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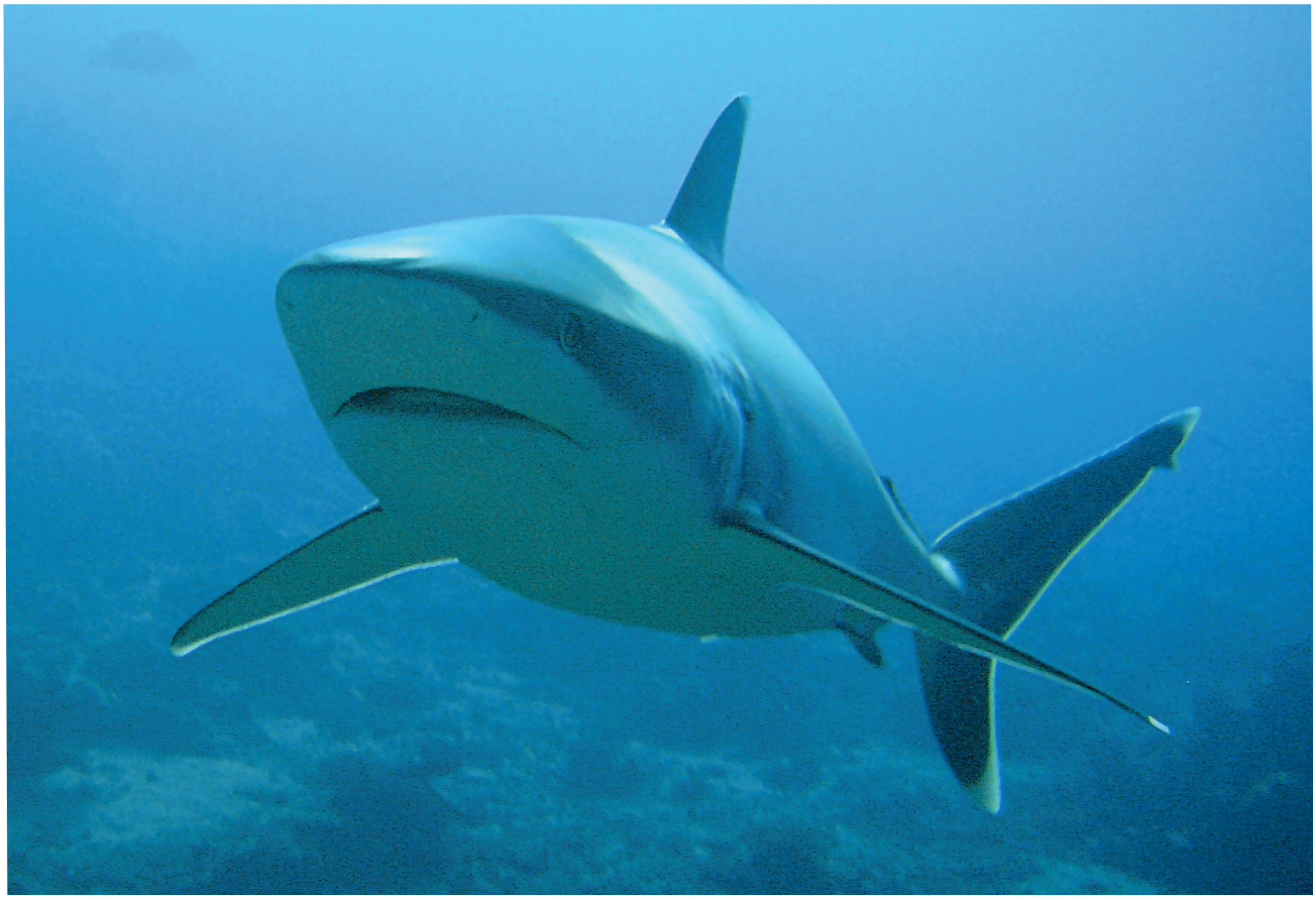

Airway management in resuscitation

CO_2 monitoring for ICU patients

Fitness to dive
- **asthma**
- **diabetes**

Lung function after scuba dives

'Tech' diving the safe way?

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PURPOSES OF THE SOCIETY

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

OFFICE HOLDERS

ADMINISTRATION

MEMBERSHIP

Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
Membership applications can be completed online at the Society's website <www.SPUMS.org.au>

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The Society's financial year is January to December, the same as the Journal year.
The 2007 subscription will be Full Members A$130.00 and Associate Members A$70.00, including GST.
There will be an additional surcharge of $8.00 for journal postage for all members living outside Australia.

The Editor's offering

This issue has a decidedly respiratory flavour to it. Management of patients on ventilators in a hyperbaric environment presents a number of challenges, summarised recently by Trytko.[1] Monitoring gas exchange during hyperbaric oxygen (HBO) therapy is not straightforward but is essential for safe delivery of HBO to these patients. Modern intensive-care-patient monitors routinely incorporate capnography to measure end-tidal carbon dioxide ($ETCO_2$). The monitor may be inside or outside the chamber, the former presenting issues of performance under pressure and electrical safety, and the latter mechanical sampling problems. Whatever device is used, correction factors must be applied to observed $ETCO_2$ values, further reducing the reliability of these observations. The device being used may not function at all under hyperbaric conditions, as occurred with one of the monitors Wolfers and Bennett wished to study in their report in this issue. Add to this our present lack of knowledge of the pathophysiological relationships of $ETCO_2$ to arterial CO_2 tension in critically ill patients under hyperbaric conditions, and we are left with a complex, poorly understood situation.

Monitoring oxygen is similarly fraught since oximetry is of limited value during HBO. Additional errors in arterial blood gas analysis may occur due to decompression of the gas sample and the observed values being outside the normal calibration range of the oxygen electrode. Accuracy of arterial CO_2 tensions is less affected by these factors but, unless analysis is immediately available, delays in obtaining results and then adjusting ventilator management may have serious repercussions for the patient. Hypercapnia may increase the likelihood of central nervous system toxicity and stress the myocardium, whilst hypocapnia during HBO may be markedly deleterious to the patient. Therefore, reliable $ETCO_2$ monitoring is important for good patient care.

Fock reports on the wellbeing of a small sample of technical divers using closed-circuit rebreathers. He adds to a growing series of small field studies using Doolette's health survey questionnaire as a less crude alternative to assessing the incidence of decompression sickness in high-risk diving situations.[2] He gives a description of 'tech' diving procedures that provides some insight for diving physicians into this growing recreational diving activity. The development of symptoms of pulmonary oxygen toxicity in three of the six divers using a PO_2 set point of 1.3 ATA (131 kPa) confirms this as a limiting factor in repetitive mixed-gas diving, and the NOAA oxygen limits should be complied with fully.

Whilst the data presented are rather limited, as is also the case with the short report on pre- and post-dive spirometry in air scuba divers by Wilson and Crockett, studies under open-water diving conditions are relatively uncommon, and it was felt that there was sufficient merit to both papers to give them space.

A resuscitation workshop focusing on airway management was conducted at the 2006 ASM. This was preceded by Chris Acott's comprehensive review of extraglottic airway devices, of which there are now a plethora on the market.

Robyn Walker provides an excellent overview of whether people with asthma should dive and, if so, how physicians might best triage and advise such candidates. Her approach is that of the experienced generalist and she ends with some clear recommendations. The Australian and New Zealand Thoracic Society had hoped that SPUMS would provide feedback on their discussion paper,[3] but to date no formal response has come from this society. Perhaps Dr Walker's recommendations should be adopted.

A major wind change is also occurring where diabetes is concerned, as readers will see from Mike Bennett's presentation at the ASM. This brings the issue into the Australian arena and he also asks what SPUMS is doing. On both these fitness-to-dive issues, SPUMS now appears out of step with most of the international diving medical community. There are now some reasonable data to work with in developing a risk-assessment-based approach to these chronic medical conditions. DAN has produced clear guidelines and SPUMS should heed Dr Bennett's call for us to work with other groups to review our position based on these new data. What do *you* think about these two issues? Is it time for a more pragmatic approach? The Committee would value your input through the letters column.

Registrations for the 2007 ASM in Tutukaka, Northland, New Zealand are slowly coming in. This will be a first-class meeting, so please get your registration in the mail soon. This particularly applies to New Zealand members – there will not be a better chance to attend a SPUMS ASM for many years to come! Northland in April is a great place for a holiday too, so combine the meeting with a decent break from work.

Michael Davis

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Front cover photo of a silver-tip shark, *Carcharhinus albimarginatus*, was taken by Dr Sherwood Smith at the Fiji ASM 2006.

Original articles

Performance of mainstream capnography under hyperbaric (243 kPa) oxygen conditions

Darren L Wolfers and Michael H Bennett

Key words
Capnography, carbon dioxide, equipment, ventilators, patient monitoring, hyperbaric oxygen

Abstract

(Wolfers DL, Bennett MH. Performance of mainstream capnography under hyperbaric (243 kPa) oxygen conditions. *Diving and Hyperbaric Medicine*. 2006; 36: 174-8.)
We evaluated the performance of the SpaceLabs Medical 90369G and 90516 capnography modules (mainstream infra-red spectroscopic capnographs) under clinical hyperbaric oxygen conditions (2.4 atmospheres absolute (243.12 kPa), FiO_2 1.0). Each module was ventilated alternately with known concentrations of carbon dioxide (CO_2) in oxygen and 100% oxygen. The input concentrations of CO_2 were varied to assess accuracy, reproducibility and stability over time. The 90516 module could not be studied as it was incapable of functioning under our conditions. The 90369G module consistently over-read but was highly predictable so that true end-tidal CO_2 (mmHg) = 0.619 x capnograph end-tidal CO_2 + 2.60 (r^2 = 1.00, P < 0.0001). The module had highly reproducible and stable results that showed no hysteresis. We conclude the 90369G capnography module is suitable for use in monitoring ventilated patients in hyperbaric practice. The correction factors are applicable only to our module, under the specific conditions of oxygen and pressure we used. We offer possible causes for the module's inaccuracy, and some putative solutions.

Introduction

There are a number of approved indications for hyperbaric oxygen therapy that involve compression of mechanically ventilated patients.[1] Capnography allows the early detection of inadvertent extubation or patient-ventilator disconnection and of hypercapnoea that may result from the change in function of mechanical ventilators known to occur with therapeutic hyperbaric pressures.[2,3] Hypercapnoea is believed to increase the risk of central nervous system (CNS) oxygen (O_2) toxicity in humans.[3] In Australia, capnography is mandatory during general anaesthesia.[4] It is also our routine practice to monitor end-tidal CO_2 in ventilated patients in our hyperbaric chamber.

Infra-red spectrography is the cheapest, most compact and most widely used of the techniques available for quantitative detection of end-tidal carbon dioxide (CO_2).[5] Side-stream (as opposed to mainstream) sampling methods are problematic under both normobaric and hyperbaric conditions.[5–8] Several authors have speculated that infra-red spectrographic capnographs may be inaccurate at therapeutic hyperbaric pressures; the only published evaluation of such a mainstream capnograph under clinical hyperbaric oxygen conditions found the capnograph gave falsely elevated readings.[9–11]

The aim of our study was to evaluate the performance of the SpaceLabs Medical capnography options 90369G and 90516 when used under clinical hyperbaric oxygen conditions. The 90369G 'add-on' module (SpaceLabs Medical, Redmond, WA, USA) is a mainstream infra-red spectrographic capnograph that may be used with both the capnograph and associated monitor placed in the chamber with the patient. In-chamber use of this module has been certified as safe by our clinical engineering department. The pressures used clinically, however, are well outside the 90369G module's operating specifications (69.7–101.3 kPa).[12] The newer 90516 module, also a mainstream infra-red spectrographic capnograph, was assessed as it is being adopted elsewhere within our institution. It has the advantage of user (not factory) recalibration.[13] Specifically, we wanted to establish the accuracy, reproducibility and stability over time of readings under clinical hyperbaric oxygen conditions, using these two modules.

The 90369G is also marketed as, and identical to, the 90367G and 90309Q add-on options and the 90515 removable module. The 90516 removable module is also marketed as, and identical to, the 90367H and 90369H add-on options (information provided by manufacturer, SpaceLabs Medical, Redmond, WA, USA).

Methods

We used customised reference beta-mix gases of various concentrations of CO_2 in O_2 with a certified analysis tolerance of +/-2% relative (Linde Gas, Yennora, NSW, Australia), to allow simulation of inspired and expired gas across a range of CO_2 concentrations. Concentrations of 1.10%, 1.66%, 2.25%, 2.75%, 3.31% and 4.29% CO_2 in O_2 were provided, delivering a pCO_2 of 20.1, 30.3, 41.0,

50.2, 60.4 and 78.3 mmHg CO_2 (1 mmHg = 0.133 kPa) respectively at our experimental conditions of 243 kPa (2.4 ATA). The reference gases were delivered to the module's mainstream sensor alternately with 100% O_2 via a custom pneumatic timer driving a flow interrupter in order to simulate a human respiratory pattern.

The 90516 capnography module could not be studied as it was incapable of functioning under our experimental conditions. The 90369G capnography module was displayed on an Ultraview 1050 (90369) Portable Bedside Monitor (SpaceLabs Medical, Redmond, WA, USA), our routine monitor. The module is capable of reporting concentration of CO_2 in both partial pressure of CO_2 (mmHg) and volume percentage of CO_2 (% CO_2); both methods of reporting were investigated throughout the experiment. The module reports both minimum inspired or baseline CO_2 – when the flow interrupter switches to 100% O_2 to simulate inspiration – and maximum or end-tidal CO_2 – when the flow interrupter delivers reference gas containing CO_2 to simulate expiration. The optional O_2 measurement cell was not used with the capnography module. The capnograph underwent calibration verification at 1.0 ATA (101.3 kPa) prior to each experimental run, as per the manufacturer's instructions.[12] The manual O_2 compensation was activated as we were using greater than 60% O_2 at all times.

All readings were taken at 2.4 ATA, with reference gases, timer/flow interrupter, capnography mainstream sensor, module and monitor in-chamber, in a compartment of our multiplace hyperbaric chamber (EBSRAY Pumps Pty Ltd, Brookvale, NSW, Australia). To ensure accurate delivery of chamber pressure of 2.4 ATA, ambient barometric pressure and temperature were recorded from a properly calibrated electronic digital barometer and thermometer, placed outside the chamber. Chamber pressure was measured on an analogue gauge (Budenberg, Sydney, Australia) with accuracy of +/- < 0.1 msw (< 1.00 kPa). Chamber temperature and relative humidity were monitored to ensure they stayed within the capnography module's operating environmental requirements.[12]

Preliminary work established the best simulated clinical measurement conditions and these were used for the experiment: respiratory rate 15 breaths per minute with an inspiratory to expiratory ratio of 1 to 3, O_2 flow of 3 $l.min^{-1}$, and CO_2 in O_2 flow of 1 $l.min^{-1}$. Ninety seconds after the introduction of a new reference gas, end-tidal CO_2 was measured in mmHg and then measured in % CO_2 a further 30 seconds later. Reference gases were all dry gases delivered at chamber temperature and readings were reported in ATPD.

Following pressurisation of the chamber to 2.4 ATA, alternating 100% O_2 and 1.10% CO_2 in O_2 were delivered to establish the accuracy of the baseline and end-tidal CO_2. These were manually recorded from the capnograph display in mmHg. Then the module was switched to report % CO_2 and the end-tidal CO_2 and chamber pressure as detected by the capnography module were recorded. This experiment was then repeated with 1.66%, 2.25%, 2.75%, 3.31% and 4.29% CO_2 in O_2 respectively.

To assess reproducibility, this procedure was repeated four times, twice with increasing reference gas CO_2 concentration and twice with decreasing CO_2 concentration. This not only gave four assessments of each input CO_2 to assess reproducibility of results but also two entire ascending then descending CO_2 runs to examine hysteresis.

To assess for stability of readings over time, an alternating CO_2 of 0 mmHg and 41.0 mmHg (at 2.4 ATA, in maximal oxygen) was delivered to the capnograph to simulate normal human respiration. This experiment was run for 90 minutes, with the baseline and end-tidal CO_2 recorded every five minutes in both mmHg and % CO_2 as well as the chamber pressure detected by the capnography module. All readings were again manually recorded from the relevant monitors.

Statistical analysis was performed using StatsDirect Statistical Software Version 1.9.8 (Iain Buchan, 2001). Accuracy data were subjected to simple linear regression and correlation analysis where appropriate. Simple descriptive statistics were used to report stability and reproducibility data. ANOVA with Tukey correction for multiple comparisons was used to detect any hysteresis in the reproducibility data. Statistical significance was accepted when $P < 0.05$.

Results

The 90369G capnograph module passed prescribed calibration verification at 1.0 ATA.[12] Chamber environmental conditions were always within the module's requirements with the exception of operating pressure.[12] Chamber and delivered-gas temperatures were 21.6–25.3 ºC throughout the experiment. Chamber pressure was within 0.52 kPa or 0.37% relative of 2.4 ATA at all times. Interestingly, the chamber pressure as detected by the 90369G capnography module was always 740 mmHg, despite the true value being 1824 mmHg (243 kPa).

Gases delivered were within the module's output range except with input of 4.29% CO_2 in O_2 when the capnograph detected end-tidal concentrations of CO_2 of 105 mmHg and 14.2%. As these values exceed the maximum reportable by the instrument this result is graphed but not included in further statistical analysis of the relationship between the actual and detected values.[12]

Baseline readings were stable at 1 mmHg regardless of the alternating concentration of CO_2 (Figure 1). The relationship between input CO_2 and end-tidal CO_2 when reported in mmHg is linear (Figure 1), with the capnograph over-reading. The correlation is highly significant ($r^2 = 1.00$, $P < 0.0001$) and linear regression shows that the true end-tidal

Figure 1
90369G capnograph accuracy (mmHg) (error bars represent +/- 1 standard deviation of the mean); dashed line – outside module's specified operating range

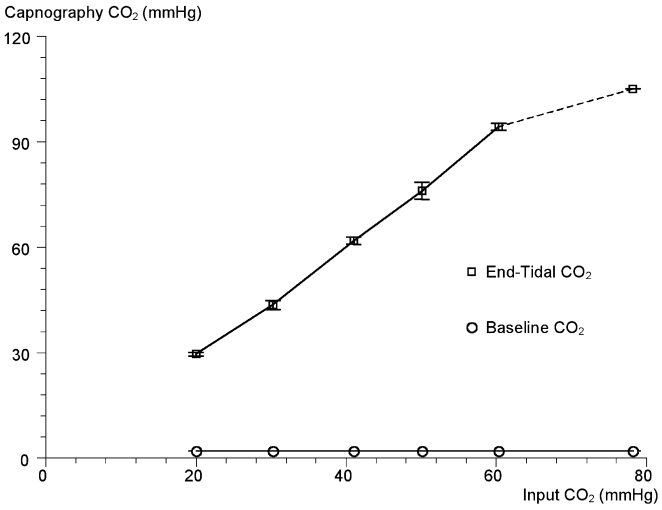
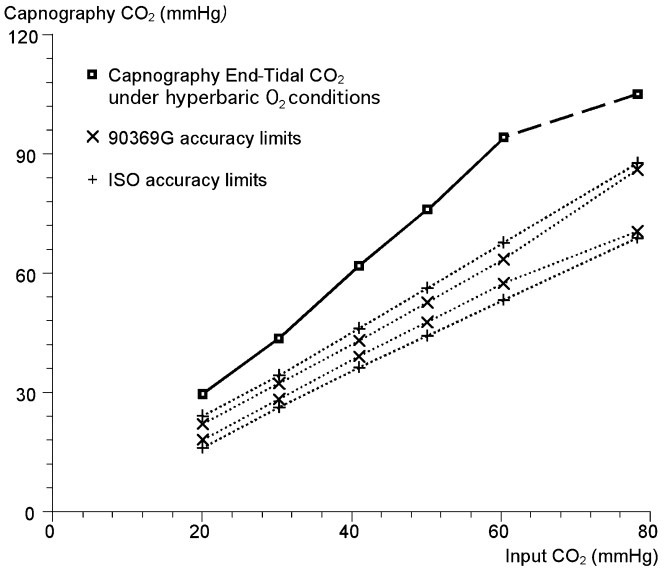


Figure 2
90369G capnograph accuracy compared with accuracy standards (dotted lines); dashed line – outside module's specified operating range



CO_2 or input CO_2 = 0.619 x capnograph end-tidal CO_2 + 2.60. There was a similar linear relationship when analysing CO_2 reported as a volume %, with the capnograph again over-reading. The correlation was highly significant (r^2 = 1.00, P < 0.0001) and linear regression shows that input % CO_2 = 0.252 x capnograph end-tidal % CO_2 + 0.146.

Results were highly reproducible on repeat testing. The baseline data showed perfect reproducibility with zero variation. The end-tidal data in mmHg showed high reproducibility with the greatest standard deviation 2.45 mmHg or 3.22% of the mean. When reporting in % CO_2 the greatest standard deviation was 0.265% absolute or 2.58% relative of the mean. ANOVA with Tukey correction for multiple comparisons indicated no hysteresis. (Maximum difference between mean ascending and descending values for each input CO_2 was 1.5 mmHg, P > 0.99 for each comparison.)

Figure 2 shows the result of the end-tidal CO_2 accuracy data in mmHg plotted against the manufacturer's limits and the ISO standard's limits for accuracy.[12,14]

The 90369G capnograph readings remained very stable when assessed over 90 minutes with 100% O_2 alternating with a single concentration of reference gas, whether reporting in mmHg or % CO_2. The baseline showed 0 mmHg absolute and 0% relative maximal drift over time. End-tidal CO_2 showed 2 mmHg CO_2 absolute maximal drift and 3.33% relative maximal drift. When end-tidal CO_2 was reported in % CO_2, absolute maximal drift was 0.6% and relative maximal drift was 7.59% over a 90-minute period.

Discussion

Both the 90369G and the 90516 modules were to be tested for suitability for use under clinical hyperbaric oxygen conditions. The 90516 module proved incapable of operating under these conditions; a reading error of the barometric pressure is the only plausible explanation.[13]

The comparison of observed values plotted against the manufacturer's limits and the ISO standard's limits for accuracy shows the 90369G module to be inaccurate under clinical hyperbaric oxygen conditions. However, our highly significant correlations between input and measured CO_2 show that corrections can be applied allowing true end-tidal CO_2 to be calculated under the conditions of this experiment (2.4 ATA, with an FiO_2 of 1.0 and the manual oxygen compensation activated). Our experiment further shows that the 90369G capnography module produces highly reproducible results with no hysteresis and very stable readings over time. Thus the SpaceLabs Medical 90369G modular mainstream infra-red spectrographic capnograph is suitable for use in the clinical hyperbaric oxygen environment.

The difference in the slope of our two end-tidal CO_2 correction equations is likely due to an error introduced through inaccurate barometric reading within the module (detecting 740 mmHg instead of 1824 mmHg) during calculation of the percentage CO_2. When this error is accounted for, the gradients of the end-tidal CO_2 equations are almost identical (input % CO_2 slope of 0.252 x 760 mmHg per 1 ATA / 740 mmHg x 2.4 ATA = 0.620 compared with input CO_2 mmHg slope of 0.619).

There are several possible explanations for the inaccuracy of the 90369G module under these conditions: problems with calibration, collision broadening due to O_2 and pressure broadening. Problems with calibration under varied atmospheric pressure are known to affect the accuracy of some infra-red spectrographic capnographs.[15,16] Whilst the capnograph passed calibration verification at 1.0 ATA, its method of calibration is unknown to us; we can only speculate that the accuracy of this calibration technique may be affected by our experimental conditions.

Collision broadening due to the presence of oxygen is known to affect the accuracy of infra-red spectrographic capnographs.[5] Molecules of O_2 and CO_2 collide causing a transfer of energy that results in a broadening of the absorption peak for CO_2 (the wavelengths where absorption of infra-red light is greatest). This causes significant under-reading of CO_2, the opposite of our experimental finding.[5] We speculate the module's manual oxygen compensation function was unable to fully compensate for the high oxygen levels in our experiment.

Pressure broadening is the broadening of the spectral absorption peaks of a gas such as CO_2 owing to an increase in the absolute pressure of the gas sample.[15] This causes a significant over-reading of CO_2.[15] Whilst the 90369G module is said to have automatic barometric pressure correction, the internal barometer was not functional under our experimental conditions.[12] Therefore, pressure broadening is highly likely to have contributed to the 90369G module's false elevation of results at 2.4 ATA. Given the likely effects of collision broadening due to oxygen and pressure broadening on the accuracy of our module, our reported correction factors should not be applied to other conditions of oxygen and pressure.

The accuracy of the 90369G module under clinical hyperbaric oxygen conditions may be improved by addressing the likely factors above. Infra-red spectrographic capnographs determine the concentration of CO_2 by comparison with a known standard, making accurate calibration essential.[5] It has been suggested that calibration should occur at each measurement pressure with a known pCO_2 and the ambient pressure manually entered into the module.[5] This will not only overcome many of the problems of calibration under varied ambient pressure, but potentially also the error due to pressure broadening. Error from collision broadening due to oxygen could also be minimised if calibration was done using oxygen as the carrier gas.[15,17] Currently the 90369G module cannot be manually calibrated so significant modifications would be required.

As an alternative, further work could be done to calculate correction equations such as ours for a large range of ambient pressures and carrier gas oxygen concentrations. Improved module barometric-pressure and oxygen sensors, accurate over the range of pressure and pO_2 found in clinical hyperbaric oxygen practice, could be incorporated. Internal module software would then complete this full-range automatic barometric pressure and oxygen compensation.

We strongly caution against applying our results directly to other capnography systems. The performance of different capnographs varies even at 1.0 ATA, whilst the relative effect of pressure/collision broadening has been shown to vary with the capnograph used.[15,18] We believe there is a case for further investigation of both our capnographs under other common hyperbaric conditions, and other available capnographs.

Acknowledgements

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Health status and diving practices of a technical diving expedition

Andrew Fock

Key words
Technical diving, safety, health surveillance, decompression, air, mixed gas, trimix, nitrox, oxygen

Abstract

(Fock A. Health status and diving practices of a technical diving expedition. *Diving and Hyperbaric Medicine*. 2006; 36: 179-85.)
The author participated in a technical diving expedition to the South China Sea primarily to dive several deep World War Two wrecks. During the expedition, diving practices and diver health were observed, and a diver health survey was completed by six of the seven divers at the end of each diving day. This survey showed a slight worsening of health scores during the first half of the expedition, which then returned to baseline levels. However, no diver reached a health score of a level (six) associated with clinical decompression sickness (DCS) in a previous study. No clinical DCS was detected or treated; however, a high level of pre-existing musculoskeletal complaints prevalent in this group made clinical diagnosis difficult for marginal symptoms. A high proportion (50%) of divers reported symptoms consistent with pulmonary and ocular oxygen toxicity. The use of closed-circuit rebreathers for 74 dives in the depth range of 50 to 70 metres' sea water, with total dive time 100.4 hours, was associated with few technical problems for a suitably trained and experienced group of technical divers.

Introduction

Technical diving has been defined as recreational diving to depths beyond recreational limits and using gases other than air and is generally recognised as a high-risk activity.[1] Originating from techniques developed by the cave-diving fraternity, technical diving has continued to evolve and now incorporates equipment such as closed-circuit rebreathers (CCRs). The ready access to this type of sophisticated equipment and to computer software to calculate suitable decompression schedules has allowed recreational divers to explore depths and areas previously only dreamed of. This exploration has come at a cost, and technical divers are an over-represented group in decompression illness (DCI) statistics. This study aimed to provide an insight for diving physicians (who potentially have to deal with emergencies relating to these activities) into the diving practices and health impact of deep, mixed-gas, repetitive diving.

Methods

This observational study was conducted during an eight-day expedition to the South China Sea in accordance with the Australian National Statement on ethical conduct in Research Involving Humans (June 1999). Informed written consent was sought from the seven divers participating in the expedition; only one diver did not consent, and no reason for this refusal was sought or given.

