# Diving and Hyperbaric Medicine

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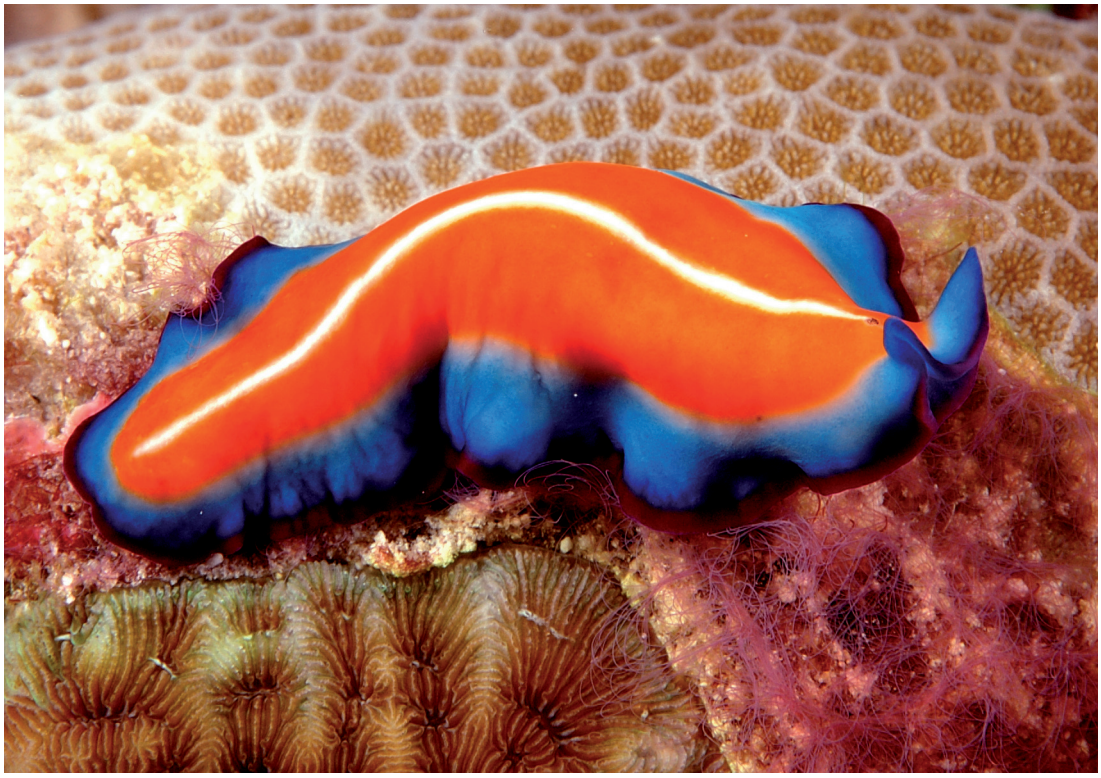

## Transcutaneous oxygen monitoring

**Project Stickybeak**

**Nitrox diving**

**More on ELTHI**

**Decompression sickness in caisson workers**

**Eulogy for Brian Hills**

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## PURPOSES OF THE SOCIETY

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

### OFFICE HOLDERS



### ADMINISTRATION



### MEMBERSHIP

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

Further information on the Society may be obtained by writing to:
SPUMS Membership,
C/o Australian and New Zealand College of Anaesthetists,
630 St Kilda Road, Melbourne, Victoria 3004, Australia
or e-mail <stevegoble@bigpond.com>
or go to <www.SPUMS.org.au>

**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.
There will be an additional surcharge of $8.00 for journal postage for all members living outside Australia.

# The Editor's offering

This issue has more of a hyperbaric medicine flavour than usual. However, the treatment of chronic wounds involves many branches of healthcare and represents a huge cost to the community. Anything that enhances healing in a cost-efficient manner is important, but we still do not know clearly the role for hyperbaric oxygen therapy (HBOT), or how best to identify those patients most likely to benefit.

The review of transcutaneous oxygen monitoring by Smart et al was originally part of a submission to the Medicare Services Advisory Committee (MSAC) in Australia. It has been substantially updated and will prove a valuable reference document for some years to come for hyperbaricists, vascular surgeons, diabetologists and others dealing with chronic wounds. It will be available on the SPUMS website in the public domain for downloading.

The interim report on the multicentre chronic wound study based at the Prince of Wales Hospital, Sydney, also stems from the MSAC review of HBOT. This is far from being a Level 1 RCT study, but will provide useful clinical data on what is happening in Australia. It is regrettable when clinical research is driven by fiscal and policy imperatives, but this is the case here. Glen Hawkins received his SPUMS Diploma for this work.

Douglas Walker has finally been able to gather sufficient data to report the 1999 Australian fatalities (December 2005 issue) and now the year 2000 deaths. When the 2001 data are reported later this year, this will represent a 30-year record. This huge resource needs further analysis (see Douglas's letter) and there are probably several research theses to be had in delving into this remarkable body of work.

Michael Lang reviews the coming of age for oxygen-enriched air – 'nitrox' – diving. I remain unconvinced of the value of nitrox over air for recreational diving, though it may have benefits for employed divers in enhancing time on the job. None of the claims regarding well-being have been substantiated scientifically, the decompression sickness rates seem similar (0.006% for nitrox and 0.007% for air; Table 7, p. 92) and nitrox is no protection for a diver who runs out of air or has an uncontrolled rapid ascent – the two main causes of diving fatalities.

**The future of *Diving and Hyperbaric Medicine***

Hyperbaric medicine is slowly coming of age. Most recompression chambers that treat divers spend much of their time treating other conditions. SPUMS has a sub-committee, the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG), that has served for over 15 years as the non-governmental organisation in Australia over issues such as Medicare payments for hyperbaric oxygen therapy. This committee consists of the medical directors of the hyperbaric units in Australia and New Zealand, but now welcomes all physicians involved in hyperbaric medicine to its annual gathering, which is usually held at the same time as that of the Hyperbaric Technicians and Nurses Association, with which the Society has close links.

Linkage with its peers is important for this Society. The SPUMS Diploma is one of the pathways towards the ANZ College of Anaesthetists Certificate in Diving and Hyperbaric Medicine. There have been approaches in recent years from both the European Underwater and Baromedical Society and the Undersea and Hyperbaric Medicine Society in the States to consider closer ties, especially the amalgamation of our publications, and the holding of the UHMS meeting in Sydney in 2004 was very successful, thanks to Mike Bennett and his colleagues. Many hyperbaricists belong to more than one of the three societies, often to all of them. The renaming of this publication is a further step in our becoming less parochial.

Linked to this is the increasingly competitive world of medical publication. Research scientists need to publish in the mainstream peer-reviewed literature. Despite our Journal being indexed on EMBASE this remains a highly negative aspect of our ability to attract good research papers. This is a Catch-22, in that without the support of research scientists, especially within Australasia, we cannot be considered for the needed next step of full Medline citation, which is largely dependent on the quality and quantity of original research published in the Journal. Without the support and commitment of society members, many of whom publish elsewhere material ideally suited to *their* journal, then the long-term outlook for the 'SPUMS Journal' is uncertain.

