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Saturation therapy for severe decompression sickness

Lung function in New Zealand occupational divers

Treatment of decompression illness in Scotland

Australian diving fatalities in 2004

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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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DIVING and HYPERBARIC MEDICINE
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Editor:
Michael Davis,
c/- Hyperbaric Medicine Unit
Christchurch Hospital, Private Bag 4710
Christchurch, New Zealand
Phone: +64-(0)3-364-0045 or (0)3-329-6857
Fax: +64-(0)3-364-0817 or (0)3-329-6810
E-mail: editor@dhmjournal.com>

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The Editor's offering

In a previous issue, a clinical audit for Scottish hyperbaric units was described, based retrospectively on a consecutive series of 104 divers presenting with probable decompression illness.[1] In this issue, the treatment and outcomes for over 500 Scottish cases are reported by Sayer et al using this audit format prospectively. These data do not reveal any startling or unexpected findings, but rather confirm many of the key features of retrospective case series reported in the past. What is important here, however, is the use of a standardised, prospective reporting procedure, allowing some useful conclusions to be drawn. Most importantly the effectiveness of US Navy treatment table 6 (USN 6, or its UK equivalent), with or without extensions, is confirmed in achieving a good outcome in the vast majority of cases. Secondly the link between the severity of symptoms at presentation and the likely outcome status for the patient is clearly seen – the worse the injury, the poorer the outcome overall. Equally important is the observation, all be it in only a relatively small number of divers (33 cases out of this series), that shorter and/or shallower oxygen tables as first treatment do not appear to provide as good outcomes as USN 6 even for mild cases. This brings into question the continued use of US Navy treatment table 5 or similar for 'Type I' decompression sickness, as widely practised around the world.[2]

The question of "where to from here?" when a diver is failing to respond or even deteriorating during recompression therapy is not resolved by this report. An accompanying paper describing the management of two divers who underwent 'saturation' therapy, one on air-oxygen and the other heliox-oxygen, with successful outcomes is interesting, but the Scottish experience is limited to only 20 divers overall in this series. The coordinated approach, with a preparedness to transfer cases between hyperbaric units with differing capabilities (only the Aberdeen unit is able to undertake saturation treatments and has full intensive care support in the associated base hospital) is important clinically and to be commended. Recently all the Scottish chambers have been upgraded to provide heliox Comex 30 treatments, based on the anecdotal evidence that heliox improves outcome in severe cases; benefits from this clinical management change will be difficult to assess.

For over three decades, this journal has published detailed case reports of diving fatalities in Australia. We continue to do so because we believe this contributes to diving safety by improving our understanding of the underlying factors at play, so that corrective action can occur where possible. Many medical readers have reaffirmed over the years that they wish to see such reports continue. There has been a major shift in the manner in which these reports are now compiled. Formerly this fell on the shoulders of a single physician, Douglas Walker, who set up Project Stickybeak in the early 1970s. From the 2004 report in this issue onwards, a team coordinated by researchers from DAN Asia-Pacific under John Lippmann and consisting of Drs Walker, Chris Lawrence, a pathologist with a special interest in the investigation of diving deaths, and Andrew Fock, a diving physician from the Alfred Hospital, will collate, analyse and report on these cases. In the near future, DAN Asia-Pacific hopes to expand these reports to include New Zealand and eventually the wider Asia-Pacific region.

The centralised review of medical fitness of New Zealand occupational divers has been described previously in a paper reporting the limited predictive value of entry-level medical examination.[3] This database was computerised a few years ago, allowing retrospective analysis of the first five-year cycle of examinations. The Auckland group now report that changes in spirometry over this time period are minor and probably of a low clinical significance, and they question the value of regular (annual) lung function testing of occupational divers. They point out, however, that this outcome may be biased by an 'healthy worker effect'.

Alf Brubakk and Andreas Møllerløkken review the interactions between bubbles and the vascular endothelium and the complex mechanisms involved that contribute to decompression sickness. They describe how these may be influenced by various factors, such as exercise, and postulate a novel approach to reducing the risks of decompression injury.

Because of the increasing number of papers being submitted to the Journal and to expand its peer-reviewing expertise, we are pleased to welcome Drs Jacek Kot from Poland and Simon Mitchell from New Zealand onto the Editorial Board. The first joint scientific meeting between the Asian Hyperbaric and Diving Medicine Association (AHDMA) and SPUMS is to be held in Malaysia in 2010. We urge members of SPUMS and EUBS to support this meeting and our Asian colleagues, whose Society has only been in existence for a few years.

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Michael Davis

The front-page picture of a 'christmas tree' worm, *Spirobranchus giganteus,* a tube-building polychaete worm, was taken in Vanuatu by Dr Martin Sayer.

Original articles

Analysis of two datasets of divers with actual or suspected decompression illness

Martin DJ Sayer, John AS Ross and Colin M Wilson

Key words
Clinical audit, decompression illness, decompression sickness, diving, recompression, treatment, hyperbaric oxygen therapy, outcome

Abstract
(Sayer MDJ, Ross JAS, Wilson CM. Analysis of two datasets of divers with actual or suspected decompression illness. *Diving and Hyperbaric Medicine*. 2009;39(3):126-32.)
Introduction: We examined national and single-centre datasets in Scotland to determine any trends in the treatment of diving-related disease and to assess how the choice of first treatment may be linked to the divers' condition on referral and on discharge.
Method: Two datasets were analysed: (1) 300 divers treated for actual or suspected decompression illness by the Dunstaffnage Hyperbaric Unit (Oban) between 1972 and 2007; and (2) 536 divers treated by the Scottish recompression chamber network between 1991 and 2003 (some data were common to both sets). The type and frequency of initial and any subsequent hyperbaric treatment used were examined. Any trends in demographics, reasons for diving, dive series profiles and condition on admission were examined.
Results: Ninety to 92 per cent of treated divers received standard or modified Royal Navy treatment table 62 (RN 62) or US Navy table 6 (USN 6) for their primary treatment. Nearly a third of the divers (32%) were rated as having a severe condition on admission; only 4% had a severe condition on discharge. Analysis of outcome versus treatment type was complicated by divers with more severe symptoms on referral tending to have a worse outcome (concomitant with their referral condition) while receiving more prolonged and complex treatments.
Conclusions: Shorter and shallower treatment tables (e.g., US Navy table 5, Royal Navy table 61), when used as first treatment, may result in poorer outcomes compared with RN 62/USN 6 treatment. Although subject to ongoing analysis, the shorter and/or shallower treatments have been discouraged as a first treatment in Scotland.

Introduction

The treatment of decompression illness (DCI) has been reviewed extensively and consists almost entirely of therapies based on re-pressurisation combined with breathing oxygen-rich gas mixtures over varying pressure/time schedules.[1–4] Recompression reduces bubble size with concomitant increase of internal bubble pressure (promoting spontaneous resolution and/or enhanced outward gas diffusion) with associated effects on adjacent tissues and secondary inflammation.[1–4] Oxygen-rich breathing gases increase inert-gas pressure differences between bubbles and external tissues while promoting recovery of hypoxic tissue damage and normal tissue function.[1–4]

Present-day therapeutic regimes for treating DCI are based mainly on treatment algorithms devised for military divers (e.g., US Navy, UK Royal Navy).[5,6] Therapeutic procedures developed in support of commercial diving operations are also sometimes employed, and modifications to standard recompression tables have been created for location-specific use.[7–9] The lack of definitive guidance for recompression therapy, coupled with ongoing changes in diving populations and their diving practices, means that periodic analysis of treatment practices may be beneficial.[10] Participants in a national registration service for emergency recompression in Scotland are required to contribute to a process of clinical audit.[11] This present account examines national and single-centre datasets being generated in Scotland to determine any trends in the treatment of diving-related disease. An initial attempt is made to assess how the choice of first treatment may be linked to the condition of the divers on referral and their subsequent condition on discharge.

Methods

The present study adheres to the procedures of implied consent operated by the UK National Health Service for clinical audit. The opinion of the Chairman of the North of Scotland Research Ethics Service was that ethical approval was not necessary for the conduct of clinical audit.

Two datasets were available to this study:

- A national dataset consisting of the audit records of 536 consecutive cases of DCI treated almost entirely by four Scottish recompression chambers (Aberdeen, Oban (Dunstaffnage), Orkney, and Cumbrae) from October 1991 to December 2003;
- A single-centre dataset made up of a summary of the clinical records of 300 consecutive cases of DCI treated at the Dunstaffnage Hyperbaric Unit near Oban from May 1972 to September 2007.

Both datasets adhered to the standardised data collection format detailed by Ross and Sayer; there was some duplication between datasets (n = 150).[11] Data entry was retrospective into both datasets prior to 1996; all entries were quality-assured at national and single-centre levels by authors of this account (JASR and CMW respectively). The total Dunstaffnage dataset (DHU_{total}) was divided into three subsets to assess any temporal variation (DHU_{100}: patients 1–100, 1972–1996; DHU_{200}: patients 101–200, 1996–2001; DHU_{300}: patients 201–300, 2001–2007).

The two databases were assessed initially for any trends in demographics, reasons for diving, dive history and clinical condition on admission. Analyses of treatments examined initial patient management such as surface oxygen, time from symptom onset to treatment, primary and secondary hyperbaric treatments, and condition on discharge.

It was expected that, in general, clinicians would choose more aggressive recompression schedules for the more severe disease states and that any analysis of the efficacy of treatment tables would be confounded by this. In addition, the predominant use of the Royal Navy treatment table 62 (US Navy treatment table 6 equivalent; RN 62/USN 6) in standard or modified forms produced a highly skewed population denominator. Nevertheless, the efficacy of the tables used in the first instance apart from the predominant RN 62/USN 6 and saturation treatment regimes was worthy of investigation. Treatment outcome was studied by comparing the recompression table groupings RN 62/USN 6, RN 62/USN 6 with extension, air or helium oxygen saturation and either hyperbaric oxygen (HBO) or RN 61/USN 5. A single US Navy treatment table 4 (USN 4) was omitted from the analysis since it was atypical. Clinical condition on referral was assessed for each treatment.

STATISTICAL ANALYSIS

Statistical analysis followed preliminary examination for normality using modified (Lilliefors) Kolmogorov-Smirnov tests with transformation where necessary; no assumptions were made for common data.[12–14] Definition of severity at presentation and outcome followed Ross and Sayer, where the most serious symptom defined patient condition.[11]

To compare treatment outcome, condition on discharge category was collapsed to 'good' outcome (no symptoms or minor pain or sensory symptoms only) and 'poor' outcome (any ataxia, any motor weakness, cerebral dysfunction or presence of a urinary catheter). The association between poor outcome and treatment method was assessed using a binary logistic regression model adjusted for time from onset of symptoms to recompression, age, the year in which the treatment took place, the condition of the patient on referral at two levels – 'mild' (pain only, sensory or ataxic symptoms) or 'severe' (motor weakness, nausea/vertigo or cerebral dysfunction) – and whether the patient's condition relapsed after treatment. Treatment efficacy was also assessed by the clinician's assessment of the immediate response to treatment.

The predictive power of the logistic regression model was assessed using receiver operating characteristic (ROC) curve analysis whereby the area under the ROC curve equates to the c-statistic. Acceptable discrimination within the model would be where c-statistic values were between 0.7 and 0.8; a value of 1.0 would be perfect discrimination.[15]

Results

Of the 536 patients examined in the Scottish dataset, 238

Table 1
Demographic and diving data for divers treated for actual or suspected decompression illness in Scotland (1991-2003) and Dunstaffnage Hyperbaric Unit (DHU; 1972-2007) (* age data: square root transformed; † depth-of-last-dive data did not conform to normality; ‡ data not collected).

	Scotland (1991–2003)	**DHU_{total}** (1972–2007)	**DHU_{100}** (1972–1996)	**DHU_{200}** (1996–2001)	**DHU_{300}** (2001–2007)
Male (%)	84	81	83	84	76
Age (y) mean*	34.4	35.0	33.4	34.7	36.7
95% CI*	+0.83, -0.82	+1.25, -1.23	+1.97, -1.92	+1.96, -1.89	+2.50, -2.42
median	34.0	34.0	33.0	35.0	34.5
range	14–73	16–77	17–66	18–62	16–77
Recreational (%)	85	84	86	86	79
Depth of last dive (m)†					
median	32	30	30	28	30
range	5–115	6–91	12–58	10–59	6–91
number (%) over 50 msw	30 (5.9)	16 (5.8)	2 (2.9)	8 (8.3)	5 (5.0)
Mean # of dives in last 48 h	‡	2.99	2.83	2.87	3.27
Sample size (n)	511–535	267–286	74–86	100	100

(44%) were treated by the Aberdeen centre, 151 (28%) were from Dunstaffnage (and were common to both datasets), 77 (14%) were from Orkney, 53 (10%) were from Cumbrae and 17 (3%) were from other treatment centres in Scotland or had received primary treatment outside Scotland prior to being transferred for secondary treatment. Overall, 96% of treated divers either made a full recovery or were left with only mild clinical residua.

The sex ratio of treated divers in all datasets was in the range of 76–84% male; 79–86% of patients treated were recreational divers (Table 1); there was no significant difference in sex ratio between the three subsets of DHU data (G-test, $P = 0.789$). Diver age ranged from 16–77 years; mean and median values ranged between 33 and 37 years (Table 1). There were no significant differences in diver age between the Scottish and DHU_{total} groups (Z-test, $P = 0.631$); mean age increased stepwise from DHU_{100} to DHU_{300} (Table 1) but the trend was not significant (Kruskal-Wallis, $P = 0.298$). The median depth of the incident (assumed last) dive in the Scottish data was not significantly different than that of the DHU_{total} group (Mann-Whitney, $P_{(same)} = 0.028$), and there were no significant differences between median depths of last dive in the DHU subsets (Kruskal-Wallis, $P = 0.833$). Nearly 6% of incident dives in the Scottish and DHU_{total} groups were deeper than 50 msw; this value ranged from 2% to 8% in the DHU subsets. The mean number of dives in the 48 h preceding the incident dive was approximately three in all groups where it was recorded (Table 1).

Of divers in DHU_{total}, 75% had received normobaric oxygen on transfer to DHU. However, there was an asymptotic trend with 84–88% receiving oxygen in the DHU_{200} and DHU_{300} groups, compared with 54% in the DHU_{100} group, which included the period prior to the widespread introduction of surface oxygen as a first-aid measure. Median (and interquartile range) time to treatment following onset of symptoms was 5.8 h (0.8–13.5) for Scottish data ($n = 470$) and 3.0 h (2.0–5.0) for DHU_{total} data ($n = 274$).

The Scottish data ($n = 535$) indicated that, on admission, 11.0% of divers had no symptoms, 25.4% had pain-only symptoms, 20.7% had altered sensation, 11.0% were ataxic, 17.8% had motor difficulties, 2.0% had bladder/rectal dysfunction and 12.0% had cerebral disturbances (including vestibular decompression sickness (DCS)). In total, 68% of divers captured in the Scottish dataset were considered to be in a mild to moderate condition on admission; 32% were considered severe. The DHU_{total} data ($n = 286$) were characterised on final diagnosis; the main category groups were 5.1% of divers being treated for omitted decompression, 4.4% for cutaneous DCS, 14.2% for pain-only, 3.6% for vestibular DCS, 59.2% for a neurological component, and 10.8% for an embolism.

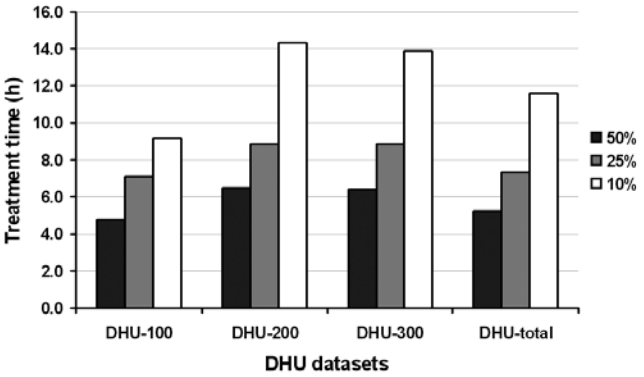
In the Scottish dataset, 90% of primary treatments ($n = 482/536$) were RN 62/USN 6; 176 of those (37%) were extended versions of the table. One USN 4 was used with poor outcome. USN treatment table 5 (USN 5) was used eight times (either as USN 5 or the Royal Navy equivalent treatment table 61, RN 61) and low pressure (range 192–243 kPa) hyperbaric oxygen treatments were used 25 times.

RN 62 was used as the primary treatment table in 276 (92%) of cases in DHU_{total} and was modified (extended) in 127 (46%) of those uses; RN 61 was used in 13 (4%) as the primary treatment. Other tables employed for primary treatment were US Navy treatment table 7 (USN 7; $n = 3$), Comex 12 ($n = 3$), USN 6A ($n = 2$), USN 5A ($n = 2$) and Royal Navy table 66 ($n = 1$); on three occasions a RN 62 was converted to RN 51, 53 or 54. In DHU_{total} there were 158 secondary treatments of which 109 were Comex 12, 15 were RN 66, 12 were extended RN 62, nine were unmodified RN 62, two were RN 61 and one was a USN 7. Total treatment times per patient in the DHU_{200} and DHU_{300} groups were significantly longer than DHU_{100} values (Kruskal-Wallis, $P = 0.007$) although there were no significant differences in the numbers of treatments (Kruskal-Wallis, $P = 0.543$; Figures 1 and 2).

Table 2
First recompression treatment (as a percentage (rounded) of patients within their respective condition grading group assigned on referral) and days spent under care in relation to the severity of the patients' condition on first contact with medical services for all-Scotland dataset

Condition on referral	USN 6 RN 62	USN 6ext RN 62ext	Helium Saturation	USN 4	HBO Low pressure	USN 7	USN 5 RN 61	Total cases (*n*)
Pain only	67	26	1	0	6	0	1	161
Sensory	61	30	0	0	7	0	2	152
Ataxia	56	36	3	0	0	4	6	36
Motor	49	38	4	0	4	0	0	71
Nausea or vertigo	38	47	11	0	0	0	4	47
Cerebral	46	39	7	1	3	3	0	69
Total cases	57	33	3	1	1	1	2	536
Days in care								
(median IQR)	2 (2–3)	3 (2–4)	8 (6–12)		2 (1–3)	12 (6–27)	2 (1–3)	

Figure 1
Total hyperbaric treatment time per patient (h) at the 10, 25 and 50 percentiles for divers treated at Dunstaffnage Hyperbaric Unit 1972–2007 (n = 300; DHU_{total}), 1972–1996 (n = 100; DHU_{100}), 1996–2001 (n=100; DHU_{200}), and 2001–2007 (n = 100; DHU_{300})



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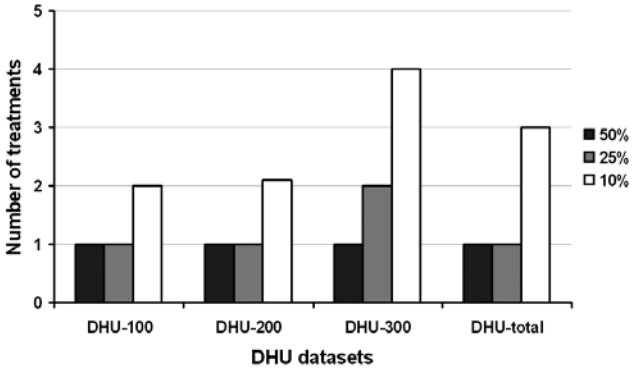

Relapses, defined as rapid and clinically significant deterioration or reversal of improvement following initial treatment, with new symptoms in most cases, occurred in equal proportions in both the Scottish and DHU_{total} groups (12%, n = 536 and 283, respectively). The time delay for relapse ranged from 0.33 to 6.20 h; although not significant, the relapse rate in the DHU_{300} group was 8% compared with a rate of 15% in DHU_{100}.

From the Scottish dataset, the type of treatment table employed for initial hyperbaric therapy was related to the severity of the presenting illness. Severe disease accounted for 28% of unmodified RN 62/USN 6 treatments, 43% of modified (extended) RN 62/USN 6 and 90% of the saturation treatments (Table 2). Only 21% of the other types of table used were associated with severe presentations and these were instead used predominantly to treat mild disease (Table 2). Longer and more aggressive forms of treatment tended to be related to a poorer patient outcome in the cases where condition on referral was more severe (Table 3).

In comparison with outcome after an unmodified RN 62/USN 6 treatment the odds for poor outcome when employing an extended RN 62/USN 6 treatment were 5.2 (95% CI 1.9, 14.1, P = 0.001) and 87.5 (95% CI 20.0, 382.3, $P < 0.001$) for a saturation treatment. The odds for a poor outcome for the other treatments were 9.0 (95% CI 1.3, 61.9, P = 0.016). Other significant factors in the analysis were age, severity on referral and relapse after treatment. The c-statistic for the regression model was 0.94 indicating a high predictive power. The initial employment of RN 61/USN 5 or low pressure hyperbaric oxygen treatments produced a much poorer response than the RN 62/USN 6 treatments (extended and non-extended; Table 4) but this does not necessarily relate to the clinical outcome at discharge.

Discussion

The data need to be considered in terms of both the general and particular approaches to handling a diving accident taken by the Scottish service. In general, first contact was rapidly

Table 3
Condition on discharge (as a percentage, rounded) and days spent in care in relation to the severity of the patients' condition on first contact with the medical services for all-Scotland database.

Days in care median (IQR)	Condition on referral	Complete resolution	Mild pain/ sensory	Motor or ataxia	Severe motor or ataxia/catheterised	Cerebral	Dead	Total cases (n)
2 (2–3)	Pain only	68	32	0	0	0	0	161
2 (2–3)	Sensory	71	28	1	0	0	0	152
2 (2–3)	Ataxia	72	22	6	0	0	0	36
3 (2–4)	Motor	59	23	7	7	4	0	71
3 (2–5)	Nausea or vertigo	60	13	23	0	4	0	47
3 (2–5)	Cerebral	61	10	10	4	13	1	69
2 (2–3)	Total cases	66	24	5	2	3	0	536
Days in care, median (IQR)		2 (2−3)	2 (2−3)	5 (4−7)	14 (8−17)	9 (4−14)		

Table 4
Response to treatment (as % rounded) against the initial treatment table; 'response to treatment' is the change in the patient's relative condition as a result of recompression treatment (i.e., a 'good' response can still be associated with a 'poor' condition on discharge)

Treatment	No symptoms at start and no change	Complete resolution	Major improvement	Moderate improvement	Slight/no improvement
RN62/USN6 (unmodified) (*n* = 306)	16	55	21	5	3
RN62/USN6 (extended) (*n* = 176)	3	42	44	7	4
Saturation Tx (*n* = 20)	0	15	60	15	10
RN61/USN5 or HBO (*n* = 33)	12	15	42	18	12

followed by a medical assessment of the victim's condition, ideally by the duty diving doctor talking to the patient or the patient's immediate carers at the scene of the accident. The available options, in order of priority and in relation to the patient's condition, were then:

- transfer to the nearest accident and emergency or trauma unit for resuscitation and stabilisation prior to assessment of the need for recompression;
- transfer to the nearest approved chamber for assessment and recompression if required;
- transfer to an accident and emergency unit or general practitioner for examination, emergency treatment if required and assessment of the need for recompression in collaboration with the duty diving doctor in Aberdeen.

The concept that rapid recompression is paramount, even in the absence of general supportive care, has never been accepted; in spite of more rapid treatment at Oban, outcomes were similar across all chambers.

Between 90 and 92% of all primary treatments delivered in the present study were standard or modified (extended) RN 62/USN 6. The other 8–10% ranged from longer or deeper tables that treated extremely severe presentation to shorter, shallower tables used mainly as precautionary treatments.

The rate of poor clinical outcome at discharge was 4% but patients who responded badly to treatment were more likely to have presented in a severe condition and, therefore, would tend to be treated by the deeper, longer therapies.[11] Subtracting that fraction of the treated population from the diving presentations in Scotland rated as 'severe' (32% of the total) shows that a considerable number of severe presentations were treated successfully with RN 62/USN 6 therapeutic tables. These oxygen tables have been highly predominant as primary treatments for DCS for well over 40 years and have replaced the previous use of single RN 61/USN 5 tables for all but precautionary or secondary therapy.[1–2] The level of satisfactory patient outcome in these data (96%) was high compared with previous similar studies.[3] However, although the major recompression schedule employed was the RN 62/USN 6 and overall outcome was good, these tables were used in the context of a recompression service with a wider capability since it was recognised that this recompression regime can be of limited efficacy in severe illness.[8,16]

During the course of this audit, a helium:oxygen Comex 30 table became available at all Scottish chambers. Throughout the audit period, intensive care at pressure and a helium:oxygen saturation capability to any required depth were available at the Aberdeen centre. If the initial condition of a patient exceeded the capability of the nearest chamber or the recompression treatment applied locally did not have a satisfactory outcome, then air transport to Aberdeen was used. Data for inter-unit transfers in the present study were too few to analyse (for example three of the last 200 DHU treatments resulted in transfer to Aberdeen; two of the last 200 DHU patients were transferred from Cumbrae). Helicopter transfers were used because other transport between treatment units is compromised by slow mountain roads and inter-island ferries (helicopter air transfer times are estimated at 16–22% of land-based times). Helicopter transfers occurred at a maximum altitude of 230 m above sea level compared with some roads having altitudes in excess of 305 m above sea level; clinically significant deteriorations were not observed in the patients undergoing transfer.

The predominance of the RN 62/USN 6 as the primary treatment table in the present study differs little from the recent accounts of Mitchell[17] and Müller et al.[18] However, this predominant use differs markedly from a review of treatment of 129 DCI cases in Italy.[19] Although that study showed that most treatments (87%) employed short oxygen/air tables, nearly 55% of that 87% were USN 5 tables or equivalents and 13% of patients were scored with residua one week post-treatment. The Scottish data show a highly infrequent employment of RN 61/USN 5 treatments with poor final outcome in only 4% of patients. However, direct comparison is inappropriate considering the lack of

equivalent information on respective severity of clinical presentation or matching of the type of treatment to the severity of presentation.

In the present study, extended RN 62/USN 6 and saturation treatments were associated with poorer outcomes on discharge from care. Those treatments, however, were used proactively with regard to the patients' response to treatment. Where a patient did not respond to an unextended RN 62/USN 6 then the treatment was extended; if patients did not respond to the RN 62/USN 6 regime (with or without extension, but usually with) then a saturation treatment was used. In this context, the immediate response to treatment is relevant (Table 4). The favourable responses to treatment for saturation and extended RN 62/USN 6 can be taken in relation to a failure to achieve an acceptable response with unextended RN 62/USN 6 treatments. If the unextended RN 62/USN 6 had not been converted, the response to treatment for that regime would have appeared worse. The same argument does not apply to the shorter and/or shallower treatments (RN 61/USN 5 and low pressure HBO) where there is doubt about their acceptability as a first-line treatment for possible DCI; their use as initial hyperbaric therapies is now discouraged in Scotland.

Both USN and RN tables are part of a recompression therapy algorithm that is pro-active and treatments should not be prescribed without knowledge of the response of the patient to recompression treatment. The algorithms also describe options to be taken in the event of failure of the patient's condition to respond to recompression. At the time of this audit, these options were largely to compress to greater depths, in air, in an attempt to get a better response. Our experience is that not only has this approach failed to produce any further response to treatment, but it also exceeded the logistic capabilities of a local unit and put attendant staff at risk of a number of factors including nitrogen narcosis, oxygen toxicity and decompression illness, and could be followed by psychological issues.

In the event of recompression failure with the RN 62/USN 6, and if the patient's condition warranted it, the table was either converted into a helium:oxygen saturation at the depth of maximum clinical response (in Aberdeen), extended into a USN 7 at 283 kPa or completed and the patient transferred to Aberdeen for further treatment with helium:oxygen saturation.[20] Although a helium:oxygen Comex 30 capability has recently become available at all Scottish chambers, it has not yet been used. While response to treatment was poorer for saturation treatments, this should be considered in relation to how the treatment was used. Saturation treatment was only initiated if an initial RN 62/USN 6 treatment had failed to produce a major response or better. Had saturation treatment not been initiated, all 20 cases in this present study would probably have been associated with only a moderate to poor response to treatment. Similar considerations apply to where RN 62/USN 6 treatments are extended.

Examining the DHU data over three sequential time periods has shown some trends in treatment patterns. On a per patient basis, total treatment time increased. However, the lack of any increase in the number of treatments given tends to suggest that this is probably caused by an increased employment of extended RN 62/USN 6 treatments. Part of the reason for extending primary tables more routinely may have been as a response to the numbers of relapses in earlier datasets.[21,22] Based on the immediate dive history, types of presentation and/or reason for diving (e.g., shellfish diving), treatments were extended more frequently. At DHU, relapse rates were halved between the DHU_{100} and DHU_{300} groups although there was a concomitant decrease in clinical severity on presentation.

