effects by means of statistical evaluations and to verify the existence of time trends similar for each individual, regardless of the measured parameter weights, the use of non-parametric tests such as Friedman and Spearman's rank tests was considered a good compromise. These tests are essentially unaffected by inter-subject variability because they are calculated on the basis of variable ranks.[15] Our sample is too small to draw firm conclusions on the hypotheses we are dealing with, but the results seem to be promising and merit further detailed study to confirm these preliminary observations.

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New cardiopulmonary resuscitation guidelines

In November 2010, the International Liaison Committee on Resuscitation (ILCOR) released revised guidelines for cardiopulmonary resuscitation (CPR) based on the most current and comprehensive review of resuscitation literature ever published.[1] In general, the changes are relatively minor, most particularly placing greater emphasis on commencing chest compressions as soon as unconsciousness is established and the victim is not breathing "*normally*", rather than attempting initial rescue breathing. The importance of promptly sending for help is stressed so that, if available, an automated external defibrillator (AED, which are increasingly common and often available in public places) can be attached as soon as possible.

A revised easy-to-read reference and memory prompt for those trained in CPR, or a training text for CPR courses, which incorporates the new recommendations is available for the diving community through DAN Asia Pacific.[2]

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ECHM Workshop report

Controversial issues in hyperbaric oxygen therapy: a European Committee for Hyperbaric Medicine Workshop

Jacek Kot and Daniel Mathieu

Key words
Hyperbaric oxygen therapy, bone necrosis, hypoxia, brain injury, autism, medical conditions and problems, clinical audit

Abstract

(Kot J, Mathieu D. Controversial issues in hyperbaric oxygen therapy: a European College of Hyperbaric Medicine Workshop. *Diving and Hyperbaric Medicine*. 2011;41(2):101-4.)
Every few years, the European Committee for Hyperbaric Medicine (ECHM) publishes its recommendations concerning the clinical indications for hyperbaric oxygen therapy (HBOT). The last recommendations were issued during the 7th European Consensus Conference on Hyperbaric Medicine in 2004. Since then, several publications have reported on the use of HBOT in some indications in which it has not yet been recommended routinely, namely aseptic bone necrosis, global brain ischaemia and autism. Patients or their families push physicians and staff of hyperbaric facilities to use hyperbaric treatment regardless of the quality of the scientific evidence. Therefore, the ECHM Workshop "*Controversial issues in hyperbaric oxygen therapy*" was convened as a satellite meeting of the 2010 European Underwater and Baromedical Society Annual Scientific Meeting in Istanbul, Turkey in 2010. For each topic, a set procedure was used: first came a general report by specialists in the topic, incorporating a review of current pathophysiological, experimental and clinical evidence. Then, there were reports from hyperbaric facilities that had gained clinical experience in that condition, followed by a general discussion with specialists present in the audience. Finally, statements regarding each topic were proposed and voted on by the audience and these were presented to the ECHM Executive Board for consideration and possible approval. In conclusion, the use of HBOT in femoral head necrosis will be proposed during the next ECHM Consensus Conference to become an 'accepted' indication; whilst the use of HBOT in global brain ischaemia and autism should retain its current ECHM recommendations, that it should be 'optional' and 'non-accepted' respectively.

Introduction

The European Committee for Hyperbaric Medicine (ECHM) workshop on "*Controversial issues in HBO*" was held as a satellite meeting of the 2010 EUBS Annual Scientific Meeting in Istanbul, Turkey. It focused on three controversial conditions that are not yet accepted as indications for hyperbaric oxygen therapy (HBOT) on the ECHM list:[1]

- aseptic bone necrosis;
- global brain ischemia;
- autism.

For each topic, there are several publications available on the use of HBOT and there are repeated requests from patients or their families to physicians and staff of hyperbaric facilities to use HBOT either in standard or experimental mode to treat these conditions. Therefore, in order to recognise the potential inclusion of these indications in the ECHM recommendations for use of HBOT,[1] general introductory reports were presented by specialists in each field with a review of current pathophysiological, experimental and clinical evidence. These presentations were followed by short communications from representatives of HBOT facilities having clinical experience in that condition. These were followed by general discussion to which specialists present in the audience contributed. Based on all presentations, reports on personal experiences and general discussion, the final statements regarding each topic were proposed and voted on by the audience. These statements were then presented to the ECHM Executive Board for approval. Therefore, the final conclusions from this workshop reflect the current position of the ECHM with regard to HBOT in these three conditions.

Aseptic bone necrosis

L Ditri (Italy), J Desola (Spain), J Von Reumont (Germany)

- Magnetic resonance imaging (MRI) is the best technique at present for diagnosing aseptic bone necrosis (ABN), particularly in the early stages of its pathology when plain radiography is negative for bone damage or collapse.
- Grading of ABN is useful for determining whether HBOT is indicated and for monitoring its effectiveness. The Steinberg classification system, which is based on MRI imaging, should be used in preference to other grading systems.[2]
- The clinical goal sought by surgical and clinical specialties is delay or avoidance of the need for hip arthroplasty.
- Use of HBOT in ABN is based on its physiological

effects: increasing oxygen availability to cells, enhancing osteoblast and osteoclast function, reducing oedema (by oxygen-osmotic pump and vasoconstriction mechanisms) and thereby reducing the intraosseous pressure. This brings about an improvement in venous drainage and microcirculation, mobilisation of stem/progenitor cells from the bone marrow by a nitric oxide-dependent mechanism and stimulation of neovascularisation for healing.
- HBOT appears to achieve significant beneficial results only in stage 1 or 2 of the Steinberg classification, before the collapse of the articular surface of the bone, so early diagnosis of ABN is mandatory.