At the same time it is vital that the Journal continues to meet the needs and interests of the society's membership, full and associate, drawn from every walk of medical life and the diving community. A past editor of the *BMJ* said that medical journals are first and foremost for their readers. The main focus of the Society has always been on diving physiology and medicine. It is important this remains so. However, we also need to cater to the needs of hyperbaric medicine.

As long as I am Editor, I will strive to ensure this journal has something of interest for everyone…but I cannot do this on my own! It is up to you, the members, to ensure that we have good 'copy' and that you contribute positively to the Annual Scientific Meetings. This issue is nearly two months late because of the limited number of submissions and the demands of peer review. As things stand at present, we may be able to produce only three issues in 2007.

So, the challenge is out there – support *your* Journal or it may disappear!

Michael Davis

**Front cover photograph of a polycladid flatworm, *Pseudoceros sp*., courtesy of Martin Sayer**

# Original article

## Provisional report on diving-related fatalities in Australian waters 2000

Douglas Walker

**Key words**
Diving deaths, scuba, breath-hold diving, diving accidents, case reports

**Abstract**

(Walker D. Provisional report on diving-related fatalities in Australian waters 2000. *Diving and Hyperbaric Medicine*. 2006; 36: 62-71.)
A total of 16 fatalities were identified from official sources as having occurred during 2000. Nine deaths were in association with freediving or snorkelling and five with scuba. There was also one death where surface-supply air was involved, and one where the equipment used was not clearly identified. The investigation of many of the first group did not, in fact, proceed to a coroner's inquest but the investigation documentation is available. The case summaries are presented with attention to the diver performance, equipment and medical factors. Important adverse factors are identified and discussed for each of these groups, with comments on possibilities for reducing the number of fatalities that occur. Many of the factors involved in these tragedies have recurred time and time again in the case histories reported over many years from Project Stickybeak.

### Introduction

Project Stickybeak is an ongoing investigation into Australian deaths associated with freediving, snorkelling and diving using scuba or surface-supply equipment, and has been running since 1972.[1–3] The case histories of 16 deaths associated with freediving, snorkelling, scuba and hookah diving in the year 2000 are reported here and summarised in Table 1.

The National Coroners Information System (NCIS) was searched and details of identified cases made available for this investigation by the State Coroners and NCIS. Not all deaths associated with snorkelling are notified to coroners for investigation by the police. Therefore, several additional sources of information needed to be interrogated including the media. Fortunately the Queensland Workplace Health and Safety (diving) department provides information on such events in the Great Barrier Reef area even when they do not fall within its area of responsibility. A few cases are known only through notification by the police, private investigators, the Divers Alert Network Southeast Asia-Pacific (DAN SEAP), or through the media.

The basic purpose of coroners' enquiries is to determine who died and why, and to make comment on the lessons to be drawn from such tragedies. Access to the information resulting from police investigations on behalf of the local coroner is an essential element of investigations such as Project Stickybeak. This documentation often contains the only reliable information, as police 'incident' investigations now elicit most of the details needed to understand the factors involved in diving-related deaths. Despite this, data are frequently missing and autopsy reports are not always as detailed as would be expected. The evidence often contains a range of estimates on depths, distances, and the experience of those involved, while the available health history of the deceased is usually minimal at best. However, Australia is fortunate in having this official support for diving fatalities research. Therefore, Project Stickybeak reports inevitably contain an element of bias in determining the most likely course of events in an accident where the documentary data reflect variation in witness recall.

### Case reports

CASE BH 00/1

This 80-year-old lady, described as mentally alert and active despite some pain from past right hip and bilateral knee replacements, was with a group of retirees from overseas. She had met none of them previously. They were on a day trip to view the Barrier Reef and received the regular talk on snorkelling during the trip out to their destination, a pontoon at a reef. She wore a buoyancy vest for her first snorkel swim but, after making a trip in the glass-bottomed boat, did not wear one for her second swim. Although there were sometimes up to 200 people in the water under the watch of a crew member in a high chair, at this time there were only about 20 in the water. Her absence was not noticed till a count was taken before the boat left the pontoon. A determined search did not find her body. There is no information concerning her past health or whether she had ever previously used a snorkel, but she was said to have been a good swimmer. The safety watcher had not observed any unusual behaviour in any of those in the water and it is unknown whether she silently sank or drifted away. The cause of death is unknown.

*Summary*

ELDERLY; APPARENTLY FIT; HISTORY PAIN FOLLOWING BILATERAL KNEE REPLACEMENTS AND RIGHT HIP REPLACEMENT; SNORKELLING; NO BUOYANCY VEST; SILENT DEATH IN WATCHED, CALM SEA; BODY NEVER RECOVERED.

CASE BH 00/2

This 79-year-old man was also an overseas visitor making a day trip to the Barrier Reef. He appeared to be fit and after the instruction talk on snorkelling he did not declare any medical conditions. There was no indication that anything was wrong till the safety lookout saw a group of three snorkellers waving to catch his attention. When he reached them he found two were supporting the third, who was now unconscious. Attempts to resuscitate him were unsuccessful.

*Autopsy*

The autopsy showed an acute myocardial infarction.

*Summary*

APPARENTLY FIT; SNORKELLING IN CALM SEA NEAR OTHERS; SUDDEN DEATH; ACUTE MYOCARDIAL INFARCTION.

CASE BH 00/3

This 30-year-old man had been spear fishing for 15 years and was known to be capable of diving to a depth of 30 metres' sea water (msw). He was with a friend but they were about 60 metres apart to avoid interfering with each other's hunting. The buddy became cold and left the water after a time to sit on the beach. Although he could not see his friend this did not worry him, as the latter was wearing a black wetsuit and his float was black. It was only after about three hours that he became concerned and persuaded a man to take him out in a boat to search, which was not successful so the police were notified. The float was found but the line could not be pulled up as it was entangled in the kelp. It was now getting dark so it was not until the next day that the police divers could search for the body. The spear gun was recovered but the spear was not found. The body was never recovered.

*Comment*

This is a typical scenario for a post-hyperventilation ascent hypoxia drowning. There was a history of him having survived such an episode in the past, being fortunate to regain consciousness on the surface on that occasion.

*Summary*

BREATH-HOLD, EXPERIENCED SPEAR FISHERMAN; SOLO; PROBABLE POST-HYPERVENTILATION ASCENT HYPOXIA; BODY NEVER RECOVERED.

CASE BH 00/4

This 31-year-old man had no admitted ill health and, like his friend, was an overseas visitor. While the friend was an experienced breath-hold spear fisherman, he himself was described as 'not confident enough to [scuba] dive'. Indeed, his friend checked his ability to swim using a snorkel for about two hours before feeling satisfied it was safe to leave him and go ashore for a shower. When he returned an hour later he could not see his friend, then he was alerted to a dive boat which had found him floating unconscious. The victim failed to respond to resuscitation efforts.