Increased treatment times per patient may have been influenced by a higher prevalence of vestibular decompression sickness but also by changes in treatment practice; more secondary treatments were provided per patient at DHU from 1996 onwards. Treatment for severe vestibular DCS at DHU is based on RN 62/USN 6 tables but has evolved to minimise exposure to 283 kPa oxygen if the patient is stable, while fully extending treatment at 191 kPa. The primary treatment is then followed with a series of Comex 12 treatments, twice daily until no significant improvement is measured. Comex 12 (223 kPa air/oxygen table) replaced RN 66 (243 kPa air/oxygen table) primarily because of safety concerns to the internal attendants although, subjectively, patients appeared to recover better breathing 223 kPa oxygen compared with 243 kPa possibly because O_2 delivery may be improved where pO_2 vaso-constriction is less. Typically, therefore, treatment of vestibular DCI at DHU consists of an initial modified RN 62/USN 6 followed over about two to four days by a series of approximately 3–8 Comex 12 treatments, with treatment continuing until no improvement is measured. A recent review of diving-related inner ear problems noted that, over the past 10–15 years, the incidence of inner ear DCS as a form of DCI has changed from rare to common with a much higher awareness of its symptomatology.[23]

Although agreeing with Mitchell about the lack of substantive evidence for any particular treatment regime, changes can be made to how the treatment is administered in general and how standard treatments can be modified to anticipate the condition and reaction of certain groups of diver.[17,21,22] Although clinical outcome with an initial RN 62/USN 6 approach was associated with good outcome statistics it cannot be said that the same standard would be achieved using these tables in isolation. Accordingly, there is no plan to limit the therapeutic capability of the Scottish service on the basis of these audit results since it is recognised that decompression illness has potentially fatal consequences, can require an intensive care approach to treatment and does not always respond to the initial RN 62/USN 6 recompression treatment.

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Martin DJ Sayer BSc, PhD, FSUT is head of the Dunstaffnage Hyperbaric Unit hosted at the National Facility for Scientific Diving (NFSD) at the Scottish Association for Marine Science laboratories near Oban in Argyll, Scotland. He is also head of the NFSD, technical advisor for the Scottish Recompression Registration Service and technical representative on the committee of the British Hyperbaric Association.
John AS Ross MB, ChB, PhD, FRCA, Honorary MFOM is a Senior Lecturer in the Department of Environmental and Occupational Medicine, University of Aberdeen Medical School, Foresterhill, Aberdeen. He is also an Honorary Consultant in Hyperbaric Medicine for NHS Grampian and the Medical Director of the Scottish Recompression Registration Service.
Colin M Wilson MB, ChB, FRCA, is the Medical Director of the Dunstaffnage Hyperbaric Unit near Oban. He is also a GP Anaesthetist at the Lorn Medical Centre and the Lorn and the Islands General District Hospital, Oban. He is currently vice-chair of the British Hyperbaric Association.

Corresponding author:
Dr M Sayer, Dunstaffnage Hyperbaric Unit
National Facility for Scientific Diving, Scottish Association for Marine Science
Dunbeg, Oban, Argyll PA37 1QA
Scotland
Phone: *+44 (0)1631-559236*
E-mail: *<mdjs@sams.ac.uk>*

The long-term effects of compressed gas diving on lung function in New Zealand occupational divers: a retrospective analysis

Christopher Sames, Desmond F Gorman, Simon J Mitchell, Greg Gamble

Key words
Occupational diving, lung function, pulmonary function, health surveillance, occupational health, medicals – diving

Abstract
(Sames C, Gorman DF, Mitchell SJ, Gamble G. The long-term effects of compressed gas diving on lung function in New Zealand occupational divers: a retrospective analysis. *Diving and Hyperbaric Medicine*. 2009;39(3):133-7.)
Objectives: Long-term effects of occupational diving on lung function are uncertain. No previous study has been conducted on New Zealand occupational divers. The aim of this study was to investigate changes in divers' lung function over a five-year period.
Methods: The lung function data of all occupational divers with two spirometric determinations separated by a five-year interval (N = 336 out of 1,475 currently registered divers) were entered into a database and analysed for changes (5.6 years mean). The trends were correlated against gender, smoking status and years of diving experience (as more accurate diving exposure data were not available). Spirometric indices were compared with predicted reference values derived from New Zealand (WRS), Australian (Gore), and American (NHANES III and Knudson) populations.
Results: Small, but significant, decreases were found in FEV_1 (0.27% against predicted per annum, $P = 0.02$) and PEF (0.47% per annum, $P = 0.04$) using the NHANES III equations. No other changes in lung function parameters reached statistical significance ($P < 0.05$) using any of the four sets of prediction equations. No changes correlated significantly with reported years' diving.
Conclusion: Observed changes to occupational divers' lung function tests over 5.6 years were small and unrelated to years of diving, which might be due to a 'healthy worker effect'. Clinical relevance is unlikely, but this requires further evaluation. There was significant disparity in normative values derived from the four sets of prediction equations and there is some consequential concern about the ongoing utility of such surveillance of New Zealand professional divers.

Introduction

Lung function is arguably most important in determining health risk for divers. Disparate results from a small number of studies of the long-term effects of diving on the lung have led to ongoing uncertainty (see Table 1). A literature search of the PubMed database seeking the MeSH terms 'Diving' and 'Respiratory function tests' found 438 articles, eight of which were longitudinal studies of professional divers' lung function (plus one preliminary report).[1–9] Neither the references quoted in these articles nor the 84 articles cited in the British Thoracic Society guidelines on respiratory aspects of fitness for diving revealed any further relevant longitudinal studies.[10]

Regulations introduced by the Department of Labour in 1999 require occupational divers in New Zealand to undergo annual surveillance of their medical fitness to dive by completing and submitting to the central medical directorate a health questionnaire which is augmented every five years by a comprehensive 'medical examination'. Audit of these diver health surveillance data is facilitated by New Zealand's relatively small population and the collection and scrutiny of the data centrally and by an expert censor panel that certifies occupational diver medical fitness.

The aim of this retrospective longitudinal cohort study was to audit lung function data collected from occupational divers over the past five to 15 years (minimum of five), and to examine any relationships with gender, smoking status and years of occupational diving experience.

Method

The inclusion criteria were that the diver was currently registered with the regulator, the New Zealand Department of Labour, and that the diver had completed at least two 'full' dive medical examinations, including spirometry, with an interim period of at least five years. The annual questionnaire and five-yearly medical examination data were uploaded to a customised database for analysis.

Lung function parameters: forced vital capacity (FVC); forced expiratory volume in one second (FEV_1), peak expiratory flow (PEF), forced expiratory flow rates at 25%, 50% and 75% FVC ($FEF_{25\%}$, $FEF_{50\%}$, $FEF_{75\%}$) and mean forced expiratory flow rate in the range 25%–75% FVC ($FEF_{25\%–75\%}$) were analysed for changes over time and against gender, smoking status and duration of diving experience.

Comparison was made with matched normative data derived from four sets of published spirometry prediction equations.[11–14] The results were expressed as the percentage change of these predicted values, which controlled for advancing diver age between measurements, as all equations are based on diver age, height and gender. Two of the sets of prediction equations (Knudson and NHANES III) were chosen because of their popularity worldwide, and two

Table 1
Comparison of longitudinal studies on lung function of divers

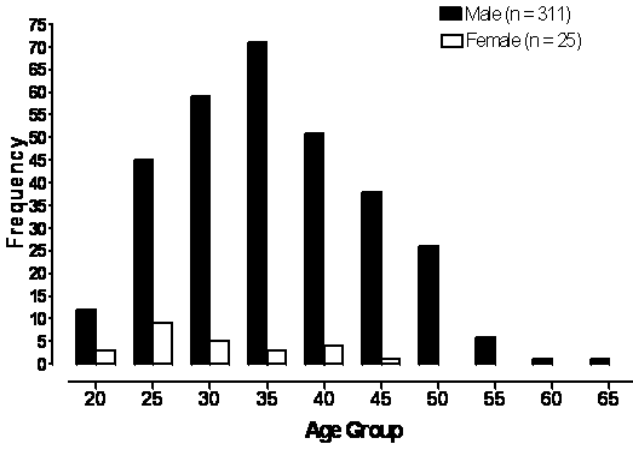
Author	Study design	Sample (*n*)	Time (y)	Outcome
Davey IS et al 1984[4]	Retrospective Epidemiological	255	5+	Change in FVC (but not FEV_1) related to max depth; decreased FEF_{75} compared with controls; evidence of airway narrowing possibly related to loss of elastic tissue
Watt SJ 1985[1]	Retrospective Epidemiological	224 123	3–4 5–9	Decrease in FVC > decrease in FEV_1; reduction in FVC significant compared with predicted norms, correlated with initial FVC (mostly above predicted norms); not correlated with age, max depth, yrs diving or weight change; no difference between smokers and non-smokers. Indicated either gradual return to normal values or pathological decrease in lung volume
Bermon S et al 1994[5]	Retrospective Epidemiological	20	8–9	Decrease in VC < decrease in FEV_1 so decrease in FEV_1/VC% over time; pronounced decrease in MMEF and MMEF/VC suggested chronic effect on small airways
Skogstad M et al 2002[3]	Prospective Controlled Cohort	77	6	Significant decrease in FVC, FEV_1, MEFRs and TI_{co} compared with reference group (policemen); reduction in FEF_{25} and FEF_{75} greater than in reference group and related to cumulative number of dives. No difference between smoking and non-smoking divers
Fitzpatrick DT et al 2003[6]	Retrospective Epidemiological	43 (shallow nitrox)	3	Initial FVC and FEV_1 greater than predicted; FVC and FEV_1 significantly increased after three years associated only with cumulative dive hours; no significant change in other parameters; likely training effect
Tetzlaff K et al 2005[7]	Retrospective Epidemiological	39 (O_2 rebreathers)	5.8	No significant change in FVC or FEV_1; hyperoxia not associated with decline in lung function
Tetzlaff K et al 2006[8]	Prospective Controlled Cohort	468	5	Baseline lung function of divers and controls (submariners) greater than predicted; decrease in FEV_1 faster if older, smoker or initially higher FEV_1; no difference in decline in FEV_1 between divers and controls
Chong SJ et al 2008[9]	Retrospective Epidemiological	116	5	Increase in % predicted FVC, FEV_1 and PEF; decrease in FEV_1/FVC ratio; no significant difference with age, smoking or years, naval service

sets (Gore and WRS) because of their local relevance.[11–14] Knudson's 1983 equations improved on his 1976 set and were derived from 697 non-smoking, healthy, white, non-Mexican-American residents of Tucson, Arizona.[11] The NHANES III (Third National Health and Nutrition Examination Survey) equations were based on data collected across the USA from a total of 20,627 subjects divided into three ethnic groups.[14] However, after selecting only those who were life-long non-smokers who could provide at least two acceptable FVC manoeuvres, the equations were derived from 7,429 subjects. For use in our study, only the equations derived from the data for Caucasian subjects older than 20 years (n = 1,349) were used. The Australian set of equations (Gore) was derived from 414 asymptomatic, non-smoking Caucasian adults from metropolitan Adelaide, South Australia, while the New Zealand set, the Wellington Respiratory Survey (WRS), was derived from 212 healthy, non-smoking Caucasian adults. Comparable reference equations for Maori and Pacific Island populations are not yet available.[12,13]

Student's paired t-test was used to test the hypothesis that there was no change in function over five years. The same test was used to find whether the baseline values of lung function tests differed from the normative means.

The relationship between dependent variables (recorded lung function) and several predictor variables (covariates

Figure 1
Age distribution of 336 New Zealand occupational divers by gender



such as gender, age, weight, smoking status and number of years' diving experience) was tested by univariate Pearson correlation coefficients and multiple linear regression analyses ($P < 0.15$ was considered necessary for inclusion in the multiple regression model). A variety of iterative procedures was used (stepwise regression, forward and backward selection and MaxR). The final model was chosen on the basis of goodness-of-fit and biological plausibility. All analyses were conducted using procedures of SAS (SAS Institute Inc. v 9.1). A *P*-value of less than 0.05 was considered significant and all tests were two-tailed.

Results

Of the 1,475 currently registered occupational divers in New Zealand, only 336 (23%) satisfied the inclusion criteria by having two sets of spirometric data separated by five years. Their demographic details are summarised in Table 2. The divers' occupational groupings were broadly categorised as commercial (148), scientific (122), sports and recreation industry (30) and military (15). At baseline, females (7.4% of the group) were on average six years younger and had 7.6 years less diving experience than males. The comparative gender/age distribution is shown in Figure1. Only 15 and 7% respectively of the divers reported their total number of dives and dives beyond 30 msw in the past year, compared to 96% who reported their total number of years' compressed gas diving.

Frequency of paired data varied according to spirometric parameter as shown in Table 3. The only significant difference over 5.6 years between smokers (defined as current and ex-smokers) and non-smokers (72.6% of the group) was a decrease of 3% in the % predicted FEV_1 in non-smokers according to the Knudson equations.

Three sets of normative value equations (Gore, Knudson and WRS) showed a 6% increase in % predicted FVC in females over the observation period. The WRS equations also showed that females had lower than predicted baseline FVC and FEV_1 values (7.4% and 8.5% respectively). Student's paired t-testing revealed no significant differences in lung function parameters when the group was stratified for age and years of diving experience (using the median value as the dividing point for young versus old and low versus high experience).

The NHANES III equations most accurately predicted the recorded values for FVC, FEV_1 and PEF and were the only equations to demonstrate significant change (for % predicted FEV_1 and % predicted PEF) for the group as a whole over the observation period. The mean changes in % predicted FEV_1, FVC and PEF, 95% confidence intervals and *P*-values are displayed in Figure 2. A comparison of changes in lung function over time employing all four prediction methods is shown in Table 4.

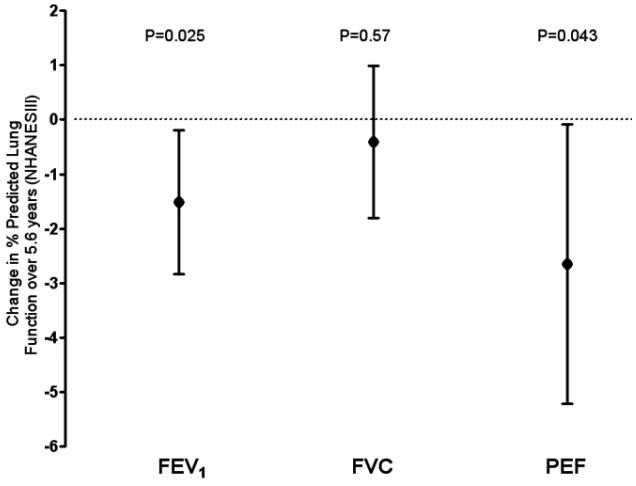
Table 2
Demographic characteristics of 336 New Zealand occupational divers at initial assessment of medical fitness for diving

	n or mean (SD)	Range
Male / Female	311 / 25	
Height (cm)	177.9 (7.1)	158–196
Weight (kg)	82.3 (12.8)	50–116
BMI ($kg.m^{-2}$)	26 (3.4)	20–36
Age (y)	35.6 (8.6)	18–65
Smoker (current)	33	
Smoker (past)	25	
Non-smoker	278	
Years' occupational diving	13.8 (8.8)	0–42
No. dives in past year ($n = 52$)	97 (117)	0–600
No. dives > 30 msw depth in past year ($n = 25$)	5 (14)	0–50
Time to 2nd examination (y)	5.6	4.8–12

Table 3
Frequency of paired data by spirometric parameter

Spirometric parameter	Number of observations	% of sample
FVC	328	98
FEV_1	330	98
FEV_1/FVC	325	97
PEF	174	52
FEF_{25}	54	16
FEF_{50}	77	23
FEF_{75}	63	19
FEF_{25-75}	70	21

Figure 2
Change in % predicted FEV_1, FVC and PEF over 5.6 y (mean) using NHANES III prediction equations; dots are the mean values and the arms represent the 95% confidence intervals



Discussion

With few exceptions, both epidemiologic and experimental studies have concluded that compressed gas diving is detrimental to divers' lung function.[15] The mechanism for the deterioration in lung function is not completely understood, but several factors have been implicated both independently and in combination. However, the small changes in lung function found in this study and in others, suggest a low likelihood of clinical significance and raise the question of the value of regular lung function testing. The two relevant and controlled prospective studies showed similarly small and probably clinically insignificant changes over a similar timeframe.[3,8] The clinical significance of respiratory function changes from diving in terms of divers' careers, quality of life and morbidity after retirement consequently remains unknown. This contrasts with the recommendations for such studies in response to the international consensus conference in Norway in 1993.[16]

The four prediction methods used here were chosen because of their relevance to New Zealand divers and their local and global popularity.[11–14,17] However, the variable results (*P*-values ranging from 0.02 to 0.97) suggest a poor fit of at least some of these equations with this dataset. The accuracy of the NHANES III equations in predicting the FVC, FEV_1 and PEF values, together with their demonstration of significant change in % predicted FEV_1 and PEF values over the observation period, implies greater accuracy but less precision than the other sets of predictive equations for this dataset.

Divers' lung function is measured on a variety of equipment and is calibrated against different sets of reference algorithms. This study showed that the most appropriate dataset for deriving normative values with which to compare New Zealand occupational divers is the NHANES III equations; it is reassuring that these equations are the most commonly used in New Zealand.[17]

The small, mostly insignificant changes and the lack of correlation with reported number of years' diving suggests a 'healthy worker effect', which is a form of sampling bias recognised since 1885. Put simply, the working population is likely to be healthier than the general population, which includes those who are not working for health reasons. Erroneous conclusions can be drawn if this is not taken into account. In the current study, the sets of 'normative' lung function prediction equations were based on groups of healthy Caucasian non-smokers with no clinical evidence of respiratory disease. No information was available on occupation. Retired divers' files were not included in this audit, but, some divers might retire early for respiratory health reasons compounding any 'healthy worker' bias.

Previous studies, such as those of Skogstad and Tetzlaff which used control groups of occupations of similar physical nature but without any diving (such as policemen or submariners), are more likely to reach valid conclusions than those based on more heterogeneous groups.[3,8]

Recent research on New Zealand occupational divers found that regular five-yearly medical examinations result in very few divers having their certificates of medical fitness changed.[18] The observation that only 22.8% of registered occupational divers met the inclusion criteria for this study suggests that few divers continue occupational diving for longer than five to ten years. The possibility of premature health-related retirement is a subject for future research.

Table 4
Comparison of mean changes in % predicted FVC, FEV_1 and PEF over 5.6 y using four prediction methods; mean (standard deviation) and *P*-values shown

	FVC (*n* = 328)		**FEV_1** (*n* = 330)		**PEF** (*n* = 174)	
NHANES III	-0.41 (12.84)	0.566	-1.51 (12.14)	0.024	-2.65 (17.10)	0.043
Knudson	0.79 (13.63)	0.292	-1.06 (12.50)	0.123	-1.75 (18.56)	0.214
WRS	0.51 (12.20)	0.451	-1.05 (11.17)	0.087	-0.12 (2.63)	0.552
Gore	-0.27 (13.00)	0.707	-1.24 (12.02)	0.062	-2.09 (16.32)	0.094

Conclusions

Decreases in occupational divers' lung function over a period of 5.6 years are minimal and of doubtful clinical significance, but, any changes may be obscured due to a 'healthy worker effect'.

The mean 5.6-year observation period for this study may have been too short to observe clinically significant changes in lung function, but, does reflect the relatively short mean duration of occupational diving careers. Future study should involve the long-term follow-up of retired divers.

Apart from anatomic lung abnormalities, or a history of previous pulmonary barotrauma (PBT), the only factor reported to be associated with an increased risk of PBT or cerebral artery gas embolism is a small FVC, and, in most cases of PBT, none of the many commonly recognised risk factors is present.[19–21] Given this, and the results of the current and previous audit on the NZ occupational diver population, it is hard to justify annual comprehensive lung function testing.[18]

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Chris Sames, BSc, BHB, MBChB, MMedSc, is Hyperbaric Medical Officer to the Naval Health Service.
Des Gorman, BSc, MBChB, MD, PhD, is Head of the School of Medicine, and
Simon Mitchell, MBChB, PhD, FANZCA, is Senior Lecturer in the Department of Aneasthesiology,
Faculty of Medicine and Health Sciences, The University of Auckland.
All three are involved in the Diving Medical Directorate to the New Zealand Department of Labour.
Mr Greg Gamble, MSc, is biostatistician in the Faculty of Medicine and Health Sciences, The University of Auckland.

Corresponding author:
Professor Des Gorman, Head of the School of Medicine
Level 12, Support Bldg, Auckland City Hospital,
FMHS, University of Auckland, Private Bag 92019, Auckland, New Zealand.
Phone: +64-(0)9-923-2940
Fax: +64-(0)9-373-7641
E-mail: <d.gorman@auckland.ac.nz>

Provisional report on diving-related fatalities in Australian waters 2004

Douglas Walker, John Lippmann, Chris Lawrence, John Houston and Andrew Fock

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

Abstract
(Walker D, Lippmann J, Lawrence CL, Houston J, Fock A. Provisional report on diving-related fatalities in Australian waters 2004. *Diving and Hyperbaric Medicine*. 2009;39(3):138-61.)
Introduction: An individual case review of diving-related deaths reported to have occurred in Australia in 2004 was conducted as part of the DAN Asia-Pacific dive fatality reporting project.
Method: The case studies were compiled using reports from witnesses, the police and coroners. In each case, the particular circumstances of the accident are provided, and also details from the post-mortem examination, where available.
Results: In total, there were 22 reported fatalities, all male. Nine deaths occurred while snorkelling and/or breath-hold diving, 10 while scuba diving, one just prior to scuba diving, one while using surface-supply breathing apparatus and one while diving with a rebreather. In this series, cardiac-related issues were thought to have contributed to the deaths of five snorkel divers and three scuba divers, and in one person who was about to go diving. Three of the deaths in breath-hold divers were likely to have been associated with apnoeic hypoxia blackout.
Conclusions: Pre-existing medical conditions, inexperience, time away from diving, inadequate supervision, and diving without appropriate training were features in several scuba deaths in this series.

Introduction

Diving is a potentially dangerous recreational activity. Tragically each year in Australia (and elsewhere) there are numerous fatalities associated with compressed-gas diving and snorkelling. Some of these accidents are unavoidable. However, many of these fatalities might have been avoided through better education, greater experience, appropriate medical screening, better equipment maintenance and design, or a modicum of common sense.

The aim of the DAN Dive Fatality Reporting Project (incorporating Project Stickybeak) is to educate divers and the diving industry and inform diving physicians on the causes of fatal dive accidents in the hope of reducing the incidence of similar accidents in the future. This report includes the diving-related fatalities between 1 January and 31 December 2004 that are recorded on the DAN Asia-Pacific (AP) database.

Methods

As part of its ongoing research into, and reporting of diving fatalities in Australia and elsewhere in the Asia-Pacific region, DAN AP has obtained ethics approval from the 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). A comprehensive search was made of NCIS to identify all diving-related cases that were reported to various State Coronial Services for the year 2004. The other major source interrogated was the DAN AP dive fatality database for scuba diving fatalities occurring in 2004. DAN AP staff routinely monitor a variety of internet sites and newspapers for diving and snorkelling fatalities. DAN sometimes also receives reports from the diving community when a fatality has occurred.

The process followed in the investigation and analysis of each case involved the following steps:

- Two of the research team (DW and JH) reviewed the police reports, witness statements and coronial reports and independently prepared a summary of each incident.
- Another researcher (JL) reviewed the two reports, investigated any discrepancies and prepared edited incident summaries.
- The incident summaries, coronial and autopsy reports were independently reviewed by three medical practitioners (DW, CL and AF), one of whom (CL) is a pathologist with extensive experience in diving autopsies.

Researchers at DAN America have recently applied the process of root cause analysis (RCA) to the investigation of diving fatalities with the aim of improving understanding of the sequence of events associated with such an accident.[1] Categories include trigger, disabling agent, disabling injury and cause of death. Sometimes the disabling injury can be more relevant to the assessment of a diving fatality than the cause of death. An example of this is a situation in which a diver suffers a cerebral arterial gas embolism (CAGE), becomes unconscious in the water and subsequently drowns. The cause of death, in this case drowning, may not provide as good an insight into the accident as the fact that the diver suffered from CAGE. We have applied this process to each case in this series.

Snorkelling and breath-hold diving fatalities

CASE BH 04/01

This victim, a 30-year-old male, was reported to have been in good health and was an experienced breath-hold diver and spear fisherman. He was spear fishing with two friends with whom he had dived for years. After a time diving near a headland, the group moved to an area off a small island where there was a reef. There was a strong current, estimated at 6 to 7 knots, and a calm sea. Visibility was described to be "*a good 10–15 metres if not more*". The dive plan was for one to remain in the boat and the other two to drift over the reef, one each side of the drifting boat. On the fourth drift, the friend in the boat noticed that the victim had not surfaced from a dive after 90 seconds; he became alarmed and alerted his other companion in the water. They pulled up the victim's float, which had his spear gun attached. It had been fired and there was a fish on the spear. The water was 15–20 metres' seawater (msw) deep and slightly murky but they thought they should be able to see the red handle of the missing man's knife if they looked down. There was no sign of the victim. He was known to have adjusted his weights in order to be negatively buoyant deeper than 10 msw.

An extensive search followed involving a police diving team who happened to be in the vicinity at the time. After two to three hours the body had not been found and the search was abandoned due to deteriorating weather. Two surfers found the victim's body floating on the surface four days later and some 50 km from where he was last seen. All of his snorkelling equipment was in place, including the weight belt with 4.2 kg of weights.

Autopsy: The body was not recovered for five days and there were severe decompositional changes. Only mild atherosclerotic changes were present in the coronary arteries (maximal 30% narrowing). Histology revealed a mild increase in interstitial fibrous tissue in the heart, of uncertain significance. He had injured his ribs the day before this dive but no chest wall injury was noted at the autopsy. The cause of death was given as drowning.
(Height = 189 cm; Weight = 71 kg; BMI = 19.9 $kg.m^{-2}$)

Comment: This is likely to have been a case of apnoeic hypoxia, with or without hyperventilation. Although it was reported that two whaler sharks were present while the group were spear fishing, and that on one occasion a two-metre shark came in very close as a fish was speared but showed no interest, the autopsy report included no mention of trauma to the victim's body. The reviewing pathologist would give the cause of death as drowning due to apnoeic hypoxia with or without hyperventilation with other significant factors including negative buoyancy.

Summary: Experienced breath-hold spear fisherman; breath-hold diving with friends to 15–20 msw; strong current; failed to surface; drowning (probably due to apnoeic hypoxia).

CASE BH 04/02

The 20-year-old male victim had multiple years of breath-hold diving and spear fishing experience, and was paired on the day of this accident with a buddy with whom he had dived over the previous two to three years. The two friends took a boat out to spear fish near to a wreck site about 28 km off the coast. The depth at this site was 20–22 msw and visibility around 15 metres, and no sharks were seen in the area. There was a mild current, which presented no problem. The victim wore a 'long-john-style' wetsuit and top with fitted hood, and used a weight belt with quick-release buckle. He wore fins, mask and snorkel and carried a spear gun with a length of rope attached to a float. The buddy later said that their usual practice was to dive one at a time "*to keep an eye out*". The victim did not appear to be having any difficulty with the dives and they managed to spear several fish.

The buddy dived to the wreck to look for an anchor that he had seen caught up there. The victim was on the surface where he appeared to be fine. When the buddy re-surfaced he saw the victim's float about 50 metres behind their boat. He waited for the victim to re-appear but when he did not do so, the buddy became alarmed. He made several unsuccessful attempts to search for the victim, both underwater and from the boat. He collected the victim's float and found the victim's speargun discharged at the end of the line. He dropped his boat anchor at this point as a reference, and released the float into the water in order to determine the direction of the current and hence the possible location of his friend. By then, he realised that the victim's chance of survival was very low. The buddy then undertook appropriate emergency call-out action and contacted authorities. A search was initiated, which lasted for three days and covered 1,140 square km but the body was never recovered.

Later checks with the family revealed that the victim had had asthma problems in the past but, although he carried and used an inhaler in earlier years, they were not aware that he had had to do so for at least a year prior to the fatal event. The family doctor had nothing on record of this, nor had he any report of the victim using an asthma inhaler.

Comment: There was no evidence of marine animal involvement, however, with no body for autopsy this cannot be excluded. This may well have been a case of apnoeic hypoxia. The victim had reported to his buddy on prior occasions that he sometimes felt dizzy after surfacing from some of his breath-hold dives. The buddy had specifically warned him not to stay on the bottom too long and to time his reserves to always be able to get back to the surface. The buddy also reported that he had never seen the victim hyperventilate prior to diving at any time.

Summary: Experienced breath-hold diver; apparently healthy; spear fishing with friend; failed to surface; no sharks sighted; body never recovered; probable drowning (possibly associated with apnoeic hypoxia).

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

ID BH	Age	Training	Experience	Dive group	Dive purpose
04/01	30	n/s	yes	BSB	Spear fishing
04/02	20	nil	yes	BSB	Spear fishing
04/03	33	trained	yes	Solo	Solo practice
04/04	61	n/s	some	BSB	Recreation
04/05	69	n/s	n/s	BSB	Recreation
04/06	60	nil	nil	BSB	Recreation
04/07	38	n/s	yes	GSB	Spear fishing
04/08	58	n/s	some	GNS	Recreation
04/09	47	nil	nil	BSB	Recreation

CASE BH 04/03

This healthy 33-year-old male overseas visitor was training to become a diving instructor. He was seen entering a swimming pool area at a residential complex and was wearing mask and snorkel. He had been swimming alone and was found unconscious on the bottom of the pool late in the evening, wearing his mask and snorkel and lying "*with his arms wrapped around a landscape rock*". Basic life support (BSL) was attempted but was unsuccessful, which is unsurprising as it was later estimated he had likely already been dead for four hours. It was reported that the victim regularly tested his breath-holding ability and had been observed practising this in the pool at his accommodation on two occasions in the preceding month. Suspicious circumstances were ruled out.