DIVING PERSONNEL

Divers were all non-smoking Caucasian males, average age 47.5 years (range 43 to 54 years). Average body mass index was 29 $kg.m^{-2}$ (range 25 to 32). The cumulative diving experience was 146 years (average 24 years, range 12–38 years). Experience on CCRs varied from one to eight years. Four divers had previously been treated for decompression sickness (DCS). Five divers described pre-existing chronic musculoskeletal injuries, mostly located in the upper limbs. Five divers consumed alcohol on most evenings, whilst one abstained completely during the expedition. No diver was seen to be intoxicated at any time. All divers hydrated actively before diving.

DIVER HEALTH SURVEY

The Diver Health Status (DHS) questionnaire developed by Doolette was used to assess the daily wellbeing of the divers.[2] The questionnaire comprises nine standardised questions. These cover five symptoms of DCS (paraesthesia, rash, balance, fatigue and pain), five health status indicators (vitality, pain, physical functioning, role limitation, and health perception) and the onset of symptoms, as well as a free response.[3] Each item is scored from 0 to 3, and the resultant summed DHS ranges from 0 to 30. This health survey has been validated against both a commercial diving group and a technical cave-diving group.[2–4] A questionnaire was filled out each evening by the divers, usually after dinner (i.e., about four hours post dive), directly into an Access® database (Office 2003®, Microsoft Corporation).[2] The participants also completed a post-expedition survey by e-mail. These data were transferred to and stored as an Excel® (Office 2003®, Microsoft Corporation) spreadsheet. The DHS scores were not calculated and analysed until after the expedition so as not to influence diving practices. Only a simple descriptive analysis was performed, without attempting to link scores to diving/decompression profiles.

DIVING PROCEDURES

All dives were conducted from a 22-metre dive boat based out of Singapore. On-board facilities included continual blending of nitrox and trimix gas mixes and pure oxygen (O_2), an electric hoist to lift divers back onto the boat and a 50-inch diameter twin-lock hyperbaric chamber, which had never been used in an emergency.

Diving was conducted over an eight-day period with usually two dives per day, except on the last day when only one dive was performed so as to allow a suitable surface interval before flying (Table 1). All dives, with the exception of this last dive, were to depths of greater than 50 metres' sea water (msw). The number of dives performed was at the discretion of the individual diver, only one diver performing all 15 dives (average 13, range 9–15). A surface interval of three hours was usual between dives on the same day. For dives to depths close to 70 msw, some divers elected to extend the surface interval to four hours.

Little formal dive planning was performed and no formal dive log was kept by the boat operator. A dive briefing was conducted before the first dive on each of six wreck sites. Two divers generally dived as a buddy pair. A further two, one being the diver who did not participate in the study, dived as a pair for some dives only. The remainder dived solo. Divers who operated in buddy pairs agreed on bottom time and bailout gases prior to diving, but, in general, formal written dive plans were not carried.

DIVING CONDITIONS

Daily air temperature varied between 30 and 35 °C, with 80% to 90% relative humidity. Surface water temperature was 30.5 °C and surface visibility approximately 20–30 m on most sites. A thermocline was present on the deeper wrecks with a drop in temperature to approximately 25 °C and visibility to about 5–10 m. Surface sea states were calm on all but one day. Currents up to two knots were experienced on most dives from about 30 msw to the surface. Divers clipped themselves to the decompression station (Figure 1), but the deeper stops during ascent along the shot line from the wreck to the station often involved considerable effort holding onto the line. Large numbers of jellyfish swept through the decompression station with the risk of envenomation.

DIVING EQUIPMENT

All divers used CCRs produced by Ambient Pressure Diving (APD), UK, five *Inspiration* rebreathers and one the smaller *Evolution* rebreather. A detailed description of the functioning of these units is beyond the scope of this article but interested readers are referred to Jeffrey Bozanic's book on the subject.[5] Three rebreathers used the new '*Vision*®' electronics, which incorporate an integrated decompression computer and a temperature monitor to assess the scrubber performance. The oldest unit on the trip had been heavily modified with the original scrubber head replaced with an after-market Hammerhead® unit (Juergenson Marine, Addison, PA, USA). This unit incorporated pre-production handsets for the Dive Rite Optima® rebreather, one of which contained an integrated dive computer. Several of the other CCRs had received minor modifications, such as mouthpieces with integrated open-circuit function, an extra oxygen (O_2) second stage, etc.

All divers carried delayed surface marker buoys which could be deployed during decompression. Two divers also

Table 1. Dive demographics

Day	Dive	Average depth	Average bottom time (h:mm)	Average dive duration (h:mm)	Man dives	Total bottom time (h:mm)	Total dive duration (h:mm)
1	1	56	0:25	1:13	6	2:34	7:19
	2	56	0:29	1:15	5	2:27	6:16
2	1	54	0:28	1:11	6	2:52	7:07
	2	55	0:34	1:33	6	3:28	9:21
3	1	53	0:36	1:33	5	3:03	7:48
	2	55	0:40	1:44	5	3:21	8:43
4	1	54	0:44	1:57	5	3:40	9:46
	2	55	0:35	1:35	3	1:46	4:46
5	1	64	0:30	1:22	6	3:00	8:14
	2	66	0:25	1:19	5	2:05	6:35
6	1	49	0:34	1:15	5	2:52	6:17
	2	52	0:42	1:26	4	2:48	5:47
7	1	53	0:23	1:04	4	1:34	4:16
	2	52	0:24	0:56	4	1:36	3:46
8	1	44	0:24	0:55	5	2:00	4:35
AVERAGE		**54**	**0:31**	**1:21**	**TOTAL 74**	**39:08**	**100:40**

carried personal emergency position radio beacons. Total weight of the diver's equipment was approximately 40–47 kg depending on configuration and the number of bailout cylinders.

GAS MANAGEMENT

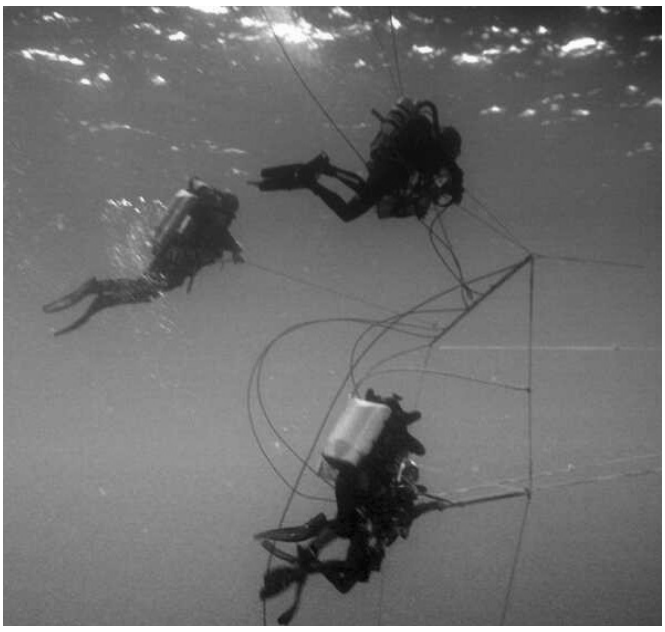
Bottom gas for all dives was trimix 15/50 (O_2 15%, helium 50%, nitrogen 35%). This was produced by a continual blending process and then stored in bank cylinders. A Haskel booster pump was used to guarantee that all diving cylinders were filled to 200 bar. All gas compositions were verified by the author using a helium/O_2 analyser (Analox®, UK). All fills were found to be within 1% of the desired values.

All divers carried an off-board 6-litre (water capacity) cylinder for 'bailout' in case of a total CCR failure. This cylinder contained either trimix (15/50) or air depending on the CCR configuration. Three divers carried an additional 6-litre bailout cylinder of trimix (15/50) for the deeper dives, for the longer planned dives and for dives involving wreck penetrations.

DECOMPRESSION PLANNING

The VR3® (Delta P Technology Ltd, Dorset, UK) mixed-gas dive computer was used by all the divers. On three of the CCRs, this was connected into the CCR loop with a fourth redundant oxygen cell for real-time monitoring of partial pressure of oxygen (PPO_2). One diver used a Sunto Viper dive computer as a back-up bottom timer. One diver carried back-up decompression tables. Three divers had fully redundant, mixed-gas decompression computers (Vision electronics, APD, Cornwall, UK).

Figure 1
An underwater decompression trapeze with bars at depths of 4.5 and 6 metres' sea water was slung underneath the boat during all dive operations. Surface-supplied oxygen was provided on the decompression station.



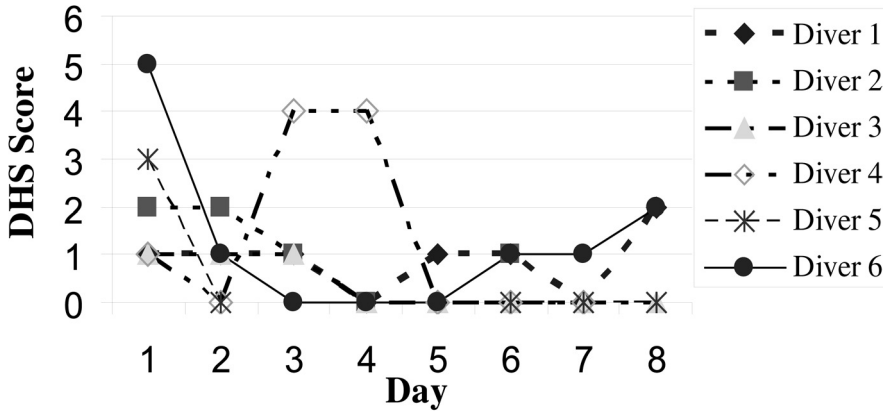
Although divers relied on their computers to provide the decompression profile, most had a fair idea of the required total dive time and final stop time for a given bottom time, based on previous experience. Two divers formally used the dive-planning function of the VR3 to predict their decompression requirements, although the results were generally not written down. Bottom time was usually planned on estimated decompression obligation rather than gas requirements. For divers carrying redundant dive computers, the decompression profile was dictated by the more conservative computer.

All VR3s utilised the Buhlmann ZHL-16 algorithm with deep stops as per the method described by Pyle.[6,7] Most divers used the 0% conservatism setting on the VR3 for all dives. One diver added a 10% conservatism factor to the algorithm for all dives, and one diver changed his setting from 0% to 10% after two days "to give more conservatism". Three divers used the inbuilt decompression computer in the *Vision* electronics, which incorporate the readings from the three CCR O_2 cells to calculate decompression requirements. This computer also utilises the Buhlmann ZHL-16 decompression method. Conservatism is altered by selecting the 'gradient factors'(allowed maximum super-saturation limits) for the deep and shallow parts of the dive.[8] One diver used the decompression computer incorporated into his Hammerhead electronics. This unit used the same decompression algorithm and method as the *Vision* decompression computer. Both the VR3s and the *Vision* tracked pulmonary O_2 toxicity units (OTU) and central nervous system (CNS) O_2 toxicity based on the methods described by Hamilton et al.[9]

All divers used a PPO_2 set point of 1.3 ATA at depth and for ascent. Three divers manually increased the PPO_2 when at 6 msw to between 1.5 and 1.6 ATA (i.e., 100% O_2). Three divers utilised the surface-supplied, open-circuit O_2 at the 6 msw stop. There was considerable variation in practice as to whether the divers told their dive computers that they had changed their PPO_2 during the final decompression stop, some opting not to do so in order to gain a measure of decompression conservatism. Two divers limited their time on 100% O_2 to 20-minute periods, performing five-minute 'low O_2 breaks' between oxygen periods.

Three divers changed the diluent from trimix to air at between 30 and 40 msw during ascent by flushing their units so as to accelerate their decompression. The other divers remained on the trimix mixture except if they changed to 100% O_2 (by using either the open-circuit O_2 or the CCR as an O_2 rebreather). No problems were encountered with the practice of diluent switching.

Figure 2
Diver health scores (DHS – Diver Health Status; diver 3 obscured by other data)



BAILOUT PLANNING

In general, insufficient open-circuit gas was carried to allow an independent return to the surface in the event of catastrophic CCR failure. Planning revolved around having sufficient gas to return to the shot line and reach the decompression station. Given that the 6-litre cylinders could be expected to last approximately 12 minutes at 56 msw, in many cases this plan would seem rather stretched! For divers who dived as buddy pairs, it was assumed that the chances of a dual catastrophic CCR failure were low, and that bailout gas would be shared in the event of an emergency. These divers also carried two bailout cylinders for deeper and longer dives. However, it was argued by some of the divers that, for penetration dives, the use of two sling cylinders increases the risks of entrapment and would impede their ability to swim. However, two divers with the dual bailout cylinder configuration swam the entire length of one wreck and back (some 500 m) at depths between 55 msw and 69 msw without physical distress.

SCRUBBER MANAGEMENT

Sodasorb® 4-8 (WR Grace & Co, Chicago, USA) with ethyl violet indicator was used for carbon dioxide (CO_2) removal. This is not the recommended CO_2 absorbent (which is Sofnolime 797) for the *Inspiration* rebreather, and is of a courser mesh. CO_2 absorbent changes were based on time of use or the integrated temperature monitor where fitted. Scrubber durations of up to six hours were reported; however, average change time was approximately 4.5 hours. All the CCRs had integrated timers used to track scrubber use. No scrubber warnings were seen on those units with scrubber monitors. The depth of the used Sodasorb as indicated by the colour change was noted to correspond closely with the scrubber monitor in the Vision electronic package.

POST-DIVE MAINTENANCE

All divers opened their CCRs after each dive to allow any condensation of water onto the O_2 cells to dry. All units were assembled well before the next dive and a positive-pressure check performed. All divers were observed to perform pre-dive CCR checks and breathe the CCR prior to the dive to activate the CO_2 scrubber. Most CCRs had their loop and counter-lungs washed out in fresh water at the end of each day. Antiseptics were not routinely used.

Results

DIVER HEALTH SURVEY

Daily scores for the Diver Health Status questionnaire are shown in Figure 2. On seven of the potential 48 diver/days (15%), three divers did not dive and a questionnaire was not completed. The maximum DHS recorded during the expedition was five in Diver six. He had a pre-existing musculoskeletal injury in his left arm. On the first day of diving, his arm received pronounced jerking on the shot line during decompression. Self treatment with an anti-inflammatory non-steroidal (naproxen) relieved his pain. Diver four developed right shoulder pain, anatomically related to an old sporting injury, after his fifth dive, giving a score of four for two days. Return to 6 msw breathing 100% O_2 did not relieve this pain. He continued diving and the pain resolved spontaneously. Most other divers had low-level grumbling pains consistent with pre-existing injuries. Three other divers with pre-existing musculoskeletal injuries reported improvement in their condition during the expedition.

DECOMPRESSION INCIDENTS

There were no incidents of divers breaching the decompression ceilings as indicated by their dive computers. Two divers developed symptoms suggestive of marginal DCS (see above). In both cases, the symptoms were clouded by pre-existing musculoskeletal injuries at the anatomical sites and by divers having hung onto the jerking shot line during prolonged decompression. In both cases, the divers continued diving for the remainder of the expedition and their symptoms resolved spontaneously. Divers all flew

Table 2
Equipment events and failures during 74 trimix technical dives

Description	Emergency	Management	Repaired
Hammerhead handset flood	Yes	Ascent using VR3 and secondary handset	No
Diluent LP hose failure	Yes	Ascent (diluent not used on ascent)	Yes
Partial scrubber flood	Yes	Ascent, semi-closed mode; surface supplied O_2 on deco	No, VR3 interface removed
VR3 electronic interface failure	No	VR3 changed to set-point PPO_2 mode	Yes, cleaned and dried
Oxygen cell failure	No	Detected on the surface	Replaced
Autoair (combined BC inflator/ regulator) damaged	No	Detected on surface	Repaired
Mouthpiece torn	No	None	Replaced
SPG(O_2) hose failure on surface	No	Removed	No
Primary torch failure	No	Back-up torch used	Yes, but failed again later
Back-up torch failure	No	No, repeated failure	

home approximately 24 hours after the last dive. No divers developed symptoms of DCS associated with or after flying.

OXYGEN TOXICITY

Three divers reported symptoms of chest tightness and a dry cough after the fourth day, consistent with pulmonary O_2 toxicity. One of these divers had an episode of mild haemoptysis and persistent coughing after prolonged O_2 use. Two of the divers using *Vision* units had O_2 toxicity warning alarms during decompression, indicating that they had exceeded the National Oceanic and Atmospheric Administration daily limits. No divers reported symptoms of central nervous system (CNS) O_2 toxicity. No divers exceeded the recommended CNS O_2 limits as calculated by their computers.

Three divers reported a change in visual acuity by the end of the expedition, notably an improvement in near vision and deterioration in distance vision. Unfortunately these changes could not be quantified.

INJURIES

Several divers sustained minor injuries from sea urchin spines or minor stings from hydroids on the wrecks. One diver suffered minor jellyfish stings about the face while on decompression. One diver developed friction ulcers on the feet from his fins, whilst another required antibiotics and drainage of a paronychia. One diver developed an upper respiratory tract infection and did not dive for two days.

EQUIPMENT PROBLEMS

There were 10 equipment failures (Table 2), none resulting in the cancellation of a dive although some dives were subsequently carried out with a reduced level of redundancy. Three incidents, involving two divers, occurred underwater and resulted in the dive being aborted. Neither diver needed to resort to open-circuit scuba or to violate their decompression obligations.

The VR3 O_2 cell interface unit caused one CCR to partially flood as a result of a displaced O-ring . This then caused a CO_2 breakthrough and failure of the scrubber monitor. The diver involved converted to a 'semi-closed' mode, where gas is vented from the loop after every fifth breath, while ascending to the decompression station where he then utilised open-circuit O_2. The VR3 interface was not subsequently used with this unit. In a second unit, corrosion of the electrical connection between the VR3 and the O_2 cell caused intermittent problems for several dives but this was resolved by the end of the expedition.

On another unit, the handset of the Hammerhead unit flooded resulting in a loss of the primary PPO_2 display, decompression data and automatic O_2 solenoid control. However, this unit had a secondary handset that provides redundant PPO_2 monitoring hence allowing manual O_2 addition. Further back-up PPO_2 monitoring and decompression data were provided by the integrated VR3. The owner of this unit elected to continue to dive the CCR manually on subsequent dives controlling his unit via the remaining handset and VR3.

One diver reported a headache associated with a very high workload at depth. His scrubber had two hours' use prior to this event.

Discussion

The ready availability of decompression software and the ease of obtaining helium have resulted in a rapid growth in technical diving. In conjunction with this boom has been the introduction in the late 1990s into the recreational arena of closed-circuit, mixed-gas rebreathers of which several models, including the APD *Inspiration*, are now available.

The reluctance of manufacturers to disclose their sales numbers makes accurate estimations of total rebreather numbers difficult. However, these units are widely used in the Northern Hemisphere and increasingly in Australasia.

Unlike open-circuit scuba, gas consumption of a CCR is essentially independent of depth. Gas is recirculated through the 'loop' via one-way valves past a 'scrubber' to remove CO_2. Oxygen levels are sensed via several oxygen cells, and O_2 is added either via a computer-controlled solenoid or via manual injection from the user, depending on the model, to maintain a constant PPO_2 in the breathing loop. Diluent gas is added to the loop to maintain loop volume as the diver descends. Gas consumption is, therefore, dependent only on the diver's O_2 consumption, with a small volume of diluent used to bring the loop to ambient pressure. Typical gas consumption during this expedition, with total dive times of approximately 100 minutes, was about 150 litres of diluent and 150 litres of O_2 per dive compared with some 6,500 litres of gas that would be expected to be consumed for a similar dive on open-circuit scuba. This represents a saving on gas costs from approximately AU$150 per day per open-circuit diver to AU$25 per day per CCR diver for gas and CO_2 absorbent.

While opencircuit dive planning is limited by the gas supply that can be carried and/or staged and the decompression needs of the dive, CCR planning is limited largely by the scrubber duration (in the case of the *Inspiration* about 4–6 hours, dependent on depth, water temperature, grade of soda lime used, etc.) and O_2 supply. This allows CCR divers large margins with regards to dive duration and contingency planning over open-circuit divers at the expense of the substantially increased complexity of the scuba system. In both cases, hypothermia may be an important factor.

Potential complications of rebreathers include hyperoxia, hypoxia, hypercarbia and 'caustic cocktail' (the last of these if water should enter the scrubber and allow alkaline soda lime to enter the breathing loop). The increased complexity both in operation and care has also come at a human cost, with a relatively high mortality rate amongst CCR divers, mostly ascribed to user error. During this expedition, there were relatively few incidents or problems with the rebreathers per se and these were largely confined to the oldest and most modified CCR in the group. The divers, being very experienced, managed these incidents without requiring external help and without the need to resort to their open-circuit bailout option. In a less experienced group of divers outcomes may have been less favourable.

The lack of formal dive planning and the high level of solo diving are both of concern. In an internet survey of *Inspiration* users in 2002, Hawkins found that 42% dived solo and almost 20% chose to carry no open-circuit bailout.[10] These behaviours appeared to correspond to divers who subsequently showed a high mortality. The lack of detailed planning was facilitated particularly by the availability of continuous decompression solutions produced by decompression computers. However, given the substantial amount of accrued decompression on each dive, the reliance on this technology alone without a written back-up plan would seem somewhat cavalier.

The type and depth of diving during this expedition was fairly typical of that being practised by recreational technical divers. Most of the dives would have been placed in the 'extreme exposure' category in the DCIEM decompression tables, with an expected DCS rate of approximately 4%.[11] In practice, no DCS was observed on this expedition or in a cave-diving group also using the Buhlmann ZHL-16 algorithm.[3] However, the numbers of dives were relatively small. Both 'forward' and 'reverse' profile dives were performed. No divers used the popular 'bubble' models, VPM or RGBM, which introduce a series of deep decompression stops and, often, reduced shallow decompression times.[12,13]

The low observed rate of DCS might be ascribed to several factors. Ideal temperature conditions were present with divers cool on the bottom and decompressing in 30 °C water, enhancing blood flow and gas elimination. This was somewhat offset by the difficulties produced by the currents experienced during decompression. The diver lift minimised the need for divers to strain getting back onto the boat. Also, CCRs maintain a constant PPO_2, keeping the 'oxygen window' optimised during decompression.[14,15] Finally the use of near 100% O_2 at the 6 msw stop would optimise the inert gas gradients and help minimise any bubbles that had formed.

No problems were encountered during the expedition in the divers who switched diluents to accelerate decompression, a practice that is controversial as it appears to be associated with a high incidence of inner ear decompression sickness (IEDCS).[16,17] Some technical diving agencies now limit the changes in inert gas concentrations during decompression. For CCR divers, as the PPO_2 is kept constant, the partial pressure of inert gases falls proportionally as the diver ascends. Switching diluent offers only a small additional reduction in decompression obligation for most dives and it would appear difficult to justify the risks of developing IEDCS for this small gain. A recent animal study has suggested that the gas kinetics of nitrogen and helium are not, in fact, as different as predicted by most decompression models.[18] If correct, this would mean that the predicted acceleration in decompression by switching gases may not occur.

Pre-existing musculoskeletal injuries in this group provided some difficulty in making a diagnosis of marginal DCS. Typically divers tend to rationalise marginal symptoms as being due to other causes and will self treat where possible. The author was not asked to formally deliberate on the exact nature of these symptoms despite his background as a diving physician being well known to the divers.

DHS scores have been correlated to decompression stress in occupational and technical diving groups, with scores of six or greater being associated with the development of clinical DCS requiring treatment.[2,4,19] No divers reached a score of six during this expedition, and none developed overt DCS. DHS has also been correlated to diving depth. Doolette found an increase of one DHS unit per 13 msw increase in depth.[3] For this expedition, scores of one to two would, therefore, have been expected and were indeed seen for most divers on most days. The lack of DCS symptoms despite the divers being in what would generally be considered relatively high-risk categories (overweight, middle-aged, relatively unfit, alcohol intake the night before diving, etc.) would imply that the decompression algorithm used and the decompression practices engaged in produced satisfactory decompression solutions within the depth/time profiles conducted. No symptoms of DCS post flying were observed despite deep, repetitive mixed-gas dives and a relatively short interval between the last dive and flying home.

The incidence of pulmonary oxygen toxicity symptoms (three divers) and of minor visual changes (three divers) is indicative of the high oxygen exposure associated with repetitive deep CCR diving. Both have been reported previously by technical divers in the popular literature. In all cases these symptoms were reported to have resolved post expedition. In two cases, divers reduced their number of dives (after the 69 msw dives) to reduce the oxygen exposure.

Conclusions

The use of CCRs for 74 man dives in the 50 to 70 msw depth range by six experienced technical divers, total time underwater of 100.4 hours, was associated with few technical problems. Diver health survey scores were five or less and no clinical cases of DCS were observed.

Acknowledgements

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Review article

The use of extraglottic airway devices in diving medicine – a review of the literature. Part 1: On-site (beach) management of near-drowned victims

Christopher John Acott

Key words
Extraglottic airway devices, oesophageal combitube, near drowning, resuscitation, review article

Abstract

(Acott CJ. The use of extraglottic airway devices in diving medicine – a review of the literature. Part 1: On-site (beach) management of near-drowned victims. *Diving and Hyperbaric Medicine*. 2006; 36: 186-94.)
On-site resuscitation for near-drowned (ND) victims has been limited to expired air resuscitation (EAR), bag mask ventilation (BMV) and intubation despite the development of the classic laryngeal mask airway (cLMA) and other extraglottic airway devices (EADs). Endotracheal intubation is the gold standard for airway control and ventilation during resuscitation; however, it requires a high degree of training, skill retention and additional equipment. In addition, BMV and EAR may be difficult because of the victim's physical characteristics and the need for an increased inspiratory pressure because of the pathophysiological effects of ND. BMV and EAR may also cause gastric inflation increasing the risk of regurgitation. A review of the relevant studies concerning the use of EADs in resuscitation and trauma was conducted to examine their suitability for use in resuscitation of ND victims. Those suitable were then compared with endotracheal intubation. The majority of the EADs reviewed lacked substantive data to support their use. However, the oesophageal tracheal combitube (OTC) and the cLMA are currently the only EADs with a Class lla recommendation from the American Heart Association. The risk of aspiration, gastric inflation and the inability to apply positive end expiratory pressure (PEEP) limits the use of the cLMA and other laryngeal masks (except the ProSeal™) in the emergency management of ND victims. Because the OTC protects the airway from aspiration, and permits gastric suction and the application of PEEP it is the EAD of choice in the management of adult ND victims (height > 117 cm).

Introduction

On-site resuscitation measures for near-drowned (ND) victims have been limited mainly to expired air resuscitation (EAR), bag mask ventilation (BMV) and intubation despite the development of the classic laryngeal mask airway (cLMA) and other extraglottic airway devices (EADs).[1] During cardiopulmonary resuscitation (CPR) the distribution of gas between the lungs and stomach during intermittent positive pressure ventilation (IPPV) in an unprotected airway has been shown to be determined by the victim's airway resistance, pulmonary compliance, lower oesophageal sphincter pressure and the peak inspiratory pressure required for ventilation.[2] The pathophysiological effects of ND of decreased lung compliance, pulmonary oedema and atelectasis will not only increase the magnitude of the intrapulmonary shunt but also increase the inspiratory pressure required during BMV, predisposing to gastric inflation and the risk of regurgitation.[3] Gastric distension limits ventilation and hence any resuscitative efforts should involve means to deflate the stomach. In addition, some of the victim's physical factors (a lack of teeth, the presence of a beard, an increased body mass index, a history of snoring or age greater than 55) may also make BMV and EAR difficult.[4] While endotracheal intubation remains the gold standard for airway control and ventilation during resuscitation, it requires a high degree of training, skill retention and additional equipment (a working laryngoscope and suction apparatus). Laryngoscopy and intubation in ND victims may also be difficult because of an obstructed view of the larynx by regurgitated gastric contents or pulmonary oedema fluid and, when attempted on the beach, environmental glare will add to the difficulty.