*Autopsy*

The autopsy revealed the presence of significant coronary atheroma with vessel narrowing, the left anterior descending showing 80% narrowing at one place, which may have been a significant factor leading to his drowning. There was no evidence of a myocardial infarction.

*Summary*

INEXPERIENCED SNORKELLER; APPARENTLY FIT; SOLO; CALM SEA; SILENT DEATH; SIGNIFICANT CORONARY ATHEROMA; PROBABLE CARDIAC DEATH.

CASE BH 00/5

A 75-year-old woman and her husband were amongst the passengers on a day trip to view the Barrier Reef. There was a talk on safe snorkelling during the outward trip and there was a request for anyone with a health problem to discuss the matter with a crew member. Neither reported a health problem and the available records fail to provide any details. On arrival at the cay there was a further briefing and instruction in snorkelling offered, but it is not known whether this couple accepted this instruction. There were two safety lookouts watching over the 40 persons in the water off the beach, one on the boat and the other on the beach. When the latter decided to take the small tender out to assist some swimmers he first arranged for another to replace him on the beach. The water was calm but there was some current. He noticed one person, who was wearing a buoyancy vest, drifting face down among the other swimmers and snorkellers and went to investigate. The unconscious woman was quickly brought to shore but could not be resuscitated. It was suggested that she may have been floating unconscious for 15 minutes before this was noticed.

*Autopsy*

Unfortunately no autopsy report is available. However, the pathologist decided this was a case of drowning but offered no reason for it to have occurred in calm water in a person wearing a buoyancy vest and close to others, so it is not possible to evaluate whether there was a cardiac cause for her death.

**Table 1. Summary of diving-related fatalities**
**(BH – breath-hold, BSB – buddy separation before incident, GNS – group not separated,**

| Case | Age | Training | Experience | Dive group | Dive purpose | Depth (metres) Dive | Depth (metres) Incident | Weight belt On | Weight belt Kg |
|---|---|---|---|---|---|---|---|---|---|
| BH 00/1 | 80 | Nil | Some | Solo | Recreation | Not stated | Surface | None | n/a |
| BH 00/2 | 79 | Not stated | Not stated | GNS | Recreation | Not stated | Surface | None | n/a |
| BH 00/3 | 30 | Not stated | Experienced + | BSB | Spear fishing | 25 | Ascent | On | 6 |
| BH 00/4 | 31 | Nil | Some | BSB | Recreation | 9 | Surface | None | n/a |
| BH 00/5 | 75 | Not stated | Not stated | GSB | Recreation | Not stated | Surface | None | n/a |
| BH 00/6 | 27 | Nil | Not stated | Solo | Recreation | Not stated | Surface | None | n/a |
| BH 00/7 | 60 | Not stated | Not stated | GNS | Recreation | Not stated | Surface | None | n/a |
| BH 00/8 | 28 | Not stated | Not stated | GSB | Recreation | Not stated | Surface | None | n/a |
| BH 00/9 | 55 | Not stated | Not stated | Solo | Recreation | Not stated | Surface | None | n/a |
| SC 00/1 | 24 | Trained | Nil | BSB | Recreation | 21 | 17 | Ditched by buddy | 7 |
| SC 00/2 | 30 | Trained | Experienced | BSB | Cray fishing | 20 | Not stated | Not stated | ? |
| SC 00/3 | 27 | Trained | Experienced | Solo | Work | 9 | 9 | On | 6 |
| SC 00/4 | 30 | Trained | Experienced | GNS | Recreation | 54 | 54 | Ditched by buddy | ? |
| SC 00/5 | 29 | Trained | Experienced | BSB | Recreation | 10 | 10 | On | 10 |
| H 00/1 | 20 | Scuba | Experienced | Solo | Work | 9 | 12 | On | ? |
| X 00/1 | 31 | Scuba | Nil | Sepn | Work | Not stated | Not stated | On | Not stated |

*Summary*

SNORKELLING; NO HEALTH HISTORY; POSSIBLY NIL SNORKEL EXPERIENCE; WEARING BUOYANCY VEST; SILENT DEATH CLOSE TO OTHERS IN CALM SEA; GOOD LOOKOUTS SAW NO DISTURBANCE TO SUGGEST ANYONE IN TROUBLE; NO AUTOPSY REPORT BUT REPORTED AS DROWNING; POSSIBLE CARDIAC DEATH.

CASE BH 00/6

This 27-year-old man was a member of an overseas group of employees visiting Australia. The record is very limited concerning the lead-up to this fatality beyond the statement that he was snorkelling off a beach when he cried out for assistance. He was apparently alone at the time. His cry was heard by a witness who was mooring an empty dive tender 30 to 50 metres from him. She saw someone waving their arms and initially thought it was to people on the beach, then realised he might be in trouble and alerted the lifeguard on the beach. Each then swam out to him to give assistance. As he was swimming out, the lifeguard observed the person appear to have a convulsion and his body come half out of the water. When reached, the person was unconscious, cyanosed, and floating face down. They placed him on a surf ski and brought him back to the beach. He failed to respond to resuscitation.

**in Australian waters in 2000**
**GSB – group separation before incident, H – hookah, SC – scuba, ? – unknown, )**

| Bouyancy vest | Remaining air | Equipment Tested | Equipment Whose | Comments |
|---|---|---|---|---|
| Off | n/a | n/a | Dive boat | Possible cardiac death. Body never recovered. |
| Nil | n/a | n/a | Dive boat | Cardiac death. |
| Nil | n/a | n/a | Own | Probable post-hyperventilation blackout. Body never recovered. |
| Nil | n/a | n/a | Hired | Cardiac death. |
| On | n/a | n/a | Dive boat | Drowned. Possible cardiac death. |
| Nil | n/a | n/a | Hired | Drowned. |
| Nil | n/a | n/a | Dive boat | Acute heart failure. |
| Off | n/a | n/a | Dive boat | Drowned. Epilepsy. |
| Nil | n/a | n/a | Not stated | Drowned. Possible cardiac death. |
| Not stated | Nil | Fault | Dive shop | Drowned. Inexperienced. Fatigue. Cold. Buddy breathing failed. Out of air. |
| Not stated | Not stated | Fault | Own | Body never recovered. |
| Nil | Not stated | Nil to test | Employer | Drowned. Commercial diver. No wetsuit. No lifeline. Fatigue. Body never recovered. |
| Not stated | ++ | NAD | Borrowed twin set | Cerebral arterial gas embolism (CAGE). |
| Not stated | + | NAD | Hired | Drowned. Aborted dive as “felt uncomfortable”. Health? |
| Nil | n/a | Fault | Employer | Drowned. Wave swamped boat. Regulator separated from hose. |
| Not stated | n/a | Fault | Employer | Possible seizure condition. |

*Autopsy*

At autopsy, his heart and coronaries were healthy, and the cause of death was given as drowning. The pathologist offered no comment concerning the cause of the ‘convulsion’.