Autopsy: This confirmed he was otherwise healthy. No defensive markings were found. Condition of the skin indicated he had likely been in the water for several hours. The lungs weighed 746 g and 689 g. There was abundant pink frothy material in the trachea and main bronchi consistent with drowning. The heart weighed 368 g and was normal. No signs of drug use were noted. Toxicology results were negative for all tests performed. The cause of death was asphyxia (sic) due to drowning.
(Height = 177 cm; Weight = 76 kg; BMI = 24.3 $kg.m^{-2}$)

Comment: The reviewing pathologist would give the cause of death as drowning due to post-hyperventilation apnoeic hypoxia. Ironically, the victim had been present at a lecture two days earlier during which a diving safety officer had presented a talk on diving fatalities, stressing the point that in all the recreational diving work fatalities he had investigated there were two common factors. Firstly, that the person had disobeyed some of the basic rules of diving that instructors spend their time relying to others and, secondly, in each case the death occurred in a situation where the person was alone. The presentation had included the quote "*don't let the sole purpose of your life be to serve as a warning to others*".

Although in this case there was no history of syncope, the negative autopsy does not rule out the possibility of long QT syndrome as a cause of cardiac arrhythmia and loss of consciousness during breath-hold diving. This cannot be detected at autopsy and if there was any past history suggestive of syncope, the only way to exclude this possibility would be to screen the remaining family for long QT by electrocardiogram (ECG).

Summary: Trainee scuba instructor; alone in pool; likely practising prolonged breath-holding; drowning (possibly due to post-hyperventilation apnoeic hypoxia).

CASE BH 04/04

This 61-year-old male was described by his wife as a proficient swimmer who had snorkelled many times before. He and his wife, both visitors from overseas, went snorkelling on a coral reef in water about 3 msw deep. The water was described as clear and with a temperature of about 23°C. There was no apparent current and it was calm. On finding an area of abundant marine life, the victim called to his wife to come over to have a look. She was swimming towards him and was about 10 metres away when she saw him suddenly go limp, his head slumping to

in Australian water in 2004; all cases were male
separated before; n/a – not applicable; n/i – not inflated; n/s – not stated

Depth (msw)	Incident (msw)	Wt belt	Wts (kg)	BCD	Cause of death
15	15	on	4.2	nil	Drowning, ?hypoxic blackout
21	n/s	n/s	n/s	n/s	Drowning, ?hypoxic blackout
n/s	n/s	nil	n/a	n/i	Drowning, ?hypoxic blackout
2.5	surface	nil	n/a	nil	Cardiac
n/s	surface	nil	n/a	nil	Cardiac, drowning
n/s	surface	nil	n/a	nil	Cardiac
18	n/s	on	n/s	nil	Trauma, shark
n/s	surface	nil	n/a	nil	Cardiac
n/s	surface	nil	n/a	nil	Cardiac

one side and his snorkel becoming submerged. She found him to be unconscious and so she took him to shore. BLS was commenced immediately on shore, with assistance from other beach users, and was pursued vigorously for 45 minutes without success.

Autopsy: The heart weighed 438 g and there was multifocal atheroma, which varied from 70% to 30% narrowing along the major coronary arteries. The lungs weighed 658 g and 588 g and were hyperinflated, but the upper airways were clear. There was no evidence of marine animal activity or bites. The cause of death was given as coronary atherosclerosis.
(Height = 180 cm; Weight = 88 kg; BMI = 27.2 kg.m^{-2})

Comment: The victim was a competent swimmer with no previous medical history, and no evidence of bites or stings. This death from ischaemic heart disease while snorkelling could have occurred during any form of exertion.

Summary: No history of health problems; silent death occurring in the vicinity of others while snorkelling; vigorous but unsuccessful BLS attempts; coronary atherosclerosis.

CASE BH 04/05

This 69-year-old male, who was a visitor from overseas, had a history of hypertension, for which he was taking amlodipine besylate, and angina, for which he was prescribed nitroglycerine. Despite this, he showed no outward signs of being unfit and was reported to have been a competent swimmer. It is unknown if he had any snorkelling experience. He and his wife joined a trip to see the Great Barrier Reef (GBR). There was a talk on snorkelling during the outward trip and passengers were asked to report any health or medication factors that might affect their safety. There is no record of him reporting his medical condition.

They were snorkelling in the roped area around the pontoon moored at a reef. He was wearing a thin wetsuit and the snorkelling equipment provided. Three lookouts watched those in the water in the roped area, ready to help anyone requiring assistance. The victim and his wife were separated at times due to choppy sea conditions. A short period of separation occurred and when next seen, the victim was floating face-down against the boundary rope and not moving. A rescue swimmer and a tender were immediately dispatched to him and he was dragged into the tender. He was apnoeic and pulseless. He was taken to the pontoon where BLS was commenced, without success.

Autopsy: The heart weighed 480 g. There was left ventricular hypertrophy (18 mm thickness, normal <15 mm). The only significant coronary atheroma was in the left anterior descending (LAD) coronary artery, whose lumen was reduced to approximately 50% by a plaque 10 mm from its origin. The heart muscle histology showed myocyte hypertrophy and some interstitial fibrosis in some areas but no myocarditis. The lungs were heavy, weighing 1220 g and 1053 g. There was a large amount of frothy fluid in the larynx and bronchi and the lungs were extremely moist with watery fluid suggestive of salt water drowning. The cause of death was given as drowning, other significant conditions were coronary artery stenosis due to atherosclerosis.
(Height = 187 cm; weight = 87.5 kg; BMI = 25.0 kg.m^{-2})

Comment: The reviewing pathologist would give the cause of death as drowning due to ischaemic heart disease (50% LAD) and left ventricular hypertrophy while snorkelling. At autopsy, there are no pathognomonic findings of drowning. The diagnosis is based on the circumstances of the death, plus a variety of non-specific anatomical findings like pulmonary oedema, which may also be caused by cardiac failure. As in cases BH04/04 and BH04/08, the degree of coronary artery stenosis reported was 50%, below the 75% stenosis usually held to be clinically significant. The left ventricular hypertrophy could be the significant other factor. The fact that he was prescribed nitroglycerine is a strong indicator of clinical ischaemic heart disease. The occurrence of a cardiac event was possibly high at any time, but of particular importance in the water especially in choppy sea conditions and/or a current.

Summary: History of angina; appeared to be otherwise 'fit'; unpredictable event occurring whilst, but not necessarily as a consequence of, snorkelling; silent death in presence of others; drowning (possibly due to cardiac arrhythmia).

CASE BH 04/06

The victim was a 60-year-old male, an overseas tourist who had come with his family to holiday on the GBR. He had a history of Parkinson's disease for which he was taking a variety of medications, including levodopa, selegiline, tolcapene, benserazide hydrochloride and adamantine.

The usual introductory information and films were shown on the outward boat trip to a pontoon on the reef. The victim omitted declaring his health condition and medications on the pre-snorkel medical questionnaire. Although he was an active swimmer, it is not known if he had snorkelled before and the family was apparently surprised by his decision to do so. He began snorkelling with his son who then exited the water briefly to have his mask adjusted. At this time, the lookout noticed that the victim was about 4 to 5 metres away and appeared to be struggling to reach out to the outer yellow float line. Another swimmer was seen to lift the line to allow him to swim under it. After appearing to regain his composure, the victim was thought to have continued to snorkel, although there were no visible signs of him kicking. He was then noted by a ship's lookout to be face-down and not moving. The lookout immediately dived in and swam to assist the victim who was now approximately 30 metres away. The victim was found to be unresponsive, with staring eyes, and did not appear to be breathing. The victim was brought to the pontoon where BLS was commenced. A doctor, present by coincidence, assisted. Despite advanced life support (ALS), the victim failed to respond.

Autopsy: Autopsy revealed the presence of a small skull defect about 20 mm across in the right mid lateral portion of the skull and a small area of compression of the anterolateral right hemisphere. Both were long-standing and indicated a past surgical procedure. The heart weighed 408 g and there was no left ventricular hypertrophy. There was no macroscopic scarring and histology revealed only small patches of predominantly subendocardial fibrosis. The coronary arteries showed 80-85% stenosis of LAD coronary artery and 50% stenosis of the other two major coronary arteries. The lungs were heavy, weighing 855 g and 689 g and exuded haemorrhagic fluid. The cause of death was stated to be "*acute cardiac failure due to atherosclerotic stenoses of the coronary arteries due to atherosclerosis*".
(Height = 183 cm; Weight = 78 kg; BMI = 23.3 $kg.m^{-2}$)

Comment: Although the victim was taking several medications to manage his Parkinson's disease, he was not apparently taking any cardiac medication. There is no information concerning the reason for the skull surgery or whether he had previously reported any cardiac symptoms. Why he became determined to leave the designated snorkelling area is unknown. This cardiac death was not necessarily related to his snorkelling and could have likely occurred in other circumstances.

Acute cardiac failure is a 'mode' of death and not an acceptable 'cause' of death according to a report from the Australian Bureau of Statistics,[2] although it is used on occasions in coroner's findings. The reviewing pathologist would give the cause of death as ischaemic heart disease due to severe narrowing of the LAD coronary artery while snorkelling. Another significant condition contributing to death may have been Parkinson's disease as the movement problems caused by this disease would probably significantly impair the victim's ability to save himself.

Summary: Overseas visitor on multiple medications for Parkinsonism; active swimmer with no known history of cardiac symptoms; unknown reason for skull surgery long ago; separation; silent death; acute cardiac failure.

CASE BH 04/07

This 38-year-old male had 15 years of spear fishing experience and was thought to be generally of good health, although he was taking medications for high cholesterol (simvastatin) and depression (paroxetine hydrochloride). He was spear fishing with two friends on the outer GBR and continued to spear fish when his two friends returned to the boat for lunch. One of the friends looked up and noticed the victim vertically upright in the water, then heard him scream. He had been snorkelling about 60 metres from the bow of the boat. No shark was sighted at any stage of the event.

Realizing that something was wrong, the friends quickly weighed anchor and went directly to him. He was surrounded by a pool of blood about 3 metres in diameter and his spear gun floated nearby, although they did not notice whether it had been fired. They quickly pulled him into the boat. The victim had a large bite wound down to the bone on his inner left thigh and involving his femoral artery. He was still conscious when pulled aboard but was extremely pale.

The friends tried to pack the wound to stop the bleeding and talked to keep him calm and prevent him from trying to get up. Emergency services were contacted by mobile phone when efforts on marine radio failed. Whilst returning to land, the victim slipped in and out of consciousness. About 10 minutes before an emergency helicopter arrived, his pulse could no longer be felt, although his eyes were open. His death was confirmed by the paramedics. It was reported that the victim had a 'bait pouch' attached to his weight belt.

Autopsy: No teeth were found in the wound but examination of the incision marks on the victim and on his wetsuit indicated that he was attacked either by a bull shark or a black whaler. Experts at James Cook University suggested its probable size to have been 1.8 to 2.2 metres. The wound showed no tearing so it was thought to be a single bite.
(Height = 174 cm; weight = 70 kg; BMI = 23.1 kg.m-2)

Comment: The presence of blood from fish that the divers had speared, and the 'bait pouch' attached to the victim's belt are likely to have encouraged the shark to attack. The manner in which the friends attempted to stop the bleeding is not clear so it is unknown whether or not more effective first aid would have made any difference to the outcome. The reviewing pathologist would give the cause of death as shark bite of left thigh while spear fishing.

Summary: Experienced spear fisherman; bait pouch on weightbelt; location far from shore hence distant from medical care; shark attack; exsanguination.

CASE BH 04/08

This victim was a 58-year-old male, with unknown previous snorkelling experience. He had recently arrived from overseas and was on a three-day cruise on the GBR with his daughter, who was a scuba diver. He was obese, with a BMI of 34.6 $kg.m^{-2}$, and had a history of hypertension for which he was taking atenolol 25 mg, enalapril maleate 10 mg, calcium aspirin 100 mg, hydrochlorthiazide 25 mg and isoride dinitrate 5 mg. It was reported that the victim had visited his doctor in the weeks preceding his journey "to have his blood pressure checked" and was assured that he was fit to undertake his forthcoming trip.

Prior to being permitted to snorkel, the victim was required to complete a medical declaration, which he chose to do in his native language, but in a manner that was comprehensible to the dive operator. The waiver he had signed contained warnings about the risks of snorkelling for people with certain health conditions, such as heart disease. It is unknown whether or not he reported his health conditions.

On the first day of the trip, he remained on board the vessel, appeared well and ate normally. Mid-morning on the second day, the victim prepared to go snorkelling with an organised group. He hired mask, snorkel and fins, donned a wetsuit top but refused an extra buoyancy aid that he was offered. After boarding the tender, he was taken approximately 30 metres from the vessel. The conditions were described as suitable to allow the victim and others to snorkel in the area and to return to the main vessel "with the aid of the wind and the surface chop". The victim requested to be taken back to the main vessel after only a few minutes in the water, stating that he felt tired. He was taken back promptly and after several minutes on board, he became breathless, vomited and collapsed. The crew immediately initiated BLS, which continued until the arrival of a rescue helicopter. Paramedics implemented ALS procedures but the victim failed to respond.

Autopsy: The autopsy revealed no injuries. The heart weighed 475 g and was mildly enlarged. The left ventricle was reported to measure between 20 and 28 mm in thickness (normal < 15 mm), There was an area of 50% narrowing in the right coronary artery. The more distally placed coronary vessels showed widespread areas of luminal narrowing but no signs of complete occlusion. Histology of the heart showed advanced ischaemic pattern fibrosis and severe atheromatous narrowing of the smaller coronary arteries. The weight of the lungs is not given and there is no comment about the presence or absence of pulmonary oedema fluid in the upper airways, making it difficult to assess whether drowning may have occurred. Histology also showed renal arterial and arteriolar nephrosclerosis, an indicator of background raised blood pressure. The cause of death was given as acute cardiac failure, coronary arterial atherosclerotic narrowing, cardiomegaly and myocardial fibrosis.
(Height = 183 cm; Weight = 116 kg; BMI = 34.6 $kg.m^{-2}$)

Comment: Fatalities occurring during exercise are observed with narrowing of between 50% and 75%, especially when there is severe atheroma in the smaller coronary arteries or left ventricular myocardial hypertrophy. The histology is very suggestive of past ischaemic fibrosis. Where there is hypertrophy of the left ventricle greater than 15 mm, there may be some problems with diastolic perfusion. In this context, it seems reasonable that a subcritical stenosis with left ventricular hypertrophy during exercise might cause myocardial ischaemia and trigger a lethal arrhythmia.

As with BH 04/06, cardiac failure is a mode, not a cause of death, which is more correctly described as due to ischaemic heart disease and left ventricular hypertrophy while snorkelling. Another significant condition contributing to death may have been hypertension. This death could have occurred in a variety of other circumstances and the victim was either unaware of the degree of his ill health, or concealed its presence.

Summary: Obese; hypertension on medication; pre-trip medical check found victim fit to travel but unknown if snorkelling was discussed with doctor; became breathless shortly after entering the water to snorkel; collapse soon after exiting water; ischaemic heart disease (death could have occurred at any time, especially with exertion).

CASE BH 04/09

The victim was a 47-year-old, apparently healthy, male who was visiting Australia with his brother and was on a day trip to the GBR. During the cruise out on the vessel, they attended the safety talks and the victim decided to undertake a 'resort dive'. He completed a medical questionnaire, reporting no ill health. He said to his brother that his doctor had told him the previous year that he was fine.

While awaiting the appointed dive, the two men joined others snorkelling. The current was reported to be fairly strong. Seven crewmen were tasked to look out for any people in distress or fatigued. Four were on the observation deck and three were in boats either inside, or close to, the marked area. The two brothers snorkelled together, the victim taking pictures. After talking about the strength of the tide and "*stinging going on in the seawater*" they took pictures of each other. There were no signs that the victim was suffering any difficulty. However, he said that he was a little tired and he would return to the pontoon to rest before his scuba dive. At this stage, they were 15–20 m from the boat. The brother then made several dives to photograph giant clams but checked and saw the victim swimming towards the pontoon each time he surfaced. Nothing seemed out of the ordinary.

Later, on returning to the pontoon, he was unable to locate his brother. One of the lookouts noticed the victim floating face-down and immobile. When a rescuer reached him, he was found to be unconscious, apnoeic and cyanotic. He was dragged into the boat where BLS was commenced. This continued for a further hour on the pontoon before the attempt was abandoned. It was estimated that about 30 minutes had elapsed from the time he entered the water until he was found.

Autopsy: There was significant ischaemic heart disease with a pinpoint narrowing of the ostium of the right coronary artery by atheroma and the left coronary and the left circumflex arteries with 50% stenoses. Histology showed similar narrowing of some small vessels but no evidence of myocarditis, significant fibrosis, or of recent infarction. Cause of death was given as myocardial ischaemia as a consequence of coronary artery stenosis due to atherosclerosis. No suspicious circumstances were noted. (Height: 170 cm; weight: 88 kg; BMI = 30.4 $kg.m^{-2}$)

Comment: While this death occurred during immersion, it was clearly an event that could have happened at any time. It is likely that the exertion of snorkelling was significant in this death due to ischaemic heart disease, as during vigorous exercise the risk of sudden death is 56 times greater than at rest in sedentary individuals and 5–25 times greater in fit individuals based on the underlying level of fitness.[3]

Summary: Apparently healthy; snorkelling in strong current; reported fatigue; silent death despite several lookouts; myocardial ischaemia.

Scuba diving deaths

CASE SC 04/01

This victim was a 44-year-old male, who was working on a luxury yacht that was visiting the GBR from overseas. He was said to be a qualified and competent diver although the level of his training was not reported. The yacht's anchor became stuck in water at a depth of 23 msw and the victim and his buddy were sent to try to free it. The divers did an initial survey dive to assess the situation and to formulate a plan. After waiting on the surface for equipment to be lowered to them, they dived again about 15 minutes later. This time they went down with a heavy line and shackle to attach to the anchor chain, which they did.

About one hour later, after having refilled their air cylinders using the ship's compressor, they dived again to free the anchor. The victim injured his right hand but it is unclear exactly how this occurred. However, the injury resulted in the victim making an immediate solo rapid ascent without warning. Observers on the vessel noted that he arrived at the surface looking unwell, although he was initially conscious. He seemed to try to speak and then began to vomit. At this point, two of the crew dived in and assisted him back to the vessel. Once on board, he was apnoeic and pulseless. BLS was immediately commenced. An emergency services helicopter arrived within the next half an hour and ALS procedures were implemented. Resuscitation attempts were halted approximately one hour after the victim was removed from the water. He was pronounced dead shortly thereafter at a mainland hospital.

Another witness, apart from the buddy, who was an experienced diver had observed the dive plan and watched the equipment prepared and had seen no fault in any of this. The victim had seemed at ease with his task as he had appeared to be during dives on previous occasions.

Autopsy: Autopsy revealed subcutaneous emphysema of the upper chest and significant amount of gas within the right side of the heart and adjacent major vessels, and the vessels of the circle of Willis together with hyper-inflated lungs strongly suggested massive air embolism. Radiological imaging showed changes in the vessels of the brain, neck, thorax and pelvis. Toxicology showed no other findings. Cause of death was massive gas (air) embolism due to diving.
(Height = 185 cm; Weight = 88 kg; BMI = 25.7 $kg.m^{-2}$)

Comment: This is a classic description of an incident of pulmonary barotrauma/cerebral arterial gas embolism (PBT/CAGE) following rapid ascent, in this case following injury while scuba diving, and sudden loss of consciousness upon reaching the surface. Although the victim was reported to have been a competent diver, it appears that he panicked when injured and is likely to have ascended rapidly without breathing adequately. The extent of the injury to his hand

or arm is unclear but it was a trigger for an unsafe ascent. Although the victim and his buddy performed three 23 msw dives in a short period of time, decompression sickness was not believed to have been a factor in his demise.

Summary: Buddy pair attempting to free a jammed ship's anchor; victim cut hand/arm; probable panic; rapid ascent; BLS and ALS unsuccessful; CAGE.

CASE SC 04/02

The victim was an apparently healthy 41-year-old male tourist from overseas. He registered for a 'resort' dive with a group visiting the GBR. He completed the required medical declaration, stating that he had no known medical conditions prohibiting him from undertaking a scuba dive. There was a talk by an instructor covering the main matters the group of four resort divers needed to know before their first scuba dive. After the instructor checked their equipment, they entered the water and assembled at a bar in the water near the stern of the boat where they practised equalizing, mask clearing, and regulator recovery. Each individual demonstrated these skills before descending to a lower bar to be photographed. Sea conditions were described as moderate with a slight current and visibility of around 15 metres.

They then linked arms and swam over towards the reef whilst slowly descending to the bottom. After about 30 minutes, one diver's air contents gauge was indicating 60 bar so it was time for him to ascend. Another pupil indicated a desire to surface also. The instructor thought the dive was rather short so offered the other two the option of continuing. To his surprise the victim decided to ascend at this time, though he had seemed to be managing well, so the instructor escorted three divers to the surface while the fourth diver remained on the bottom. The depth of the water at this time was variously described as 3–8 msw.

At the surface, the victim and one of the others mentioned having experienced some trouble breathing from their regulators. The instructor ascribed this to their being nervous and to the effort of swimming, believing this to be quite common among 'resort dive' pupils. After noting that the lookout on the boat, some 20–40 meters away, had seen the group on the surface, and that the wind and tide would assist their return, the instructor asked the three whether they were able to return together on the surface to the vessel before re-descending to the fourth student. The victim and his companions then commenced their return swim. A short time later, the victim was located on the surface by another crew member. He was unconscious and cyanotic, with neither snorkel nor regulator in his mouth. Although his weightbelt was in place, he was quite buoyant as his buoyancy compensating device (BCD) was inflated. The victim was brought back aboard to the boat where BLS was commenced. A doctor and paramedic who were on a nearby vessel came over to assist and took over the resuscitation efforts. Oxygen and an automated external defibrillator (AED) were available but it is unknown whether or not these were used. Resuscitation efforts were continued for approximately 35 minutes but the victim failed to respond.

Autopsy: The heart weighed 381 g and showed mild left ventricular hypertrophy. There was severe ("pinpoint") atheromatous stenosis of the origin of the left circumflex coronary artery and moderate stenosis of the ostium of the right coronary artery. Ischaemic changes were seen in the myocardial fibres but no acute infarction. There was diffuse fatty change in the liver. The cause of death was ischaemic heart disease.
(Height: 177 cm; Weight: 79 kg; BMI = 25.2 $kg.m^{-2}$)

Comment: This death resulted from pre-existing ischaemic heart disease due to severe atherosclerosis and could have occurred in many circumstances, unrelated to diving, although the exertion was probably significant. The abandonment by the instructor on the surface breached recognised training protocols.

Summary: Apparently healthy man; 'resort dive'; abandoned on surface by instructor; silent death at surface; significant but apparently unknown coronary artery disease; ischaemic heart disease.

CASE SC 04/03

The victim was a 31-year-old male who was uncertified and inexperienced, having only completed around six to eight dives in total. He had learned what he knew about diving from a friend who had reportedly been diving more than 20 years and claimed to have completed between 200 and 300 dives. The victim was described as safety-conscious and competent and, although it had been suggested to him by others with whom he had dived previously (including the person with whom he had 'trained'), that he should do a formal diving course, he had not done so. The pair went diving for crayfish from a private vessel. Another friend, who had no knowledge of diving, tendered the boat whilst the two dived. He had been told to look out for them when they resurfaced. It was reported that the victim's buddy, the diver who had 'trained him', had set up and checked the victim's gear, which included air cylinders charged to 270 bar.

There was a delayed start to the dive as the victim was initially under-weighted and an additional 1.3 kg weight was placed in each pocket of his BCD. The sea was calm and visibility around 15 metres. They descended and followed the bottom out to a depth of about 15 msw, staying together for about 30 minutes. They then became separated but soon reunited when the victim's buddy found him with his head in a hole trying to extract a crayfish. The buddy swam off about 25 metres and when he returned there was no sign of the victim and he presumed that his friend had swum off in search of other crayfish and so was not concerned. About 10 minutes later, he heard a boat's motor and believed that

Table 2. Summary of scuba, rebreather and surface-supply diving-related
BNS – buddy not separated; BSB – buddy separated before problem; BSD – buddy separated during; GSB – group separated before; GSD – group separated during; expcd – experienced; tnd = trained;

ID SC	Age	Training	Experience	Dive group	Dive purpose	Depth (msw)
04/01	44	trained	expcd	BSB	Work	23
04/02	41	nil	nil	GSB	Resort	8
04/03	31	some	slight	BSB	Crayfish	15
C04/04	32	trained	some	BSB	Recreation	3
04/05	26	trained	some	GSB	Recreation	13
04/06	53	trained	some	GSD	Recreation	30
04/07	55	trained	nil	BNS	Recreation	18
04/08	37	some	nil	GSB	Class	3.3
04/09	36	some	nil	GSB	Class	10.5
04/10	32	trained	some	GSB	Recreation	12
RB						
04/01	55	trained	expcd	GSB	Recreation	46
SSBA						
04/01	24	some	some	GSB	Crayfish	30
Pre-SC						
04/01	65	n/s	expcd	BNS	Recreation	n/s

this indicated that the victim had surfaced and was being picked up. Noticing that he had 70 bar of air remaining, the buddy began a slow return to shallower waters but after about 5 to 10 minutes he saw the victim lying face-down on the bottom at a depth of 12 msw. He was unconscious with regulator out of his mouth and mask missing. After being unable to inflate the victim's BCD as there was no remaining air supply, the buddy inflated his own BCD and brought the victim to the surface. The buddy then commenced in-water rescue breathing while he waited for the boat to pick them up. Rescue breathing was continued until they reached shore where they were met by an ambulance and a local doctor. BLS attempts were unsuccessful.

The equipment was later found to be in good working order apart from the lack of air in the tank. The dive computer indicated that the ascent alarm had been triggered; however, the dive profile could not be downloaded.

Autopsy: The pathologist did not obtain a CT check prior to autopsy but did inflate the lungs underwater (no air escaped), noting the absence of subpleural emphysema, then aspirating the ventricles. He obtained 20 ml of gas from the right ventricle and 60–80 ml from the left. Gas was found in the inferior vena cava, the aorta, and the left and right atria (with less in the right). There was mediastinal emphysema. The coronary arteries showed minimal atherosclerosis, and the foramen ovale was probe patent. Cause of death was given as pulmonary barotrauma/cerebral arterial gas embolism due to an out-of-air situation while scuba diving.
(Height: 172 cm; Weight: 77.8 kg; BMI = 26.3 $kg.m^{-2}$)

Comment: Given that the victim was discovered dead on the bottom, it is possible that the gas detected at autopsy represents post-mortem off-gassing. Unfortunately, the ascent was not witnessed, hence one of the best diagnostic criteria for PBT/CAGE was absent.

fatalities in Australian waters in 2004; all cases were male
problem; n/a – not applicable; n/i – not inflated; nad – nothing abnormal discovered; n/s – not stated;
+ sufficient air (to surface safely); ++ 1/4–1/2 full tank; +++ >50% full; CAGE – cerebral arterial gas embolism

Incident (msw)	Wt belt	Wts (kg)	BCD	Remaining air	Equip test	Cause of death
23	n/s	n/s	n/s	+++	nad	CAGE
surface	on	n/s	infl	++	n/s	Cardiac
12	on	7	n/i	nil	nad	CAGE
3	on	10	infl	+++	nad	Cardiac?
ascent	on	n/s	n/i	+++	fault	CAGE
5	on	11	n/i	nil	nad	Drowning
surface	on	n/s	n/i	++	nad	Cardiac, post dive
3	on	11.2	n/i	nil	faults	Drowning
ascent	on	n/s	n/i	+++	faults	CAGE
surface	on	18	n/i	+++	faults	Drowning
46	on	3	nil	ok	faults	Drowning
n/s	on	2	nil	++	faults	Drowning
surface	n/a	n/a	n/a	n/a	nad	Cardiac, pre dive

Summary: No formal training; some instruction; inexperienced; hunting crayfish; separated; out-of-air situation with probable rapid ascent; PBT and CAGE.

CASE SC 04/04

This 32-year-old male was obese and had a history of HLA B27 spondyloarthritis. He was a trained open water diver but had "not dived for quite some time" and was keen to get back into diving. The dive was planned with a group of work colleagues, one of whom was to be the victim's dive buddy as they had dived together previously. Prior to the outing, the victim hired a tank and weight belt.

The chosen location required a 50-metre walk down a steep path, which the victim appeared to manage without any problems. The group rested for 10 minutes, then checked each other's equipment before entering the water. The entry point was off a rock ledge requiring judgment of the optimal point of the surge before jumping in. Conditions were choppy, visibility was described as 10–15 metres and there was an incoming tide that would tend to carry them towards the planned exit point.

The first group of three divers entered the water from the ledge and swam off. The victim was the next to enter the water, just before his buddy. He descended two to three metres, then ascended again very shortly after, reaching the surface before his buddy had submerged. He removed his regulator and said "*I'm not feeling good*" and that he "*wanted to go in*". By this time they were about 15 metres from the rock shelf. When they reached this shelf the victim found he could not climb out as it was too steep and waves were knocking him onto the rocks. His buddy thought he was beginning to panic and saw him pulling at his exposure suit as if it was too tight. As they swam to find an easier place

to exit, the victim pulled his mask off. His buddy urged him to continue to use his regulator and saw that there was some air in his BCD. The buddy later said it would not have been possible to exit onto their water entry ledge as it was too high.