Based on a literature review[3–7] and on the personal experience of experts, if HBOT is considered for ABN, then the number of HBOT sessions should be about 60–80 in no more than four consecutive months, at a pressure of 243–254 kPa (2.4-2.5 bar) for a time of 90 minutes' oxygen (O_2) breathing. This should be combined with the use of crutches to ensure complete non-weight bearing during the entire treatment course. In particular, the recent publication of a double-blind, controlled randomised trial (RCT) raises the level of clinical evidence for HBOT in ABN to level B ("*double-blind controlled, randomised studies, but with methodological flaws; studies with only small samples, or only a single study*").[7,8] Based on this literature review, Ditri proposed that ABN be included in the ECHM list of recommended indications for HBOT.

Three short reports were then presented on their personal clinical experience by Desola, Ditri and Welslau/von Reumont. Ditri (unpublished observations) reported that in 329 patients with mild to moderate ABN of the femoral head, femoral condyle and other sites (stage 1 or 2 in the MRI Steinberg classification) treated with long-term HBOT (40–100 HBOT sessions at 253 kPa (2.5 Ata), in conjunction with conventional non-surgical therapy (physical therapy, crutches, osteointegration, pain control) full recovery or significant improvement was observed in almost 70% of patients, while deterioration or lack of any change was observed in 30% of cases. Based on this observational and uncontrolled study, the protocol for a prospective multicentre research project has been prepared and is now ready for implementation.

Welslau and von Reumont (unpublished data) reported on an ongoing, prospective, uncontrolled study on HBOT in ABN involving the knee or talus. 333 patients with MRI-verified diagnosis (with ARCO classification) have received HBOT (from 6 to 62 HBOT sessions at 253 kPa) in conjunction with standard conservative therapy. In this group, out of 269 patients with ABN of the knee (ARCO grades varying from 1 to 4), 91.5% were symptom free or had only mild discomfort following HBOT, whilst 8.5% continued to deteriorate or had no change in clinical status.

The final outcome appeared to vary from centre to centre and country to country. The process of bone healing is slow, so one of the possible explanations for different outcomes is the number of HBOT sessions given. In all cases, MRI should be used before and after HBOT treatment to monitor the result of therapy.

After the general discussion, the audience was split fairly evenly between those who supported the inclusion of ABN on the ECHM list and those wishing to wait until more clinical data had been published.

Global brain ischaemia

B Ratzenhofer (Austria), D Mathieu (France), J Desola (Spain)

Cerebral metabolic rate is about 4 ml O_2 $100g^{-1}$ min^{-1}, with approximately half spent on maintenance of synaptic activity and half on maintenance of basal cellular function, such as protein/neurotransmitter synthesis, trans-membrane ionic gradients, anabolic enzyme reactions and Na^+/K^+ pump activity.

Over the past 30 years, it has been established that:
- there is a linear relationship between local cerebral blood flow (CBF) and brain tissue pH over a PO_2 range from 20–400 mmHg;
- reduction of PO_2 causes a decrease of local pH leading to vasodilatation and increased CBF;
- after an acute cerebral hypoxic episode, areas of heterogeneous perfusion remain, leading to chronic and varying degrees of O_2-deprivation (the so-called 'penumbra' in stroke, for example).[9]

Several thresholds for cerebral ischaemia are evident:
- at 50 ml $100g^{-1}$ min^{-1} neuronal function is normal;
- at 50–30 ml $100g^{-1}$ min^{-1} inhibition of protein synthesis, selective gene expression, neuronal loss and lactic acid generation start;
- at < 30 ml 100 g^{-1} min^{-1} there is glutamate release, acidosis and ATP decline;
- at < 20 ml 100 g^{-1} min^{-1} infarction commences;
- at < 10 ml 100 g^{-1} min^{-1} K^+/Ca^{++} homeostasis is disrupted.

The heterogeneity of the brain in terms of circulation and metabolism is a challenge in the evaluation of the effects of HBOT. The ischaemic penumbra, the region where CBF is insufficient to maintain normal function, but just enough to maintain structural integrity, is the therapeutic target for HBOT.

The potential mechanisms of neuroprotection by HBOT include:
- increased oxygen delivery to neurons;
- stimulation of apoptotic inhibitors and free radical scavengers;

- inhibition of leukocyte adhesion through inhibition of ICAM-1;
- decreased breakdown of the blood-brain barrier;
- decreased oedema through oxygen-mediated vasoconstriction;
- stimulation of angiogenesis.[10]

Since the previous ECHM Consensus Conference in 2004, two new publications concerning global cerebral ischaemia in humans have appeared and were reviewed.[11,12]

Clinical experience was then presented by Desola and Mathieu. Desola (unpublished observations) reported that 34 previously healthy patients in coma (with one exception of a conscious patient with cortical blindness) after acute anoxic brain injury were treated with HBOT (60 min 100% O_2 at 233–283 kPa, once daily up to a median of 40 sessions, range 20–60), with a latency of 12 days to two months. In all cases, HBOT was started after patients failed to respond to standard care. Regardless of severity, improvement was observed in all patients, 28 with complete and six with partial recovery. Delay to HBOT treatment also did not appear to influence the final outcome.