*Comment*

There is a total absence of any information concerning his swimming and snorkelling experience or where the other members of his group were at this time. The sea conditions were described as far from inviting as it was raining, overcast, windy, with a 0.8 metre swell, so his decision to enter the water alone is strange.

*Summary*

SNORKELLING; UNKNOWN HEALTH AND SNORKEL HISTORY; SOLO; ADVERSE SEA CONDITIONS; SUDDEN CRY FOR HELP; CONVULSION THEN UNCONSCIOUS; DROWNED.

CASE BH 00/7

While visiting a Great Barrier Reef island with her daughter, this 60-year-old lady joined a snorkelling trip to a reef pontoon. There was no history of ill health according to her daughter and she appeared to be behaving in a normal manner till she stood up on some coral and said she was

feeling short of breath. She was noticed to have lost most of her normal colour so those nearby called the glass-bottomed boat to collect her. Arrangements were made to transport her by a float plane to the nearby resort island but during transfer she stopped breathing and expired-air breathing was started. Circulatory arrest ensued and cardio-pulmonary resuscitation was commenced, but she failed to respond. There is an alternative report of the incident which describes the onset of her symptoms being as she rested on the pontoon after her swim.

*Autopsy*

The autopsy showed there was 60% narrowing of the left anterior descending coronary artery but no evidence of thrombus. There was histological evidence suggestive of chronic myocardial ischaemia. There was marked thickening of both cusps of the mitral valve with ballooning of the anterior cusp. The lungs were moderately congested and oedematous.

*Comment*

This being regarded as a 'death from natural causes' there was no coronial involvement, and no further investigation took place. Death appears to have been due to acute cardiac failure. It is possible that immersion pulmonary oedema was a factor, initiating the fatal conclusion, but this was not recorded as having been considered.

*Summary*

SNORKELLING; APPARENTLY HEALTHY WOMAN; ONSET BREATHLESSNESS LEADING TO CARDIAC ARREST; ACUTE CARDIAC FAILURE; CORONARY ATHEROMA AND MYOCARDIAL ISCHAEMIA.

CASE BH 00/8

This 28-year-old man had a history of epilepsy, which he claimed was well controlled by his medication. He had declared this condition when booking his 'adventure holiday' trip. He was in a group which had gone white-water rafting, mountain biking, and backpacking before commencing this 'scuba and snorkel' trip to visit the Great Barrier Reef. They were on a motor schooner and received the regular safety talks. The diving instructor on the boat refused his request to undertake a 'resort dive' but found nothing in the rules which required him to be prohibited from snorkelling. He claimed to have made a number of scuba dives previously, but this did not dissuade the instructor from his decision concerning the 'resort dive'. He was not wearing a buoyancy vest, a decision permitted because it would have prevented him from leaving the surface. Although all had been told to dive or swim with a buddy he had separated from his. He had been in a group of others but none had noticed any disturbance, though one noticed his stillness 'but was reassured when he saw him start kicking'. There was a safety watch of those swimming near the boat and when he was first noticed to be too still in the water it was thought he was taking underwater photographs. Then he was seen to be drifting with his arms and legs hanging down. The instructor quickly entered the water to check the situation and the skipper brought the safety tender. They found he was unconscious, face down, with his snorkel still in his mouth. It was difficult to get him into the tender as he was described as 'a large man' and it was necessary for them to use a harness to pull him into the tender. Resuscitation attempts were unsuccessful.

*Autopsy*

The autopsy showed drowning in a healthy person, assumed to be as a result of an epileptic episode.

*Comment*

Wearing a buoyancy vest would not have prevented him from drowning as it would have floated him face down. The safety watchers cannot be faulted as there was no disturbance apparent to draw attention to him. Although his full medical history is not known there was a holiday history of recent strenuous physical exertion without problems so his acceptance as a snorkeller can be defended.

*Summary*

SNORKELLING; EPILEPTIC WELL CONTROLLED ON MEDICATIONS; KNOWN GOOD EXERCISE TOLERANCE; SILENT DROWNING AMONG OTHERS DESPITE GOOD SAFETY WATCHERS; PROBABLE EPILEPTIC FIT.

CASE BH 00/9

This 55-year-old man had an unfortunate family medical history, his father and brother having both died from heart disease. He had recently been started on anticoagulants for his hypertension but he appeared to be healthy. No details are available about his experience or ability as a swimmer or snorkeller. His family were sitting on the beach watching him snorkel round a wreck and occasionally waving to them. Then they noticed he was floating without moving and became alarmed. His son paddled out on an inflatable raft but was unable to drag him onto it. The police were notified and the rescue helicopter arrived about 35 minutes later and dropped a diver who brought him back to the beach. Resuscitation efforts were unsuccessful. His mask was up on his forehead when he was reached.

*Autopsy*

The autopsy showed mild atheroma in the right coronary artery but 70–80% narrowing in the left, with patchy myocardial fibrosis lesions. However, there was no evidence of acute myocardial infarction so his death was attributed to drowning, although possibly from an initial cardiac factor.

*Comment*

The fact that his mask was on his forehead when first found is a possible marker for a panic reaction or awareness that he was in some kind of trouble.

*Summary*

SNORKELLING; SOLO; SUDDEN SILENT DEATH; SIGNIFICANT CORONARY ARTERY NARROWING; DROWNING; POSSIBLE CARDIAC FACTOR.

CASE SC 00/1

It is particularly tragic and ironic that this 24-year-old woman was making an escorted dive intended to help her improve her confidence and ability, a present from her boyfriend. She had trained overseas in warm waters and made two subsequent dives, during one of which she had experienced problems. The dive leader was a divemaster and was aware of her inexperience. Her assigned buddy was a slightly more experienced diver, a person who had taken an advanced diver course. The dive shop supplied all their equipment and they were told about the proposed dive, water entry to be from a rock ledge followed by a snorkel out with inflated BCDs before descending. The area was described as being shallow and protected. This would be her first 'rock entry' dive. The weight on the belt was an estimate made in the shop and no buoyancy check was made. They were told to indicate when their contents gauge reading fell to 100 bar pressure.

There were nine customers with a divemaster as dive leader, and a trainee divemaster as support. The dive leader, who was buddying a diver known to be liable to stray, was in the lead and the others followed in buddy pairs in line behind him, with the trainee divemaster acting as a back marker. The water was described as rough at the entry point, visibility 'not good', and cold. A police witness later said he believed the water conditions were too rough for snorkel swimming. There was a current against them during their outward underwater swim and this, in combination with her inexperience, led her to use her air faster than her buddy and most of the other divers. The dive leader split the group when she showed him that her remaining air was down to 100 bar. She and one other were to return to shore with the 'back marker', then her buddy decided it was the correct thing for him to remain with her although still having about 160 bar. They were slowly ascending on their return swim and were at about 12–13 msw when her 'low air' situation became acute. Her buddy offered his 'octopus' regulator to her and they began to buddy breath, sinking down to the sea bed at 21 msw as they did so.