Resuscitative efforts to improve the victim's oxygenation will require all or some of the following:
- increase in the inspired oxygen fraction (FiO_2)
- application of IPPV with or without positive end expiratory pressure (PEEP) to decrease the magnitude of pulmonary shunt
- tracheal and oropharyngeal suction to clear some of the pulmonary oedema fluid to enable ventilation.[3]

A plethora of EADs have been marketed since the release of the cLMA (Table 1), some of which have been shown to be superior to BMV during resuscitation and CPR.[5,6] However, all are untried in the first-aid management of ND victims. Because there are no data concerning the use of the cLMA or any other EAD in the 'on-site' management of the ND victim a literature review of their characteristics

Table 1
Other extraglottic airway devices released for use since the classic laryngeal mask airway (cLMA)[5]

- Pharyngeo-tracheal lumen airway (1984)
- Oesophageal tracheal combitube (1986)
- Flexible laryngeal mask airway (1991)
- Cuffed oral pharyngeal airway (1992)
- Intubating laryngeal mask airway (1997)
- Glottic aperture seal airway (1998)
- Laryngeal tube airway (1999)
- ProSeal laryngeal mask airway (2000)
- Airway management device (2000)
- Soft seal laryngeal mask, Portex™ (2002)
- Streamlined liner of the pharyngeal airway (2002)
- Laryngeal tube suction airway (2002)
- PAxpress oropharyngeal airway (2002)
- COBRA perilaryngeal airway (2003)
- Elisha airway device (2003)
- Easy tube (2003)

Table 2
Desirable characteristics of any airway device used for 'out of hospital' resuscitation of near-drowned victims

- Easy insertion by non-anaesthetists
- Blind insertion
- Used in difficult airway scenarios
- Minimal or no aspiration risk
- Negligible side effects (sore throat, dysphagia, hoarseness, blood contamination)
- Cricoid pressure friendly
- Easily converted to tracheal tube placement
- Minimal gastric inflation with IPPV
- Able to use PEEP
- Able to suction trachea
- Able to insert gastric tube and deflate the stomach
- Data confirming use in CPR
- Able to be secured once placed
- Paediatric size available

was conducted to predict their suitability for use in airway management of ND victims, particularly in clinical situations requiring endotracheal intubation which are or have been proven difficult.

Methods

A medical literature search was conducted for relevant studies of the EADs listed in Table 1 using the clinical criteria outlined in Table 2. These criteria were modified from the ideal airway device characteristics proposed by Charters.[7] The relevant data, comparison with endotracheal intubation and conclusions regarding each EAD's suitability for 'beach resuscitation' are tabulated in Table 3.

Review of the current extraglottic airway devices

THE CLASSIC LARYNGEAL MASK AIRWAY

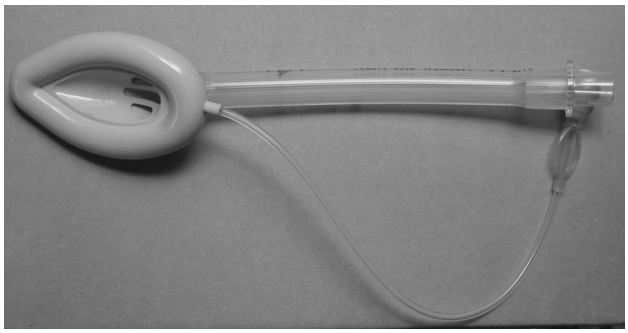
The cLMA (Figure 1) is a ventilatory device that provides a conduit from outside the lips to the laryngeal opening and has added a new dimension to airway control. The cLMA is easily inserted and secured. Since its commercial release in the United Kingdom in the 1980s, it has gained wide international acceptance in anaesthesia practice

Table 3. Comparison of various EADs to endotracheal intubation for use in 'beach' resuscitation (see text for full names of devices and other terms)

	cLMA	OTC	pLMA	SLIPA	LTA	ETT
Easy insertion	Yes	Yes	+/-	Yes	Yes	No
Blind insertion	Yes	Yes	+/-	Yes	Yes	No
Use in CPR*	Yes	Yes	Yes	Yes	Yes	Yes
Aspiration risk	Yes	No	No	No	No	No
Gastric inflation	Yes	No	No	+/-	No	No
Gastric tube insertable	No	Yes	Yes	No	No	Yes
CP friendly	No	No	No	Nd	No	Yes
IPPV	+/-	Yes	Yes	Yes	Yes	Yes
PEEP (up to +10 cm)	No	Md	Yes	Nd	Nd	Yes
CVS side effects	+	+++(+)	+	+	Md	+++(+)
Easily converted to ETT	No**	Yes	No**	No**	No**	—
Suction trachea	No	Yes	No	No	No	Yes
Securable once placed	No	Yes	No	No	No	No
Used in difficult airway	Yes	Yes	Yes	Nd	Nd	Yes
Paediatric size	Yes	No	Yes	No	Nd	Yes
Ease of training	Yes	Yes	+/-	Ld	Nd	No
Recommended	Y/N	Yes	Yes	Md	Md	

* includes manikin studies; ** bougie or fibrescope required, blind intubation through device occasionally successful
CP – cricoid pressure; Nd – no data; Ld – limited data; Md – more data and studies needed; Yes/No – better than BMV

Figure 1
The classic laryngeal mask airway (cLMA). When the cuff is fully inflated following correct insertion, the cLMA occupies the hypopharynx and rests against the upper oesophageal sphincter behind the cricoid cartilage. The cuff and bowl seal the laryngeal inlet. The cLMA's sides face the pyriform fossae and the epiglottis rests inside the bowl or under the proximal cuff at the junction of the cuff and airway tube.



both in routine cases and the management of the difficult airway.[6,8]

There are still reservations concerning the use of the cLMA for controlled ventilation and the prevention of aspiration.[8,9] Its role in trauma management is controversial; however, there are data suggesting better oxygenation and airway control than BMV.[6,8] Despite these reservations it has been reported to have provided an effective emergency airway in a variety of crisis situations and hence it is now considered a primary option for the management of the difficult airway by the American Society of Anesthesiologists (ASA),[10] the European Resuscitation Council,[11] and the British Difficult Airway Society.[12]

A meta-analysis of 10 studies containing 700 patients revealed that cricoid pressure (CP) not only impeded the insertion of the cLMA but also impeded ventilation after successful insertion of the cLMA. These data were applicable to any type of laryngeal mask.[6]

DISPOSABLE SOFT SEAL LARYNGEAL MASK

Portex™ released the soft seal LMA in 2002.[5] It differs from the cLMA in that it is made from polyvinyl, has a deeper bowl, blunter distal cuff, no aperture bars and a wider airway tube fused to a larger part of the bowl. There are contradictory data comparing it with the cLMA regarding ease of insertion.[13]

INTUBATING LARYNGEAL MASK AIRWAY

The intubating laryngeal mask (iLMA) functions in the same manner as the cLMA and hence offers inadequate airway protection. It was designed to facilitate either blind or fibreoptically assisted intubation in the difficult airway scenario.[14,15] Even inexperienced operators find the iLMA easy to insert and achieve ventilation.[16] One study suggested that the iLMA was inserted faster than the cLMA with a greater proportion achieving ventilation after their first attempt.[17] There are limited data on the use of the iLMA in CPR and only one study evaluating its use in children.[18] It may offer an advantage over the cLMA when a patient needs to be intubated. When used in the pre-hospital setting it will need to be replaced upon arrival at hospital, but at present the majority of hospital personnel are unfamiliar with it.

THE OESOPHAGEAL TRACHEAL COMBITUBE

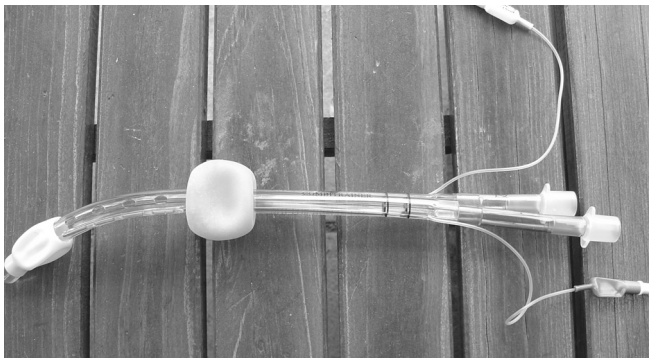
The oesophageal tracheal combitube (OTC) is a double-lumen, double-cuffed, polyvinyl EAD that can be used as the primary or as a secondary 'rescue airway' (Figure 2). It can function as an alternative ventilatory device to bag mask ventilation, the cLMA or endotracheal intubation.[19] The ASA,[10] American Heart Association,[19] and the European Resuscitation Council[11] have included the OTC in their guidelines as an emergency rescue airway device. The OTC is available in two sizes: 37F and 41F. The 37F is now recommended for use in the majority of patients greater than 117 cm in height. There is no paediatric size available at present.[20, 21]

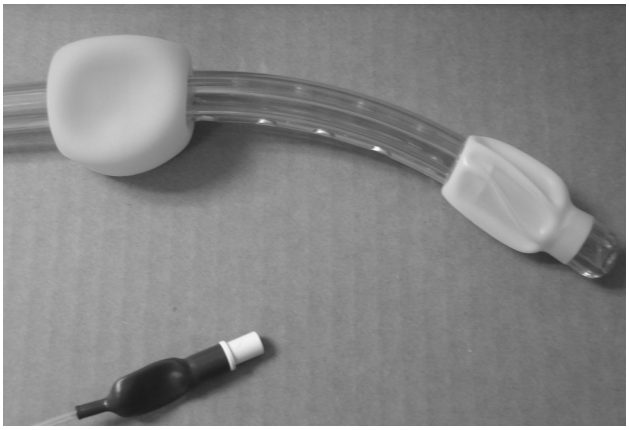
The two separated short, proximal, colour-coded tubes (numbered 1 and 2) unite to form one tube with a double lumen. These two proximal tubes each have a 15 mm connector and are of differing length (Figure 2a). The longer blue tube (numbered 1) is blind at the distal end but has eight small ventilatory side ports located midway along the joined single lumen (Figure 2b). The shorter clear tube (numbered 2) is open at its distal end and resembles an endotracheal tube (ETT). The double rings marked just distal to the junction of the two proximal colour-coded tubes should be at the level of the patient's teeth or alveolar margins when the OTC is correctly placed. The diameter of the 37F is 14 mm at its distal end (Size 8 ETT is 12 mm).[19–21]

The large proximal oropharyngeal latex cuff seals the upper airway while the smaller distal oesophageal-tracheal cuff will seal either the oesophagus when in the oesophageal position or the trachea when in the tracheal position. Various studies have been published concerning cuff volumes and pressures.[22] However, the potential risk of impaired oropharyngeal venous blood flow and swelling of the oropharyngeal soft tissues by the oropharyngeal balloon can be prevented by deflating the balloon to the minimum volume required for an airtight seal and routinely measuring cuff pressures.[20,22]

Insertion technique for the OTC is described in Table 4. During insertion there is little movement of the head and cervical spine and, therefore, it has been reported to be suitable for securing the airway in patients with either a fractured or abnormal cervical spine or difficult intubation. However, some insertions do require elevation of the chin

Figures 2a and 2b
The oesophageal tracheal combitube (OTC). Note the two cuffs, the larger pharyngeal cuff and the distal oesophageal (or tracheal) cuff, with the ventilating holes between.





and tongue.[23–25] Cricoid pressure cannot be applied while the OTC is being inserted, but insertion has been successfully performed in a vomiting patient without aspiration.[26] Contra-indications for use include patients with intact gag reflexes, known oesophageal pathology, following ingestion of caustic substances, supraglottic tumours or stenosis and unfamiliarity with its use.[19]

The OTC provides adequate ventilation and oxygenation in either oesophageal or tracheal positions even during CPR.[27] The oesophageal position is preferred and has been reported to occur in 89–95% of occasions. In this position ventilation occurs through the longer blue tube via the eight pharyngeal perforations, while in the tracheal position ventilation is via the shorter clear tube. Studies have shown there is almost 100% recognition by paramedic staff of oesophageal or tracheal placement.[28]

Patients ventilated with identical ventilatory parameters via an oesophageally placed OTC generated higher arterial oxygen partial pressures than patients ventilated with an ETT. This is probably due to a slower increase in inspiratory pressure and a positive end expiratory pressure effect of approximately 2 cm H_2O caused by the increased expiratory resistance associated with the perforations in the oesophageal limb of the OTC.[29] In the tracheal position the oropharyngeal cuff can be deflated; however, it is recommended that this cuff is inflated during transport to prevent dislodgement unless secured in another way.

Difficulty with ventilation has been recorded due to partial obstruction of the ventilatory perforations because of too deep an insertion of the pharyngeal tube in the oesophagus, or glottic obstruction due to downward displacement of the epiglottis by the inflated proximal oropharyngeal cuff. Withdrawing the OTC in increments of 2–3 cm can restore ventilation.[19,30]

Table 4
Insertion technique for the oesophageal tracheal combitube (OTC)

1 Bend the portion of the OTC between the cuffs in order to augment the preformed curve and maintain this bend as long as possible prior to insertion (a modified Lipp manoeuvre).
2 Blind insertion in the midline in a caudal direction along the tongue; avoid pushing against the hard palate and posterior pharyngeal wall. A laryngoscope can also be used to assist insertion.
3 Head preferably in the neutral position.
4 The OTC is inserted until the patient's teeth or alveolar margins lie between the double rings distal to the junction of the two proximal tubes.
5 The oropharyngeal cuff is inflated first with 50–85 ml of air followed by the oesophageal/tracheal cuff with 8–10 ml.
6 Attach a ventilating bag to the longer blue tube 1 and confirm chest ventilation by auscultation of the chest listening for bilateral lung sounds and epigastrium confirming an absence of gastric insufflation. In addition an oesophageal detector device, capnometry and colorimetric breath indicators can be used to verify the position of the OTC.
7 Ventilate via the colourless shorter tube 2 if there is an absence of chest breath sounds, a failure to detect carbon dioxide via capnometry, or gastric inflation.
8 In the absence of ventilation via either tube check the position of the teeth or alveolar margins in relationship to the two proximal rings, deflate cuffs and adjust accordingly.
9 The most common insertion problem is too deep an insertion. A failure to ventilate after adjustment requires a further cuff deflation and withdrawal of the OTC in increments of 2–3 cm checking ventilation each time until it is achieved.

Several studies have shown that the skill retention required to insert the OTC is easier to retain over time when compared with the cLMA and endotracheal intubation. However, the period of time required before retraining has varied in different studies and is more likely to be related to the airway skills used on a daily basis by paramedics.[6,31]

The OTC is primarily intended for emergency use and should not be left in situ for more than eight hours. Complications of the OTC, such as oesophageal and pyriform fossa tears, haematomas, dysphagia and sore throat occur infrequently.[30,31] The reported increase in airway morbidity may be explained by the unphysiological high cuff pressure, which may be prevented by deflating the cuffs to the minimum volume required for an airtight seal and routinely monitoring intra-cuff pressures.[22,32]

Intubation can be performed with the OTC in place protecting the airway from aspiration. If it is in the tracheal position an exchange catheter bougie technique is used with an appropriately sized bougie to enable it to be placed in the OTC's tracheal lumen. If the OTC is in the oesophageal position the oropharyngeal cuff is deflated, and the OTC pushed to the left followed by laryngoscopy and intubation; the distal cuff is left inflated until intubation is achieved.[19–21]

THE EASY TUBE

The Easy tube (EzT) was released in Europe in 2003. It is a double-lumen tube similar to the OTC but is latex free. Ventilation is via a single large orifice situated between the oropharyngeal and oesophageal cuffs and allows the passage of a fibreoptic scope, bougie or suction catheter.[33] There are two sizes (28 and 41) for use in patients greater than 90 cm in height. The tip of the size 41 is the same as that of a standard 7.5 mm ETT, and the tip of the size 28 as for a standard 5.5 mm ETT. The tip of the EzT resembles the end of an endotracheal tube and is less bulky than the OTC. There are limited data on its use at present. A recent study has shown it to be effective in the 'difficult airway' scenario in either anaesthesia or the pre-hospital setting.[33]

PROSEAL LARYNGEAL MASK AIRWAY

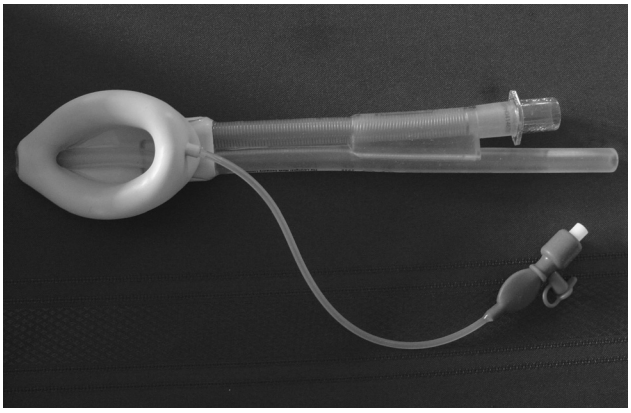
The ProSeal (pLMA) is a major advance in airway control compared with the cLMA. It allows ventilation at higher airway pressures, protects against gastric insufflation and aspiration, allows insertion of a gastric tube and has a built-in bite block (Figure 3).[34] It has four main components: a bowl-shaped mask, pilot balloon inflation line, an airway and drainage tubes. The airway tube is shorter and narrower than that of the cLMA (9 mm) and hence has a 20% greater airway resistance. The drainage tube traverses the floor of the mask opening at the mask tip.[34,35] There are paediatric sizes available.

Digital insertion is recommended with the head in the intubating position (neck flexed, head extended) using either a metal introducer or a gum-elastic bougie-guided technique.[34–6] The pLMA is more difficult to insert digitally than the cLMA because of the larger cuff, which leaves less room in the mouth for the index finger; however, this difficulty is eliminated when the metal introducer or the bougie-guided technique is used (both these techniques have the advantage that a finger is not placed in the patient's mouth).[37] Using the introducer made insertion of the pLMA easier than that of the cLMA in patients with manual in-line neck stabilization.[38] Haemodynamic responses to insertion (whatever the method) are similar to those seen with insertion of the cLMA with an increase in mean arterial pressure and heart rate of about 20%.[5]

The pLMA is an improvement on the cLMA for controlled ventilation and can be used effectively for the application of 10 cm H_2O PEEP during IPPV without any detectable gas leak or gastric inflation.[39] The improved airway seal is thought to be due to the larger wedge-shaped ventral cuff, deeper bowl with the dorsal cuff pushing the ventral cuff firmly into the periglottic tissues.[35,37] A correctly positioned pLMA theoretically protects the airway from aspiration; however, comparison of the proposed increased safety of the pLMA with that of the cLMA in a patient with an aspiration risk will probably remain unproven. Therefore, it is important to identify the correct position of the pLMA by the performance of a series of simple tests.[34,40] The drainage tube allows insertion of a gastric tube for drainage of the stomach. Failure to insert a gastric tube via the drainage tube can be due to malpositioning, inadequate tube lubrication or herniation of the dorsal cuff compressing the drainage tube in the bowl.[41]

There are no clinical case reports of the use of the pLMA in the trauma setting but it has been reported as a rescue device after failed intubation during rapid-sequence intubation.[42]

Figure 3
The ProSeal laryngeal mask airway (pLMA). The pLMA differs from the cLMA in that it is bulkier and has a gastric drainage tube passing through the bowl. This drainage tube allows the passage of an oral-gastric tube for drainage of the stomach.



Manikin studies comparing various laryngeal masks with tracheal intubation, OTC, laryngeal tube suction airway (LTSA) or BMV during simulated CPR showed that the pLMA functioned as well as the tracheal tube, OTC or LTSA but better than BMV or the other laryngeal masks (cLMA, iLMA and the disposable LMA).[5,35]

The pLMA is not designed to replace the ETT in patients who are at risk of aspiration but it offers several important advantages over the cLMA:

- it isolates the gastrointestinal tract from the airway[34,35]
- when correctly positioned its design makes gastric inflation unlikely and a gastric tube can be inserted to aspirate or deflate the stomach[34,42]
- it has a built-in bite block[34]
- its airway sealing pressure is 50% greater (10.8 cm H_2O) than the cLMA[34,42]
- up to 10.0 cm H_2O PEEP can be applied without gastric inflation[39]
- a wider bowl without aperture bars makes the view of the glottis with a fibrescope easier and allows for easier intubation[35,42]
- malposition of the pLMA can be detected by a series of simple tests.[5,34,40]

LARYNGEAL TUBE AIRWAY AND LARYNGEAL TUBE SUCTION AIRWAY

The laryngeal tube airway (LTA) is a single-lumen, silicone tube with two, low-pressure cuffs (oropharyngeal and oesophageal) and a ventilation port between these two. It is autoclavable and can be used up to 50 times. Six sizes are available (from neonates to large adults) but usually a size 4 is adequate for adults. The cuffs are inflated by a single pilot balloon either via a cuff inflator or with a 100 ml syringe with marks for the recommended volumes for each size of the LTA. The single ventilation orifice is positioned between the two cuffs and when correctly positioned lies behind the larynx. The orifice is large enough to allow for fibreoptic bronchoscopy and suctioning. A disposable version is now available. [43]

It is inserted in the midline until resistance is felt; the patient's head can be in either the neutral or intubating position. The cuffs are then inflated. When correctly placed, the LTA lies along the midline of the tongue with the distal tip in the hypopharynx. The proximal non-latex cuff seals the upper pharynx and the distal cuff the oesophagus.[43] Studies show that it prevents aspiration, is atraumatic and can be used for IPPV; however, it is not a satisfactory device for spontaneous ventilation.[44] There are no data concerning the application of PEEP. When used by experienced personnel, the LTA is comparable to the cLMA and pLMA in ease and time of insertion.[45] Studies comparing the cLMA with the LTA have shown that the incidence of complications was similar but the LTA required more adjustments to obtain a clear airway.[43] Exchange for an ETT using an exchange catheter and a fibreoptic bronchoscope has been reported.[43]

It is as effective during CPR as a bag mask or endotracheal intubation,[46] but there are only limited reports (five cases) of the successful use of the LTA in out-of-hospital CPR.[47] There are no data concerning its use in trauma or in children.

Concern about the blind distal end causing an oesophageal rupture during regurgitation led to the LTSA being developed. The LTSA has two tubes, one for ventilation and the other to allow the passage of a gastric tube for gastric decompression and suction.[43] The efficacy of the LTSA has yet to be determined.

GLOTTIC APERTURE SEAL AIRWAY

The glottic aperture seal airway (GASA) was introduced in 1998.[5] It is not easy to insert but is reported to incur less gastric inflation compared with the cLMA when used for IPPV.[48] Insertion requires the use of a broad semi-flexible retractable blade to elevate the epiglottis anteriorly while the GASA is passed behind the blade until resistance is felt. The blade is then removed and the foam cuff allowed to align itself with the glottic inlet.[5] The foam cushion seals behind the epiglottis and arytenoids. Insertion is more traumatic than with the cLMA.[48] At present this airway is not readily available and there are limited data concerning its use.

COBRA PERILARYNGEAL TUBE

The cobra perilaryngeal airway (COBRA) consists of a tube, a standard 15 mm adaptor at one end, an inflatable cuff (which requires deflation prior to insertion) and a softened distal end (shaped like a Cobra's head). The distal end has slotted openings on one side which, when correctly positioned in the hypopharynx, are opposite the laryngeal opening.[5,49] The appropriate size for the patient's weight is marked on the tube. It is inserted blindly along the midline of the tongue. A recent study was abandoned because of lung aspiration of gastric contents in two subjects.[50]

STREAMLINED LINER OF THE PHARYNGEAL AIRWAY

The streamlined liner of the pharyngeal airway (SLIPA™) is a new, inexpensive, disposable EAD designed to seal the airway without the use of an inflatable cuff and has features designed to reduce the aspiration risk. Shaped like a hollow boot, it is made of soft plastic and hence flexible, allowing it to be 'squeezed' between the teeth in limited opening situations. Insertion is easy but requires the flat side to face the patient's back, the jaw to be lifted forward and the device lubricated. Once inserted the flatter hollow portion (which consists of the heel, toe and bridge sections) faces the laryngeal inlet. The 'central' bridge fits into the pyriform fossae at the base of the tongue. The toe of the chamber slips easily into the entrance of the oesophagus where it seals against the crico-pharyngeal sphincter. The heel anchors the SLIPA™ in position.[5,51]

Comparative study of 120 patients by Miller and Light demonstrated that the SLIPA™ compared favourably with the cLMA in ease of insertion, ventilatory capacity, post-extubation morbidity, haemodynamic changes associated with insertion, and prevention of aspiration if secretions or blood accumulated in the pharynx or if regurgitation occurred.[51] Airway seal equalled the cLMA but gastric inflation is possible with IPPV if too small a size is used. There are six adult sizes. The size is estimated by measurement of the patient's translaryngeal diameter and its comparison with the SLIPA's diameter.[51] At present it is not readily available and more studies are needed.

PHARYNGO-TRACHEAL LUMEN AIRWAY

The pharyngo-tracheal lumen airway (PTLA) is a double-cuffed, double-lumen tube which allows ventilation following placement in either the oesophagus or trachea. The operator inflates the two cuffs orally. It has been used successfully in pre-hospital CPR and as a method of emergency airway management. The PTLA is not readily available and has limited data supporting its use.[5,52]

PHARYNGEAL AIRWAY EXPRESS

The pharyngeal airway express (PAxpress) was released recently and has few data concerning its efficacy and safety. It consists of an anatomically curved polypropylene tube, an inflatable midsection circular cuff and a non-inflatable gilled conical cuff at the distal end. It is easily inserted, is atraumatic to the upper airway, allows effective IPPV with a low risk of gastric inflation but is haemodynamically stressful during insertion.[5,53] Only one size, for adults greater that 40 kg, is manufactured, and it is not readily available.

AIRWAY MANAGEMENT DEVICE

The original airway management device (AMD) was released in 2000. It was similar in appearance to the LTA with a blind distal end but had two pilot balloons for cuff inflation. It is inserted in a similar manner to the LTA. Studies concerning its use, however, were unfavourable – tongue congestion, airway obstruction following insertion and regurgitation being reported.[54] It has subsequently been modified, the sizes have changed making it easier to choose an appropriate size for an adult, and the inflated cuffs have been modified in size and shape. A direct comparison with other EADs is needed but a recently published study of 50 patients showed that the modified AMD was easy to insert, atraumatic, and provided a reliable patent airway that could be suitably used in anaesthesia.[55] More studies are required in the pre-hospital and CPR situations.

Discussion

The majority of the EADs reviewed:

- failed to meet the criteria outlined in Table 2
- lacked substantive data concerning their use in CPR, trauma and anaesthesia and/or
- had small patient numbers in published studies.

The OTC, SLIPA™, pLMA and LTSA have limited data to support their use in resuscitation; however, the OTC and cLMA are the only EADs with a Class lla recommendation from the American Heart Association (the weight of evidence/opinion is in favour of its usefulness/efficacy).

The problems associated with the use of the cLMA and other laryngeal masks in emergency management – the lack of airway protection from aspiration, conflict with the use of CP, the risk of gastric inflation with IPPV, particularly if high inspiratory pressures are needed, and the inability to apply PEEP and decompression or suction of the stomach – are not associated with the pLMA. Its design isolates the respiratory tract from the gastrointestinal tract and allows IPPV with PEEP without a substantial airway leak or gastric inflation and allows the passage of a gastric tube to decompress the stomach. Few complications have been reported in association with its use but it needs securing once positioned and it is not easily replaced with an ETT. There are also other potential limitations for the use of the pLMA for resuscitation: it is more complex to understand, more difficult to insert and must be correctly positioned for it to be used safely. In addition, there are no data, at present, on its use in resuscitation. Its main use is that it acts as a 'bridge' between the use of a cLMA and endotracheal intubation and if the user is trained and skilled then it is potentially a very useful EAD in the trauma/resuscitation situation.

Gastric suction and deflation of the stomach cannot be performed if the SLIPA™ is used and there are no data concerning its ease of replacement with an ETT or the use of PEEP. There are no data on the use of the LTSA with PEEP and once positioned it needs to be constantly monitored to ensure that it remains correctly placed. More data are needed before the SLIPA™ and LTSA are routinely recommended for use in CPR or trauma and hence they are not recommended for beach resuscitation of the ND victim.