Mathieu (unpublished observations) reported that, in a group of 305 patients after near-hanging, the following prognostic factors were found to be statistically associated with poor outcome: no lid reflex; cardiac arrest on site; coma > III or GCS <= 5, no light reflex and no HBOT.

During general discussion, the point was raised that combined hypothermia and HBOT therapy should be investigated for clinical efficacy. It was concluded that treatment with HBOT may possibly reduce mortality and neurological sequelae in term neonates with hypoxic-ischaemic encephalopathy. Despite widely differing study designs and clinical reports, the data suggest a beneficial effect. However, the optimal HBOT dosage and onset of treatment are yet to be defined. Because of the poor quality of reporting, a high-quality RCT is needed to investigate these findings.

After the general discussion, a majority of the audience voted for leaving the current recommendation for use of HBOT as it is, which is 'optional', in post-anoxic encephalopathy.[1]

Autism

N Motavalli (Turkey), J Schmutz (Switzerland)

- Studies on the pathogenesis of autism are far from claiming a single intervention method as a 'cure' for the disorder.
- Treatment approaches should focus on enhancing socio-emotional and communicative abilities as well as self-help skills in this group.
- The professionals should inform parents about the lifelong nature of the disorder and importance of appropriate early education programmes.

Schmutz presented a literature review of current evidence concerning HBOT use in autism.[13–15] It was concluded that some studies showed efficacy of HBOT in autism, but there were serious design limitations and a possibility that there was a strong influence not by HBOT itself but rather by associated learning and care procedures.

During general discussion, the majority of workshop participants confirmed that they had received requests from families for HBOT in the treatment of autistic children. With isolated exceptions, these requests were generally declined. However, the growing practice of 'mild hyperbaric therapy' (conducted in inflatable chambers with low pressures with air, installed in family homes) was recognised. This has been addressed in position statements by the Undersea and Hyperbaric Medical Society and the Australia and New Zealand Hyperbaric Medicine Group.[16,17]

The workshop confirmed that autism is not accepted as an indication for HBOT, as there is insufficient evidence currently. HBOT should only be used in autism within the framework of an ethically approved clinical trial. Use on compassionate grounds in individual cases should only occur if there is believed to be an acceptable scientific rationale, if the patient/parents, referral physician(s) and third party payer (if any) have been extensively informed about the potential risks and questionable benefit, and if treatment is at no cost to the patient.

Conclusions

- There is a good rationale for HBOT in aseptic bone necrosis, based on pathophysiological mechanisms. The personal experience of hyperbaric medicine specialists and cumulative clinical data confirm that HBOT can improve clinical outcome in Steinberg stage 1 or 2 cases. Therefore, approval for the use of HBOT in femoral head necrosis should be proposed during the next ECHM Consensus Conference as an 'accepted indication'.
- There is a good rationale for using HBOT in global brain ischaemia based on pathophysiological mechanisms. However, since the last ECHM Consensus Conference there have been only limited clinical data published. Therefore, there is no reason to change the current status of the ECHM recommendation, which is 'optional'.[1]
- Currently there is no clear rationale for HBOT in autism based on clinically proven pathophysiological mechanisms. The few studies on the use of pressurised environments (not always compatible with the definition of 'hyperbaric oxygen therapy') for autism have serious methodological limitations. Therefore, autism should remain as a non-accepted indication for HBOT. HBOT should be used only within the framework of an ethically approved clinical trial. Treatment of individual patients may occasionally be acceptable on compassionate grounds only with the agreement of all parties after extensive informed discussion and at no cost to the patient.

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Critical appraisal

Hyperbaric oxygen therapy improved both pain scores and range of motion in patients with early idiopathic femoral head necrosis (Ficat stage II)

Bottom line
1. Pain and active range of motion were improved at 20 treatments, and more so after 30 treatments of HBOT.
2. An observational follow up for seven years suggested this effect may persist for at least that long, and may avoid hip replacement or other surgical intervention.
3. Only the findings at completion of the HBOT course are comparative differences between groups.

Citation
Camporesi E, Vezzano G, Bosco G, Mangar D, Bernasek T. Hyperbaric oxygen therapy in femoral head necrosis. *J Arthroplasty*. 2010;25:118-23.

Three-part clinical question
For adults with idiopathic femoral hip necrosis, does the application of hyperbaric oxygen therapy result in the improvement or stabilisation of symptoms such that hip replacement can be delayed or avoided?

Search terms
Hyperbaric oxygen therapy, femoral head necrosis

The study
Double-blinded, randomised, controlled trial with intention to treat.

The study patients
Adult patients with idiopathic femoral head necrosis (Ficat stage II) diagnosed by X-ray and confirmed with MRI. Patients with a history of alcohol abuse, trauma to the hip or steroid administration were excluded.
CONTROL GROUP
(n = 10; 9 analysed): Hyperbaric exposure breathing 21% oxygen (air) at 243 kPa, for 60 minutes once daily, Monday to Friday, for six weeks (30 treatments total).
EXPERIMENTAL GROUP
(n = 10; 10 analysed): As above, but breathing 100% oxygen at 243 kPa.

The evidence
See Table 1.