After a short time the buddy's contents gauge bleeped, warning of a critically low state. They were holding onto each other while he was attempting to drop her weight belt when his contents gauge bleeped a warning he was about to run out of air. He dropped his own weights and started a low air/out-of-air ascent, the two becoming separated at this time. His ascent technique was fortunately successful and he suffered no serious ill effects, though required treatment for salt-water aspiration. His friend failed to surface and her body was not found for three days. The weight belt was off when she was found. It is apparent that he had managed to ditch her weight belt, but her wetsuit was older than the one he was wearing and provided less buoyancy so she remained on the sea bed. The reason he had not recognised his seriously low air situation was because his gauge was reading 25 bar too high, a fact only discovered later.

*Autopsy*

The cause of death was given as drowning. It should be noted that the body was not recovered for three days.

*Summary*

SCUBA DIVING; TRAINED; INEXPERIENCED; CURRENT AND ROUGH WATER AFFECTED RATE OF AIR USE; OLDER WETSUIT LESS BUOYANT, NO BUOYANCY CHECK BY DIVE SHOP; LOW-AIR STATE THEN OUT OF AIR SO BUDDY BREATHING; SEPARATION WHEN BUDDY OUT OF AIR; BUDDY'S CONTENTS GAUGE INACCURATE.

CASE SC 00/2

Both this 30-year-old man and his brother were trained and experienced scuba divers, and on this occasion were diving for crayfish off a small island. The sea was calm, there was some tide flow, and the visibility was poor, only two metres. They anchored in 20-metre deep water and started hunting separately in the rock crevices. These were filled with kelp. After about half an hour of separation the buddy began to worry about his brother's failure to return to their boat and made a search for him. Both this and all subsequent searches were unsuccessful and his body was never found, so the reasons for his death are unknown.

*Summary*

SCUBA DIVING; EXPERIENCED DIVER; DIVING IN KELP FOR CRAYFISH; BUDDY SEPARATION; BODY NEVER FOUND.

CASE SC 00/3

This 27-year-old professional diver was employed to lead a three-man team tasked with repositioning cyclone moorings. Due to the limited baggage capacity of the plane in which they travelled to join the base ship of this deployment, they were unable to take their wetsuits or other diving equipment except for the Kirby Morgan unit owned by this diver. When they arrived they found that the company's Kirby Morgan unit had been damaged because of poor care and his set was

to be used. There were no wetsuits so they were to wear overalls while diving, the voice communication equipment did not work, and they were limited to one surface-supplied breathing apparatus (SSBA) and only one scuba set as only a reserve compressor was operating at the time. No head protection was available as would have been required for the proposed tasks. Their job was to unshackle the anchor chain from the anchor shanks and attach a lifting cable. It was necessary to uncover the chain from the bottom material and the job was undertaken with each of his two fellow divers in turn singly, using first SSBA then scuba. The victim was using scuba on the fatal dive. The sea had become rough and they had to dive from a dinghy. The line tender became alarmed when he saw the line start to drift away, then he found it had become detached from the diver. After a delay, due to having to return to the support tug to get the SSBA, a search was made. This was supplemented by a surface search for bubbles, but neither the diver nor his equipment was ever located.

*Comment*

The reason for this accident is unknown but before his final descent he was noticed to be breathless and showing signs of tiredness. The work depth was insufficient to implicate narcosis but fatigue and possibly hypothermia may have affected his behaviour. Lack of proper equipment for the job was clearly a contributing factor – no wetsuit, unattached lifeline, no buoyancy aid, no protective helmet, no voice communication to the surface, no retention strap on his regulator. All these matters were required to comply with commercial diving regulations. Witnesses considered him unfit before final dive descent. Because neither the diver nor any of his equipment was recovered it is not possible to determine whether the problem was contaminated air, a head injury from the lifting equipment, a health factor, or simple loss of the regulator from his mouth.

*Summary*

EXPERIENCED DIVER; SOLO; SUBSTANDARD EQUIPMENT; DISCONNECTED LIFELINE; FATIGUE FACTOR; DIVER ACCEPTED USE OF POOR EQUIPMENT SO EMPLOYER ESCAPED LEGAL LIABILITY FOR THE DEATH; BODY NEVER RECOVERED.

CASE SC 00/4

The victim, aged 30, and her husband had trained overseas three to four years previously and subsequently made about 200 dives, over 50 being deeper than 40 msw, their deepest to 46 msw. A friend invited them to make a wreck dive with him and his buddy with whom he had made a number of dives on this wreck. It was a deep dive, 55 msw, and he took care to warn them of the possible dangers of such a depth. He noted they had single tanks so loaned each of them twin 72 cu ft tank sets, with separate regulator on each tank, and stressed that they should abort the dive at any time they wished to do so. It is not known if they had ever previously used such sets. It was planned for them to ascend before the more experienced divers, though the latter were to descend first. Her descent was delayed by her difficulty in venting air from her drysuit.

When they reached the wreck she appeared to lack her usual composure so their friend, despite the 'OK' sign she gave him, decided to abort the dive and return to the surface with her. He held her wrist, then he checked the position of the anchor line, and when he looked back he saw she had lost consciousness. He immediately inflated her BCD, then found she was tethered by her secondary regulator to the wreck's aerial so he ditched her weight belt and the regulator pulled free, the latter just missing her husband as it did so. A rapid ascent followed and when they were about half way to the surface she had two convulsions and went limp.

The friend attempted to slow their ascent rate by venting her BCD but was unsuccessful, the air expansion rate exceeding the venting. At 15 msw he chose to let her go and make a computer-gauge-regulated decompression stop before continuing his ascent to the surface. There he saw her floating face up. The others helped him to get her into the boat and commence CPR but there was no response.

*Autopsy*

A CT scan before the autopsy showed the presence of air in the right side of the heart and also in her thighs. A small tumour of no clinical importance was noted in the liver.

*Comment*

The convulsion as she ascended may have been evidence of a cerebral air embolus. The reason for her sudden loss of confidence and then of consciousness at depth is unknown but cold water and narcosis may have played a part.

*Summary*

SCUBA DIVING; TRAINED; EXPERIENCED TO 46 MSW; DEEP WRECK DIVE TO 55 MSW; COLD, DARK AND NARCOSIS FACTORS; LOSS OF CONSCIOUSNESS AT DEPTH; EQUIPMENT CAUGHT ON WRECK; UNCONTROLLED ASCENT AFTER BUDDY INFLATED HER BCD AND DITCHED WEIGHT BELT; CONVULSION DURING ASCENT; GOOD ASSISTANCE FROM OTHERS; CEREBRAL ARTERIAL GAS EMBOLISM.