The OTC compares favourably with the use of an ETT in the emergency setting. The main limitations to the use of the OTC are a lack of any paediatric sizes (although it can be used in patients of a height greater than 117 cm – a 9- or 10-year-old child), the latex oropharyngeal cuff, the intra-cuff pressures and its reported rare complications of oesophageal and laryngeal damage. It is important to realise that the efficacy of the airway seal obtained with the OTC may vary with the individual's laryngopharyngeal anatomy and, therefore, using a fixed cuff inflation value is not recommended. The cuffs should be inflated until an acceptable airway seal is obtained and intra-cuff pressure monitoring should become routine when available. The ease of insertion, the lack of the need of any additional equipment, protection of the airway from aspiration, the ability to deflate the stomach in either the oesophageal or tracheal positions, and the ability to apply IPPV and probably PEEP make the

OTC the first choice in the resuscitation of ND victims with a height greater than 117 cm.

Recommendation of a particular EAD for the resuscitation of ND children is difficult. Several choices are available, none fulfilling all requirements. If the operator is skilled in the use of the pLMA then this would be the EAD of first choice. The LTSA has merit but more data on paediatric patients are needed. If the clinical situation dictates that the only choice is between using the cLMA and BMV then the cLMA should be used because it does offer some airway protection and better oxygenation than the BMV.

Conclusions

Environmental circumstances, victim size and operator experience all dictate which airway device can be used for resuscitation of the ND victim. This review indicates that the OTC and pLMA are suitable. More data on the LTSA are needed. The OTC is the EAD of choice in teenage or adult ND victims while the pLMA can be used in adults or children if the resuscitator is suitably trained and skilled.

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Short communication

Pre- and post-dive spirometry assessment of recreational scuba divers. A pilot field study

Anne Wilson and Alan Crockett

Key words
Scuba diving, diving research, pulmonary function

Abstract
(Wilson A, Crockett A. Pre- and post-dive spirometry assessment of recreational scuba divers. A pilot field study. *Diving and Hyperbaric Medicine*. 2006; 36: 195-7.)
Purpose: Pre- and post-dive spirometry were conducted by recreational scuba divers in order to determine whether there were acute changes in divers' forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) or the FEV_1/FVC ratio following a dive. Previous studies have been conducted in artificial conditions using hypertonic saline and using professional diving equipment rather than that used by recreational divers.
Methods: Data were collected from qualified scuba divers at six different dive locations. Spirometry was undertaken prior to the dive and within 30 minutes of completing the dive using an Easyone® spirometer.
Results: There were 26 male (72.2%) and 10 (27.8%) female divers. No significant changes in lung function were detected post dive (P = 0.94). However, 8 (22%) divers had pre-dive FEV_1/FVC ratio values below normal signifying mild airways obstruction, and 23 (63.8%) were overweight.
Conclusions: Although there was no significant change in divers' FEV_1/FVC ratio following a scuba dive to indicate bronchial hyperresponsiveness due to salt-water aspiration, further studies using techniques for measuring airways resistance during tidal breathing may be more appropriate for testing this hypothesis.

Introduction

Reporting on human factors associated with scuba-diving fatalities in Australia and New Zealand, Edmonds and Walker pointed out that salt-water aspiration in the conscious diver was an unverifiable factor that relied on data from others and was obscured in the event of drowning.[1] As such, the lack of information on the prevalence of bronchial hyperresponsiveness in the sport-diving population presents difficulties in setting reasonable recommendations for medical standards. The following pilot study was conducted to ascertain whether seawater aspiration during a routine dive might increase the probability of bronchoconstriction. A search of the literature did not reveal any studies that had been conducted on recreational divers in the field.

Methods

Approval was received from the University of Adelaide Human Research Ethics Committee. NHMRC guidelines were adhered to. A convenience sample of 56 qualified divers was recruited through scuba clubs, shops and at dive sites. After giving informed consent, participants completed a short questionnaire requesting relevant health history information and personal demographic data.

Spirometric data were compared with Australian predicted normal limits.[2] Results were grouped according to spirometry variables (e.g., normal (predicted) forced vital capacity (FVC) *versus* abnormal FVC) and individual variables (e.g., age and height). Easyone® spirometers were used to ascertain divers' forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1). An FEV_1/FVC ratio of less than 75% was regarded as abnormal. Assessment was undertaken prior to the dive and within 30 minutes of completing the dive. The spirometry results were assessed according to established standards for lung function testing with spirometry.[3] These criteria revealed more unacceptable results than the quality-assurance algorithm of the spirometer. Data were compared with normal limits and results grouped according to spirometry variables and demographic variables (e.g., age and height). Student's paired t-tests were applied for comparison of nominal data between groups.

Survey forms were de-identified and data entered into SPSS® V.13 for management and analysis of descriptive statistics and frequencies.

STUDY DIVE PROFILES

Dives were conducted at six different sites and included both shore and boat dives. Depths ranged from 3 to 28 metres' sea water (msw). Fourteen dives (39.2%) were conducted under 12 msw and 17 (47.6%) over 21 msw. Length of dives ranged from 25 minutes to over an hour. Efforts were made to take post-dive spirometry measures as soon as possible after the dive.

Results

DEMOGRAPHIC DATA

Of the 36 divers (see below) analysed, ages ranged from 15 to 68 years with a mean of 43 years; 15 (42%) were 46 to 55 years of age. There were 26 male (72%) and 10 (28%) female subjects. According to body mass index (BMI) scales, 18 (50%), were overweight and five (14%) were obese (BMI => 30). Four subjects (11%) were current smokers and nine (25%) were former smokers.

MEDICAL HISTORY

At the time of the dive, four (11%) divers reported they had a respiratory illness. A variety of allergies were reported: drugs – two (6%), animals – two (6%), dust, metal and pollen – three (8%), and nuts – one (3%). Two subjects (6%) reported taking decongestant nasal spray and Sudafed medication before a dive.

Five subjects (14%) indicated they had never undergone a dive medical. For those who had, the mean interval since was five years (median one year, 18 divers; range 1–30 years).

DIVING HISTORY

Diving experience ranged from under one month to 46 years, with a mean of 12.4 years. The average number of dives conducted each month per person was seven (mean) with a range of less than one to 15 per month.

SPIROMETRY

Of the 56 divers recruited, data from 20 divers was either incomplete or rejected on technical grounds.[3] Both pre- and post-dive spirometry data met the standards for acceptability and repeatability for 36 divers (64%) and were analysed.

Critical incidents that affected data collection included one case of ear barotrauma and several of seasickness. Complications due to rough seas and seasickness affected subjects' ability to perform post-dive spirometry.

Compared with before the dive, no statistically significant differences in spirometry measurements were detected post dive (Table 1). Nevertheless, eight (22%) divers had pre-dive FEV_1/FVC ratio values below normal, signifying mild airways obstruction.

Discussion

This pilot study sought to provide information on the prevalence of any acute lung function changes associated with recreational diving. The experience, average age and mean body mass index of the population were found to be consistent with divers of other studies.[4] Of interest were the findings relating to obesity, fitness and medications used.

This study has contributed to new knowledge by being undertaken in the field, as opposed to in the laboratory environment. Some previous studies have investigated expired airflow limitations in professional scuba divers or changes to lung function as a result of exposure to hyperoxia at depth and to decompression stress resulting in venous gas micro-embolism during ascent. Several studies examining bronchospasm and respiratory function in scuba divers with known respiratory dysfunction and allergic respiratory conditions have been identified.

However, only two of these studies considered the relatively shallow dives of sport scuba divers and the pattern of resultant lung function changes that may occur. In addition, these studies were conducted in artificial environments utilising chemical substitutes for seawater.[5,6]

Cirillo et al studied the effects of scuba dives on airway responsiveness in non-asthmatic, atopic subjects and concluded that there is a relationship between the development of early airway hyperresponsiveness and atopic subjects.[6] However, this relationship has also been demonstrated in non-diving atopic subjects. Tetzlaff et al studied 18 male sport divers in a hyperbaric chamber wearing full-face masks rather than using oral demand valves.[5] The study concluded that atopic divers were more susceptible to the effects of diving on lung function than divers without an atopic history and suggested that the mechanical and physiological loads of scuba diving are associated with a

Table 1
Differences between pre- and post-dive spirometry data in 36 scuba divers; mean differences with 95% confidence intervals and paired t-test probabilities are shown
(FVC – forced vital capacity; FEV_1 – forced expiratory volume in 1 second)

Parameter	Mean difference	95% confidence intervals of the difference	P values
FVCpre – FVCpost	-0.066	-0.051 to +0.183	0.261
FEV_1pre – FEV_1post	+0.013	-0.111 to +0.137	0.835
FEV_1/FVCpre – FEV_1/FVCpost	+0.973	-3.075 to +1.129	0.354

reduction in airways conductance. Effects on respiratory function were consistent with small airways dysfunction, which may lead to long-term effects on respiratory function in scuba divers.[5]

In addition, given that half of the divers in the present study were aged 46 years and over, it is reasonable to anticipate that, as the diving population ages, divers will have health needs that require appropriate management to keep them healthy and active. It is imperative that information on risks related to diving be disseminated to the diving public.[7] In 2004, the South Australian coroner reported that of five diving deaths all were not medically fit to dive, due to specific medical conditions, cardiovascular unfitness or being overweight.[8] The coroner's recommendations included regular medical assessments for recreational divers. The use of spirometry during routine medical assessment by general practitioners may detect unforeseen problems.

Conclusions

There was no significant change in divers' pre- and post-dive FEV_1/FVC ratio indicating bronchial hyperresponsiveness due to salt-water aspiration. Due to difficulties faced in the field such as fatigue and seasickness, studies using techniques for measuring airways resistance during tidal breathing may be more appropriate for testing this hypothesis. The incidental findings of unfitness and obesity warrant investigation by further studies.

Acknowledgements

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Diving Medicine Special Interest Group

A diving medicine special interest group has been formed recently within the RACP. Its mission is to promote diving medicine and the specialty of occupational medicine.

Objectives:
- To develop, implement and manage a Certificate of Competency (COP) in diving medicine
- To develop, implement and manage a website of interest and use to practitioners who have an interest in diving medicine
- To conduct a session or lecture relevant to diving medicine as part of each year's AFOM ASM

Proposed regulations for the COP in diving medicine:
- Medical practitioner
- Medical scientist or educationalist at doctoral level who has a primary interest in diving medicine
- Acceptable postgraduate qualification in diving medicine (e.g., SPUMS Diploma, University of Auckland PG Diploma or Masters degree)
- Ongoing commitment to diving medicine

For further information or to join the diving medicine SIG please contact:
<afom@racp.edu.au>

The diving doctor's diary

A case of diving-induced pulmonary oedema

Peter Glanvill

Key words
Immersion, pulmonary oedema, scuba diving, case reports

Abstract

(Glanvill P. A case of diving-induced pulmonary oedema. *Diving and Hyperbaric Medicine*. 2006; 36: 198-200.)
An interesting case of acute immersion pulmonary oedema in a fit middle-aged woman is presented. Intermittent dyspnoea and cough occurred over a seven-year diving history in temperate but not tropical waters. Cool surface weather conditions prior to symptomatic dives may have been a contributing factor.

C, a married pharmacist aged 48, originally contacted me in late August 2001, after encountering medical problems relating to a recent dive.

She had a history of rheumatic fever aged 12, which resulted in restricted physical activity for several months and then her taking penicillin for about two years. There were no apparent sequelae. Several years previous to contacting me she sustained a depressed fracture of the facial bones necessitating surgical repair of both malar and nasal bones, including metal and plastic prostheses, with a satisfactory cosmetic result. She took no medication. She made a point of keeping physically fit with a variety of activities including regular visits to the gym and yoga. Family history is interesting in that her mother always refused to swim in UK waters telling her children she got the "dreaded lurgy". When her daughter was older she told her that she fainted if she entered cold water although could swim in the warm waters of the Mediterranean. Her mother was a physically active individual having been a ballet dancer and is still alive and well. C's daughter has problems when eating ice cream – it makes her feel short of breath.

C learnt to scuba dive in 2000 in Mexico and Thailand and then made a few dives in the UK, none of which she described as being "successful" (Table 1). She found concentration difficult during the dives, which she made in a 7 mm semi-dry suit. At the time she attributed the problems in concentration to difficulty in adapting to the more cumbersome nature of the suit and the extra weights that were required.

Four days prior to contacting me in 2001 she had dived with normal scuba gear, breathing air, to a maximum depth of 18 msw off the south-west Cornish coast (sea temperature 15 °C) – her fourth UK dive. After 15 minutes she ascended from 18 metres, making a fairly rapid journey from 10 metres to the surface as she had lost concentration completely and "felt strange". She then became acutely breathless, felt fatigued and coughed up pink, frothy sputum. She had no neurological symptoms, surgical emphysema or voice changes. She was given oxygen and transferred by boat to shore and then by ambulance to the nearest hospital. She says her oxygen saturation was reportedly low at transfer and that inspiratory crackles were apparently heard over the lungs. She was treated with oxygen and intravenous frusemide. Within four hours her symptoms had remitted and she felt well, with normal blood pressure and oxygen saturation. A chest X-ray was taken on admission and she reported that it showed "fluid on the lung".

She had contacted me because she wished to know when she could return to diving. My initial reaction was that she had had pulmonary barotrauma, but I also wondered about transient pulmonary oedema. Subsequent examination by a chest physician who had some knowledge of diving medicine could find no evidence of pulmonary abnormality and he advised her that she could recommence diving. During the following year she dived uneventfully in the Red Sea and the Far East (Table 1).

She next contacted me in May 2002 after a dive to 14 msw in Portland Harbour, Dorset (sea temperature 13 °C). Coincidentally she was with the same dive buddy and boat skipper as at the time of the previous episode.

She felt unwell at 20 minutes, breathless at depth after 25 minutes and, after a rapid ascent, coughed up pink, frothy sputum on the surface. She was given oxygen and a helicopter transfer to a recompression facility at Poole only 20 minutes away. She was not recompressed but observed overnight having been given intravenous frusemide and oxygen. She felt that the diuretic therapy made her feel worse by dehydrating her. Those treating her were reported to be mystified as to the cause of her symptoms.

I now felt certain she was suffering from cold-water-induced pulmonary oedema and sought the advice of Dr Peter Wilmshurst, consultant cardiologist and member of the UK Sport Diving Medical Committee, copying the enquiry to

her general practitioner who would have to make any formal referrals. In the event she was referred back to the chest physician who had seen her previously.

He performed full lung-function tests and an echocardiogram, all of which were normal. He was not aware of the syndrome of cold water pulmonary oedema but felt that she was clearly putting herself at considerable risk by continuing to dive. C decided to continue diving at her own risk with a full-face mask. This was the state of play in July 2002.

In October 2006, preparatory to this report, I contacted her with regard to her subsequent diving activities particularly in view of the four-year lapse our last contact.

She reported that she had taken her advanced open water PADI course in the UK using a full-face mask and had experienced no problems with dives up to 24 msw in water temperatures ranging from 15 to 18 °C for dive times of up to 40 minutes. She had also dived in Bali, Lombok and the Maldives using ordinary scuba diving equipment for these warm-water dives (Table 1).

Finding the full-face mask uncomfortable she decided to try a dive in the UK with a 'big eye' mask supplemented with a dose of antihistamine (acrivastine) taken 15 minutes prior to the dive. During the summer of 2003 she continued diving in the UK and later in the year dived in Kenya (Table 1).

She did not do any foreign diving that winter but started UK diving again in the summer of 2004 using the large mask/antihistamine combination that appeared to have been successful the previous season. Her first dive on a cool, overcast evening (17 °C) ended in her feeling slightly short of breath with a cough, but she was asymptomatic on three subsequent dives in water of 16 °C. At the end of 2004 she spent a week diving in Egypt (Table 1).

In 2005 she again did no diving until July when, after five uneventful sea dives in the UK, she dived on a cool evening to a depth of 16 msw for 40 minutes in a water temperature of 18 °C. Thirty minutes into the dive she described herself as feeling "very strange" and then developed a cough and dyspnoea. She clipped herself to her buddy and they made a controlled ascent. After breathing oxygen on the surface she

Table 1
Record of patient's diving experience 2000–2006 (WS – wetsuit; SDS – semi-dry suit; DS – dry suit)

Year	Location	Sea temp < 19 °C	No. of dives	Symptoms	Equipment	Comments
2000	Mexico and Thailand	No	17	Nil	Scuba + 5 mm WS	Training dives
2001	UK	Yes	3	Poor concentration	Scuba + 7 mm SDS	First cold-water dives
	UK	Yes	1	Loss of concentration, exhaustion and breathless on surface, pink sputum	Scuba + 7 mm SDS	Rapid ascent from 10 msw Treated by evacuation, O_2 and diuretics.
	Red Sea	No	15	Nil	Scuba + 5 mm WS	
2002	Sipadan	No	20	Nil	Scuba + 5 mm WS	
	UK	Yes	1	Malaise, breathlessness underwater, pink frothy sputum on surface	Scuba + 7 mm SDS	Helicopter evacuation to hyperbaric unit. Treated with IV diuretics and O_2
	UK	Yes	15	Nil	Full-face mask + 7 mm DS	Advanced PADI open water training
	Bali/Lombok	No	19	Nil	Scuba + 5 mm WS	
2003	Maldives	No	25	Nil	Scuba + 5 mm WS	
	UK	Yes	9	Nil	'Big eye' mask + 7 mm SDS	Pre-dive acrivastine
	Kenya	No	17	Nil	Scuba + 5 mm WS	
2004	UK	Yes	3	Slight breathlessness and cough on one dive	'Big eye' mask + 7 mm SDS	Low ambient temperature pre-dive + acrivastine
	Egypt	No	12	Nil	Scuba + 5 mm WS	
2005	UK	Yes	6	On sixth dive, malaise, cough and dyspnoea with pink sputum on surface	'Big eye' mask + 7 mm SDS	Pre-dive acrivastine. Oxygen and rest. Cool evening.
	Manado	No	20	Nil	Scuba + 5 mm WS	
2006	Egypt	Yes	10	Nil	Scuba + 5 mm WS	Nitrox dives – "Clearer headed" post dive

discussed the situation with the skipper (who had witnessed the previous two episodes) and declined an emergency evacuation to hospital on the basis that the symptoms were identical to those that had remitted spontaneously previously. She was driven home and had made a full recovery within four hours with no further treatment and returned to work the next day.

She has since dived in the Far East and in March 2006 did a nitrox course in Egypt with no problems. She has no plans to dive in UK waters in the future as she feels that the risk to herself and the inconvenience to her companions is too great, but she plans to continue diving overseas in warm waters where she has never been symptomatic. As she pointed out, the standard dive declarations do not mention immersion or cold-induced pulmonary oedema!

She made some observations of interest on her symptoms in that prior to the episodes of dyspnoea she was aware of feeling vaguely unwell with a loss of concentration. She had also noticed that the air temperature appeared to have some influence on her attacks of oedema in that they had all occurred when she had dived after being exposed to lower than normal ambient temperatures prior to the dive. She also observed that after using nitrox she felt much livelier than she had breathing air, which leads me to suspect she was suffering from mild hypoxia on more UK dives than she realised but was able to compensate due to her physical fitness and increasing experience. Peter Wilmshurst (personal communication) observed one very physically fit diver (a marathon runner) with a large right-to-left shunt who could tolerate a remarkably low oxygen saturation, suggesting that there is a process of adaptation presumably akin to that occurring in high-altitude climbers.

Commentary

The phenomenon of diving-induced pulmonary oedema is still not well recognised and, in this case, it was only after the second episode that the problem was diagnosed. The trigger in this case appears to be exposure to water colder than 19 °C and, more specifically, exposure of the facial skin to cold water. It is interesting that the episodes occurred not only in colder UK water but also, as the subject observed, on dives where she had been exposed to lower ambient surface temperatures either because of a long boat trip to the dive site in a cooling wind or because the dive was conducted in cooler conditions such as the evening. She has speculated that the presence of significant amounts of metal in her cheek bones might have increased her susceptibility. I suspect that the self-administration of antihistamine had little to do with reducing the number of episodes she sustained.

Previous case reports have described subjects with latent or undiagnosed disease but C remains in good health and specifically is normotensive. I think this makes her case particularly interesting.

It is also worth noting her comment that there is nowhere on a self-declaration form to indicate that one has suffered from this disorder and although it is rare perhaps this needs to be considered. She has made an informed decision based on her experience to continue diving, but only in warm waters.

Reviewing this case report caused me to consider that a number of unexplained diving fatalities could be the consequence of the diver developing pulmonary oedema. Nothing is more likely to induce panic and irrational behaviour than acute breathlessness. C's experience hints at the possibility that the phenomenon may also occur subclinically, the hypoxia resulting in poor decision making and again the possibility of error on the diver's part.

Further reading

- Scubadoc's Diving Medicine Online. Pulmonary edema of diving [monograph on the Internet.] Available from: <http://www.scuba-doc.com/puledem.htm>. Accessed 31 October 2006.

This website describes the phenomenon of pulmonary oedema for the layman.

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This article describes a survey of 1,250 divers with 460 responders, only one of whom had a history suggestive of pulmonary oedema.

- Wilmshurst PT. Cardiovascular problems in divers. *Heart*. 1998; 80: 537-8.

The author describes cases of pulmonary oedema precipitated by diving. He recommends that affected individuals should not dive but that those who insist on continuing to dive should take nifedipine 5 mg pre-dive. At the time of writing this report he was not aware of any further episodes of pulmonary oedema occurring in those who took nifedipine pre-dive.

- Wilmshurst PT. Pulmonary oedema induced by emotional stress, by sexual intercourse, and by exertion in a cold environment in people without evidence of heart disease. *Heart*. 2004; 90: 806-7.

The author describes further cases of pulmonary oedema triggered not only by diving but also by emotional stress and sexual intercourse. He suggests a neurohumoral process producing flash hypertension and acute left heart failure as a possible mechanism.

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Cochrane corner

Hyperbaric oxygen therapy for acute coronary syndrome: a systematic review of randomised controlled trials

Michael H Bennett, Nigel Jepson and Jan Lehm

Key words
Hyperbaric oxygen therapy, cardiovascular, evidence, Cochrane library, review article

Abstract
(Bennett MH, Jepson N, Lehm J. Hyperbaric oxygen therapy for acute coronary syndrome: a systematic review of randomised controlled trials. *Diving and Hyperbaric Medicine*. 2006; 36: 201-7.)
Background: During an ischaemic event, hyperbaric oxygen therapy (HBOT) will improve oxygen supply to the threatened heart and may reduce the volume of heart muscle that will perish. This may reduce death rate and other major adverse outcomes following acute coronary syndrome (ACS). This review assesses the randomised clinical evidence for benefit or harm from HBOT in this setting.
Methods: We performed a systematic search of the literature and made a pooled analysis of predetermined outcomes where possible.
Results: There was a trend towards a decrease in the risk of death with HBOT (relative risk 0.64, 95% CI 0.38 to 1.06, P = 0.08). There was evidence from individual trials of reductions in the risk of major adverse coronary events (MACE) (RR 0.12, 95% CI 0.02 to 0.85, P = 0.03; NNT 4, 95% CI 3 to 10) and some dysrhythmias (RR 0.59, 95% CI 0.39 to 0.89, P = 0.01; NNT 6, 95% CI 3 to 24) following HBOT. The time to relief of pain was reduced with HBOT (mean difference 353 minutes shorter, 95% CI 219 to 488, P < 0.0001).
Conclusions: For people with ACS, the addition of HBOT reduced the risk of MACE and some dysrhythmias, and reduced the time to relief from ischaemic pain, but did not significantly reduce mortality. The review was hampered by modest numbers of patients, methodological shortcomings and poor reporting. More research is needed. The routine application of HBOT to these patients cannot be justified from this review.

Introduction

Cardiovascular disease remains the leading cause of death in developed countries, and is predicted to become the disease with the greatest global burden by 2020.[1] In the United Kingdom, coronary heart disease is the most common cause of premature death, causing 125,000 deaths from approximately 274,000 episodes in 2000 at a community cost of around £10 billion.[2,3] Because myocardial infarction (the presence of two out of three of: chest pain, ECG changes and cardiac enzyme rise) is not always diagnosable during an acute event, unstable or persisting ischaemic heart pain (angina) with or without infarction is described as acute coronary syndrome (ACS).

The main underlying problem in coronary heart disease is atherosclerosis – a degenerative process characterised by the formation of plaques comprising platelets, cells, matrix fibres, lipids, and tissue debris in the vessel lumen. While such plaques are often complicated by ulceration of the vessel wall with obstruction to blood flow, such ulceration is not necessary for plaques to be problematic.[4] An unstable plaque (coronary atheroma vulnerable to rupture and fissure, and associated with thrombus formation) can lead to an acute coronary syndrome without the artery being totally occluded, and infarction may follow.[5] A significant proportion of patients admitted with acute myocardial infarction (AMI) will suffer a major morbidity or mortality, even when thrombolysis or angioplasty is used to relieve the obstruction.[6]

Hyperbaric oxygen therapy (HBOT) has been proposed as an adjunctive measure to improve outcome following ACS, being first reported in a canine experimental model in 1958,[7] and in a human in 1964.[8] Several uncontrolled human studies have been published since, generally with indications of benefit measured as a reduction in mortality or improvements in haemodynamic or metabolic parameters.[9,10]

The administration of HBOT is based on the argument that the myocardium is hypoxic, and that HBOT can reverse that hypoxia in areas that are marginally perfused. This effect is achieved by greatly increasing the diffusion gradient down which oxygen moves from the blood to the myocyte. Improved oxygen availability may also improve outcome through the effects of oxygen as a modulator of tissue repair. Oxygen has been shown to increase the expression of antioxidant enzymes in both tissues and plasma through an increase in glutathione levels,[11,12] to reduce the degree of lipid peroxidation[13] and to prevent the activation of neutrophils in response to endothelial damage, thus modifying ischaemia-reperfusion injury.[14]

Despite over 40 years of interest in the delivery of HBOT in

these patients, relatively little clinical evidence exists for the assertion that such an intervention improves outcome.

Methods

Using specific search strategies for a wide range of sources, we aimed to locate all randomised controlled trials that investigated the effect of HBOT for ACS. Any trial administering HBOT between 1.5 ATA and 3.0 ATA with treatment times between 30 minutes and 120 minutes on at least one occasion was eligible.

Each reviewer independently assessed the electronic search results and selected potentially relevant studies. Disagreements were settled by examination of the full paper and consensus. To assess methodological quality and detect potential sources of bias we used the methods detailed in section six of the *Cochrane handbook for systematic reviews of interventions*.[15] To allow an intention-to-treat analysis we extracted the data reflecting the original allocation group where possible. Disagreements were again settled by consensus.

Important clinical outcomes were predetermined and each trial accepted into the review must have reported at least one of the following: death, major adverse coronary events (MACE – this includes death, recurrent MI or need for urgent revascularisation by coronary artery bypass graft (CABG) or percutaneous coronary angioplasty), significant dysrhythmia, onset of cardiac failure, time to relief of cardiac pain, size of infarct area, magnitude of cardiac enzyme changes, left ventricular function, length of stay, myocardial perfusion, quality of life (QOL), re-admission, costs for the delivery of care or adverse effects of therapy.

STATISTICAL ANALYSIS

Following agreement, the data were entered into Review Manager® 4.2.1. (Cochrane Collaboration, Oxford, UK). For dichotomous outcomes such as mortality, we calculated relative risk (RR) with 95% confidence interval (CI). A statistically significant difference from control was assumed when the 95% CI of the RR did not include the value 1.0. For continuous outcomes such as the mean time to pain relief for each group, we calculated the mean difference (MD) between groups with 95% CI. We used a fixed-effects model where problematic heterogeneity between the studies was not likely, and a random-effects model where such heterogeneity was likely. Heterogeneity was deemed problematic if the I^2 analysis suggested more than 30% of the variability in an analysis was due to systematic differences between trials rather than chance alone.[16] Consideration was then given to the appropriateness of pooling and meta-analysis. Number needed to treat (NNT) with 95% CI was calculated when the relative risk estimates were statistically significant.