The buddy swam with the victim, holding his tank and pulling him along on his back. He encouraged him to relax and to keep breathing from his regulator. When the regulator fell out of the victim's mouth, the buddy replaced it, then removed his mask and snorkel from his hand. On reaching the lower ledge the victim and his buddy tried in vain to exit the water. This required significant exertion for both divers and the victim began to panic when he was unsuccessful. The buddy then realized he needed to release his friend's weights before he would be able to pull him out of the water, which he did. At this time the victim seemed to be relaxed, floating with his head out of the water. He then developed a blank look with his eyes open and distant, and he spat out the regulator. The buddy kept the victim's head above the water and called for help, which soon arrived. He attempted to pull the victim from the water but this was only achieved when more helpers arrived and four were needed for this task as the victim was so heavy.

The buddy placed the victim on his side and "*about a cup-full of water and yellow matter came out of his mouth*". He was then rolled face-up and BLS was commenced. Another diver soon arrived and together they maintained BLS until ambulance paramedics arrived. Shortly after this, a rescue helicopter arrived carrying a doctor who, together with the paramedics, implemented ALS. However, the victim failed to respond.

It was only later, at work, that the buddy learned that his friend had been unwell with arthritis the previous week. They had dived together three times in the past, the last occasion three years prior. The buddy thought that the victim had put on a lot of weight since last wearing his semi-dry suit. In his opinion, it had been too tight. He later suggested that the victim should probably not have been diving that day. Later examination of the victim's diving equipment found it functioned correctly and the air was suitable for use.

Autopsy: The autopsy revealed no evidence of subcutaneous emphysema, pneumothorax, or serious coronary disease and a 'cause undetermined' finding was recorded. Although there was 2 cc of small air bubbles in the right atrium, ventricle, and pulmonary arteries, this was ascribed to post-mortem changes – the heart was opened underwater. Toxicology tests show that he had used cannabis in the recent past and had taken rofecoxib and pseudoephedrine. There was 3% carbon monoxide saturation of the blood, which was consistent for that of a smoker. There was no evidence of trauma beyond minor grazes probably sustained when he was pulled from the water. The maximum coronary lumen stenosis was 10%. Histology showed delicate focal scarring in the anterior wall of the left ventricle but there were no changes in other locations. The liver showed mild macrovesicular steatosis. No comment was made concerning arthritis or his obesity. The cause of death was not determined at autopsy.

Subsequent review of the case by a physician and a pathologist with diving medicine experience suggested that cardiac arrhythmia was the most likely cause of death. Features of concern were the presence of pseudoephedrine on toxicology, the HLA B27 spondyloarthritis with a possible hypercoagulable state associated with the arthritis just before death, minimal recent diving experience, recent weight gain and a tight wetsuit. Screening of the family for long QT was recommended even though ECGs of the diver had not demonstrated a long QT interval. The cause of death was given as cardiac arrhythmia.
(Height = 174 cm; Weight = 105.5 kg; BMI = 34.8 $kg.m^{-2}$)

Comment: At the time of this case, there was publicity about the potential cardiac risk for those taking rofecoxib and the victim had been taking this, in addition to other medications, for his chronic arthritic disease. His family brought this to the Coroner's notice and in consequence his medical records were reviewed. These showed a long history of arthritis, diagnosed in 1995 as Reiter's Syndrome. He was intermittently on various non-steroidal anti-inflammatories, salazopyrin, prednisolone, and atropine eye drops for episodes of uveitis. Opinions were obtained from a cardiologist and a pathologist with a special diving-medicine interest. They noted the medications, his obesity and lack of fitness, the structural changes in the heart, the strenuous activity in reaching the water's edge and the tight-fitting semidry suit. It was noted that physical evidence of a myocardial infarction takes time (at least four hours) to develop. Various suggestions, such as coronary thrombosis due to platelet aggregation associated with the spondyloarthritis, a long QT Type 1 or a 'small-vessel disease' syndrome, were made, and it was suggested the family be investigated for some of these conditions. Both specialists agreed this was probably a cardiac arrhythmia death. This case demonstrates the difficulty of diagnosis of a functional abnormality, for example cardiac arrhythmia, at autopsy when there is no structural abnormality.

Summary: Trained but no recent experience; obese with long history of medication, including rofecoxib for spondyloarthritis; tight semi-dry suit; difficult access to dive site; reported feeling unwell immediately on entering water; buddy rescue attempt; probable cardiac arrhythmia.

CASE SC 04/05

This 26-year-old male came from overseas on a diving holiday with three friends. He had obtained his open water diving certification at home some months earlier and, although described as an enthusiastic diver, he was thought to have done only six dives prior to this trip, the deepest being to 18 msw.

The trip was for three days on a live-aboard vessel in tropical

GBR waters. The victim aborted a dive on the first day as he was grossly under-weighted and had exhausted himself trying to stay underwater. He reported feeling tired and seasick. On the second day, he decided not to join the first dive as it was too early in the morning, but dived later that morning with two friends and a dive guide.

The vessel was anchored in water 10 to 12 msw deep. Conditions were described as "good", with visibility of around 20 metres, no swell and only a small surface chop. Underwater, the divers swam into a slight current. After about five minutes, one of his friends saw the victim standing on his fins on the sandy bottom and wrote on his slate telling him not to kick up the sand. The victim made some sort of a signal in response, possibly a hurried 'OK' before swimming, his kicking appearing to be weaker than expected. It was a further few minutes before his absence was noticed and the dive leader, who was a divemaster-in-training, started to ascend to look for the missing diver and noticed the victim on or near the surface above him. He was face-up and sinking from the surface, the regulator was not in his mouth and his mask and snorkel were missing. The leader grabbed the victim by his BCD, placed the regulator back into his mouth, inflated his own BCD and they both ascended to the surface from a depth of 4–5 msw. The leader described this as a struggle as the victim was heavier than him. On reaching the surface, he removed the victim's weight belt, enabling him to remain buoyant.

Meanwhile, the look-out on the boat had seen the victim surface briefly and then disappear. The rescuer in the water called and signaled for help then checked for, and found no, signs of life so he began in-water rescue breathing while the tender came to their aid. The victim was brought aboard the tender and quickly taken to the main vessel where BLS was commenced. Supplemental oxygen was provided (15 litres per minute via a resuscitation mask). Initially a lot of water was drained from the victim's mouth. Ventilation was complicated by the fact that the victim had clenched teeth as well as continuous frothing and regurgitation of water and stomach contents. Bloody fluid was noted coming from his ears. A doctor and nurse (tourists themselves) arrived from another vessel and continued with the BLS. After a total of some 90 minutes the doctor ceased resuscitation attempts.

The victim's dive computer indicated a maximum depth of 13.8 msw and a dive time of 12 minutes. There was 110 bar of air remaining in the cylinder. The rented equipment was reasonably serviceable except for the contents gauge which was inoperable, stuck at a constant reading of 90-100 bar. The cylinder air was found to be "suitable for use" when tested.

Autopsy: The autopsy was performed two days after death. The CT of the head and neck revealed gas in both internal carotid and vertebral arteries as well as within the basilar artery and within branches of those vessels. Extra-axial air was seen anteriorly in each middle cranial fossa. The hard palate was high-arched and the left side canted up laterally. The findings suggested repair or part-repair of a cleft palate. The CT of the trunk showed intra-cardiac gas and gas within the origins of the great vessels of the aortic arch and a small amount in the mid-descending thoracic aorta. An air-fluid level was seen in the distal trachea and within the right and left main bronchi. There was no pneumothorax. In the abdomen, there was also gas in the aorta and major arterial branches and in the spleen and liver, neither of which was enlarged.

The heart weighed 330 g, the coronary arteries were small in calibre and showing no significant atheroma. There was gas in the ventricles, especially the left ventricle, and in the arch of the aorta. There was a comment that histology of the heart revealed "chronic myocarditis", although the extent, and hence, the possible significance of this change is not clear. The lungs weighed 700 g and 600 g and had the appearance and consistency of "e*mphysema aqueosum*", typically associated with drowning. The liver had a number of pale areas, chiefly subcapsular, suggestive of impending necrosis due to ischaemia (likely due to obstruction of peripheral blood supply from blockage by bubbles). The cause of death was given as cerebral and generalized gas embolism with drowning due to probable uncontrolled ascent during a scuba diving accident.
(Height = 185 cm; Weight = not recorded; BMI = unknown)

Comment: The cause of death as PBT/CAGE was probably due to a rapid ascent. Other significant factors may have been feeling unwell prior to the dive and a faulty contents gauge giving misleading information. Because this autopsy was completed two days after death, it is possible that some of the gas seen could be due to post-mortem off-gassing or decomposition. The descriptions of the liver and spleen are suggestive of either off-gassing or decomposition. There are some features that do not completely fit the diagnosis of CAGE: (1) the diver may have been unwell prior to ascent, (2) there are changes consistent with drowning, and (3) there was microscopic chronic myocarditis. One of the other passengers noted afterwards that he saw the victim before he entered the water and "*he didn't seem relaxed. I think it might be best to call him tense*". His actions as described by his buddy cannot be readily explained; he had adequate air even should he have believed the contents gauge rather than the ease of his air supply. Panic appears to have overwhelmed him, possibly in consequence of anxiety concerning his diving ability and exacerbated by his residual ill health.

Summary: Prior sea sickness and fever symptoms; trained but very inexperienced; appeared anxious before dive; strange behaviour, then separation and solo ascent; seen to sink from surface; weights not ditched; adequate air but faulty contents gauge; comment that histology of the heart revealed "chronic myocarditis" although the extent and hence the possible significance of this change is not clear; CAGE.

CASE SC 04/06

This victim was a 53-year old male described as a "big man". Although he reportedly suffered from migraines, tinnitus and impaired vision, he had no history of cardiovascular disease. He gained his open water diver qualification three years earlier and his logbook noted only four dives since then, although it is likely that he had made several more, including two confirmed dives with his former instructor to 23 and 34 msw respectively. This instructor had advised him to always use a large (2800 to 3360 L) cylinder because of his "enormous" air consumption, especially on deep dives. On this occasion, the victim borrowed a smaller cylinder (2240 L) of air at a pressure of 220 bar. He also borrowed a regulator and BCD. He was wearing a 5 mm wetsuit and had approximately 11 kg of weight on his belt.

A group of seven divers boarded a chartered vessel and were taken to a dive site with a maximum depth of 30 msw. Other, shallower, sites had been discussed but were apparently rejected by the boat skipper. Sea conditions were described as choppy and several people on board were reportedly sea-sick, although the victim was not one of these. It was reported that the dive briefing, delivered by the skipper, was scant and a dive leader was hastily selected just prior to entry.

The divers entered the water (the victim made a head-first water entry for some unknown reason) and separated into two groups once they reached a ledge at 24 msw depth, the victim being in a group with two other divers. Visibility was reported to be limited until reaching the bottom where it was possibly 10 metres. There was a current and at least one diver later described the water as cold. The victim became separated from his buddies at one point before they retrieved him. One of the victim's buddies kept a watch on his contents gauge after noticing his use of air was far more than his own, and, after they had been diving about 20 minutes, he noticed that the victim's gauge was reading 110 bar and initiated their return to the anchor rope. When they reached the anchor, one of the buddies realised it was the wrong one but decided that they should ascend up it anyway. The victim's gauge now read 60 bar. When they had reached about 10 msw depth they came to end of the rope and realised that it had been cut previously and was floating free. Both his buddies decided to make a 5-metre safety stop and inflated their delayed surface marker buoys (DSMB). However, the victim continued to the surface and did not re-descend even after one of his buddies reached him and tugged at one of his fins. They stated later that they saw him start to fin towards the dive boat some 20–30 metres away.

The skipper noticed him at the surface, low in the water, facing towards the boat in the choppy water, and then turned to watch the other divers as they surfaced alongside their DSMBs. He concentrated on picking up the other divers, none of whom attempted to swim back to the dive boat in the choppy water. When he next looked he could no longer see the victim and assumed that he had chosen to re-descend, either to make a safety stop or to return below the surface to avoid the rough water. Some time passed before any concern was expressed at the victim's continued absence and a visual check made of the surface. Nobody considered performing an underwater search. After about 20 minutes a call was made and a police boat arrived soon after. An intensive search was made but it found only one of the victim's fins before the conditions deteriorated and the search was called off. His body was found by divers the next morning, lying on the sea bed. All his equipment, except for one fin, was present, his BCD was deflated and his cylinder empty. No faults were found with the equipment when later tested.

Autopsy: X-ray of the head and neck showed some air in neck vessels (it was not identified whether arteries or veins) but none in the heart. At autopsy, the heart weighed 420 g. There was no atherosclerosis of the coronary arteries. The lungs were heavy, weighing 1240 g and 1120 g and there was frothy fluid in the upper airways. There was bruising in the right and left trapezius muscle at the back of his neck at autopsy, which may have been due to a blow from his tank when he made his head-first water entry. Cause of death was given as consistent with drowning.
(Height = 192 cm; Weight = 118 kg; BMI = 32.0 $kg.m^{-2}$)

Comment: This appears to have been a drowning following an out-of-air situation while scuba diving. Other significant conditions contributing to death were excessive air consumption, loss of a fin and negative buoyancy. There were many safety-adverse factors present but ultimately the lethal one was likely to be the failure to drop his weight belt and use his snorkel after he surfaced in somewhat rough water some distance from the dive boat. Adverse factors included the omission of a dive leader and a subsequent absence of checking on the experience of those intending this deeper dive. He was using borrowed equipment and neither asked for nor received advice on its use although the dive organizer, its owner, was aware that he was relatively inexperienced. His history of excessive air use was not disclosed, and the use of a smaller cylinder than he was used to led to an out-of-air situation. His two buddy divers undertook an appropriate monitoring role, but observed no signs of nitrogen narcosis or panic or of his being over-weighted. His medical history of migraine, tinnitus, and some visual impairment was not considered significant.

The skipper failed to recognize the need to monitor the diver he saw "at the surface, low in the water" and could be criticized for not picking him up sooner, but there were soon other divers requesting collection and he could not have known that this diver was out of air. It is apparent the buddies could not have prevented his omitting the safety stop and he failed to indicate at the surface that he was in need of assistance.

Summary: History of migraine, tinnitus and impaired vision; trained but inexperienced; heavy air user; borrowed equipment with smaller cylinder than used to; site changed

prior to dive; careful buddies in group of three; omission of safety stop; separation and alone at surface; probable out of air; failure to signal for assistance, release weight belt or inflate BCD; loss of a fin; lack of trip/dive leader; incorrect role of commercial skipper; drowning.

CASE SC 04/07

The victim was a 55-year-old male who was visiting Australia and holidaying with his wife at the GBR. They both had a medical examination prior to the trip and were both declared to be fit to dive. He was taking valsartan for mild hypertension and bupropion hydrochloride to help him stop smoking. He had been certified to dive for approximately 12 months but it is unknown how many dives he had done.

They were on the first day of a three-day trip on a live-aboard vessel on one of the outer reefs. The couple dived with a group to a depth of 18 msw when the wife had a mild panic attack and indicated to the dive leader she wanted to surface. They soon surfaced and found that the boat was a long way off and had to swim the long distance to the boat. About 10 minutes after boarding the boat, the victim felt sick and vomited. His wife had noticed that "*he was really sweaty*" and told him to go to the upper deck. A short time later he collapsed on the deck and had a seizure. The crew attempted to help him and when they discovered him to be unconscious and apnoeic with no palpable pulse they began BLS with oxygen supplementation, apparently using a bag-valve-mask. An air ambulance arrived and paramedics implemented ALS while the victim was being airlifted to hospital, although he died en route. All of his dive gear was tested by water police and found to be in working order. His air supply was satisfactory.

Autopsy: The heart weighed 375 g. There was an unstable plaque in the right coronary artery with haemorrhage into a plaque and thrombus formation as well as 50% narrowing of the coronary arteries by atherosclerosis. There was focal subendocardial fibrosis. No intravascular gas was detected on CT scan. The lungs were heavy, weighing 1177 g and 1137 g, with haemorrhagic fluid in the distal airway and cut surface of the lung. There was renal arterial and arteriolar nephrosclerosis. The cause of death was given as acute cardiac failure as a result of thrombus in the right coronary artery due to atherosclerosis of the coronary arteries.
(Height = 174 cm; Weight = 74 kg; BMI = 24.4 $kg.m^{-2}$)

Comment: The actual mechanism of death in this man was probably ventricular fibrillation (VF) due to myocardial ischaemia in a person with undiagnosed coronary artery disease. There may also have been secondary drowning due to loss of consciousness from the arrhythmia based on the lung findings.

Summary: Overseas tourist on first day of live-aboard dive trip; significant exertion during long surface swim to boat; had undergone diving medical prior to trip and had been passed fit to dive; collapsed soon after exiting water; probable cardiac arrhythmia due to myocardial ischaemia.

CASE SC 04/08

This 37-year-old male open-water student had no significant medical history and had passed a dive medical examination. He was described by one of his family as a non-swimmer. As part of his training, he was required to do a 200-metre survival swim without aids. There was no time limit on the swim and he struggled to do it, reportedly taking some 30 minutes. He was also supposed to tread water for 10 minutes but was too exhausted to attempt this. On a subsequent day, before completing the pool swim test, he participated in two open water dives at a pier in calm conditions and completed these without any apparent problems.

On the day of the accident, the victim did another dive at the same location as his previous dives. A different instructor was leading this dive and the instructor was apparently unaware that the victim had not completed the treading water test. The conditions on the day were windy, with a surface chop of 0.5–1 metre. Visibility was variously reported to be 5–10 metres, and the depth at the site approximately 5 msw. Some witnesses described the prevailing conditions as very unsuitable for open-water trainees; however, the instructor assessed it to be safe and went ahead with the dive, despite some warnings from at least one bystander who was also an instructor.

There were four students, including the victim and his buddy, and a certified diver joined the group in order to gain some additional dive experience. It was reported that the victim's pressure gauge read 220 bar prior to the dive. The 12.3L cylinder had a capacity of 2700 L at this pressure. The group did a high-water entry from the pier. On entry, the victim's octopus regulator free-flowed and his cylinder slipped down in the BCD mount. These problems were remedied by the instructor. The instructor also needed to assist the victim in venting air from his BCD. After a short time, the group submerged to practise controlled emergency swimming ascents. The victim appeared to have problems re-descending after this ascent and was last seen apparently trying to vent air from his BCD. The instructor then assessed the trainees sharing air with their octopus regulators. The depth was just over 3 msw. He assessed the first buddy pair and then signaled to the next two divers to perform the skill, believing these divers to be the victim and his buddy when in fact one of them was the certified diver. After this skill was done, the instructor realised that a diver was missing and surfaced to search for the missing diver.

There was no sign of the diver on the surface so he re-descended at the shot-line and soon located the victim face-down on the bottom. The BCD was deflated and neither of the BCD-integrated weights had been released. When the instructor turned the victim over, he noticed that his mask and regulator were still in place but he was unresponsive

and apnoeic. He was unable to inflate the victim's BCD so he inflated his own to bring them both to the surface. The victim's mask and regulator appear to have been displaced during this time. Once on the surface, the instructor called for help and was soon assisted by bystanders. The rescuers towed the victim to shore and commenced BLS but ventilations were complicated by froth and regurgitation of water and stomach contents, and by seaweed in the airway. Two trained rescuers continued BLS until paramedics arrived but the victim failed to respond.

When examined, the cylinder was empty. The first stage line pressure was slightly high and the breathing resistance of the second stage regulator was slightly lower than specified. In addition, the low pressure inflator did not function smoothly. However, none of these minor faults was thought to be sufficient to cause substantial problems.

Autopsy: The heart weighed 304 g and the coronary arteries were widely patent. No gas was detected on dissection or radiology. The lungs were heavy, 1146 and 1022 g, the upper airways contained a moderate amount of frothy fluid and the lungs appeared hyperinflated and concealed the cardiac outline. All lobes showed florid congestion and oedema. The cause of death was stated as "*finding in keeping with drowning*."
(Height = 182 cm; Weight = 76 kg; BMI = 22.9 $kg.m^{-2}$)

Comment: The phrase "*finding in keeping with drowning*" reflects the difficulty of diagnosis of drowning at autopsy, as drowning has no pathognomonic features. The diagnosis is based on the circumstances of the death, plus a variety of non-specific anatomical findings such as pulmonary oedema. The reviewing pathologist would give the cause of death as drowning due to poor swimming skills and low air supply while scuba diving.

This trainee was a very poor swimmer and had failed to tread water for the time required. He should not have been allowed to attempt the open water dives prior to completing the pool swimming tests satisfactorily. However, there appears to have been a communication failure within the dive operation and information about the victim's lack of completion of required tests was not conveyed to the instructor who took the victim on these dives. As a result, the victim was permitted to dive. In addition, it is likely that the conditions on the day of this fatal dive were unsuitable for trainees, especially one with such poor aquatic skills. His cylinder reportedly contained approximately 2700 L of air initially and was empty when the victim was found. Given the maximum depth of 5 msw and a dive time of approximately 16 minutes, it is highly unlikely that he breathed all of this gas but rather that substantial air must have escaped from the equipment. Whether this was prior to or after the victim became unconscious is unknown. However, an anxious diver can consume air very quickly. He had not released his integrated weights.

Summary: Open water student; very poor water skills possibly incompatible with safe diving; incomplete water skills assessment; rough surface conditions; separated from group; found unconscious on seabed; BCD deflated and weights on; BLS unsuccessful; drowning.

CASE SC 04/09

This victim was a 36-year-old obese male who had suffered from an embolic stroke from an unidentified source 16 years earlier. He reported minimal residual problems but did suffer from migraines, sometimes with vomiting and visual field losses. He was assessed as 'fit to dive' by a doctor trained in diving medicine and was explained the risks of diving with his medical history. He agreed to accept the doctor's advice that he never dive deeper than 18 msw or make more than two dives in any day, that he always ascend slowly, and always make a safety stop.

He was undergoing open water diver training and on the day before the accident he completed two open water pier dives without incident. He completed another shallow, incident-free pier dive earlier on the day of the accident. Although he told his buddy that he was exhausted, sunburned and did not feel like diving, he persisted. The victim, his buddy, the instructor and some additional divers and divemasters undertook a boat dive at a popular dive site – an annulus of rocks in a current-prone area. The maximum depth in the area was approximately 11 msw. Surface conditions were described as choppy with visibility of 5 metres. The group entered the sheltered water inside the annulus and planned to swim around the rocks, initially swimming with the current. A bystander noticed that, on the surface prior to diving, the victim was breathing erratically and "*fiddling with his equipment*". The divers submerged and swam around the end of the rocks. The victim was grouped with his buddy and the instructor, as part of the larger group. The instructor was in front, followed by the buddy and then the victim. The buddy reported that the victim did not appear to be having any problems. However, when they again encountered the current, this time against them, the victim disappeared from view. His buddy quickly pointed this out to the instructor who then surfaced to locate the victim.

Observers on a nearby boat saw the victim surface rapidly. After a short time he waved to the people on the boat and called for help, complaining that he couldn't breathe and thought he was going to die. Staff on the boat acted quickly to remove him from the water, assisted by the instructor who had surfaced and swum over to assist the victim. The victim was floating on his back, apparently unconscious with his regulator out and water coming out from his mouth. On board, he was apnoeic and pulseless so the instructor began BLS. Water and regurgitated stomach contents made it difficult to attain a clear airway for ventilation and it was several minutes before it was possible to give proper ventilations. Once the boat arrived at the jetty, paramedics applied ALS but the victim failed to respond.

When tested, the victim's second stage regulators were noted to have a much lower breathing resistance than that specified by the manufacturer. This could lead to air surging from the demand valve if the user attempted to breathe deeply. The BCD inflation hose connection was faulty. However, there is no evidence that equipment issues contributed to this accident. The dive computer indicated that he had done a rapid ascent. There was still 110 bar of air remaining in the cylinder.

Autopsy: The heart weighed 440 g and the coronary arteries were widely patent. There was 10–15 ml of gas in the right ventricle when opened underwater, consistent with the diagnosis of PBT/CAGE. There was a patent foramen ovale with a small oval window 5 x 3 mm. Examination of the brain revealed gas bubbles in the circle of Willis, consistent with CAGE. The brain showed no macroscopic damage from the previous stroke. Cause of death: cerebral arterial gas embolism (CAGE).
(Height = 191 cm; Weight = 111 kg (clothed in diving suit); BMI = 30.4 $kg.m^{-2}$)

Comment: Although it is unknown whether or not this victim's previous stroke had impacted on this accident, given the autopsy findings, this seems unlikely. Although his buddy was aware that the victim was feeling unwell prior to the dive, it is unclear if the instructor was aware of this. Despite the prevailing currents at this dive site, it is one that was commonly used for training. It is likely that when the victim encountered difficulty swimming against the strong current, he panicked and ascended rapidly.

Summary: Open water student; previous cerebrovascular accident but assessed fit to dive by diving physician; strong current; separated from instructor and others; surfaced rapidly; unconscious shortly after surfacing; prompt BLS complicated by regurgitation; CAGE.

CASE SC 04/10

This reportedly apparently healthy 32-year-old male had learned to dive in tropical waters about 18 months earlier, the course consisting of four open water dives. He had not dived since. His buddies booked him on a boat dive in temperate southern waters with a dive charter operator. He hired a scuba cylinder, BCD and weight belt. He had planned to conduct a shallow shore dive earlier that day to re-orientate himself to diving and familiarise himself with the equipment but was too buoyant and aborted the dive. He returned to the dive operator's store and obtained extra weights and a different regulator. He now had 18 kg of weight, which included one 2 kg weight in each pocket of his BCD. He did not attempt the shore dive again.

The victim and his friends later joined other divers on the dive charter boat and were taken to a popular 'slack water' dive site. However, on arrival at the site, some witnesses asserted that the divers had to hurry into the water as the current had already begun to run. The site was an underwater hole, beginning at a depth of 12 msw and dropping to a maximum 34 msw. The victim, the last diver to enter the water, initially tried to jump in without his fins on, telling a crew member that he would put them on in the water. He was told to put his fins on in the boat, which he then did then entered the water over-weighted, with his mask on his forehead, his BCD inflator hose unattached and BCD deflated, and without his regulator in his mouth.

He submerged briefly, re-surfaced and struggled for a short time on the surface, apparently attempting to inflate his BCD, before sinking. His buddies (who had been waiting for him several metres down the shot-line) found him unconscious with his mask off on the sea bed at about 12 msw depth and brought him to the surface. His total time underwater was approximately five minutes. When he was brought aboard the dive boat there was froth coming from his mouth but there were some initial indications of breathing and pulse. However, these soon ceased and BLS was commenced, without a response. The rescuers had difficulty using unfamiliar oxygen equipment: a bag-valve-mask for which the rescuers were untrained.

Inspection of the equipment indicated that both first and second stage regulators were in need of servicing, the scuba feed was 'sticky' and that it would have likely been difficult for an inexperienced diver to attach the scuba feed hose to the buoyancy compensator. Despite this, the coroner concluded that "*the equipment was in reasonable condition and unlikely to have contributed to the death of an experienced diver*". The dive operator was subsequently prosecuted by the State Workcover Authority for breaches of the Workplace Health and Safety Regulations and was fined $200,000. The operator subsequently went into liquidation and the fine was not paid.

Autopsy: The autopsy was performed two days after death. The brain was mildly heavy, weighing 1670 g. There was a 10x10x7 mm oligodendroglioma in the right basal ganglia of the brain. The heart weighed 465 g which is normal for his weight. The left ventricle was 15 mm in thickness and there was a bicuspid aortic valve without significant aortic stenosis. The coronary arteries showed 60% stenosis of the LAD coronary artery. Gas was detected in the right but not the left ventricle. The pathologist concluded that the oligodendroglioma, the bicuspid aortic valve and the 60% coronary artery stenosis were probably incidental to the death. The cause of death was given as drowning.
(Height = 182 cm; Weight = 108 kg; BMI = 32.6 $kg.m^{-2}$)

Comment: The cause of death was likely to have been drowning due to equipment problems (i.e. mask and regulator not in place during entry, negative buoyancy (buoyancy compensator not connected and BCD uninflated)). The role of the other pathology in the death is unclear.

Many factors conspired to cause this rather predictable

accident. The diver was inexperienced, had not dived for an extended period, was diving in more difficult conditions than those under which he was trained and was reportedly rushing to get into the water. His buddies did not assist him with gearing up or check him prior to entry, instead waiting for him to meet them underwater. In addition, the divemaster, who was unaware of the victim's lack of diving experience, failed to properly check his equipment prior to his entry. This incident might not have occurred had his buddies or the divemaster taken the time to ensure his equipment was in place and functional prior to entry.

Summary: Inexperienced; rushing to get ready; poor communication by dive operator and poor supervision by divemaster; no buddy check prior to entry; entered with mask on forehead, regulator out of mouth, BCD deflated and hose unattached, and over-weighted; buddies waiting underwater; ventilations attempted using unfamiliar oxygen equipment; drowning.

Rebreather diving death

CASE RB 04/01

This 55-year-old male had begun diving seven years earlier. He had completed numerous diving specialty courses, including the use of a rebreather, and dived regularly and often, having logged over 250 dives in his first two years since certification and diving regularly since, often using his rebreather.