CASE SC 00/5

This 29-year-old man had been scuba diving for 13 years, though not in the previous six months, and his buddy had only slightly less experience. The buddy hired the equipment for the day. They made their water entry off a rocky shore

and swam out beyond rough water before they descended, the depth being about 10 msw. A short time later, the victim indicated he wanted to ascend and at the surface, where the water was choppy, he said "I don't feel comfortable". They held onto a cray pot float while his buddy talked to him to help calm him. After a time he said he felt OK and they again descended. At a depth of 3 msw he changed his mind and again surfaced, saying "I can't do it, I have to get out". He was so intent on this that he ignored his buddy's advice to swim to the nearest safe exit area a short distance away, instead swimming to the closest rocky ledge. The water here was a turbulent surge over the rocks but the buddy made a safe exit. When he looked back he saw his friend floating on his back out to sea and imagined he had changed his mind about trying to exit here. After taking off his equipment he went and found a surf lifesaver and asked that a 'rubber ducky' be sent to collect his friend. When he was reached they found he was unconscious and started CPR as soon as they brought him ashore. He did not respond to resuscitation.

*Autopsy*

The autopsy revealed evidence of drowning and the presence of non-significant localised subarachnoid haemorrhage over his left cerebral hemisphere. The latter observation was in keeping with a history of a motorcycle accident at the age of 18, which had caused a left parietal skull fracture complicated by a subdural haemorrhage needing surgical evacuation. A more recent back injury was blamed for his occasional back pain and 'migraine headaches'. His only recent visits to his doctor had concerned a headache and 'sinus symptoms'.

*Comment*

His true swimming and scuba ability is unknown, as is whether he found the sea conditions more severe than he felt competent to meet and for this reason he panicked, failing to inflate his BCD or ditch his weight belt in the process and drowned. His wife reported he had two nocturnal episodes of chest tightness during the previous two weeks, their significance unknown.

*Summary*

SCUBA DIVING; TRAINED AND EXPERIENCED; NIL DIVING FOR SIX MONTHS; ABORTED DIVE; UNSAFE CHOICE OF EXIT AREA; PROBABLE PANIC REACTION TO SEA CONDITIONS; FAILED TO DROP WEIGHTS OR INFLATE BCD; DROWNED.

CASE H 00/1

This 20-year-old man was, like many employed in the harvesting of *beche de mer*, untrained in hookah diving and had never had a 'diving medical'. The diving system employed was for the boat to tow three dinghies from each of which, depending on the depth, a diver would breath-hold dive or use hose supply from a compressor in the dinghy to collect the *beche de mer*. There was doubt concerning the reliability of these compressors such that the skipper had his personal one. It was known that the outboard engine of the dinghy from which the victim was diving was difficult to restart but was reliable once started. There was a second diver with him but when he knew the local depth was 20–25 msw he said it was too hard to breathe at that depth and transferred to another boat to cadge a light for a cigarette. It was a short time later that the accident occurred. A large wave nearly swamped the dinghy and while the crewman was bailing the water out the diver decided to start his dive and jumped out. This made the bow rise and water come in over the stern. The crewman then noticed the hose to the diver was trapped between the transom and the outboard's leg. It was necessary to lift the engine out of the water to free it, but it would first be necessary to stop it as it would run too hot if the inlet for its water cooling was out of the water. Fortunately a diver from another dinghy came and got it free and he was required only to let the engine idle.

While this was happening the boat drifted and suddenly the hose went slack and spun around in the water showing that there was no longer anything connected to it. The crewman checked that the diver had not surfaced, then gave the alarm. The skipper, who had been in one of the other dinghies, checked he had enough fuel for his compressor, then descended to search for him. His first three dives were unsuccessful before he found the victim on the sea bed, his weight belt on and catch bag and regulator lying nearby. He ditched the belt and brought the drowned diver to the surface. Depth here was 10–15 msw.

*Autopsy*

The finding was of drowning without evidence of any medical disease or barotrauma.

*Comment*

The lack of training or experience with hookah, whilst not the critical element leading to this death, may have been contributory. It is apparent that the victim either never thought to ditch his weight belt or was so shocked by the loss of his air supply and contact with the surface that he inhaled water before considering this option. The air compressor had many adverse features but these were not implicated in the tragedy. There were three critical issues in addition to his lack of training: he was not wearing a bailout bottle, he had no lifeline, and there was an insecure coupling between his supply hose and his regulator unit. This last fault made it impossible to treat the hose as a lifeline as it separated when so used in an attempt to pull the diver to the surface. The legal responsibility of the company which ran this particular harvesting of *beche de mer* is a moot point, because the ownership of the compressor was disputed and the divers were described as being self-employed. The absence of

enforcement of workplace safety requirements certainly deserves attention.

*Summary*

HOOKAH; UNTRAINED BUT REPORTEDLY EXPERIENCED SSBA DIVER; CONNECTION HOSE TO REGULATOR UNIT INSECURE; HOSE CONNECTION SEPARATED; NO BAILOUT BOTTLE; NO LIFELINE; FAILED TO DITCH WEIGHT BELT; SAFETY REGULATIONS IGNORED BY DIVER'S EMPLOYER AND GOVERNMENT; DROWNED.

CASE X 00/1

This 31-year-old diver had completed his basic recreational scuba training course less than three months before he commenced employment with this pearl farming enterprise. He had received no instruction and there was no supervision of him on this his first day at work. There were three divers employed to attach pearl panels to an underwater fence line. He apparently experienced some difficulty with his regulator and entered the water last but appeared to be working in a normal manner when seen from time to time by the other divers. His failure to join the other divers when they completed their tasks led them to check on him and to find him on the sea bed. The incomplete nature of the information available makes it impossible to be certain whether he was using scuba or SSBA equipment.

*Autopsy*

The autopsy reportedly noted he had a congenital brain abnormality which may have predisposed him to having a seizure but further details are not available. This was possibly his first use of hookah diving apparatus. The firm was prosecuted, pleaded guilty, and was fined $10,000.

*Summary*

HOOKAH OR SCUBA – UNKNOWN; UNTRAINED; NIL EXPERIENCE OR INSTRUCTION; NEW EMPLOYEE; SEPARATION UNDERWATER; POSSIBLE MEDICAL CAUSE FOR SEIZURE FOUND AT AUTOPSY; DROWNED.

**Discussion**

SNORKEL USERS AND BREATH-HOLD DIVERS

These fatalities fall into two clearly defined types, either breath-hold dives spear fishing (BH 00/3) or swimming while wearing a mask and snorkel (the remainder). The critical factors in the two groups clearly differentiate them. The cause of death of the spear fisherman was probably post-hyperventilation hypoxic syncope resulting in drowning. The danger of hyperventilation to increase one's underwater duration is well documented, but such deaths are sufficiently infrequent to be discounted by divers. Unfortunately survival from a non-fatal blackout incident does not appear to be persuasive in teaching avoidance of 'excessive' hyperventilation.