We planned sensitivity analyses for missing data using best-case and worst-case scenarios for imputing outcome. We also considered subgroup analysis based on the inclusion or otherwise of thrombolysis in both arms of the trial, the nature of comparator treatment modalities, the dose of oxygen received, the presence or absence of cardiogenic shock and the site of the myocardial infarction.

Results

THE INCLUDED STUDIES

The initial search produced ten possible relevant randomised comparative trials. After appraisal of the full publications, five of these reports were accepted into the review.[17-21] Shandling 1997 and Stavitsky 1998 are reports from the same study, the Hyperbaric Oxygen Therapy for Myocardial Infarction (HOTMI) Study, but they report different outcomes and so have both been included. These trials included a total of 425 participants, 210 receiving HBOT and 215 control (see Table 1 for a summary of the characteristics of these studies).

All studies involved the administration of 100% oxygen at a pressure of 2 atmospheres absolute (ATA) for between 30 and 120 minutes; however, the total number of treatment sessions varied between studies. The lowest number administered was a single session (Stavitsky 1998; Swift 1992), while the highest was a maximum of 16 treatments within 48 hours (Thurston 1973). All trials included participants with acute myocardial infarction and Sharifi et al 2004 also included individuals presenting with unstable angina. Only Swift 1992 described allocation concealment and blinded subjects to allocation with a sham HBOT session. The time from presentation to enrolment varied from 'within one week' (Swift 1992) to 'within 24 hours' (Thurston 1973) and 'within six hours' (Stavitsky 1998; Shandling 1997). Sharifi 2004 did not state any time. The primary purpose of three of these reports was the treatment of AMI with HBOT, while for Swift 1992 it was the use of HBOT in AMI patients to identify myocardial segments capable of functional improvement, and for Sharifi 2004 the effect of HBOT on re-stenosis following percutaneous coronary interventions.

All trials excluded those unfit for HBOT, but in addition Stavitsky 1998 and Shandling 1997 excluded subjects who were not suitable for thrombolysis (e.g., recent stroke), those with previous transmural AMI and those in cardiogenic shock, while Swift 1992 excluded those with uncontrolled heart failure and/or significant ongoing angina. Thurston 1973 excluded subjects over 70 years old and those presenting when there was no HBOT chamber available. Sharifi 2004 excluded those who continued to show evidence of ischaemia after 30 minutes of medical treatment.

All patients required a clinical diagnosis of AMI for enrolment in these studies except in Sharifi 2004, who also enrolled subjects with unstable angina. All patients in that study had presumed coronary arterial lesions where a percutaneous stent was indicated and so were a more highly selected subset of ACS patients.

Table 1
Characteristics of included studies (AMI – acute myocardial infarct ; LVEF – left ventricular ejection fraction; MACE – major adverse coronary events; RCT – randomised controlled trial; TOE – transoesophageal echo)

Study	Methods	Participants	Interventions	Outcomes
Stavitsky 1998[18]	Multicentre RCT. No blinding. 16 were excluded after randomisation.	138 patients enrolled in emergency room with clinical diagnosis of AMI and eligible for thrombolysis.	Control: Thrombolysis, aspirin, heparin and IV nitroglycerine. HBO: Same plus 2 ATA O_2 for two hours.	Death, time to pain relief, enzyme changes, LVEF.
Shandling 1997[19]	As for Stavitsky 1998.	82 patients.	As for Stavitsky 1998.	Length of stay.
Sharifi 2004[17]	RCT, no blinding. 5 crossed after allocation.	69 patients with AMI or unstable angina. Excluded if pain or S-T segments unresolved after 30 min.	Control: Stenting, aspirin, heparin and clopidogrel. HBOT: Same plus 2 ATA O_2 for 90 minutes at 1 and 18 hours.	MACE, adverse events.
Swift 1992[20]	RCT with 2 active-arm patients for each control. No loss to follow-up. Subject and assessor blinding.	34 patients with a clinical diagnosis of AMI within one week, plus abnormal wall motion on TOE.	Control: Echo, followed by 2 ATA air for 30 mins and repeat echo. HBOT: Same but 2 ATA O_2.	Improved LV function on echo.
Thurston 1973[21]	RCT, no blinding after allocation.	221 patients with strong clinical suspicion of AMI at admission. 13 later excluded.	Control: "Coronary care including oxygen by mask." HBOT: 48 hours of cycling from 2 ATA O_2 for 2 hours, then 1 ATA air for 1 hour.	Death, significant dysrhythmia, adverse effects.

The follow-up period varied from the period immediately following HBOT (Swift 1992), to three weeks (Thurston 1973) and eight months (Sharifi 2004). Stavitsky 1998 reported mortality to discharge from hospital. Study quality was generally assessed as low and quality was not used as a basis for sensitivity analysis.

Swift 1992 reported no losses to follow up or any violation of treatment protocol. Stavitsky 1998 and Shandling 1997 reported 16 subjects withdrawn from analysis after allocation to groups (four became unstable, four generated incomplete data, three were enrolled after six hours in violation of inclusion criteria, two showed no cardiac enzyme rise, two received an incorrect treatment protocol and one refused to have HBOT). Thurston 1973 similarly did not report data on 13 subjects who were withdrawn for misdiagnosis or being aged more than 70 years in violation of inclusion criteria. The group allocation was not indicated for any of the withdrawn patients in either of these studies.

Sharifi 2004 excluded nine subjects allocated to HBOT from the analysis, five of whom were crossed over to the control arm after declining to receive HBOT. The other four participants required CABG or did not have a lesion suitable for stent, while there were also four subjects excluded from the control group for the same reasons. None of the included studies specifically indicated an intention-to-treat approach, and such an approach was not possible for Sharifi 2004 as five subjects crossed from HBOT to control for analysis.

CLINICAL OUTCOMES

Statistical pooling was not possible for the majority of pre-planned outcome measures due to lack of suitable data. Problems included the small number of studies, modest number of patients, and the variability in outcome measures employed.

Three trials reported the number of subjects who died at any time after enrolment (Sharifi 2004; Stavitsky 1998; Thurston 1973), involving 391 subjects, with 186 (48%) allocated to standard treatment plus HBOT and 205 (53%) to standard therapy alone (Figure 1). Fewer subjects died following HBOT, but the difference was not statistically significant (18 (9.7%) versus 29 (14.1%), RR 0.64, 95% CI 0.38 to 1.06, P = 0.08), nor was there any statistically significant reduction on subgroup analysis for those presenting in cardiogenic shock (RR with cardiogenic shock 0.57, 95% CI 0.3 to 1.09,

P = 0.09, RR without cardiogenic shock 0.65, 95% CI 0.35 to 1.2, P = 0.17). The overall comparison was sensitive to the allocation of withdrawals (best-case RR of death with HBOT is 0.42, 95% CI 0.26 to 0.70, P = 0.0008).

MACE were reported only by Sharifi at eight months (61 subjects), with one subject (4%) suffering a MACE following HBOT versus eight subjects (35%) in the control group (RR 0.12, 95% CI 0.01 to 0.61, P = 0.01). This result was also sensitive to the allocation of withdrawals (worst-case RR 0.56, 95% CI 0.23 to 1.40, P = 0.22). The number needed to treat (NNT) to avoid one extra MACE was 4, (95% CI 3 to 10).

Thurston (1973, 208 subjects) reported the incidence of significant dysrhythmia (complete heart block, ventricular fibrillation or asystole). It is not clear if the numbers reported reflect individuals who suffered these events, or the number of events in total. Overall there were 25 such events reported in the patients receiving HBOT versus 43 such events in the control group, and patients receiving HBOT were significantly less likely to suffer one of these dysrythmias (RR 0.59, 95% CI 0.39 to 0.89, P = 0.01; NNT 6, 95% CI 3 to 24). Again, this result was sensitive to the allocation of withdrawals (worst-case RR 0.73, 95% CI 0.50 to 1.06, P = 0.10). Separate analyses for each of the three dysrythmias suggested HBOT patients were significantly less likely to suffer with complete heart block (RR 0.32, 95% CI 0.12 to 0.84, P = 0.02), but not ventricular fibrillation (RR 0.78, 95% CI 0.36 to 1.71, P = 0.54) or asystole (RR 0.73, 95% CI 0.73 to 1.56, P = 0.42) (Figure 2).

Stavitsky (1998, 81 subjects) reported a statistically shorter mean time to pain relief in the HBOT group (261 minutes versus 614, MD 353 minutes, 95% CI 219 to 488, P < 0.0001) but not significantly lower creatine phosphokinase (CPK) level at 12 and 24 hours, nor the maximum CPK level recorded (e.g., maximum CPK in HBOT group 1,698 units versus 2,111 units with control, MD 413, 95% CI -982 to 156, P = 0.15).

Two trials reported improvements in left ventricular (LV) function; however, pooling was not appropriate. Swift 1992 reported the number of individuals in whom improved function could be demonstrated on echocardiography following HBOT. Twelve out of 24 (50%) showed improved function in at least one segment following HBOT versus 0 with control (RR 0.09, 95% CI 0.01 to 1.4, P = 0.09). Stavitsky 1998 reported left ventricular ejection fraction (LVEF) at discharge (mean LVEF with HBOT 51.7% versus 48.4% with control therapy, MD 3.3%, 95% CI -1.1 to 7.6, P = 0.14).

Shandling 1997 reported the length of stay in the first 63 subjects of the HOTMI study. The mean days' stay in hospital for the HBOT group was 7.4 days versus 9.2 days for the controls. This difference was not statistically significant (MD 1.8 days, 95% CI 3.7 to -0.1, P = 0.06).

With regard to the adverse effects of therapy, two trials (Sharifi 2004, Thurston 1973), involving 269 subjects, reported that one patient suffered tympanic membrane rupture in the HBOT group versus none of the controls (RR with HBOT 4.56, 95% CI 0.19 to 107.54, P = 0.35). Three trials (Sharifi 2004, Shandling 1997, Thurston 1973) involving 335 subjects reported a zero incidence of neurological oxygen toxicity in all arms. Thurston reported a significant incidence of claustrophobia in the monoplace setting, 15 subjects (15%) with claustrophobia requiring cessation of therapy in the HBOT group versus none in the control group (RR 31.6, 95% CI 1.92 to 521, P = 0.02).

ECONOMIC ANALYSIS

None of the included trials made any attempt at economic analysis. Using the effectiveness estimates from this review, combined with data reported by Gomez-Castillo,[22] the cost of avoiding a single extra episode of MACE by using HBOT is estimated at $AUD6,080 (95% CI $4,560 to $15,200) assuming five treatments, and $AUD18,240 (95% CI 13,680 to 36,480) assuming 15 treatments (in fact, Sharifi used only two treatments). This estimate should be interpreted with caution given the paucity of data from which it is drawn.

Discussion

There is limited evidence that HBOT reduces the incidence of both MACE and complete heart block, and reduces the time to relief from angina when administered to patients with ACS. Although there was a trend toward favourable outcomes, there were no reliable data from these trials to confirm or refute any effect of HBOT on mortality, length of stay or LV contractility. Only four trials with 425 participants were available for evaluation using our planned comparisons, and meta-analysis was not appropriate or possible for a number of these. Other problems for this review were the poor methodological quality of most of these trials, variability in entry criteria and the nature and timing of outcomes, and poor reporting of both outcomes and methodology. In particular, there is a possibility of bias due to different anatomical locations and extent of myocardial damage on entry to these small trials, as well as from non-blinded management decisions in all except Swift 1992.

Patient inclusion criteria were not standard, and poorly reported in some trials. Only Stavitsky and Swift clearly indicated the time at which the inclusion criteria were applied. There was significant variation both in oxygen dose during an individual treatment session, and in the number of sessions administered to each patient. While all trials used some form of 'standard' cardiac therapy in a dedicated unit designed to maximise outcome, these comparator therapies were generally poorly described and could not form the basis of a meaningful subgroup analysis.

Pooling of data for clinical outcomes of interest could

Figure 1
Forest plot of the risk of death with HBOT; subgroup analysis by presence or absence of cardiogenic shock

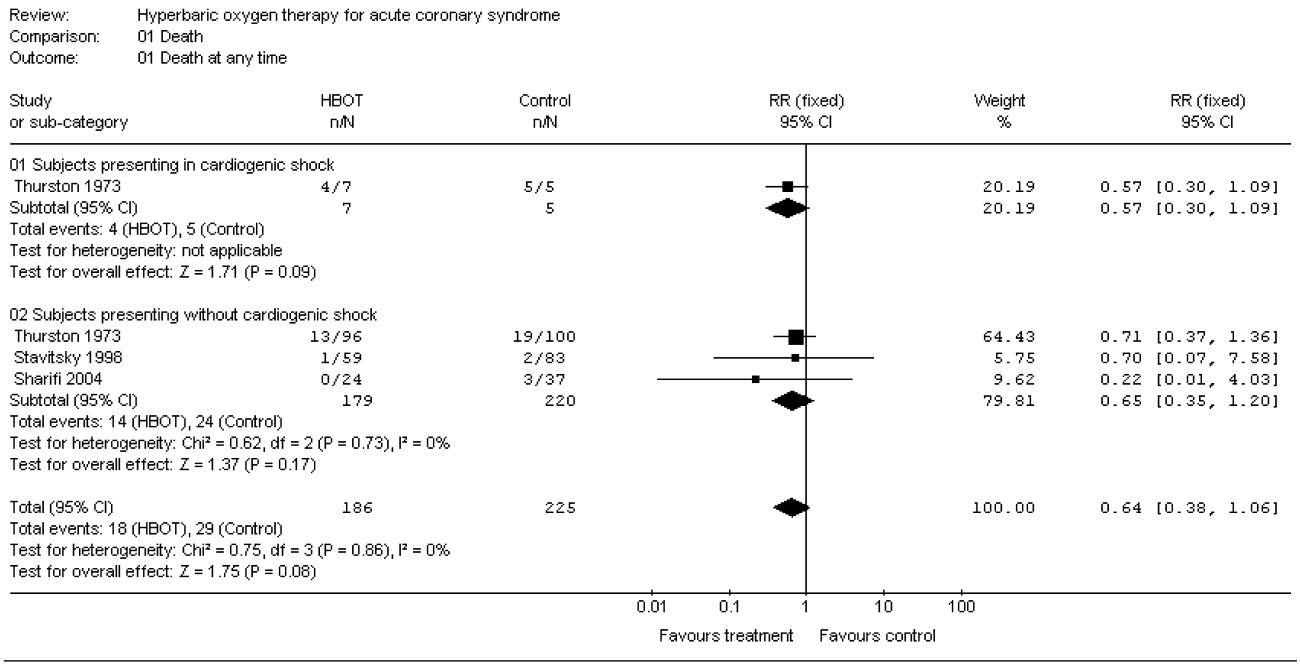
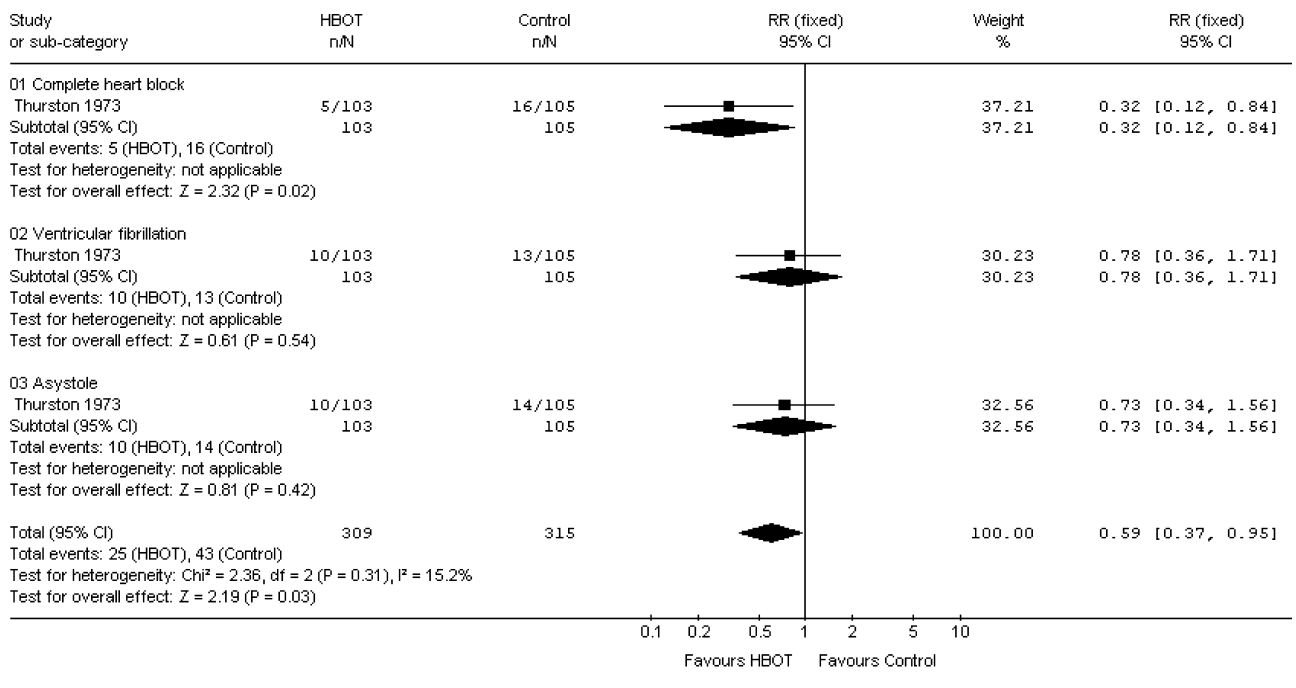


Figure 2
Forest plot of the risk of significant dysrhythmias with HBOT; subgroup analysis by the nature of dysrhythmia



be performed only with respect to the risk of death and adverse effects. While the risk of dying was not significantly improved following HBOT, there was some trend in that direction (RR 0.64, P = 0.08) and the absolute risk difference of 3.2% suggested an NNT of around 31 patients in order to save one life by the addition of HBOT. Only one trial (Thurston 1973) reported the fate of those presenting in cardiogenic shock, and while there was no statistically significant difference between groups in this small sample, it is worth noting that all survivors were from the HBOT group (three from seven subjects versus none from five). The one small study that reported MACE rather than death alone (Sharifi 2004) also suggested better outcome with the use of HBOT. This possible treatment effect would be of

great clinical importance and deserves further investigation. At present, given the small numbers and the sensitivity of the risk of both death and MACE to the allocation of withdrawals, this result should be interpreted with caution. The routine adjunctive use of HBOT in these patients cannot yet be justified by the clinical evidence.

Given the indicative findings of improved outcomes with the use of HBOT in these patients, however, there is a case for large randomised trials of high methodological rigour in order to define the true extent of benefit (if any) from the administration of HBOT. Specifically, more information is required on the subset of disease severity and timing of therapy most likely to result in benefit. Given the activity of HBOT in modifying ischaemia-reperfusion injury, attention should be given to combinations of HBOT and thrombolysis in the early treatment of acute coronary events and the prevention of re-stenosis after stent placement.

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The Cochrane Library is available at: <http://www3.interscience.wiley.com/cgi-bin/mrwhome/106568753/HOME>. Reprints of the full-text version are available online from this site.

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Guest Speaker

Neal Pollock, PhD

Themes

From mountain high to ocean deep – the physiological challenges of extreme environments
Workshop: Medical aspects of technical diving

Neal Pollock is a research physiologist at the Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, Durham, NC. He is also heavily involved in DAN International and was chief editor of the recent DAN guidelines for scuba diving and diabetes. He has worked regularly in Antarctica and been involved in high-altitude physiology studies. He thus brings a wealth of expertise to our meeting and is an excellent speaker.

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SPUMS notices and news

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2 The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved Hyperbaric Medicine Unit.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, and in a standard format, for approval by the Academic Board before commencing their research project.
5 The candidate must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

The candidate must contact the Education Officer to advise of their intended candidacy, seek approval of their courses in Diving and Hyperbaric Medicine and training time in the intended Hyperbaric Medicine Unit, discuss the proposed subject matter of their research, and obtain instructions before submitting any written material or commencing a research project.

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

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice (available at http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm) or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documented evidence of approval by an appropriate research ethics committee. It is expected that the research project and the written report will be primarily the work of the candidate.

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Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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SPUMS Annual General Meeting 2007

The SPUMS AGM 2007 is to be held in the Marina Room, Oceans Resort, Tutukaka, Northland, New Zealand, at 1700 hr, Friday 20 April 2007.

Agenda

Apologies:
Minutes of the previous meeting:
Minutes of the previous meeting will be posted on the meeting notice board and appeared in *Diving and Hyperbaric Medicine*. 2006; 36(3): 162-6.
Matters arising from the minutes:
Annual reports:
President's report
Secretary's report
Education Officer's report
Annual financial statement and Treasurer's report
Subscription fees for 2007:
Treasurer to propose a motion
Election of office bearers:
Nil
Appointment of the Auditor:
Business of which notice has been given:
Nil

Report on Australian and New Zealand Standards Occupational Diving Committee Meeting

Held on Monday 29 May 2006 at Standards Australia House, Sydney

Opened: 0930 hr

1 Australian and New Zealand Standard 2299.1 Occupational Diving Operations Part 1: Standard Operational Practice

The 2006 version for public comment has been available on the Australian Standards website and publicly released since 2006. Closure for comment was 6 March 2006. At the meeting a number of public comments were addressed. They can be summarised as follows:

Stand-by diver preparedness in diving operations
This generated a lot of discussion; however, for occupational diving the stand-by diver is required to be immediately available to attend the operational diver should there be an accident requiring assistance.

a Use of DCIEM tables
i The new standard stops just short of mandating the use of DCIEM tables; however, it has now moved away from the use of other tables and recommends use of DCIEM tables.

b Retaining records
i The new standard will mandate that records are maintained for a minimum of 12 months; however, individual diving operations will be required to comply with State Legislation. Previously the standard mandated 7 years, which was not consistent with some State or Federal Legislation.

c Use of buoyancy compensators in professional diving
i Where surface-supply breathing apparatus is used – buoyancy compensators will be recommended based on a risk assessment; however, they are not mandatory. It was pointed out that in professional diving, where there is risk of buoyancy compensators malfunctioning in an enclosed environment or amongst potential entanglement hazards, there could actually be a risk to life.
ii Buoyancy compensators when using scuba equipment for operational diving are mandatory.

d Recompression chambers
i This clause is being completely re-written now to incorporate the following:
ii On-site recompression chambers continue to be mandatory if diving is greater than 30 metres.
iii All other diving operations are risk-rated based on the overall risk of the operation. This would take into account whether or not a chamber should be less than or greater than 2 hours' availability.

e Risk assessment
i A major revamp of the Australian Standard has taken place to require risk assessment in advance of the diving operation. This places a lot more responsibility on the diving operators to produce a risk assessment, and treat all available risks and not proceed should the risk be too high. In addition a risk assessment form is in the process of being produced that would provide a template for those without this paperwork to see the level of documentation that is required prior to the diving operation. Most of this information is part of mandatory occupational health and safety legislation anyway and it has just been updated in the Australian Standard.

GENERAL COMMENT

The Australian and New Zealand Standard has generally been updated and improved with its referencing to particular legislation. Overall, there are a few changes that will be of impact to medical practitioners. The Australian Standard medical form has been slightly changed so that it is only four pages. It also includes a section for the diver to authorise the assessing doctor to contact the diver's GP for information relating to their diving health. Most diving doctors will not note any major differences with the medical assessment forms.

2 Review of the summary of comment for draft Australian Standard 2815.5 Training and Certification of Occupational Divers Part V: Dive Supervisor

This document is a continuation of the 2815.XXX series, which governs the training of occupational diving in Australia. The first four parts of the series are as follows:

2815 Part 1: Basic air diving without tools and to depth less than 30 metres
Part 2: Air diving to 30 metres using underwater tools
Part 3: Air diving to 50 metres including wet bell diving
Part 4: Saturation diving.

Part 4 now covers the training and certification of the dive supervisor. This is complementary to current series.

The Dive Supervisor Part 5 Standard covers the training course content, the role of the dive supervisor, the implementation and monitoring of occupational health and safety programmes, managing dive illness and dive emergencies, dive plan operations, conducting dive operations, plant equipment and maintenance procedures, people management, supervision of tools and explosive in a

dive operation, wet bell diving supervision, and development of trading plans associated with the dive supervisor's work.

Medical practitioners working in the field of occupational diving medicine should be familiar with these standards. They are an integral part of the development of appropriate training standards for the occupational diving industry in Australia.

3 International Standards Organisation TC228/WG1. Title – Tourism Services – Diving Services

I reported on the developments of the International Standard in my last report to the SPUMS Executive and Membership. The Occupational Diving Committee of Australian Standards registered strong objections to the International Standards, which emanated from Europe and covered the competencies and training of recreational scuba divers through to recreational instructors. There were many deficiencies in these Standards, which appeared to be very light on detail compared with those produced in Australia. Despite our objections, we were but a small component of the countries voting, which were dominated by those European countries from which the Standards had originated. As such the Standards are now being worked into public documents for comment at international level.

There appears to be some political pressure for Australian Standards to adopt International Standards where these are equivalent to or in the absence of similar Australian Standards.

THE PROCESS FROM HERE

Once the public documents are circulated, the Australian Standards Occupational Diving Committee will again register their objections to these International Standard documents.

It appears that we do not have to take up these Standards unless there is a review of similar Australian and New Zealand Standards in the recreational diving industry in the near future. There is also the possibility of extensively modifying the Standard should it be deficient in the view of the Occupational Diving Australian Standards Committee. At this point there is nothing further to report on the International Standards document: however, it may become a significant issue in the future, especially if there is an attempt to force the document upon the Australian diving community.

4 Future directions

This was a two-day meeting and unfortunately I was able to attend only the first day. Issues to be covered on Day 2 included DCIEM ascent rates; commencement of revision of AS2299.2, including nitrox scientific diving; recommendations from the Queensland Coroner after a recreational diving death; and Standards Australia strategic plan. I will report on these other issues after the minutes are released.

The Occupational Diving Committee will now focus its attention on the rest of the Australian Standard 2299.2, .3, and .4, as well as commencing a review of the training certification 2815.1 through to 4.

Dr David Smart
SPUMS Representative, Australian Standards for Occupational Diving

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Minutes of the Annual General Meeting of the Australian and New Zealand Hyperbaric Medicine Group, Saturday 26 August 2006

Opened: 1700 hr

1 Present
B Long, Wei Ch'ng, G Hawkins, M Davis, M Bennett, D Griffiths, J Lehm, D Smart, D Wilkinson

2 Apologies
M Walker, M Hodgson, I Millar, B Trytko, H Oxer, B Wong, B Webb, A Gibson, B Boch, B Spain

3 Welcome
Dr Smart welcomed Dr Richard Smerz DO as a visitor to the meeting and thanked him for his involvement. Dr Smerz was one of the HTNA visitors for 2006.

4 Office bearers
Nominations were called for positions of Honorary Chair and Secretary. Dr Smart was nominated for Chair (Drs Long/Bennett). Dr Wilkinson was nominated for Secretary (Drs Smart/Long). No further nominations were received, no vote was required.

5 Minutes of the 2005 AGM
That the minutes be accepted (Drs Smart/Hawkins). Carried.

6 Business arising
Any matters arising will be discussed on the current agenda.

7 Address by Chair of ANZHMG (Dr Smart)
7.1 Funding for hyperbaric medicine in Australia
This has been a critical issue for hyperbaric medicine units Australia-wide over the last eight years since the intervention of MSAC in funding. ANZHMG MSAC Submission 1054 was completed in May 2003 by the 1054 Supporting Committee, which included four members of the ANZHMG. The recommendations from

the Supporting Committee were that soft-tissue radiation injury/necrosis should be fully funded and that hypoxic non-diabetic problem wounds should be funded for three years whilst data collection on problem wounds occurred across all hyperbaric units in Australia.