Comments
1 Patients who received sham therapy were offered HBOT immediately after completion of the initial 30 treatments. From that time, therefore, this was an observational study.
2 Outcomes were measured after 10 and 20 treatments as well as on completion. The magnitude of effect increased at each time point, suggesting a dose-effect relationship.
3 These results were arrived at using a correction for multiple comparisons.
4 Similar findings in range of motion for flexion, abduction and adduction, but flexion did not reach statistical significance.
5 The authors reported no adverse effects in either group.
6 All 17 patients followed to seven years reported minimal pain and no decrease in activities of daily living; none had received arthroplasty. Nine had repeat MRI and all these showed radiographic improvement.

Appraised by: M Bennett, Monday, 02 May 2011
E-mail: *<m.bennett@unsw.edu.au>*

Key words
Hyperbaric oxygen, bone necrosis, outcome, critical appraisal

Source
<www.hboevidence.com>

Table 1
Major outcomes for idiopathic femoral head necrosis following 30 60-min air or oxygen treatments at 243 kPa

Non-event outcomes	Time to outcome/s	Control group	Experimental group	*P*-value
Pain score (VAS 0 to 10; median scores estimated from graph)	30 treatments (6 weeks)	5.3	1.3	0.002
Active range of movement (median degrees and range)	30 treatments (6 weeks)	7.0 (0–10.0)	35.5 (26.0–45.0)	< 0.001
Stabilometry (% difference in load distribution between affected and unaffected limb)	30 treatments (6 weeks)	18 (10–42)	12 (6–22)	0.64

From the recent literature

Comparison of two modes of delivery of first aid training including basic life support

John Lippmann, Patricia Livingston and Melinda J Craike

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Abstract

Aims: Flexible-learning first aid courses are increasingly common due to reduced classroom contact time. This study compared retention of first aid knowledge and basic life support (BLS) skills three months after a two-day, classroom-based first aid course (STD) to one utilising on-line theory learning at home followed by one day of classroom training (FLEX).

Methods: In this prospective, randomised controlled trial, 256 participants with internet access and no first aid-related training for at least five years were randomly allocated to a STD or FLEX course. Assessment was conducted immediately after training and three months later. Each participant was allocated a theory and a BLS score, which were averaged and summed to create an equally-weighted 'combined score' of first aid knowledge and skills.

Results: There was no significant difference in theory scores between the STD and FLEX groups immediately after training and after three months. STD participants had significantly higher BLS scores immediately after training ($P = 0.001$) and three months later ($P = 0.046$). Males had significantly higher BLS scores after training ($P < 0.001$), but not three months later ($P = 0.2$). Participants > 46 years had significantly lower BLS scores than younger participants ($P < 0.001$). There was no significant difference in combined scores between the STD and FLEX groups or between genders, education or age groups either immediately after training or three months thereafter.

Conclusion: After replacing one day of classroom-based training with on-line theory training, there was no significant difference in the first aid competencies of the study population, as measured by an equally-weighted combined score of basic life support and first aid theory.

Published with kind permission from: Lippmann J, Livingston P, Craike MJ. Comparison of two modes of delivery of first aid training including basic life support. *Health Education Journal*. 2010. Published online before print November 24, 2010, doi: 10.1177/0017896910386208.

Key words

First aid, resuscitation, training, World Wide Web

The database of randomised controlled trials in hyperbaric medicine maintained by Dr Michael Bennett and colleagues at the Prince of Wales Hospital Diving and Hyperbaric Medicine Unit is at:

<www.hboevidence.com>

Letter to the Editor

The death of buddy diving?

David Cooper raises concerns in his letter regarding the potential implications for recreational diving of the Watson case.[1] Before we draw any conclusions, however, one must be aware that this is a complex case, the plea and verdict were subject to negotiation and the outcome possibly perverse. Gabe Watson was not tried before a jury for his wife's death, because he pleaded guilty to manslaughter, where death is an unintended consequence. Thus the evidence was never tested in court. He was convicted under a rare Queensland criminal code statute, detailed by Dr Cooper, and enacted in 1899 prior to Australian federation and not used in a criminal trial until this case.[2] This statute is not in the criminal code in any other Australian jurisdiction.

Watson had some incriminating circumstantial evidence against him for murder, but the Queensland Director of Public Prosecutions felt that a guilty verdict, (murder beyond reasonable doubt), was very unlikely so the charge was reduced to manslaughter.[3] Then Watson returned from the USA voluntarily for the trial on this lesser charge. Watson's father stated pragmatically: "*faced with the prospect of a long trial against the state with unlimited resources, an unknown outcome in a place far from our home, we elected the 'guilty plea' option so as to have our son returned to us at the earliest possible date*." One of the judges at the sentence appeal was surprised that Watson had pleaded guilty.[3]

Watson's diving behaviour should have been better, but Dr Michael Bennett stated in expert evidence that his three-year lack of diving made him "*relatively inexperienced*".[3] Watson now faces a possible murder trial in Alabama, as he was deported following sentencing. This case remains controversial, and few conclusions can be drawn from it. "*Rare cases make bad law*".[4]

References

1 Cooper PD. The death of buddy diving. *Diving and Hyperbaric Medicine*. 2011;41(1):38.

2 *Criminal Code Act 1899* (Qld) s 290. Available from: <www.legislation.qld.gov.au/LEGISLTN/CURRENT/.../CriminCode.pdf>. Chapter 27. p. 203.

3 *Death on reef mystery*. Available from: <http.://www.theage.com.au/national/death-on-reef-mystery-20100716-10eb1.html>.

4 *Do cases make bad law?* Available from: <lawreview.uchicago.edu/issues/archive/v73/summer/schauer>.