The victim and four others set out to dive on a wrecked barge about two kilometres offshore on a sandy bottom at about 50 msw. Conditions were said to be "ideal" – calm, little current, no surge and with around 15 metres visibility. Each diver had done his own breathing gas preparation, calculated his own dive profile and looked after his own gear, There was a mix of gear in use – some divers, including the victim, used rebreathers and others used twin-tank open-circuit scuba. As is fairly common practice in deep technical diving, the divers descended with no particular buddy system. The barge had a small cabin at both bow and stern, entry being through a hatch, which covered each opening and was significantly more restricted over the bow cabin. One of the last divers to see the victim alive was moving towards the bow of the vessel when he saw the victim backing into the tight bow cabin hatchway, feet first at a depth of 46 msw. Despite being a very tight fit, one that the other divers would not attempt, the victim managed to enter the cabin without having to remove any of his gear in the process. As there was no room for anyone else in the bow compartment this diver waved to the victim and swam off elsewhere.

This group's practice was that each diver would leave a marker (strobe light) at the bottom of the ascent line. As each diver left the bottom, he collected his marker and, on this day, the agreement was that the last diver would ensure the release of the anchor line. Concern was raised when, at least 10 to 15 minutes after all the other divers had surfaced, they realised that the victim had not released the anchor from the wreck. He should have surfaced or at least have been decompressing by this time. However, there was no sign of the victim on the surface, so one of the divers re-descended to the wreck and quickly found the victim. He was floating upside down (the air in his drysuit having gone to his feet), separated from and above his equipment, which was lying on the deck of the wreck. He was still attached to the gear by his dry-suit inflator hose, with no mouthpiece in his mouth. He was unconscious and apparently lifeless. Knowing that the victim had very likely been in the water for too long to survive and being aware that he did not have sufficient gas supply to recover the victim, this diver returned to surface. The boat skipper and another diver then descended to recover the body.

The victim had been diving with a rebreather. The diluent cylinder was found to be empty. It appeared that he had removed his gear to exit the wreck through the tight opening. Most of his diving weight was in the rebreather with very little on his belt. On releasing his gear, the buoyancy of his drysuit may have caused him to float above his gas supply and it is probable that he would not even have been able to reach the sling bottle used as back-up gas.

Later testing of the rebreather unit revealed that it operated appropriately according to the operating manual. One concern related to the type and status of the soda lime used in the rebreather. Evidence implied that this may have been of low quality or had perhaps been used already in an earlier dive. If so, then some degree of carbon dioxide intoxication was a possibility. It was officially concluded that efficacy of the equipment used was unlikely to have contributed to the fatality. The official conclusion about the tragic turn of events was that:
"*It was the aforementioned factors: poor decision by (the victim) to enter the hatch, the need to remove the rebreather prior to exiting the hatch, the fact that the deceased had embraced a lone diving policy, and the sudden and unrecoverable change in the deceased's buoyancy, that have combined to produce the ultimately fatal result*."

Autopsy: The autopsy was performed three days after death. The heart weighed 460 g and the coronary arteries showed 50% narrowing of the LAD coronary artery. There was mild myocardial perivascular fibrosis. Gas bubbles were detected in the vessels on the surface of the brain, the basilar artery and the pia mater, and within the heart ventricles. However, it could not be determined whether this was due to post-mortem off-gassing from bringing the body to the surface from depth, or decomposition from the three day post-mortem interval. The lungs weighed 1000 g and 800 g and there was frothy fluid in the upper airways. There was a 10 mm laceration on the posterior scalp. Toxicology was negative for alcohol and all drugs tested. Carboxyhaemoglobin level was 1%. The cause of death was given as drowning.
(Height = 186 cm; Weight = 85.5 kg; BMI = 24.7 $kg.m^{-2}$)

Comment: Removal of a CCR in order to attempt a difficult exit following penetration of a wreck is a very dangerous manoeuvre, leading in this case to death. The role of the 50% stenosis of the LAD is hard to evaluate but in a hypoxic situation could be the difference between survival or death. The small laceration on the scalp could have resulted from an impact during the attempted exit.

The diver who had seen the victim trying to squeeze through the hatch had signaled "you're mad" but the victim had just shrugged his shoulders and gone through. The witness did not think anyone wearing a rebreather would attempt this as it was too tight an opening. He considered the victim "*a bit of a loner*" who would "*go off by himself and attempt to negotiate tight places others would not attempt*" and said he had seen him on previous dives trying and failing to enter this cabin. The critical factor in this fatality appears to be the diver's personality, his desire to push his limits and break the safety rules. Removing his equipment while alone at depth in order to exit a narrow hatch was a predictably dangerous action in the absence of any buddy to assist him safely complete the manoeuvre.

Summary: Well-trained and experienced technical diver using a closed-circuit rebreather; diving separately in group on deep wreck; most weights worn on rebreather; removed rebreather to exit wreck; too buoyant without rebreather and lost and then floated above gas supply; drowning.

Surface-supply death

CASE SS 04/01

The victim was an apparently healthy 24-year-old male who was a 'backpacker' from overseas. He held an advanced diver certification and was reported to have made 70 dives, the deepest to 45 msw. He had answered an advertisement in the local paper to become an ocean harvester, collecting sea cucumbers or crayfish off the ocean floor. However, there is no evidence he had ever used surface supply (hookah) equipment, or performed drift diving to collect sea cucumbers, before the fatal dive.

On the day of the accident, there were four other similarly inexperienced divers on the boat who were to be introduced to the work. The five new employees were given an operations manual to read. They then signed an employee declaration stating they agreed to accept these standards. Their induction into the work involved a demonstration of the equipment, information on how to manage it, how to respond to loss of work-line, use of the bail-out bottle and decompression procedure. They did not use any of the equipment. However, a short demonstration dive was made by the nominated head diver.

That afternoon, after the introductory session, a working dive was conducted. The group consisted of one of the 'experienced' crew divers (who were not recorded as holding any occupational diving or harvesting qualifications) and two of the neophytes. It appears that the intention was harvesting rather than training or observation of their performance. Indeed, it is doubtful whether they would have been able to readily observe the actions of the two new employees because of positioning and other tasks involved. The weather was calm with a strong current running, visibility underwater a maximum of 15 metres and water temperature 29^{o}C.

The first of these dives passed without incident, with two of the new divers involved in its topside management. The victim participated in the second of these dives and the boat skipper was the experienced diver in the trio at depth. It was later reported that the victim looked a bit nervous and vague as he prepared for the dive and had required assistance to correctly don and position his equipment. He chose not to wear a wetsuit, only a tee-shirt and shorts (although this was in breach of the employer's policy), reducing his weights accordingly. He required help to clip his hose to his weightbelt, a difficult task wearing gloves.

The second dive was planned for 25 minutes' bottom time and a maximum depth of 35 msw. Nitrogen narcosis problems were expected to be mild. One of the new divers, acting as the on-deck tender, later reported that the victim had commenced his dive by swimming through the hoses of the diver next to him, contrary to their instructions, and causing the hoses to become entangled. He stated that he did not think this would have caused a problem and did nothing about it. This on-deck tender had only been trained that day. Two of the divers surfaced at the planned dive time, after a decompression stop. One of them brought up the victim's air hose and regulator and only then was it realised a diver was missing. The hope was that he had ascended using his pony bottle but a surface search was unsuccessful. The catch bags were raised and the victim's bag was found to also contain his neck bag (a small catch bag carried by the diver), indicating that he had at some stage swum back to reach it.

During the dive, none of the others divers or tenders had seen him. When the diving equipment and lines were retrieved, it was noted that the victim's line and air hose appeared to be in a knot and when his regulator was examined, the mouthpiece was missing. It is usual for it to be held in place by a plastic cable or a metal clamp and one witness stated there was a plastic tie around it before the dive commenced. A later check of the rest of the equipment revealed two without the essential cable ties, and on some others the cable ties were too loose to ensure retention. It was noted that there was no evidence of an attempt to use the bail-out bottle.

A formal search of the dive area the next day found the body on the sea bed. All of the victim's equipment was in position except for his mask. The victim's bail-out bottle had not been turned on and its regulator was still stowed in position. Massive tissue damage of his upper chest and face was present, which was later attributed to post-mortem shark action.

Autopsy: The autopsy was performed 5 days after death. The pathologist noted there was evidence of post-mortem changes and damage from both shark and sea lice. There was no evidence of physical injury or health factors as the cause of death. The heart weighed 275 g and the coronary arteries showed only a focal 30% narrowing in the LAD coronary artery branch. The lungs weighed 310 g and 264 g and showed decompositional changes. The cause of death was stated as drowning.
(Height = 183 cm; Weight = 86 kg; BMI = 25.7 $kg.m^{-2}$)

Drift diving procedure

The drift diving procedure was for the boat to maintain a slow (1 knot) forward speed using a drogue to maintain steerage as it towed from two to four divers. There was a boom extending from each side of the boat, each one held the working lines, air lines and oxygen (for use at the decompression stop), all of which were controlled from the vessel. Each working line, two from each boom, consisted of a down-line weighted by chains which were kept just above the sea bed. A large catch bag was attached to this and a 50-metre line trailed back above the sea bed from it. Each diver worked his own line, pulling himself along the line or swimming back to the bag as he caught his quarry. Each of the air-lines had a Tema brand end connection. A non-locking karabiner was whipped to the hose at the diver's end. Separate hoses were used for the oxygen delivery system. Two divers shared a single delivery hose.

Each diver's weight belt had a stainless steel 'D' ring to serve as the attachment point for the air hose karabiner. His regulator then connected to the quick-connect fitting of the hose and a bail-out or pony bottle was worn in a backpack. To use this, the diver had to reach back with his left hand to turn the cylinder on, and, with his right hand behind his back, pull the regulator free from its restraining cable ties. As the cylinder was worn inverted, it effectively required a reversal of the usual rotation direction needed to open the valve. This procedure requires practice. It was possible for the karabiner to be accidentally released if the lines or hoses were twisted over the karabiner gate. Also, the weight belt could not be easily released independently of the air hose, which would create a problem if the diver needed to ditch the weight belt quickly and ascend using his usual air supply from the hose. As there were several 'D' rings on the harness which held the bail-out bottle, a safer attachment area was available but not used. All of the divers wore half-face masks but none had the safety factor of a head strap to retain the mouthpiece if it became displaced.

Comment: It is especially difficult to diagnose drowning in the presence of decomposition as the typical lung changes are lost, but the most likely cause of death was drowning following loss of the diving regulator due to an inadequate harness system while surface-supply diving.

This fatality was investigated by a local Workplace, Health and Safety Inspector and the report listed numerous serious breaches of the regulations governing diver safety. The victim was untrained in the use of surface-supply diving equipment. He had never practised the use of a bail-out bottle air supply or of the oxygen decompression procedure. He was wearing a half-face mask without strap to retain it should he lose consciousness. He had his line and air hose attached to his weight belt rather than to a backpack holding his bail-out bottle. There was no communication with the surface or a stand-by diver at the surface. The regulator mouthpiece was not securely attached. He was not wearing a wetsuit or other protection, putting him at risk of avoidable injury. The karabiner was non-locking and could be easily detached. The anxiety of this first dive, and the probable presence of some nitrogen narcosis, would further reduce any safety margin. Following the investigation, the company was prosecuted and despite imperfect legislation at the time, was fined AUD$6,000. The skipper, who was part owner and supervising diver on the day, was fined AUD$10,000. This was the third occasion this company had been prosecuted.

Summary: Experienced recreational diver; no known health problems; using hookah for first time and inadequately trained in its use; first dive as commercial sea harvester; inadequate supervision; air hose entangled and regulator mouthpiece likely to have been loose and become detached; mask may also have become detached; out of sight of other divers; nitrogen narcosis possible factor; body found following day; drowning.

CASE PRE-SC 04/01

This victim was a 65-year-old male who was an experienced and active diver over many decades. He and two friends were in a small, inflatable boat and heading out to go diving at one of their regular dive sites, leaving the shore late in the morning. After about 20 minutes, the victim fell forwards in the boat and was found to be unconscious and apnoeic. The friends headed back to shore, which took twenty minutes, attempting BLS en route. On reaching shore, BLS was continued on the beach, with supplemental oxygen, by various bystanders including some doctors and nurses. Paramedics arrived after approximately 30 minutes but the victim failed to respond to ALS.

Autopsy: The autopsy showed severe ischaemic heart disease. The heart weighed 470 g. There was severe focal calcific atherosclerosis with stenoses of the left main LAD, left circumflex and right coronary arteries. In addition there was a thrombus in the right coronary artery. Histology showed a healed sub-endocardial infarct and contraction band necrosis but no neutrophil infiltrate. Cause of death was given as ischaemic heart disease.

Comment: It was unknown whether or not the victim had recently seen a doctor or was on any medications. However,

his buddy stated that the victim had been complaining of constant "indigestion" and that, lately, he had seemed lethargic. This death could very likely have occurred at any time. Although this incident did not occur during a dive, and is therefore not strictly classified as a diving death, it is included here to highlight how an existing medical condition can be implicated in a diving accident. Had this person become unconscious in the water a short time later, it would have been classified as a diving death, and, unless an appropriate autopsy was conducted, which is unlikely in many parts of the world, the cause of death may well have been attributed to drowning.

Summary: Experienced diver; pre-existing ischaemic heart disease; collapsed prior to dive; unstable plaque in right coronary artery.

Discussion

BREATH-HOLD DIVERS AND SNORKEL USERS

In previous Project Stickybeak reports , there were several deaths which were most likely attributable to apnoeic hypoxia.[4–10] Despite this phenomenon being well described and discussed both within and outside of the freediving community many young breath-hold divers still seem to fail to realize the dangers of pre-dive hyperventilation and pushing their apnoeic times to the limits. That this problem is also relatively common amongst the 'elite' breath-hold divers who are often highly educated on these matters is probably an indication that education alone will not see this problem disappear. Equally, as will be discussed later with regards to one of the technical diving deaths, experience may well work against these individuals as, the more often they "get away with it" the more likely they are to push the limits further. Blackout with little or no warning can occur before, during or soon after ascent. Unless a rescuer is immediately at hand, drowning will be the most likely result. Even the relative safety of a swimming pool can prove dangerous if there is no supervision, as was the case with BH 04/03.

CARDIAC-RELATED DEATHS

Cardiac-related issues appear to have been instrumental in the deaths of five snorkel divers (56%) and four of the compressed gas divers (31%) in this series. Of these nine divers, only four were known to have been undergoing treatment for cardiovascular conditions (BH 04/05, BH 04/06, BH 04/08 and SC 04/07). The victim in Pre-SC 04/01 appears likely to have been suffering cardiac symptoms over recent times but there is no evidence that he had visited a doctor for this. BH04/08 represents another case where an individual has significant medical conditions but would appear to be able to conduct normal activities of daily life, only to die with the relatively minor challenge of snorkelling in tropical waters, whilst, in BH04/06, the severity of the atheromatous lesions apparent on autopsy would generally be considered to be non-critical clinically.

This phenomenon of almost silent cardiac death without what would usually be considered clinically significant coronary lesions in divers and snorkellers would appear to be a recurring theme in diving fatalities. Dysrhythmias, sudden death and pulmonary oedema are becoming increasingly recognized in the setting of immersion.[11–13] As with many unobserved diving-related deaths, the exact agonal sequence of events in many cases will never be known with certainty and we can but speculate on the exact cause of death.

Given the high incidence of cardiac-related diving fatalities in 'older' snorkellers and divers, it seems reasonable to suggest that, over the age of about 45 years, they should be strongly encouraged to have their cardiovascular health periodically assessed by a doctor, preferably one who is well aware of the cardiovascular stressors associated with diving and snorkelling. However, even a full cardiac evaluation may not reveal the presence of a risk that should have precluded these in-water activities.

REFRESHER DIVES / SUPERVISION OF DIVERS

Diving accidents in divers who have just returned to diving are not uncommon. Any diver who has not dived for an extended period needs to have the opportunity to re-orientate to diving by undertaking an appropriate refresher dive(s) under favourable circumstances. This is especially important for an inexperienced diver, divers in whom dive fitness is a potential issue, and/or for one who is planning to dive in more demanding conditions than previously experienced.

Although the victim in SC 04/10 had conducted a brief, unsupervised 'orientation' dive prior to the fatal event, without suitable supervision and education from a qualified person, this was of little value as the problems that it highlighted were not adequately addressed. When required to enter the water quickly for the subsequent dive, he was totally unprepared. The events during the subsequent dive raise the difficult issue of duty of care of the dive operator and divemaster in the setting of an ostensibly qualified diver. There is an ongoing debate about the relative responsibilities of qualified divers and the dive professionals with whom they dive. Some argue that a qualified diver must take full responsibility for themselves and accept any and all risks associated with their diving activities. Others, including most of these authors, believe that those who take others diving, whether within a commercial setting or otherwise, owe a duty of care to the divers, and that the level of this duty is inversely proportional to the experience of the diver.

Had the divemaster been aware of this victim's inexperience, he may well have more carefully observed him and, hopefully, would have ensured that he had his mask on, regulator in his mouth (air on) and that his BCD was functional and inflated prior to entry. However, one would have thought that the victim's lack of experience was evident by his behaviour, and it is rather alarming that the divemaster or skipper did not prevent him from entering the water with

his gear as it was. Had this been done, this accident may well have been prevented. Equally, one may question how a diver of this experience was allowed on the boat in the first place without the operator conducting a 'check out' dive under suitable supervision.

A divemaster, by acting as the 'gate-keeper' prior to entry, has a good opportunity to prevent some avoidable mishaps. The divemaster in this case was not made aware of the victim's lack of experience as this information had not been relayed from the dive shop staff to the boat staff, in breach of the dive operator's own written procedures. This failure to follow appropriate procedures led to the prosecution of the dive operator by the local workplace authority. The successful prosecution led to an audit of all local recreational dive operations by the workplace authority. Operators were required to produce evidence of having appropriate procedures in place, regular documentation of the checks included within these procedures and safe, regularly serviced equipment, along with a variety of other requirements.

Inadequate supervision also appears to have been a factor in SC 04/06. This situation can often arise with a so-called 'bare-boat charter' where a boat and non-divemaster skipper are chartered by a dive group and no-one in particular is appointed to oversee the divers. As a result, an inexperienced diver can miss out on required supervision.

An instructor must be constantly vigilant and maintain close contact with his or her students at all times. This can sometimes be difficult to achieve, especially in poor conditions and / or with many students. It was unfortunate that the instructor in SC 04/08 initially failed to realise that the student was missing from the group until it was too late. Extra certified divers with a student group should be clearly identifiable and well-briefed on appropriate separation from the students in order to avoid a mix-up, as occurred here. The victim in SC 04/02, who was undertaking a resort dive, was also separated from his instructor at what appears to have been the considered decision of the instructor. This turned out to be a poor decision, although the victim may well have died despite the immediate presence of the instructor.

POOR AQUATIC SKILLS

Diving is an aquatic sport, and a minimal swimming capability would appear to be fundamental. There is no logic to having a swimming test then allowing a student who fails this test to proceed to open-water dive training. SC 04/08 is a tragic example of just such a student diver who was unprepared for diving in the circumstances into which he was taken. He was described as a "non-swimmer" and had failed to complete the required basic aquatic skills tests. He should not have been taken on this dive, especially into conditions that were described by several other instructors as relatively rough and inappropriate for open water students.

LACK OF APPROPRIATE TRAINING

Dive fatality reports not infrequently include cases of divers who were untrained, or inadequately trained for the diving activities undertaken. The victim in SC 04/03 was uncertified and inexperienced. Learning from a friend who is not a trained instructor is inadequate as there is a lack of the usual 'checks and balances' required when being trained and certified by a licensed instructor. The employer of the victim in HH 04/01 obviously failed to provide adequate training and supervision and these actions no doubt contributed greatly to the victim's demise.

BUDDY SYSTEM IN TECHICAL DIVING

Technical diving is increasing in popularity. Technical divers are taught to be self-sufficient, a laudable attribute; however, divers who dive alone or without a reasonable buddy system continue to be well-represented in diving death statistics.[14] Overconfidence and a history of 'having got away with it' often encourages such individuals to push their limits a little further each time until a situation becomes unrecoverable. The 'same ocean' buddy system where divers know who is about but do not specifically stay close to one another is common practice amongst technical divers. As well demonstrated here, unless there is a proper close buddy system, there is in reality no buddy system at all.

The victim in RB 04/01 was well known for penetrating tight spaces. As an experienced diver he should have been aware of the buoyancy issues surrounding taking off his gear at depth, His decision to use the small gas supply in his CCR rather than that in his 'bail-out' cylinder is also an interesting point. In the post-event analysis of this diver's equipment there was some suggestion that the CO_2 canister may have been near exhaustion. Discussion of this finding with the dive operator revealed that the victim was due to leave on holiday after this dive and was unlikely to have had a freshly charged CO_2 scrubber. Whether this was an influencing factor in his decision to remove his equipment to extricate himself rather than wait for assistance must remain a matter of speculation. Even highly competent, self-sufficient technical divers should be aware that not all situations are self-recoverable. The authors would suggest that even for this group, buddy diving makes for a more enjoyable and safer experience.

CARRYING OF BAIT BAG OR TETHERING FISH TO BODY

It was reported that the victim in BH 04/07 was wearing a bait pouch. The practice of carrying bait bags and/or tethering fish near to the body was relatively commonplace many years ago. However, having captive marine life in one's possession underwater does appear to increase the likelihood of shark attack, and, when this became better

known, its popularity waned.[15] It is a practice that should continue to be discouraged.

FAMILIARITY WITH OXYGEN / RESUSCITATION EQUIPMENT

It is not sufficient to have fulfilled the criteria of having emergency equipment available if there is no one who is trained or available to use it. This was vividly demonstrated in the case of SC 04/10. The victim was reported to be initially breathing when brought aboard the boat and given that he was relatively young, apparently healthy and had only been submerged for several minutes there may have been a brief window of opportunity where a successful resuscitation was possible. Unfortunately, the staff who were present did not appear to have been sufficiently familiar with the operation of the resuscitation equipment available, nor was the diving instructor who came aboard from another dive boat to perform the resuscitative efforts. The unit available was a bag-valve-mask device with a mask of a type that is difficult to obtain a seal with during positive pressure ventilation in inexperienced hands. In addition, the rescuer was also unable to use this type of mask for mouth-to-mask ventilation when he tried to revert to this.

Failure to achieve an adequate mask seal and perform effective ventilations is common with bag-valve-masks devices when used by infrequent operators, and especially when used by a single operator.[16–18] Studies suggest that it is more effective to provide oxygen-supplemented rescue breathing using a resuscitation mask with oxygen inlet.[16,19,20] It is important that oxygen equipment is chosen carefully so that it is not only effective, but is relatively simple to operate by those who are likely to use it. Dive leaders should be appropriately and thoroughly trained and well-practised in the equipment they are likely to be required to use in an emergency. Rescuers should also be prepared to quickly abandon equipment if they are having trouble using it and to revert to basic rescue breathing while equipment issues are resolved by others, if possible.

POST-MORTEM FINDINGS OF PULMONARY BAROTRAUMA / CEREBRAL ARTERIAL GAS EMBOLISM

There were four deaths in the scuba divers attributed to PBT/CAGE making it the single most common cause of death apart from drowning. It is difficult to distinguish the cause of gas found at autopsy between CAGE, post-mortem off-gassing, decomposition and resuscitation. The reviewing pathologist is increasingly restricting this diagnosis of PBT/CAGE to cases where there is a witnessed history of rapid ascent to the surface followed by loss of consciousness. In the absence of this history, especially where there is a delay in imaging or autopsy, these cases should probably be classified as possible or probable PBT/CAGE. The CT scan needs to be done within the first eight hours post mortem to be of value.[21]

PROBLEMS WITH DIAGNOSING DROWNING AT POST MORTEM

The contribution of drowning to the death of people with schaemic heart disease can be difficult to assess, in part due to the non-specific nature of the pathology of drowning. For this reason, it is important to include such information as the macroscopic appearance of the lungs, the weight of the lungs and the presence or absence of pulmonary oedema in the upper airways.

ROOT CAUSE ANALYSIS

A summary of the root cause analysis is shown in Table 3.

Snorkel and breath-hold divers

Unsurprisingly, exertion was suspected to be the major 'trigger' in all five of the cardiac-related snorkelling fatalities. Other potential triggers to a cardiac event might include a tight wetsuit and possibly aspiration of water through the snorkel. The disabling injury was thought to be the same as the cause of death in all but BH 04/05 where it is believed the victim may have become unconscious as a result of a cardiac event and subsequently drowned.

Compressed gas divers

Again, exertion appeared to be a key trigger in the cardiac-related incidents. Loss of air supply and negative buoyancy were major triggers in the drownings. In the incidents involving diving using compressed gas, it is believed that the disabling injury was the ultimate cause of death in all cases reviewed. The main disabling injury and cause of death was drowning (five of 12 cases), followed jointly by CAGE (four cases) and cardiac-related deaths (three cases).

Conclusions

- There were 22 reported diving-related fatalities during 2004, which include nine deaths while snorkelling and/or breath-hold diving, 10 while scuba diving, one shortly prior to scuba diving, one while using a closed-circuit rebreather and one while using surface-supply breathing apparatus.
- Causal factors associated with these deaths include apnoeic hypoxic blackout from extended breath-hold diving; cardiac disease, whether diagnosed or not; lack of training; inexperience or lack of recent diving experience; poor supervision and poor aquatic skills.
- The main disabling injury with snorkellers was a cardiac incident (five of nine cases). With scuba divers, the main disabling injuries were CAGE and asphyxia (each with four of 12 cases) and cardiac incidents (three cases).
- Factors that may reduce mortality in the future include improved medical screening of older divers; cessation of the practice of hyperventilation prior to breath-hold diving; closer supervision of inexperienced divers,

Case	Trigger	Disabling agent	Disabling injury	Cause of death
BH				
04/01	Extended breath-hold	Sudden loss of consciousness	Asphyxia	Drowning
04/02	Extended breath-hold	Sudden loss of consciousness	Asphyxia	?Drowning (body not recovered)
04/03	Extended breath-hold	Sudden loss of consciousness	Asphyxia	Drowning
04/04	Exertion	Cardiovascular disease (coronary atherosclerosis)	Cardiac incident (probable cardiac arrhythmia due to myocardial ischaemia)	Cardiac-related (ischaemic heart disease)
04/05	Exertion	Cardiovascular disease (coronary atherosclerosis; left ventricular hypertrophy)	Cardiac incident (?cardiac arrhythmia)	Drowning
04/06	?Exertion; ?water inhalation via snorkel	Cardiovascular disease (coronary atherosclerosis)	Cardiac incident (probable arrhythmia due to myocardial ischaemia)	Cardiac-related (ischaemic heart disease)
04/07	Bait on belt; dying fish	Shark attack	Trauma (transection of left femoral artery)	Trauma (shark bite of left thigh)
04/08	Exertion; ?tight suit; ?anxiety	Cardiovascular disease (coronary atherosclerosis; left ventricular hypertrophy)	Cardiac incident (ischaemic heart disease; left ventricular hypertrophy)	Cardiac-related
04/09	Exertion in current	Cardiovascular disease (coronary atherosclerosis)	Cardiac incident (probable arrhythmia due to myocardial ischaemia)	Cardiac-related (ischaemic heart disease)
SC				
04/01	Trauma (hand/arm injury)	Rapid ascent	CAGE	CAGE
04/02	Exertion; stress	Cardiovascular disease (coronary atherosclerosis)	Cardiac incident (probable arrhythmia due to myocardial ischaemia)	Cardiac-related (ischaemic heart disease)
04/03	Insufficient gas	Probable rapid ascent	CAGE	CAGE
04/04	Exertion; tight semi-dry suit	Other medical condition (HLA B27 spondyloarthritis; ?hypercoagulable state)	?Cardiac incident (?cardiac arrhythmia)	?Cardiac-related (?cardiac arrhythmia)
04/05	?Anxiety (feeling unwell; faulty contents gauge)	Rapid ascent	CAGE	CAGE
04/06	Out of air; negative buoyancy	Loss of air supply	Asphyxia	Drowning
04/07	Exertion from long surface swim	Cardiovascular disease (coronary atherosclerosis)	Cardiac incident (probable cardiac arrhythmia due to myocardial ischaemia)	Cardiac-related (ischaemic heart disease)
04/08	Unknown (?loss of air supply; ?lack of buoyancy)	Loss of air supply	Asphyxia	Drowning
04/09	Panic (feeling unwell; current; separation)	Rapid ascent	CAGE	CAGE
04/10	Buoyancy problem (negatively buoyant); disabled BCD; regulator out	Loss of air supply	Asphyxia	Drowning
RB				
04/01	Separation from gas supply	Loss of air supply	Asphyxia	Drowning
SS				
04/01	Separation from gas supply	Loss of air supply	Asphyxia	Drowning

Table 3. (opposite page)
Root cause analysis for 21 diving fatalities in Australian waters in 2004; BH – breath-hold diver; SC – open-circuit scuba diver; RB – rebreather diver; SS – surface-supplied diver

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out-of-practice divers or divers who are inexperienced in the particular environment; better screening by dive operators and better communication within dive operations and between buddies.

Conflict of interest

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

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Douglas Walker, MB, ChB, MM, has been the principal researcher for Project Stickybeak since its inception in 1972, a founder member of SPUMS and Editor of the SPUMS Journal from 1974 to 1990.
John Lippmann, BSc, Dip Ed, OAM, is Executive Director of the Diver Alert Network Asia Pacific (DAN AP).
Chris Lawrence MB, BS, FRCPA, is Director – Statewide Forensic Medical Services, Royal Hobart Hospital, Tasmania.
John Houston, MSc, is a researcher at DAN AP.
Andrew Fock, MB, BS, FANZCA, Dip DHM, is a senior specialist for the Hyperbaric Services, The Alfred Hospital, Melbourne.