In 2004, the ANZHMG unearthed that MSAC had changed the conclusions of the report so that neither condition was recommended for funding. After political lobbying by members of ANZHMG, a Ministerial 3C determination allowed funding for hypoxic non-diabetic problem wounds and soft-tissue radiation injury and necrosis for three years from 1 November 2004 to 31 October 2007. MSAC corresponded with ANZHMG stating that if funding was required beyond 2007, a full submission through MSAC was required once again. MSAC also stated that it was essential that the submission occur at least 12 months in advance of the expiry date.

At this point, data collection with the problem wound study and also the HORTIS trials is only in its early to mid stages because of the requirement for 12 months' follow up. Interestingly the final 1054 report has had international flow-on effects; recently USA health insurers placed soft-tissue radiation injury in the "experimental" area of funding. This has caused uproar in the USA and finally it might motivate the Americans to contribute research in the field.

7.2 Research

7.2.1 Congratulations to Mike Bennett and David Smart for receiving their Doctor of Medicine Degrees in the last 12 months. These academic degrees are very important for a research and academic base of the specialty.

7.2.2 The Wesley Hyperbaric Trust has been set up with David Smart as a Trustee and Mike Bennett on the Scientific Board.

7.2.3 The HTNA has set up a Research Trust, with Martin Hodgson and David Wilkinson on the Selection Panel, to facilitate research for HTNA members, including ANZHMG members.

7.2.4 Despite lack of funds, two Units in Australia have signed on for the HORTIS Trial (RHH and the Wesley), and the Proctitis arm is now closed.

7.2.5 Other achievements in research include:

- Publication of the interim results of the first 110 cases in the Problem Wound Study – congratulations to Glen Hawkins. This research was commenced as a result of initial recommendations by MSAC.
- David Smart, Mike Bennett and Simon Mitchell have recently published a review of transcutaneous oximetry and recommended evidence-based guidelines for its use in selecting patients for hyperbaric oxygen treatment in the SPUMS journal, *Diving and Hyperbaric Medicine*. This paper was presented on Thursday 24 August 2006 at the HTNA Conference.

7.3 SPUMS issues

South Pacific Underwater Medicine Society Journal has changed its name to *Diving and Hyperbaric Medicine*. The emphasis remains appropriately on diving medicine. All hyperbaric units in Australia are requested to start contributing papers to SPUMS. The Editor, Dr Mike Davis, has had considerable difficulties in attracting papers in 2006 leading to the possibility of only three journals for the year – a previously unheard of situation. *Diving and Hyperbaric Medicine* is registered on EMBASE but is not Index Medicus listed. The only way we can get it to a higher level with Index Medicus is through publication of high-quality papers. There has also been some discussion taking place about the amalgamation of SPUMS with EUBS in the academic sense for journal production.

7.4 Other research links

Ian Millar has been establishing links with Monash with a possibility of setting up a data registry that could be used for hyperbaric oxygen in Australia. There is also a proposal for a randomised controlled trial in problem wounds post-surgical amputation.

7.5 Other issues

There has been a push to move low-pressure hyperbaric chambers into Australia using the terminology of 'mild hyperbaric oxygen therapy'. These use low-pressure air for patients (based on the cerebral palsy trial emanating from North America). The placebo group in the cerebral palsy trial also had some benefit– the Hawthorne effect. A potentially dangerous spin-off of this move towards 'mild hyperbaric oxygen therapy' is the use of portable collapsible chambers to deliver treatment. These have not been subject to the rigours of Australian Standards and may be hazardous.

7.6 ANZHMG list of indications

Significant amendments to the ANZHMG list of indications are proposed as part of the agenda, and will be published in *Diving and Hyperbaric Medicine* once Committee agreement is achieved.

8 Timing of AGM

Last year the AGMs of the ANZHMG and the SIG were held concurrently. In view of the differing goals of these two organisations it was felt that future meetings should be held separately. It was suggested that the ANZHMG AGM should be held at the HTNA Annual Scientific Meeting.

9 MSAC report and Federal Government funding issues

Refer 7.1.

10 Hyperbaric Problem Wound Study

It was noted that an article on the first cohort of subjects in this study was published in the Journal (*Diving and Hyperbaric Medicine*). Glen Hawkins was congratulated for his efforts. This is considered an interim report with data contributed by only three facilities. As this is to be considered representative of practice at the national

level, the involvement of other units was strongly encouraged.

11 HORTIS

This international study continues with the involvement of two facilities in Australia. The Proctitis arm is now closed, an encouraging abstract has been presented and publication is anticipated.

12 ANZHMG/SIG list of approved indications for HBO

Dr Smart proposed changes to the current list, including retitling to "Consensus Recommendations" and several alterations to specific indications. It was felt that this topic was going to require considerable thought and discussion and would probably be best addressed via e-mail.

ACTION: Dr Smart to e-mail members to achieve consensus.

13 Introductory Course in Hyperbaric Medicine

This course continues to be successfully run from the Prince of Wales Hospital with considerable input from Drs Walker and Smart. Dates for 2007 are from 26 February to 9 March.

14 Hyperbaric facility accreditation

While facilities were encouraged to apply for accreditation, Dr Smart stated he would defer issues related to accreditation to the SIG. There was some discussion as to whether the ANZHMG should be involved in accreditation, Dr Long pointed out that in the USA the UHMS is involved in all aspects of hyperbaric practice. Other opinion compared the relationship with the SIG to that of the College of Anaesthetists and the ASA, with the SIG responsible for academic standards and QA and the ANZHMG being more political with lobbying and promotional activities. Some division between these differing goals was seen as desirable. For the timebeing, the SIG will deal with this issue.

15 Australian Standards report

Dr Smart reported that a review of AS/NZS2299.1 was expected in 2007 or thereabouts, followed by a review of AS2299.2. Recent changes in the European code, which have flowed into ISO documents, may have influence on some aspects of the Australian Standard. The implications are at this point uncertain. The New Zealand position is also uncertain.

16 Diving and Hyperbaric Medicine (SPUMS Journal)

The change in journal name was noted and contributions from everyone encouraged. The journal is registered on EMBASE but listing on Index Medicus will require continued high-quality submissions.

17 Minimum data set

Discussion of this item was joined by Al Blake, an epidemiologist associated with Burnett and Monash University. His involvement will be in development of software to support a national data set. Three phases in this process were identified.

- Amongst the disparate collection of computerised databases currently in use, Dr Webb is looking to identify commonality and hopefully the parameters of a minimum data set.
- Development of computer software based on above.
- Dr Bennett suggested that each unit should commit to "signing on" and support its use at a national level.

ACTION: Dr Wilkinson will coordinate tabulating the various computer software programmes used in existing databases around Australia and forwarding this information to Al Blake.

18 Clinical trials

The multicentre clinical trial meeting was held separately. A proposal for HBO in partial foot amputation was raised. Further discussion was planned via e-mail.

ACTION: Dr Millar to circulate proposed protocol to members.

19 HTNA issues

Dr Smart attended the HTNA AGM. He discussed a desire to ensure the timing of HTNA AGM did not conflict with the EUBS meeting usually held about the same time. He also requested that information about future HTNA meetings be disseminated in plenty of time to allow more doctors to arrange attendance.

20 Other business

No issues raised.

Closed: 1835 hr

Dr David Wilkinson, Honorary Secretary, ANZHMG

Extension to 3C Ministerial Determination under Item 13015, hyperbaric oxygen therapy

Dr Stephen Blamey, Chair of the Medical Services Advisory Committee (MSAC), has written to the Australian Healthcare Association (7 November) agreeing to a three-year extension to the original 3C Ministerial Determination (1/11/04 – 31/10/07). This will ensure MBS funding (under Item 13015) for hyperbaric oxygen therapy for soft-tissue radiation injury and radio-necrosis and hypoxic problem wounds in non-diabetic patients until 1/11/2010. In the letter Dr Blamey acknowledges the request for further time to complete research into the 'sustainability and credibility' of these treatments. MSAC has now asked that data be made available in May 2009.

Dr David Smart is to be congratulated on his hard work as Chairman of ANZHMG, a sub-committee of SPUMS, to achieve this extension.

SPUMS Annual Scientific Meeting 2006

Are asthmatics fit to dive?

Robyn M Walker

Key words
Asthma, fitness to dive, scuba diving, safety, medical conditions and problems, pulmonary function, bronchial provocation testing, review article

Abstract

(Walker RM. Are asthmatics fit to dive? *Diving and Hyperbaric Medicine*. 2006; 36: 213-19.)
There are many theoretical reasons why asthmatics should be at increased risk when scuba diving: exertion, inspiration of cold and dry air, increased respiratory effort from regulator resistance and increased density of gas. Diving could provoke an acute attack of asthma or increase the asthmatic's susceptibility to pulmonary barotrauma. However, an asthmatic's sensitivity to many of the stimuli encountered during diving is reduced following treatment with inhaled steroids so that lung function and bronchial responsiveness fall within the normal predicted range. The SPUMS medical states that any current evidence of asthma is a contra-indication to diving. The United Kingdom Sports Diving Medical Committee guidelines, however, recommend that individuals who are currently well controlled and have normal pulmonary function tests may dive if they have a negative exercise test. Is it time for a new recommendation regarding fitness to dive for asthmatics?

A brief review of asthma

Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T-lymphocytes, macrophages, neutrophils, and epithelial cells. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversed either spontaneously or with treatment. The inflammation also causes an increase in bronchial responsiveness to a wide variety of stimuli.[1]

ASTHMA PREVALENCE

Asthma is becoming increasingly common in Western countries, affecting between 15 and 35% of children aged 13–14 years.[2] The National Asthma Council Australia estimates that about 40% of all Australians will have respiratory symptoms consistent with asthma at some time in their lives.[3] The European Community Respiratory Health Survey (ECRHS) states that the prevalence of asthma in centres from the United Kingdom, New Zealand and Australia varies from 7.5 to 11.9%.[4] During 1982–1992 in the age range from five to 34 years, the overall annual age-adjusted prevalence rate of asthma increased from 34.6 to 52.6 per 1000 in the United States.[5] Since that time the increase in prevalence in the USA has slowed somewhat.

NATURAL HISTORY

For most children, wheezing before the age of six years is probably a benign condition reflecting smaller airways that will improve or resolve in a few years. In a substantial minority of infants, however, wheezing episodes are probably related to a predisposition to asthma.[6]

Robertson reports that the outcome of childhood asthma is dependent on the pattern of asthma through childhood.[7] Episodic asthma in childhood tends to resolve in adolescence and through mid-adult years, with no impairment of lung function. Persistent asthma is more likely to continue into adult years, with modest impairment of lung function that occurs early in the disease process and is not progressive, despite continuing symptoms.[7]

Taylor et al evaluated the frequency and risk factors for relapse of asthma in a group of 18-year-old patients with previous asthma but in remission.[8] Approximately one-third of study members (35%) with asthma in remission at 18 years of age relapsed by 21 or 26 years of age. Atopy and lower forced expiratory volume in one second (FEV_1)/ forced vital capacity (FVC) ratio at 18 years of age were significant independent prognostic factors for relapse in multiple logistic regression analyses. Increased responsiveness to methacholine or bronchodilator at 21 years of age was more common among those with relapse, but the positive and negative predictive values for a previous positive methacholine challenge test result at 15 years of age were low. Asthma after relapse was generally mild. Totally new adult asthma developed by 26 years of age in 9% of study members who had no asthma or wheezing at any time up to 18 years of age. Taylor et al concluded that subsequent relapse of previously diagnosed asthma in remission at 18 years of age occurs in one in three young adults. Such relapse is not easily predicted, especially by measurements of airway responsiveness to methacholine.[8]

RISK FACTORS FOR ASTHMA

Abnormal bronchial responsiveness is a central feature in the definition of asthma; however, not all patients with bronchial hyperresponsiveness to a pharmacological agent have symptoms of asthma.[9] Atopy, exposure to indoor allergens, outdoor pollution, exposure to tobacco smoke, respiratory infections and obesity are all known risk factors for the development of asthma. Common trigger factors for asthma include allergens, respiratory infections, irritants, chemicals, physical activity and emotional stress.

EXERCISE-INDUCED BRONCHOCONSTRICTION

Exercise is a trigger of bronchoconstriction in patients with asthma.[10] The prevalence of exercise-induced bronchoconstriction in patients with asthma has been reported to range from 40-90 per cent.[11] Whilst the severity of exercise-induced bronchoconstriction in many asthmatics may relate to underlying hyperresponsiveness to pharmacological agents, some can have exercise-induced bronchoconstriction yet not have bronchial hyperresponsiveness to a pharmacological agent.[12] Thus in many patients with mild, episodic asthma and minimally increased airways responsiveness, even strenuous exercise does not cause significant bronchoconstriction. Exercise-induced bronchoconstriction results from the thermal and osmotic effects of conditioning large volumes of relatively cool, dry air in a short time during vigorous activity.[11,13] Exercise-induced bronchoconstriction most commonly occurs post exercise, following a period of initial bronchodilation during exercise. Bronchoconstriction begins within three minutes post exercise, generally peaking within 10–15 minutes, and resolves spontaneously over 3–60 minutes depending on the severity.

DIAGNOSIS

The clinical diagnosis of asthma is based on the symptomatic triad of cough, shortness of breath and wheezing occurring simultaneously. Spirometry and, in particular, the FEV_1 measure the degree of airflow obstruction. Administration of a bronchodilator is indicated if the baseline spirometry reveals obstruction. However, many patients do not present with the classical picture described above, and many asthmatics have normal spirometry despite the presence of asthma symptoms. Other patients give a past history of asthma but deny current symptoms. Bronchial provocation testing may be useful in these situations (Table 1).[14]

BRONCHIAL PROVOCATION TESTS

Bronchial provocation tests include the pharmacological agonists methacholine and histamine that act directly on receptors on bronchial smooth muscle causing it to contract and the airways to narrow. Whilst these tests have good sensitivity they are not specific for identifying asthma. Bronchial provocation tests that act indirectly to cause airway narrowing are more specific for identifying current airway inflammation and include exercise, eucapnic hyperventilation of dry air, and inhalation of hypertonic aerosols such as saline and mannitol. The bronchial hyperresponsiveness documented in response to indirect stimuli is associated with the presence of inflammation that is responsive to inhaled corticosteroids. Tests that use indirect stimuli have been used successfully to assess both the response to treatment and the withdrawal of treatment with inhaled steroids.[15] Approximately 50% of asthmatics lose their bronchial hyperresponsiveness to these stimuli following 3–6 months' treatment.[16] Twenty per cent of people with a past history of asthma, no current symptoms or current use of medication, with normal spirometry and who have been passed medically fit to dive have bronchial hyperresponsiveness to hyperosmolar saline.[17]

TREATMENT

Current asthma management plans encourage the use of peak expiratory flow (PEF) monitoring in patients with moderate to severe asthma. The utility of PEF to measure airflow limitation, however, is not particularly good, as variability among individuals is very large. The variability of PEF readings in healthy non-asthmatic individuals can be up to 31% in children and 19% for adults limiting the value of the measurements. However, PEF is useful in monitoring changes or trends in the patient's lung function.[18] Pharmacologic treatment of asthma includes the use of short- and long-acting inhaled beta-agonists, inhaled and oral corticosteroids, mast-cell stabilizing agents and leukotriene-modifying agents.

Intermittent use of short-acting inhaled bronchodilators is recommended for symptomatic relief for those with mild intermittent asthma and prophylactically prior to a known exposure in order to prevent the symptoms, e.g., exercise-induced asthma. Mast-cell stabilizing drugs taken immediately prior to exercise constitute effective

Table 1
Usefulness of bronchoprovocation testing

- Failure to show airway hyperresponsiveness strongly argues against diagnosis of asthma
- Airway hyperresponsiveness may be the sole evidence of airways dysfunction
- Airway hyperresponsiveness is quantitatively associated with the presence and severity of disease
- The occurrence of airway hyperresponsiveness in an asymptomatic person may help predict the future development of asthma
- The degree of airway hyperresponsiveness in a symptomatic person can have prognostic and potentially therapeutic implications
- The periodicity of asthma exists in parallel with changes in the degree of airway hyperresponsiveness.

preventive treatment and provide additive protection when used in combination with a beta-agonist.[19] Regular anti-inflammatory medications (e.g., inhaled corticosteroids) are recommended when symptoms become frequent. Inhaled corticosteroids reduce inflammation and by doing so reduce bronchial hyperresponsiveness to many triggers of asthma. Long-acting oral bronchodilators, e.g., theophylline, are effective when used in combination with anti-inflammatory therapy. However, they have a narrow therapeutic index, a relative lack of bronchodilator potency and their use is associated with frequent side effects. Drugs such as theophylline cause pulmonary vasodilatation and may reduce the bubble-filtering effect of the lungs and, therefore, increase the risk of venous bubbles becoming arterialised.

SUMMARY

In summary, in Australia, the United Kingdom and the United States, asthma has become increasingly prevalent in the age group of potential scuba-diving candidates. Mild asthma in childhood is likely to mean mild disease as an adult; remission in teenage years does not equate to lifelong remission and totally new asthma may present in adult years. This implies that there must be some individuals in Australia and New Zealand who were passed medically fit to dive in their teenage years who have later gone on to develop adult-onset asthma (and probably have not had another diving medical). Similarly it could be inferred that there are current divers whose asthma was in remission or not identified at the time of their diving medical.

The predictive value of bronchial hyperresponsiveness testing with methacholine in identifying those individuals with a previous history of asthma who are currently asymptomatic and who would likely relapse is low and therefore is not a useful screening test. Provocation testing by indirect challenge identifies those with bronchial hyperresponsiveness but who may have no current symptoms of asthma. However, bronchial hyperresponsiveness to a direct challenge is indicative of currently active asthma.

Asthma and scuba diving – why the concern?

There are many theoretical reasons why asthmatics should be at increased risk of an exacerbation of their disease when scuba diving. The exertion of diving may promote bronchospasm (exercise-induced asthma) and the inspiration of cold and dry air may cause release of inflammatory mediators in the airways triggering an attack.[20] The increased respiratory effort required as a consequence of regulator resistance and the increased density of gas at depth increases the work of breathing. Gotshall et al have demonstrated that compressed-air breathing via a scuba regulator at ambient pressure unimmersed increased the severity of exercise-induced bronchoconstriction in asthmatics but not in non-asthmatics.[20]

Diving could provoke an acute attack of asthma on the surface or, if at depth, increase the asthmatic's susceptibility to pulmonary barotrauma. Pulmonary barotrauma is the clinical manifestation of Boyle's law as it affects the lungs and is the result of over-distension and rupture of the lungs by expanding gases during ascent. The gas trapping and the increased airway resistance of asthmatics may predispose them to potentially fatal pulmonary barotrauma of ascent.

For these reasons, Australian and New Zealand diving medical practitioners have considered asthma to be a contra-indication to diving. However, the evidence to support these theoretical risks is limited (Table 2). Edmonds reports that asthmatics are over represented in diving fatality reports,[21] whilst Glanvill et al report no mortality and little morbidity

Table 2
Studies of asthma in diving and its prevalence in relation to fatal and non-fatal diving accidents

Author	Report	Outcome
Edmonds 1991[21]	Series of 100 diving deaths in Australia and New Zealand	9 deaths despite < 1% of divers reported having asthma
Neuman et al 1994[23]	5% prevalence of asthma in recreational divers in the USA	Fatal accident rate of 1 asthmatic in 2,132 deaths
DAN report 1996[24]	Retrospective review of DAN accident data 1988–1994	23/369 cases of arterial gas embolism and 123/2720 cases of decompression illness had coexistent asthma
Corson et al 1991[25]	Retrospective review of DAN accident data 1987–1990	16/196 divers with AGE had asthma, (AGE: current asthmatics odds ratio (OR) 1.98), 30/755 with DCS Type 2 had asthma (DCS 2: current asthmatics OR 1.16), neither statistically significant
Glanvill et al 2005[22]	Longitudinal cohort study of 100 UK divers with asthma	12,697 dives, 20 divers reported problems during diving, 12 reported wheeze underwater; one person reported two episodes of DCI (later confirmed to have a PFO). Current UKSDMC and BTS guidelines would have excluded all of the divers who reported either wheezing underwater or problems on the surface.

in a series of 12,697 dives.[22] Recent advances in asthma therapy may explain some of these differences; however, current diving death statistics in countries where medical examinations are not mandatory do not report a significant excess of asthma-related diving deaths.

Current position

SPUMS

SPUMS has consistently advocated the requirement for all diving candidates to undergo a diving medical examination by a medical practitioner with experience in diving medicine.[26] The SPUMS diving medical states that

"*A full (respiratory) history and examination should be normal. Any abnormal findings should be fully investigated. Such investigations should include provocation testing if any doubt concerning the possibility of bronchial hyperreactivity exists. Particular attention must be paid to any condition that might cause retention and trapping of expanding gas in any particular part of the lungs during decompression (e.g., asthma). The following conditions may disqualify:*

i any chronic lung disease past or present

ii any history of spontaneous pneumothorax, penetrating chest injuries or open chest surgery

iii any fibrotic lesion of the lung that may cause generalised or localised lack of compliance in lung tissue

iv any evidence of obstructive disease e.g., current asthma, chronic bronchitis, allergic bronchospasm.

All divers shall have a pulmonary function test to establish the FEV_1 and FVC. An FVC or FEV_1 of more than 20% below predicted values and/or a FEV_1/FVC ratio of less than 75% requires further assessment."[26]

AUSTRALIAN STANDARD FOR RECREATIONAL DIVERS

The Australian Standard AS 4005-2000, Training and Certification of Recreational Divers, reiterates the advice in the SPUMS medical but in addition states that

"*asthma...is an absolute contra-indication to breathing air under pressure. A normal FEV_1/FVC ratio but clinical signs of bronchospasm, especially on forced deep, rapid ventilation, is an indication of unfitness to dive. Treatment with drugs is not suitable as the effects can wear off underwater and the combined effects of pressure and bronchodilator drugs are uncertain*."[27]

THORACIC SOCIETY OF AUSTRALIA AND NEW ZEALAND

The Thoracic Society of Australia and New Zealand advised in 1993 that spirometric tests before and after bronchodilation should be performed on all intending divers. If there is an increase in FEV_1 of more than 15% post-bronchodilator bronchial provocation testing should be performed on a subsequent occasion. Intending divers with a history of current asthma should be advised not to dive. Intending divers with a past history of asthma and asthma symptoms within the previous five years should be advised not to dive. Those who have had asthma in the past, but who are asymptomatic and have normal spirometric tests and have taken no medication at all in the past five years should proceed to bronchial provocation testing.[28]

This advice has recently been reviewed.[15] Anderson et al note that since 1993 there has been a nationwide effort to improve asthma education, inhaled steroids are more widely available and more frequently used, lung-function tests are more commonly requested and the tests are more sophisticated. Self-monitoring of symptoms is more common and many asthmatics own peak flow meters. Anderson et al also state that

"*the finding of bronchial hyperresponsiveness or bronchial hyperreactivity in a significant proportion of healthy young adults, with a past history of asthma, seeking employment in occupations excluding current asthma, or seeking permission to use drugs before sporting events, supports the need for objective testing before clearance to dive.*"[15]

They recommend those bronchial provocation tests that involve the stimulus to which the intending diver is exposed, either exercise or eucapnic hyperpnoea of dry air and non-isotonic aerosols. In their experience, if either of these challenges produces symptoms, the intending diver is immediately aware of the potential for the same thing to occur whilst diving and may voluntarily withdraw from scuba training.

Anderson et al also state that bronchial hyperresponsiveness to exercise, eucapnic hyperpnoea of dry air and hypertonic aerosols has been demonstrated to be reduced over weeks by treatment with inhaled steroids. They make no recommendation as to whether treated asthmatics with no symptoms and negative bronchial provocation tests should be certified fit to dive.[15]

Anderson et al conclude that the 1993 approach that scuba should be disallowed for anyone with a history of symptoms and medication for asthma within the last five years should be re-evaluated in light of improved medication regimes, the ease with which lung-function and bronchial provocation tests can be performed and the move towards the informed risk assessment model. They strongly recommend the measurement of bronchial hyperresponsiveness in those individuals with a past history of asthma but no current symptoms and good lung function.

BRITISH THORACIC SOCIETY GUIDELINES

The British Thoracic Society established a working party to formulate national recommendations for assessment

of fitness to dive.[29] Their specific recommendations with respect to assessment of respiratory fitness include

> "*FEV_1, FVC and PEF should be measured. FEV_1 and FVC should normally be greater than 80% of predicted and the FEV_1/FVC ratio greater than 70%. Routine measurement of expiratory flow-volume loop, exercise testing, or bronchial provocation testing* [is] *not considered necessary although these tests may be useful in specific cases*."

Specific recommendations on asthma include

> "*subjects with asthma should be advised not to dive if they have wheeze precipitated by exercise, cold or emotion. Subjects with asthma may be permitted to dive if, with or without regular inhaled anti-inflammatory agents, they are free of asthma symptoms, have normal spirometry and have a negative exercise test. Subjects with asthma should monitor their asthma with regular twice daily peak flow measurement and should refrain from diving if they have active asthma (symptoms requiring relief medication in the 48 hours preceding the dive), a reduced peak expiratory flow (more than 10% fall from best value), or increased peak flow variability (more than 20% diurnal variation)*".[29]

The discussion accompanying the guidelines expands to state that there has been no prospective testing of the relationship between bronchial hyperresponsiveness and risk in divers and current evidence does not support the routine use of bronchial provocation testing in assessing fitness to dive. However, they do recommend an exercise test and advise that a step or free running test to raise the heart rate to 80% of maximum followed by measurement of FEV_1 at 1, 3, 5, 10, 15, 20, and 30 minutes after exercise is acceptable. A decrease in FEV_1 of 10% or more from the baseline is abnormal and a decrease of 15% or more is diagnostic of exercise-induced bronchoconstriction and would contradict diving.

UNITED KINGDOM SPORT DIVING MEDICAL COMMITTEE

The United Kingdom Sport Diving Medical Committee advises the British Sub-Aqua Club, the Sub Aqua Association and the Scottish Sub-Aqua Club on aspects of medical fitness to dive. Their guidelines state that

> "*asthma may predispose to air-trapping leading to pulmonary barotrauma and air embolism, which may be fatal. An acute asthma attack can also cause severe dyspnoea which may be hazardous or fatal during diving. These theoretical risks should be fully explained to the asthmatic diver. There is little if any evidence that the mild, controlled asthmatic who follows the guidelines below is at more risk: Asthmatics may dive if they have allergic asthma but not if they have cold, exercise or emotion induced asthma. All asthmatics should be managed in accordance with British Thoracic Society Guidelines. Only well controlled asthmatics may dive. Asthmatics should not dive if he/she has needed a therapeutic bronchodilator in the last 48 hours or has had any other chest symptoms*."[30]

Discussion

The dichotomy between the Australian/New Zealand and United Kingdom approaches to asthmatics diving deserves further discussion. Whilst both groups acknowledge the relevance of potential risks for asthmatics when diving, the United Kingdom approach places the decision in the intending diver's hands. Whilst the UK approach is to exclude asthmatics with exercise-induced asthma, evidence previously presented in this paper indicates that up to 90% of patients with symptomatic asthma have some degree of exercise-induced bronchoconstriction. However, if their symptoms are controlled (with or without anti-inflammatory medication) and they have normal spirometry and a negative exercise test, they are permitted to dive. However, unless the exercise test is conducted under standard conditions its reproducibility would be expected to be low. The UKSDMC advise that the medical examiner should perform an exercise test such as the 18-inch (43 cm) step test for three minutes, or running outside (duration not stated) to increase the heart rate to 80% of maximum (210 minus age in years beats per minute). A decrease in PEF of 15% at three minutes post exercise is evidence of exercise-induced bronchoconstriction and indicates disqualification. Exactly how the general practitioner would assess if the required heart rate was reached and maintained at 80% during this test is not explained. The British Thoracic Society guidelines on exercise testing are more rigorous yet may prove a logistic challenge for some general practices and therefore require specialist referral. It is then up to the patient to monitor their symptoms and lung function and for the medical practitioner to provide guidelines on when not to dive.