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Key words
Diving deaths, legal and insurance, Letters (to the Editor)

The poetry doctor

Amazing air

When on scuba I can't respire without an apparatus.
To regulate air from a tank to ambience; to inflate us.

Refrain: Amazing air inflates my lungs and lets me breathe – inspiring.
Without this air I would soon die, so aptly termed expiring.

It works so well I can descend to the deep and back.
Unless the air supply does end, a catastrophic lack.

I gasp and gulp and gag in fright, however hard I suck.
My buddy is nowhere in sight. I curse my rotten luck.

Panic starts to take its grip. I blame my poor dive plan.
As consciousness begins to slip, its reasons I do scan.

My regulator's sprung a leak? I've dived too long, too deep?
My air content I did not seek, my vigilance asleep?

My training fires despite my doubts. I drop my belt then fin.
As I rise I breathe out, avoiding CAGE within.

I break the waves and what a treat to breathe in nature's air.
Nothing ever could taste so sweet, a life-sustaining fare.

I'm so relieved I've thwarted death I say a thankful prayer.
Appreciating my every breath, never again to run out of air.

John Parker

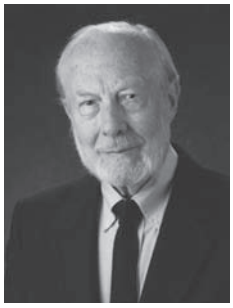
Christine Cridge and DDRC

The Diving Diseases Research Centre (DDRC), Plymouth, has recently appointed Dr Cridge as its new Director, incorporating the roles of Medical Director and Chief Executive.

A general practitioner with the Postgraduate Diploma of Medical Science in diving and hyperbaric medicine from The University of Auckland, Dr Cridge has worked in both the HMS Haslar, Gosport, and DDRC hyperbaric units in the past. She returns from a recent appointment as Medical Director with Hyperbaric Health in Perth, Western Australia.

Obituary

Professor Christian J Lambertsen



May 1917 – February 2011

Dr Christian Lambertsen, MD, DSc (Hon), died at his home in Newtown Square, Pennsylvania on 11 February 2011.

Chris received his Bachelor of Science from Rutgers University in 1938 and his MD from the University of Pennsylvania in 1943. During his medical school period, he invented the initial self-contained closed-circuit oxygen rebreathing apparatus used by the US military for neutral buoyancy underwater swimming and diving, the Lambertsen Amphibious Respiratory Unit (LARU). As a student, he aided the early Office of Strategic Services (OSS) in establishing the first cadre of US military operational combat swimmers. Dr Lambertsen became a US Army medical officer on graduation from medical school in early 1943, and immediately joined the OSS Maritime Unit on active duty through its period of function in World War II. He joined the University of Pennsylvania Medical Faculty in 1946, and became Professor of Pharmacology in 1952. Whilst a faculty member, he combined diving research with further development of underwater rebreathing equipment for the Army and Navy. In 1967 he served as Founding President of the Undersea Medical Society (now Undersea and Hyperbaric Medical Society). Dr. Lambertsen is recognised by the Naval Special Warfare community as "*The father of US combat swimming*." His hand has touched every aspect of military and commercial diving. Dr Lambertsen's active contributions to diving began during WWII and became even more progressive in the post-war period through the evolution of the US Navy Deep Submergence and Naval Special Warfare developmental programme.

Please send condolences to: Lambertsen Family, c/o Chris Lambertsen, 3500 West Chester Pike, Suite 129, Newtown Square, PA 19073-4101.

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Alf Brubakk:
I met Chris Lambertsen a number of times and even worked with him on problems related to decompression. Even as a student, he was involved in developing diving procedures and he was instrumental in developing the rebreather that was used by the Navy Seals. I once saw a letter from the Secretary of War, telling the Dean of the University not to give Chris a problem if he spent too much time away from his studies, he was essential to the war effort! I can hardly believe he is dead. Once, about 10 years ago, when I commented on the problem of finding successors in our field, he said to me that he did not see the problem, as he was still going strong! He also allowed me to go through his personal archive; a lot of his unpublished work on isobaric counter-diffusion is still very relevant. For my generation, Lambertsen was like a beacon. He has influenced a whole generation of scientists; we have an obligation to continue the good work.

David Elliott:
The end of an historic era and a career that defies any summary … what a multifaceted contribution Chris leaves. Dick Vann gave a presentation in London some years back on CJL, which included an account of when he came to the UK in the dark days of World War II to make an operational assessment of the secret British 'underwater canoe', and how he then returned to the USA having had to memorise the classified instruction manual.[2] Creating his Institute at the University of Pennsylvania and the Underwater Physiology Symposia series were achievements later crowned by his foundation of the Undersea Medical Society. Frank Butler has reviewed some of Chris's early work.[3]

Noemi Bitterman:
It is hard to believe that Chris Lambertsen will not be with us. We know that time will come for everyone, but somehow it seemed Chris was above the rules, and he would stay alive for ever. Indeed, only a few weeks ago, I sent him the traditional New Year card for 2011. I have plenty of great memories, letters with his unreadable handwriting and pictures, but it will take time to go through it all. The symposium that Steve Thom and I co-chaired, "*Oxygen 2002 – Honoring the life time work of Christian J Lambertsen",* in La Jolla was a great two-day event presenting the multidisciplinary aspects of oxygen and saluting "*the father of oxygen*".[4] We will never forget him.

