Diving and Hyperbaric Medicine

The Journal of the South Pacific Underwater Medicine Society (Incorporated in Victoria) A0020660B

ISSN 1833 - 3516
ABN 29 299 823 713

SPUMS

Volume 36 No. 1
March 2006

Autopsies for diving fatalities

Asthma and diving – TSANZ discussion paper

DAN Diabetes Workshop

More on RDPs

Dirty diving

Print Post Approved
PP 331758/0015

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.
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The Society's financial year is January to December, the same as the Journal year.
The 2006 subscription will be Full Members A$132.00 and Associate Members A$66.00, including GST.
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The Editor's offering

In the analysis of diving fatalities, it has been said frequently that drowning is not a diagnosis, a cause must be looked for. Causation is often difficult to establish. Many potential factors – bad diving practice and poor decision making, contributing environmental conditions, concomitant medical problems, etc – combined with second-hand knowledge, conflicting eye-witness reports, inadequate police investigations and incomplete autopsy procedures mean that the truth is elusive in many cases. This is partly because the investigations may be conducted, with the best of intentions, by people who know little or nothing of scuba diving and with little guidance as to how to go about such investigations. As a result, surveys of diving fatalities such as those by the Divers Alert Network[1] in the USA and Project Stickybeak[2] in Australia have been hampered in their analysis of causation by the gaps in the information and the failure to ask the right questions.

This was also frequently true of coroners' autopsies in divers. Diving autopsies are undertaken to determine the immediate cause(s) of death, and to explore and identify potential underlying causes. Whilst drowning is often the terminal event, this is a diagnosis of exclusion made at the end of a full examination of the evidence and the deceased's body. In the 20-year review (1980–2000) of New Zealand fatalities, only about half the autopsies were conducted in accordance with recommended guidelines.[3] In Australia and New Zealand, this is now changing. New police guidelines for diving fatality investigations, combined with detailed autopsy advice and the involvement at an early stage in the investigation of physicians experienced in diving medicine should ensure that as much information as possible is gleaned from each such tragedy.

The Royal College of Pathologists of Australasia has for many years promulgated guidelines for autopsy in divers, but getting investigators to use this information has been a problem. Recently, Drs Lawrence and Cooke updated this information, which is freely available on the College website (www.rcpa.edu.au). However, it was felt that this excellent monograph needed wider promulgation amongst the diving medical community to enhance understanding of what should be involved and the difficulties inherent in interpreting autopsy findings.

Linked to this topic (we do not want them to die) is the on-going debate regarding whether asthmatics should scuba dive and if so what criteria should be used in assessing their suitably or otherwise. Widely differing views have been promulgated in the past by such learned groups as the Thoracic Society of Australia and New Zealand (TSANZ)[4] and the British Thoracic Society Fitness to Dive Group.[5] Recently, the TSANZ revisited this issue in a discussion paper in the *Thoracic Society News*, which drew a vigorous response from one respiratory physician.

This was timely as it had been intended to invite several respiratory clinicians and scientists to contribute to just such a debate in the *SPUMS Journal* in the near future. Instead, we re-publish the TSANZ material in this issue for members to consider. Please contribute to this debate. We would like to think that some combined recommendations could come out of the two societies working together on this thorny issue. It is noteworthy that some recent papers with new data do not appear in the bibliography of the TSANZ material.[6,7]

In 2003, the Society Executive was asked to review the need for medical assessment of users of a new underwater tourist device, the 'Scubadoo'. Quoting from the minutes of the Committee teleconference of 5 August 2003, "*the concensus…was that Scuba Doo should follow the guidelines set out by Workplace Health and Safety*." Dr Simpson, following a single-subject study suggests otherwise here. As he points out, the data could be predicted from basic principles (succinctly laid out by Camporesi and Bosco[8]), and he argues that the risks involved in this activity are minimal. What do you think? Registrations are going well for this year's ASM. Keep them coming!

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Michael Davis

Front cover photo of two technical divers using underwater scooters taken by Dr Simon Mitchell at about 40 msw depth at the Poor Knights Islands, where the 2007 SPUMS ASM diving will be held.

Review article

Autopsy and the investigation of scuba diving fatalities

Chris Lawrence and Clive Cooke

Key words
Diving deaths, scuba accidents, snorkelling, investigations, autopsy, review article, reprinted from

Abstract
(Lawrence C, Cooke C. Autopsy and the investigation of scuba diving fatalities. *Diving and Hyperbaric Medicine*. 2006; 36: 2-8.)
The investigation of diving fatalities is multi-faceted, and the pathologist has a central and critical part to play. It is important that the pathologist has knowledge of and understands the physiological risks and possible pathological changes associated with diving, and should be aware of other facets of the investigation. This review article reproduces the guidelines for autopsy in diving accidents on the Royal Australasian College of Pathologists website (www.rcpa.edu.au).

The investigation of the diving fatality is multi-faceted, involving inquiry into a number of areas, including:

i the past medical history of the deceased
ii the past diving history of the deceased (including satisfactory completion of appropriate training)
iii the circumstances of the dive (including water conditions, the dive profile, the diving techniques used, local dangers)
iv the equipment used
v the events before and after the fatal incident (including recent use of alcohol or drugs)
vi the medical findings at post mortem examination of the deceased

The pathologist has a central and critical part to play. In order to interpret the autopsy findings it is important that the pathologist has knowledge of and understands the physiological risks and possible pathological changes associated with diving. Additionally, prior to the post mortem examination, the pathologist should be aware of other facets of the investigation, including the results of examination of the dive equipment used by the deceased.

It may be helpful to seek assistance from the diving physician at the local hyperbaric medicine unit, or to have undertaken one of the underwater medicine courses offered by the Royal Australian College of Anaesthetists, the Adelaide Hospital Underwater Medicine Course or the Australian Navy Underwater Medicine Course.

Diving in Australia most commonly involves self-contained underwater breathing apparatus (scuba), or snorkelling/ breathhold diving. The use of surface supply breathing equipment or 'hookah' gear is common in commercial and recreational fishing and investigation of these deaths requires careful attention to previous training, examination of the settings of the equipment and review of the quality of air for carbon monoxide. Rebreather equipment is used by the military and by underwater photographers because of the absence of expired gas bubbles. Particular risks include failure of the scrubbers to remove carbon dioxide, hypoxia and oxygen toxicity seizures when using oxygen re-breather circuits at oxygen pressures of greater than 1.5–1.8 ATA. Using pure oxygen this can occur in as little as 9 metres of sea water (msw).[1] There is also a small but rapidly growing cadre of recreational divers using rebreathers despite the dangers associated with depth and gas toxicity.[2] This group has had a high mortality and morbidity so far, compared with ordinary recreational diving. Commercial saturation diving and military diving present particular problems and require specialised knowledge of the practice of this kind of diving. Most of the diving fatalities are recreational scuba divers and snorkellers.

Causes of death in divers

The usual immediate cause of death is **drowning** which probably accounts for between 52 and 86 per cent of the fatalities.[3] Drowning is the terminal event but it is important for the investigation to explore and identify potential underlying cause(s) (see summary):

i inability to swim
ii fatigue
iii panic
  • inadequate training
iv decreased level of consciousness
  • intoxication
  • nitrogen narcosis
  • seizures e.g., oxygen toxicity seizure
  • cerebral arterial gas embolism
  • hypercapnoea
v natural disease – acute myocardial infarct, asthma, diabetes, epilepsy
vi trauma
vii entrapment in caves or wrecks
viii physical disability
ix equipment malfunction

In over half of the drownings in one study, there were no external signs of distress, i.e., the drowning was silent.[4] Unfortunately, there is no diagnostic test for drowning at autopsy; it is a diagnosis of exclusion made at the end of a thorough post mortem medical examination.

Considering some of these potential underlying causes in further detail:

Decreased level of consciousness

Each 10 metres' sea water (msw) produces approximately 1 atmosphere (ATA) of pressure (i.e., at 30 msw the diver is subject to an ambient pressure of 4 ATA). During deeper dives some gases at the higher partial pressures become toxic. Nitrogen at depths of over 30 msw produces **nitrogen narcosis** (impairment of intellectual and neuromuscular function) which was reported to contribute to 9% of fatalities in ANZ studies.[5] Divers exposed to **oxygen** at pressures of greater than 1.5–1.8 ATA for some period can suffer seizures and drowning.[6] Similarly at depth, **carbon dioxide** retention and toxicity may depress consciousness and lead to death by drowning.

Among snorkellers, unconsciousness due to **breath holding following hyperventilation**, sometimes colloquially termed **shallow water blackout**, is a common cause of drowning. It is aggravated by the hypoxia due to ascent. The snorkeller hyperventilates then dives. During descent the increased ambient pressure maintains the partial pressure of oxygen despite consumption. However, during ascent in addition to the oxygen consumed the ambient pressure also drops, producing a very rapid drop in the partial pressure of oxygen and in oxyhaemoglobin and loss of consciousness that can result in drowning.

In divers using compressed gases, **pulmonary barotrauma and cerebral arterial gas embolism (PBT/CAGE)** probably represents the next largest group of fatalities (13–24%). Boyle's Law states that, at a constant temperature, the volume of a gas is inversely proportional to the pressure. Pulmonary barotrauma followed by cerebral arterial gas embolism (PBT/CAGE) may occur in a diver who makes an uncontrolled ascent, especially without exhaling. The volume of the gas in the lungs expands during ascent as the ambient pressure falls. If the diver does not exhale, air is forced from the airspace into the pulmonary circulation to the heart and hence into the cerebral circulation (CAGE). Pulmonary barotrauma has been reported in dives in as little as two metres of water. The history of the diver coming to the surface rapidly, crying out and then losing consciousness within seconds to minutes is characteristic of this condition. Because of the loss of consciousness there is often evidence of drowning as an agonal event. Pulmonary barotrauma less commonly causes pneumothorax; however, CL has observed a tension pneumothorax in a diver with asthma, possibly due to air trapping.

A number of deaths have occurred during **very deep dives** (50–80 msw) using compressed air. Death appears to be a consequence of loss of consciousness due to a combination of increase work of breathing, nitrogen narcosis, oxygen toxicity, hypercapnoea and possibly impaired venous return to the heart. These cases are usually brought to the surface rapidly and inevitably show significant post mortem decompression or 'off gassing'. It is important to recognise that the diver was unresponsive before ascent and that pulmonary barotrauma and cerebral arterial gas embolism (PBT/CAGE) was not the underlying problem.

Decompression sickness (the bends) is a rare cause of death in amateur divers, but a common cause of morbidity in divers. The symptoms may occur minutes to hours after the dive. Given well-developed retrieval services, deaths are rare. The bubbles that cause this process are frequently not detectable in the live patient, and would probably not be obvious at autopsy. Unfortunately, post mortem decompression or off gassing is common and is a major cause of artefactual gas at autopsy (see below).

Natural disease, particularly **ischaemic heart disease**, can cause sudden death or drowning especially in the older diver. **Asthma** is regarded by many as a contra-indication to diving. This remains a controversial issue; however, asthmatics make up around 1–2% of divers while 9% of the deaths in one ANZ series were in asthmatics.[7] **Idiopathic pulmonary oedema** associated with scuba diving[8] is well described in living divers and it seems reasonable that this could cause fatalities, but these cases are probably classified as drowning at autopsy.

Physical injury including head injury from boat propellers or rocks can lead to drowning. The incidents of shark attack on divers are rare but appear to be increasing.[9] Bites from fish, eels, sea snakes, blue ringed octopus, stings from fish, cone shells, coelenterates and urchins are uncommon but should be looked for; it is doubtful if envenomation from a bite or sting may of itself explain the fatality. However, naturally the circumstances and/or the pain of the bite may result in loss of the self control required to dive safely.

Overall, the investigation of the fatality may be expected to identify multiple interacting problems that have combined to cause death. These problems usually fall into one of four groups:

i **Medical factors** including pathology, psychology (panic, fatigue) and physiology (lack of physical fitness),
ii **Diving techniques**, lack of air, failure to stay in visual contact with a buddy,
iii **Equipment problems**, faults, misuse and loss of equipment, and
iv **Environmental factors**, current, depth and visibility.

Most investigations reveal a critical error in judgement by the diver or failure to follow recommended safe diving

procedures. Commonly identified factors in Australian diving fatalities include low air or out-of-air situations (56%), buoyancy problems (over-weighted, failure to ditch weight belt) (52%), panic (39%), salt-water aspiration (37%), tidal currents or surge (36%) and fatigue (28%).[10] Vomiting (10%), drugs (7%) and hypothermia, entrapment and loss of equipment such as the facemask often contribute to drowning.

Post mortem examination

1. THE HISTORY

The post mortem examination of the body of the deceased should not proceed until the pathologist is satisfied that there is a good understanding about the circumstances of the death. Naturally, there is some urgency for the post mortem examination to proceed, to minimise the amount of post mortem change obscuring important pathological findings. Accordingly, there should be open and early communication between various experts involved with the investigation – inquiry police officer; officer examining the dive equipment; police divers; dive physician.

In most cases it is useful to obtain the following before offering a final opinion as to the cause of death:

1. Police and witness statements
- Police reports to the Coroner
- Statements from other divers and boat crew
- Dive profile, depth, duration, weather and current conditions of dive
- When did the diver start to have problems? During descent, on the bottom, during ascent, after the dive.
- Did the diver ascend rapidly?

2. Diving history of diver
- Diving log and experience
- Diving certification
- Diving medical/past medical history especially ischaemic heart disease, asthma, diabetes and epilepsy

3. Examination of equipment
- How much air is left in the tank? Composition (especially in technical diving)? Presence of carbon monoxide?
- Regulator/tank/BC including testing under relevant conditions
- Dive computer log down loaded (this is the best evidence of a rapid ascent)
- Was the diver using too much weight on the weight belt?
- Additional information should be sought if the diver was using a 'rebreather'

4. Autopsy (preferably by pathologist with experience of diving fatalities)
- Report of X-ray or CT scan taken before autopsy
- Autopsy findings including descriptions of site and approximate volume of gas
- Histology of relevant organs especially lungs, heart and brain
- Toxicology including carbon monoxide, alcohol and drug screen

2. BODY STORAGE

The body of the deceased is often transported to the mortuary with part of the diving equipment still present – a wetsuit, fins, mask, weight belt. Because of the insulating effect of the wetsuit it is common for the body of the deceased to show early post mortem decomposition changes, despite refrigeration. The pathologist should consider reviewing and documenting appropriately (including photography) the external appearance of the body at the time of first receipt at the facility; the wetsuit may then be removed enabling satisfactory refrigeration of the body of the deceased.

3. RADIOLOGICAL EXAMINATION FOR GAS AS PART OF THE POST MORTEM EXAMINATION

Early X-ray examination of the body of the deceased should be undertaken. Important accumulations of gas may be demonstrated such as pulmonary cysts, pneumothoraces, mediastinal emphysema, intravascular gas (PBT/CAGE).

1. **CT scan** is a very sensitive way of detecting small amounts of gas in the body. It requires rapid access to a body CT scan. The CT scan will show gas in the cerebral arteries and in the right and left ventricles of the heart. Small amounts of gas in the liver are mostly a decomposition artefact.

2. **Erect X-ray of chest and abdomen** will show relatively large amounts of gas present in the right ventricle (an air-fluid level in the right ventricle or pulmonary trunk), aorta and neck veins. X-rays of the head will show gas in the cerebral vessels, while X-rays of the limbs will show gas in veins, joints and soft tissue in decomposition and post mortem off gassing.

In pulmonary barotrauma and cerebral arterial gas embolism (PBT/CAGE) you are looking for gas in the cerebral arteries and the left ventricle of the heart. Gas is also seen in the right ventricle. Finding gas in the right ventricle appears counter intuitive, however; it has been suggested that in PBT/CAGE the gas emboli pass through the capillaries and veins and become trapped in the pulmonary vein/right ventricle. Large amounts of gas can also be seen in the right ventricle in off gassing, decomposition and resuscitation.

We have used both CT scan and erect chest X-ray with success. The CT scan is more sensitive, but it can be very difficult to interpret the significance of small volumes of gas. CT or MRI scans can be useful to detect bubbles due to decompression sickness in the spinal cord.

Unfortunately the presence of intra-vascular gas is common and is not specific for PBT/CAGE. In a study of 13 diving fatalities,[11] intra-vascular gas was detected in 12 of the 13 cases, while only four cases had a history strongly suggestive of PBT/CAGE and three were possible CAGE.

Intra-vascular gas can also be due to:

1 Decomposition – the bacteria in the body produce gas after death. This can be seen in the portal/hepatic veins as little as 12 hours after death. If not refrigerated the body will show extensive gas both intra-vascular and in soft tissue within 36 hours. Hydrogen and methane in the recovered gas are an indication of decomposition, provided a gas-tight Hamilton syringe is used and no blood enters the syringe.
2 Resuscitation – following resuscitation with an endotracheal tube and positive ventilation, significant gas in the heart was detected in chest X-ray in 5 of 13 non-diving fatalities.[11]
3 Post-mortem decompression or **off gassing** – during a deep dive the tissue absorbs nitrogen. If the diver then ascends rapidly and dies, or dies on the bottom and is brought quickly to the surface, nitrogen bubbles will form in the tissues and vessels. This process will produce both intra-vascular and soft tissue gas, and should theoretically be distinguishable from CAGE by the presence of gas in muscles and joints. In practice it is difficult to identify a PBT/CAGE in the presence of post-mortem decompression. It remains to be established how deep and how long the dive needs to be to see off gassing. Preliminary data from Townsville suggest that it may occur experimentally after 45 min at 18 msw (Griffith D, personal communication, 2005).

We do not subscribe to the view that the only thing that intra-vascular gas indicates is that the person has been diving. Detecting and measuring intra-vascular gas is important in establishing that PBT/CAGE could have occurred. However, it is necessary to have supporting evidence. The following are the criteria we use for diagnosis.

Major criteria for PBT/CAGE are:

1 History of a rapid ascent followed by loss of consciousness.
2 Gas in the left side of the heart, circle of Willis, coronary and retinal arteries.
3 Low probability of off gassing or decomposition.
4 Mediastinal or subcutaneous emphysema limited to the peri-thoracic area (e.g., supra-clavicular area) and/or pneumothorax.

Minor criteria are:

5 Low air or panic situation.
6 Student or novice divers.
7 Over-inflated buoyancy jacket or ditched weight belt.
8 Dive computer evidence of a rapid ascent.
9 Other evidence of barotrauma, subcutaneous emphysema or pneumothorax.

4. AUTOPSY

Once certified dead, the body of the deceased should be placed in a sealed body bag and transported as soon as possible to the autopsy site. The loss of any equipment e.g., mask, weight belt or fins should be noted. The equipment should be sealed with the valves closed to retain the breathing gas for analysis and transported as soon as possible for examination.

External examination

A conspicuous plume of white foam around the nose and mouth (pulmonary oedema fluid) is commonly seen in drowning. This may disappear quickly, so early examination of the body is essential. Compression marks around eyes/nose and small conjunctival haemorrhages usually indicate mask squeeze, suggesting inadequate equalising during descent, possibly while unconscious. Examination of the eardrum with an otoscope may show perforation (an event which usually occurs during descent). Biting of the tongue and lips may indicate fitting (also check the mouthpiece). Haemorrhagic (i.e., with bruising) abrasions and bruises on the face and limbs indicate injuries that occurred before the circulation stopped. They may include trauma due to rocks or animal bites. Post mortem injury due to animal scavenging is common around the lips and eyes and is recognised by lack of haemorrhage in the underlying tissues.

Initial dissection

Past recommendations have included initial dissection of the cranial and chest cavities underwater. Whilst opening the head and chest under running water does allow the direct demonstration of gas in the heart and vessels, **the process is cumbersome, difficult without specialised equipment, potentially dangerous to the prosector and assistant because it is hard to see and, in the end, of dubious validity** as the sawing of the skull, out of water, severs vessels which may introduce air into the venous system. The radiological demonstration of gas prior to any dissection is more reliable than either 1 or 2 below.

1. PRIMARY OPENING OF THE ELEVATED CHEST AND ASPIRATION OF THE HEART

This follows radiological documentation of gas by CT scan or erect chest X-ray.

The neck block is placed under the shoulders elevating the chest, causing the gas to collect in the superiorly positioned right ventricular outflow track and proximal aorta. The skin of the neck and chest is reflected taking care not to cut the neck veins. The sternum is removed cutting the costal cartilages with a scalpel and the pericardial sac is opened with scissors. The four chambers of the heart are then aspirated with a needle and syringe keeping the needle in the upper most point of the chamber. The volume of gas in

each chamber is recorded. Alternatively the gas can be collected under a water seal. The gas volumes are then correlated with the CT scan or X-ray findings. If gas is present the right ventricle will bulge out from the pericardial sac.

We do not test for pneumothorax, as past experience indicates that it is uncommon in diving fatalities and if present, will usually show up on the erect chest X-ray or CT scan.

2. DISSECTION UNDER WATER

The scalp is incised then reflected under running water, the skull is cut carefully avoiding cutting the dura and the brain is removed underwater (see "Post mortem Technique in Fatal Diving Accidents". Royal College of Pathologists of Australasia Broadsheet No.27).

The chest can be opened underwater, or the heart can be opened after filling the opened pericardial sac with water.

Head and neck

If the chest is opened before the head, the carotid arteries should be tied off at the base of the neck. The head is opened and the presence of air in the cerebral arteries and dural veins is noted.

The middle ears can be removed, fixed in formalin then decalcified and serially sectioned to show damage to the middle and inner ear. An ENT surgeon may appreciate the opportunity to assist with this examination. Given concerns regarding tissue retention, in practice, we now examine the eardrum using an otoscope and leave the middle ear intact, unless there is other evidence of a significant middle or inner ear problem – it should be recognised, however, that this approach may miss potentially important middle and inner ear pathology.

Formal neuropathology examination of the brain is advisable (and of the spinal cord if spinal decompression sickness is suspected). In some PBT/CAGE cases, there are occasional small perivascular haemorrhages in the brainstem on the floor of the fourth ventricle, of uncertain significance. The minimum time for formalin fixation of the brain to enable optimal neuropathology examination appears to be about 48 hours' immersion in 20% formalin. If, for local reasons, the Coroner (or other legislated authority) is not prepared to authorise brain retention for formal neuropathology examination, then examination of the fresh brain should nonetheless proceed in the same systematic and careful manner – the small perivascular haemorrhages of PBT/CAGE are still identifiable. Naturally, where resuscitation is initially successful and the diver survives for some time, the likelihood of identifying pathological changes in the brain and spinal cord increases.

Chest and abdomen

The finding of air in the heart, inferior vena cava and portal vein are described above.

Over-expanded lungs that cover the heart and show the impressions of the ribs may be seen with drowning and in conditions where there is peripheral air trapping such as asthma and deep aspiration of vomitus; this may also be a feature of respiratory resuscitation.

Water in the stomach and florid pulmonary oedema in the trachea and lungs also suggest drowning.

The lungs can be inflated with air underwater to find air leaks that suggest pulmonary barotrauma. Where indicated, inflation of the whole lungs with formalin allows detailed examination for apical bullae with associated haemorrhage in an otherwise normal lung. This may be the source of an unexpected PBT/CAGE.

The heart should be examined closely for coronary atherosclerosis and other cardiac anomalies which can cause sudden death.

The foramen ovale of the heart should be tested for probe patency as this may give rise to paradoxical air emboli.

Musculo-skeletal system

In long term and commercial divers we have in the past examined the femoral head, but found no pathology. With X-rays of the joints we tend now only to examine if the joint appears radiologically abnormal.

Histology

Comprehensive microscopic examination of all organs should be undertaken.

Divers who are kept alive for some hours may show significant pathology in the heart and central nervous system including small infarcts in the cardiac muscle and spinal cord.

Other tests

Preserved blood and urine should be submitted for alcohol, a drug screen and carbon monoxide.

There is no reliable test for drowning.

Diatoms remain the best chance for a diagnostic test for drowning; however the test is not routinely available, as it requires working with concentrated acid and experience in distinguishing the species of diatom. A sample of water from the site, blood, bone marrow, kidney and lung should be collected if this test is contemplated.

Comparison of chloride levels in right and left ventricular blood has fallen into disrepute. In a salt-water drowning sampled soon after death where no cardiopulmonary resuscitation occurs there will sometimes be a 17mmol.l^{-1} increase in the left ventricle chloride level.

Acknowledgement

We would like to thank Carl Edmonds for his help, guidance and inspiration over the past decade and Jim Caruso for his notes (Caruso J L, Bell M. The medicolegal investigation of a recreational diving fatality. *American Academy of Forensic Science Workshop*, 2002) and his insights.

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Radiology images courtesy of Dr Robin Harle, Department of Radiology, Royal Hobart Hospital, Hobart, Tasmania.

and is endorsed by The Royal College of Pathologists of Australasia (RCPA).

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Summary of features of the common causes of death

(i) Drowning

Equipment	- Leaking or poorly maintained equipment - Loss of equipment e.g., mask, fins - Carbon monoxide in air mix - Faulty CO_2 scrubber in a rebreather - Technical diving use of hypoxic bottom mix on the surface or oxygen-enriched mixtures at depth
Autopsy	- Pulmonary oedema fluid in mouth, trachea and lungs - Over-expanded lungs covering the heart, with impressions of the ribs - Salt water in the stomach (check sodium and chloride levels) - The amount of water inhaled may be variable. Dry drowning is in our experience rare in salt water.