The larger group consisted of swimmers using snorkels, often for the first time, visitors from out-of-State. These deaths occurred, in the majority of recent instances, despite alert safety watchers. These cases indicate both the serious difficulty of recognising a swimmer in trouble in a crowd of others, in particular when there is no outward sign of any problem, and the problem of sudden death in the apparently healthy. Although two of this group had obtained and worn buoyancy vests initially, one had removed her vest before making her last water entry. As these vests tend to float the wearer face down, they have a limited safety function in an unconscious wearer. If they were designed to keep the wearer face up they would be unsuitable for anyone trying to swim and examine the underwater world.

The most common contributing health factor was cardiac with four, possibly five, dying from this cause. It is probable that it was also critical in the fatality where the body was never found. Whether a person with an epileptic history should be permitted to swim, even if accompanied by a conscientious buddy, is a contentious problem and any decision involves consideration of the risk versus quality-of-life factors. It is certainly easier to have a blanket prohibition on all epileptics swimming, but the 'evidence basis' justifying any decision is debatable: even the most healthy can drown. No explanation has been offered for the sequence of events in case BH 00/6 but adverse sea conditions, his possible first use of a snorkel, and being solo, would explain his panic and have resulted in the commotion the witness labeled as 'a convulsion'.

The problem of death among snorkel-swimming visitors to the Great Barrier Reef is a major concern to those involved in taking visitors to view the reef and to tourism authorities generally. It is not clear what action can be taken to effectively reduce these fatalities, as a large proportion are in the age group where unpredictable cardiac events are most common. The majority of victims appeared to be healthy, though a few had failed to reveal their true health history.

The wearing of the type of life jackets suitable for snorkel swimming is of negative benefit should loss of consciousness occur. It is apparent that even the most conscientious safety watch of a group in the water will fail to benefit those who lose consciousness without any outward sign, the only alerting factor being the absence of activity for longer than expected. The safety watchers employed to supervise the safety of swimmers and snorkellers on trips to the Barrier Reef appear to have acted efficiently and are to be commended for the difficult task they perform. The organisations running day trips to the Barrier Reef appear to be taking active steps to ensure efficient safety watch procedures. Whether there is a need to require watchers to hold certificates in life saving is not proven on the evidence

presently available, though boats which carry defibrillators ensure those likely to use them have training. As it would be neither practical nor necessarily effective to require a specialist cardiologist to examine all visitors aged over 45 who intend to snorkel swim on the reef, it may be necessary to accept that such deaths are unavoidable until new predictive tests are developed.

SCUBA DIVERS

There were five fatalities identified in association with scuba diving, of which one was due to pulmonary barotrauma/air embolism as indicated by autopsy findings and the clinical history. In one case there was a low-air factor, while the other two divers had adequate remaining air. Inadequate experience and panic are once again apparent.

In case SC 00/3 an unwillingness to risk job loss is the most probable explanation of the number of work-safety violations found in this accident, and indeed helped to protect the employer from the legal consequences of their failure to follow 'best practice' workplace management. In the absence of the victim's body and equipment it is not possible to know the critical factor in this death.

While in case SC 00/4 the diver had shown symptoms suggestive of CAGE during her ascent, she had lost consciousness prior to this for reasons that were never defined but probably included anxiety due to this being her deepest dive, nitrogen narcosis, the cold water, and use of a borrowed twin-cylinder scuba unit. It is probable that this was the first time she had used such equipment, which had a separate regulator for each cylinder.

In the cases where drowning was the given cause of death, the circumstances of each were unique. The dangers of ignoring the 'nanny' advice on safe diving practices is demonstrated, as also the factors of panic, running out of air, and inexperience. The concordance of several adverse factors in these scenarios supports the common belief that the greater the failure to strictly observe advised safe diving procedure the less is the margin of safety.

Scuba diving-related deaths frequently show the presence of multiple risk factors. Rarely does a single adverse factor result in a fatality. Inexperience, absolute or relative, is a predictable factor and this includes those who have not dived recently, as are buddy separation and low-air situations. These breaches of good diving practice are best tackled through training protocols. Buddy breathing cannot be relied on as a safe and sure alternative to monitoring the contents gauge, even assuming the gauge is reading accurately.

SURFACE-SUPPLY SYSTEMS

There is no tradition of zero tolerance of unsafe conditions in the pearling industry, indeed from an examination of these two cases it appears workplace safety is ignored at all levels of the industry. In case X 00/1 complete lack of experience and a possible medical problem leading to a seizure were factors, but unfortunately complete details of this fatality are not at present available. There is clearly an immediate need for a simple 'cross-over' course for those wishing to take up employment in this industry when the only prior training has been recreational scuba diving and not hookah diving.

The examination of the deaths using hookah equipment clearly shows that using faulty equipment can be fatal, and that experience of recreational scuba diving is inadequate preparation for this type of diving. It is a sad fact that no action has been taken to require the completion of a certificated course for divers employed in the pearling, crayfish, and *beche de mer* industries, or enforce the requirement to hold a certificate for employment in this industry.

Permission to access data from the police investigations of these deaths on behalf of the local coroner is a vital element in any investigation into areas where intervention may reduce the occurrence of fatalities. Where such investigations take a particular note of the medical history, training, experience, and equipment factors their value is enhanced. One area of difficulty arises where a snorkeller, independent of a commercial enterprise, is decided to have died 'from natural causes' and, very naturally, the cases are not further investigated. Their omission from later reviews is unavoidable but there is nothing to suggest the critical factors in such cases differ from those here considered. The diving community is greatly indebted to coronial and other sources for their understanding and active support of this ongoing investigation.

**Acknowledgements**

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**References**

1 Walker D. *Report on Australian diving deaths 1972-1993*. Melbourne: JL Publications Ltd; 1998.
2 Walker D. *Report on Australian diving deaths 1994-1998*. Ashburton, Victoria: Divers Alert Network (DAN) S.E. Asia-Pacific Ltd; 2002.
3 Walker D. Provisional report on diving-related fatalities in Australian waters 1999. *SPUMS J*. 2005; 35: 183-93.

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# Review articles

## Transcutaneous oximetry, problem wounds and hyperbaric oxygen therapy

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**Key words**
Transcutaneous oximetry, wounds, chronic wounds, hyperbaric oxygen therapy, diabetes, review article

**Abstract**

(Smart DR, Bennett MH, Mitchell SJ. Transcutaneous oximetry, problem wounds and hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2006; 36: 72-86.)

**Introduction:** Transcutaneous oximetry measurement (TCOM) is the process of measuring the partial pressure of tissue oxygen ($P_{tc}O_2$) via a heated electrode placed upon the skin.

**Aim:** We aim to describe the use of TCOM to define tissue hypoxia and normal ranges for $P_{tc}O_2$, and correlate TCOM with clinical outcomes for wounds treated with hyperbaric oxygen therapy (HBOT).

**Methods:** A structured literature search covering the past 25 years was performed using the MeSH terms: blood gas monitoring; transcutaneous; wound healing; peripheral vascular disease; diabetes; and hyperbaric oxygenation. We critically appraised all relevant papers and, using our synthesis of the data, present our recommendations for the use of TCOM in the assessment of problem wounds for HBOT, and for further research.