In 2009 Dr Lambertsen transferred his extensive literature and research collection to the UHMS Charles W Shilling Library. The UHMS and the Rubicon Foundation (<http://archive.rubicon-foundation.org>) have been organising this before it is processed into the Shilling Library by the Duke University Medical Center Library and Archives staff.

References

1 *Christian J Lambertsen*. From Wikipedia, the free encyclopedia. Available at: <http://en.wikipedia.org/wiki/Christian_J._Lambertsen>.
2 Vann, RD. Lambertsen and O_2: beginnings of operational physiology. *Undersea Hyperb Med*. 2004;31(1):21-31.
3 Butler FK. Closed-circuit oxygen diving in the US Navy. *Undersea Hyperb Med*. 2004;31(1):3-20.
4 Oxygen 2002. The Lambertsen symposium. *Undersea Hyperb Med*. 2004;31(1):1-183.

Key words
Obituary, general interest, rebreathers/semi-closed circuit

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates (updated October 2008)

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

1 The candidate must be medically qualified, and be a current financial member of the Society.
2 The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved facility. The list of approved facilities providing two-week courses may be found on the SPUMS website.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval *before* commencing their research project.
5 The candidate must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.
6 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, published in *Diving and Hyperbaric Medicine* 2010; 40(2):110-2.
7 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.
8 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 <http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm>) or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documented evidence of approval by an appropriate research ethics committee. It is expected that the research project and the written report will be primarily the work of the candidate, 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 2008, 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 the Education Officer:
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

SPUMS website news

There was a slight surprise for everyone in the New Year in the form of a renewal e-mail for SPUMS. The reminder was sent out automatically by a section of the software that had been activated without our knowledge by a security update. The message was a little 'generic' and I apologise to anyone who was confused or offended by the message, as it caught us by surprise as well! We have subsequently found the generic message and hopefully next year a nicer version will be sent out to remind members to renew.

We have been trying to reduce the workload for both Steve Goble (the Administrator) and myself in getting the membership database under control. I am progressively implementing automated tasks and the next one will be checking the Diving Doctors database against the membership database. Please note that to be on the diving doctors database you have to be a member of SPUMS. There will be two months grace to renew membership prior to doing the auto-check, but after February each year, if you are not a member then you will be taken off the Diving Doctors List. Please check your membership details are correct and current, as the professional address is what is available to people searching the database.

New features to be implimented over the next year are:
a diving and hyperbaric jobs section in the forums;

- better access to position papers and SPUMS paperwork;
- encouraging people to post in the forums, including resident experts for people who have questions in diving and hyperbaric medicine;
- promotional material for SPUMS including a PowerPoint presentation for use at local meetings for all members and non-members to use and view.

This is on top of the features that we already have, such as full copies of *Diving and Hyperbaric Medicine* and information on the SPUMS ASM and other meetings around the region and world.

So the website is slowly crystallising towards a user-friendly, accessible part of your SPUMS membership, as my time permits. Suggestions are welcome, so e-mail me at <webmaster@spums.org.au>.

Glen Hawkins, SPUMS Webmaster

Key words
World Wide Web, medical society

ANZCA Certificate in Diving and Hyperbaric Medicine

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 Diploma in Diving and Hyperbaric Medicine.
3 **EITHER:**
   a Completion of the Diploma of the South Pacific Underwater Medicine Society, including six months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS
   b **and** Completion of a further 12 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 18 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA
   d **and** 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:
<www.anzca.edu.au/edutraining/DHM/index.htm>

Dr Margaret Walker, FANZCA
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine

EUBS European Underwater and Baromedical Society

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EUBS European Underwater and Baromedical Society

EUBS 37th ANNUAL SCIENTIFIC MEETING 2011

24–27 August 2011
Gdansk, Poland

Hosts: The National Centre for Hyperbaric Medicine, Gdynia
Venue: The Medical University of Gdansk

Honorary Patron:
Lech Wałęsa,
President of the Republic of Poland (1990-1995) and Nobel Peace Prize Laureate 1983

Zdzislaw Sicko, Chairman of the Organising Committee, **E-mail:** <zsicko@ucmmit.gdynia.pl>
Jacek Kot, General Secretary of the Organising Committee, **E-mail:** <jkot@gumed.edu.pl>

Scientific Committee: Alessandro Marroni (IT) (Chairman), Mike Bennett (AU), Alf Brubakk (NO), Maide Cimsit (TR), Jordi Desola (ES), Peter Germonpré (BE), Yoram Grossman (IL), Michal Hajek (CZ), Erik Jansen (DK), Jacek Kot (PL), Folke Lind (SE), Daniel Mathieu (FR), Peter Radermacher (DE), Maria Wujtewicz (PL)

Main topics:

Diving physiology and medicine, non-dysbaric disorders
Research in deep diving and dysbaric diving disorders
Basic research and clinical hyperbaric medicine
Hyperbaric safety, technology and organisation

Satellite meetings:

24 August: ECHM Workshop on "*HBO in Emergency Medicine*"
24 August: Workshop on "*Validation of decompression computers*"
(these are parallel meetings)
28 August: Divers Alert Network "*Divers' day*"
Working group meetings during conference:

- EUBS Executive Committee
- ECHM Executive Board
- ECHM Board of Representatives
- EDTC Medical Committee
- IDAN Board of Directors
- DAN Board of Directors
- PHYPODE Consortium Board

Conference office: (registration, accommodation, travel, payment)
Phone: +48 46 856 30 13
Fax: +48 46 856 30 13
Mobile: +48 604 203 244
E-mail: <office@eubs2011.org>

Visit our website:

<www.EUBS2011.org>
and register today

International Congress on Hyperbaric Medicine

The 17th International Congress on Hyperbaric Medicine was held in Cape Town, South Africa, 16–19 March 2011. In these notices we print the minutes of the ICHM Governors' Meeting held during the conference.