(ii) Pulmonary barotrauma/cerebral arterial gas embolism (PBT/CAGE)

History	- Inexperienced diver, out of air, night dive, panic situation - Rapid ascent followed by sudden loss of consciousness
Equipment	- Rapid ascent on dive profile downloaded from dive computer
CT scan	- Gas in the left ventricle and cerebral arteries - Mediastinal emphysema - Pulmonary barotrauma
Autopsy	- Gas in the left ventricle and cerebral arteries - Bullae and dilated airspaces with haemorrhage in the lungs

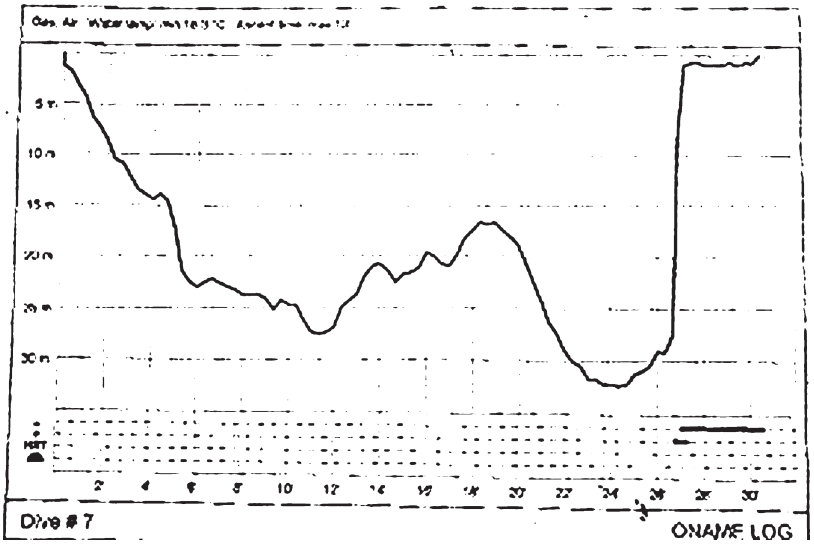


Figure 1
Dive profile downloaded from dive computer. The diver attempted to breath from buddy's buoyancy compensator then ascended 29 metres in 20 seconds, surfaced coughing and became unconscious. Cause of death was pulmonary barotrauma/cerebral arterial gas embolism (PBT/CAGE).

Figure 2
This X-ray is of a diver who died after a 43 msw rebreather dive. He was found on the bottom with his mask off shortly after leaving the surface. He had set his equipment incorrectly and had a P_iO_2 at depth well in excess of 202 kPa. His rescuer put an octopus regulator in his mouth and purged gas into him all the way to the surface. Gas-fluid levels are seen in the heart and gastro-intestinal tract.

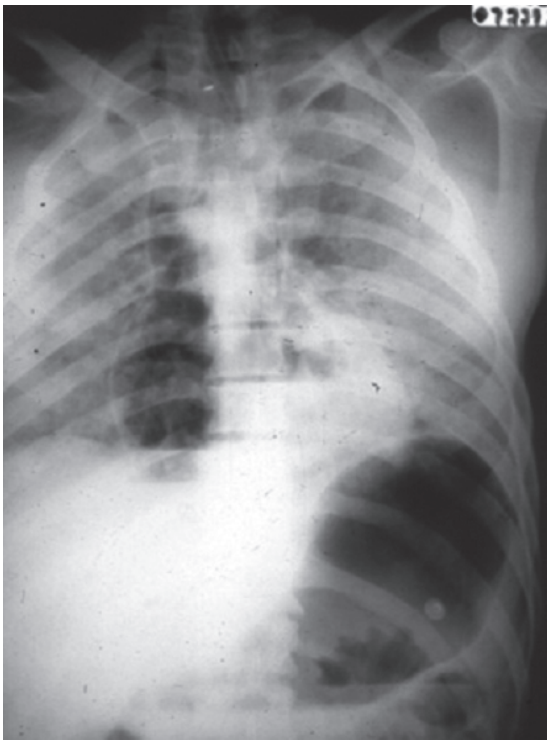
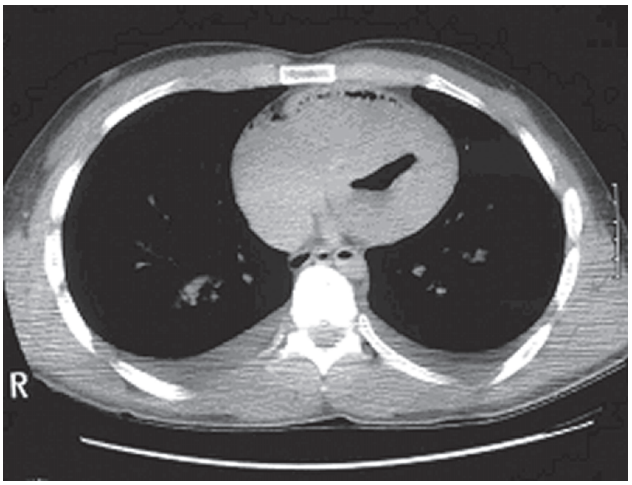
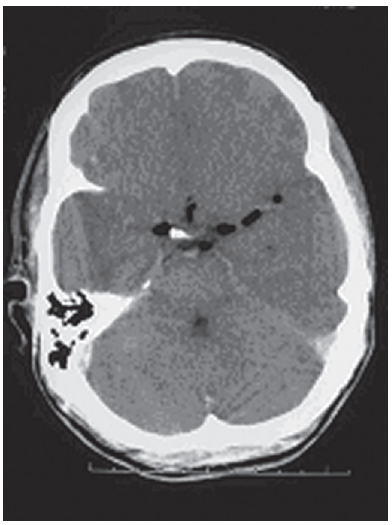


Figure 3
CT scan of chest and brain showing gas in the left ventricle and right middle cerebral artery in a diver who had separated from his buddy, was low on air and ditched his weight belt. The ascent was unwitnessed but the history and findings together suggested pulmonary barotrauma, with cerebral arterial gas embolism (PBT/CAGE).





Short communication

Medical standards for the use of 'Scubadoo' – a discussion paper

Graham Simpson, Janine Ferns, Trevor Knight and Malcolm L Heron

Key words
Recreational diving, diving, safety, respiratory, Scubadoo, risk management

Abstract
(Simpson G, Ferns J, Knight T, Heron ML. Medical standards for the use of 'Scubadoo' – a discussion paper. *Diving and Hyperbaric Medicine*. 2006; 36: 9-11.)
'Scubadoo' is a novel recreational diving device which operates at a fixed depth of three metres' sea water (msw). The diver is free to move in an air-filled dome replenished by continuous air flow from a scuba tank which is an integral part of the device. Calculations show that the equilibrium concentration of carbon dioxide and oxygen in the dome depend on the volumetric air flow from the compressed-air cylinder. Experiments carried out with an air flow of 20 $l.min^{-1}$ gave gas mixtures consistent with the calculations. This provides the basis of safe design for the air supply to the dome. The medical issues that may arise in the use of the Scubadoo device are discussed. Most of the medical issues which apply to scuba diving are minimised here because of the rigid three msw depth limit. Pulmonary barotrauma and also bronchospasm for asthmatics are theoretically possible but are considered to be low-risk conditions in the context of the use of the device. Epilepsy and other conditions likely to cause sudden loss of consciousness are the only contra-indications to use of the device. It is concluded that Scubadoo should not be subjected to the same medical restrictions as scuba diving.

Introduction

The 'Scubadoo' is an underwater recreational device, relatively new to the dive industry. The device consists of a submersible scooter-like vessel that is steerable and powered by an electric motor. The passenger sits astride the seat and an acrylic dome fits over the whole upper body of the passenger (Figure 1). The dome is air filled so that the passenger's shoulders and head are above the water level. Air is replenished at a constant rate from an integral scuba tank. The vessel is maintained in an upright position partly by the weight of the battery and motor at the base, and also by the buoyancy of the air in the acrylic dome, with the additional safety device of a line to a surface buoy attached to the dome. The vessel is operated at a fixed depth of three metres' sea water (msw). The electric motor and propeller provide slow forward motion and the vessel can be steered by the passenger.

Normal practice is to have a diving instructor wearing scuba gear supervising each Scubadoo device. The device is steered around a short tour underwater for approximately 20 minutes. The question has been raised as to what medical standards should apply to potential users of this device. We have undertaken some simple calculations and some in-water testing of the device to try to clarify the situation.

Potential medical problems

Users of the Scubadoo are breathing compressed air at the ambient pressure of one atmosphere plus three msw. There is no second-stage regulator and the user's head is in the dome surrounded by approximately 25 l of air. Fresh air is supplied to the dome from a compressed-air scuba cylinder at a constant flow rate of 20 $l.min^{-1}$. Potential problems, therefore, are:

1 decompression sickness
2 hypoxia because of inadequate air replacement
3 carbon dioxide retention because of inadequate flushing of the dome
4 barotrauma to the ears and/or sinuses
5 pulmonary barotrauma
6 exacerbations of pre-existing medical conditions, particularly asthma
7 panic.

Theoretical calculations

To investigate some of these problems further, calculations were undertaken regarding the expected alteration of gas composition in the dome using expected oxygen (O_2) consumption rates and carbon dioxide (CO_2) production rates for an adult at rest. It was assumed that oxygen removal rate would be 300 $ml.min^{-1}$ and carbon dioxide production would be 250 $ml.min^{-1}$. The approximate volume of the airspace is 25 l. Calculations were performed for three different inflow rates from the compressed-air bottle of 20, 10 and 5 $l.min^{-1}$. In summary, it was shown that the volume of air in the dome does not influence the final levels of oxygen and carbon dioxide, although the smaller the volume, the more rapidly these equilibrium levels are approached. The calculated equilibrium values using the O_2 and CO_2 figures for an adult at rest given above and the flow rate of 20 $l.min^{-1}$ are: equilibrium O_2 concentration

Table 1
Changes in temperature, humidity and CO_2 over time in a just-submerged Scubadoo, using a continuous 20 l.min^{-1} fresh-air flow rate

Time (min)	Temperature (°C)	Relative humidity (%)	CO_2 (%)*
0	33.3	60.5	0
1	33.9	73.8	0.82
3	34.9	78.0	1.09
5	36.4	84.0	1.24
8	35.9	87.0	1.24
11	36.7	92.4	1.52
13	37.2	88.0	1.66
16	37.2	87.0	2.07
19	37.9	83.6	2.22
22	38.7	79.8	2.08
25	41.3	76.6	2.07
27	41.4	79.4	2.35

*corrected for relative humidity, temperature and saturated vapour pressure of water

19.49%, equilibrium CO_2 concentration 1.29%. If it is assumed that, perhaps because of psychological stress or the minor physical exertion involved, the CO_2 production is doubled these figures change only slightly, with equilibrium CO_2 level rising to 2.51%. The critical factor determining the equilibration levels was the gas inflow rate. A flow of 10 l.min^{-1} approximately doubled the equilibrium CO_2 level but remained safe; however, flow levels below this could cause problems. Using 20 l.min^{-1}, 90% of the equilibration levels were reached in less than 12 minutes and values at the end of the 20-minute dive would be very close to the calculated equilibration levels. For a further explanation see *Bennett and Elliott's physiology and medicine of diving.*[1]

Methods

Experiments were undertaken with a Scubadoo in a swimming pool with a volunteer. An air replenishment flow rate of 20 l.min^{-1} was used. Measurements were made of CO_2 levels in the dome air using an end tidal CO_2 meter (BCI International Capnocheck Model 20600A1) and of the temperature and relative humidity of air within the bell using a Centre 311 humidity and temperature meter Model RS-232. Measurements were made throughout a 27-minute 'dive'. Because of limitations of the monitoring equipment the Scubadoo dome actually remained at the surface rather than being completely submerged to 3 msw. The CO_2 levels were corrected for the alterations in temperature and relative humidity to allow for the vapour pressure of water under changing conditions.

Results

The results are shown in Table 1. The observed changes in

Figure 1
Artist's impression of Scubadoo™ device

CO_2 show close agreement with the calculated results, with a final CO_2 concentration of about 2.3%. The temperature in the dome rose significantly whilst relative humidity remained at approximately 80%.

Discussion

The final carbon dioxide concentration of about 2.3% would suggest that CO_2 production in the subject was greater than the basal assumed rate of 250 ml.min^{-1} but did not exceed 500 ml.min^{-1}. The calculated oxygen content of the dome atmosphere would thus be over 19%. The rise in temperature is explained by the fact that the experiment was performed at the surface on a sunny day. It would be anticipated that temperature would not rise so much with the dome completely submerged. Of interest is that the

relative humidity of the air in the bell remained around 80% despite its constant replenishment with drier air from the scuba tank.

ISSUES

We are now able to address some of the potential medical problems related to operation of a Scubadoo device.

Decompression sickness
Exposure to nitrogen at a pressure of three msw for 20 minutes is insufficient to produce decompression sickness.

Barotrauma
Descent of three msw is unlikely to cause significant ear or sinus squeeze even without equalisation. Nevertheless, we would recommend that the participants be instructed in equalising techniques. The accompanying diver can easily signal through the transparent dome that equalisation should be done and check that the occupant is not distressed.

Pulmonary barotrauma
Pulmonary barotrauma is a theoretical possibility in a rapid ascent from three msw. However, it seems difficult to envisage a situation where this could occur with a Scubadoo, which is essentially hanging on a surface buoy at this depth and is negatively buoyant. The only way that this would be a problem is if the occupant exited the dome suddenly and ascended rapidly holding his or her breath. In a one-to-one supervision situation this would seem to be extremely unlikely to occur.

Hypoxia and carbon dioxide retention
Both calculation and direct experimental evidence based on a flow rate of 20 $l.min^{-1}$ (less than that used in practice, 25 $l.min^{-1}$) show that the air replenishment rate is adequate to keep O_2 levels in excess of 19%. Allowing for the fact that this in practice would be in a slightly increased pressure of three msw the partial pressure of O_2 in inspired gas would be effectively unaltered. CO_2 levels do rise, but not to the level where any symptoms would be anticipated or to a level where there would be respiratory stimulation by hyperventilation. A recent study of head hoods under hyperbaric conditions supports these conclusions.[2]

Medical conditions
The medical condition that causes the most debate regarding scuba diving is, of course, asthma. The theoretical objection to asthmatics scuba diving is of precipitation of an attack of bronchospasm by the scuba diving environment. Precipitants usually mentioned include exercise, inhalation of cold, dry air and the possibility of inhalation of an aerolised mist of sea water caused by a faulty regulator. Though considered spurious by some, this last point clearly is irrelevant in relation to the Scubadoo as there is no second-stage regulator and the occupant's head and shoulders are well clear of water and breathing is normal. Scubadoo does not involve significant exertion and our experiment has shown that the air in the dome is both warm and moist. Therefore, there seems no reason for asthma to be regarded as a contra-indication to use of the device. As far as other medical conditions go, the lack of an important exertional component and the close supervision provided would seem to render the device safe for almost anyone with the exception of patients suffering from epilepsy or other conditions likely to cause sudden loss of consciousness.

Panic
The underwater environment can produce panic and this seems to be the main potential problem of the Scubadoo. This would need to be dealt with by adequate instruction and supervision of Scubadoo users.

Conclusions

For the reasons outlined above, it is our opinion that the Scubadoo should not be subjected to the same medical restrictions as scuba diving. Assuming adequate instruction and supervision, the device should be available to anyone of adequate size to use it, and medical conditions, with the exception of epilepsy, should not be regarded as a contra-indication.

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The world as it is

Summary of knowledge and thinking about asthma and diving since 1993*

Discussion paper for the Thoracic Society of Australia and New Zealand, November 2004

Sandra D Anderson, Robert Wong, Michael Bennett and Lutz Beckert

This discussion paper has a currency of 5 years from the date of publication unless superseded.

Introduction

The 1993 TSANZ discussion paper appears to have been one of the first to be published by a Thoracic Society.[1] This probably reflects the high prevalence of asthma in the Australian and New Zealand population and the high morbidity and mortality associated with the disease at that time. Since 1993 there has been a nationwide effort to improve education of doctors and patients about asthma and its treatment through advertising campaigns. Inhaled corticosteroids are more available and more widely used than they were 10 years ago. Lung function tests are more commonly requested and the testing apparatus used in laboratories is more sophisticated, permitting measurements of flow at low lung volumes. Self-monitoring of symptoms is more common and many asthmatics own peak flow meters. The finding of bronchial hyperresponsiveness (BHR) or bronchial hyperreactivity in a significant proportion of healthy young adults, with a past history of asthma, seeking employment in occupations excluding active asthma, or seeking permission to use drugs before sporting events, supports the need for objective testing before clearance to dive. It is now clear that BHR to hyperpnoea of dry air, an absolute contraindication for scuba diving, can be associated with normal values for resting spirometry.[2] It is also clear that responses to pharmacological challenges cannot be used to exclude BHR to dry air or to hyperosmolar aerosols.[3] In 2004 most public hospital laboratories and many private laboratories provide a choice of bronchial provocation tests. These tests are now well standardised relative to 1993.[4-8] The bronchial provocation tests recommended are those that involve the stimulus to which the intending diving is exposed, either exercise or eucapnic hyperpnoea of dry air and non-isotonic aerosols.[8]

Prevalence of Respiratory Disease

There is now excellent documentation of the prevalence of asthma in Australia and New Zealand.[9-12] Some 18-20% of school children aged 8-13 years have BHR to histamine or exercise.[12-14] This group of children would now be at the age of those applying to dive and the high prevalence underscores the importance of using appropriate tests to identify those at risk during diving. Asthma is a term that covers a disease with a wide variation in severity of symptoms and BHR. These hallmarks of asthma will change over time and in response to treatment. It is clear however that BHR usually precedes symptoms of the disease and remains long after treatment has relieved symptoms and lung function has returned to normal. Exercise-induced asthma is a good example of this.[15,16]

The presence of other respiratory diseases in the Australian and New Zealand community are not as well described as asthma, with the exception of cystic fibrosis. However, anyone with a history of disease or symptoms consistent with a respiratory problem should be referred for assessment of lung function, including spirometry, lung volumes, diffusing capacity and bronchial provocation testing.

The Role of Bronchial Provocation Tests

The role of the bronchial provocation test in the assessment of the diver or intending diver should be to identify those persons who would be at risk from acute airway narrowing during the activities associated with diving. Those who have demonstrable BHR should be told that they may be at increased risk of pulmonary barotrauma (PBT), and the details of the possible consequences of this should be explained.

There are two types of tests used for bronchial provocation referred to as direct and indirect. The direct challenges 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. Bronchial hyperresponsiveness to these agents is not an

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Footnote

* The physics of diving was presented in Jenkins C, Anderson SO, Wong R, Veale A. Compressed air diving and respiratory disease. *Med J Aust*. 1993; 158: 275-9 and is available from the TSANZ Office [www.thoracic.org.au].

absolute contraindication for diving because the BHR is not specific for identifying asthma and it is commonly recorded in healthy people without symptoms,[11] elite athletes,[17-19] and in smokers.[20] Elite swimmers[17,21] and people who dive regularly using scuba also have a high rate of BHR to inhaled histamine and methacholine so that it would seem unfair to exclude someone from diving on the basis of BHR to these agents.[22, 23] It is not known if the BHR in divers is a consequence of airway injury from breathing dry air over long periods of time,[24] but this is suggested by the trend towards an association between BHR and number of compressed air dives performed.[22]

In contrast, the indirect challenges are specific for identifying currently active airway inflammation. This has been demonstrated by the BHR to these challenges being reduced over weeks by treatment with inhaled steroids.[25-31] The indirect challenges include exercise, eucapnic hyperpnoea of dry air (EVH) and challenges with hypertonic aerosols.[6-8] These challenges cause release of mediators from inflammatory cells in the airways,[32,33] probably in response to an increase in airway surface osmolarity. These mediators, which include leukotrienes and prostaglandins, are potent and on a molar basis only one thousandth of the dose is required to induce the same degree of airway narrowing as histamine or methacholine.[34] The fact that these potent mediators are involved in these challenges may serve to explain why they are more sensitive for identifying people with exercise-induced asthma compared with methacholine or histamine.[14,35] The BHR to indirect challenges is associated with the presence of inflammatory cells[29] and has been used successfully to assess response to treatment and withdrawal of treatment with inhaled corticosteroids.[25,30,36]

For these reasons over the last 10 years bronchial provocation using indirect challenges has become more widely used and reported for assessment of asthma for diving and for assessment for some occupations where exercise-induced bronchoconstriction is unwanted (e.g. NSW Police Force). Findings from a recent study of athletes suggest that pharmacological stimuli should no longer be permitted as a sole measurement of BHR to assess suitability to dive because they do not exclude exercise-induced bronchoconstriction which is an absolute contraindication to diving.[14,35] Strenuous exercise is frequently required of the diver, and there are many opportunities for accidental aspiration of sea water to occur. Inhaling dry air from a tank or fine aerosols of hyperosmolar saline through a faulty valve are stimuli also encountered by the diver. Nowadays many laboratories have the facility to exercise patients and could easily include a dry air challenge during exercise or eucapnic voluntary hyperpnoea in their assessment. The techniques for these tests have also been standardised,[4,6-8] and there has been a big increase in the number of laboratories in Australia which have experience with the challenges using aerosols of non-isotonic solutions, and in particular 4.5% saline.

There is a growing interest in challenge with eucapnic voluntary hyperpnoea to replace exercise testing for EIB in the laboratory. This test lends itself particularly well to assessment for scuba diving. Most laboratories in Australia would not use the cold air protocol of Assoufi et al as used by Tetzlaff et al.[22,37] Rather the EVH tests would be carried out for 6 minutes with a target ventilation of 30 times the measured FEV_1.[6] The higher ventilation and the longer time (2 minutes) appear to compensate adequately for the reduction in temperature of the inspired air.[38] High ventilation rates are easily achieved during EVH. Both the ventilation rate achieved and sustained and the water content of inspired air are important determinants of EIB.[39] To exclude EIB for divers it is suggested that the ventilation achieved during testing is > 55 $l.min^{-1}$ because divers are often required to exercise at 2 $l.min^{-1}$ oxygen consumption. Thus, simple exercise by step testing breathing room air without the appropriate measurement of ventilation and dry inspired air condition would not be an adequate test to exclude EIB in an intending diver. It has been clearly shown that sports specific exercise is far more potent than laboratory based testing.[40] It is for this reason that EVH is now recommended for testing athletes.[38,41]

It is particularly important to note that dry air and hyperosmolar aerosols can provoke cough in people with a history of asthma. In our experience, if inhaled hyperosmolar aerosols of saline or hyperpnoea with dry air causes a reduction in FEV_1 or excessive coughing the intending diver is immediately aware of the potential for the same thing to occur whilst diving and usually voluntarily excludes himself from diving with scuba. This voluntary abstention is important and takes the pressure off the examining physician by members of the family or friends trying to encourage a person with a history of asthma to dive.

The airway responses to 4.5% saline have been documented in one laboratory in a group of 180 intending divers with a past history of asthma who had been cleared medically fit to dive.[42] A positive response consistent with currently active airway inflammation could be demonstrated in 30 or 17% of these applicants who were without current symptoms of asthma, who were taking no medications and

Table 1
Hypertonic (4.5%) saline challenge in 180 prospective divers with a past history of asthma but considered medically fit to dive, subject to BPT[42]

	Fall < 15%	Fall > 15%
n	150	30
% Fall FEV_1	4.5 ± 3.7	22.3 ± 6.5
FEV_1 % Pred	106.3 ± 14.0	100.3 ± 13.7
FVC % Pred	105.5 ± 11.0	105.1 ± 12.1
FEV_1/VC %	79.8 ± 8.4	76.1 ± 8.8
FEF_{25-75}% Pred	81.7 ± 23.5	69.6 ± 20.4

Table 2
Subjects wishing to dive with scuba who responded negatively to questions on asthma yet had a 15% fall in FEV_1 to hypertonic saline (PD_{15}) a value consistent with a diagnosis of currently active asthma

Question	Answer	PD_{15} to 4.5% Saline	
		n	%
1. How would you describe your asthma over the past 3 months?	None	21	10.0%
3. How often do you have symptoms from your asthma at present?	None for the past 3 months	22	10.3%
4. In the past 3 months, have you woken at night because of wheezing, chest tightness or cough?	No	43	20.2%
5. How many days in the past 2 weeks have you had a morning wheeze or chest tightness?	None	46	21.6%
6. Does exercise trigger your asthma?	No	37	17.4%
7. How often do you usually use your bronchodilator?	None for 3 months or more	29	13.6%
9. In the last 3 months, have you used your bronchodilator?	No	45	28.0%

without a recent history and who had no other medical reason to be prevented from diving (Table 1).

Since the report of these findings referrals by practitioners to the laboratory appear to have changed in that intending divers, with a history of asthma, are referred to the laboratory for assessment before, rather than after, they have their medical examination. This is both time saving to the doctor and potentially cost saving to the subject and provides important medical information to a significant number of applicants.

At one teaching hospital in Sydney an analysis was made of responses to a self-administered questionnaire in some 212 intending divers after they had each had a challenge test with 4.5% saline. They were aged 27.7 years and 44% were females. The group had a mean % predicted FEV_1 of 104% (95% CI 102.2 to 105.8). Fifty-four of the 212 recorded a 15% fall in FEV_1 in response to 4.5% saline. The mean provoking dose to cause a 15% fall in FEV_1 was 9.7 ml (7.2, 13.2) and the FEV_1% predicted in this group was 98.9% (95.8,102). Many with BHR to 4.5% saline gave negative answers to the questionnaire relating to asthma in the last three months and did not take medication (Table 2).

The lesson from this is that BHR can be common in the absence of symptoms. From the analysis of the questionnaire we found a number of questions that are useful and with each positive response the likelihood of BHR to 4.5% saline increases. These include: self-classification of asthma severity, self-admission of asthma triggers, particularly dust mite and cats, and use of bronchodilator more than once per month but less than once per two weeks.