Corresponding author:
John Lippmann
DAN AP, P.O. Box 384 Ashburton,
VIC 3147, Australia
***Phone:** +61-(0)3-9886-9166*
***Fax:** +61-(0)3-9886-9155*
***E-mail:** <johnl@danasiapacific.org>*

Review article

The role of intra-vascular bubbles and the vascular endothelium in decompression sickness

Alf O Brubakk and Andreas Møllerløkken

Key words
Decompression, bubbles, Doppler, endothelium, stress, exercise, review article

Abstract

(Brubakk AO, Møllerløkken A. The role of intra-vascular bubbles and the vascular endothelium in decompression sickness. *Diving and Hyperbaric Medicine*. 2009;39(3):162-9.)
Although decompression procedures have been improved over the years, decompression still remains a significant problem in diving. While there is universal agreement that the basic problem of decompression is gas coming out of solution, forming bubbles when pressure is reduced, the exact mechanism of decompression injury is not known. Furthermore, the wide variety of clinical symptoms and the significant difference in individual susceptibility makes identification of the mechanisms involved difficult. Using ultrasound, vascular gas bubbles have been detected in most decompressions, and these bubbles can act on the endothelial lining of blood vessels resulting in impaired endothelial function. Normal endothelial function is a major indicator of cardiovascular health and thus a reduction in vascular bubble formation and hence the risk of endothelial injury is an important goal in decompression. Even if vascular gas bubbles may not be the only adverse effect of decompression, vascular gas bubbles and their adverse effects on the endothelium may be a useful model for decompression injury. This review claims that endothelial dysfunction may be a possible main mechanism for neurological decompression injuries and describes some of the effects of vascular gas bubbles on the endothelium. Furthermore, as the formation of vascular gas bubbles can be significantly influenced by physical exercise and the use of nitric oxide, a novel approach to reducing the risk of decompression injury is suggested.

Defining adverse effects of decompression

Following return to atmospheric pressure after a dive or an exposure to altitude, clinical symptoms and signs can occur. These vary from mild to very severe including death, and have been given a variety of names: decompression sickness (DCS), decompression illness (DCI), 'niggles', aeroembolism, nitrogen disease, diver's palsy, and compressed air illness to name a few. The most commonplace name, the 'bends', is attributed to the posture adapted by fashionable ladies at the turn of the 20th century, the 'Grecian bend'. This posture, bending forward at the waist would give some comfort to a diver in acute pain. As described by Ferris,
"*the term is used to denote the syndrome of pain and disability, localized in the locomotor system ... The pain is usually localized in the joints and may radiate up and down the extremities involved ... The subjective pain is usually described as being a deep aching pain, difficult to localize, which – once it begins – usually progresses in intensity with periods of waxing and waning ... When severe it is associated by functional impairment of the involved extremity, with a feeling of numbness and weakness of the part and with faintness.*"[1]
Or as was described by Behnke,
"*The major symptoms and signs of decompression sickness are pain (bends), asphyxia (chokes) and paralysis. Minor effects are rash and fatigue. The parts of the body chiefly involved are the extremities (bends), cardiorespiratory system (chokes) and the spinal cord.*"[2]
Even today, there is probably little to add to Behnke's 1951 description, with the possible exception that the brain may be more frequently involved and that extreme fatigue may be a more serious sign than previously thought.[3]

Traditionally, the symptoms following decompression (dysbarism) have been categorised according to their anatomical location and severity:

- Type I (mild): muscle and joints, skin, lymphatics, malaise / fatigue
- Type II (serious): spinal, cerebral, vestibular, cardiopulmonary ('chokes')
- Arterial gas embolism
- Barotrauma.

This classification implies that the different categories are well defined disease entities and that there is reasonable agreement between doctors about the classification. However, studies have demonstrated that there is considerable uncertainty between experts about classification.[3] For instance, many cases of cerebral DCS cannot be distinguished from arterial gas embolism or vestibular barotrauma. Furthermore, several studies have shown that joint symptoms alone are uncommon; they are usually accompanied by central nervous symptoms.[4,5] According to Diver Alert Network (DAN) data, 40% of symptoms are neurological, 13% are

vestibular and 22% pain.[6,7] Extreme fatigue can be classified as a harmless sign or be a sign of subclinical pulmonary embolism. Therefore, the term 'decompression illness' was suggested to include both decompression sickness and arterial gas embolism.[8] It was further suggested that the disease should not be classified as Type I and Type II, but instead described according to clinical symptoms and their development. Using this classification scheme, a high degree of concordance between different doctors was reached.[9]

However, the classification debate loses some of its importance when we realize that all clinical signs of DCS are treated similarly by using oxygen and pressure. Nevertheless, in discussing the more general problems related to the effects of decompression, several other definitions may be used:

- Acute clinical symptoms requiring treatment in individuals who have been exposed to a reduction in environmental pressure
- Acute clinical symptoms in individuals who have been exposed to a reduction in environmental pressure
- Organic and/or functional decrements in individuals who have been exposed to a reduction in environmental pressure
- Vascular gas bubbles without clinical symptoms in individuals exposed to a reduction in environmental pressure.

The first definition is the one traditionally used and is incidentally the one used to evaluate the effectiveness of decompression procedures. This is probably quite accurate if serious symptoms occur. However, decompression illness requiring treatment is a rare disease. In commercial diving, the incidence of treated DCI is probably below 1%.[10] In recreational divers, the incidence appears to be much lower – about 0.01–0.05%.[11] However, these general numbers hide the fact that, even in commercial operations, DCS shows considerable individual differences/variability (see below).

Even if it is uncommon, a large proportion of divers have been treated for DCI. In a survey of divers in an off-shore diving company in 1985, 38% of the divers with 1–9 years' experience and 62% of those with 10–24 years of experience had been treated.[12] A survey of a large population of Norwegian divers showed that 3% of the recreational divers and 28% of the experienced professional divers had been treated for DCI.[13] For many years, there has been anecdotal evidence that clinical symptoms of DCI are considerably under-reported. In the Norwegian survey, 19% of the sports divers, 50% of the professional air divers and 63% of the saturation divers reported that they had had symptoms that had not been treated with recompression; a majority of these symptoms being neurological.[13] Interestingly, there was a statistical relationship between this and later minor CNS symptoms.

Newer data from DAN have shown that, in recreational divers with DCS, pain is only present in about half of the cases, that injuries of the spinal cord and symptoms from the lungs are quite common and that 17% had experienced extreme fatigue.[7] Fatigue has been described as a sign of subclinical pulmonary embolism, further supporting the theory that vascular gas bubbles may be an important factor in neurological DCS. If, however, the symptoms are less marked, considerable under-reporting is likely, and the second of the four definitions above perhaps provides a more accurate description.

The third definition includes both acute and chronic changes related to decompression. These may be related to acute clinical symptoms or develop sub-clinically. A recent consensus conference determined that such changes, even in individuals with few or no reported symptoms, have been found in the bones, central nervous system and the lungs of divers.[14]

The last definition is similar to the so-called 'silent bubbles' described by Behnke.[2] The term silent refers to the fact that these bubbles do not lead to acute clinical symptoms. Most will probably not regard this as DCS. However, the fact that such bubbles are present during most decompressions is similar to the situation in many infectious diseases with detectable pathological flora and few or no symptoms. The question still remains whether these bubbles have an effect on the organism. There is little information about the real incidence of long-term effects of diving, nor is there any agreement about the possible mechanisms for such effects.

Describing the possible signs and symptoms following decompression does not provide a full understanding of DCS, the presentation of which is protean in nature and severity, and the prognosis varies markedly between patients. A doctor with considerable experience in treating DCS commented that "*the signs and symptoms of DCS are more varied than the symptoms of syphilis and diabetes together*".[6]

The majority of cases of DCS can be classified as a disorder, "*a disruption of normal physical or mental functions*".[15] Probably only neurological DCS fits the dictionary definition of a disease: *"a condition of an organ, part, structure or system of the body in which there is incorrect function resulting from the effect of heredity, infection, diet or environment. A disease is a serious, active, prolonged and deep-rooted condition"*.[15]

We have proposed the term 'adverse effects of decompression' (AED) as a useful indicator of decompression stress and decompression risk.[16] This is supported by Thalman who suggested that "*minor symptoms like fatigue and transient niggles must be considered as they probably indicate a higher level of decompression stress as completely asymptomatic tables*".[17]

Decompression stress

Stress is a concept that comes from physics, describing the effect of forces against a resistance. However, in medicine and biology, biological stress, a concept developed by Hans Selye, is defined as a general pathophysiological response, where similar symptoms and signs develop in response to a variety of agents and conditions. This phenomenon is termed the 'general adaptation syndrome'.[18] The term has also been used for a long time in psychology to describe the effects on the body that indicate a strong negative psychological or physical pressure or tension exceeding the mental or behavioural resources of the individual.

Decompression acts as a stressor, and decompression stress is the effect on the organism of the physical and physiological factors accompanying decompression. Even without any acute signs and symptoms, vascular gas bubbles can be an indicator of the magnitude of stress. In view of the fact that the majority of dives lead only to minimal symptoms despite the formation of gas bubbles, a major aim in developing safer procedures would be to provide an indication of the risk for injury in a particular dive.

Vascular gas bubbles as a marker of decompression stress

It is generally acknowledged that the injuries to the organism related to decompression in diving are caused by gas bubbles, and that the 'bends' is the reaction of the body to bubble formation. The amount, duration and location of the gas phase will influence the risk of acute symptoms and the degree of injury. Following this, it is also reasonable to assume that a reduction in the gas phase will reduce the risk of injury, both acutely and in the longer term. The evolution of a gas phase within the body is outside the terms of reference for this paper and readers are referred to a useful summary.[6]

Gas bubbles are formed in the vasculature on most decompressions, as only very low levels of supersaturation in the body appear to be needed for bubble formation.[19] The evaluation of new decompression procedures is increasingly based on bubble detection in the pulmonary artery using ultrasonic techniques.[20] While it is possible that bubbles in the tissue may also play a role in neurological DCS, vascular bubbles are probably the main cause of serious symptoms from the lungs and the central nervous system.[2,21–24] Since gas bubbles may be observed by Doppler or through ultrasonic imaging in the circulation in a majority of divers, and since divers are regularly exposed to such bubbles, it is important to determine their effect, how bubble formation can be reduced and how possible harmful effects can be prevented.

At present, there is only one practical way of evaluating bubble formation, namely by monitoring bubbles in the venous system.[20] As all blood enters the lungs through the right side of the heart, it is also reasonable to assume that bubbles in the pulmonary artery might be a good indicator of the total amount of free gas in the body. Whilst the sensitivity and specificity of pulmonary artery Doppler-detected bubbles is somewhat limited in predicting clinical DCS, there is general agreement that the risk of DCS increases with increasing numbers of bubbles.[25] For air dives, DCS is always accompanied by vascular bubbles, if all monitoring sites are considered.[26] There is even stronger support for the observation that the lack of detectable pulmonary artery bubbles is associated with a low risk of DCS.[20] If no bubbles are observed following air dives, any clinical symptoms or signs seen are probably not caused by DCS.[27] However, it must be borne in mind that bubble detection is performed intermittently so that bubbles may be missed. Even if the detection of vascular bubbles has the advantage of being an objective indicator, better tools are needed as the same decompression stress may produce quite different amounts of separated gas in different individuals.

In aviators with localized joint pain from DCS, gas could be seen in peri-articular and peri-vascular tissue spaces, and there was a correlation between the occurrence of gas and pain. Ferris and Engels demonstrated in the 1940s that strain and muscular activity were correlated with joint pain in altitude DCS.[1] Local compression could reduce or remove the pain in many cases, and pain could be eliminated by occluding arterial inflow to the limb. This suggests that a diver complaining of joint pain has most likely been exposed to two types of decompression stress, namely tissue gas in and around the joint and intravascular gas in the pulmonary circulation.

Decompression risk

In order to develop safer procedures for all divers, it is important to evaluate whether one diver can have a higher risk of DCS compared to another diver. In the literature, several risk factors such as obesity, age and physical activity have been claimed to influence decompression risk.[6] It has also been claimed that differences in work load, temperature and blood flow may have a significant effect upon decompression outcome.[28] In a major study of tunnel-workers in the United Kingdom by Colvin, those who had been treated for DCS were compared to workers who had no symptoms.[29] Four per cent of the work force contributed to 50% of the treatments for DCS, with no differences in work activities between the groups. Similar findings were reported in a small study from the 1950s, whilst Walder noted similar findings in the 1940s but with a slighter higher incidence (18%) of DCS.[30,31] Considerable differences in DCS incidence were noted between different companies in the 2003 report, indicating that operational factors may also play an important role.[29] No relationships between the occurrence of DCS and pre-clinical findings were observed; the only significant factors identified being absolute pressure and the duration of exposure. This is similar to the findings of Shields et al in North Sea divers, where the depth and

duration of the dive (expressed as p√T, p in bar and T in minutes bottom time) were related to the incidence of DCS regardless of which decompression tables were used.[32]

In two separate studies, we performed the same dive (18 metres' sea water (msw) for 80 minutes) in two groups of similarly aged, well-trained military divers who underwent the same training and activities.[33,34] There was a significant difference in vascular bubble formation in the two studies, the amount of vascular bubbles in the two groups differing by a factor of approximately twenty. At present, we have no explanation for these findings. According to Lanphier et al, long, shallow or short, deep dives both have a high incidence of DCS pulmonary symptoms ('chokes').[35] The main conclusion from these studies is that, in any particular group of divers, there is a small percentage (approx 5–20%) that has a significantly higher risk for being injured than the rest of the group. These data also demonstrate that traditional pre-clinical testing of the divers will not necessarily identify those who are most susceptible.

The vascular endothelium and bubbles

The vascular endothelium plays a vital role in homeostasis and is recognized as an organ with important autocrine and paracrine functions. The endothelial cells produce a large number of both vasoconstriction and vasodilating substances, which act on the underlying vascular smooth muscle. Probably the most important endothelial-derived relaxing factor is nitric oxide (NO). NO is produced by the endothelial isoform of nitric oxide synthase (eNOS). In addition to relaxing vascular smooth muscle, NO counteracts the formation of atherosclerosis through inhibition of leukocyte adhesion and invasion, smooth muscle proliferation, platelet aggregation and inflammation.[36] Abnormalities in one or more of the pathways that ultimately regulate the availability of NO may lead to endothelial dysfunction. Endothelial dysfunction, as defined by impaired endothelial-dependent vasodilatation, has been identified as an independent risk factor and a strong prognostic marker of long-term cardiovascular morbidity and mortality in latent and manifest cardiovascular disease.[37,38]

Several studies confirm that bubbles will damage or reduce endothelial function in a dose-dependent manner.[39,40] In the cerebral circulation, they lead to injury of the blood-brain-barrier within minutes.[41,42] We hypothesise that "*the main mechanism for dysfunction or injury to the central nervous system after decompression is the effect of bubbles on the vascular endothelium.*[43]

In one of the studies mentioned above in navy divers, where a dive to 18 msw led to little bubble formation, a reduction in arterial endothelial function was observed and these divers had reduced endothelial function even before they performed the dive.[33] This may indicate that diving has a long-term effect on endothelial function, but other lifestyle effects may also be involved.

Activation of the endothelium will lead to production of so-called endothelial micro particles (EMP).[44] Such activation has been observed in a number of cardiovascular diseases and after using a heart-lung-machine, and it is not unlikely that gas bubbles may lead to such activation.[45] Madden and Laden showed that bubbles formed during decompression may interact with the endothelium resulting in a loss of integrity which results in an increased shedding of EMP into the circulation.[46]

Studies have shown that circulating activated micro-particles can reduce endothelial function, and it has been suggested that EMP may be used as a marker of endothelial stress.[47,48] The reduction in endothelial function is probably caused by a reduction in NO production.[49] Activated endothelial cells have an increased expression of adhesion molecules (VCAM, ICAM and E-selectin), and activation of C5a leads to an increased expression of such adhesion molecules after about four hours.[50] This is in agreement with our findings that the reduction in endothelial function could be observed between one and six hours after exposure to gas bubbles, and that gas bubbles lead to an increase in C5a in a dose-dependent manner.[40,51] In a recent study, increases in vascular cell adhesion molecule (VCAM) and induced cell adhesion molecule (ICAM) were observed in the blood of divers five minutes after surfacing, persisting for 24 hours.[46]

Heat shock proteins (HSP) are formed in the body when the organism is exposed to stressors such as hyper- or hypoxia, heat, cold, exercise and some heavy metals or drugs. HSP have important functions in controlling the folding and structure of proteins and protecting the organism from injury.[52] However, in some cases, expression of HSP may contribute to injury. Saturation divers are exposed to considerable stress (e.g., hyperoxia, hard physical work and exposure to infections), which could potentially lead to an increase in HSP over longer periods of time. Of particular interest is the exposure to bacteria, as infections are still a serious problem in saturation diving operations.[53] Certain bacteria, e.g., *Pseudomonas aerogenosa*, which is common in saturation diving, produce HSP that is strongly antigenic and may trigger a significant immune response.[54] If the bacterial flora in the diving habitats can produce such an immunological response, this would indicate that saturation diving may carry a higher risk of endothelial damage by bubbles than other types of diving. The above also raises the interesting question whether hyperoxia and the stress of the dive *prior* to decompression play an important role in determining the outcome of decompression.[55]

Prevention of injury

Traditionally the reduction of bubble formation to prevent DCS has been achieved by changing stop times during decompression. Even if the procedures used today have a low incidence of DCS, we have demonstrated theoretically and experimentally that there is still considerable room for improvement in decompression procedures by reducing

the amount of vascular gas bubbles formed.[56,57] These observations suggest a novel and more efficient way of reducing the formation of intravascular gas bubbles and hence reducing the decompression stress.

In a number of studies in rats, we have shown that the amount of vascular bubbles following a dive and the incidence of DCS can be significantly reduced by performing severe physical exercise 20 to 24 hours before the dive.[58] This effect has disappeared after 48 hours, while exercise closer to the dive has no effect. Exercise with increase in blood flow and shear stress will increase the production of NO, which also affects the properties of the endothelial surface.[59,60] We have been able to show that bubble production is increased by blocking NO and that the bubble-preventing effect of exercise can be simulated by exogenous NO.[58,61,62] The same effect of exercise in reducing bubble formation with exercise 20 hours pre-dive has also been shown in a group of divers performing a dive to 18 msw.[63] The above findings were quite surprising and have significantly changed our opinion on how bubbles are formed and how their formation may be controlled.

It is assumed that bubbles grow from so-called gas-filled bubble nuclei which are about 1μ in diameter, since *de novo* formation of bubbles requires high supersaturation pressures that do not occur in diving.[64] These nuclei are not stable in blood, but on a hydrophobic surface such bubbles will remain stable more or less indefinitely.[65] Hydrophobic areas exist on the endothelial surface in the form of caveola, where the production of NO is also localized.[66] A reduction of surface tension on such a surface will increase the number of stable nuclei.[67] We have previously shown in a pig model that there is a relationship between surface tension of serum and bubble production and that a small reduction in surface tension will increase bubble production significantly.[68] Finally, there is a significant increase in caveola and NO after exercise.[69] The effect of this could be that exercise, by increasing NO production, will lead to microbubble detachment from the endothelium, thus allowing them to be transported to the lungs by the blood and actually reduce their number available for future growth during decompression. We believe that variations in surface tension and/or NO production may be one factor that explains the large intra- and inter-personal variations in bubble formation observed in divers.

It has also been shown that an increase in low-density lipoproteins will decrease NO production.[70] Even if obesity does not seem to be related to DCS risk in Colvin's study, food preferences could have an effect.[29] This might explain why repeated exposure will reduce the risk for DCS.[2] An interesting fact about this adaptation is that it is very specific – if the depth of the dive is changed, the adaptation is lost. This raises the interesting possibility that epigenetic mechanisms might be involved.[71]

As mentioned above, blocking NO production promotes bubble production, and heavy exercise 20 hours before the dive prevented this.[61] This study was performed in rats weighing less than 280 g. When heavier animals were used (>300 g) this effect could no longer be seen. Acute heavy exercise increases blood lipids by approximately 30% immediately after exercise is finished, then, over the next hours, blood lipids are gradually reduced and this effect has disappeared after a few days.[72,73] This effect is more pronounced in the trained than the untrained rat and is also dependent on the intensity of exercise. Twenty-four hours after exercise HDL is increased.[74] This could be a mechanism to explain why exercise 24 hours before a dive protects lean but not fat animals that are NO blocked. In the lean animals, with lower lipid levels, exercise will reduce blood lipids, allowing hydrophobicity to be reduced sufficiently to allow washing out of bubbles, while the effect is not strong enough in the heavier animals with blocked NO production. This mechanism could also explain why heavy exercise shortly before decompression could increase bubble formation. If more bubble nuclei adhere, there are more nuclei available for bubble growth.

Injury by bubbles may be preventable through other mechanisms. As described above, HSP are formed in the body when the organism is exposed to a number of stressors. The protective effect is strongest from a few hours to a day after the stress episode.[52] HSP90 is involved in the production of NO.[75] We have shown in rats that increasing body temperature to 42^{O}C 24 hours before the dive, reduces mortality by 50% and that this exposure increased HSP70 but not HSP90 and eNOS.[76] Exercise will also have an effect on HSP expression: moderate exercise increased HSP70 by 2,100% 48 hours after the last exercise bout.[77,78] These studies also showed that exercise reduced apoptosis. HSP70 was also found increased in animals showing signs of DCS.[79]

The incidence of DCS has been reduced over the last 40 years, but the relative number of incidents of DCS involving the CNS has increased. While supersaturation has been a major focus in nearly all research within this field, future research should perhaps focus more on biochemical pathways to uncover the secrets of the bubbles, both in their generation and their pathophysiological effect.

Conclusions

In this review we have concentrated on vascular bubble formation, its detection and effects. We suggest that damage and/or reduction in endothelial function due to the passage of gas bubbles is a central mechanism for the development of serious decompression injury and possibly also for the long-term effects of diving. We further suggest that these effects may be both influenced and prevented. Decompression stress, as defined above, can be used to describe the risk of dysfunction and injury after decompression, but we are well aware that this is only a part of the decompression problem.

The majority of divers do not show any acute clinical signs of DCS. Although the risk of clinical symptoms increases with increasing depth and duration of exposure, only a small proportion of divers develop clinical signs in spite of significant bubble formation. At present, there are no reliable ways of identifying, prior to the dive, those individuals who account for the majority of DCS cases. As diving is performed to greater depths and for longer periods, the search for these identifying factors should be given high priority. One possible approach could be based on the observation that vascular gas bubble formation appears to be significantly influenced by prior physical exercise, and that this mechanism is related to NO production.

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Alf O. Brubakk, MD, PhD, is Director of, and Andreas Møllerløkken, PhD, is a post-doctoral researcher in the Baromedical and Environmental Physiology Group (BAREN), Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Trondheim, Norway.
Corresponding author:
Alf O Brubakk
Department of Circulation and Medical Imaging
Medical Technology Centre
7489 Trondheim
Norway
***Phone:** +47-(0)73-598904*
***Fax:** +47-(0)73-598613*
***E-mail:** <alf.o.brubakk@ntnu.no>*

Case reports

Saturation treatment in shore-based chambers for divers with deteriorating cerebro-spinal decompression sickness

Colin M Wilson, John AS Ross and Martin DJ Sayer

Key words
Recompression, saturation treatment, heliox, hyperbaric oxygen therapy, cerebro-spinal decompression sickness, divers, diving

Abstract

(Wilson CM, Ross JAS, Sayer MDJ. Saturation treatment in shore-based chambers for divers with deteriorating cerebro-spinal decompression sickness. *Diving and Hyperbaric Medicine*. 2009;39(3):170-4.)
Nearly 4% of all primary recompression treatments in Scotland employ saturation tables (helium/oxygen-oxygen/air or oxygen/air alone). These cases usually involve divers presenting at the surface who then develop deteriorating spinal cord injury with varying degrees of cerebral involvement. Treatment is delivered either through immediate saturation therapy or through conversion of failing or failed primary treatment. The basic principles and delivery protocols of saturation treatment are outlined. A case study from both types of treatment is presented to illustrate the forms of decompression sickness that may require saturation treatment and how the treatments are initiated and evolve.

Introduction

In an analysis of treatment protocols for emergency recompression of divers in shore-based chambers in Scotland, treatments based on saturation were reported for approximately 4% of all cases (range 1–6% across the treatment centres employing saturation).[1] Two forms of saturation therapy are used in Scotland: helium/oxygen-oxygen/air ('heliox') and oxygen/air ('air') saturations. The basic principle of both treatment protocols is that entering saturation extends greatly the duration of treatment at pressure in circumstances where the alternative is returning to the normobaric situation before the presenting condition has improved or even stabilised.[2] The heliox tables have the further advantage of being able to increase the depth of compression with reduced oxygen partial pressures and there may be benefits in some cases of using pressures greater than 284 kPa.[3] In addition, some hyperbaric clinicians believe there are therapeutic advantages to 'heliox' over oxygen alone, though this is supported only by anecdotal evidence. The present account outlines methods and considerations for delivering the two types of saturation treatment. Two case reports are presented that required saturation treatment, one heliox and one air. These selected examples demonstrate the types of presentation that result in saturation.

Methods

The oxygen/air saturation treatments delivered have followed the US Navy treatment table 7 (USN 7) protocols; these are outlined in detail elsewhere.[4–6] The delivery of USN 7 at Dunstaffnage was dependent on the continuous presence of a doctor trained in diving medicine and a team of 4–6 external chamber operators. USN 7s were run with only one internal attendant; extra attendants were locked in and out when necessary using standard air decompression schedules. Saturation treatment was not embarked upon if there were too few external operators available to support the treatment or where there were too many internal attendants already inside the chamber.

The helium/oxygen-oxygen/air saturation treatments began with an initial compression to 284 kPa (18 metres' sea water, msw) on air. Where there was no response from the patient, the doctor in charge would complete any medical interventions before further compression on 2% O_2 in He to the depth of relief (ranging from 405 to 608 kPa, 30 to 50 msw). The 2/98 O_2/He mix was used for pressurisation only; the oxygen content of the therapeutic gases delivered ppO_2 levels of 2.0 bar or less and was determined by the depth of relief; chamber atmosphere oxygen levels were 41 kPa (0.4 bar) at storage depth and 51 kPa (0.5 bar) during the decompression. An extra nurse was locked in at this time with the added requirement of an additional nurse externally for logistic support. Each 24 h period required two life support technicians externally. A minimum period of 24 h was required at the chosen storage depth to ensure saturation; during this period, treatment gas sessions were delivered following the USN 7 format (repeated cycles of four 25-minute treatment gas with 5 min air break session for 2 h, then air only for 2 h).

Decompression on the helium saturations follows the USN 7 format but allowing for entry into decompression from deeper initial storage depths. Decompression from storage depth to 253 kPa (15 msw) was at a rate of 60 $min.m^{-1}$; from 15 msw to surface was at a rate of 80 $min.m^{-1}$. Decompression was suspended between midnight and 0600 each day. Permission was obtained from the subjects of both case reports for inclusion in this account.

Case reports

CASE 1: AIR SATURATION TREATMENT FOR SPINAL DCS

A 41-year-old male with no past medical history of note, except a possible allergy to the stinging jellyfish *Cyanea capillata* (Linnaeus, 1758) for which he carried self-administrating adrenaline, was diving in the Sound of Mull on the west coast of Scotland. He reported that he had been using tables to control his decompression though there was some doubt as to the reliability of this. His dive computer became 'lost' during transfer; all depths and times reported below are, therefore, recollections of the patient who had obvious signs of confusion, backed up only in part by information from his diving companions.

The first, uneventful dive was at approximately 1800 on the Friday evening to a maximum depth of 23 msw for a total time of 25 min. The following day, after a surface interval of 19.5 h, he carried out a second dive to a maximum depth of 28 msw; after 10 min he started ascending. At approximately 20 min dive time, buoyancy control problems resulted in a rapid ascent to 6 msw. An over-correction of his buoyancy caused him to descend again to 28 msw. Gaining control of his buoyancy he ascended again, stopping at 9 msw and 6 msw for 10 min each. He reported some panic during these stops because of the presence of numbers of stinging jellyfish before surfacing at 1250 after a total dive time of approximately 50 min.

Following a short swim back to the dive boat he felt unwell and required assistance to exit the water. Within 10 min of surfacing he developed loss of sensation in his legs; this deteriorated quickly and he was unable to move his legs against gravity on making shore less than 10 min later. At 1320 he was seen by the local doctor who confirmed paraplegia and commenced him on high-flow oxygen although failed to establish intravenous (IV) access. He was evacuated urgently to the Dunstaffnage Hyperbaric Unit (DHU) using the Oban lifeboat with an experienced diving doctor on board.

On arrival at the DHU at 1505, he was conscious, fully orientated in time and space and demonstrated a complete paraplegia with a sensory level to touch and pin prick at his waist. Additionally there was marked weakness to flexion and extension of his arms with weakness of grip of the left hand being worse than the right. Cranial nerve examination and serial sevens were normal. Prior to recompression IV access was established and he received two litres of normal saline over the next two hours.

Recompression was started at 1630 using the Royal Navy treatment table 62 (RN 62) protocol. After three standard oxygen/air cycles and one oxygen extension at 284 kPa (18 msw) there were no discernable objective or subjective signs of improvement. Given the serious nature of his condition at this stage, the treatment was converted at 1745 to a USN 7 at 287 kPa (60 feet sea water, fsw). In addition, he was given dexamethasone 80 mg intravenously.