The Thoracic Society of Australia and New Zealand strongly recommends the use of bronchial provocation testing by hyperpnoea or hypertonic aerosols in the assessment of individuals with a past history of asthma but no current symptoms and good lung function. This testing will, however, identify those whose bronchial hyperresponsiveness is likely to be resolved by treatment with inhaled steroids. The newly available mannitol test (which is approved by the Australian Therapeutic Goods Administration) should be added to the list of existing bronchial provocation tests.

Advances in asthma treatment have occurred over the last 20 years and if medication reverses or abolishes bronchial hyperresponsiveness then theoretically the adequately treated asthmatic should be at no greater risk than the non-asthmatic. It is very unlikely that a prospective double-blind randomised trial will be conducted to prove this theory but there are sound theoretical reasons to support this statement (just like the theoretical reasons that argue against people with asthma diving).

Anderson reports that at least 50% of well-controlled asthmatics on steroids are negative to challenge by exercise, hypertonic saline and mannitol after 12 hours off all medication.[31] There may be some benefit in conducting challenge testing on prospective asthmatic divers after at least 12 hours off medication as this gives some confidence that the inflammation has resolved and the short-term effects of vasoconstriction will have dissipated.

It appears clear that an assessment of bronchial responsiveness is required for the prospective diver with treated asthma or with current symptoms but normal spirometry. It does not appear so clear that provocation testing should be mandatory in all of those with a past history of asthma, no symptoms and normal spirometry. It should, however, be kept in mind that 20–30% of those are likely to develop bronchial hyperresponsiveness to exercise or the inhalation of hyperosmolar aerosols that may be encountered during diving.[17,32] Many of these individuals will have bronchial hyperresponsiveness on indirect challenge testing but the relationship to morbidity or mortality when diving is not proven. Measurement of airway hyperresponsiveness would at least provide both the general practitioner and the patient a measurable marker on which to assess the perceived level of risk.

The UKSDMC position appears rational with the caveat that exercise or other indirect challenge testing (eucapnic hyperpnoea, inhaled hypertonic saline or mannitol) is an acceptable way to assess current asthmatic status and this testing should be done at least 12 hours after the last dose of asthma medication was taken. Whatever test is used it should be quantifiable and be reproducible under controlled conditions. The inhaled mannitol test (Aridol™, Pharmaxis Ltd., Frenchs Forest, NSW) may allow many more general practitioners to undertake bronchoprovocation testing in their surgeries and provide a greater degree of sensitivity and specificity than the exercise test as proposed by the UK authors.

The diving medical practitioner must explain all potential risks and give detailed written guidelines on how the individual should monitor their symptoms and when they should not dive. The individual diver must fully understand the instructions and accept responsibility for their actions.

Recommendations

1 Moderate to severe asthmatics should not dive due to the unpredictability of their disease, and the potential risk from pulmonary barotrauma and an exacerbation of their disease either underwater or on the surface.
2 There may be a subset of asthmatics who, either on anti-inflammatory medication or not, have a negative response to either exercise or indirect airway challenge, have normal lung function, are asymptomatic and who are fit to dive. These individuals should use PEF to monitor their lung function. The requirement of symptomatic relief with inhaled bronchodilators within 48 hours excludes diving.
3 Consideration should be given to the measurement of bronchial hyperresponsiveness in those patients with a past history of asthma and who have no symptoms and normal spirometry.
4 All potential divers must be informed of the potential risks of diving and the additional risk active asthma may pose. Written guidelines should be provided and the individual should accept responsibility for following these guidelines.

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SPUMS Award, HTNA ASM 2006

The SPUMS Award for the best presentation by a member of the Hyperbaric Technicians and Nurses Association Annual Scientific Meeting, held in Townsville in August 2006, was given to Carol Baines, Clinical Nurse Manager, Department of Hyperbaric and Diving Medicine, Royal Hobart Hospital. Her presentation, on behalf of Corry van den Broek and herself, was entitled
"Maggots! Can they handle the "pressure"?

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

<www.hboevidence.com>

Diabetes and diving: where to now for SPUMS?

Michael H Bennett

Key words
Diabetes, scuba diving, safety, fitness to dive, medical conditions and problems

Abstract

(Bennett MH. Diabetes and diving: where to now for SPUMS? *Diving and Hyperbaric Medicine*. 2006; 36: 220-5.)
The current guidelines of the Society explicitly preclude insulin-dependent diabetics from undertaking scuba diving. This stance is based on a perception that diabetics are at high risk of serious injury or death as a result of diabetic end-organ complications or unanticipated hypoglycaemia. It is also possible that some symptoms of hypoglycaemia may mimic decompression illness. In reality, however, several dive training organisations have developed programmes that allow some insulin-dependent diabetics to dive. These programmes seem to be highly successful and several formal reports suggest that, when using strict guidelines, there is a low incidence of problematic hypoglycaemia in these divers. This address to the SPUMS ASM in 2006 suggests that we should carefully consider these programmes and critically re-examine our position.

This talk on diving with diabetes largely summarises the recommendations of the DAN/UHMS Workshop held in June 2005, in which Simon Mitchell and I participated.[1] The executive summary and guidelines promulgated from that meeting were reprinted earlier this year in this journal.[2] We are discussing only recreational diving, so nothing in this address has any direct bearing on occupational diving. To begin with, I will discuss briefly the potential problems faced by diabetics who wish to dive.

There are four potentially problematic areas for divers with diabetes. First, the seriousness of hypoglycaemia underwater leading to a reduced level of consciousness and clouding of judgement is obvious to anyone here. Many insulin-dependent diabetics are unaware of impending important hypoglycaemia, and this has been one of the real sticking points for those of us who have been generally against this kind of activity for people with diabetes.

Second, thermal and exercise stress while diving can develop unpredictably, so anticipating needs and modifying insulin doses and/or sugar intake correctly can be difficult. Third, there is also a clear potential for symptomatic hypoglycaemia to be confused with decompression illness and vice versa.

Finally, hyperglycaemia may be a problem, although it seems unlikely that a diver would be getting to that state and still be diving. People with diabetes who run high blood sugar levels (BSLs), many of them non-insulin dependent, are prone to a wide range of complications and end-organ damage that might compromise their ability to dive safely. Dehydration from osmotic diuresis if running at high BSLs, may increase the risk of decompression sickness.

Therefore, there are problems if BSLs are either too low or too high, so if we are going to be positive about diving with diabetes, then this must involve pretty tight control of BSLs. The chronic complications and end-organ damage common in the diabetic population will also impact on their 'fitness to dive'.

The current SPUMS diving medical form states, "*Diabetes requiring medication with insulin is a contra-indication to diving.*"[3] On the SPUMS website there is a statement on diabetes, which contains much of a general nature to non-medical specialists as well as a clear direction to members of the Society regarding diabetes.[4] In parts, this states,

> "*Physicians who are sympathetic…often quote examples of world-class athletes who have diabetes…the diving environment is totally different from the athletic field or tennis court…On the athletic field, the blood glucose level can be easily maintained…consumption of (sugar) in the course of a dive is not as readily achieved.*
> *There are occasions when (diving) becomes exceedingly stressful and there is a need for unplanned, severe, sustained exercise. A diabetic whose blood sugar is controlled either with insulin or other oral agents would be incapable of maintaining such an exercise level and should be guided into less exacting pursuits.*
> *The insulin-dependent diabetic is prone to hypoglycaemia resulting in loss of consciousness and decompression illness and consequently should be advised against diving.*"

It probably will not surprise you to know that there are plenty of people with diabetes out there diving. There are organisations, some such as Camp DAVI (run by the Diabetic Association of the Virgin Islands) in existence for many years, that hold regular diving training for people with diabetes. Other examples on the Web are the Utila Community Clinic in Honduras (<http://www.aboututila.com/ScubaInfo/Diabetic-Scuba-Diver-Protocol.doc>), the YMCA (<http://www.ymcascuba.org/ymcascub/diabetic.

html>) and the British Sub-Aqua Club, where Dr Chris Edge has been an active promoter (<http://www.ukdiving.co.uk/information/medicine/diabetes.htm>).

As a Society we last reviewed the subject of diving with diabetes at the 2000 ASM.[5] It was pointed out that there are many people with diabetes who dive, apparently with a low risk of adverse events. Mitchell and Taylor in their paper advocated a review of the Society's absolute medical edict against diving with diabetes. The Diabetes Australia statement in 1994 on diving and diabetes was consistent with the SPUMS position. Recently, however, Diabetes Australia asked Simon Mitchell and me to provide them with a summary of the current thinking and data in the field. Their medical advisory panel is now considering the DAN/UHMS recommendations and guidelines, which (in a slightly modified form) is what we submitted rather than the SPUMS statement.

Let us review what is happening around the world. In the USA, there are several organisations actively promoting diving in insulin-dependent diabetes; the YMCA has a published protocol for divers with diabetes and there is an SSI programme available. Camp DAVI has been operating since the late 1980s with about 700 dives reported, and has developed some very detailed protocols (<http://www.diabetesnet.com/visle.php>). Participants must have an HbA_{1c} running at less than 9% and no symptomatic hypoglycaemic events requiring treatment or requiring third-party intervention for one year.

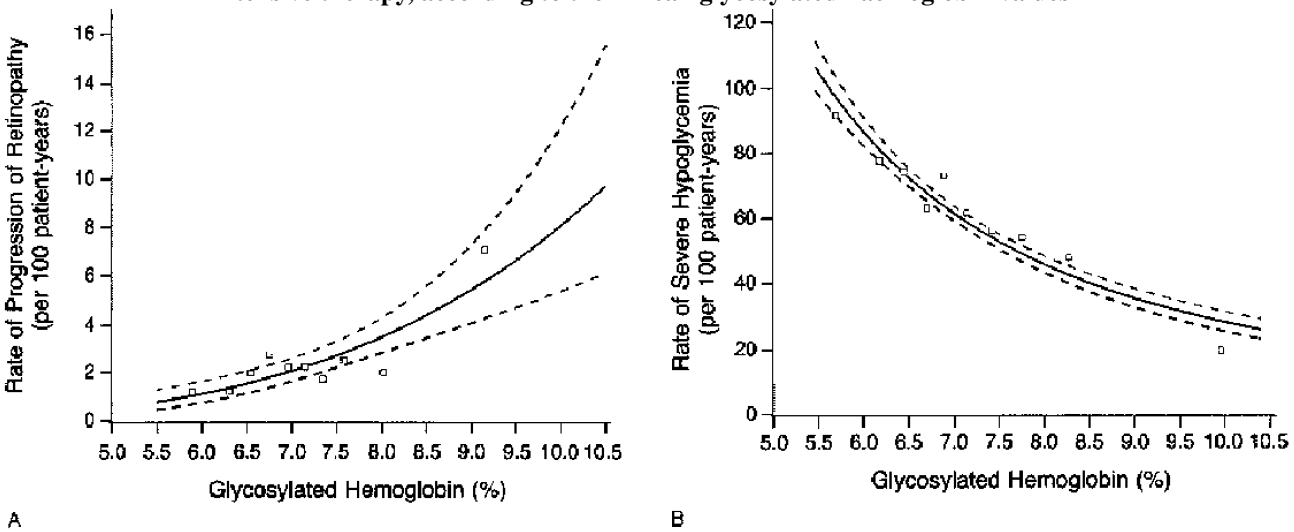
The goal BSL they are looking for is 8 to 10 $mmol.l^{-1}$ immediately prior to diving. BSLs are measured at 60 and 30 minutes and immediately pre-dive, and the BSL must have been rising or stable across that time. All diving is restricted to no-decompression diving, and participants must carry a glucose source. Their ideas were based on those of medical advisors in the diabetic field, which in turn were based largely on data from the Diabetes Control and Complications Trial Research Group in 1993.[6] In essence, the tighter you run your control, the lower your HbA_{1c}, the greater chance you have of a symptomatic hypoglycaemic event. On the other hand, retinopathy and most of the other complications of diabetes are much less common when you have tight glycaemic control. So, this is a balance between two differing needs, one short-term and one long-term.

DAN published guidelines in 2005 that are very similar to those of the DAN/UHMS Workshop (Table 1[1]) and Camp DAVI. From 1997 DAN has been running an observational study of 83 divers: 43 well-controlled insulin-dependent diabetic divers having a total of 555 dives and a control group of 40 divers having 504 dives.[7] No symptomatic hypoglycaemic events were recorded, but 7% of the divers with diabetes had a blood sugar of less than 4 $mmol.l^{-1}$ at some stage. Interestingly 1% of the non-diabetic divers also recorded BSLs of less than 4 $mmol.l^{-1}$.

Both the French and British have published guidelines as have a number of other countries. A British database going back to 1991 documented 447 divers, with a median HbA_{1c} of 7.6%, who recorded 14,000 dives.[8] There were two deaths reported, both in non-insulin-dependent diabetics. One was a middle-aged man who suffered a myocardial infarction. The other death in a fit, young person remains unexplained, and so is worrying. There was only one symptomatic hypoglycaemic episode during a dive, which was treated underwater with ingestion of glucose paste.

It is fair to point out that SPUMS has taken no action since 2000. Now the word is out on the street, there are literally dozens of websites where diabetics are saying "Now we

Figure 1
Risk of sustained progression of retinopathy (A) and rate of severe hypoglycaemia (B) in the patients receiving intensive therapy, according to their mean glycosylated haemoglobin values[6]



can go diving". One increasingly important issue will become the application of the anti-discrimination and equal-opportunities legislation to our refusing to entertain people with diabetes diving. Clearly the current SPUMS position on diabetes and diving has become very different to that of much of the rest of the world. The question arises as to what the Society should do?

The sensible course would be to have an appropriate group of interested people, knowledgeable in the area, review all

Table 1
Guidelines for recreational diving with diabetes - summary form*

Selection and surveillance
- Age >=18 years (>=16 years if in special training program)
- Delay diving after start/change in medication
  - 3 months with oral hypoglycaemic agents (OHA)
  - 1 year after initiation of insulin therapy
- No episodes of hypoglycaemia or hyperglycaemia requiring intervention from a third party for at least one year
- No history of hypoglycaemia unawareness
- HbA_{1c} <= 9% no more than one month prior to initial assessment and at each annual review
  - values > 9% indicate the need for further evaluation and possible modification of therapy
- No significant secondary complications from diabetes
- Physician/Diabetologist should carry out annual review and determine that diver has good understanding of disease and effect of exercise
  - in consultation with an expert in diving medicine, as required
- Evaluation for silent ischaemia for candidates > 40 years of age
  - after initial evaluation, periodic surveillance for silent ischaemia can be in accordance with accepted local/national guidelines for the evaluation of diabetics
- Candidate documents intent to follow protocol for divers with diabetes and to cease diving and seek medical review for any adverse events during diving possibly related to diabetes

Scope of diving
- Diving should be planned to avoid
  - depths > 100 fsw (30 msw)
  - durations > 60 min
  - compulsory decompression stops
  - overhead environments (e.g., cave, wreck penetration)
  - situations that may exacerbate hypoglycaemia (e.g., prolonged cold and arduous dives)
- Dive buddy/leader informed of diver's condition and steps to follow in case of problem
- Dive buddy should not have diabetes

Glucose management on the day of diving
- General self-assessment of fitness to dive
- Blood glucose (BG) >=150 $mg.dl^{-1}$ (8.3 $mmol.l^{-1}$), stable or rising, before entering the water
  - complete a minimum of three pre-dive BG tests to evaluate trends
    - 60 min, 30 min and immediately prior to diving
  - alterations in dosage of OHA or insulin on evening prior or day of diving may help
- Delay dive if BG
  - < 150 $mg.dl^{-1}$ (8.3 $mmol.l^{-1}$)
  - > 300 $mg.dl^{-1}$ (16.7 $mmol.l^{-1}$)
- Rescue medications
  - carry readily accessible oral glucose during all dives
  - have parenteral glucagon available at the surface
- If hypoglycaemia noticed underwater, the diver should surface (with buddy), establish positive buoyancy, ingest glucose and leave the water
- Check blood sugar frequently for 12-15 hours after diving
- Ensure adequate hydration on days of diving
- Log all dives (include BG test results and all information pertinent to diabetes management)

* For full text see: Pollock NW, Uguccioni DM, Dear GdeL, editors. *Diabetes and recreational diving: guidelines for the future*. Proceedings of the UHMS/DAN 2005 June 19 Workshop. Durham, NC: DAN; 2005.

the implications of adopting some or all of the guidelines and recommendations of the DAN/UHMS Workshop. Such a group would consist of diving physicians and diabetologists. I was convinced by the legal experts and the patient pressure groups at the DAN/UHMS Workshop that it would be very sensible to involve them in this process too. This fits best into a risk-assessment framework, which would move the Society, in Des Gorman's words, "from policeman to health adviser."

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Audience participation

Haller, Victoria: I think it is a great idea putting some of the onus back onto the diver, rather than on the medical practitioner and the diving instructing people. If a diabetic diver from, say, Europe comes to Australia, who then takes the onus if they run into any diving problems and in looking after their diabetes?

Bennett, Sydney: I don't have the answer to that, I don't know if someone else in the room does?

Standon, Australia: As a dive-shop operator, as long as they have a certification card, I assume that they have been trained appropriately and they have the appropriate awareness and I take them diving.

Bennett, Sydney: What would you do if they came in and said here's my insulin, here's my dive card?

Standon, Australia: I would then have issues with whether or not my staff are trained to look after these people and, taking that on board, telling them that I either do or do not have the staff trained appropriately. Most of them have a basic awareness, but certainly trying to give insulin injections or anything like that…All they could do is shove a few jellybeans down their throat. Apart from insurance and duty-of-care considerations, it's not really a problem. Once again, they have been trained, they have their certification card, they should be aware of their condition and its ramifications and we will do whatever we can to help them.

Bennett, Sydney: It should be said, though, that someone doing that would clearly be diving outside those guidelines, because they would be concealing it from the dive leader, the dive group organiser.

Henderson, USA: One thing that came to mind for me as far as the discrimination issue is concerned, is what does Australia, New Zealand and the States do for pilots who are diabetic? It would seem to me that if these countries have imposed limitations for pilots who are diabetic one could adopt those guidelines, or parts of those guidelines, and hopefully avoid some of the discrimination issues that might arise.

Bennett, Sydney: You are absolutely right and there was a representative from the aviation authorities, who was a specialist in that area, at the Workshop. A lot of the recommendations were based on criteria for holding a private pilot's licence. I am not sure about professional pilots.

Meehan, Cairns: In Queensland, all certified divers have to fill out a declaration form, declaring if they have any medical illness or condition, or if they are on medications. So anyone who is an insulin-dependent diabetic is identified and the situation there is that we are regularly called for advice. It is

very much you follow the guidelines; the dive instructor or the dive team leader does need to know they're diabetic and does need to have training in how to look after those divers, and the diver has to have their own buddy who knows how to look after them. Are there any PADI schools in Australia who are going to teach dive instructors and divers to dive effectively with diabetes?

Richardson, PADI Worldwide: I don't believe so. Mike, you have laid down the gauntlet here as you normally do, and I see an opportunity here for the Society. This is a closeted issue, just like asthma. I understand that there is an increasing problem with both asthma and latent diabetes in the developed world, obesity and lots of allergens and so on. So there is a likelihood of more people doing their own web search, and the diving instructional community really does not have first-hand expertise. There's certainly a call for educational seminars. I am positive that our operators would love to attend if they were led by informed physicians; Dr Chris Edge has demonstrated that in the UK.

I was just thinking that a useful tool for the individual diver would be some sort of work slate. We developed one for dive accident management some years ago. If SPUMS wanted to it could develop an algorithm, the world might applaud that. I think this is food for thought. Alternatively you could simply say do not dive, which would save a lot of trouble! However, there is a growing clientele of sport divers and diving instructors who might be very interested in algorithm flow charts for both asthma and for diabetes. Right now people are doing that on their own. You might make a very relevant impact on the recreational diving community.

Fulton, New Zealand: As a GP, if I had a patient whose HbA_{1c} was consistently up around 9%, I would think that that patient was very poorly controlled. Should it not be tightened up more than that?

Bennett, Sydney: There were certainly plenty of people at the Workshop who would agree with you. The people who actually work most with divers with diabetes suggested that making it much tighter than that would unreasonably limit the number of people who could get into the water. They have had that group diving quite successfully and did not see why they should be excluded. Everyone agreed that poorly controlled diabetics shouldn't be in the water, so we came out with this compromise number of [HbA_{1c}] 9%, but it is absolutely open to debate and interpretation. I actually started off by suggesting less than 6%, which was shouted down with howls of derision as being far too tight, too restrictive.

Long, NSW: I'd like to follow up what Fulton said. Recommendations for good diabetic control are now less than 7% and the reason for those recommendations is to avoid end-organ disease. So there is an implication in what you are saying that so these guys can dive it does not matter if they get retinopathy at the end of it all. That is the implication of bringing the number up, when there has been a big drive to get everybody below 7%.

Christie, Victoria: Obviously people who have diabetes and who meet those guidelines are going to need some kind of extra education and training about their diabetes before they actually dive. I wonder is there any specification in the guidelines, about whether it would be the diving instructor, the diving doctor doing the dive medical or in fact a combination of both who would have that responsibility?

Bennett, Sydney: No, we did not deal with that. At the moment, with these specialised groups, this function is taken on by diving instructors, usually with diabetes, who have developed the strong interest in it and have involved diving doctors and diabetologists to advise them on the details. It is difficult to see how that could work over a whole system where diabetics were going to have free access to every dive school in the country. The suggestion by Simon Mitchell and Lynn Taylor was that you would need to run special courses. However, the industry pointed out how difficult that would be to get off the ground. So I do not think anyone knows the answer to how you are going to integrate into dive training in Australia at least.

Waterson, Narimbula: Just a comment on the HbA_{1c}; the 9% figure seemed to come out of that Virgin Islands project which, from the website you showed, included teenagers. In general, teenagers run with higher HbA_{1c} measurements because they are usually less reliable at that stage of their lives and I guess they are less concerned about retinopathy and nephropathy when they are 16 years old. In my experience, endocrinologists are happy with their teenagers running higher because of hypos being such an issue in the growth period. I wonder if that is where that leniency came from?

Bennett, Sydney: You are probably right. I take your point, but it is a guideline for who should be allowed to dive, not a recommendation for diabetic management. However, if there is a potential for it to be interpreted that way then maybe we should think carefully about how we put it, even if we let that number in.

Acott, Adelaide (President SPUMS): In your recommendations you suggested gathering a few people together to put recommendations to the SPUMS Committee. As President of the Society, I want to know when are you going to start?

Bennett, Sydney: Well, once we've discussed the budget, I'll get underway.

Acott, Adelaide: What budget?

Smart, Hobart: There is a huge logistical issue in the actual medical process for this, and it is likely to be expensive for the diver. That aspect is going to have to be taken

into account. It is certainly going to be a problem for any international divers who are diabetic coming to Australia and their primary physician is back in another country and they want the quick dive medical for their Great Barrier Reef holiday they have paid $20,000 for.

I like Drew's idea with the slates, but we also need information in terms of a calculation of risk. I believe that in this circumstance family members would need to come in as the risk sharers and understand what is going on. Finally, information for dive buddies is essential. It is okay to release guidelines, but the practical implications of the operation are going to be huge.

Meehan, Cairns: In Cairns there are a couple of dive instructors who are trained through the International Association of Handicapped Divers from Europe, and my understanding is that in that Society they do train instructors to train divers who have diabetes, plus a whole lot of other issues. Does PADI provide courses for certain instructors to train people with certain medical conditions or handicaps?

Richardson, PADI Worldwide: We cooperate with the International Association for Handicapped Divers. What I am suggesting is, it depends whether SPUMS wishes to evolve guidelines and integrate these into the diving community or not. If you wanted to create a model like that, I can assure you that PADI would cooperate fully in getting the word out to divers and diving professionals and try to bring them to educational sessions. The problem now is that there are all these opinions and guidelines floating around the Internet and elsewhere that are not really harmonised and people are taking matters into their own hands. If you feel that you want to evolve guidelines, with Australia as a model, I am sure Henrik [Nimb] and his training department at PADI could do a lot to help you along those lines locally.

Sharp, Perth: In the UK system diabetic divers have both the UK medical form and a diabetic form they must fill out, and which the GP has to fill out, as well as this other diabetic form which the GP and the endocrinologist also complete. That all goes to Dr Chris Edge or Dr Phil Bryson for review. Many of the UK sport-diving medical referees thought that it was all just too much hard work. In England, the Sports Diving Medical Committee medical questionnaire is done every year, so the auditing workload is considerable. After 14 years, Dr Edge said he had had enough, let the diabetics dive, here are our guidelines.

Davis, New Zealand: Thank you everyone who has contributed. I think we do need to set up a group, and the issue of funding for that is a genuine one. Mike's comment about that was not facetious at all. Perhaps through the Diabetes Society is the way to seek at least partial funding for setting up a joint committee to investigate these issues. That would be my feeling. I think Drew's idea of a slate is a very sensible one. Slates like that do work from a practical point of view and that gives us a new path for SPUMS to explore in assessing the data. I don't think there is much doubt that, as a diving medical society, we do have to change our stance. It will be interesting to see how this develops.

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'South Pacific Underwater Medicine Society'.
Credit card facilities are not available for this purchase.

Contact: Steve Goble, Administrative Officer
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Articles reprinted from other sources

Expired carbon monoxide (CO) as a marker of CO poisoning and its application in determining treatment end-points [Abstract]

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**Abstract of the thesis submitted for the degree of Doctor of Medicine of the University of Tasmania
Satisfied requirements on 27 August 2005**

Abstract

(Smart DR. Expired carbon monoxide (CO) as a marker of CO poisoning and its application in determining treatment end-points [Abstract]. *Diving and Hyperbaric Medicine*. 2006; 36; 226-7.)
Carbon monoxide (CO) is a colourless, odourless toxic gas that is able to substitute for oxygen at many levels in the oxygen cascade. CO poisoning is responsible for nearly a quarter of suicide deaths in Australia, and hundreds of individuals sustain non-fatal poisoning every year. Up to two thirds of individuals who survive CO poisoning have long-term neurological or cognitive impairment. Despite years of study by medical researchers, a reliable marker of acute CO poisoning severity that correlates with outcome has not been identified. Oxygen is known to be an antidote to CO poisoning, yet there is significant debate regarding the dose required, and the treatment duration. The end-point of CO excretion from the body is the lungs. Measurement of expired CO has been documented since the 1980s; however, there has been limited study of ECO in poisoned patients.

In this research ECO was investigated as a marker of CO poisoning, and its application in determining treatment end-point. A low-cost, portable and non-invasive apparatus was successfully developed for measurement of ECO, oxygen concentration and minute volume. The apparatus was then evaluated in a variety of settings, for adults and children, and to establish baseline ranges for non-smokers, smokers and poisoned individuals, breathing air, NBO and HBO. The technique of measuring ECO was further investigated to determine the relationship between ECO and COHb, and for the diagnosis of CO poisoning. The apparatus was evaluated in the clinical setting to determine pulmonary CO elimination kinetics. A prospective series of CO-poisoned patients was enrolled to determine if acute ECO levels correlated with clinical outcomes and to assess whether unrecordable ECO was a suitable marker of treatment end-point. In this research, expired oxygen concentration was also monitored, to ensure that all individuals received the stated dose of oxygen.

Baseline levels of ECO were found to be very low in healthy, non-smoking volunteers, and in non-smoking divers treated for decompression illness, consistent with the observation that most CO derives from exogenous sources. Smokers had higher baseline ECO than non-smokers, and smoker ECO levels correlated positively with the number of cigarettes smoked per day, and negatively with the time since last cigarette.