In the last five years there has been an increasing interest in carrying out tests of BHR with hypertonic aerosols or eucapnic voluntary hyperpnoea in a number of special groups. These include recruits for the NSW Police Force[43] and young athletes being evaluated for exercise-induced bronchoconstriction.[44] Many of these subjects have marked BHR to eucapnic voluntary hyperpnoea (EVH), a surrogate challenge used to identify exercise-induced bronchoconstriction in the presence of normal values for FEV_1 and FVC.[2,43,44] The results of the findings in a group of young adults are given in Table 3.

In another study in recruits positive to EVH (% fall in FEV_1 of 25.2% +/- 11.0) the values for FEV_1% predicted were 98.8% +/- 13, with an FEV_1 / VC ratio of 79% +/- 8.6 and FEF_{25-75}% predicted 80.4% +/- 22.7, all within the normal range. In another group in the same study the lung function in those positive to hypertonic saline (PD_{15} 6.9 ml CI 4.9, 9.6% fall in FEV_1 21.4 +/- 4.2) was FEV_1% predicted 98.8% +/- 11.6, FEV_1/FVC 76.8 +/- 9.7, FEF_{25-75} 73.6% +/- 24.2).[43] Whilst BHR to hyperpnoea or 4.5% saline cannot be excluded by normal values for these indices those with

Table 3
Eucapnic voluntary hyperpnoea – + 20 to 25 °C for 6 min at VE 22 to 30 x FEV_1 96 athletes < 31 years referred for possible EIB (# P < 0.004, * P > 0.05)[44]

n =	31 +ve	65 -ve
FEV_1 % Pred	105 ± 16	109 ± 13#
FVC % Pred	111 ± 15	109 ± 15#
FEV_1/FVC	81 ± 8	86 ± 6*
FEF_{25-27} % Pred	85 ± 24	101 ± 18*
VE L/min	94 ± 24	97 ± 23#
% MVV (FEV_1*35)	69	69
% Fall FEV_1	23 ± 13	4 ± 3*
% Fall FEF_{25-75}	36 ± 18	12 ± 7*

abnormal values, particularly of FEF_{25-75}, are more likely to have BHR. The data in the recruits have demonstrated that with good adherence to a medication regimen, the BHR to EVH and 4.5% saline can be reduced to within the normal healthy range with eight to nine weeks' treatment with 800-1000 micrograms of budesonide daily. It is important to note that budesonide only came onto the market in Australia in 1992 and that there has been a significant improvement in asthma treatment since publication of the last discussion paper.

There is recent evidence that flow rates at low lung volumes are lower in divers and are inversely related to the years of diving.[45] This same author found that those with PBT also had low flow rates at low lung volumes[46] so that measurement of these flow rates may now be considered an important part of the assessment of lung function. A reduction in FEF_{25} or FEF_{25-75} of > 20% of the predicted values could contribute to risk of PBT. However, Russi has pointed out that no firm conclusions can be drawn between PBT and pre existing lung function or structural abnormality.[47] This emphasises the importance of paying attention to the flow rates at the lower lung volumes.

There is some evidence that pulmonary abnormalities are associated with increased risk of decompression illness and obstructive airway disease has been identified as an independent risk factor.[48] Also there is evidence of patchy airway closure in asthmatics demonstrated by single photon emission tomography after inhaling technegas.[49] Airway closure results in gas trapping (an increased residual volume) and increases risk of PBT. Hyperinflation, measured by an increase in thoracic gas volume, has also been demonstrated acutely in response to challenge with 4.5% saline with the volume increasing from 4.3 l (SD 0.6) to 5.3 l (SD 1.1) at the time the fall in FEV_1 was 18% (SD 23).[42] These data suggest that even small reductions in FEV_1 can lead to hyperinflation and gas trapping and a reduction of 15% in FEV_1 should not be considered just borderline.

If people with a past history of asthma, but without significant BHR to challenge by hyperpnoea or hypertonic aerosols and with normal lung function, are thought to have a relatively low risk of problems and permitted to dive, they should be instructed to be aware of change in their asthma status over time. Asthma exacerbations can occur particularly following viral infections and exposure to allergens to which the subject may be sensitised. Asthmatics cleared to dive should be informed that there is a risk that their asthma may become active again. They should also be informed to ensure that filtered air is used in filling their compressed air tanks as there are reports of allergen particle exposure to unfiltered air.

The British Thoracic Society Guidelines[48] suggest that measurements twice daily of peak flow should be made by asthmatics during the diving season variability may be useful but this index of asthma has limitations.[50] For example lack of variability does not exclude exercise-induced bronchoconstriction a feature that is accepted by the diving medical community as an absolute contraindication to diving. We suggest that asthmatics may require further referral to the laboratory for testing during their diving careers.

Guidelines and Recommendations for Diving from Other Countries

Some guideline and discussion papers have recently been published and the reader is referred to these for more information on recommendations[48] and data relating to asthmatics and diving incidents.[51] Recommendations have also been made by medical personnel involved in research in diving medicine.[52,53]

The British have tended to be more tolerant towards asthma, and the findings of Farrell & Glanville (1990) may explain this continued tolerance. Similarly, the analysis by Neuman et al[53] in the USA suggests that there is only a 1.6 fold increase risk for arterial gas embolism in asthmatic subjects increasing to 1.98 in those with a current history of asthma. Neither of these increases reached statistical significance. No similar data are available in Australia for this type of analysis, although there are data on the medical conditions of recreational divers who dive despite medical contraindications. Taylor found 2.6% of 346 recreational divers reporting current asthma and 22.8% reporting current allergies but other respiratory disease was rare with only one subject reporting chronic obstructive lung disease and two others reporting previous history of a pneumothorax.[54]

Morbidity and Mortality

Project Stickybeak is an ongoing investigation seeking to document all types and severities of diving related accidents (www.spums.org.au/diveinfo.htm). It reports 88 deaths in Australia between 1980-1990. There are some recent Canadian data on risk of decompression illness and death where the denominator used was air fills. The findings suggest that incidence of death is 0.002% of dives and for decompression illness 0.01% of dives.[55]

Conclusion

With respect to asthma the approach by most Respiratory Physicians is a conservative one, with scuba to be disallowed for anyone with a history of symptoms and medication for asthma within the last five years. It would now seem to be an appropriate time to re-evaluate this suggestion in the light of the improved medication regimens, the ease with which lung function and bronchial provocation tests can be performed in Australia and New Zealand, and the move towards the informed risk assessment model. Given that bronchial hyperresponsiveness to the very stimuli experienced by the diver (hyperpnoea of dry air during exercise and possible aspiration of sea water) occurs in a

significant number of people with a past history of asthma but no current symptoms and good lung function, measurement of BHR to these stimuli is strongly recommended. The results of these tests should be part of the risk assessment approach to diving where the subject needs to acknowledge that they have been informed of potential risks and hazards of diving with asthma.[56]

The Australia and New Zealand Standard

The Australian Standard for diving is still appropriate (AS/NZ 2299.1:1999) although it has been enlarged and the respiratory section is reproduced in the Appendix below. The conditions shall require a careful risk assessment and may be a consideration for disqualification.

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Additional sources of reference available on the Internet

There are now a number of diving websites that provide useful information. The website for the South Pacific Underwater Journal (http://www.spums.org.au), for example, provides the medical forms required for passing a recreational diver. Others from overseas include The British Sub Aqua Club (www.bsac.com) and Divers Alert Network (www.diversalertnetwork.org). For people who are excluded from scuba diving for their asthma we suggest a referral to the Asthma Australia website (www.asthmaaustralia.org.au) for education materials.

Appendix

The following Appendix is taken from AS/NZS 2299.1: 1999. The information given in this appendix is relevant only to examination of individuals considering recreational scuba diving. Document AS 2299-1992 should be referred to for criteria for medical examination of individuals intending to commence diving as an occupation.

K4.11 Respiratory System

The respiratory system should be examined as follows:

(a) Particular attention must be paid to any condition that might cause retention and trapping of expanding gas in any part of the lungs during decompression. This includes the following conditions which are considered to be contraindications to diving.
  (i) Any chronic lung disease, past or present.
  (ii) Any history of spontaneous pneumothorax, perforating chest injuries, or open chest surgery.
  (iii) Any evidence of obstructive airway disease for example asthma, chronic bronchitis, allergic bronchospasm.
  (iv) Any fibrotic lesion of the lung that may cause generalised or localised lack of compliance in lung tissue.
  (v) Any chest X-ray signs of pulmonary adhesions, tenting effects, emphysematous change, cysts, blebs or bullae.

A past history of asthma may be acceptable, but this and any other cases of doubt indicate the need for specialist opinion. Such opinion should include inhalational challenge testing if there is any doubt about the possibility of bronchial hyperreactivity.

(b) A large plate posterior-anterior chest X-ray should be performed at the initial examination. Chest X-ray should be performed subsequently as part of a fitness review following serious chest infection, and should be considered at intervals not exceeding five years for divers with extensive exposure to diving or any respiratory risk factor.

(c) Pulmonary function tests shall be conducted as follows, using equipment capable of reading to at least 7 litres:
  (i) At initial examination, all divers shall have pulmonary function tests to establish forced expiratory volume at one second (FEV_1) and forced vital capacity (FVC), recording the best of three measurements. This test should be repeated at intervals not exceeding five years.
  (ii) An FEV_1 or FVC of more than 20% below predicted values or FEV_1/FVC ratio of less than 75% may indicate increased risk of pulmonary barotrauma and is an indication for specialist assessment.

While five-yearly chest X-ray and pulmonary function tests are considered sufficiently frequent for healthy divers with no risk factors for pulmonary disease, more frequent screening is indicated for smokers, and divers who had abnormalities noted on previous examination.

If no other abnormality is present, a finding of fitness may be allowable if additional specialist pulmonary function tests and opinion do not find any fixed or intermittent outflow obstruction that might predispose to pulmonary barotrauma.

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Key words
Scuba diving, fitness to dive, respiratory, asthma, review article, medical society, reprinted from

Discussion Paper On Asthma And Diving published in *Thoracic Society News*, Vol 14 Issue 4 A Response By Dr Graham Simpson

The discussion document by Anderson et al makes some interesting points about changing opinions regarding asthma and scuba diving over the last eleven years. One point it fails to address is whether this is actually a real problem. Scuba diving is a remarkably safe sport - safer, for example, than rock fishing on the New South Wales coast (see www.safewaters.nsw.gov.au). Anderson et al quote an estimate of one death for every 50,000 dives. It is known from many sources that the vast majority of diving accidents relate to stupidity, poor training, equipment malfunction or usually some combination of these factors. A very careful analysis of Australian diving deaths implicated medical conditions in a very small fraction of fatal diving accidents. Of these the majority involved cardiac events in middle aged or elderly men.[1–3] There is no good evidence that the proportion of asthmatics in the scuba diving population is any different from that in the general population and at least some evidence that many clinical asthmatics continue to dive without difficulties. A recent study from New Zealand of over 20 years of diving deaths found only four cases in whom asthma was even suggested as a contributing cause without any evidence for this assertion being available in any case.[4]

Despite considerable effort no study has demonstrated that asthmatics are at a statistically significant increased risk of death or barotrauma when scuba diving. If there is an increased risk, then it is relatively small and in absolute terms minute.[5]

Why then are we so concerned? Unfortunately a lot of responsibility for this must be placed on the TSANZ 1993 position paper. This elegantly and lucidly describes the theoretical reasons why asthmatic bronchospasm may create localised air trapping and thus pulmonary barotrauma and it is largely as a consequence of this paper that the Australian diving medical standard, which is enshrined in law in Queensland, puts such stress on asthma and bronchial provocation testing.

The history of medicine is sadly littered with examples of detailed theoretical argument which, when belatedly put to the test, are shown to produce exactly the opposite result of that expected. Fluid loading for acute tubular necrosis, HRT to prevent ischaemic heart disease and routine post-operative radiotherapy for lung cancer are only a few examples. In fact the only pulmonary function abnormalities convincingly demonstrated to predict pulmonary barotrauma are a low vital capacity and decreased pulmonary compliance - in fact the opposite of those seen in obstructive lung disease.

Bronchial provocation testing

Australia has put a unique emphasis on the role of bronchial provocation testing in the exclusion of prospective scuba diving candidates. There has been an almost emotional attachment to the use of hypertonic saline, presumably because the sea is salty. Both the 1993 and current paper refer to aspiration of fine aerosols of hypertonic sea water through faulty regulators. It must be remembered that to perform a hypertonic saline challenge one needs an ultrasonic nebuliser as no jet nebuliser yet devised has a sufficiently high output. The idea that a regulator could become faulty underwater such that it produces an output of respirable particles of saline at about double that of the best purpose designed nebuliser one can buy is akin to suggesting that a dive torch could develop a short circuit and transform itself into a DVD player. Aspiration of sea water into the upper airway is also invoked but it seems unlikely that enough water could be aspirated deeply enough into the lungs to cause widespread bronchospasm by altering airway surface osmolality. In any event this sort of aspiration occurs far more commonly when swimming, snorkelling or surfing and as yet these activities are free from medical regulation. Anderson quotes a study of hypertonic saline challenge in intending divers with a past history of asthma where 17% were advised against diving because of a positive result.[6] In the same issue of the SPUMS Journal we reported a study in a group 50 experienced scuba divers with over 70,000 logged dives.[7] Strict application of the criteria for respiratory fitness suggested by the standard Australian diving text books and recently again supported by the current president of the SPUMS[9] would have resulted in 46% of this group being excluded from scuba diving training. Interestingly the exclusions did not include three of the five divers with currently active clinical asthma. Contrary to Anderson's suggestion, more of the divers failed the hypertonic saline challenge than the histamine challenge.

The problem with bronchial provocation testing is that bronchial smooth muscle is present for a reason and if sufficiently provoked will produce bronchospasm in anyone. The difficulty is in knowing how much provocation to apply. Eucapnic voluntary hyperpnoea (EVH) has become the accepted test for use in elite athletes who wish to use bronchodilators during competition. These individuals, however, do not have asthma in the sense which any respiratory physician would understand it and it is a considerable leap of the imagination to apply results from these super fit athletes to the average recreational scuba diver. Contrary to repeated statements in the discussion paper, scuba diving is essentially a very relaxed pastime and high levels of exertion are rarely required. If they are, it is usually at the surface where scuba divers are joined in their predicament by swimmers, snorkellers and surfers. To put things in perspective, the EVH test suggested by Anderson et al (six minutes of 85% of MVV) is not applicable to a scuba diver at 20 m of sea water depth, because that level of ventilation would empty the average scuba tank in less than six minutes. Even in elite athletes

the levels of ventilation achieved in the EVH are significantly higher than that normally achieved at maximal exercise. The British Thoracic Society guidelines have surely got this right with a suggestion that the exclusion criterion should be significant wheeze precipitated by moderate exercise. It is perhaps unfortunate that the debate on asthma and diving in Australia is being dominated by individuals with particular expertise and interests in bronchial provocation testing. It is of note that the patent for the use of mannitol in bronchial provocation testing, which has been suggested to replace EVH, is held by the Health Authority which employs the principal author of the Discussion Paper.

Accurately calculating the 'Number Needed to Test' by EVH or mannitol to prevent a single SCUBA diving accident is not possible as even active asthma has not been shown to increase risk but the number is likely to be many thousands. The converse of this is that thousands of people would be barred from a voluntary activity for no good reason. The informed consent approach is clearly the right one with regard to medical assessment for scuba diving. A simple explanation of the possible potential problems of a history of asthma and the true magnitude of the risk involved would however seem to make more sense than the mandatory use of expensive and time consuming tests.

Conflict of interest

The author has demonstrable exercise induced bronchospasm and chooses to continue to scuba dive.

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A Reply to the Letter from Dr Graham Simpson in Respect to the Discussion Paper on Diving (Published *Thoracic Society News*, Vol 14 Issue 4)

This Reply by S D Anderson is endorsed by co-authors R Wong, M Bennett & L Beckert

Dr Simpson has started the discussion on this paper and raises some important points about diving and asthma. First he draws our attention to the lack of statistics regarding problems with diving and asthmatics. Perhaps part of the explanation for this failure to find asthmatics amongst diving accidents is simply because they are not equally represented in the diving community. Although Dr Simpson makes reference to the fact that "the proportion of asthmatics in the scuba diving population" is no different from the general population this statement is not supported by the recent survey of Australians diving published by Taylor. {Taylor, 2002 #1585} Another factor is that diving is a sport with a high attrition rate and many people who register to dive do so only once. Thus there is likely to be some 'healthy worker' effect in play. We support the measurement approach because it informs the intending diver and is likely to reduce the risk that people with currently active asthma will be represented in the statistics.

Dr Simpson also makes reference to the fact that Australia "has put a unique emphasis on bronchial provocation." This is probably a result of the high prevalence of asthma in Australia, the high level of research interest in the disease, the ready opportunity to dive within Australia, the ease with which a person with asthma can be assessed in a laboratory at relatively low cost, and the increasing awareness of duty of care and informed consent. In other countries, such as the USA, pharmacological stimuli are more commonly used to assess bronchial hyper-responsiveness (BHR). Diving doctors in the USA have not been tempted to introduce tests with pharmacological agents for diving assessment as they are simply not specific for identifying asthma and healthy people can also respond to these agents. There is a high rate of BHR to pharmacological stimuli in divers and cross-country skiers and skaters probably as a result of airway injury from dry air breathing. As Dr Simpson quite correctly says in reference to pharmacological challenge, "if sufficiently provoked [it]

will produce bronchospasm in anyone". There is no suggestion to include pharmacological challenges to assess intending divers in the discussion paper.

The principal author's interest in diving came about as a result of referrals to the laboratory for testing following the publication of a paper in 1981 that reported that the airways of people with asthma were responsive to changes in osmolarity. {Schoeffel, 1981 #291} By the 1990's the numbers of referrals to the laboratory were significant and the findings for hypertonic challenge in 180 intending divers were published by SPUMS in 1995.{Anderson, 1995 #408} At this time Project Sticky Beak reported 88 fatalities between 1980-1990 in Australia and New Zealand so the community was rightly concerned about death and diving. The referrals for testing with hypertonic saline came from the local doctors assessing people for diving and the laboratory simply provided a service. "The emotional attachment to hypertonic saline" to which Dr Simpson refers is simply a reflection of the stimulus to which most divers are exposed. Should the diver be exposed to fresh water then water would be the osmotic stimulus used in the laboratory. We do not advertise the availability of testing for divers. The laboratory personnel performing the bronchial provocation tests have no role in the decision to or not to dive. Further the principal author on the paper does not serve on any committee that makes decisions standards and recommendations by SPUMS re bronchial provocation testing and diving. The authors of the text book and the review on diving referred to by Dr Simpson have presumably based their opinion on their experience of referring people for such testing and the published literature.

The referrals by the doctors seemed to be a logical extension of concept of duty of care to inform an 'asthmatic' of the risks associated with diving. The tests became more readily available across the country because they were more specific for identifying currently active asthma not because they could be used to identify potential for problems whilst diving with SCUBA. We thank Dr Simpson for drawing attention to the mannitol test that was not described or recommended in the paper as it is not available commercially. However the potential for this test to be used for assessing divers at the time of consultation in the future would be a logical extension of its application to assessment of asthma in general.

By performing an appropriate bronchial provocation test the potential diver with possible asthma becomes informed of the possibility of the same stimuli causing an attack of asthma. It has been our experience in the laboratory that people who have positive response to hypertonic saline are very willing to absent themselves from diving. It should be noted that a proportion of these people are quite relieved at having an excuse not to dive as relatives and partners enthusiastically try to get them to take up diving.

Dr Simpson is right to refer to the magnitude of the stimulus of hyperpnoea. As pointed out in the paper a ventilation of 55 $l.min^{-1}$ or more is likely to exclude someone who would suffer EIB when exercising at the surface with all their equipment suggesting that higher values may not be necessary for testing. However the higher the ventilation, in relation to possible maximum achievable, the more confident one can be of excluding EIB. The eucapnic voluntary hyperpnoea test and the target ventilation (30 times FEV_1) is recommended in response to criticism that exercise testing in the laboratory does not necessarily exclude the diagnosis of EIB in people with mild disease.

In respect to volume of hypertonic saline aerosol, airways can narrow after 30 seconds of exposure to an ultrasonic aerosol in a person with asthma. The estimated volume of airway surface liquid covering the first 10 generations of airways is less than 1 ml. This means that only a small amount of salt water (< 3 ml) needs to be aspirated to provoke airway narrowing in a person with even the mildest response to hypertonic saline (PD_{15} of 22.5 ml). The PD_{15} in those people with a past history of asthma was 9.7 ml and only 15% of this volume would have deposited in the lower airway, the rest remains in the valve and tubing or is swallowed. The aerosol does not have to penetrate 'deeply into the lungs' and only needs to reach the first few generations of airways to change the osmolarity and provoke a response in people with sensitive airways.

We have made some suggestions for assessing people with asthma or a past history of asthma knowing that this disease varies spontaneously over time and in response to treatment and that there is a wide variation in severity within the population. Testing provides an opportunity to have a level playing ground. Rather than be concerned about the 17% of people with a past history of asthma being considered at risk of diving Dr Simpson should pause to consider that 83% were cleared to dive.{Anderson, 1995 #408} An extension of this finding would be testing of asthmatics currently receiving treatment as BHR to dry air and hypertonic stimuli can be normalised in many asthmatics by treatment with inhaled steroids. Giving people whose asthma is controlled by treatment the 'go ahead' to dive is the opportunity that arises from measurement and informed consent of the risk.

The most striking thing about asthma in young people today, relative to 10 years ago, is the presence of BHR to dry air and hypertonic aerosols in those with normal spirometry. This was demonstrated in the data in our paper.{Anderson, 2004 #2077} In the absence of airflow limitation at rest the simple bronchodilator test for reversibility is not available to the practitioner. Further, it does not appear that any index of spirometry is a predictor of BHR to dry air or hypertonic saline. The knowledge that only a small volume of saline or a few minutes of dry air breathing can provoke airway narrowing in asthmatics with normal lung function is the driving force behind the recommendation to measure BHR to the stimuli to which the diver is exposed.

We would agree with Dr Simpson that informed consent "is clearly the right one with regard to medical assessment." Our conclusion remains that "these tests should be part of the risk assessment approach to diving where the subject needs to acknowledge that they have been informed of potential risks and hazards of diving with asthma." Our question to Dr Simpson is "What alternative approach would he propose to assess the suitability to dive of people with a history of asthma?"

Key words
Scuba diving, fitness to dive, respiratory, asthma, review article, medical society, reprinted from

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Editor's comments:

Readers are invited to contribute their thoughts on this subject to a letters forum in the next issue. Asthma will also be discussed at the June ASM, led by Robyn Walker. SPUMS will provide feedback to TSANZ from these two inputs, from which a joint concensus may be promulgated.

The diving doctor's diary

Tales from the South Pacific – diving medicine in Vanuatu

Richard Harris

Key words
Decompression illness, travel medicine, general interest

Although I borrowed my title from the famous book by James Michener, my story of two years as a doctor in the island nation of Vanuatu doesn't have quite the ring of romance and adventure as his classic tale. However, as an anaesthetist from Adelaide in South Australia, I have found that my time here has certainly turned out to be a great adventure for my family and me.

When the position of anaesthetist at Vila Central Hospital in Port Vila came up through AusAid (www.ausaid.gov.au), I was living the quiet life giving anaesthetics in private practice in Adelaide and working part-time in diving and hyperbaric medicine at the Royal Adelaide Hospital. A few months later I was working well outside my comfort zone in a developing country…a regular jack of all trades and a master of none.

Vanuatu relies heavily on the tourist industry for a large proportion of its GDP, and many of its visitors come here to scuba dive or otherwise enjoy the tropical waters. Several dive operators work the waters around Port Vila, catering for recreational scuba diving, which is strictly limited to 40-msw, single-tank, no-decompression dives on the numerous local reefs and wrecks (many of which are purpose-sunk artificial reefs).

In the northern island of Espiritu Santo, the famous shipwreck of the *SS President Coolidge* lies on a sand shelf from 20 msw at the bow to 70 msw at the stern. This site is unique in the world because the local diving code of conduct allows certified divers of any level to perform decompression dives using air to depths of 60 msw, as long as the dive operators assess them as competent to do so. This competence is established by working slowly down the wreck to increasing depths and penetration during the divers' stay.

Human nature and financial pressures being what they are, this means that occasionally divers will find themselves out of their depth in terms of their qualifications and experience. Despite the potential for accidents that this scenario might project, only three deaths have ever occurred on the wreck (none of them whilst diving under the direct supervision of a dive shop), in the many thousands of dives that are performed here every year. In addition, only one or two episodes of decompression illness (DCI) present for treatment each year arising from *Coolidge* dives.

A small but steadily growing technical diving contingent visits the wreck each year, but at this stage only one of the dive shops caters for this group. Opportunity for live-aboard diving is very limited in Vanuatu, with only one operator running trips intermittently at the time of writing.

Aside from the recreational dive industry, a small amount of commercial diving takes place in Vanuatu. Inshore commercial dive operators do work on ships' moorings, sea walls and the like, free-diving fishermen collect sea cucumber, and a significant aquarium-fish industry owned by an offshore company uses local and Philippino divers to catch fish by hand using scuba and hookah equipment. I have had no professional dealings with these groups during my time in Vanuatu.

Port Vila is lucky enough to have a twin-lock hyperbaric chamber owned by Hyperbaric Health Services, and run by

the Australian-trained paramedics at Promedical (a private ambulance company). The chamber was formerly at the Alfred Hospital in Melbourne and will be well known to some. It started its new life in Vanuatu based on Espiritu Santo, but for lack of medical and technical staff it was moved to Port Vila. It is well maintained and staffed, and I felt very comfortable using it for the administration of hyperbaric oxygen treatment (HBOT) to 2.8 ATA.