Within 30 minutes, he reported some return of sensation with small movements to his toes; he passed 750 ml of urine. By 2000, he was able to lift his legs to 90 degrees against gravity. At this point, the table protocol prescribes a 2 h air break; mid-way through this break, he was able to stand unsteadily unsupported with his eyes open but could not walk. Following completion of the next 2 h oxygen session (7.5 h under pressure) he had return of normal power in his arms and could just walk, though with a broad-based gait and very unsteady Romberg test.

During the next set of oxygen cycles, he started to develop a cough but on completion at 0400 (day 2; 11.5 h under pressure) he could walk slowly with more confidence, and a less broad-based gait. The 0600 assessment prior to commencement of further oxygen showed improved gait and demonstrated careful, slow heel-toe walking; he could heel-shin slide while standing on one leg. Unfortunately at this stage (0615), oxygen breathing had to be stopped because of pronounced coughing and retrosternal chest pain. He had received over 1650 units of pulmonary toxic dosage (UPTD). Intravenous fluids were suspended having received 5 L and he was taking oral fluids well.

His condition remained stable on air, and the ascent from 287 kPa was commenced following 14.5 h at that depth. During the first few hours of ascent he continued to have an irritant cough and he described a number of pains in his back (which he associated with an old injury) and lower leg (attributed to a previous fracture). By 1700 (day 2, 24.5 h under pressure) he was at 200 kPa (32 fsw); his condition was essentially unchanged, he described his head feeling less clear and simple mental arithmetic challenges showed some impairment. He was given a further dose of dexamethasone and, as his chest symptoms had lessened, he was recommenced on oxygen-breathing cycles. These were continued until 2300 (day 2) but suspended overnight to permit sleep. The following morning (day 3), after a good sleep, neurological examination was unchanged, and he surfaced at 1930 (day 3; 51 h under pressure) and was transferred to the Oban hospital for post-recompression monitoring.

During his hospital stay, he underwent psychometric examination in which he showed major loss of semantic memory (vocabulary), and was slightly confabulatory, with perseveration that raised suspicion of a recent compromise of the cerebral circulation. He was discharged on day seven with normal motor power, slight ataxia on walking, a small area of sensory loss on his right foot and no ongoing discomfort or pain. He was advised not to dive again, which we believe he has adhered to.

CASE 2: HELIUM SATURATION TREATMENT FOR SPINAL DCS

A 70-year-old male carried out a shore dive, his first dive for 14 weeks, on the Scottish east coast. This was the deepest and longest of ten dives since having an aortic valve replacement 10 months previously, for which he was on warfarin. The dive commenced at 1448 to a maximum depth of 26.5 msw with a total dive time of 36 min including a one-minute stop at 6 msw. Within 10 min of surfacing and while on the way back to his car, he developed upper back discomfort with bilateral leg weakness and numbness from the thighs distally. He no longer had sufficient strength in his legs and collapsed to the ground with uncontrolled leg jerking. 100% oxygen was commenced and the local emergency services were contacted. Transfer to the Aberdeen Hyperbaric Medical Unit was organised by helicopter with the patient arriving at 1800. During transfer he was described as having weakness of all limbs, though he had some subjective improvement on oxygen.

On admission, examination demonstrated mild upper limb ataxia, bilateral up-going plantar reflexes, absent abdominal reflexes, paraesthesia over the feet, a wide-based, ataxic gait, and a blood pressure of 190/110. All blood haematology and biochemistry were normal, and his INR level was 2.9 (daily target range 2.5–3.0). He was able to pass urine. Upward progression of the paraesthesia in his legs was noted prior to recompression. Subsequently it became known that he had a history of spinal shock following a rugby accident in his 20s.

Intravenous fluids were commenced and he was compressed at 1905 to 284 kPa (18 msw) using the USN 6 protocol. Continued hypertension was noted while under pressure and he became unable to pass urine but declined urinary catheterisation. With the IV fluid resuscitation continuing, his urinary retention progressed, precipitating and then aggravating myoclonic jerking of his legs. This settled when urinary catheterisation was finally permitted but complicated neurological assessment regarding the progression of decompression illness to the extent that it was not really possible. At a pressure of 192 kPa (9 msw) it was again difficult to assess his neurological progress. Although there had been some improvement, walking was with unsteady gait and paraesthesia was present from his thighs distally. He surfaced following a fully extended USN 6 treatment at 0225, at which point examination showed his condition to be about the same as he had been at 18 msw prior to going into retention and worse than prior to recompression. He was returned to the wards for monitoring with a further recompression planned for later that day.

At 0630 (day 2) he was reported to have had a sudden deterioration in his condition, with nausea, and examination found him to have a sensory level at his xiphisternum (T4/5), unable to sit with hip extension power grade three, and finger-nose ataxia . At 0900, he was recompressed on a Comex 30 protocol. Soon after starting 50% oxygen in helium at 405 kPa (30 msw) the patient reported that the deterioration in his condition had halted. At the end of the first hour, considerable improvement in muscle power was noted by the attendant nurse but it was clear that the patient was by no means back to normal. At this stage, the Comex 30 table requires decompression to 344 kPa (24 msw). In view of the substantial and continuing improvement, however, it was decided to convert his treatment to helium saturation and he was further compressed to 507 kPa (40 msw). Treatment at that depth consisted of breathing cycles of 25 min using 35% oxygen/helium with 5 min breaks for four cycles at a time, repeating these cycles after a two-hour air break. In terms of O_2 levels between heliox treatment sessions, the target level was 41 kPa (0.4 bar) at storage depth and 51 kPa (0.5 bar) during the decompression.

While still at 40 msw (507 kPa), he developed a raised blood glucose requiring sliding scale intravenous insulin administration and he was also given low molecular weight heparin (LMWH) after discussion with his cardiac surgeon. His treatment gas cycles were suspended at midnight and recommenced first thing in the morning of day 3. At 1030 on day 3 (having had just over 24 h at 507 kPa) reassessment found him to be stable, able to stand on his own albeit with a shuffling gait. Decompression was started with continuation of the treatment breathing cycles with 50% oxygen in helium at 28 msw (385 kPa) and 100% oxygen at 18 msw (284 kPa). Decompression was completed on day 5 after 78.5 h under pressure. He could walk with an ataxic gait with normal leg power and had a negative Romberg test. He remained catheterised and it became clear on the ward that he was unable to control his anal sphincter.

He was discharged to the rehabilitation unit on day 12 with his LMWH having been changed back to warfarin following a normal transcranial Doppler assessment. He was fully mobile, being able to manage stairs well and was independent but still had some sensory loss distal to his lower abdomen. He remained catheterised and had to use rectal suppositories. Four months after discharge he was essentially back to normal apart from some reported sexual impairment with erectile dysfunction. One year after discharge, he reported being back to normal and had taken up yachting. Eight years after discharge, he reported enjoying good health, being very active, but missing scuba diving.

Discussion

The present account summarises the theoretical basis for saturation recompression treatment, outlines the methodology of delivery and gives two case studies as example of how saturation can be employed. All cases where saturation has been used in Scotland have involved patients with intractable or deteriorating neurological problems. In some cases, saturation has been preceded by serious relapse following initial treatment; all cases entering saturation will either have failed to respond to the initial stages of shorter

treatment tables or have been predicted to fail. Both types of saturation described here ('heliox' and 'air') have defused difficult clinical situations and negated subsequent relapse using well-tried and safe decompression schedules.

Whether saturation treatment is more beneficial to the final outcome than other treatments is the subject of ongoing appraisal in Scotland. Nearly all USN 7 treatments carried out have resulted in pulmonary oxygen toxicity for the patient, which may affect the optimum therapeutic effect through impaired lung function.[6] USN 7 use is considered to have little benefit *per se* apart from providing more time at depth (maintaining some bubble compression) to permit use and possible outcome of adjunctive measures. The Scottish Chamber Network treatment algorithm now has two options: (1) stabilisation using standard or extended RN 62/USN 6 treatment followed by immediate helicopter transfer on surfacing to Aberdeen (1–1.5 h transfer) for subsequent treatment, probably using heliox saturation; or (2) treatment using 30 msw (405 kPa) 50:50 heliox tables, such as the Comex 30, which have been used previously for treating spinal cord decompression injury.[7,8]

Heliox saturation treatments provide many more options to the treating centre in terms of enhanced flexibility for selecting treatment depth (added bubble compression) and duration (without concomitant oxygen toxicity). However, in these cases, as well as providing additional time at depth for adjunctive therapy, there are the possible added therapeutic benefits of helium breathing mixtures.[9–11] The use of heliox at pressure possibly causes shrinkage of nitrogen bubbles in a number of tissues, though after an initial expansion.[9,10] Heliox use may give some spinal cord function protection and cause some air bubble shrinkage in white matter compared with an initial growth using oxygen.[11] However, some mathematical models suggest that helium may prolong existence of bubbles in spinal and fatty tissues with isobaric counter-diffusion during the switch from high-pressure air to heliox.[12] Animal models imply that heliox is not beneficial in the treatment of respiratory decompression sickness.[13,14] Animal models of helium use in decompression illness, however, generally study isobaric administration of helium gas mixtures. In clinical practice, heliox combined with increased ambient pressure should limit or remove the risks of bubble growth and the treatment has been described as beneficial in man.[15] Oxygen acts as a vasoconstrictor and whereas the RN 62/USN 6 treatments use 2.8 bar ppO_2 (284 kPa) at the maximum treatment pressure, heliox treatments employ ppO_2 levels of 2.0 bar or less at absolute pressures of 4 bar (405 kPa) or more. Perfusion-dependent nitrogen elimination decreases secondary to vasoconstriction induced by increasing oxygen pressures.[16]

Saturation treatments should only be considered where the treating facilities are adequate and the patient has significant neurological injury.[5] Vestibular DCI may not be an indication, for example, but in cases of severe spinal cord illness where deterioration is continuing at the key treatment depth, saturation provides an additional option. However, saturation treatments do carry risks to both the patient and the internal attendant that need to be balanced against the potential outcome. By its very nature, saturation prevents accelerated bail-out procedures that may be necessary in case of events such as external fire or patient mortality. If compression to depths of greater than 18 msw is thought necessary, however, the use of heliox avoids effects such as nitrogen narcosis and pulmonary oxygen toxicity in attendants while they attempt to conduct the complex care necessary for a seriously injured diver. The internal attendant(s) should be well hydrated at all times, encouraged to exercise/walk frequently during the treatment and adhere to some form of post-treatment health observation procedure. The patient will have elevated risks of deep vein thrombosis, pulmonary embolism and stress peptic ulceration. Para-/quadriplegic patients will require frequent turning; some psychological effects may be evident in patients that are becoming more aware of their condition and likely outcome in addition to contending with physical impairments such as bladder and bowel incontinence. In addition to maintaining maximum carbon dioxide levels in the chamber to below 0.5% surface equivalent (0.5 kPa), atmospheric oxygen depletion can become important at shallower depths in the treatment and must be monitored and adjusted accordingly.

Although some studies have described the need for additional compression treatment following saturation,[13] in most cases completion of a saturation table will conclude the compression phase of recompression therapy. Where saturation delivers an improved outcome, the treatment cost must be measured against the alternative long-term expenditure on patients with significantly impaired cerebro-spinal outcome.

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Colin M Wilson MB, ChB, FRCA, is the Medical Director of the Dunstaffnage Hyperbaric Unit near Oban. He is also a GP anaesthetist at the Lorn Medical Centre and the Lorn and the Islands General District Hospital, Oban. He is currently vice-chair of the British Hyperbaric Association.
John AS Ross MB, ChB, PhD, FRCA, Honorary MFOM is a Senior Lecturer in the Department of Environmental and Occupational Medicine, University of Aberdeen Medical School, Foresterhill, Aberdeen. He is also an Honorary Consultant in Hyperbaric Medicine for NHS Grampian and the Medical Director of the Scottish Recompression Registration Service.
Martin DJ Sayer BSc, PhD, FSUT is head of the Dunstaffnage Hyperbaric Unit hosted at the National Facility for Scientific Diving (NFSD) at the Scottish Association for Marine Science laboratories near Oban in Argyll, Scotland. He is also head of the NFSD, technical advisor for the Scottish Recompression Registration Service and technical representative on the committee of the British Hyperbaric Association.
Corresponding author:
Dr CM Wilson, Lorn Medical Centre, Soroba Road
Oban, Argyll PA34 4HE
Scotland
***Phone:** +44-(0)1631-563175*
***E-mail:** <colinwilson@tiscali.co.uk>*

Severe Irukandji-like jellyfish stings in Thai waters

Peter J Fenner and John Lippmann

Key words
Envenomation, jellyfish, narine animals, case reports

Abstract

(Fenner PJ, Lippmann J. Severe Irukandji-like jellyfish stings in Thai waters. *Diving and Hyperbaric Medicine*. 2009;39(3):175-7.)
Over recent years, there have been more widely-reported sightings of chirodropids and carybdeids in Thailand. There has also been an increased awareness and documentation of fatal and severe non-fatal jellyfish stings occurring in Thai waters. Although the victims are usually swimming or wading in shallow water, divers are also at risk. Despite generally wearing some protective coverings while diving in the tropics, parts of a diver's body often remain exposed, and divers can and do sustain severe and/or life-threatening jellyfish stings. In December 2007 and January 2008, two serious cases of envenomation in divers in Thailand were reported to Divers Alert Network Asia-Pacific (DAN AP). Both of these victims displayed some typical symptoms of an Irukandji-like syndrome. Similar to Australia, appropriate measures need to be taken by the Thai authorities to warn locals and tourists alike of the possible presence of dangerous jellyfish, and suitable prevention and management strategies need to be established and implemented to minimise morbidity and mortality.

Introduction

Cubozoans are cuboid jellyfish (box jellyfish) consisting of two main groups (Classes):

- Chirodropids – box jellyfish with multiple tentacles arising from each of the four lower corners of the bell. Their deadly venom has caused human fatalities throughout tropical waters worldwide.[1–5]
- Carybdeids – box jellyfish with just one tentacle arising from each corner; may measure just a few millimetres to 250 mm bell size. Many of these species can produce symptoms of the Irukandji syndrome (see below).

Various chirodropids occur throughout the tropical waters of the Indo-Pacific Ocean: westwards to the Maldive Islands; eastwards through southern India, Myanmar, Indonesia, the Malaysian archipelago (east and west coasts), Gulf of Thailand, Cambodia, Vietnam, Brunei, Sarawak, Sabah, the Philippines and Solomon Islands; and northwards from the Tropic of Capricorn in Australia to Okinawa, Japan.[4,6] Chirodropids appear mainly in the summer months in their northern and southern geographical regions – often during the local rainy cyclonic or monsoonal area. They are most commonly found around sandy beaches close to mangrove areas. Their season is longest at the equator, where they may be present throughout the year, to just summer months as they extend down further towards the tropics.[3]

Serious and sometimes fatal jellyfish stings have always occurred in the Indo-Pacific, including Thailand.[7,8] The greatest risk is to the indigenous people whose existence includes fishing and gathering marine animals from the surrounding seas as part of their staple diet. Regularly facing dangers such as venomous snakes and marine animals is inherent in their day-to-day life, and a fatalistic approach is normal. Such deaths are not publicised and relatively unknown and are usually only discovered after diligent local research.

Carybdeids are also present in these areas and are responsible for serious envenomation, often resembling an Irukandji, or Irukandji-like syndrome. The basic Irukandji syndrome symptoms include severe low back pain, generalised muscle cramps, vomiting, sweating, anxiety, headaches and palpitations. A severe Irukandji-syndrome also occurs with extreme hypertension and/or toxic heart failure. These symptoms, although somewhat bizarre, are accountable – Irukandji venom produces potent sodium agonist catecholamines, particularly noradrenalin, from the synapses of the autonomic nervous system.[9] Less commonly, severe autonomic dysfunction and/or distressing neuropathic pain particularly in the jaw and the lower legs may be experienced.[10,11]

Early reports of the Irukandji syndrome in divers came from north Queensland in Australia.[12] However, since then, DAN AP has received correspondence from divers who have suffered Irukandji-like symptoms after diving in Thailand, Indonesia, and the Philippines. In December 2007 and January 2008, two serious cases of envenomation, both occurring in Thailand, were reported to DAN AP, both cases displaying some of the more serious characteristics of an Irukandji-like syndrome. Both divers gave permission for their cases to be reported.

CASE 1

The first case involved a 35-year-old experienced dive instructor, diving off the island of Koh Tao. At the safety stop, he hung his left arm over the mooring line to support his camera. He then surfaced, boarded the boat, pulled down his wetsuit top, and felt a stinging sensation like a cigarette burn on his inner elbow left arm. Here there was a small, raised red mark. One to two minutes later, the pain moved

up his arm and he had "waves of a strange sensation" across his upper and lower back. He describes "*the wind blowing across my shoulders and being incredibly sensitive to it to the point of pain*". Two to three minutes after the initial sting, the pain radiated to his lower back, becoming more intense and then spreading down his legs. He became weak in the legs, having to kneel down, and then had difficulty breathing and started to get severe chest pain. By 3–5 minutes the breathing difficulties increased and the pain became "*unbearable*" – he was unable to talk, his body felt "*heavy*" and he was unable to move.

Forty-five to 60 minutes later he arrived at a 'treatment clinic'. He was able to talk but still had moderate chest pain and lower back pain. Staff felt and heard an irregular heart beat and so he was evacuated to a hospital. During the trip he had an altered state of consciousness, waves of pain and difficulty breathing; later he was unable to remember much in this period. On arrival at the hospital emergency ward, the examining doctors suspected a heart attack due to chest pain and irregular heart beat shown on ECG. Blood tests showed no enzyme rise suggesting infarction, but high levels of creatinine, previously reported in some Irukandji, or Irukandji-like stings, suggesting some cardiac stress.[10] Treating doctors felt it was a severe allergic reaction to the envenomation. Treatment consisted of IV 5% glucose and 'analgesia' (unknown agent). The next day, he felt better, with reduced chest and muscle pain but he now had jaw pains (attributed to clenching his teeth the previous day).

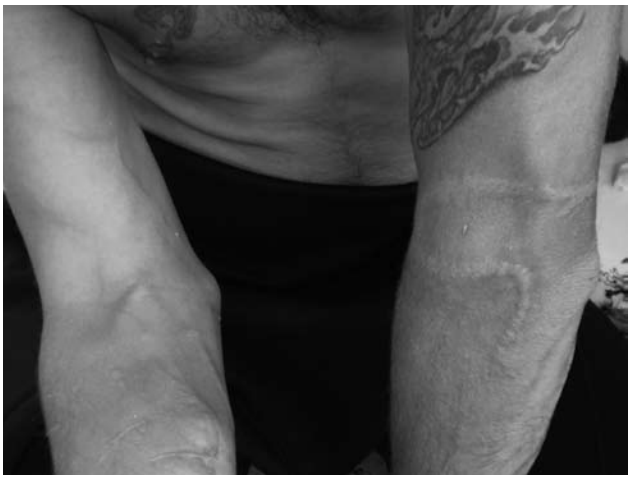
After two months, he continued to experience shooting pains, had altered sensation on his shins and tired very easily, needing much more sleep than normal. Even a year later, he still experienced recurring leg pain with altered sensation on his shins, chest discomfort and general exhaustion.

CASE 2

The second case involved a very fit 40-year-old British tourist diver who was diving near Pattaya. He was wearing a sleeveless suit without hood. While ascending from the dive up an ascent line, he suddenly felt a sharp pain on the back of his head. Reaching back, he felt a tentacle which became caught in the current and wrapped around one arm, and then the other. He described the pain as burning and very severe, scoring it at 10/10 on the pain scale. The tentacle was around 70 cm long, a brownish appearance with tinges of purple and with white spots. The victim immediately went to the surface and was helped onto the boat where staff promptly poured vinegar over the wound area and removed the remaining traces of tentacle.

He soon became nauseated, began to vomit and had severe epigastric abdominal cramps, the pain again scored at 10/10. He began to shiver, developed a bad headache, felt very dizzy, experienced tightness across the chest and became dyspnoeic, followed by a brief period of unconsciousness. He was placed on oxygen but continued to suffer from vomiting, severe abdominal cramps and arm and head pain while he was being rushed to hospital by boat and then bus. He described his symptoms as "coming in waves"; all typical of the Irukandji syndrome.

Figure 1
The arms of Case 2, with an obvious tentacle mark over the left antecubital fossa; the offending tentacle is likely to have come from a large carybdeid



On admission to hospital some three hours after being stung, he was hypertensive, still complaining of epigastric abdominal cramps. There were spiral erythematous marks with surrounding inflamed painful skin lesions over both arms and scalp. (Figure 1). After analgesia and anti-inflammatory medication (unknown agent), the head and arm pain decreased but he still had abdominal colic. He was discharged after 18 hours and returned to his hotel room at noon. However by 1600, the severe abdominal cramps returned; he vomited blood and returned to hospital, where hyoscine butylbromide 20 mg iv; metoclopramide 20 mg iv; pethidine 50 mg iv; esomeprazole 40 mg iv 12 hourly; cephalexin 500 mg q.d.s.; fexofenidine 60 mg b.d. and betamethazone-N cream were administered. He was discharged the following day and made a slow but steady recovery over a period of several weeks.

Discussion

These stings, although similar to the "basic" Irukandji envenomation, had further symptoms that have been reported and are not "the usual" Irukandji syndrome (author, PJF database of case studies). Case 1 had severe neuropathic pains (autonomic neurotoxicity).[13] Neuropathic pains are often helped by medication such as pregabalin, and this would be worth consideration to help reduce or prevent this very unpleasant feeling.[14] Jaw pain and burning leg pains have been described in severe Irukandji-like stings and are neuropathic in origin.[11]

News of fatalities and severe stings appears sometimes to be suppressed by tourism bodies and operators in the more

underdeveloped countries, concerned about the impact on their businesses and the local economy. Also, unlike Australia's comprehensive world news coverage, any reports of fatal jellyfish stings are far less likely to be publicised, unless the fatality involves an overseas tourist in which case foreign media may become involved.

However, in more recent times, when foreign tourists receive severe stings, especially if they are fatal, with worldwide media via the internet and similar facilities, the facts are rapidly transmitted worldwide. In addition, DAN AP has actively been canvassing its members and others in the diving community, to notify any serious stings and to send photographs of jellyfish seen during dives in tropical areas. The rapidly increasing publicity occurs much to the discomfort of tourism and government officials who are suddenly presented with events of which they had little or no previous knowledge. Recently in Thailand, there have been ongoing crisis talks with government officials, tourism chiefs, and Australian jellyfish experts, trying to deal with the publicity of a spate of deaths and severe stings from jellyfish and, hopefully, to establish management systems to try to prevent stings and better manage those that do occur in the future.

Conclusions

Both chirodropids and carybdeids are present in Thai waters; as such, severe and sometimes fatal stings do occur. As most divers do not wear full body protection while diving in the tropics, they, like other water users, are exposed to the risk of serious envenomation. Stings and even fatalities will never be prevented. However, the old maxim, 'prevention is better than cure', means that tourists must be made aware of the danger and alternatives made available to them, as is done in Australia. Honest and accurate educational material needs to be freely available and provided by all tourism agencies arranging holidays in Thailand and all countries in the Indo-Pacific region where the problem exists. Similar educational materials should be freely available at airports and resorts. Beaches should have restricted access with walkways leading down to them and signs warning of the possibility of dangerous jellyfish should be displayed in all languages of tourists using that resort area and/or translation easily available by digital access; the language should include phonetic. Vinegar should be made freely available on all beaches and provision of stinger-resistant nets and suitably trained lifeguards will greatly reduce the possibility of stings. For areas where nets are unavailable, swimming pools will make excellent substitutes. If people insist on swimming or entering the water in high-risk areas for snorkelling, diving, water skiing and other in-water activities, lifeguards and the provision of protective clothing by tourism operators should be considered mandatory. Such measures would greatly reduce the possibility of a fatality and are unlikely to detract from tourism, indeed they may well enhance it due to the safety measures provided.

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Peter J Fenner, AM, MD (London), FACTM, FRCGP, is Associate Professor, School of Public Health, Tropical Medicine and Rehabilitation Sciences, James Cook University, Queensland.
John Lippmann, OAM, BSc, DipEd, is Executive Director of the Divers Alert Network Asia-Pacific.

Corresponding author:
Peter Fenner
Travel Medicine Mackay,
PO Box 3080, North Mackay
Queenland 4740, Australia.
Phone: *+61-(0)7-4957-4724*
Fax: *+61-(0)7-531241*
E-mail: *<pjf@occupationalhealthmackay.com.au>*

Letters to the Editor

Online literature database for diving and hyperbaric medicine

Many colleagues already know about the 'Rubicon Foundation' database project (<http://archive.rubicon-foundation.org>), a great project that provides easy access to a vast amount of relevant diving medicine literature from many sources, including articles from the *SPUMS Journal* up to 2003. This letter is to inform SPUMS members about a long-standing project that is a nice addition to this.

Because of legal restrictions, it is not easy to obtain complete articles for free without disregarding copyright. The EUBS has solved this problem for posters and presentations presented at the EUBS annual scientific meetings by allowing the German Society for Diving and Hyperbaric Medicine (GTÜM) to incorporate presentations into their literature database *GTUEMLIT* and to provide access through the members-only area of the EUBS homepage.

GTUEMLIT also provides information about the literature in diving medicine, hyperbaric oxygen therapy, and submarine medicine which can be found in large databases like MEDLINE, EMBASE and others. GTÜM employs a documentation specialist to keep the database up to date. In addition to these well-known sources, we have also included the so-called 'grey literature' – that not listed in the databases mentioned above. For German-speaking colleagues, we have included all articles published in the GTÜM periodical *Caisson*. In total, over 34,000 publications are now included. Also included is a search engine and all articles can be exported as text file (rtf) or xml file. In many cases these export formats allow import of publication data into one's own literature database due to 'medline compatibility'.

Until now, *GTUEMLIT* was only accessible for members of GTÜM and EUBS. Because of this restriction, it is legally possible to provide access to all articles in full length where copyright is with EUBS or GTÜM. Every article published in *Caisson* or EUBS ASM proceedings can be downloaded as a PDF file in the original format including all tables, graphs, and pictures.

We believe there is enough room in the WorldWideWeb for two diving medicine database projects with different focuses and advantages – the Rubicon Foundation Archive and *GTUEMLIT*. Our idea is to open *GTUEMLIT* to SPUMS members. SPUMS Executive Committee members recently were provided with access to our database to get a personal impression of how it works.

If SPUMS is interested, we would need to agree that:

- SPUMS will provide us with digital data of articles published in *SPUMS Journal* / *Diving and Hyperbaric Medicine* to integrate these data into *GTUEMLIT* and
- GTÜM in return provides *GTUEMLIT* access to all SPUMS members.

For copyright reasons, access to the *GTUEMLIT* database will be restricted to members of EUBS, GTÜM and SPUMS only. There will be no additional costs for SPUMS members, this will be covered by GTÜM. The GTÜM Executive Committee hopes you like our project and that SPUMS will join us in the near future!

Dr. med. Wilhelm Welslau, President GTÜM
Seeboeckgasse 17, A-1160 Wien
E-mail: *<welslau@gmx.at>*

Reply:

This matter was considered at the SPUMS Executive Committee meeting in May 2009, and received unanimous support. Since then, Professor Weslau has been provided with volumes 1–30 of the *SPUMS Journal* (to year 2000) and this will be built on further over coming months. This service for SPUMS members will become available hopefully early in 2010 via a secure link in the members-only section of the new Society website once this is launched. On behalf of SPUMS members, I would like to express our gratitude to the GTÜM for this generous offer.

Michael Davis
Editor, Diving and Hyperbaric Medicine

Key words
Data, underwater medicine, hyperbaric research, writing – medical, medical society, letters (to the Editor)

Deaths from breath-hold diving

Whilst admiring the scientific approach and methodology revealed in Schagatay's paper on competitive apnoea diving,[1] I am of sufficient age to remember the earlier days of recreational breath-hold and scuba diving when the diving magazines reported each fresh record depth achieved [*Editor's note: They still do!*]. Then sanity prevailed and there was an agreement that such efforts could be fatal and should not be publicised, as such only encouraged others to try to exceed the achieved depths.

In this paper, the first sentence of the abstract reads "*Ever since the first deep diving competitions were organised, there has been debate about when the ultimate limits of the human apnoeic performance will be reached*". As the endpoint, in the absence of extremely efficient back-up services, is death, there is argument in favour of the prohibition of such competitions and for research to be

limited to strictly controlled conditions. This is particularly important as swimming activity can continue for a time after consciousness has been lost, a fact noted in swimming pool fatalities.

Having read so many reports of the deaths of breath-hold swimmers in swimming pools, and of highly experienced spear fishermen in the sea and known one who survived with marked loss of cerebral function, I believe that physiologists have a social duty to ensure their efforts are not viewed as encouraging public competition in this activity. Although this information has value in increasing our present imperfect understanding of the complex physiology involved, there should also be a recognition of the unintended flow-on effects of such research. While this paper records the results of carefully supervised dives it may be viewed as justification by others for their efforts to become successful in such competitions but who practice, for financial reasons, without the necessary safety support.