Breathing air and NBO, a strong positive linear relationship between the ECO and COHb was observed for non-poisoned smokers, poisoned individuals and pooled data. Expired CO concentration increased in proportion with increasing FiO_2 for 0.21 (air) to 1.0 (NBO). While breathing 100% oxygen, increasing ambient pressure from 1 ATA to 2.8 ATA did not alter the ECO concentration (ppm) in each breath. However, elimination of CO was greatly enhanced due to the increased density of gas at higher pressures. Each tidal volume at 2.8 ATA actually contains 2.8 times as many molecules of CO compared with the same tidal volume at 1 ATA ambient pressure. When poisoned subjects breathed NBO and HBO, significant amounts of ECO were detectable when the COHb was unrecordable using the biochemical method. This suggested that ECO more accurately reflected remaining CO in body stores than COHb; however, this might have resulted from the limits of the biochemical method for detecting low levels of COHb (< 2%). Concurrent measurement of expired oxygen provided useful confirmation that the intended 100% oxygen dose was delivered to all treated individuals.

ECO was a useful non-invasive test to diagnose acute (< 6 hours) CO poisoning, when ECO values were > 40 ppm. For ECO values of 7 ppm to 40 ppm, clinical information would be needed to separate mildly poisoned individuals from smokers. Expired CO and COHb were equally effective in identifying acutely poisoned individuals, from smokers and non-smokers. Critical values of ECO > 40 ppm or COHb > 7% were shown to be highly specific for CO poisoning.

Expired CO demonstrated single-stage exponential elimination kinetics in both NBO and HBO treatment environments. CO elimination in HBO was significantly faster than NBO. There was a seven- to ten-fold variation in CO elimination between individuals in either treatment (NBO or HBO). Based on these findings, current empirical regimens may over-treat some individuals and under-treat others. The half-lives determined for ECO elimination were longer than those determined for COHb. This suggests that elimination of CO via the breath may be slower than elimination from Hb. If unrecordable ECO proved useful as a treatment end-point, this would allow treatment to be tailored to the individual's acute CO load.

In the clinical series of acutely poisoned patients, there were a high number of males sustaining CO poisoning from deliberate self-harm. These individuals had longer exposures, greater neurological toxicity, and were more likely to have LOC than accidental exposures. The greater toxic effect and higher CO body load was most likely due to breathing leaded petrol exhaust containing high CO levels to attempt suicide. In keeping with their greater neurological toxicity, there was a positive correlation between ECO, COHb levels, and the severity of poisoning. The ECO measurement breathing oxygen correlated significantly with the severity of neurological impairment in the ED. This provided support for ECO levels as a useful guide to acute clinical poisoning severity. However, acute ECO and COHb levels measured in the ED were not predictive of outcome at three months. This may have been affected by significant delays in transferring patients for HBO treatment.

Just over 28% of patients had poor outcomes at three months, using unrecordable ECO as a treatment end-point. At this point, patients who had abnormal neurological or cognitive function remained abnormal at three months. Unfortunately the treatment end-point using ECO did not prevent cases of DNS, or the need to provide follow up for CO-poisoned patients. The occurrence of DNS after all CO had been removed suggests that DNS may result from mechanisms other than direct CO toxicity.

Poor outcomes were associated with delays to study entry, suicide attempts, motor vehicle exhaust as a source of CO and acidosis measured in the ED. Individuals with LOC did not have a significantly worse outcome than those remaining conscious during their CO exposure. HBO- and NBO-treated patients had similar levels of PNS, however the HBO group had a lower incidence of DNS – an unexpected finding. Because the study was not randomised, it was not possible to conclude this is a definite treatment effect. Compared with NBO, HBO treatment led to faster removal of CO, and shorter treatments.

Measurement of ECO constitutes a novel non-invasive method of monitoring of acute CO poisoning. It has potential to complement existing methods of monitoring acute CO poisoning, and may be useful as a non-invasive test to diagnose CO poisoning. Clinical outcomes in this series compared favourably with other series of similar severity poisoning in the literature. However, further research using a randomised controlled trial is required to determine if unrecordable ECO is a useful guide to treatment end-point.

Key words
Carbon monoxide, clinical toxicology, toxicity, hyperbaric oxygen, morbidity, reprinted from

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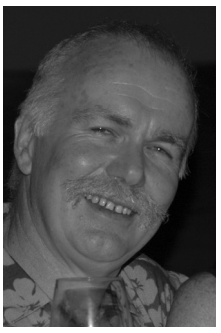
Faces from the 2006 ASM, Fiji

Steve Goble,
SPUMS Administrator

Dr David Griffiths,
Townsville

Dr Denise Blake,
Townsville



Guy Williams,
SPUMS Treasurer

Australian Resuscitation Council Guideline 7: Cardiopulmonary resuscitation

This guideline is applicable to adults, children and infants.

Cardiopulmonary resuscitation (CPR)

Cardiopulmonary resuscitation is the technique of rescue breathing combined with chest compressions. The purpose of cardiopulmonary resuscitation is to temporarily maintain a circulation sufficient to preserve brain function until specialised treatment is available.

Rescuers should start CPR if the victim has no signs of life (unconscious, unresponsive, not moving, and not breathing normally). Even if the victim takes occasional gasps, rescuers should suspect that cardiac arrest has occurred and should start CPR.[1] [Class A; LOE IV]

COMPRESSION VENTILATION RATIO

No human evidence has identified an optimal compression-ventilation ratio for CPR in victims of any age.[1,2] Interruptions to compressions should he avoided with evidence suggesting that previous compression-ventilation ratios resulted in too much "hands off time" [LOE IV]. Evidence also demonstrates that over ventilation occurs even by trained responders.[1]

A universal compression-ventilation ratio of 30:2 (30 compressions followed by 2 ventilations) is recommended for all ages regardless of the numbers of rescuers present.[1,2] Compressions must be paused to allow for ventilations.

This compression ventilation ratio has been selected to:
- Increase the number of compressions;
- Minimise interruptions to compressions;
- Prevent excessive ventilation;
- Simplify teaching;
- Maximise skill retention;
- Maintain international consistency.

[Class A; LOE IV]

STEPS OF RESUSCITATION

Initial steps of resuscitation are:
DRABCD
- Check for **danger** (hazards/risks/safety);
- Check for **response** (unresponsive/unconscious);
- Opening the **airway** (look for signs of life — call 000/ Resuscitation team);
- Give rescue **breathing** (give two rescue breaths if not breathing normally);
- Give 30 chest **compressions** (almost 2 compressions/ second) followed by 2 breaths;
- Attach an AED (automated external **defibrillator**) if available and follow the prompts.

When providing 30 compressions (at approximately 100/ min) and giving 2 breaths (each given over 1 second per inspiration), this should result in the delivery of 5 cycles in approximately 2 minutes.
[Class A; LOE Expert Consensus Opinion]

DEFIBRILLATION

The Australian Resuscitation Council recommends the use of an AED if available (refer to Guideline 10.1.3).

CHEST COMPRESSION ONLY

If rescuers are unwilling or unable to do rescue breathing they should do chest compressions only. If chest compressions only are given, they should be continuous at a rate of approximately 100/min.[1] [Class A; LOE 111-2]

MULTIPLE RESCUERS

When more than one rescuer is available ensure:
- That an ambulance has been called (000);
- All available equipment has been obtained (e.g., defibrillator);
- Frequent rotation of rescuers is undertaken (approximately every 2 minutes) to reduce fatigue. [Class A; LOE Expert Consensus Opinion]

DURATION OF CPR

The rescuer should continue cardiopulmonary resuscitation until:
- Signs of life return;
- Qualified help arrives;
- It is impossible to continue (e.g., exhaustion);
- An authorised person pronounces life extinct.

[Class A; Expert Consensus Opinion]

RECOVERY CHECKS

Evidence has demonstrated that interruption of chest compressions is associated with poorer return of spontaneous circulation and lower survival rates and that both lay and health care professionals experience difficulty in determining presence or absence of pulse in collapsed victims. Therefore, rescuers should minimise interruptions of chest compressions and CPR should not be interrupted to check for signs of life.[1] [Class A; LOE IV]

RESUSCITATION IN LATE PREGNANCY

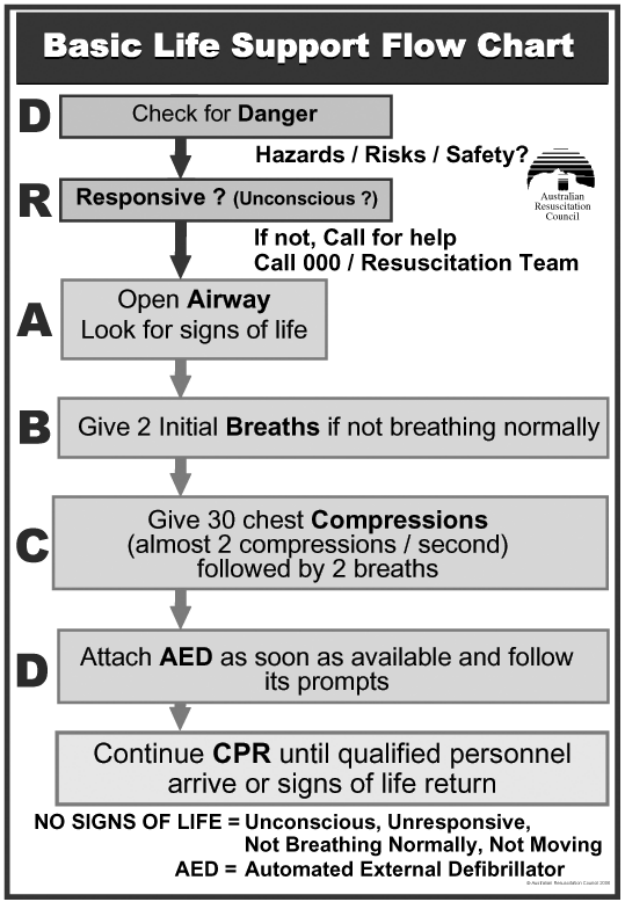
In the obviously pregnant woman the pregnant uterus causes pressure on the major abdominal vessels when she lies flat,

reducing venous return to the heart. The pregnant woman should be positioned on her back with her shoulders flat and sufficient padding under the right buttock to give an obvious pelvic tilt to the left.[3] [LOE: Expert Consensus Opinion] [Class A; LOE Expert Consensus Opinion]

Additional notes:

Distension of the stomach may occur when the rescuer either blows too hard or blows when the airway is partially obstructed so that air enters the stomach rather than the lungs. If the stomach is distended, DO NOT APPLY PRESSURE TO THE STOMACH. If air is forced into the stomach, some stomach contents can be forced up into the mouth and airway and thus into the lungs.

Regurgitation is the passive flow of stomach contents into the mouth and nose. Although this can occur in any person, regurgitation and inhalation of stomach contents is a major threat to an unconscious person. It is often unrecognised because it is silent and there is no obvious muscle activity. Vomiting is an active process during which muscular action causes the stomach to eject its contents. In resuscitation, regurgitation and vomiting are managed in the same way by prompt positioning of the victim on the side and manual clearance of the airway prior to continuing rescue breathing.



Currency and assessment of CPR skills

CPR skills performance has been shown to decline rapidly following initial achievement of competency.[4] The Australian Resuscitation Council recommends that CPR skills are reassessed at least annually. [Class A; LOE Expert Consensus Opinion]

The Australian Resuscitation Council recognises that training organisations are required to assess CPR competency. ARC recommends that assessors be cognisant to the intent of the resuscitation community that any attempt at resuscitation is better than no attempt. As such, assessment should focus on adequate CPR and not on the technicalities of achieving set figures or rates. [Class A; LOE Expert Consensus Opinion] (refer to Guideline 9.1.1)

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This guideline is reprinted with kind permission from The Australian Resuscitation Council Online Guidelines, Section 7 – Cardiopulmonary resuscitation, February 2006. Available online at <http://www.resus.org.au> (last accessed 18 December 2006).

Comments on the revised ARC Guidelines

In March 2006 the Australian Resuscitation Council (ARC) released updated guidelines for Basic and Advanced Life Support.[1] The changes were based on extensive evaluation of the current resuscitation evidence by the International Liaison Committee on Resuscitation (ILCOR).[2] Evidence has shown that:

- the single most important factor in survival from a sudden cardiac arrest may be early defibrillation
- any attempt at resuscitation is better than none
- all guidelines be simplified to eliminate time-wasting procedures – for example the carotid pulse is no longer palpated because the 'no signs of life' equals being unconscious, unresponsive, not breathing normally and not moving.

Therefore defibrillation via an automatic external defibrillator (AED) has been added to these new basic life support

(BLS) guidelines. This may have important ramifications for those providing first-aid assistance to injured divers. In the future, all dive boats may be required to carry an AED and have attendants trained in its use. AEDs are simple to use; once the pads have been correctly placed the rescuer is prompted by the machine. AEDs have been used safely by lifeguards/savers on the beach, at swimming pools or even in inflatable boats; they are, therefore, safe in the aquatic environment.[3,4]

Other important changes to the BLS guidelines are:

- the compression–ventilation ratio is now 30:2 irrespective of the number of rescuers or if the victim is an adult, child or infant
- the compression rate is 100 compressions per minute
- the term 'rescue breathing' (RB) has replaced the term 'expired air resuscitation' (EAR)
- RB is no longer a stand-alone procedure – all victims receive cardiopulmonary resuscitation (RB and chest compressions) regardless
- the initial five (5) breaths have been replaced by two (2), although this varies from country to country.

An AED is simple to use – so update your skills now!

Note that there may be minor differences between these Australian guidelines and those elsewhere and one should check with local national resuscitation guidelines.

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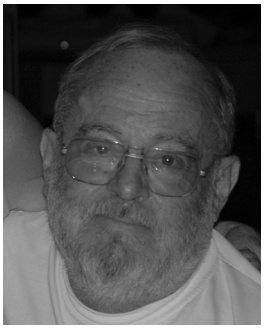
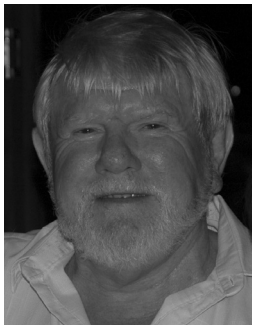
Chris Acott
President, SPUMS

Santa at Fiji ASM

Unsubstantiated rumours suggest that Dr & Mrs Santa Claus attended the 2006 SPUMS ASM incognito. We hope members may recognise them amongst the faces below. Happy Christmas and a healthy, safe New Year to all!








Other registrants thought it was all a scam!



Letters to the Editor

Mannitol bronchial challenge testing and scuba diving

Dear Editor,

I was interested at your decision to publish Dr Anderson's letter about the mannitol bronchial provocation test.[1] I remain puzzled as to its relevance to scuba diving. As I have observed previously, bronchial smooth muscle evolved in order to contract and narrow the airways and can be made to do so in anyone if sufficient stimulus is applied. The level at which this bronchial responsiveness is labelled hyperresponsiveness and thus identified as a disease state seems to be arbitrary.

The mannitol test has been used along with eucapnic voluntary hyperventilation to try to document abnormalities in elite athletes who wish to use bronchodilators to improve their performance. This has been accepted by major sporting bodies to try to limit the very high use of bronchodilators by athletes. However, the problem is that mannitol and other tests of exercise-induced bronchospasm correlate very poorly with either reported symptoms or diminished performance in elite athletes such that some athletes with positive tests have no symptoms and others with symptoms and diminished performance have no bronchospasm on testing.[2] It is not clear, therefore, which group actually has a significant clinical problem.

So far as scuba diving goes, there is no evidence that either asthma or bronchospasm induced by testing with either pharmacological or non-pharmacological agents has any adverse outcomes in relation to barotrauma, decompression illness or mortality. Dr Anderson refers to individuals who are relieved at having an excuse to avoid scuba diving in the form of a positive bronchial provocation test.[3] I think that most doctors doing diving medicals would find this an unusual situation, and the vast majority of prospective divers who fail their medical are actually deeply disappointed. As the positive predictive value of bronchial provocation testing for adverse events in scuba diving must be so low as to approach zero, it would seem that introduction of a new test at this time is not sensible.

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Graham Simpson
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Conflict of interest

I am currently an investigator in a multicentre trial looking at the use of mannitol manufactured by Pharmaxis in the treatment of bronchiectasis. I am not personally in receipt of any financial benefit from Pharmaxis in relation to this study nor have I any financial interest in Pharmaxis or other pharmaceutical companies.

Key words
Bronchial provocation testing, asthma, fitness to dive, letters (to the Editor)

Editor's note: This is the last submission that the journal will take on the specific issue of mannitol for bronchial provocation testing. However, letters regarding the SPUMS policy on asthma and diving, and related to Dr Walker's paper in this issue, are welcome.

Decompression sickness following breath-hold diving

Dear Editor,

Gemp and Blatteau point out in a recent case report that decompression sickness (DCS) is a possibility in breath-hold (BH) divers and advised that anyone who experiences unusual symptoms after BH diving should seek medical attention.[1] They describe a fit, young sailor in the French Navy who performed repetitive dives to 10–18 metres' sea water over 60–90 minutes, made 10–12 unassisted dives, each dive lasting 1–2 minutes with surface intervals of 5–6 minutes. Ascent times were 15–20 seconds.

The dive profiles should theoretically preclude such a person from developing DCS. However, due to forceful Valsalva manoeuvres, he suffered dizziness, visual disturbance, tightness in the chest with dyspnoea, flushed face and numbness of all limbs and the right side of the face. These symptoms appeared two hours after surfacing and lasted about one hour. He was discovered to have a patent foramen ovale (PFO) on subsequent investigation.

Again, as in previous reports, symptoms were transient: they came on some two hours after the series of BH dives and totally disappeared after an hour, and without treatment. Nonetheless, he was offered hyperbaric oxygen therapy (220 kPa) for 120 min together with IV fluids and oral aspirin (250 mg) and buflomedil (400 mg).

Whilst it is uncommon to suffer DCS from BH diving, it does occur.[2] This case lends support to the view that even such benign profiles with 'shallow' dives and 'adequate' surface intervals can nevertheless produce sufficient inert gas burden to produce nitrogen bubbles within the body that cause problems, particularly if one has a PFO.

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Key words
Breath-hold diving, decompression sickness, letters (to the Editor)

The poetry doctor

The rise and fall of a bubble

Hi, I am a bubble.
I once was just plain gas,
But now I've grown some substance
I even have a mass.
I don't know how it happened.
I was inert and slow
Then a sudden rising
Caused my form to grow.
You may think me hollow,
A weak-walled void created,
But with my surface tension
My ego gets inflated.

It lets me become rash
And get beneath your skin
To irritate and niggle
And scare you deep within.
Or, I'll grab a joint
To cause a deep-set pain
Until you're bent in agony
As if it is inflamed.
But best of all is when I move
Into the blood to flow,
And even take a shortcut
Through a PFO.
I'll head up into the brain
Where I can embolise
To confuse and fatigue
Or numb and paralyse.

I am amazed at what I do,
That I can cause such stress.
They even have a name for me
They call me DCS
But wait...what is happening?
I feel a great unease.
The outside pressure's going up.
I think I'm going to squeeze.
I'm getting so much smaller,
A force I can't resolve.
My life has just gone to pot.
Oh blast...I've just dissolved...

John Parker
<www.thepoetrydoctor.com>

Book review

Handbook on hyperbaric medicine, first edition

D Mathieu (ed)

812 pages, hardback
ISBN 1-4020-4376-7
Dordrecht: Springer; 2006.
Copies can be ordered online at <www.amazon.com>
Price: US$199.00

The Europeans have been active in the last two years in publishing their work. The European College of Hyperbaric Medicine (ECHM) Collection, Volumes 1 and 2 were reviewed recently in this journal[1] and now this textbook is intended as a state-of-the-art reference for the rational use of hyperbaric oxygen (HBO). It is the product of the Cooperation on Scientific and Technology (COST) programme, an initiative to implement and improve cooperation between scientists within the European Union. The HBO initiative (Action COST B14) was launched in 1998 and, under the chairmanship of Professor Mathieu of the University of Lille, has culminated in this book. The foreword states "*this handbook is intended as a reference document for researchers and clinicians alike – to be used both in the research laboratory and in everyday hyperbaric clinical practice; it also provides support material for teachers and will assist students in obtaining ECHM level II and III qualifications in hyperbaric medicine*."

This is truly an international collaboration with 60 contributors from 19 countries, stretching from Finland to South Africa, the French West Indies to Israel. Interestingly no scientists and only one physician from the United Kingdom contribute (to a single chapter), although another is a co-editor of the first of the three main sections. These three sections are devoted to the physical and pathophysiological bases of HBO, the clinical indications for HBO and the practice of hyperbaric medicine. Each is subdivided into a series of chapters written by experts, whilst the clinical indications section is further subdivided into recommended, optional and controversial or non indications for HBO.

At 800 pages long, this is not a text to read from cover to cover, but to use, as advocated, as a reference book. This reviewer has managed to read only about half the chapters and, therefore, cannot vouch for the entire text. The only other comprehensive textbook in hyperbaric medicine[2] was last published in 1999 so there has been a growing need for an up-to-date, authoritative publication. Professor Mathieu and his collaborators are to be congratulated on an excellent monograph that achieves their goals very well.

From a clinician's viewpoint (I am not a laboratory scientist), I found the information and commentaries in almost all the sections that I read to be informative and well presented in a logical manner, and that they often extended my knowledge and understanding. The third section provided an interesting insight into European hyperbaric medicine practice, including the approach to training and certification of personnel. As the programme director of a post-graduate, university-based course for diving and hyperbaric physicians, I found much useful material to assist in the preparation of our programme, and this will become one of our recommended textbooks.

Each chapter has an extensive international bibliography, including both English and non-English papers and lacking the tendency of USA publications to focus predominantly on American literature. However, such referencing needs to be contemporary, and this is not always the case. For instance, the most recent reference in the chapter on necrotising soft-tissue infections is for 1997 – an inexcusable failure to review the most recent literature in an important topic. Likewise, for the chapters on the effects of HBO on the cardiovascular system and on microorganisms and host defences, the most recent references are for 2000. By contrast, over half of the 93 references for the chapter on ischaemia-reperfusion injury are for later than 2000, including several from 2005. These differences are not sufficiently explained by the current extent of research in these areas, and such deficiencies need correction in future editions.

Presentation of the text is first class, with each chapter clearly laid out in sub-sections. Inevitably there is a degree of repetition between chapters written by different authors on related topics, but this is not pronounced, different approaches often complementing rather than mimicking each other. Despite English not being the first language for almost all authors, instances of awkward or incorrect usage are tolerable; though as a journal editor, I consider the two English-speaking editors could have done a better job of this – 'caelioscopy' instead of laparoscopy and 'high pressures of insufflation' instead of high inflation pressures (referring to mechanical ventilation of patients) are just two examples taken at random.

Searching for specific items can sometimes be daunting if using the index. For example, there are over 90 instances of the term 'decompression' listed. Once a chapter had been read, I found specific points again easily because of the clear subdivision of each chapter. There are relatively few typographical errors, and tables and diagrams are relevant, reasonably laid out and legible. However, the quality of photographs is generally disappointing, many being too small and of a poor standard. The book's cover disintegrated quite early suggesting the binding is inadequate.

This text is an important contribution to the hyperbaric literature for which the Europeans must be congratulated. It should be in the personal library of all physicians responsible for the care of patients undergoing HBO.

References

1 Mathieu D, editor. *The ECHM Collection, Volumes 1 and 2*. Flagstaff, AZ: Best Publishing Company; 2004 and 2005.
2 Kindwall EP, Whelan HT, editors. *Hyperbaric medicine practice*, 2nd edition. Flagstaff, AZ: Best Publishing Company, 1999.

Michael Davis
Editor, Diving and Hyperbaric Medicine

Key words
Hyperbaric oxygen therapy, textbook, book reviews

SPUMS

ANZ HYPERBARIC MEDICINE GROUP of SPUMS

INTRODUCTORY COURSE IN DIVING AND HYPERBARIC MEDICINE

Dates: 26 February to 9 March 2007
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Contact for information:
Ms Gabrielle Janik, Course Administrator
Phone: +61-(0)2-9382-3880
E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

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Diving and Hyperbaric Medicine

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For further information or to enrol contact:
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Royal Adelaide Hospital, North Terrace
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Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lmirabel@mail.rah.sa.gov.au>

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For information and application forms contact:
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Middle Head Road, Mosman, 2088 NSW, Australia
Phone: +61-(0)2-9960-0572
Fax: +61-(0)2-9960-4435
E-mail: <Sarah.Sharkey@defence.gov.au>

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For further information, please visit our website <www.eubs2007.org> or contact:
Dr Adel Taher, Secretary General of 33rd EUBS Annual Scientific Meeting
E-mail: <info@eubs2007.org>
Mobile: +20 12 212 4292 (24 hours)

BRITISH HYPERBARIC ASSOCIATION ANNUAL MEETING 2007

Dates: 01 to 04 November 2007 (pre-meeting diving programme 29 October to 01 November)
Venue: Oban, Scotland

For information contact: BHA 2007, Dunstaffnage Hyperbaric Unit, Scottish Association for Marine Science, Oban, Argyll, Scotland PA37 1QA
E-mail: <info@bha2007.org>
Website: <www.bha2007.org>

AUSTRALIAN AND NEW ZEALAND COLLEGE OF ANAESTHETISTS ANNUAL SCIENTIFIC MEETING

Venue: Melbourne Exhibition and Convention Centre

The Diving and Hyperbaric Medicine Special Interest Group session will be held on 28 May, 1530–1700 hr

For more information contact:
<anzca2007@meetingplanners.com.au>
or: <margaret.walker@dhhs.tas.gov.au>

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Dates: 09 to 11 August 2007
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Guest speakers Associate Professor Mike Davis, Professor Des Gorman, and Mr Dick Clarke

For further information contact: Czes Mucha
E-mail: <cmucha@mail.rah.sa.gov.au>
Phone: +61-(0)8-8222-5121
Fax: +61-(0)8-8232-4207

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Dates: 14 to 16 June 2007
Venue: The Ritz-Carlton, Kapalua, Maui

General information and online registration can be found at <http://www.uhms.org/Meetings/AMMeetingsMain.htm>

For additional information:
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Phone: +1-410-257-6606 extn 104
Fax: +1-410-257-6617
E-mail: <lisa@uhms.org>

ASIAN HYPERBARIC & DIVING MEDICAL ASSOCIATION (AHDMA) THIRD SCIENTIFIC MEETING 2007

Dates: 12 to 15 April 2007
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Registration details and further information contact:
E-mail: <secretary@ahdma.com>
Website: <www.ahdma.com>

Instructions to authors

(revised December 2006)

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 3,000 words or fewer. Inclusion of more than five authors requires justification as does more than 30 references per major article. Case reports should not exceed 1,500 words, with a maximum of 15 references. Abstracts are also required for all case reports and review papers. 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 should 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 columns should be as tab-separated text rather than using the columns/tables options or other software and each submitted double-spaced as a separate file. No vertical or horizontal borders are to be used.
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The Journal reference style is the 'Vancouver' style (*Uniform requirements for manuscripts submitted to biomedical journals*, updated July 2003. Website for details: <http://www.icmje.org/index.html>). In this system references 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 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.

There should be a space after the semi-colon and after the colon, and a full stop after the journal and the page numbers. Titles of quoted books and journals should be in italics. Accuracy of the references is the responsibility of authors.

Any manuscript not complying with these requirements will be returned to the author before it will be considered for publication in *Diving and Hyperbaric Medicine*.

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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The DES numbers are generously supported by DAN-SEAP

PROJECT STICKYBEAK

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 regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organisation to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence) to:
Dr D Walker
PO Box 120, Narrabeen, NSW 2101, Australia.
Enquiries to: <diverhealth@hotmail.com>

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-SEAP website: <www.danseap.org>
They should be returned to:
DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

DIVING-RELATED FATALITIES RESOURCE

The coronial documents relating to diving fatalities in Australian waters up to and including 1998 have been deposited by Dr Douglas Walker for safe keeping in the National Library of Australia, Canberra. Accession number for the collection is: MS ACC 03/38.

These documents have been the basis for the series of reports previously printed in this Journal as Project Sticky-beak. They are available free of charge to *bona fide* researchers attending the library in person, subject to an agreement regarding anonymity.

It is hoped that other researchers will similarly securely deposit documents relating to diving incidents when they have no further immediate need of them. Such documents can contain data of great value for subsequent research.

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All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of SPUMS.

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