In the past two years I have treated only five divers in the chamber (with a total of 15 treatments). Two of these had clinically significant spinal sensory-motor DCI, and both made a good recovery with appropriate first aid, extended USN Treatment Table 6, intravenous lignocaine (in one case) and follow-up HBOT treatments (repetitive 18.60.30).[1]

As the chamber is not on the hospital grounds, the patient either stayed at a hotel between treatments or, in the case of the one diver given lignocaine, was admitted to the private medical clinic in Port Vila for monitoring. The majority of divers held DAN insurance and insurance-related delays were minimal, significantly reducing stress on the part of all involved. A further two divers were assessed as having mild, resolving decompression sickness (DCS) and were not treated, and three other divers were assessed as not suffering from DCS.

As a physician in diving medicine in Vanuatu, I was consulted by telephone on numerous occasions by local dive shops with fitness-to-dive queries. I was reluctant to see all the divers in question due to the voluntary nature of my diving medical work; however, I was happy to provide a telephone advice service. Naturally, without seeing the prospective divers myself the advice I gave was extremely conservative in nature. I did, however, see a number of patients with injuries secondary to diving or swimming including ENT problems, marine envenomations and bites, fish poisoning and salt-water aspiration/near drowning.

Some cases of note that come to mind? There were several cases of ciguatera and scromboid poisoning. The red bass (*Lutjanus bohar*) is a prime culprit in Vanuatu. Two shark attacks occurred during my time, one fatal and one nearly so, two stingray envenomations, a moray eel bite and a variety of jellyfish stings. On a personal note, I had brushes with the magnificent sea urchin (*Asthenosoma ijimai*) and the devil stinger (*Inimicus caledonicus*). Both very painful and best avoided! Multiple coral cuts, producing ulcers despite the best of care, leave me with a nice collection of scars on my legs as a permanent reminder of my stay.

Sadly I leave the beautiful shores of Vanuatu in January 2006, returning to Australia to resume work as an anaesthetist. But I know I now have the taste for adventure, and I am already scheming about the next trip away!

Reference

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Richard Harris, BM, BS, FANZCA, DipDHM, at the time of writing was working as an anaesthetist and in diving medicine at Vila Central Hospital, PMB 9013, Vanuatu.

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The salvage of the *Wahine*: an exercise in occupational medicine

Anthony G Slark

Key words
Occupational diving, occupational health, decompression sickness, computers, safety, history

Abstract

(Slark AG. The salvage of the *Wahine*: an exercise in occupational medicine. *Diving and Hyperbaric Medicine*. 2006; 36: 24-7.)
In 1968, the inter-island ferry *Wahine* sank in the entrance to Wellington Harbour in a wild storm with the loss of 51 lives. Following a further storm, attempts to refloat the vessel were abandoned. This report, written over 30 years ago by Tony Slark as the diving medical consultant to the salvage operation, describes some of the medical aspects involved in the cutting up and clearing of the wreck between 1968 and 1973. It includes two detailed case descriptions of decompression sickness. From April 1970, Dr Slark introduced the SOS decompression meter to control all diving operations, with over 10,800 hours of diving time being completed without any further incidents of decompression sickness.

Introduction

The inter-island ferry *Wahine* sank in Wellington Harbour in a wild storm in April 1968 with [the] loss of 51 lives. It was initially hoped that the salvage of the vessel could be performed by refloating the whole hull with foam and compressed air. However, a further storm in May 1969 shifted the vessel breaking it up to such a degree that the original concept of salvage had to be abandoned in favour of raising the individual broken sections. However, further deterioration of the hull occurred and eventually the system of salvage necessary has been the piecemeal cutting of the hulk into sections capable of being lifted from the bottom by the support vessel *Holm Park* anchored beside the wreck. This has required the formation of a team of professional divers working constantly on the wreck to cut it into manageable sections.

During the initial period when the wreck remained in one piece lying on the sea-bed, the diving work consisted primarily of the removal of vehicles and cargo, and the cutting down of the superstructure. Twelve divers were employed for a period of approximately 3,000 hours underwater. [During] this time there was no organised system of decompression, but no bends resulted. It can be assumed that much of the diving took place at depths less than necessary for the production of a sufficient degree of nitrogen supersaturation to make decompression sickness possible. The average working depth was in the region of 40–50 feet of sea water (fsw).

When work was begun after the wreck had broken up, the maximum working depth increased to 70 fsw. Again, no organised system of decompression was used. During this time two divers suffered attacks of decompression sickness. Four divers only constituted the team during this time, which extended from October 1969 to March 1970, and they put in about 1,400 hours of work underwater.

Case report one

On 2 March 1970, the senior diver of the team, a man aged 52 years, a professional of many years' experience working underwater, surfaced about 4 pm after a total of 4 hours at 60 fsw. He did not perform any decompression time. The American Naval Tables would have suggested a total ascent time of some 82 minutes and the Royal Naval Tables [a] total decompression time of 90 minutes. Even with such a decompression time the tables are reckoned to have a possible 10% bends rate for such prolonged exposures.

The diver first noticed a pain in his left biceps, forearm and wrist with some pain in his left groin. The pain started some three hours after surfacing and he felt it became really unbearable in the early hours of the morning. He recognised that he was suffering from decompression sickness having had similar bouts before, and arrangements were made by the Salvage Master for him to be flown direct to Auckland first thing in the morning so that recompression could be offered him if necessary. He came by ordinary commercial flight which meant that he suffered further decompression stress during the flight for the cabin is only pressurised to an equivalent level of 5,000 ft. However, he did not remark upon any exacerbation of his symptoms.

On arrival he was able to walk into the consulting room and give a good account of the circumstances. He described his pain as being more severe than he had felt on any previous occasion, but did not admit to any neurological symptoms. His bladder function was normal. General medical examination revealed no abnormal signs, apart from brisk tendon reflexes throughout, and bilateral upgoing plantar responses. Since he described one major previous bend as having a considerable neurological content this finding was ascribed to the earlier incident, for which incidentally he had not received treatment with recompression.

He was asked whether he felt able to put up with the pain while a further medical examination took place – he said he would rather not. I would have wished to have done a chest X-ray, electrocardiogram and full blood screening, but he was quite blunt in his lack of enthusiasm for any delay. Since analgesia would have confused the decision-making process whilst under recompression treatment, I decided to recompress him forthwith. He was put under pressure at approximately 10.30 that morning and we tried initially the 'minimal-pressure oxygen recompression' schedule,[1] which involved initial compression to a depth of 60 fsw on pure oxygen for 20 minutes with breaks in air after each 20 minutes for 5 minutes. This latter is designed to prevent oxygen poisoning. Towards the end of the first oxygen period it became obvious that he was receiving little benefit, and it was therefore decided to proceed with the longer air table. He was therefore compressed further to a level of 165 fsw on Table 5B of the Royal Naval Diving Manual.[2] After a short spell at this depth he noted a very great relief in the pain and following this, decompression on the schedule proceeded uneventfully. He left the chamber at 10.30 that evening complaining only of a very slight tenderness of the left upper arm and with virtually no other abnormalities apart from the minor neurological signs noted previously. He was kept under observation at the Naval Hospital for the following day, during which time routine medical examinations were performed, including chest X-ray and electrocardiogram and blood screening. Apart from an ESR of 40 [sec] and a haemoglobin of 13 [g.100ml^{-1}] there were no abnormalities detected. He did not have a platelet examination or blood lipid screening as would now be my practice.

It was noted that he had a marked limp. And examination of his legs revealed shortening of the left leg of approximately three quarters of an inch. There were no symptoms relevant to this and he had what seemed a fairly full range of painless movement. It was decided to perform a full X-ray screening of him for the possibility of aseptic [bone] necrosis. This was confirmed by the X-rays, which showed a very widespread involvement with virtual complete destruction of his left hip and widespread necrosis throughout the long bones and many infarcts involving joint surfaces in other parts.

Because of this he was advised most strongly that in future his diving should be confined to supervision. It is interesting that his pattern of diving i.e., that of doing long periods at relatively shallow depths was similar to the pressure changes experienced by tunnel and caisson workers in whom aseptic [bone] necrosis is a far more common finding. He was, however, the sort of older worker who always wishes to show the younger generation that he can do more and do it better. He did not in fact follow my advice and continued his diving for a further fairly extensive period. The Salvage Master said he had had the limp for 15 years. He would often actively refuse to undergo decompression and cut it short if he could. Many of the old school divers regarded bends as some lack of courage. This attitude, of course, influenced the younger divers.[3] I understand, however, that his arthritis has still not caused him much pain though he has now retired from diving completely.

Case report two

The following week another diver of the team, a man aged 25 years, was sent to us with similar symptoms. He had been diving at a depth of 65 feet for 3.5 hours without decompression. This exposure according to the Royal Naval Tables should have required a total decompression time of 115 minutes, and on the US Naval Tables for exceptional exposures a time of ascent of 179 minutes. He stated that 15 minutes after surfacing severe pain had begun in his left shoulder, a less severe pain in the left elbow and some slight numbness of the fingers of his left hand. There were no other significant signs on examination and the central nervous system seemed quite normal in all respects. He was accordingly recompressed again. Initially an attempt was made to treat him on the minimal-pressure oxygen table but with no relief. Once more we recompressed further on the much longer deep air table and he stated fairly soon after being at 165 ft [sw pressure]. that he had considerable relief of his symptoms. Decompression according to Table 5B continued uneventfully and he was removed from the chamber the next day complaining only of some slight ache in the left shoulder. Further examination was performed at the Naval Hospital including electrocardiogram, blood investigation and a full series of X-ray examinations. None of these revealed any significant abnormalities and he was therefore returned to Wellington and has since continued to dive, and is now their second in charge.

Management of diving operations from April 1970

It was obvious that with two cases occurring in such a short space of time, both illustrating a complete absence of proper precautions for decompression following prolonged time underwater, some review of the safety precautions offered the workers on the salvage operation was required.

It was obvious that the senior diver's long personal apparent immunity to decompression sickness had allowed a rather casual approach to develop in the team, and I think that it was very much a matter of familiarity breeds contempt. The Salvage Master himself had faith in his chief diver with whom he had worked for many years, and had had no reason hitherto to concern himself much with the detail of the safety aspects of the diving side to the work. He also wished to avoid undermining his authority which he thought was precariously maintained. However, the complete disruption of the operation of a small team which was occurring because of the neglect of the standard of diving patterns could only result in a great deal of further trouble. There was therefore a considerable tightening up of all safety procedures, and all further diving in the next period between March and [April 1970] was worked strictly under the United States Navy Standard Air-Decompression Tables with surface interval credit tables and repetitive dive time-tables.

Each diver was provided with a depth gauge and complete records of dive times and depths were recorded in a log book. This system prevailed for a month during which time 278 hours of time were recorded.

This system of diving in salvage circumstances has grave disadvantages. Firstly on a salvage operation it is extremely difficult for any diver to perform one long dive at one consistent depth. He must move around varying his depth and position, he may have to return to the surface for different articles of equipment, and therefore may involve himself in a pattern of repetitive dives which complicate his decompression schedule to a considerable degree. Maximum depths and maximum times are routinely used for the calculation of decompression schedules as they should be and very often from a commercial point of view the divers have 'run out' of diving time before the end of the working day. Furthermore calculations with repetitive dive sheets, three different diving tables, the calculations of surface interval credits, even the simple difficulty of pencil and paper work and minor arithmetic on a wet and windy diving platform are very prone to errors. It seemed to me that this would inevitably lead to a further crop of "bends" as well as having disadvantages from the commercial point of view to the operators. I therefore recommended

1 the use of decompression meters for each individual diver suggesting that, together with their use, log books be maintained as accurately as possible
2 that repetitive dives be kept to the absolute minimum and of the briefest duration possible
3 that as far as possible, the decompression meters be used to indicate when the dive should be completed without the need for decompression stops.

From the institution of this pattern of diving operation in April 1970 until the end of July of 1973 something over 10,800 hours of diving time had been completed without any incident involving decompression sickness. One diver was killed by an underwater explosion on 25 October 1972, but apart from this accident the pattern of operations has been one of very considerable safety which bears very good comparison with any other similar pressure work. For instance in most pressure works involving caisson or tunnels an incidence of 2% bends rate is considered quite acceptable, and in many instances the number of cases has been considerably greater.

I would not, however, wish to recommend the uncritical use of decompression meters for commercial diving, nor indeed for any other sort of diving. They are but one method of monitoring a dive pattern and should in general be used with a full knowledge of other systems in addition. Certain points regarding the decompression meter have to be borne in mind.

1 It seems to be rather safer for shallower dives. The tables are safer for deeper dives. The crossover safety point seems to be in the region of about 90 fsw.
2 The meter is definitely less safe than tables when surface intervals occur over a period of longer than 6 hours.
3 Short, deep repetitive dives on the meter are likely to be dangerous.
4 There is a proven instrument variation.
5 The supposed 6 hour 'memory period' of the decompression meter is much less than the real excess nitrogen elimination period of the body – which is probably greater than 24 hours.

Decompression meters should only be used with a full understanding of their limitations. Nevertheless, the value of a simple instrument eliminating the need for calculation on a wet and windy surface with pencil and paper give it practical advantages which may in many cases outweigh its potential disadvantages. This is of particular significance in salvage work where in most cases the diving is likely to take place in depths less than 100 fsw.

I paid a visit to *MV Holmpark* myself in September and October of 1971 with a view to instructing the diving team in various safety procedures including the safe use of the decompression meter. I examined the site of work underwater and made continuing arrangements for the safety and medical supervision of the diving team, in addition to the provisions required by the Department of Labour code of practice for underwater operations. All divers were subsequently issued with a plastic card indicating vehicle management if decompression sickness might be suspected, and a list of telephone numbers relevant for assistance. A designated Medical Officer was appointed to conduct all routine pre-employment examinations and regular assessments on continuing fitness for diving. A high standard of fitness and safety has been maintained subsequently, apart from the one accident already mentioned. In addition even though a great deal of time has been spent by the divers in water possibly suffering from considerable contamination, they have been remarkably free of otitis externa.

Discussion

There are several points of interest in these two cases and the subsequent safety procedures adopted after their occurrence. Both cases were simple bends, classified currently as Type 1, without neurological or other involvement. Recently it has been our unfortunate lot to have to deal with a large number of very much more seriously injured divers, though in almost all cases these have been the result of much deeper dives for much briefer periods. Although both these divers exceeded the safe diving times for surfacing without decompression by a very great degree they did not receive the severe neurological involvement that has often occurred with other divers, even though in both cases there was a delay in treatment and an air evacuation involving some considerable additional decompression stress. Neither case responded adequately to the minimal-pressure oxygen recompression tables, and [both] required the older high-pressure air tables for their

adequate treatment. I personally ascribe this to the delay in the institution of treatment, and although I have in all cases tried to use the shorter tables initially, I have only found them of benefit when instituted very rapidly after the onset of symptoms. I think this displays some inadequacy in the assessment of the tables when they were originally introduced. It is obvious that, in testing therapeutic tables, patients cannot be subjected to a delay in treatment. I think it relevant that the diving pattern and pressure-time changes involved were similar to those for caisson and tunnel workers and that this case of aseptic necrosis should have occurred in a diver performing such work. Finally although during the past years the decompression meter has come in for a great deal of criticism by professional and amateur divers alike, this safety programme, when the instrument was used with a fairly full knowledge of its limitations, shows how valuable it can be. The salvage firm is intending to use the same system for the diving on the salvage operation on the *Seawise University* in Hong Kong.

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Dr Anthony George Slark, MB, BS (Lond), DPH, DIH, DObst, FAPHM, MRCGP, MFOM, deceased, at the time of writing this report, was the Senior Medical Officer to the RNZN Hospital HMNZS Philomel, Auckland, and Diving Medical Advisor to the Department of Labour.

This verbatim report is published posthumously with the kind permission of Eileen Slark and her family.

Automatic decompression meters

Carl Edmonds

Once again we hear of divers needing treatment for decompression sickness which occurred following routine decompression in accordance with an automatic decompression meter. There have been three such cases treated at the School of Underwater Medicine this year, and the records show many others occurring over the last few years since their general acceptance by the public as safe alternatives to the "tables".

It never ceases to amaze me how divers place such blind faith in mechanical gadgetry! It seems that one can write almost anything in a diving magazine, and there will be gullible divers eager to accept every word as "gospel". Such has been the sales spiel on these DCMs (see *Skin Diver Magazine* Nov. 1967 and Nov. 1970).

The DCMs in common use today make no allowances for individual variability in physiology, and strict adherence to the meter's decompression schedule is bound to result in some cases of decompression sickness (DS). Similarly there is no allowance made for this factor with recognised RN or USN decompression tables – however, the records here are evident. Providing the table is followed exactly, the rate of development of DS in divers is never greater than 2–3%. I'm sure the record of divers on the DCMs is nowhere near as good – certainly not in my experience.

For some time, we have been asked – especially by ex-patients treated for DS after following the DCM schedules – to evaluate these meters and publicise the results. At long last we have managed to obtain 12 such meters (10 secondhand and two brand new and never exposed to pressure/water) and have started evaluation testing. This has been conducted on a basis compatible with practical diving to depths varying in 20 ft increments from 60 ft to 200 ft. The results are far from being completed; however, several significant features are already outstanding. These are inconsistencies which are evident when the DCMs are tested in a 'wet pot' and show

- that the decompression schedules recommended by individual DCMs for identical dive (depth/time) factors vary considerably,
- that the decompression schedules recommended by the same DCM for identical dives vary considerably – and this followed a much longer than normal non-dive period, and
- that the decompression schedules recommended by the DCMs in some cases were more conservative (time wise) than corresponding RN or USN tables; and yet in others were far outside the limits of staging according to the tables.

These features are apparent on single ("bounce") dives – repetitive dive testing has only just commenced, and results are unknown as yet. The fact that variables such as movement of the DCM (tapping, vibration, etc.) sunlight (warmth, etc.) are known to markedly affect the non-dive recovery period of the DCM, is sure to create interesting variations when these tests are finalised.

In the meantime, it would appear that our best advice to divers concerning these DCMs is to never rely on them for any dive in excess of 120 ft or for any repetitive dives, and to follow the most conservative regime when the DCM is compared to a recognised decompression table, (i.e.) dive with both table and meter, and decompress according to the deepest first stop and longest decompression times.

Certainly these techniques will make diving more complex for "fools" – but anyone who dives to depths in excess of 100 ft and thinks all is rosy when following a DCM is a fool. Deep diving in a hostile environment requires careful planning and thoughtful techniques, and no mechanical mechanism exists which can always reliably predict decompression schedules for divers at various depths for variable periods. Surely, it is safer to err conservatively and stick to the "deepest depth X longest time" method. There are many ex-patients who can recommend this practice from personal experience with DCMs which failed.

The full results of the tests on the DCMs will be printed in the SPUMS newsletter when completed.

This article is reprinted from Edmonds C. Automatic decompression meters. *SPUMS J*. 1973; 3: 9.

Editor's comment:

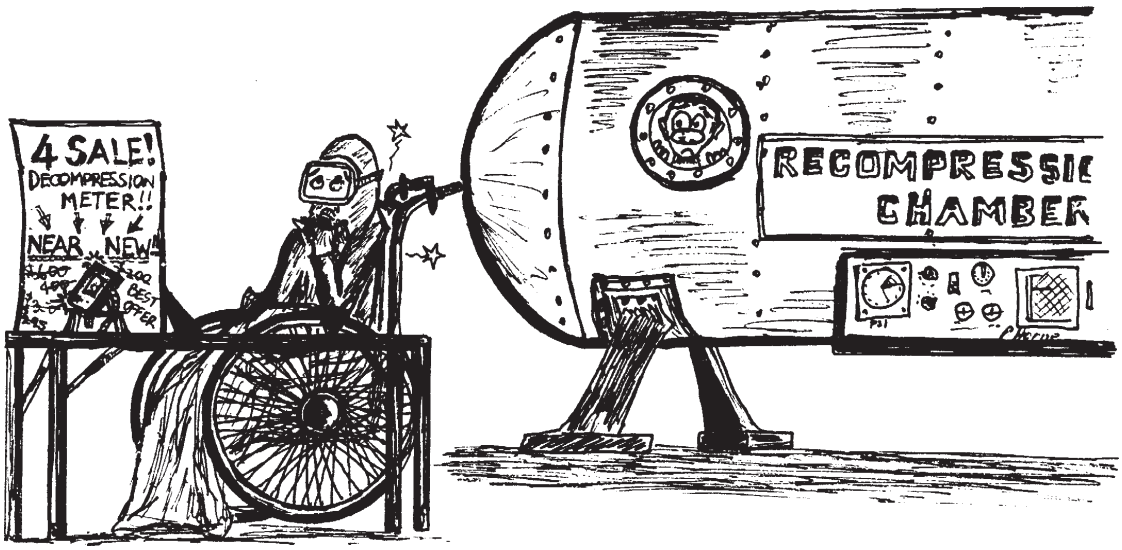
Eileen Slark kindly donated all of her husband's diving medical teaching slides, papers, case records and books to the Occupational Medicine Unit, Department of Medicine, The University of Auckland. The report above was found amongst these papers, whilst Carl Edmonds' brief contemporaneous article on decompression meters was published over 30 years ago in this Journal. The SOS meter was often referred to in those days as the 'Bendomatic'!

I felt the juxtaposition of the two articles would be interesting historically, and that it highlights the common disparity between theory and practice in diving. Edmonds' research, particularly his detailed later work on the Orca EDGE™ computer, showed the unreliability of the early generations of dive computers (and made him no friends in the diving industry!). Nevertheless, divers got on with the job, utilising these tools, inadaquate as they were, with seeming success. Tony's report demonstrates why he was held in high regard in New Zealand by his peers, and by military, occupational and recreational divers alike.

Mike Davis

The SOS decompression meter

Cartoon by Peter Harrigan reproduced from the cover of the *SPUMS J.* 1987; 17 (3)



Articles reprinted from other journals

Diabetes and recreational diving: guidelines for the future

Workshop proceedings, 19 June, 2005

Neal W Pollock, Donna M Uguccioni and Guy de Lisle Dear

Guidelines for diabetes and recreational diving

A workshop addressing issues of diabetes and recreational diving was jointly sponsored by the Undersea and Hyperbaric Medical Society (UHMS) and Divers Alert Network (DAN) to bring together experts and interested parties from within and beyond the international diving community. The meeting was held on June 19, 2005 in Las Vegas, Nevada, USA, following the UHMS annual scientific meeting. The objectives of the workshop were to review the existing data and, as warranted by participant support, to develop consensus guidelines to address diabetes and recreational diving. More than 50 individuals from seven nations, mostly clinicians and researchers, participated in the discussions.

Limitations: 1) The discussion was restricted to recreational diving. The issues concerning professional diving require future, separate deliberations. 2) This is a set of guidelines, not rules. The participants agreed that appropriate and justifiable differences in acceptable procedures may exist and that interest groups must have the flexibility to use the guidelines as they best serve their community's needs.

The guidelines were divided into three sections: 1) selection and surveillance of people with diabetes in scuba diving; 2) scope of diving by people with diabetes; and 3) glucose management on the day of diving. Individual divers must bear responsibility for their health and safety and for adherence to established guidelines developed to improve their protection and that of their dive partners. Divers with diabetes are encouraged to participate in relevant research studies to expand the data available concerning diving with diabetes. Anyone with questions should consult with physicians knowledgeable in both diving medicine and diabetes care.

Section 1. Selection and surveillance

Those evaluating persons with diabetes for medical fitness to dive must first ensure that no other exclusionary conditions (e.g., epilepsy, pulmonary disease) exist. The physiological demands of diving must then be considered. Coronary artery disease is a leading cause of death in the largely non-diabetic diving population. Immersion may result in increased myocardial wall stress. There may also be a reduced awareness of ischaemic symptoms. People with diabetes are at higher risk of medical complications such as myocardial infarction, angina and hypoglycaemia than the general diving population. Such risks are exacerbated by the fact that many dive sites are quite isolated from medical aid. While only some medications increase the risk of hypoglycaemia, all persons with diabetes are at risk of secondary complications of the disease.

Recreational scuba diving may be undertaken by candidates otherwise qualified to dive who use medication (oral hypoglycaemic agents [OHAs] or insulin) to treat diabetes provided the following criteria are met.

1.1. Age 18 years and over (limit may be lowered to 16 years if special training* is available)
*special training will include dive training programs designed specifically to meet the education needs of individuals with diabetes and, desirably, to include participation by parents and/or responsible family members or guardians.

1.2. For a new diver at least three months have passed since the initiation or alteration of treatment* with OHAs or one year since the initiation of treatment with insulin. An established diver using OHAs who is started on insulin should wait at least six months before resuming diving.
*"alteration of treatment" is defined as a change in medication(s) or dosage(s) that could result in significant deviations from current status (changes likely to include only moderate change from current status would be described as "adjustment of treatment").

1.3. There should have been no episodes of hypoglycaemia or hyperglycaemia requiring intervention from a third party for at least one year, and no history of hypoglycaemia unawareness. Note: certain OHAs (e.g., metformin, acarbose), when used on their own, do not predispose to hypoglycaemia.

1.4. Glycosylated haemoglobin (HbA_{1c} - a measure of plasma glucose stability over the past two to three months) should be <= 9% when measured no more than one month prior to initial assessment and at each annual review. If $HbA_{1c} > 9\%$ the diver should contact his/her diabetes specialist for further evaluation and modification of therapy.