Reference

1 Schagatay E. Predicting performance in competitive apnoea diving. *Diving and Hyperbaric Medicine*. 2009;39(2):88-99.

Douglas G Walker, principal researcher for Project Stickybeak since its inception in 1972, is now retired from medical practice but continues an active interest in the investigation and reporting of diving fatalities.
E-mail: *<diverhealth@hotmail.com*

Key words
Breath-hold diving, freediving, safety, deaths, writing – medical, letters (to the Editor)

Reply: Increased reporting improves safety

Freediving is a growing recreational and competitive sport. My answer to Dr Walker's concern that reporting on competitive freediving may encourage others to try this on their own is that only by increasing the general knowledge of the dangers of the sport will we be able to avoid them. Having taught freediving to children for nearly three decades, I am aware of the possible risks and how to avoid them.

Any responsible person can freedive without the risk of drowning by abiding to two simple rules of freediving.

- Never dive alone.
- Never hyperventilate before diving.

For those interested in learning how to do it properly, there are many diving clubs and schools teaching freediving at basic and advanced levels.

However, when freediving is taken to its extremes in competitions, it does, as do several other sports, include calculated risks and necessitates good backup safety systems to deal with them. For the past six years, I have worked with the world elite of freediving or "Apnea" in physiological studies at competitions. In safety discussions with the athletes, I have been impressed by the concern taken and the methods developed by these divers to avoid injury. There have been no accidents in organized competitions during that time, and much has been learned about safety from these elite divers, which can contribute to improved safety in recreational diving. My recent review of the physiological limits of static apnoea, however, did not focus on safety aspects, as several other recent papers have dealt with this.[1]

I respect and share Dr Walker's desire to avoid injury and fatalities, but do not understand how this could be achieved by not writing about the physiology of freediving. Instead, should we not increase the reporting and improve common knowledge? Dr Walker's comments basically imply that we should not write about this sport at all, as it may be dangerous to uninformed people. For me, this is a contradiction; by understanding and reporting the risks we may instead avoid them. I do not believe freediving will go away if we as physiologists and physicians decide not to report and study it.

Describing the achievements of elite apneists with depth records now beyond 100 msw and breath-hold times over 10 minutes' duration, I believe will not encourage individuals to try this for themselves, as the records achieved are so clearly beyond normal performance. We still report Everest climbs without including the warning "*don´t try this on your own*", even though high altitude climbing probably involves a far greater risk than diving, breath-hold or scuba. It is obvious that this cannot be done without proper knowledge and training, and only by certain individuals and with serious safety measures taken to support their activities.

Reference

1 Pollock NW. Breath-hold diving: performance and safety. *Diving and Hyperbaric Medicine*. 2008;38(2):79-86.

Erika Schagatay, Professor
Mid Sweden University, Östersund, Sweden
E-mail: <Erika.Schagatay@miun.se>

Key words
Breath-hold diving, freediving, safety, deaths, writing – medical, letters (to the Editor)

Book review

Textbook of hyperbaric medicine, 5th edition

KK Jain, editor

Hardcover, 578 pages
ISBN 978-0-88937-361-7
Hogrefe & Huber; 2009
Available direct from publisher <www.hogrefe.com>
Price US $ 199.00 / € 143.95

This edition is slightly longer than the last one. Eighteen specialists are involved in 14 chapters, the remainder being written by Jain. Many diseases mentioned are not recommended for hyperbaric oxygenation (HBO) by European authorities, whilst others that are accepted are omitted. I would have expected an extensive discussion of internationally recommended HBO indications. A summary of indications (Table 8.2) lists some contradictory and potentially dangerous indications (e.g., lung diseases). An 'approved' ECHM indication list on page 455 is incorrect.

The chapter on physical, physiological and biochemical aspects of HBO gives very basic information, focussing on details of neurological disorders. The effects of diving and high pressure on the human body should not be learned out of this book, with errors in the text on diving-related problems and treatment tables for diving accidents, misinterpretation of references and unclear practical treatment guidelines for diving accidents. A very short chapter on exercise under HBO conditions could be included elsewhere, as could the hypoxia chapter (again with a neurological focus) because the content is at least partly repeated elsewhere in the text.

Jain describes the biochemical mechanisms of pulmonary and central nervous system oxygen toxicity covering the broad range of modern expert evidence about free radicals, HBO-induced generation of reactive oxygen species, and mechanisms of oxidative metabolic changes and neurotransmitters as mediators of oxygen toxicity; for a hyperbaric clinician, there is not so much practical advice, though contra-indications and complications are discussed in a clear, comprehensive way. It would be great to have included some guidelines on fitness for hyperbaric staff.

Concerning drug interactions, Jain states that animal studies cannot always be applied to humans, yet the majority of references are on animal studies! If someone is interested in the experimental HBO animal bibliography, they would be updated after reading this book. The chapter about carbon monoxide poisoning gives a clear explanation about diagnosis and rationale for HBO. The various techniques of brain imaging to assess CNS lesions and neurological sequelae are described. Recent evidence (experimental and clinical) for general clinical management is mentioned. In the infections chapter, some diseases (e.g., AIDS, tuberculosis, and leprosy) are questionably listed for HBO treatment, yet some infections for which HBO is strongly recommended are not discussed in adequate depth. The possible role of HBO in chronic Lyme disease is appropriately discussed in a chapter by Drs W and C Fife, who also discuss ethical considerations of using HBO for unproven indications and informed consent for 'off-label' use. This is clinical reality for a physician involved in daily HBO treatment.

Neurological indications for the use of HBO, most of them not approved by European or USA authorities, are extensively described by Jain. Management of stroke, HBO in global cerebral ischaemia/coma, neurosurgery, multiple sclerosis, and cerebral palsy show the wide range of some specialists' opinions about HBO in the treatment of neurological disorders. These recommendations are largely based more on personal experience of the authors than on clinical evidence-based data. Cardiovascular disease, gastroenterology, endocrinology, paediatric surgery, orthopaedics, otolaryngology, obstetrics and neonatology, geriatrics, rehabilitation and sports medicine follow a similar pattern: information about accepted indications for HBO is embedded along with statements about HBO and various diseases unsupported by evidence.

There is a useful chapter on ophthalmology offering practical information for the hyperbaric clinician. Ophthalmologic contra-indications for HBO are comprehensively described and there is a good, contemporary bibliography. In a new chapter on organ transplantation, the addition of HBO to the transplant process is recommended. Camporesi reviews anaesthesia in the hyperbaric environment, and some emergency indications for HBO are discussed by Van Meter and Harch. An overview of HBO worldwide includes the USA, Japan, and "the rest of the world" (consisting of just Germany, China, and Russia)!

The book has a clear, didactic structure. It is easy to find single topics, sections have a well-arranged layout and pictures and tables are well presented. The language is easy to understand. The bibliography of about 2,000 references has been updated.

This textbook may lead to erroneous conclusions about HBO if used as a reader's sole reference on diving and hyperbaric medicine. However, it is sometimes interesting to read exotic opinions. I found many statements had a different viewpoint to those of most of my European HBO colleagues. This book provides extraordinary insights into a wide range of possible and improbable applications of HBO in medicine.

Roswitha Prohaska
Consultant for Diving and Hyperbaric Medicine, Vienna.
E-mail: *<prohaska@gmx.org>*

Key words
Hyperbaric oxygen, treatment, indication, textbook, book reviews

Continuing professional development

Continuing professional development and continuing medical education in *Diving and Hyperbaric Medicine*

Michael Bennett

Introduction

As from this issue of *Diving and Hyperbaric Medicine*, we hope to publish continuing professional development/ continuing medical education (CPD/CME) activities on a regular quarterly basis in every issue, with the questions relating to clinical cases that may be encountered. The cases will alternate between those relating to diving medicine and those to hyperbaric medicine, and will often refer to articles published in the same issue of the Journal. Each quarterly CPD activity or unit should take about an hour to complete.

Each unit will involve the candidate answering a series of questions and these will need to be posted by e-mail to a nominated CPD coordinator. At present, this has only been set up for members of the ANZCA Diving and Hyperbaric Medicine Special Interest Group, for whom the CPD coordinator will be Associate Professor Mike Bennett, <M.Bennett@unsw.edu.au>. On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. Each CPD unit will expire within 24 months of its publication to ensure that additional, more recent data has not superceded the activity.

Eventually we hope that this programme will be web-based on the Societies' websites, and will become widely available to other members of SPUMS and EUBS.

Accreditation statement

To complete a unit successfully, 80% of the questions must be answered correctly. These activities published in association with *Diving and Hyperbaric Medicine* are accredited by the Australia and New Zealand College of Anaesthetists CPD Programme for members of the ANZCA Diving and Hyperbaric Medicine Special Interest Group (SIG) under 'Learning Projects: Category 2 / Level 2: 2 Credits per hour'.

Intended audience

The intended audience consists of anaesthetists and other specialists who are members of the ANZCA SIG in Diving and Hyperbaric Medicine. However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation.

Objectives

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

Faculty disclosure

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

Do I have to pay?

All activities are free to subscribers.

How can I subscribe to the Journal?

To subscribe to DHM, you must be a current member of either the South Pacific Underwater Medical Society or the European Undersea and Baromedical Society. For membership enquiries, please visit <www.spums.org.au> or <www.eubs.org>.

We would value any suggestions or contributions from members of SPUMS and EUBS to help make this a successful educational venture.

Sample CPD activity

To get things started, read some of the following articles about diagnosis and treatment of diving injuries, then complete the following activity. Iin this exercise, each multiple choice question has a single most correct answer.

REFERENCE ARTICLES

1 Edmonds C. Diving and inner ear damage. Editorial. *SPUMS Journal*. 2004;34(1):2-4. (N.B., There is a typological error on page 3, left column, paragraph 4, first line, which should read "*The pathophysiology guides the treatment of IEBt*.")

2 Edmonds C. Inner ear barotrauma: a retrospective clinical series of 50 cases. *SPUMS Journal*. 2004;34(1):11-4.

3 Klingmann C, Plinkert PK. The inner ear and diving. *SPUMS Journal*. 2005;35(1):52-3.

4 Klingmann C, Benton P, Schellinger P, Knauth M. A safe treatment concept for divers with acute inner ear disorders. *Laryngoscope*. 2004;114(11 I):2048-50.

5 McGeoch G. Two divers with acute vertigo and loss of balance. *Diving and Hyperbaric Medicine*. 2007;37(1):40-1.
6 Mitchell S. Severity scoring in decompression illness. *SPUMS Journal*. 2005;35(4):199-205.
7 Müller PHJ, Beuster W, Huhn W, Knessl P, Roggenbach HJ, Warninghoff V, et al. The world as it is: Diving Accident Guidelines of the German Society for Diving and Hyperbaric Medicine: summary version. *Diving and Hyperbaric Medicine*. 2008;38(4):212-7.
8 Wong R, Walker M. Diagnostic dilemmas in inner ear decompression sickness. *SPUMS Journal*. 2004;34(1):5-10.

A DIVER WITH NAUSEA AND DIZZINESS

Evolving case notes 1

You are on-call in the hyperbaric unit over the weekend and get a call around lunchtime from a dive shop around 150 kilometres away down the coast. They have a boat arriving at their jetty in about ten minutes from a dive site nearby and have a sick diver on board. All they know is that he is complaining of dizziness and nausea and is unable to walk without assistance.

Question 1: Which of the following is in your initial differential diagnosis?

A. Acute sea sickness
B. Inner ear decompression illness
C. Middle ear barotrauma
D. Inner ear barotrauma
E. All of the above

The dive shop has called the local ambulance and they should arrive soon after the boat docks. They want to know what they should do for the diver as the boat comes in and they wait for the ambulance.

Question 2: What is your advice for care on the boat and the dock?

A. Give oxygen, encourage him to walk around to keep the circulation going and improve off-gassing, and give fluids orally
B. Lie flat, give oxygen and allow oral fluids if he wants them
C. Lie flat, avoid oxygen as the mask will worsen nausea, and encourage oral fluids
D. Encourage mobilisation to avoid venous stasis while dehydrated, give oxygen and fluids
E. Do any of the above to keep them busy until the ambulance arrives

Evolving case notes 2

The boat arrives and his buddy rings with more details. First dive of the day, experienced open water diver with around 100 dives. Caller noticed his buddy slow to descend with trouble clearing ears. Down successfully but not happy and they abandoned dive after 15 minutes at the planned max depth of 20 metres' sea water (msw). Slow ascent complicated by one vomit at 5 msw leading to abandonment of safety stop and a rapid ascent to surface. He was unable to stand on the boat and complained of nausea and head spinning. He was quite distressed and unhappy at first but a bit calmer now.

Question 3: What is your order of differential diagnosis now (most likely first)?

A. Inner ear decompression illness, middle ear decompression illness, inner ear barotrauma
B. Contaminated gas, inner ear barotrauma, inner ear decompression illness
C. Inner ear barotrauma, inner ear decompression illness, alternobaric vertigo
D. Inner ear decompression illness, alternobaric vertigo, inner ear barotrauma
E. Alternobaric vertigo, middle ear barotrauma, inner ear barotrauma

Evolving case notes 3

The ambulance has arrived and transported the diver to your emergency department where he is being given oxygen and intravenous fluids. He has been well on the journey – conscious, alert and orientated, but is still complaining of vertigo and nausea. You come down to assess him. It is now three hours since he came out of the water.

Question 4: Which of the following most accurately summarises your assessment procedure?

A. Complete a short history and get him into the chamber as soon as possible.
B. Take a full and detailed history followed by complete neurological examination including sharpened Romberg's and heel-toe walking.
C. Take a full and detailed history, a complete physical examination with emphasis on the audiovestibular, neurological and respiratory systems as far as possible without standing him up. Consider supine chest X-ray and emergency opinion of ENT surgeon.
D. Take a full and detailed history followed by complete neurological examination, chest X-ray with inspiratory and expiratory films, full blood count, urea electrolytes and creatinine, blood sugar. Review by neurology registrar when results are available.
E. Take a full and detailed history, a complete physical examination with emphasis on the audiovestibular, neurological and respiratory systems as far as possible without standing him up. Plan recompression to 30 metres on 50% oxygen/helium for presumed inner ear decompression illness.

Evolving case notes 4

History is consistent with the story as already relayed to you. The diver reports a sudden 'popping' in his left ear with some pain as he was forcefully clearing his ears, followed by immediate disorientation in the water. He "didn't know

which way was up" and felt nauseous. He thought it would settle but never really improved and he abandoned dive after about 15 minutes. Very short rapid ascent from about 4 msw, but exhaled all the way. Examination suggests reduced hearing acuity on the left. The tympanic membrane on the left appears mildly injected. He has rotatory nystagmus.

Question 5: Which of the following is most true of this situation?

A. He most likely has an inner ear barotrauma involving rupture of the round window and peri-lymphatic fistula, but might well have decompression illness. We must avoid recompression and treat with surface oxygen and anti-emetics only.

B. He most likely has an inner ear barotrauma involving rupture of the round window and peri-lymphatic fistula. The chance of decompression illness is remote and the most appropriate therapy is strict bed rest, avoidance of any increase in middle ear pressure and consideration by ENT team of surgical repair.

C. He most likely has neurological decompression illness with some degree of middle ear barotrauma. He should be planned for recompression with a US Navy Treatment Table 6 or similar 283 kPa (60 feet, or 18 msw) 100% oxygen table. He may need assistance to clear his left ear and compression should be slow and careful.

D. He most likely has inner ear decompression illness given these signs and a history of rapid ascent. There is some degree of middle ear barotrauma. He should be planned for recompression with a US Navy Treatment Table 6 or equivalent 283 kPa (18 msw) 100% oxygen table. He may need assistance to clear his left ear and compression should be slow and careful.

E. He most likely has an inner ear barotrauma involving rupture of the round window and peri-lymphatic fistula. The chance of decompression illness is low and he should be immediately scheduled for operative repair of the round window.

For ANZCA SIG members, send your answers to Mike Bennett at <M.Bennett@unsw.edu.au>. Other readers are welcome to work through this material but cannot be assessed at this time.

Key words
MOPS (maintenance of professional standards), scuba diving, inner ear decompression sickness, inner ear barotrauma

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SPUMS notices and news

Minutes of the SPUMS Executive Committee Meeting 15 Nov 2008 at Prince of Wales Hospital Hyperbaric Unit

Opened: 0915h

Present: M Bennett, S Squires, G Hawkins, M Davis, D Smart, G Williams and S Lockley

Apologies: C Acott and V Haller

1 Minutes of previous meeting

Minutes accepted for Executive Committee Meeting, Liamo Resort, PNG held 28 May 2008, with corrections at 6.3 regarding wording for credit card application (Proposed: Dr Bennett, Seconded: Dr Williams).

2 Matters arising from previous minutes

2.1 Discussions have begun and processes underway for a joint venture allowing EUBS and SPUMS members to access a combined database.

2.2 Copy of the current Constitution to be mailed by Dr Williams to Secretary for distribution and comment by the Committee.

2.3 Recommend date stamp all documents and give version number, so modifications can be tracked.

3 Annual Scientific Meetings

3.1 ASM Guidelines for the Convenor are to be reviewed. Action: Dr Hawkins to email to Secretary to circulate current guidelines to Committee for comment.

3.2 ASM 2009

3.2.1 Dr Smart summarised current planning. The venue is Snorkelers Cove Resort, Iririki Island, Vanuatu. The conference centre, accommodation and facilities have been visited by Dr Davis and determined to be suitable.

3.2.2 Budget discussed for 50 participants. Registration costs will depend on numbers with minimum 50 delegates required to break even.

3.2.3 Keynote speakers will be Dr Bruce Spiess and Dr Michael Taplin.

3.2.4 Letter to be sent out re: ASM registration as a minimum of 25 delegates required prior to December 2008.

3.2.5 Advertisement in DHM. Action: Dr M Bennett and Dr D Smart.

3.2.6 SPUMS to pay DHM Editor's airfare to inspect facility.

3.2.7 Penalise late registrants at earlier cut-off date (that matches cut-off date for booking payment).

3.3 ASM 2010

3.3.1 Dr Hawkins summarised planning to date. Joint meeting planned with Asia Hyperbaric Medicine. Possible locations discussed and included SE Asia: Philippines, Thailand and Malaysia.

3.3.2 Discussed in more detail Perhentian Islands in South China Sea, with website of possible location provided for review (<www.perhentian.com.my>).

3.3.3 Transport would be from Kuala Lumpur (flights and buses).

3.3.4 Options for guest speakers discussed, with two speakers proposed and yet to be formally requested.

3.4 ASM 2011 and ASM 2012

3.4.1 Possibility of combined meeting with EUBS in 2011 discussed.

3.4.2 Expressions of interest for ASM Convenor for 2011 and 2012, to be sought through advertisement on website and advertisement in next Journal. Action: Dr Bennett and Dr Hawkins to provide request for expression of interest.

4 Journal report

4.1 Good range of material has been submitted, including 38 papers for 2008, which is double previous amount.

4.2 Loss of editorial assistant has increased the workload of the Editor, including assistance with proof reading. Members of the Committee have been requested to assist in this regard. In addition, the Editor may lose the current journal e-mail address due to retirement from hospital position.

4.3 Difficulties continue in manuscripts for ASM being submitted in a timely manner and obtaining SPUMS Committee material for the Journal.

4.4 Recommendation that links with other societies (including South East Asian, German, South African, etc) be enhanced.

4.5 Recommend the following Committee decisions be addressed:

4.5.1 Medline application

4.5.2 Advertising policy guidelines to be formulated. Committee has agreed on commercial advertising to be introduced. Action: Dr Davis to draft guidelines.

4.6 Letter circulated regarding payment of Journal Editor with a draft proposal submitted for consideration.

4.7 Editor requested approval of cost for DHM computer upgrade. All outlined costs of upgrade were approved by the Committee.

5 Treasurer's report

5.1 The Committee has formally approved the issue of an initial credit card to Dr Williams. Additional credit cards will be issued to Dr Lockley and Dr Davis for SPUMS expenditures, with a limit for each card of $2,000.

5.2 EUBS/DHM payments and shipping continue to be delayed. To be monitored.

6 Education Officer's report

6.1 SPUMS Diploma: structure and format of papers, and processes involved were discussed. Projects require pre-approval. Education Officer to liaise with current Diploma candidates regarding individual progress.

6.2 Progress of candidates currently being assessed or to be awarded was summarised.

6.3 Recommend invite awarded candidates to present at ASM.

6.4 'Young Investigator Award' discussed: incentive to encourage diving/hyperbaric research to attend ASM and present research.

6.5 Accreditation of Diving Medicine courses to be undertaken by the Education Committee under direction of the Education Officer.

6.6 Further details to be forwarded for publication by the Education Officer in regard to Diploma requirements and accreditation of Diving/Hyperbaric courses (detailed in attached report).

7 Correspondence

7.1 Book prize request was approved. It was recommended that guidelines for the selection of a book prize be developed.

7.2 Letter received from the New South Wales Government Attorney General's Department outlining recommendations made by Magistrate Crews in relation to a diver death that occurred in 2008. These recommendations were taken into account in review and development of the SPUMS Guidelines on Medical Risk Assessment for Recreational Diving.

8 Other business

8.1 Formal resignation received from Dr Sharkey. Her position as Committee Member is now open. Dr Hawkins and Dr Williams will reach the end of their terms in 2009, and their positions will also be open. A call for nominations for Committee Members (2) and Treasurer will be published prior to the next AGM.

8.2 Website redevelopment presented by Dr Hawkins

8.2.1 Current cost of website is excessive in comparison to other contracts available.

8.2.2 An upgrade would be expensive as the system components are outdated.

8.2.3 Presentation by Dr Hawkins including fees, benefits, current limitations of present system.

8.2.4 Committee approved the change of provider outlined for website redevelopment.

8.3 Informit Medical On-line Publishing have requested inclusion of DHM on their database. A link on their site to Rubicon database was suggested. Action: Dr Bennett to provide response letter.

8.4 Dr Bennett discussed the inclusion of CME activities in the DHM Journal. An example of case exercises was provided to the Committee. The proposal will require a CME co-ordinator and a CME Committee to review the cases, with the development of a variety of cases. This proposal will be discussed further at the next meeting.

8.5 Conflicts of interest within the Committee were discussed and it was proposed that conflicts of interest be declared at the commencement of a meeting. A decision will then be made by the Committee in regard to whether the Committee Member should remain. This was agreed on by the Committee.

8.6 International Society of Undersea Biomedicine would request SPUMS to join. Dr Bennett to follow progress.

8.7 Efforts to improve membership were discussed and include development of a brochure and membership package. This will be an item on the agenda of the next AGM.

8.8 Request received from Hyperbaric Health for corporate membership. Discussed subscription fees for corporate membership. A policy will be developed for future corporate memberships. Committee approved corporate membership for Hyperbaric Health. Dr Hawkins abstained from this decision.

9 Next meeting

The next meeting is planned for May 2009, at Snorkelers Cove, Vanuatu.

Closed: 1315h

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Full details of the requirements **for candidates (updated October 2008)** for the SPUMS Diploma are available on the SPUMS website <www.spums.org.au>
or from the Education Officer, Assoc. Professor Smart.
E-mail: <*david.smart@dhhs.tas.gov.au*>

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

Please be advised that examinations for the ANZCA Certificate in Diving and Hyperbaric Medicine will be held as follows:

Written Exam:	Monday 9 November, 2009
Oral Exam:	Friday 27 November 2009

All eligible candidates are encouraged to sit.

For further information or to register, please contact:
Cherie Wilkinson
Phone: +61-(0)3-8517-5312
E-mail: <cwilkinson@anzca.edu.au>

EUBS European Underwater and Baromedical Society

EXECUTIVE COMMITTEE (as of September 2009)

PRESIDENT
Dr. Peter Germonpré
Centre for Hyperbaric Oxygen Therapy
Military Hospital Brussels
B-1120 Brussels, Belgium
Phone: +32-(0)2-264-4868
Fax: +32-(0)2-264-4861
E-mail: <p.germonpre@gmail.com>

VICE PRESIDENT
Prof. Costantino Balestra
Environmental & Occupational
Physiology Laboratory
Haute Ecole Paul Henri Spaak
91, Av. C. Schaller
B-1160 Auderghem, Belgium
Phone & Fax: +32-(0)2-663-0076
E-mail: <daneuben@skynet.be>

IMMEDIATE PAST PRESIDENT
Prof. Alf O. Brubakk
NTNU, Dept. Circulation & Imaging
N-7089 Trondheim, Norway
Phone: +47-(0)73-598904
Fax: +47-(0)73-597940
E-mail: <alf.o.brubakk@ntnu.no>

PAST PRESIDENT
Dr. Noemi Bitterman
Technion, Israel Institute of Technology
Technion City
Haifa 32000, Israel
Phone: +972-(0)4-829-4909
Fax: +972-(0)4-824-6631
E-mail: <noemib@tx.technion.ac.il>

HONORARY SECRETARY
Dr. Joerg Schmutz
Foundation for Hyperbaric Medicine
Kleinhuningerstrasse 177
CH-4057 Basel, Switzerland
Phone: +41-(0)61-631-3013
Fax: +41-(0)61-631-3006
E-mail: <joerg.schmutz@hin.ch>

MEMBER AT LARGE 2009
Dr. Andreas Møllerløkken
NTNU, Dept. Circulation & Imaging
N-7089 Trondheim, Norway
Phone: +47-(0)73-598907
Fax: +47-(0)73-598613
E-mail: <andreas.mollerlokken@ntnu.no>

MEMBER AT LARGE 2008
Dr. Peter Knessl
Steinechtweg 18
CH-4452 Itingen, Switzerland
Phone: +41-(0)44-716-7105
E-mail: <pknessl@bluewin.ch>

MEMBER AT LARGE 2007
Dr. Phil Bryson
DDRC, The Hyperbaric Medical Centre
Tamar Science Park, Research Way
Derriford, Plymouth
Devon, PL6 8BU, United Kingdom
Phone: +44-(0)1752-209999
Fax: +44-(0)1752-209115
E-mail: <phil.bryson@ddrc.org>

HONORARY TREASURER & MEMBERSHIP SECRETARY
Ms. Patricia Wooding
16 Burselm Avenue
Hainault, Ilford
Essex, IG6 3EH, United Kingdom
Phone & Fax: +44-(0)20-85001778
E-mail: <patriciawooding@btinternet.com>

NEWSLETTER EDITOR
Dr. Peter HJ Mueller
Dudenhofer Str. 8C
D-67346 Speyer, Germany
Phone & Fax: +49-(0)6232-686-5866
E-mail: <pete@ejuhm.de>

The EUBS website is at
www.eubs.org
Members are encouraged to log in

Annual Scientific Meeting 2010
Preliminary Announcement

Dates:14–18 September 2010

Earlier dates were not possible because of Ramadan (6 August–7 September) followed by a religious feast.
Venue: The Marmara Hotel, Istanbul, Turkey

The Registration Desk opens from 14 September 2009.

Preliminary enquiries to:
Prof. Maide Cimsit, Istanbul University
Secretary General, EUBS ASM 2010
E-mail: <mcimsit@istanbul.edu.tr>
or
Ms. Mine Cuca, Figur Congress
Ayazmaderesi Cd. Karadut Sk. No: 7
34394 Dikilitas, Istanbul, Turkiye
Phone: + 90 212 381 46 88
Fax:+ 90 212 258 60 78
Gsm: +90 533 955 34 81
E-mail: <minecuca@figur.net>

2009 ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS' UNDERWATER MEDICINE COURSE

Dates: 16 to 27 Nov 2009
Venue: HMAS PENGUIN, Sydney
Cost: AU $2,008 (including accommodation costs, at HMAS PENGUIN) or AU $705 (without accommodation)

The Medical Officer's Underwater Medicine 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 workshop covering the key components of the diving medical.

For information and application forms contact:
Mr Rajeev Karekar for Officer in Charge,
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Rd, Mosman, 2088 NSW, Australia
Phone: +61-(0)2-99600572
Fax: +61-(0)2-99604435
Email: <Rajeev.Karekar@defence.gov.au> with cc to <Sarah.Lockley@defence.gov.au>

The Hyperbaric Research Prize

The Hyperbaric Research Prize encourages the scientific advancement of hyperbaric medicine and is awarded annually whenever a suitable nominee is identified. It will recognise a scholarly published work or body of work(s) either as original research or as a significant advancement in the understanding of earlier published science. The scope of this work includes doctoral and post-doctoral dissertations. The Hyperbaric Research Prize is international in scope. However, the research must be available in English. The Hyperbaric Research Prize takes the form of commissioned art piece and US$10,000 honorarium.

For detailed information please contact:
Baromedical Research Foundation
5 Medical Park, Columbia, SC 29203, USA
Phone: +1-803-434-7101
Fax: +1-803-434-4354
E-mail: <samir.desai@palmettohealth.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

(revised March 2009)

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, including SPUMS Diploma theses, will be subject to peer review. Accepted contributions will be subject to editing.

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: <spumsj@cdhb.govt.nz>

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.

The text should be double-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. Measurements are to be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included. Abbreviations may be used once they have been shown in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is up to 3,000 words. Including more than five authors requires justification, as does more than 30 references. Case reports should not exceed 1,500 words, with a maximum of 15 references. Abstracts are required for all articles. Letters to the Editor should not exceed 500 words with a maximum of five references. Legends for figures and tables should generally be less 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 may 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 May 2007. 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 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.

Place a full stop after the journal name and at the end of the reference. Titles of books and journals should be in italics. Accuracy of the references is the responsibility of authors.

Any manuscript not complying with the above requirements will be returned to the author 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 should be available if requested.

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Full 'Instructions to authors' can be found on the EUBS and SPUMS websites (revised June 2009).

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)
010-4500-9113 (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
+1-919-684-8111
+52-5-629-9800 (America-Mexico))

LATIN AMERICA
+1-919-684-9111 (may be called collect; Spanish and Portuguese)

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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hawthorn@snap.com.au