For all enquiries regarding the ICHM, please contact the Secretary, A/Prof Michael Bennett: <m.bennett@unsw.edu.au>

Minutes of Governors' Meeting, International Congress on Hyperbaric Medicine, held in Cape Town, SA, 18 March 2011

Meeting opened: 1304 h

Present: Mike Bennett, Nina Subbotina, Thanasawat Chaiyakul, Gang Wang, Folke Lind, Lorenz Lampl, Frans Cronje, Albert van Den Brink, Andy Branfield, Peter Bennett, Pieter Bothma, John Feldmeier, Dirk Jan Bakker, Jacek Kot, Jordi Desola, Joseph Ting

Apologies: Alessandro Marroni, Zdzislaw Sicko, Gao Chunjin, Dr Pisarella

1. Office bearers:
President: Dr Pisarella (represented at this meeting by Nina Subbotina)
Immediate Past President: Frans Cronje
Executive Director: Alessandro Marroni
Secretary/Treasurer: Michael Bennett
Members at large: John Feldmeier (term 2), Zdzislaw Sicko (term 2), Jordi Desola (term 1), Folke Lind (term 2), Pieter Bothma (term 1), Peter Germonpré (term 1), Lorenz Lampl (term 1).

2. Frans Cronje opened the meeting and introduced members to each other. Dirk Bakker took the place of Alessandro Marroni as Executive Director for this meeting.

3. 2014 Congress: Representations were made for Buenos Aires and Orlando, Florida. John Feldmeier nominated and MB seconded the motion to accept the proposal from Buenos Aires, Argentina – date in 2014 to be advised. Accepted unanimously. Details will be posted as soon as possible on the website.

4. As a result of this decision, Nina Subbotina accepted the role of President on behalf of the absent Dr Pisarella.

5. 2017 Congress: An informal discussion raised the possibility of a combined meeting with the UHMS (John Feldmeier) in the USA, a meeting alone or in combination with the EUBS in Amsterdam (Dirk Bakker) or a meeting in Stockholm (Folke Lind).

6. Notes from the Secretary: Mike Bennett outlined the developments on several fronts since our last meeting:

6.1 Finances: the ICHM has no money. The previous Secretary/Treasurer (Fred Cramer) had been financing the newsletter and website personally.
6.2 New members: nil to report.
6.3 Web site: still operational and updated with current meeting dates.
6.4 Newsletter: MB had discontinued the newsletter as this had been produced without external finance. He has negotiated a new arrangement with the journal *Diving and Hyperbaric Medicine* to have one or two pages of ICHM news and business twice yearly. Members of the ICHM are encouraged to join either the South Pacific Underwater Medicine Society or the European Undersea and Baromedical Society in order to receive this journal.

7. Thanks: The meeting formally thanked Frans Cronje and his team for the wonderful meeting. A vote of thanks was also offered for the outgoing committee members Jochen Freier, Professor Kawashima and Professor Gao Chunjin.

Meeting closed: 1400 h

Norwegian University of Science and Technology Diving Medicine Course

Dates: 17–23 September 2011
Venue: Trondheim / Stokkøya, Norway

The course is designed to partially fulfil the requirements for qualifying as a Diving Medical Physician (Level IIa), and emphasises hands-on experience in handling diving accidents and organisation of diving expeditions. The course will follow the training standard of DMAC/EDTC.
The following requirements must be fulfilled before being accepted to the course:

- MD
- Successful completion of an internet e-learning course "*Fundamentals of diving medicine*" or completion of an accredited course "*Medical examiner of divers*"
- Diving certificate according to Euro-Norm 14153-1/ ISO 24801-1from any recognised agency

Applications must be received by 01 August 2011
For further information contact:
<http://baromedicine.eu/divemed>

Royal Australian Navy Medical Officers' Underwater Medicine Course 2011

Dates: 07–17 November 2011
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: TBC (Approx AUD2,100 including accommodation at HMAS PENGUIN or AUD800 without accommodation)

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> with copy to: <Peter.Smith33@defence.gov.au>

Hyperbaric Technicians and Nurses Association Annual Scientific Meeting 2011

Dates: 14–17 September 2011
Venue: Crowne Plaza, Coogee Beach, Sydney, Australia

Hosts: Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital

Guest Speakers:
- Tim Smith, Deputy Director, Heritage Branch, NSW Dept of Planning
- Paul Sheffield, Chair, UHMS Education Committee
- Harriet Hopf, Professor and Director of resident research training, University of Utah
- Claude Wreford-Brown, Virginia Mason Medical Center, Seattle
- A/Prof David Smart, Medical Director, Hobart Hyperbaric Medicine Unit

Workshop: 14 Sep 2011
Discussion/consensus on deep therapeutic tables

Further details available soon at:
<http://www.htna.com.au/conference.htm>

The Australia and New Zealand Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine

Dates: 20 February–02 March 2012
Venue: Prince of Wales Hospital, Sydney, Australia