1.5. There should be no: retinopathy worse than nonproliferative; significant autonomic or peripheral neuropathy; nephropathy causing proteinuria; coronary artery disease or significant peripheral vascular disease. Patients with retinopathy, peripheral vascular disease and/or neuropathy have a higher risk of sudden death due to coronary artery disease. Retinal haemorrhage could be

precipitated by small changes in mask pressure during descent and ascent or equalizing manoeuvers. Patients with neuropathy may experience exaggerated hypotension when exiting the water. Peripheral vascular disease may alter inert gas washout and predispose an individual to limb decompression sickness.

1.6. No more than two months prior to the first diving medical assessment and at each annual evaluation, a review is conducted by the candidate's primary care physician (knowledgeable in treating diabetes) who must confirm that: criteria 1.3 – 1.6 are fulfilled; the candidate demonstrates accurate use of a personal blood glucose monitoring device; and that the candidate has a good understanding of the relationship between diet, exercise, stress, temperature, and blood glucose levels.

1.7. No more than two months prior to commencing diving for the first time and at each annual review, a diving medical examination is completed, preferably by (or in consultation with) a doctor who has completed an accredited post-graduate diving medical examiner's course*. The review report completed by the primary care physician must be available. It is strongly recommended that formal evaluation for silent ischaemia be undertaken for candidates over 40 years in accordance with US American Heart Association/ American College of Cardiology or equivalent guidelines. *any accredited course (one certified as fulfilling certain standards by a national and/or regional professional association) in diving medicine is acceptable

1.8. At the diving medical examination, the candidate acknowledges in writing the receipt of and intention to use the diabetic diving protocol; the need to seek further guidance if there is any material that is incompletely understood; and the need to cease diving and seek review if there are any adverse events associated with diving suspected to be related to diabetes.

1.9. Steps 1.1 – 1.8 must be completed annually, using the same physicians where possible. After the initial evaluation, periodic surveillance for silent ischaemia can be in accordance with accepted guidelines for evaluation of diabetics.

Section 2. Scope of diving

Persons with diabetes selected according to Section 1 of this document who satisfactorily complete a recognized diver training course are considered suitable for recreational diving. The following stipulations and strong recommendations regarding diving activity and methods apply.

2.1. It is recommended that dives do not involve depths greater than 30 meters of sea water (100 fsw), durations longer than one hour, compulsory decompression stops, or take place in overhead environments. The depth limit is to avoid situations in which narcosis could be confused with hypoglycaemia. The time limit is to moderate the time blood glucose would remain unmonitored. The decompression and overhead environment limits are to avoid situations in which direct and immediate access to the surface is not available.

2.2. Divers with diabetes should dive with a buddy/leader who is informed of their condition and is aware of the appropriate response in the event of a hypoglycaemic episode. It is recommended the buddy does not have diabetes.

2.3. It is recommended that divers with diabetes avoid combinations of circumstances that might be provocative for hypoglycaemic episodes such as prolonged cold and arduous dives.

Section 3. Glucose management on the day of diving

Divers with diabetes who are selected according to Section 1 of this document, and who participate in appropriate diving activity as specified in Section 2, should use a protocol to manage their health on the day of diving. Note: the blood glucose monitoring protocols are applicable to people with diabetes whose medication may put them at risk of hypoglycaemia.

3.1. For every day on which diving is contemplated, the diver should assess him or herself in a general sense. If he or she is uncomfortable, unduly anxious, unwell in any way (including seasickness), or blood glucose control is not in its normal stable pattern, DIVING SHOULD NOT BE UNDERTAKEN.

3.2. The suggested goal for the diabetic approaching any dive is to establish a blood glucose level of at least 150 $mg.dl^{-1}$ (8.3 $mmol.l^{-1}$), and to ensure that this level is either stable or rising before entering the water. The workshop recommends that this be determined by three measurements of blood glucose, ideally taken 60 minutes, 30 minutes and immediately prior to diving. Diving should be postponed if blood glucose is < 150 $mg.dl^{-1}$ (8.3 $mmol.l^{-1}$), or there is a fall between any two measurements.

a. Where relevant, strategic and individually tailored reductions in dosages of OHA medication or insulin on the evening prior or on the day of diving may assist in meeting these goals. Initial testing of individual protocols should be conducted under very controlled circumstances.
b. Where relevant, a regimen of incremental glucose intake to correct inappropriate pre-dive levels or trends may assist in meeting these goals.

3.3. It is recommended that diving should be postponed or cancelled if blood glucose levels are higher than 300 $mg.dl^{-1}$ (16.7 $mmol.l^{-1}$).

3.4. Divers with diabetes should carry oral glucose in a readily accessible and ingestible form at the surface and

during all dives. It is strongly recommended that parenteral glucagon is available at the surface. The dive buddy or other person at the surface should be knowledgeable in the use of glucagon. If symptoms or indications of hypoglycaemia are noticed underwater, the diver should surface, establish positive buoyancy, ingest glucose and leave the water. An informed buddy should be in a position to assist throughout this process. Use of an "L" signal with the thumb and index finger of either hand is recommended as a signal for suspected hypoglycaemia.

3.5. Blood glucose levels should be checked at the end of every dive. Appropriate response to the measured level can be determined by the individual mindful of his or her plans for the rest of the day. It should be noted that the requirements for blood glucose status outlined in 3.2 remain the same for any subsequent dive. In view of the recognized potential for late decrements in blood glucose levels following diving it is strongly recommended that the level is checked frequently for 12-15 hours after diving.

3.6. Divers with diabetes are strongly recommended to pay particular attention to adequate hydration on days of diving. Elevated blood glucose will lead to increased diuresis. While the data are limited, there is some evidence from divers with diabetes that an increase in haematocrit observed post-dive (suggesting dehydration) can be avoided by deliberate ingestion of fluid.

3.7. Divers with diabetes should log all dives, associated diabetic interventions and results of all blood glucose level tests conducted in association with diving. This log can be used to refine future planning in relation to diving.

Executive summary

Historically, the diving medicine community has maintained a conservative position and concluded that insulin-requiring diabetes mellitus (IRDM) should be an absolute contraindication for participation in scuba diving. Dissent for this view has grown over the last 20 years. Recognizing that a substantial number of divers are diving successfully with diabetes – either openly or surreptitiously – has led many to believe that it is time to acknowledge this fact and reexamine the position concerning diabetes and diving.

This diabetes and diving workshop was jointly sponsored by the Undersea and Hyperbaric Medical Society (UHMS) and the Divers Alert Network (DAN) to bring together experts and interested parties from within and beyond the international diving community. Co-organizers were Dr Guy Dear, Dr Neal Pollock and Ms Donna Uguccioni. The meeting was held on June 19, 2005 in Las Vegas, Nevada, USA, following the UHMS annual scientific meeting. The objectives of the workshop were to review the existing data and, if deemed appropriate by discussants, to produce consensus guidelines addressing diabetes and recreational diving. More than 50 individuals from seven nations, mostly clinicians and researchers, participated in the discussions. The list of participants and their affiliations are found at the end of the proceedings document.

Nine invited speakers described data and experience gathered from around the world. Dr Guy Dear (USA) provided the opening remarks. Mr Steve Prosterman (USVI) provided an invaluable description of his personal experience both with diabetes and with diabetes and diving. Dr Eugenio Cersosimo (USA) presented an overview of the current state of the art in clinical management of diabetes mellitus. Dr Chris Edge (UK) reviewed 14 years of data, totaling approximately 14,000 dives, from United Kingdom divers diving with diabetes. Dr Dan Lorber (USA) represented the American Diabetes Association and presented an overview of discrimination and legal advocacy issues pertinent to persons with diabetes. The final paper appearing in this document was edited and approved by the ADA advocacy group. Ms Donna Uguccioni (USA) reviewed 12 years of data gathered through DAN-affiliated efforts, including surveys, workshops and observational studies. Dr Duke Scott (USA), the medical director for the YMCA SCUBA program, described the American YMCA program that has been used to train persons with diabetes to dive for the past 10 years. Dr Alexis Tabah (France) shared research data from two field studies on divers with diabetes conducted in France and reviewed the recently developed national regulations allowing recreational diving by persons with diabetes. Dr Warren Silberman (USA) described the US Federal Aviation Administration's nine-year-old policy allowing special issuance of medical certificates to individuals with diabetes for third-class (noncommercial) aviation licenses. Dr Simon Mitchell (NZ) closed the presentation portion of the meeting by delivering a draft list of guidelines for diving with diabetes developed from the published literature.

The edited transcript of the workshop reveals the depth of discussions and controversy surrounding each of the guidelines presented below. Some points were easily settled and others more contentious, but all were finally decided through compromise and consensus. The general level of agreement for each point is indicated in this summary.

The workshop participants agreed that the available data supported the position that at least some individuals with diabetes might reasonably be allowed to dive. There was no open dissent on this fundamental issue. The discussion focused on the specifics of who and how.

Two important issues were raised at the start of the discussion. The first concerned the scope of the deliberations. It was agreed that the discussion was to be

limited to recreational diving. The issues concerning professional diving require future, separate deliberations. The second issue concerned the nature of the product that would be produced by the group effort. It was agreed that a set of guidelines, not rules, would be generated. The participants agreed that appropriate and justifiable differences in acceptable procedures may exist and that interest groups must have the flexibility to use the guidelines as they best serve their community's needs.

The draft list delivered by Dr Mitchell served as a "straw man" to guide the discussion. The consensus guidelines, like the draft form, were grouped under three sections: selection and surveillance, scope of diving, and glucose management on the day of diving.

The **selection and surveillance** section began with general text indicating the importance of screening for other exclusionary conditions (e.g., epilepsy, pulmonary disease) and careful consideration of the context in which diving might be conducted. This includes immersion, the potential for extremely remote diving locations, and the high normal risk of cardiac involvement in diving fatalities. The section then addressed limits on age (18 years or older with the possibility of lowering to 16 years with special training), frequency of medical evaluation (at least annually), minimum periods of time from point of initiation or alteration of treatment to start or return to diving (three months from initiation or alteration of treatment with oral hypoglycaemic agents and one year since the initiation of treatment with insulin), allowable history of hypoglycaemic or hyperglycaemic events requiring third-party intervention (none within past year), hypoglycaemia unawareness (no history allowed), recent glycosylated haemoglobin (HbA_{1c}) scores (further evaluation and possibly modification of therapy recommended for values > 9 percent), and secondary complications (none can be significant). The section also addressed the importance of having candidates demonstrate a good understanding of diet, exercise, stress, temperature and blood glucose levels and the need for silent ischaemia screening. Finally, the section addressed the need to have candidates agree to follow diabetic diving protocols and to stop diving and seek review for any adverse events that may be related to diabetes.

Several aspects pertaining to the minimum age for training were discussed. The merits of involving family members in training and in providing positive reinforcements to persons with diabetes were recognized. The need to be consistent with applicable public rules was also discussed.

The selection of appropriate minimum durations between initiation or alteration of treatment was contentious. Discussants favored a variety of intervals. The final wording reflected the more conservative position.

The importance of disqualification based on recent history of extreme hypoglycaemia, hyperglycaemia or hypoglycaemia awareness were widely accepted. Similarly, the importance of a solid understanding of the disease, personal responsibility and a willingness/ability to conduct appropriate self-monitoring were all widely accepted. The option to recommend disqualification based on a history of emergency visits to hospital for any condition related to the diabetes was discussed and rejected.

The necessity for an HbA_{1c} criterion was contentious. Some felt that it was not an appropriate criterion; others preferred a range of high and low cutoff values. Key considerations included recognition that the tightest control might be associated with a greater frequency of hypoglycaemic events and the utility of the measure for counseling purposes. The final wording reflected a relatively inclusive limit.

The discussion of secondary complications of diabetes reflected the importance of monitoring and protecting the long term health of persons with diabetes. The relatively high frequency of cardiac involvement in diving incidents and the potential for accelerated development of coronary artery disease in persons with diabetes was addressed with a strong recommendation for silent ischaemia screening for candidates over 40 years of age. The guideline text regarding secondary complications and silent ischaemia screening was kept general in recognition of the limitations of available research data and potential regional/national differences in screening and evaluation standards. This section is expected to evolve as additional data become available.

The value of annual medical evaluation and the importance of the diver taking personal responsibility in managing his or her disease were generally accepted. There was discussion regarding the appropriate recommendations for physician training. While the abilities of fully trained diabetologists and diving medical officers were appreciated, practical limitations on the availability of specialty-trained physicians were also recognized. It was decided that accepting physicians knowledgeable in treating diabetes and physicians who had completed any post-graduate course in diving medicine was appropriate at this time.

The **scope of diving** section addressed limits on dive depth (100 fsw [30 msw]), decompression obligation and overhead environments, dive time (< 60 min), the need to inform dive partners of their condition and the appropriate response to adverse events, the diabetic status of the buddy diver (recommended to not have diabetes), and recommendations on avoiding situations that may promote or exacerbate hypoglycaemic events.

The discussants widely agreed that divers with diabetes should avoid situations which restrict direct access to the surface (notably dives with obligatory decompression or in overhead environments), those that could create conditions potentially confused with hypoglycaemic symptoms (specifically nitrogen narcosis), and those expected to

increase the likelihood of hypoglycaemic events (e.g., prolonged cold and arduous dives).

There was more debate regarding maximum dive times. Positions favored a variety of recommended maximums and the discretion of the individual. The final wording on the dive time reflects a compromise between the extreme views. Options to include guidelines on a maximum number of dives to be carried out in a given day and/or a minimum surface interval between dives were discussed and rejected. The discussants widely agreed that it is important for divers with diabetes to inform their dive partners of their condition. There was more debate regarding the propriety of two divers with diabetes diving together. A comment

Table 1: Guidelines for recreational diving with diabetes - summary form[1]

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)

[1] 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.

was made that two divers with diabetes may be diving in a larger group. The final wording of the recommendation favored a conservative position.

The **glucose management on the day of diving** section began by noting that the blood glucose monitoring protocols are applicable to people with diabetes whose medication may put them at risk of hypoglycaemia. The section then addressed the importance of self-assessment to ensure readiness to dive, as recommended for all divers, and several procedures specific to diabetes management. Advance preparation included individually tailored pre-dive modification of oral hypoglycaemic agents or insulin and carbohydrate ingestion, attention to hydration, and critical health self-assessment prior to diving. Measurement standards included minimum pre-dive blood glucose levels (150 $mg.dl^{-1}$ [8.3 $mmol.l^{-1}$]), repeated pre-dive blood glucose measures to ensure a stable or rising trend (nominal planned monitoring at approximately 60 min, 30 min and immediately pre-dive), maximum pre-dive blood glucose levels (300 $mg.dl^{-1}$ [16.7 $mmol.l^{-1}$]), and post-dive blood glucose testing (repeated throughout a 12-15 hour post-dive period). Intervention supplies to have available included ingestible oral glucose and parenteral glucagon. Record keeping addressed logging dives, blood glucose test results and diabetic interventions associated with diving.

The discussants widely agreed on the importance of most elements in this section: a self-assessment of health preceding diving; both divers and buddies carrying a readily accessible and ingestible form of oral glucose; divers surfacing before ingesting glucose if needed; the availability of parenteral glucagon at the surface; the strategic tailoring of medication regimens in conjunction with diving (worked out in advance of diving or under highly controlled circumstances); serial pre- and post-dive blood glucose checks; incremental correction of sub-optimal blood glucose levels; adequate hydration; and the logging of dives and all information pertinent to diabetes management.

Controversy surrounded the reasonable frequency of pre-dive blood glucose measures, the need to specify pre-dive blood glucose ranges, the optimal and acceptable ranges of pre-dive blood glucose, and the appropriate duration of post-dive blood glucose monitoring. Arguments for recommending minimal obligatory monitoring and greater freedom for the diver were largely based on the record of relatively trouble-free diving by minimally monitored persons with diabetes registered in the United Kingdom. Arguments for greater obligatory monitoring and tighter controls favor the potential for the guidelines to be more useful to persons, both divers with diabetes and medical professionals, who may have less experience with diabetes management and/or diving. The final wording of the recommendations reflected the conservative position of requiring repeated blood glucose tests and definitive minimum and maximum values.

The draft text of the guidelines was completed by the end of the workshop. The draft text was refined after the meeting by the workshop planners. The refined text and an edited transcript of the discussion were then distributed to participants electronically. Each was invited to provide comment. Changes were circulated to stimulate further electronic discussion. The guidelines provided at the beginning of this proceedings document represent the final text produced after integration of all input received. An abbreviated version of the final guidelines is in Table 1.

The participants in this workshop viewed the guidelines as a work in progress. We fully expect further refinements or even substantial modifications as our understanding of the issues involved in diving with diabetes evolves. It is important that any individual who has questions should consult with physicians knowledgeable in both diving medicine and diabetes care.

Future progress will be facilitated by efforts in two directions. The first is continued support and promotion of initiatives to collect data relevant to diabetes and diving. The second is development of programs and relationships to educate individuals with diabetes who are diving or interested in diving and those who might be professionally involved with divers with diabetes. The latter group includes certifying agencies, dive professionals, medical monitors addressing qualification issues and emergent needs, and the general diving public.

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Workshop jointly sponsored by the Undersea and Hyperbaric Medical Society and Divers Alert Network

Reprinted with kind permission from Pollock NW, Uguccioni DM, Dear G de L, editors. *Diabetes and recreational diving: guidelines for the future*. Proceedings of the Undersea and Hyperbaric Medical Society/Divers Alert Network 2005 June 19 Workshop. Durham, NC: Divers Alert Network; 2005. p.1-9.
ISBN 978-0-09673066-9-8

Key words
Scuba diving, diabetes, blood sugar level, fitness to dive, DAN, meeting, reprinted from

Hidden dangers

Contaminated water diving: the risks divers don't want to acknowledge

Steven M Barsky

Many commercial divers are unaware of the dangers of diving in polluted water. Some bodies of water don't appear polluted, yet have high levels of biological or chemical contamination. In other environments, divers sometimes mistakenly believe that the water itself will dilute the hazard to a low level. In both settings, divers can be dead wrong.

The same hazardous materials that concern haz-mat personnel topside should concern divers underwater. These hazards include biohazards, toxic chemicals, and radiation. However, the situations that divers encounter these hazards in underwater are very different from those on the surface.

The main difference in dealing with a hazardous material underwater is that in many cases, the hazardous material floats in the water around the diver. This means that unless the diver equips himself properly, the material may enter the diver's mouth through the regulator in his helmet. It may also get in his eyes through leaks in a band-mask hood or a helmet's neck dam, and touch his skin through his wet suit. Compare this to a liquid spill topside, where the chemical puddles in the street, and it's easy to appreciate the increased risks.

Toxics that float on top of the water, such as gasoline, also present a serious hazard to the diver. The diver must pass through them to dive or exit the water. Substances that sink in water are those most likely to collect as pockets of pure chemical substance on the bottom. Concentrated chemicals are obviously very hazardous.

Aside from the risks, the biggest problem in contaminated water diving is that many divers don't want to acknowledge the dangers present at the sites where they dive. Conceding that these risks are present means that they must be dealt with intelligently and many divers don't want to make the effort or spend the money to take the proper precautions. Over the short or long term, such disregard can have fatal effects.

Biological pollutants are the most common

Biological pollutants are probably the most common form of hazardous materials encountered by divers. Three main classes of biological contaminants are of concern to divers. Bacteria are single celled creatures that exhibit characteristics common to both plants and animals. Protozoans are single celled animals. Viruses are organisms that take over the chemistry of a host cell in living creatures to reproduce themselves.

Faecal coliforms are a disease-producing bacteria found in human and animal faeces. They are universally present in the water wherever there is raw sewage, or inadequate sewage treatment. The maximum safe level of this organism is considered to be 200 organisms per 100 milliliters of water. Swallowing water that contains faecal coliforms can produce severe, disabling diarrhoea. It might not kill you, but it will result in lost time from work.

Whenever heavy rains fill storm drains and cause waste treatment plants to exceed their capacity, millions of gallons of raw sewage spill into nearby waterways, sometimes closing beaches for several weeks. Such occurrences are common throughout the United States and other countries.

A commercial diver who dives in a waterway that contains high numbers of faecal coliforms should be equipped with the right equipment. Ordinary commercial diving gear is usually not enough. In the United States, even many public safety divers (fire, sheriff, police) now wear vulcanized rubber dry suits and full-face masks or diving helmets for protection from biological contaminants.

If faecal coliforms are present, it's a safe bet there are probably several other forms of biological pollution as well. Other dangerous bacteria include cholera, *Vibrio vulnificus* and *Aeromonas hydrophilla*. Cholera is a good example of a bacteria that can survive in sea water.

Vibrio vulnificus is an extremely potent marine bacteria that can also cause death. It enters the body through the mouth or raw wounds. *Aeromonas hydrophilla* also infects open cuts in the body and is commonly found in harbour waters. *Aeromonas* infections have been fatal if not properly treated.

Like bacteria, many protozoans pose serious threats to divers. For example, eight different species of *Acanthamoeba* occur in polluted waters. This deadly single celled organism causes inflammation of the spinal cord, with death as the end result. *Giardia lamblia*, another protozoan, causes intestinal pain, diarrhoea, and high fever.

Today, there are almost no streams in even remote areas that do not contain *Giardia*. This comes as a result of the high number of campers and backpackers using these areas. It is unsafe to drink water from any lake or stream in these areas unless it is treated. It is equally unsafe to swim or dive in these waters and accidentally swallow any liquid.

One of the most commonly known viruses, Hepatitis type A survives outside the body in both fresh and salt water. In Hepatitis A, the subject's liver will become inflamed. Like other disease producing organisms that spread through contact with raw sewage, hepatitis can be found in faecal matter.

Chemical hazards

When divers think of hazardous materials emergencies, they often think about accidental spills of toxic chemicals. However, in many situations, divers also face serious threats from low level, long term pollution of waterways. Less obvious threats lurk in the form of pesticides and fertilizers that have drained into irrigation ditches or even water traps on golf courses.

A chemical hazard commonly found in all harbours and marinas is the variety of residues from boat bottom paints that have been used over the years. These bottom paints were designed to kill or inhibit the growth of marine life. They have been used on both large and small vessels. The same chemicals that discourage marine growth are hazardous to humans.

One of the primary components of these anti-fouling paints is an organotin compound known as tributyltin, more commonly known as "TBT". There are 20 TBT compounds; 9 are used in boat bottom paints.

TBTs dissolve into fats, giving them the ability to move across the membranes of living cells. This trait is what makes them effective in killing marine organisms, such as barnacles. TBT tends to collect in the silt found on the bottom of harbours.

Almost all the research that has been done on TBTs has concentrated on the effect of these chemicals on marine creatures. However, in a report by the Brookhaven National Laboratory in the US, they note that chemicals in this class have toxic effects on the human central nervous system, blood, liver, kidneys, heart, and skin.

More alarmingly, the scientists noted that while people react to a single acute dose of TBTs, repeated sub-toxic doses also produce negative reactions. This suggests a cumulative effect, where low doses keep adding up in a diver's body after repeated exposures.

Since many commercial dives take place in harbours, TBTs should be of concern to divers and dive supervisors, especially divers working on ship's hulls. A scientist for the US Environmental Protection Agency (EPA) has labelled TBT as the "*most toxic chemical ever deliberately added to the marine environment*". In 1988, the EPA banned the use of TBTs on non-aluminum vessels under 82 feet in US waters. Tributyltin use is restricted in some countries.

Although tributyltin breaks down in clear waters, it persists much longer in murky harbour waters. The by-products of TBT's decay are also harmful. It may be years after TBT is banned worldwide before it no longer can be detected in the marine environment.

PCBs (Polychlorinated biphenyls) also pose serious potential threats to divers. Although PCBs are now banned in many countries, they were widely used in electrical and hydraulic equipment, paints, plastics, and other compounds. PCBs still continue to pollute many sites and numerous divers have been exposed to PCBs.

Divers who work around wooden piers and wharves should also beware of the dangers of creosote. Many wooden pilings are treated with creosote to prevent wood decay. Creosote also discourages marine worms from boring holes in the pilings. Unprotected divers can get chemical burns from brushing against pilings that are coated with creosote.

Certain chemicals are so dangerous that no diver should consider working around them. These chemicals include, but are not limited to, the following:

- Acetic anhydride
- Acrylonitrile
- Carbon tetrachloride
- Chlordane
- Cresol
- Dichloropropane
- Epichlorohydrin
- Ethylbenzene
- Methyl chloride
- Methyl parathion
- Perchloroethylene
- Styrene
- Trichloroethylene
- Xylene

Blood, urine, and stool samples are recommended pre and post dive when divers expose themselves to specific known chemical toxins. In addition, tests of the divers' lung capacity are merited in cases where chemicals are known to affect the divers' breathing ability.

It's only in the last few years that the risks of diving in polluted water have been scientifically correlated with cancer in divers. Dr Elihu Richter, head of the unit of Occupational and Environmental Medicine at Hebrew University School of Public Health and Community Medicine in Jerusalem, was the principal author of a paper which detailed the chemical exposure of 682 Israeli Navy divers working in the Kishon River since 1948. The Kishon River is highly polluted with heavy metals and other contaminants.

Richter and his team found a much higher level of cancer in these divers than in other control populations. Exactly what caused the cancer in so many Israeli Navy divers is unknown, but there was a strong correlation between diving in the Kishon and cancer that cannot be explained by other causes. In the United States we have anecdotal reports of cancer among dive team members in San Diego and Michigan, but there have been no studies undertaken to establish a scientific cause and effect relationship between diving and disease in most parts of the world.

By far, the largest source of pollution in most places is what is termed 'non-point source pollution.' This is a combination of everything that washes into our rivers, streams, and oceans from all 'normal' sources, including tire dust from cars, leaking oil and gasoline, faecal matter from household pets, pesticides and fertilizers used in agriculture as well as personal gardens.

Radioactive hazards

Radioactive substances are most likely to enter the marine environment through industrial accidents. However, the possibility also exists that someday terrorists may dump radioactive material into a drinking water supply or a harbour. Of course, some divers work inside nuclear plants, but they are usually well protected due to stringent monitoring in these environments.

Thermal hazards exist too

Aside from the risks of exposure to hazardous materials, dry suits and helmets also create thermal hazards for the diver. These hazards are exactly the same as haz-mat personnel face topside. They include fluid loss, heat cramps, and heat exhaustion.

During the time the diver dresses in before the dive, and during decontamination, heat stress can be a severe problem. If the diver works in cold water, some of the heat stress will be relieved during the dive. Moving from very warm surface climates into cold water, and back to hot surface temperatures, is stressful in itself.

If the diver works in warm water there is no relief from heat stress. Overheating may be a very real danger. Commercial divers who work in warm waters should carefully evaluate these conditions and plan dives accordingly.

In extended contaminated water diving operations in warm weather the diver's physiology should be monitored. These include heart rate, body temperature, and weight. Measurements of these functions should be taken before and after diving.

Experiments have been conducted at the National Institute of Occupational Health, in Sweden, on the effectiveness of diver cooling using an ice filled vest. The divers in the study wore dry suits similar to those used for contaminated water diving.

Underneath the dry suit they wore a vest fitted with 46 small pockets, each of which was filled with a block of ice in a plastic bag. At water temperatures of 107 degrees F, the divers were able to complete dives that were 15-30 minutes longer when equipped with the ice filled vest. Further tests will need to be performed to determine safe exposure times for using such systems.

Selecting the right equipment for contaminated water diving

One of the basic tenets of contaminated water diving is to never dive unless you know exactly what pollutants are present. In reality, we know that many people do not take the time to find out what risks are present in the water. In certain circumstances, this could be fatal.

In order to protect yourself as fully as possible, the ideal combination of equipment is a vulcanized rubber dry suit with a mating helmet and dry gloves. Keep in mind that even with this gear, there is no one set of equipment that will protect you from all types of chemical hazards. There's also no gear that will protect you from strong sources of radiation.

Free-flow helmets are generally considered very good protection from contaminated water because a positive pressure is maintained inside the helmet. However, demand helmets can also be used successfully, provided the breathing system is equipped with a redundant exhaust system to help prevent a back-flow of contaminants in the breathing system.

The interface between the diving helmet and the dry suit is extremely critical. Ideally, the helmet should mate directly to the suit, quickly and easily. Yet, the connection must be positive and secure. The system should be designed so that few, if any, contaminants are trapped between the helmet and the suit when the two are separated after the dive.

Dry suits for contaminated water diving should be made from a material that has a smooth, non-porous outer surface. The material must not absorb or trap contaminants. For diving in biologically polluted water, vulcanized rubber dry suits are usually considered the best choice.

Dry glove systems consist of a set of cuff rings as well as the gloves (or mittens). The cuff rings come in pairs of inner and outer rings. The inner ring is machined from hard plastic. It goes inside the sleeve of the dry suit where the sleeve attaches to the wrist seal. The outer ring is made from rubber. It slips over the sleeve and compresses the suit over the inner ring. The dry gloves or mittens snap into position over the outer ring.

If you are planning a dive in a chemical environment, it is essential to know the chemical compatibility of your equipment compared to the substances you will encounter. Some manufacturers have produced chemical compatibility tables that will give you the acceptable exposure time, in minutes, for their equipment. 'Permeation time,' which is the time it takes for a particular chemical to make its way through a piece of gear at the molecular level, is an essential issue for you to evaluate.

Evaluating tables like these is the only way to make an intelligent decision whether the risks on a particular dive are acceptable or not. Less scrupulous manufacturers have published results for their products that rate chemical compatibility as 'good' or 'acceptable.' Information like this is NOT adequate to plan a dive in a chemically contaminated environment. (Note: Chemical compatibility tables are available from some manufacturers as well as in the book, *Diving in High-Risk Environments*.)

Keep in mind that your exposure time is limited by the 'weakest' piece of equipment you plan to use. Since helmets and suits are made from many different types of materials, you must evaluate your entire diving ensemble, including suit, regulator diaphragm, exhaust valve, dry suit zipper, umbilical, etc. Making the wrong decision could cost you your life.

It's also important to remember that the chemical tests conducted by all testing agencies are always conducted on new, unused equipment. Diving equipment that has been previously exposed to other chemicals may fail unexpectedly.

Get the right training

Training for contaminated water diving operations is a complex process. There is no single expert on this topic. Instead, it takes the combined talents of many different people to put together a strong training program. Ideally, the staff for a training course in contaminated water diving would include a biologist, a chemist, a haz-mat specialist, and a commercial diver.

The critical points in the hands-on training for this type of diving include properly dressing and leak testing the diver's gear, and learning the correct procedures for decontamination following the dive. Since tenders may also need to be protected from fumes or chemicals encountered while tending the diver, they will need to be trained in the proper use of personal protective equipment topside.

Acknowledge the risks

Diving always involves risks and can never be made 100% safe. However, you increase your risks when you refuse to recognize that certain types of dives entail additional risks beyond what's considered "normal." Take the time to educate yourself and develop a healthy scepticism so that you'll be properly prepared the next time someone asks you to dive in a high-risk environment.

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Key words
Occupational diving, environment, toxins, infectious diseases, irradiation, operations, diving, reprinted from

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Cochrane corner

A systematic review of the use of hyperbaric oxygen therapy in the treatment of acute traumatic brain injury

Michael H Bennett, Barbara E Trytko and Benjamin Jonker

Key words
Cochrane library, brain injury, hyperbaric oxygen, reprinted from

Abstract
(Bennett MH, Trytko BE, Jonker B. A systematic review of the use of hyperbaric oxygen therapy in the treatment of acute traumatic brain injury. The Cochrane Database of Systematic Reviews 2004, Issue 4, Art. No. CD004609.)
Introduction: We aimed to assess the randomised clinical evidence for the benefits and harms of adjunctive hyperbaric oxygen therapy (HBOT) for acutely brain-injured patients. HBOT can improve oxygen supply to the injured brain and reduce both cerebral oedema and cerebrospinal fluid pressure and might therefore result in a reduction in patient death and disability.
Methods: We performed a systematic search of the literature for randomised controlled trials and made pooled analyses of pre-determined clinical outcomes where possible using Cochrane Collaboration methodology. We included adults with serious closed head injury requiring admission to an intensive care environment and included trials must have compared a standard therapy with adjunctive HBOT to standard therapy alone following randomised allocation. We pre-determined important clinical outcomes and assessed them when reported in the primary studies.
Results: Four trials contributed to this review (382 participants, 199 receiving HBOT and 183 control). Pooled analysis suggested a significant reduction in the risk of dying when HBOT was added (RR 0.69, 95% CI 0.54 to 0.88, NNT = 7, P = 0.003), but no statistically significant increase in the chance of a favourable clinical outcome (RR 1.94, 95% CI 0.92 to 4.08, P = 0.08).
Conclusions: HBOT reduced the risk of death but did not clearly increase the chance of favourable clinical outcome. Routine application of HBOT to these patients should not be justified from this review. More research of high methodological rigour is needed in order to confirm or refute the findings of this review.

Introduction

Traumatic brain injury (TBI) is a significant cause of premature death and disability. There are at least 10 million new head injuries worldwide annually and these account for a high proportion of deaths in young adults.[1,2] In the US, 2% of the population (5.3 million citizens) are living with disability as a result of TBI[1] and this places considerable medical, social and financial burden on both families and health systems.[3] Any intervention that may improve the chance of a good functional outcome is therefore worthy of study.

Hyperbaric oxygen therapy (HBOT) is one such intervention. HBOT is the administration of 100% oxygen at environmental pressures greater than 1 atmosphere absolute (ATA), an absolute pressure of 101.3 kPa. This involves placing the patient in an airtight vessel and increasing the pressure within that vessel while administering 100% oxygen for respiration. In this way, it is possible to deliver a greatly increased partial pressure of oxygen to the tissues. At 2 ATA (202.6 kPa) for example, patients with reasonable cardiopulmonary function will have an arterial oxygen tension of over 1000 mmHg, and a muscle oxygen tension around 221 mmHg.[4,5] Administration of HBOT is therefore based on the potential for reversing tissue hypoxia and modifying secondary neurological effects.

Following primary injury, there is ongoing injury to the brain through a variety of mechanisms including hypoxia, reduced cerebral blood flow (ischaemia), release of toxic levels of excitatory neurotransmitters, impaired calcium homeostasis and elevated levels of cytokines (secondary injury).[6,7] In addition oxygen extraction is increased in the hours following injury.[8]

Hypoxic neurons performing anaerobic metabolism result in acidosis, unsustainable reduction in cellular metabolic reserve,[9] loss of the ability to maintain ionic homeostasis, free oxygen radical accumulation, degradation of cell membranes and eventual secondary cell death.[10,11] When hypoxia is severe enough, these changes occur rapidly, but there is some evidence that these effects can sometimes occur over a period of days.[12]

A therapy able to increase oxygen availability in the early period following TBI may therefore improve long-term outcome. HBOT is also thought to reduce tissue oedema by an osmotic effect,[13] and any agent that has a positive effect on brain swelling following trauma might also contribute

to improved outcomes. On the other hand, oxygen in high doses is potentially toxic to normally perfused tissue, and the brain is particularly at risk.[14] HBOT may therefore do more harm than good in some patients.

Since the 1960s, there have been scattered reports that HBOT improves the outcome following brain trauma.[15] HBOT has been shown to reduce intracranial pressure (ICP) in brain-injured patients,[16,17] improve grey matter metabolic activity on SPECT scan,[18] and improve glucose metabolism.[19] Some studies suggest that any effect of HBOT may not be uniform across all brain-injured patients. For example, Hayakawa demonstrated that CSFP rebounded to higher levels following HBOT than at pre-treatment estimation in some patients, while others showed persistent reductions.[17] It is possible that HBOT has a positive effect in a sub-group of patients with moderate injury, but not in those with extensive cerebral injury. Furthermore, repeated exposure to hyperbaric oxygen may be required to attain consistent changes.[20]

Clinical reports have attributed a wide range of improvements to HBOT including cognitive and motor skills, improved attention span and increased verbalisation.[16,18] These improvements are, however, difficult to ascribe to any single treatment modality because HBOT was most often applied in conjunction with intensive supportive and rehabilitative therapies.

The purpose of this review is to assess the randomised clinical evidence for the benefit or harm of adjunctive HBOT in the treatment of acute TBI. This paper is based on a Cochrane review first published in The Cochrane Library 2004, Issue 4. Chichester, UK: John Wiley & Sons, Ltd (www.thecochranelibrary.com). Copywrite Cochrane Library, reproduced with permission. Cochrane reviews are regularly updated as new evidence emerges and in response to comments and criticisms. The Cochrane Library should be consulted for the most recent version of the review.

Methods

It was our intention to identify and review all randomised controlled trials (RCTs) concerning the treatment with HBOT of any patient with TBI in the first days following injury. We included all trials using hyperbaric oxygen administered in a compression chamber above 1.5 ATA (152 kPa) and for treatment times between 30 and 120 minutes on at least one occasion. For the comparator therapy, we accepted any standard treatment regimen designed to maximise brain protection and promote recovery from TBI. We did not include studies where comparator interventions were not undertaken in a specialised acute care setting.

Specific search strategies were developed to identify eligible reports from database inception to August 2004 in MEDLINE, EMBASE, the Cochrane Central Register of Controlled Trials (CENTRAL) and the Database of Randomised Controlled Trials in Hyperbaric Medicine (DORCTIHM). The latter is a specifically targeted database of clinical evidence in the field (<http://www.hboevidence.com>).

Medical subject headings (MeSH) and main key words used were 'hyperbaric oxygenation', 'head injuries, closed', 'head injuries, penetrating', 'craniocerebral trauma' and 'coma- post head injury', with variants of the main key words and free text terms also applied. No restrictions to language were made. Relevant hyperbaric textbooks, journals and conference proceedings were hand searched. Experts in the field were contacted for published, unpublished and ongoing RCTs. Additional trials were identified from the citations within obtained papers.

We pre-determined the following clinically important outcomes for assessment, and all included studies must have reported at least one of these: functional outcome measures (e.g. Glasgow Outcome Scale, GOS), death, activities of daily living (ADL) or quality of life (QALY) measures. In

Table 1
Summary of Jadad score from[21]
(each criteria scores or deducts one point if satisfied, giving a quality score from zero to five)

Criteria	Description
Randomisation	**The study is described as randomised, including using words such as 'random', 'randomised' or 'randomly'**
Additional	The method of randomisation is described and appropriate (e.g. use of random number table)
Deduction	The method of randomisation is described and is inappropriate (e.g. use birth date)
Double blinding	**The study is described as double-blind**
Additional	The method of double-blinding is described and appropriate (e.g. use of placebo or sham therapy)
Deduction	The method of double-blinding is described and is inappropriate (e.g. use observably different placebo)
Description of withdrawals	**There is a description of any dropouts or withdrawals during the course of the study**

addition we recorded the following indirect outcomes: intracranial pressure (ICP), magnetic resonance image (MRI) findings, computed tomography (CT) findings and cost-effectiveness. Any reported adverse events of HBOT were also recorded.

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 applied the quality scale of Jadad (Table 1).[21] We also recorded the adequacy of allocation concealment. If any relevant data were missing from trial reports, we attempted to contact the authors. To allow an intention to treat analysis we extracted the data reflecting the original allocation group where possible. Disagreements were again settled by consensus.

STATISTICAL ANALYSIS

Following agreement, the data were entered into Review Manager® 4.2.1. (Cochrane Collaboration, Oxford, UK). For dichotomous outcomes such as the proportion of participants who died, we calculated Relative Risks (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 change in ICP 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.[22] Consideration was then given

Table 2
Characteristics of included studies (GOS - Glasgow outcome score)

Study	Methods	Participants	Interventions	Outcomes
Artru 1976[28]	Method of randomisation not stated. No blinding reported. Jadad score 2.	60 participants, 31 HBOT and 29 control. Inclusion depended on availability of chamber. Stratified into nine categories of severity and pathology.	Control: 'Standard care' included hyperventilation and frusemide. HBOT: above plus 2.5 ATA oxygen for 1 hour daily for 10 days, followed by 4 days rest and repeat if not responding.	Death Unfavourable outcome Adverse effects
Holbach 1974[29]	Quasi-random, no blinding. Jadad score 1.	99 participants, 49 HBOT and 50 control. Included: history of closed head injury and comatose with 'acute midbrain syndrome'.	Control: 'Usual intensive care regimen'. HBOT: above plus 1.5 ATA oxygen for 60 mins daily. Total dose not stated.	Death Complete recovery
Ren 2001[26]	Method of randomisation not stated. No blinding. Jadad score 1.	55 participants, 35 HBOT and 20 control. Included: closed head injury with GCS < 9. Randomised on day 3 post admission after condition stabilised.	Control: Standard care plus dehydration, steroids and antibiotics. HBOT: above plus 2.5 ATA for a total of 400 to 600 minutes every 4 days, repeated 3 or 4 times.	Favourable GOS Change in GCS
Rockswold 1992[27]	Method of randomisation not clear, medical assessors blind. Jadad score 2.	168 participants, 84 HBOT and 84 control. Included: closed head injury with GCS < 10 for > 6 hrs, < 24 hrs.	Control: 'Intensive neurosurgical care according to a comprehensive protocol'. HBOT: above plus 1.5 ATA oxygen for 1 hour every 8 hours for 2 weeks or until waking or death (ave 21 treatments).	Death Favourable outcome (GOS 1 or 2) ICP Adverse events

Table 3
Summary of pooled outcomes

Outcome	Studies	N HBOT/Control	Efficacy data[a] with 95% CI, P-value and NNT
Good functional outcome at four weeks	Holbach 1974[29] Artru 1976[28]	80/79	RR 2.66, 95% CI 0.73 to 9.69
Good functional outcome at final follow-up	Holbach 1974[29] Artru 1976[28] Rockswold 1992[27] Ren 2001[26]	199/183	RR 1.94, 95% CI 0.92 to 4.08
Death at any time after enrolment	Holbach 1974[29] Artru 1976[28] Rockswold 1992[27] Ren 2001[26]	199/183	[b]RR 1.46, 95% CI 1.13 to 1.87 NNT 7, 95% CI 4 to 22
Development of any significant respiratory symptoms	Artru 1976[28] Rockswold 1992[27]	115/113	[b]RR 0.06, 95% CI 0.01 to 0.47 NNH 8, 95% CI 5 to 15

[a]RR: Relative Risk, NNT: number needed to treat, NNH: number needed to harm
[b]Significant outcomes (statistical difference is assumed if the 95%CI does not include the value 1.0)

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 and study quality. We also considered subgroup analysis based on age, oxygen dose, comparator therapy used, and the nature and severity of injury.

Results

THE INCLUDED STUDIES

The search in August 2004 yielded 23 articles of which seven were considered to be possible randomised human trials dealing with the treatment of TBI with HBOT. Two were excluded because they were incomplete reports of included trials,[23,24] and one because it enrolled only participants with non-acute injuries.[25] Four publications therefore met our inclusion criteria.[26-29] One trial[29] used a sequential system for allocation that may not have been truly random. The total number of participants enrolled was 382, 199 receiving HBOT and 183 control.

All four trials enrolled participants with closed head injury, but inclusion criteria varied. Rockswold[27] accepted those with a Glasgow Coma Score (GCS) of less than 10 for between six and 24 hours, Ren[26] accepted participants with a GCS of less than nine for up to three days after trauma. The other two older trials did not specify inclusion criteria, other than 'closed head injury and comatose'.[28,29] Treatment pressures (1.5 to 2.5ATA, or 152 to 253.3 kPa), time schedule (60 to 90 min), and number of sessions (10 to 40) of HBOT differed between studies. Similarly, there was some variation in comparator therapies and the time to final assessment. Individual study characteristics are given in Table 2.

No study described the method of randomisation, clearly concealed allocation from the individual responsible for randomisation or employed a sham therapy. Study quality was generally assessed as low and was not used as a basis for sensitivity analysis.

CLINICAL OUTCOMES

Statistical pooling was not possible for many of the 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. The data are summarised in Table 3.

PRIMARY OUTCOMES

Good functional outcome

Good functional outcome was defined in these studies as any one of the following: GOS < three,[26] 'return of consciousness',[28] 'complete recovery'[29] or classified as 'independent'.[27] Two trials reported this outcome early (0 to 4 weeks) following the course of therapy[28,29] and involved 159 participants. 29 (36%) were described as having a good outcome in the HBOT group versus 11 (14%) in the control group. Pooled analysis suggests however, that there is no significant difference between groups (RR with HBOT: 2.66,

95% CI 0.73 to 9.69, P = 0.06). There was evidence of significant heterogeneity between these studies (I^2 = 72%) and this result is performed using a random effects model (Figure 1).

Ren reported a significant improvement in the chance of a good outcome at six months' review[26] (RR 2.8, 95% CI 1.4 to 5.5, P = 0.004), while at one year, Rockswold did not[27] (RR 0.98, 95% CI 0.73 to 1.3, P = 0.87). When combining all trials at final outcome, 109 participants (51%) in the HBOT group had a good outcome versus 61 (34%) of controls, however this difference was not statistically significant (RR 1.94, 95% CI 0.92 to 4.08, P = 0.08). This result is very likely to be subject to important heterogeneity between trials (I^2 = 81%) and should be interpreted very cautiously.

Subgroup analysis by treatment pressure suggested the application of a high treatment pressure (2.5 ATA or 253.3 kPa) was associated with a better outcome than the application of a low treatment pressure (1.5 ATA or 152 kPa) (high pressure RR 2.07, 95% CI 1.15 to 3.72, P = 0.003, low pressure RR 2.12, 95% CI 0.35 to 12.78, P = 0.11). This result is unconvincing given the high probability of important heterogeneity remaining between the two low pressure trials (I^2 = 89%) and the similar estimate of RR in these two groups.

Mortality

Three trials reported this data at some time (Holbach at 12 days, Artru and Rockswold 1992 at 12 months) involving 327 participants. There was significantly increased mortality with control therapy (RR 1.46, 95% CI 1.13 to 1.87, P = 0.003). Heterogeneity between studies was low (I^2 = 0%). The NNT to avoid one death by applying HBOT was 7, 95% CI 4 to 22 (Figure 2).

No trials reported on activities of daily living, quality of life measures, CT or MRI findings, progress of GCS or cost-effectiveness.

SECONDARY OUTCOMES

Intracranial pressure

Only Rockswold reported the effects of therapy on ICP.[27] The effect of HBOT was complicated by a change in the experimental protocol during the period of recruitment. While overall there was no difference in the mean maximum ICP between the two groups (MD 3.1 mmHg lower with HBOT, 95% CI -9.6 mmHg to +3.4 mmHg), the authors noted higher than expected ICP in the early HBOT participants. As this was likely to represent pain from middle ear barotrauma (MEBT), the last 46 participants recruited to

Figure 1
Forest plot for risk of good outcome at final follow-up

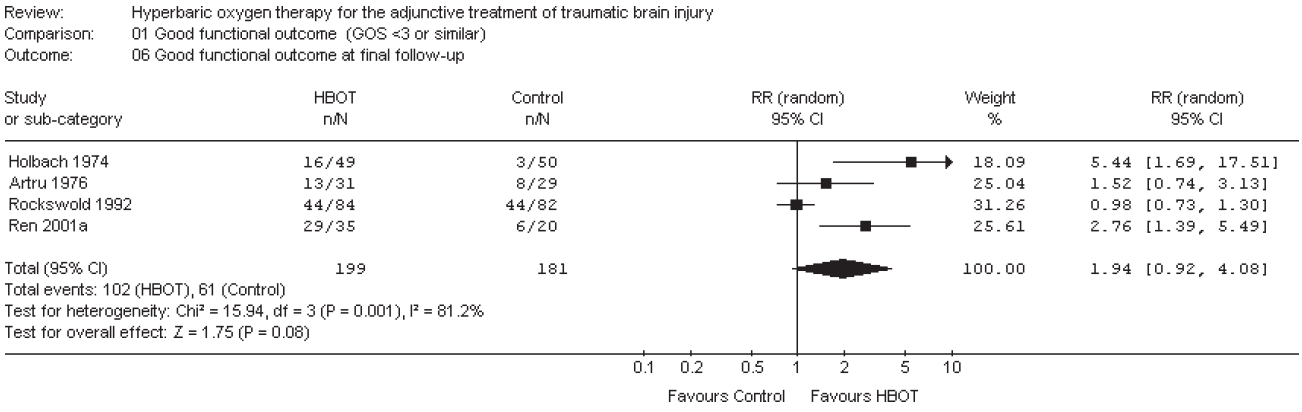
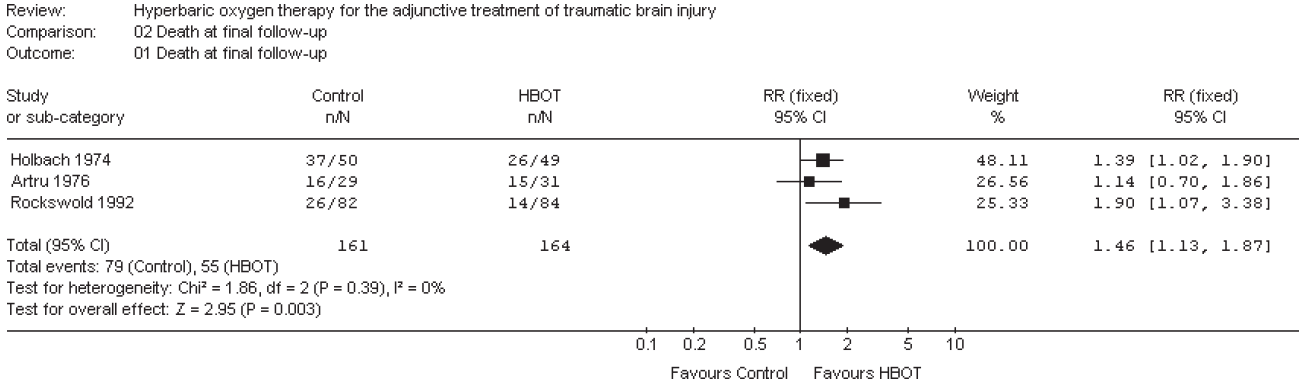


Figure 2
Forest plot for risk of death at any time after enrolment



HBOT had pre-compression myringotomy tubes inserted to allow free equalisation of middle ear pressures. Comparing the standard care group with the HBOT subjects with and without myringotomy, there is a significant lowering of ICP with HBOT plus myringotomy, but no difference without myringotomy (MD with myringotomy -8.2 mmHg, 95% CI -14.7 mmHg to -1.7 mmHg, P = 0.01; without myringotomy MD +2.7 mmHg, 95% CI -5.9 mmHg to +11.3 mmHg, P = 0.54).

Adverse effects

Rockswold reported generalised seizures in two participants in the HBOT group versus none in the control group (RR 0.2, P = 0.3) and a further two with haemotympanum from MEBT (RR 0.2, P = 0.03).