Course content includes:
- History of hyperbaric oxygen
- Physics and physiology of compression
- Pressure-related injuries (barotraumas, decompression illness)
- Accepted indications for hyperbaric oxygen therapy
- Wound assessment, including transcutaneous oximetry
- Gas toxicities, marine envenomation, drowning
- Practical sessions, including assessment of fitness to dive
- Visit to HMAS Penguin
- Visit to the NSW Water Police

This course is approved as a CPD Learning Project by ANZCA – Cat 2, Level 2 – 2 credits per hour (Approval No. 1191)

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

Royal Adelaide Hospital

Medical Officers' Course 2011
Units 1 and 2: 28 November–09 December

Diver Medical Technicians' Full Course
Units 1, 2 and 3: 07–25 November

DMT Refresher Course
Units 1 and 2: 29 August–09 September

Accommodation at a reasonable price is available for a limited number of registrants. Costs available on enquiry.

Contact for information:
Lorna Mirabelli, Senior Administrative Assistant
Hyperbaric Medicine Unit
Royal Adelaide Hospital, North Terrace
Adelaide, SA 5000
Australia
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lorna.mirabelli@health.sa.gov.au>

EUBS 38th Annual Scientific Meeting 2012 Preliminary announcement

Dates: 12–15 September 2012
Venue: Sava Centar, Belgrade, Serbia

ECHM Consensus Conference on Hyperbaric Medicine
11–12 September

Enquiries to: Mariana Sedlar
E-mail: <chm@scnet.rs>

Scott Haldane Foundation

The basic course (Part I plus Part II) complies fully with the current EDTC/ECHM curricula, and the different advanced courses offer modules to achieve Level IIa status according to the EDTC/ECHM guidelines.

17 September: Refresher course Fitness to Dive (AMC, Amsterdam, NL)
07–08 October: 17th Advanced course, Dive safety (NL)
12–19 Nov (departure 10 Nov): Basic course, diving medicine Part I (Palau)
19–26 Nov (departure 17 Nov): 19th Advanced course (Palau)
26 Nov– 03 Dec (departure 24 Nov): 19th Advanced course (Palau)

For further information: <www.scotthaldane.nl>

Oxygen and Infection: European Committee for Hyperbaric Medicine (ECHM) Conference Proceedings

Free video lectures from the May 2009 Stockholm meeting are available for your iPhone or computer
<www.hyperbaricoxygen.se>

5th Karolinska Postgraduate Course in Clinical Hyperbaric Oxygen Therapy
14 lectures on fundamental concepts and front-line knowledge in the clinical use of HBO.

ECHM Conference 'Oxygen and Infection'
22 lectures and three panel discussions are available on topics such as necrotizing fasciitis and the diabetic foot.

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

Inter-university Diploma in Diving and Hyperbaric Medicine, France

For further information go to:
<http://www.medsubhyp.org> or
<http://medecine.univ-lille2.fr/format/diu/hyperbar.htm>

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_/_Termine/Kurse.html>

British Hyperbaric Association Annual Scientific Meeting 2011

Dates: 20–22 October 2011
Venue: QinetiQ Haslar, Haslar Marine Technology Park, Gosport

Hosts: QinetiQ Hyperbaric Medicine Unit, St Richard's Hospital, Chichester

Full information will be on the BHA website in the near future <http://www.hyperbaric.org.uk>

Contact for further information:
Dr Mark Glover
E-mail: <maglover1@qinetiq.com>
Phone: +44-(0)1243-776621

Undersea and Hyperbaric Medical Society

45th Annual Scientific Meeting
Preliminary announcement

Dates: 20–23 June 2012
Venue: JW Marriott Desert Ridge Resort, Phoenix AZ
Contact: www.uhms.org

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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

Instructions to authors

(Short version, updated November 2010)

Diving and Hyperbaric Medicine welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing. An accompanying letter signed by all authors should be sent.
Contributions should be sent to:
The Editor, Diving and Hyperbaric Medicine,
C/o Hyperbaric Medicine Unit, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand.
E-mail: *<editor@dhmjournal.com>*

Requirements for manuscripts
Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Microsoft® Office Word 2003. Paper submissions will also 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 supplied. The text should generally be subdivided into the following sections: an 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 or single-spaced, using both upper and lower case. Headings should conform to the current format in *Diving and Hyperbaric Medicine*. 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. **Case Reports** should not 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 wordprocessor document, only their position indicated. No captions or symbol definitions should appear in the body of the table or image.

Table data may be presented either as normal text with tab-separated columns (preferred) or in table format. No gridlines, borders or shading should be used.
Illustrations and figures should be submitted as separate electronic files in TIFF, high resolution JPG or BMP format. If figures are created in Excel, submit the complete Excel file. Large files (> 10 Mb) should be submitted on disk.
Photographs should be glossy, black-and-white or colour. Colour is available only when it is essential and will be at the authors' expense. Indicate magnification for photomicrographs.

References
The Journal reference style is the 'Vancouver' style (Uniform requirements for manuscripts submitted to biomedical journals, updated August 2009. Website for details: <http://www.nlm.nih.gov/bsd/uniform_requirements.html>). References must appear in the text as superscript numbers at the end of the sentence after the full stop.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples of the 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 being considered for publication.

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

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Full instructions to authors (revised June 2010) may be found on the DHM Jounal, EUBS and SPUMS websites.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

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

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

EUROPE
+39-06-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-07740-251-635

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>

DIVING INCIDENT MONITORING STUDY (DIMS)

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

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

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