Two trials reported participants with significant pulmonary effects.[27,28] Rockswold reported ten individuals with rising oxygen requirements and infiltrates on chest x-ray, while Artru reported five patients with respiratory symptoms including cyanosis and hyperpnoea so severe as to imply 'impending hyperoxic pneumonia'. Overall, therefore, 15 patients (13% of those receiving HBOT) had severe pulmonary complications while no such complications were reported in the standard therapy arm. This difference is statistically significant (RR 0.06, 95% CI 0.01 to 0.47, P = 0.007). There was no indication of heterogeneity between trials (I^2 = 0%) and this analysis suggests we might expect to treat eight patients with HBOT in order to cause this adverse effect in one individual (NNH 8, 95% CI 5 to 15).

Discussion

This review has included data from four trials and we believe these represent all randomised human trials in this area, both published and unpublished, at the time of searching the databases. We found some evidence that HBOT reduces mortality following closed head injury, but cannot be confident that the addition of HBOT to standard therapy increases the chance of recovery to independence. The single trial looking at ICP as a proxy for beneficial effects did suggest that ICP was lower immediately following HBOT when patients had received middle ear ventilation tubes. These tubes avoid MEBT on compression – a highly painful and stimulating condition that might be expected to raise ICP, regardless of the underlying brain injury. Any clinical benefit may come at the cost of significant pulmonary complications. These complications are rare in general hyperbaric practice[30] and may be related specifically to the head injuries suffered by these patients.

Only 382 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 these trials, variability and poor reporting of entry criteria, the variable nature and timing of outcomes, poor reporting of both outcomes and methodology and the long time period spanned by the four trials (27 years). In particular, there is a possibility of bias due to different times to entry in these small trials, as well as from non-blinded management decisions in all trials.

We had planned to perform subgroup analyses with respect to age, oxygen dose, nature of comparative therapies and the severity of injury. The paucity of eligible trials and poor reporting suggested the majority of these analyses would not be informative, and we only performed subgroup analysis with respect to treatment pressure for the proportion of individuals achieving a good outcome. No standard severity index was employed uniformly across these trials, no standard injury pattern was established, and only Rockswold and Ren described the time at which the inclusion criteria were applied.

While 13% of participants in two of these trials suffered significant pulmonary complications, this is unusual, and HBOT is generally regarded as a relatively benign intervention. There are few major adverse effects (pulmonary barotrauma, drug reactions, injuries or death related to chamber fire), and a number of more minor complications that may occur commonly. Visual disturbance, usually reduction in visual acuity secondary to conformational changes in the lens, is very commonly reported – perhaps as many as 50% of those having a course of 30 treatments.[31] While the great majority of patients recover spontaneously over a period of days to weeks, a small proportion of patients continue to require correction to restore sight to pre-treatment levels. The second most common adverse effect associated with HBOT is barotrauma, usually MEBT, although other sites include the respiratory sinuses and dental cavities. Most episodes of barotrauma do not require the therapy to be abandoned. Less commonly, perhaps once every 5,000 treatments, HBOT may be associated with acute neurological toxicity manifesting as seizure.[30]

While we have made every effort to locate further unpublished data, it remains possible that this review is subject to a positive publication bias, with generally favourable trials more likely to achieve reporting. With regard to long-term outcomes following HBOT and any effect on the quality of life for these patients, we have located no relevant data.

Conclusions

We conclude there is limited evidence that HBOT reduces mortality in patients with acute TBI, but no clear evidence of improved functional outcome. The small number of studies, the modest numbers of patients, and the methodological and reporting inadequacies of the primary studies included in this review demand a cautious interpretation. We do not believe routine use of HBOT for these patients is justified by this review.

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 or classification most likely to benefit from this therapy and the oxygen dose most appropriate. Any future trials would also need to consider appropriate sample sizes with power to detect expected differences, appropriate and carefully defined comparator therapy, use of an effective sham therapy, effective and explicit blinding of outcome assessors, appropriate outcome measures including all those listed in this review, careful elucidation of any adverse effects and the cost-utility of the therapy.

Acknowledgements

We acknowledge the assistance provided by the Cochrane Injuries Group, and particularly of Katharine Ker and Paul Chinnock, in the production of this review.

The results of a Cochrane review can be interpreted differently, depending on people's perspectives and circumstances. Please consider the conclusions presented carefully. They are the opinions of review authors, and are not necessarily shared by The Cochrane Collaboration.

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ANZCA Citations

ANZCA Citations have been awarded to:
Dr Carl Edmonds
Dr Peter McCartney, and
Dr John Williamson
for their contributions to Diving and Hyperbaric Medicine. The South Pacific Underwater Medicine Society extends its congratulations.

The poetry doctor

Beware below blues

The sea is full of danger.
For me it is a fact
For whenever I go diving
I always get attacked.

The lion fish is lurking
Looking oh so tame
As I guide it with my hand
To fit my photo frame.

The jelly fish drifts passively,
Its tentacles so slim,
Yet as I swim through their mass
They wrap around my limbs.

The octopus just ogles me,
So serene and calm
As I admire its blue rings
Whilst it nestles in my palm.

The cone shell waits so patiently.
It shows no fire or fear
As I pick and pocket it
As a souvenir.

The stone fish sits so stoically
With camouflage so neat
As I walk the shallow reef
With unprotected feet.

The hydroid seems so innocent
So soft and fine and thin
As I gently fin past it
And brush my ankle skin.

As the sharks patrol the reef
I watch them with alarm
As they speed at me bare teethed
My speared fish underarm.

I am so scared to dive below.
It's full of dangerous things.
Please tell me how I can avoid
These bites and spines and stings?

I wrote this after brushing my ankle on a stinging hydroid. These stings always give me grief and afterwards I thought how stupid I am not to wear bootees every dive. A few days later I was bitten by a red back spider as I put my boot on in my shed. I was immensely grateful for the four ampoules of antivenene used to ease this particular reminder of how important it is to be cautious both in and out of the water.

John Parker
<www.thepoetrydoctor.com>

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates

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

1 The candidate must be medically qualified, and be a financial member of the Society of at least two years' standing.
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 andguidelines 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.

The Academic Board reserves the right to modify any of these requirements from time to time. The Academic Board consists of:
Dr Chris Acott, Education Officer, Professor Des Gorman and Associate Professor Mike Davis.
All enquiries should be addressed to the Education Officer:
Dr Chris Acott,
30 Park Avenue
Rosslyn Park
South Australia 5072
Australia
E-mail: *<cacott@optusnet.com.au>*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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Minutes of the SPUMS Executive Committee Teleconference held on 9 October 2005

Opened: 0900 hr

Present: Drs C Acott (President), S Sharkey (Secretary), G Williams (Public Officer), D Smart (ANZHMG Representative), M Davis (Editor), C Lee (Committee Member), D Vote (Committee Member)

Apologies: Drs R Walker (Immediate Past-President), A Patterson (Treasurer)

1 Minutes of the previous meeting (31 July 2005)
Moved that the minutes be accepted as a true record. Proposed Dr Sharkey, seconded Dr Vote, carried.

2 Matters arising from the previous minutes
2.1 The issue of formalising the functions required of the SPUMS Administrator is progressing. Current roles and functions are to be reviewed by the Committee at the next meeting with a view to formalising this arrangement in the form of an independent contractor agreement. ACTION: All.

2.2 Finalisation of the final figures from the 2004 ASM remained outstanding in view of the absence of the Treasurer for further comment. Action: Dr Patterson to advise of final accounts for 2004 ASM in particular whether the refunds had been reflected in the P&L.
2.3 The irregularities in the 2005 ASM financial reports require investigation. ACTION: Dr Patterson to pursue this issue with the Convenor and Administrator.
2.4 Audit of SPUMS equipment being progressed. ACTION: Dr Sharkey.
2.5 Confirmation of status of overseas representatives required. ACTION: Dr Sharkey.

3 Annual Scientific Meeting 2006

3.1 Preliminary timetable was proposed and agreed. AGM on the Wednesday night, Gala Night on the Friday night with workshops on the Monday, Tuesday and Thursday nights.
3.2 Registration fees: $450 members; $570 non-members; $180 partners.
3.3 CME points from relevant colleges are being sought.
3.4 Proposed workshops include variety of airway and ventilation procedures. Assistance in workshop delivery by Anaesthetic members is welcome. Proposed presentations include diabetes, PFOs and other shunts, asthma, breath-hold diving and immersion physiology, obesity, airway devices and resuscitation, reverse dive profiles, evolving problem sessions (FTD and emergency management of diving presentations).
3.5 Dr Williams advised that Consumer Affairs had agreed to slight delay in this year's AGM outside the rules.
3.6 2007 Scientific Meeting to be held in New Zealand – Convenor Dr M Davis; Co-convenor Dr S Mitchell. Meeting is confirmed for the third week in April 2007, venue is Tutakaka. The conference will have a predominantly physiological theme. Organisation of the 2007 conference is being progressed by Dr Davis.

4 Journal report

4.1 Discussions occurred regarding the issues relating to profit reduction due to lower membership numbers over last year. This included recognition of certain obstacles to be overcome for these reductions to be lessened. E.g., need to write personally to all old non-renewed members; need the new website to be up and running; possible need for the journal amalgamation to go ahead. In view of membership reduction and therefore financial considerations, Dr Davis declined the offer of an honorarium increase as editor at present.
4.2 Journal name update will take place in the New Year with the new volume. This will include the Australian National Library being informed and EMBASE Indexing.
4.3 CD production has been discussed with the printer. A searchable PDF CD covering the past 5 years is possible for A$900; additional to this, John's 30 volumes will incur a small fee resulting in approximately $1000 total price. A charge per CD could be added onto the membership fees and would incur a small profit – this could be available during 2006. A master CD by SNAP printers could be available for burning further CD copies – the commercial production option was preferred. Proposal approved.
4.4 New Zealand account status: NZ$2,800. The software update can be paid for from this account.
4.5 Outstanding contributions to the Journal are required urgently.

5 Education Officer's report

5.1 No new diplomas have been awarded.

6 Correspondence

6.1 Letter received from ANZCA SIG requesting that an ANZCA SIG member sits on the SPUMS Education Board for authorisation of the SPUMS Diploma – for Special Interest Group members of ANZCA. The request was endorsed by the majority of the Committee.

7 Other business

7.1 The Committee were informed that the current Treasurer had advised his desire to resign from the position on completion of this calender year. The Committee would prefer that he remain in this position but wish him well in his future endeavours if he is unable to remain. Successor is yet to be determined. With respect to Dr Patterson's current role in acting as Convenor of the 2006 ASM, he also advised that he would be happy to hand over that task if the Committee can find someone to assume this role.
7.2 Australian Standards Report: Dr Smart reported on the proceedings of the recent AS meetings. This report is included as an annex to these minutes.
7.3 ANZHMG phone conferences (one per year) agreed to be paid for by SPUMS.
7.4 HTNA prize dually awarded to Helen Mullins from Fremantle: A review of visual acuity changes in patients receiving more than 20 treatments; Anne Sydes from Wesley: A case series of pyoderma gangrenosum.
7.5 Congratulations extended to Des Gorman who has recently accepted the appointment as Head of School of Medicine at the University of Auckland.

Closed: 1052 hr

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>

Combined meeting of Australian Standards SF017 Occupational Diving and CS083 Recreational Diving

Held on Monday 19 September 2005

A combined meeting was held at Australian Standards in Sydney to discuss a number of International Standards drafts which have been proposed from the International Standards Organisation (ISO) covering the health and training of recreational divers at the following levels:

1 Supervised diver – to 12 metres
2 Autonomous diver
3 Dive leader (divemaster)
4 Instructor level 1 (Assistant instructor)
5 Instructor
6 A broader standard covering providers of training

The Australian equivalent is the recreational 4005 series.

The ISO standards cover terms and definitions, competencies, prerequisites, theoretical knowledge, personal and specific scuba skills and assessment of the recreational divers. The standards also were very light on defining the amount of theory required. Surprisingly these standards did not even define that they were designed to train people on air.

The International Standards presented to the Committee were significantly lacking in detail and inconsistent regarding the need for diving medicals prior to undertaking a course. For example there were three different wordings regarding health requirements with the lowest standards applicable for entry-level divers. The following are quoted from the draft standards:

> Supervised Diver and Autonomous Diver: "*Documented evidence shall be obtained that the student has been medically screened as suitable for recreational diving by means of an appropriate questionnaire or medical examination. In case of any doubt or at the scuba instructor's discretion, students shall be referred to proper medical resources. If the student is not examined by a physician the student shall be obliged to confirm by signature that he or she has understood the written information given by the scuba instructor on diseases and physical conditions which may pose diving related risks*."

> Dive Leader (divemaster): "*Documented evidence shall be obtained that the student has been medically screened as suitable for recreational diving. NOTE In some countries and training organisations a medical examination is mandatory*."

> Scuba instructors level 1 and 2: "*Scuba instructor candidates shall be medically screened as suitable for diving according to procedures laid down by a competent medical authority. If such procedures are not specified scuba instructor candidates shall provide evidence of a diver medical examination not older than one year unless the medical doctor who has carried out the examination specifies longer validity*."

After working through the documents word by word, the CS083 Australian Committee rejected the documents, with a detailed submission forwarded to ISO. The ISO standards fell far short of the existing 4005 series Australian Standards in their detail relating to definitions, emergency equipment and procedures, risk assessment, and the standards of supervision required for the divers. The Committee's position was that all divers covered by the standards required diving medicals.

There are some interesting processes taking place in relation to the International Standards. The ISO series we examined evolved from the European Standards Committee, with some origins from the tourism and leisure sector. There is also an attempt to fast-track the ISO standards. There also appears to be some pressure on Australian Standards as an organisation to adopt international standards, even when our own standards have greater detail and have been more thoroughly worked.

Australia is only a single voting member in a larger body containing over 20 countries. It is likely that, although our objections to the ISO documents will be heard, we will be unable to influence the final ISO standards published. Once ISO standards are published there is likely to be political pressure for Australia to adopt them because they cover areas in common with Australian Standards such as the 4005 series. The only option we have if ISO does not listen to our input, will be to provide appendices and additions to the standards to suit the Australian conditions.

It is of note that the AS2299.3 covering professionals working in the recreational industry did not have an International Standards equivalent and the detail covered in this standard is far in excess of the detail covered in the International Standard No.6.

Overall, I have significant concerns about the International Standards process allowing adequate Australian input given our substantial experience in recreational diving in this country.

Dr David Smart
SPUMS Representative, Australian Standards (Occupational)

Standards Australia Meeting SF017

Held on Tuesday 20 September 2005

Topics discussed
Revision of Australian Standard 2299.1 Occupational Diving Standard Occupational Practice
Review of draft 2815.5 Training and Certification of Occupational Divers Part 5 Dive Supervisor

The main areas covered under the Occupational Diving Operations were:

1. Recompression chamber support:
In the absence of clear evidence outlining how recompression chamber support should be provided to the on-shore and off-shore industries as well as scientific diving, an expert consensus was agreed to. The situations for commercial diving requiring a chamber within two hours were defined. A second time period greater than two hours for chamber support was defined with some restrictions on diving practices applying. This simplified the Standard from 3 columns to 2 in relation to chamber support. The scientific diving community was also provided with risk assessment guidelines which would define the situation where scientific diving required a chamber in less than two hours, for example: risk of entanglement, use of specialised tools, decompression diving, diving greater than 30 metres and risk of rapid ascents. A detailed risk assessment form was also developed for assessing risk in relation to diving in accordance with AS2299.1. The medical fitness to dive form has been slightly revamped but would not be significantly different from the existing 2299 form.

2. The AS2299.1 (2005) form will also be released for public comment, probably at the end of the year.

3. This meeting also reported that the AS2299.4 has been released as an official standard and is available to the public for purchase. This covers film and photographic diving.

4. Review of the Training and Certification of Occupational Divers Part 5 Dive Supervisor:
This occurred at the meeting and a consensus was agreed to allow this form to be released for public document and public comment.

Future business of the Committee will require a review of the AS2815.1.2.3.4 series and a further review of the Scientific Standard 2299.2.

Dr David Smart
SPUMS Representative, Australian Standards (Occupational)

SPUMS Annual General Meeting 2006

Notice of the Annual General Meeting of SPUMS to be held at The Pearl South Pacific Resort, Pacific Harbour, Fiji, at 1800 hrs, Wednesday 7 June 2006

Agenda

Apologies:
Minutes of the previous meeting:
Unratified minutes of the previous meeting will be posted on the meeting notice board and appeared in the *SPUMS J*. 2005; 35: 97-101.
Matters arising from the minutes:
Annual reports:
President's Report.
Secretary's Report
Education Officer's Report
Presidents' Committee Report
Annual Financial Statement and Treasurer's Report:
Proposal regarding subscription fees for 2006:
That the annual subscription rates for membership of the Society be set at AUD130.00 plus GST for Full Members and AUD70.00 plus GST for Associate Members with effect from January 2007.
Proposed: Dr A Patterson; Seconded: Dr C Acott
Reasons:
Rising costs of running the Society and producing its Journal make the increase in subscription rates inevitable. The subscription rates have been held at present levels for some four years, in the face of inexorable increases in costs. The proposed new subscription rates reflect a very modest rise compared with CPI increases or inflation over the same period. I commend the new rates to members.
Election of office bearers:
Nominations have been called for the positions of Treasurer and one committee member.
Appointment of the Auditor:
Business of which notice has been given:
1.Motions re Consumer Affairs-required amendments to constitution to comply with Victorian State legislation.
Proposed: Dr Williams Seconded: Dr Sharkey

2.Motion re adoption of model rules for publishing of Minutes
Proposed: Dr Walker Seconded: Dr Sharkey

3.Motion re additional membership category for retired members
Proposed: Dr Walker Seconded: Dr Sharkey

4.Nomination of Martin Sayer as Full Member
Proposed: Dr Davis Seconded: Dr Acott

Reverse dive profiles

Reverse dive profiles: the making of a myth. A response

Michael A Lang and Charles E Lehner

Key words
Reverse dive profiles, decompression sickness, evidence

Abstract
(Lang MA, Lehner CE. Reverse dive profiles: the making of a myth. A response. *Diving and Hyperbaric Medicine*. 2006; 36: 51-5.)
The original aims of the Reverse Dive Profile Workshop were to challenge the reasoning behind FDPs and to generate an understanding as to where the historical objection to RDPs originated. While there was a lack of definitive experimental evidence advocating RDPs, it was the lack of evidence prohibiting them that was the issue. In their review article 'Reverse dive profiles: the making of a myth', Edmonds, McInnes, and Bennett fail to impose the desired level of uncertainty on the subject of RDPs, in the context of the Workshop's findings and conclusion, and have added little to the debate that took place at the Workshop. We find no reason for the diving communities to prohibit reverse dive profiles within the no-decompression limits for dives less than 40 msw (130 fsw) and depth differentials less than 12 msw (40 fsw).

In their review article 'Reverse dive profiles: the making of a myth',[1] Edmonds, McInnes, and Bennett conclude that the results of a workshop report[2] revoke established procedures advocating forward dive profiles (FDPs) and promote reverse dive profiles (RDPs) as safe and equivalent alternatives. The authors have added little to the debate that took place at the Workshop. Four pages of criticism of an historical document supplemented by five paragraphs of "new data" fail to impose the desired level of uncertainty on the subject of RDPs, in the context of the Workshop's findings and conclusion.

The original aims of the Reverse Dive Profile Workshop were to challenge the reasoning behind FDPs and to generate an understanding as to where the historical objection to RDPs originated. In the Proceedings of the Workshop, we summarised the evolution of the prohibition of RDPs, defined either as two dives performed within 12 hours in which the second dive is deeper than the first; or, as the performance of a single dive in which the latter portion of the dive is deeper than the earlier portion. The collective knowledge and experience of the highly talented body of workshop participants were not likely to be overcome by a predetermined agenda, as implied by Edmonds et al.[1]

The workshop data

While Edmonds et al point to the lack of definitive experimental evidence advocating RDPs, it is the lack of evidence prohibiting them that is the issue. Although we agree that RDPs have become more prevalent in recent years, the ability of divers to manage an acceptable probability of decompression sickness (pDCS) will clearly depend on the extent to which their profiles approximate the prescribed dive computer algorithms and concomitant decompression obligations. The rationale for the ban against RDPs reviewed at the Workshop indicated that it, also, was based on opinion (and theory) rather than evidence. In the absence of supporting evidence, the necessity of a ban was called into question. Forward profiles are not banned even though we know they have been reported to cause DCS.

Accepting the paucity of experimental data directly addressing the reverse profile issue, the Workshop also succeeded in demonstrating that the traditional recreational diving recommendation (deep then shallow) was similarly lacking in sufficient evidence to justify its abolition. We also showed that RDPs were included in the validation of several tables and dive computer algorithms. Edmonds et al appear to discount these historical data, preferring instead to assume that the safety of FDPs is now being revoked in favour of RDPs.

The scientific, commercial, and military operational diving profiles are well documented and an outcome is ascertained for each profile (DCS/no DCS). In that vein, we argue that these operational exposures in fact constitute data and are not opinion based. The scientific diving community's diving data are scrutinized and recorded for US regulatory purposes by mandate of the Department of Labor.[3] From 2000–2005, we have seen no increase in DCS cases from RDPs. Vann et al reviewed the Project Dive Exploration (PDE) data and found no evidence that RDPs had higher DCS risk than FDPs for diving as conducted by the PDE volunteers.[4] Millions of dives are being done each year around the world and we have no idea what the predominant approach to diving is. FDPs may well be favoured due to the historical ban on RDPs. However, information from chamber operations shows that the predominant profiles of divers presenting are FDPs. The hypothesis that there exist

no physiological data prohibiting reverse profiles within the envelope of the Workshop's conclusion stands. Operational data from the diving communities clearly show that FDPs were preferentially driven by logistical and mechanistic considerations for over a half century. Neither the US Navy nor commercial diving operational procedures specifically prohibit reverse profile diving.

The authors quote the Convenor as stating "*Does it really matter in which order dives are conducted as long as one keeps track of nitrogen loads and performs adequate decompression?*" They continue "*The follow-up question that remained unanswered was: do RDPs and FDPs actually have the same decompression obligations, and can we therefore apply the same decompression requirements to them?*" This is incorrect. They ignore what was stated about keeping track of nitrogen loads. On the contrary, FDPs and RDPs were repeatedly recognised as *not* requiring comparable decompression. Edmonds et al misinterpret our conclusion by testing "mirror" profiles, yet nowhere in the findings and conclusion, or in the body of the Proceedings, did we imply that RDPs that were mirror images of FDPs could be safely undertaken. This appears to be the tangent that the authors embarked on.

Edmonds et al have inserted into their argument observations by Huggins, who hinted at the potential for more severe DCS with RDPs from chamber treatment observations,[5] and St Leger Dowse et al, who analysed UK female divers' log books and indicated that symptom rates were higher in those using RDPs.[6] These observations are valid, but in the context of the authors' argument, they are not evidence. Their text suggests that these data support the notion that DCS severity and symptom rates are greater with RDPs. However, as they point out, neither data set reached statistical significance. The odds ratio for Huggins' data was 1.21 (95% CI 0.68, 2.13), arguably not even close to statistical or clinical significance. Furthermore, there was insufficient detail in the data to control for dive profile, maximum dive depth, or any other risk factor.

Regarding the restrictions agreed upon at the Workshop, these were inserted into the conclusion in order to be conservative, and to obtain consensus (since not all participants opined that the RDP 'ban' should be completely abolished). With the stipulations as stated, there was in fact 100% agreement (of 49 participants).

Indeed, Edmonds et al's assertions represent exactly the kind of conclusion that can arise without historical perspective. Presented with the same literature we searched to examine the gradual evolution of the ban on reverse dive profiles, we are optimistic that the authors would similarly conclude that there exists a lack of definitive experimental evidence supporting this ban. However, diving operational history with RDPs can be neither ignored nor changed.

From the modelling perspective presented at the Workshop we remain convinced that it does not matter what the pattern of profile exposure is provided two things are taken care of: quality decompression according to the last exposure, and not unwittingly creating bubbles at an early stage, which are then ignored.

The animal experiments

Edmonds et al's evidence for the making of a reverse-profile myth resides in a series of animal experiments. However, the myth-debunking extrapolation to humans, or to the Reverse Dive Profiles Workshop findings and conclusion, is inappropriate. As reported, this study's results have no bearing on the real world of diving.

Dive severity can influence the conclusions of a study. The key question is when do the dive profiles become severe enough to show a significant difference between RDPs and FDPs? This question can be answered only by recording human dive profiles during field use and documenting the outcomes. Is it possible that the authors made up their minds about RDPs and constructed experiments to support their preconception? We agree that under some circumstances RDPs can be hazardous but that has yet to be demonstrated in humans. The inapplicability of their animal study to humans is the greatest weakness of their review article.

Many models will demonstrate that for the same dives, 'deep' followed by 'shallow' will produce higher tissue inert gas tensions, and will therefore require different decompression procedures. This is reflected in standard decompression algorithms, such as the US Navy Standard Air Decompression Tables. That mirror-image RDPs demand an equal decompression obligation to FDPs is argued by default and no cogent mechanistic explanation is offered by the authors for the experimental design of their animal dives. If they imply that RDPs in a repetitive series incur the same decompression obligation as FDPs, they must reconcile their scenario with the observation that there exists no dive computer algorithm or table that would allow such profiles without significantly altering the pDCS. The experiment designed by Edmonds et al to excommunicate the workshop findings does not take into account any type of handicap in repetitive diving. Both Huggins[7] and Gerth and Thalmann[8] estimated DCS risk on profiles within the algorithms' required decompression parameters. For the repetitive dive scenario they took into account the handicap accumulated due to the previous dive (FDP or RDP). In order to maintain the same level of DCS risk in a repetitive dive, the current dive must be shorter, shallower, or start after a longer surface interval (SI).

A bubble model would prescribe the following if a diver intended to repeat a FDP series (30 msw/30 min, 15 min SI, 20 msw/30 min, 15 min SI, 10 msw/30 min) in reverse order. To keep the dive depths and bottom times constant, the surface intervals would have to be extended as follows:

- surface interval after first dive (10 msw/30 min): 90 min
- surface interval after second dive (20 msw/30 min): 120 min

These modifications would provide a predicted DCS risk that was approximately equal for FDPs and RDPs.

The authors state "*our findings suggest that multi-level and repetitive dives performed in the established forward profile manner are less hazardous than those performed in the reverse profile mode*." However, to imply that a Haldanian-based dive computer will allow hazardous profiles is incorrect and misleading.

Edmonds et al successfully tested nitrogen levels at the surface following these four profiles:

- 36 msw/30 min to 24 msw/30 min to 12 msw/30 min
- 30 msw/40 min
- 30 msw/40 min, SI 15 min, 20 msw/40 min
- 30 msw/40 min, SI 15 min, 20 msw/40 min, SI 15 min, 10 msw/40 min

Using the maximum tested surface nitrogen loading for tissues with halftimes ranging from 5 to 480 minutes thus established, we have the following things to say about the profiles that proved hazardous:

- for the RDP multi-level dive that begins with 12 msw/30 min to 24 msw/30 min, *no* remaining time was allowed for a subsequent descent to 36 msw. The study's results from 30 minutes at this depth causing 50% casualties come as not unexpected, and;
- for the RDP repetitive dive that consisted of 10 msw/40 min, SI for 15 min, 20 msw/40 min, SI for 15 min, then descent to 30 msw, only 19 min were allowed as compared to the tested 40 min that produced 33% DCS.

Thus, diving shallowest first (RDP) converts a FDP that barely requires decompression to a dive that requires much decompression, underscoring the 'practical' reasons divers perform FDPs. The question is whether the second dive, *if proper decompression is executed,* is as safe as the first dive. In this case, we would not want to venture a guess (i.e., a borderline 'no-stop dive' versus a properly executed decompression dive), but certainly to decompress the second (RDP) dive the same way as the first (i.e., 'no stop') is unsafe and not what the Workshop recommended.

Conclusion

We find no reason for the diving communities to prohibit reverse dive profiles within the no-decompression limits for dives less than 40 msw (130 fsw) and depth differentials less than 12 msw (40 fsw).

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Michael A Lang, BSc, is the Director of the Smithsonian Marine Science Network and Scientific Diving Program, and was the Guest Speaker at the SPUMS ASM, The Maldives, 2005.

Charles E Lehner, BA, PhD, is Assistant Scientist and Director of the Biotron Diving Physiology Laboratory, at the University of Wisconsin, Madison, Wisconsin, USA.

E-mail: *<celehner@facstaff.wisc.edu>*

They were the convenors of the Reverse Dive Profile Workshop held at The Smithsonian Institution, October 29–30, 1999.

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Office of the Under Secretary for Science,
PO Box 37012,
Washington, DC, 20013-7012, USA

E-mail: *<Langm@si.edu>*

Drs Edmonds, McInnes and Bennett reply:

The response of Lang and Lehner to our article on "Reverse dive profiles: the making of a myth" is welcome, shedding more light as it does on the intended meaning of the Workshop recommendations.[1] We think their response makes it clear that we are in agreement about the facts. It is on the interpretation of these facts that we disagree, and the primary reason for our article was to illustrate, by documenting the statements of other delegates, that we are not alone in interpreting the final recommendations as contentious. We attempted to put the recommendations into perspective, highlighting the qualifications and doubts expressed in the proceedings of the Workshop.

Having organised and edited the Workshop, Lang and Lehner are in a position to appreciate the controversial nature of the problems of comparing the relative safety of forward dive profiles (FDP) with that of reverse dive profiles (RDP). They appreciate the limitations of the data, as described in their letter, but others who just read and accept the findings and recommendations of the Workshop may not. Interpreted literally, the recommendations indicate no increase in DCS with RDP compared to FDP, and that the no-decompression limits are the same. Lang and Lehner claim that it does not matter what the pattern of the profile is, as long as there is adequate decompression. We agree. It is axiomatic. If you decompress adequately, you are much less likely to get decompression sickness (DCS), irrespective of the profile, and without any qualification.

Our objections were not so much to the absence of evidence in either direction (safety of FDP vs RDP), but to the *implication* that the two dive profiles are equivalent. RDPs impose different decompression requirements than FDP dives. We have never proposed the prohibition of RDPs, only (like Lang and Lehner) the application of appropriate (and different) decompression. This difference in decompression obligation was unfortunately glossed over in the summarised findings and recommendations promulgated.

We believe this is the explanation for subsequent publicity in the diving literature, which we quote in our article and which uses the Workshop as authority, that dismisses the significant differences in decompression requirements between RDPs and FDPs. This interpretation is inadvertently encouraged by Lang and Lehner in their own summaries: "*There is no convincing evidence that RDP within the no-decompression limits lead to a measurable increase in decompression sickness*".[1] There is in this statement an assumption that all readers will understand that a different (and unstated) decompression requirement will operate in the two situations. We are sure this was not an intentional omission, and that the workshop participants understood this assumption very well. Perhaps so well that it seemed to be stating the obvious and did not therefore require clear elucidation.

If the recommendations stipulated that FDPs and RDPs had different decompression obligations and that one cannot extrapolate from one to the other, there would have been no need for our article. Unfortunately the Workshop is now being quoted as indicating no difference between FDPs and RDPs.

We also agree that some decompression algorithms in dive computers attempt to make allowance for an added risk with RDPs. We just do not know which ones, if any, achieve this effectively. What is needed is good experimental research to investigate the safety of a variety of algorithms. Because of the nature of the problem, we believe this is only achievable through appropriate animal models. Such models are inevitably imperfect and require extrapolation to the human experience. They are, however, superior in some respects to anecdotal reports of human diving experience where the algorithm in use is only one of the variables influencing outcome. The best assessment of safety is likely to be a synthesis of both types of investigation.

Areas in which we must agree to disagree, and which we discuss in our paper, include the historical development of the FDP recommendations, the logistics of applying the 40 metres' sea water (msw) maximum depth and 12 msw differential gradient as recommended, and the appropriateness of some of the data presented in the Workshop.

Lang and Lehner imply a plethora of new data on RDPs from scientific divers from 2000–2005, and the scrutinised monitoring of these with only a minor DCS risk. In fact, the 2005 article gives no data on RDPs and approximately two thirds of the scientific dives are at depths less than 9 msw.[2] The argument is a little circular. To support the Workshop's recommendations for the relative safety of RDPs they refer to new scientific diver data and direct us to the *SPUMS Journal* article for the data.[2] In this article there are no such RDP data and the Workshop is referenced.

We suggest another revised RDP recommendation, which complies with the data available both before and after the Workshop:

> *"RDPs have different decompression requirements to FDPs, and these requirements should be validated for both decompression tables and decompression computer algorithms before use."*

References

1 Lang MA, Lehner CE, editors. *Proceedings of the Reverse Dive Profile Workshop*; 1999 Oct 29-30. Washington, DC: Smithsonian Institution; 2000.
2 Lang MA. The USA scientific diving medical and safety experience. *SPUMS J*. 2005; 35: 154-161.

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Key words
Reverse dive profiles, decompression sickness, letters (to the Editor)

Letters to the Editor

Bearded ghouls and scientific meetings

Dear Editor,

With respect to Dr Harris' article in the December issue,[1] the pain from the bearded ghoul's sting appeared to be resistant to the use of hot water. I was wondering how hot the water was? My clinical experience with stings from similar fish (scorpion fish and stonefish) indicates that the temperature of the water is crucial – warm water produces no relief but hot water produces initial relief but the pain reappears as the temperature of the water decreases. However, I am the first to admit that our knowledge of the action of these venoms is only 'the tip of the iceberg' and perhaps some venoms are resistant to first-aid hot-water treatment.

I was interested in the use of a sural nerve block for pain relief. I have used this nerve block for pain relief in these injuries with great success. However, I have had to combine it with a tibial nerve block for full relief in what appears to be the area involved in the photograph; the medial side of the foot is supplied by both the sural and medial plantar (a branch of the tibial nerve) nerves, but it is a poor photograph.

Where Dr Harris' thoughts[2] on the SPUMS AGM are concerned, I agree with the Editor's reply. I note Dr Harris' opinion is based on attendance at one meeting (in 'statistical terms' expressed as n = 1). The SPUMS Committee is trying to improve the ASM but we do need participation from members to submit presentations and attend. Perhaps we can look forward to seeing and hearing from Dr Harris at future ASMs. Past onshore meetings have not been successful but this will be tested again in the future.

Dr Christopher J Acott
President, SPUMS
E-mail: *<cacott@optusnet.com.au>*

References

1 Harris R. A fishy tale from Port Vila (with a sting in the tail). *SPUMS J*. 2005; 35: 225.
2 Harris R. Time to change the Annual Scientific Meetings (ASMs). *SPUMS J*. 2005; 35: 227.

Key words
Envenomation, marine animals, medical society, meetings, letters (to the Editor)

Maintenance of Professional Standards (MOPS)

Dear Editor,

The following MOPS points have just been approved by the Australian and New Zealand College of Anaesthetists:

The "Introductory Course in Diving and Hyperbaric Medicine" presently held at Prince of Wales Hospital, Sydney has been approved under Code 161, Category 4 (Learning Project) for 100 CME points. The approval number for this activity is 02116 and is ongoing.

Jan P Lehm, Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Randwick, NSW 2031, Australia
E-mail: *<lehmj@sesahs.nsw.gov.au>*

Key words
Letters (to the Editor), meetings, MOPS

Possible long term health effects of diving at work (the ELTHI study)

Dear Editor,

I read with appreciation David Elliott's synopsis of our first report on this project.[1] Three reports have now been compiled, which can be found on the Health and Safety Executive's website,[2–4] and I write to summarise our findings to date. We studied United Kingdom professional divers who had passed a fitness-to-dive medical before 1991. In the first report there were three main conclusions. The major factors affecting health-related quality of life were work-related accidents for both divers and the control group of offshore workers. There was a very high prevalence of noise-induced hearing loss (close to 50%) in both groups with a weak association with saturation diving in divers. Eighteen per cent of divers as opposed to 6% of control subjects reported cognitive complaint which was associated with work as a welder and diving experience. Cognitive complaint was associated with a moderate reduction in health-related quality of life of the same order of magnitude as that associated with loss of a spouse or divorce but there was no evidence that there was more work-related disability in divers.

A follow-up study considered welding fume as a possible causative factor for cognitive complaint and looked again at the data from the first study to determine possible causative factors.[2] There was no relationship between exposure to welding fume and cognitive complaint in divers, implying that divers who weld are exposed to something else that increases risk of cognitive complaint.

In further analysis of the data from the initial study, it was clear that cognitive complaint was associated with experience of the oilfield diving techniques, saturation, mixed-gas bounce and surface-oxygen decompression diving in unadjusted models. When the analysis was adjusted for possible causative factors other than diving, however, only mixed-gas bounce remained associated with complaint. Other significant associations were with work as a welder, neurological decompression illness, more than one report of exposure to contaminated breathing gas and reported exposure to "a lot of petrochemical solvents or paints".

The present picture regarding cognitive complaint in UK professional divers is of an effect associated with oilfield diving but not necessarily with the act of diving itself since, although there was an association with neurological decompression illness, there were more important relationships with reports of exposure to toxins at work. There was a robust relationship, however, with experience of mixed-gas bounce diving. Although this technique is infrequently used in industry, this does have implications for 'techie' divers and deep, mines-clearance divers.

A follow-up study is planned to look at progression and to try to get a quantitative estimate of toxin exposure, but it may well be that improved occupational hygiene in oilfield diving will be the most effective means of improving safety for the profession.

John AS Ross
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E-mail: *<j.a.ross@abdn.ac.uk>*

References

1 Elliott D. Commentary on the ELTHI diving study. *SPUMS J*. 2005; 35: 106-8.
2 Macdiarmid JI, Ross JAS, Taylor CL, Watt SJ, Adie W, Osman LM, et al. Co-ordinated investigation into the possible long term health effects of diving at work. Examination of the long term health impact of diving: the ELTHI diving study. Research Report 230. London: HMSO; 2004. Available at: www.hse.gov.uk/research/rrhtm/rr230.htm
3 Macdiarmid JI, Ross JAS, Semple S, Osman LM, Watt SJ, Crawford JR. Further investigation of possible musculoskeletal and cognitive deficit due to welding in divers identified in the ELTHI Diving study. Research Report 390. London: HMSO; 2005. Available at: www.hse.gov.uk/research/rrhtm/rr390.htm
4 Osman LM, Adie W, Cairns J. Attitudes to safety culture among professional divers and offshore workers. Research Report 159. London: HMSO; 2003. Available at: http://www.hse.gov.uk/research/rrhtm/rr159.htm

Key words
Letters (to the Editor), diving at work, occupational diving, occupational health, health surveillance, hearing, psychology, performance

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DVD review

The duty of care

Produced by PADI Asia Pacific, Training and Education Department, 2004
Far North Queensland Film Company
Length: 66 minutes
Price: AUD18.69
Available on request from PADI Asia Pacific
Website: <www.padi.com>

The duty of care is a sobering insight into a civil court case resulting from a hypothetical diving accident. In the accident, inexperienced diver "Mark" was undertaking his first deep dive on a PADI Advanced Open Water course. He experienced problems with excessive air consumption, which led to an out-of-air ascent and, ultimately, his death. Introduced by Richard Evans (Quality and Risk Management, PADI Asia Pacific), the court case provides an excellent forum to present the facts of the accident, and to examine the events before and after the diver's death. A number of errors were highlighted that led to dive instructor "Roger" receiving a finding of negligence against him. The courtroom scene is given authenticity by contributions from magistrate Trevor Black and barrister Kevin Priestley, and by PADI staff who perform roles in the case.

Mark had limited diving experience before enrolling in the course. He had problems with his buoyancy on a navigation dive the day before the accident. This initial dive was not directly supervised by Roger, but instead supervision was delegated to "Jim", a divemaster. Jim noted Mark's problems but did not communicate these to Roger, and Roger did not specifically inquire about any difficulties with the students before embarking on the deep dive the next day.

Before the deep dive Mark said he was nervous, but was not fully interrogated by the instructor. During the descent, Mark had difficulty with ear clearing, contributing to a delayed ascent and increased air consumption. His instructor did not confirm that Mark was OK before completing the descent. The dive was planned for 28 metres but reached 32 metres; beyond the limit of the PADI standard. Air consumption was not checked in any of the students until it was discovered that Mark had 70 bar left in his cylinder.

At this point Roger delegated "Yuki" (another inexperienced student) to accompany Mark to the surface. The instructor did not accompany the divers during the ascent. They ascended away from the anchor line and during the ascent, Mark ran out of air, attempted unsuccessfully to buddy breathe with Yuki, and then made an uncontrolled ascent to the surface. He was unconscious, and then sank before the dive boat could rescue him.

The court found that Roger had a duty of care to Mark, and that he failed to properly supervise and monitor Mark's dive. It also expressed concerns regarding Mark's failure to follow PADI standards. A ruling of negligence followed.

Nearly half the DVD is devoted to commentary by experts, including Dr Simon Mitchell (Diving Medicine Specialist), Chris Coxon (Dive Safety Expert, Queensland), Sharon Daniels (Clinical Psychologist), Michael Gatehouse (Solicitor and Diving Litigation Specialist). They all make the point that it would have been easier to defend Roger had he followed the PADI standards. David Strike (author of *Diving and the media*) provides perspective and advice on how to deal with the media. A useful contribution is also made by insurance executive Rob Veal, who points out the need for insurance and the likely cost of this event if individuals are not insured. The expert commentary finishes with a description of the aftermath of a dive accident, demonstrating how stressful and protracted the process can be for those involved, and the impact on all concerned.

Overall the DVD is a quality production and is relevant to the Australasian situation. It provides a detailed insight into diving risk management using the practical example of a fatal diving accident. The DVD would be suitable for all industry participants who derive their income from instructing or leading dives, and should also be of interest to recreational divers in general, because it covers the responsibilities that buddies have towards each other.

David Smart, MD, FACEM, FIFEM, FACTM, FAICD, Dip DHM is Medical Co-director, Department of Diving and Hyperbaric Medicine, Royal Hobart Hospital

Key words
Video (and DVD) reviews, recreational diving, legal and insurance, PADI, general interest

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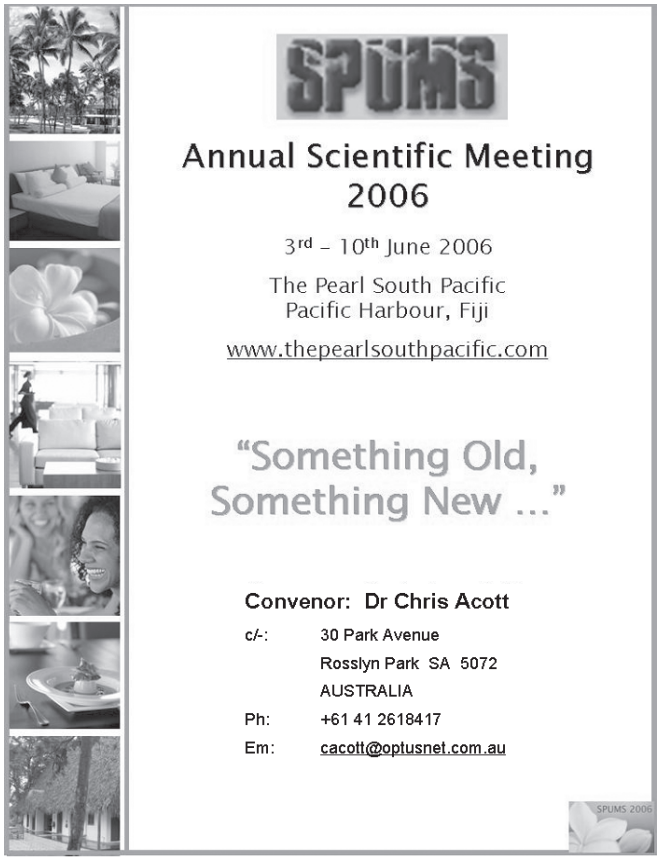
Diving-related fatalities resource

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 Stickybeak. 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.



SPUMS Journal CD

The SPUMS Journal, volumes 1-30, is available on CD.

To read and print these documents Adobe Acrobat Reader (version 3 or later) is required. This may be downloaded free of charge from the Adobe web site <www.adobe.com>.

The CD is available to members for Aust $25 (incl. GST or overseas mailing). The cost to non-members and institutions is Aust $90 inclusive.
Cheques or money orders should be made payable to:
'South Pacific Underwater Medicine Society'.
Credit card facilities are not available for this.

Contact: Steve Goble, Administrative Officer
E-mail: <stevegoble@bigpond.com.au>

INTERNATIONAL CONGRESS ON DIVING & HYPERBARIC MEDICINE, MUSCAT 2006
Hosted by the Royal Navy of Oman

Dates: 2 to 7 December 2006
Venue: Muscat, Oman

International Faculty includes Professors David Elliott, Des Gorman and Richard Moon

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For additional information:
Sultanate of Oman, Said Bin Sultan Naval Base, Medical Center, Royal Navy of Oman
PO Box: 839, Postal Code: 111 Muscat
Phone: +968-26-346832
Fax: +968-26-346367
E -mail: <drhassan@dhm.org.om>
Website: <www.dhm.org.om>

HYPERBARIC TECHNICIANS and NURSES ASSOCIATION
14th ANNUAL SCIENTIFIC MEETING
Hosted by the Townsville Hospital Hyperbaric Unit

Dates: 24 to 26 August 2006
Venue: Jupiters, Townsville

Registration/Enquiries:
<TSV-HTNA2006@health.qld.gov.au>
Phone: +61-(0)7-4796-2080
Fax: +61-(0)7-4796-2082

SPUMS ANNUAL SCIENTIFIC MEETING 2007

Preliminary Announcement

Dates: 16 to 22 April 2007
Venue: Oceans Resort, Tutukaka, Northland, New Zealand
Co-convenors: Mike Davis and Simon Mitchell

Guest Speaker
Dr Neal Pollock, Duke University
Theme
From mountain high to ocean deep
The physiological challenges of extreme environments

EUROPEAN UNDERWATER AND BAROMEDICAL SOCIETY

32nd Annual Scientific Meeting 2006

Dates: 23 to 26 August 2006
Venue: Radison SAS Hotel Norge, Bergen, Norway

Scientific Secretary: Prof Einar Thorsen, Haukeland University Hospital, Bergen
For additional information contact:
FJELL OG FJORD KONFERANSER AS
E-mail: <silje@fjellogfjord-konferanser.no>
Website: <www.eubs.org>

UNDERSEA and HYPERBARIC MEDICAL SOCIETY

Annual Scientific Meeting 2006

Dates: 22 to 24 June 2006
Venue: Hilton in the Walt Disney World Resort
Orlando, Florida
For additional information:
Lisa Wasdin
c/o Undersea and Hyperbaric Medical Society
PO Box 1020, Dunkirk, Maryland 20754, USA
Phone: +1-410-257-6606 extn 104
Fax: +1-410-257-6617
E-mail: <lisa@uhms.org>

ANZ COLLEGE OF ANAESTHETISTS ANNUAL SCIENTIFIC MEETING

Preliminary Notice

Date: 13 to 17 May 2006
Venue: Adelaide Convention Centre, South Australia
Hyperbaric Special Interest Group session
- Iatrogenic cerebral arterial gas embolism
- Cost analysis of HBOT

For further information contact:
Dr Margaret Walker <margaret.walker@dhhs.tas.gov.au>

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE COURSES 2006

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Basic	26/06/06	to	30/06/06
Advanced	03/07/06	to	07/07/06

October/November 2006

Basic	23/10/06	to	27/10/06
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DMT Full Course
October 2006 3 weeks, 9/10/06 to 27/10/06

DMT Refresher Course
To be announced

For further information or to enrol contact:
The Director, Hyperbaric Medicine Unit
Royal Adelaide Hospital, North Terrace
South Australia 5000 or
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lmirabel@mail.rah.sa.gov.au>

4TH KAROLINSKA POST GRADUATE COURSE IN CLINCAL HYPERBARIC OXYGEN THERAPY

Dates: 26 to 28 April 2006
Venue: Karolinska University Hospital, Stockholm, Sweden

Online information and registration: <http://www1.stocon.se/karolinskaHBO/9/34386.asp>

For additional information contact the Secretariat:
Stockholm Convention Bureau AB
Box 6911
SE-102 39 Stockholm, Swedend
Phone: +46-8-5465-1500
Fax: +46-8-5465-1599
E-mail: <confirmation@stocon.se>

DUIKMEDISCH CENTRUM MEDICAL ASPECTS OF DIVING ACCIDENTS AND ILLNESSSES

Dates: 16 to 20 October 2006
Venue: Diving Medical Centre, Royal Netherlands Navy

For information contact::
Diving Medical Centre, Royal Netherlands Navy
P O Box 10.000,
1780 CA Den Helder, The Netherlands
Phone: +31-223-653214
Fax: +31-223-653148
E-mail: <mj.veen@mindef.nl>

Instructions to authors

(revised June 2005)

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 Word 97 for Windows. 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 be subdivided into the following sections: an **Abstract** of no more than 250 words, **Introduction**, **Methods**, **Results**, **Discussion**, **Acknowledgements** and **References**. Acknowledgments should be brief. References should be in the format shown below. 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.

The preferred length for original articles is 3,000 words or less. 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 10 references. Abstracts are also required for all case reports and review papers. Letters to the Editor should not exceed 500 words (including references, which should be limited to five per letter). 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.
Tables are to be in Word for Windows, tab-separated text rather than using the columns/tables option or othersoftware and each saved as a separate file. They should be double-spaced and each in a separate file. No vertical or horizontal borders are to be used.
Illustrations and figures should be in separate files in TIFF or BMP format. Our firewall has a maximum size of 5 Mb for incoming files or messages with attachments.
Photographs should be glossy, black-and-white or colour. Posting high-quality hard copies of all illustrations is a sensible back-up for electronic files. Colour is available only when it is essential and may be at the authors' expense. Indicate magnification for photomicrographs.

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.

References
The Journal reference style is the 'Vancouver' style *(Uniform requirements for manuscripts submitted to biomedical journals*, updated July 2003. Web site 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 and 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 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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DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1-800-088-200 (in Australia)
+61-8-8212-9242 (International)
The toll-free number 1-800-088-200 can only be used in Australia

NEW ZEALAND
0800-4-DES111 or 09-445-8454 (in New Zealand)
+64-9-445-8454 (International)
The toll-free number 0800-4-DES111 can only be used in New Zealand

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.

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.

PROJECT PROTEUS

The aim of this investigation is to establish a database of divers who dive or have dived with any medical contra-indications to diving. At present it is known that some asthmatics dive and that some insulin-dependent diabetics dive. What is not known is how many. How many with these conditions die is known. But how many dive safely with these conditions is not. Nor is the incidence of diving accidents in these groups known.
This project is under the direction of Dr Douglas Walker and Dr Mike Bennett. The investigation has been approved by the Ethics Committee of the Prince of Wales Hospital, Randwick, approval number 01/047.

If you are in such a group please make contact. All information will be treated as CONFIDENTIAL.
No identifying details will appear in any report derived from the database.

Write to: Project Proteus
PO Box 120, Narrabeen, NSW 2101, Australia.
E-mail: <diverhealth@hotmail.com>

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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.

CONTENTS

Diving and Hyperbaric Medicine Volume 36 No. 1 March 2006

Diving and Hyperbaric Medicine is indexed on EMBASE

Printed by Snap Printing, 166 Burwood Road, Hawthorn, Victoria 3122