**Results:** Normal chest $P_{tc}O_2$ is 60–70 mmHg, which is similar to limb values. TCOM values do not change significantly with age in healthy individuals but limb values are reduced in diabetes, peripheral vascular disease and in limb elevation. TCOM has been validated in predicting wound healing, and successful vascular reconstruction and amputation level, as well as in confirmation of the need for amputation. TCOM is a more effective marker of disease than Doppler assessment or ankle-brachial indices. Thirty-eight studies since 1982 suggest that hypoxia is defined as $P_{tc}O_2$ = 10–40 mmHg. A single critical value for tissue viability has not been determined. $P_{tc}O_2$ increases with increasing partial pressure of inspired oxygen ($P_IO_2$), and is markedly elevated during HBOT. TCOM values progressively increase during a course of HBOT. While low $P_{tc}O_2$ values breathing air confirm wound hypoxia, they do not predict outcome with HBOT. Breathing 100% oxygen at ambient pressure is somewhat predictive of outcome – if wound $P_{tc}O_2 < 35$ mmHg, 41% fail to heal; while a $P_{tc}O_2 > 200$ mmHg breathing hyperbaric oxygen is the best single discriminator between success and failure of HBOT (74% reliable). Using the available data, we suggest clinical guidelines.

**Conclusions:** TCOM is useful to identify patients with problem wounds who may respond to HBOT. Poor quality of the available clinical studies limits the interpretation of the available evidence. A large, multicentre prospective study is required that correlates TCOM using a standard protocol with initial wound grades and clinical outcomes.

### Introduction and aims

Oxygen plays a vital role in wound healing. Many cellular processes such as fibroblast replication, collagen synthesis, neutrophil degranulation and bacterial killing are dependent on the presence of tissue oxygen in partial pressures greater than 30–40 mmHg.[1,2] Tissue hypoxia has been demonstrated in wounds that fail to heal promptly; such wounds are collectively called 'problem wounds'.[3] Measurement of tissue oxygenation is a component of the comprehensive evaluation of problem wounds. Invasive electrodes were first pioneered by Clark and then miniaturised by Silver.[4,5] Hunt first described measurement of tissue oxygenation in humans over 40 years ago.[6] Non-invasive transcutaneous oximetry (TCOM) probes were developed almost a decade later.[7] Since Hunt's original work, there has been considerable evolution of the process, using invasive and non-invasive techniques, summarised in a recent review.[8]

TCOM is the process of measuring oxygen tension (partial pressure) on the skin surface, and was originally used in neonatology.[9] TCOM *estimates* tissue oxygenation by measuring the diffusion of extracellular oxygen into a heated sensor on the skin. TCOM is the only non-invasive measure of tissue oxygenation currently available and avoids the risks of invasive measurement such as tissue disruption around the wound, infection and exacerbation of non-healing lesions.

Sheffield and Workman reported the use of invasive electrodes to confirm hypoxia in chronic wounds and demonstrated improvement in tissue oxygenation when breathing hyperbaric oxygen (HBO).[10,11] TCOM may assist the rational selection of patients who may benefit from hyperbaric oxygen therapy (HBOT) thus allowing more selective application of limited hyperbaric resources.[12] This assumes that TCOM accurately reflects wound tissue oxygenation and that improvement in tissue oxygenation

**Table 1**
**Abbreviations and definitions**

| Abbreviation | Meaning | Further explanation |
|---|---|---|
| TCOM | Transcutaneous oxygen measurement | |
| $P_aO_2$ | Arterial oxygen partial pressure | Oxygen partial pressure measured in arterial blood |
| $P_{tc}O_2$ | Transcutaneous oxygen partial pressure | Oxygen partial pressure measured by transcutaneous oximetry |
| $P_wO_2$ | Wound oxygen partial pressure | Oxygen partial pressure measured in the wound |
| $P_IO_2$ | Inspired oxygen partial pressure | Oxygen partial pressure in the inspired gas |
| HBOT | Hyperbaric oxygen therapy | Delivery of oxygen under pressure as a therapeutic modality |
| ATA | Atmospheres absolute | The pressure relative to a vacuum |
| RPI | Regional perfusion index | The ratio of $P_{tc}O_2$ in the region of interest, (e.g., near a problem wound; peri-wound) relative to a reference 'normal' $P_{tc}O_2$ (e.g., in the chest). This is expressed mathematically as RPI = $P_{tc}O_2$ peri-wound/$P_{tc}O_2$ chest. |
| BPI | Bilateral perfusion index | The ratio of $P_{tc}O_2$ in the region of interest in one limb relative to the reference opposite limb $P_{tc}O_2$ |

will accelerate healing in hypoxic wounds. This paper will critically examine the evidence underpinning these assumptions. Table 1 summarises the abbreviations and definitions that are used in this review.

The aims of this paper are to:
- define the normal ranges for tissue oxygen partial pressures
- review studies where TCOM has been used to define tissue hypoxia
- correlate $P_{tc}O_2$ with clinical outcomes
- review the role of TCOM in the assessment of problem wounds for HBOT
- develop an assessment algorithm based on available evidence from the literature.

We will also identify unanswered questions in order to facilitate further research.

## Methods

We intended to capture all clinical trials and case series involving TCOM in the setting of problem wounds and HBOT from 1982 to 2006. Electronic searching used the MeSH terms: blood gas monitoring; transcutaneous; wound healing; peripheral vascular disease; diabetes; and hyperbaric oxygenation. We searched Central (the Cochrane database of controlled clinical trials), Medline (PubMed), DORCTIHM, CINAHL and EMBASE.

We also hand-searched books covering the combination of problem wounds and hyperbaric oxygen therapy, as well as proceedings and workshops from the Undersea and Hyperbaric Medicine Society, the European Underwater and Baromedical Society, the South Pacific Underwater Medicine Society and international congresses on hyperbaric medicine. Finally a search was undertaken of references identified in papers located by the initial search.

RESULTS OF THE SEARCH

Our initial search identified 178 papers of interest. Of these, we rejected 33 review papers with no new data and 47 that did not present relevant data. A further seven could not be located. The remaining 91 papers contributed to this review.

## Results

The TCOM procedure, its limitations and issues affecting accuracy have been described in detail elsewhere.[13,14] Figure 1 provides a schematic summary of the process and Figure 2 demonstrates sensors applied around a problem wound.

It is important to note that TCOM *does not* measure the wound oxygen tension ($P_wO_2$) but rather the oxygen tension in the skin *surrounding* the wound (i.e., peri-wound $P_{tc}O_2$). This is a potential limitation of the procedure as wound oxygenation is likely to be lower than the $P_{tc}O_2$ adjacent to the wound. We have been unable to identify a head-to-head comparison of transcutaneous oximetry with invasive oximetry, either for raw data tissue oxygen measurements, or in relation to clinical outcomes. It is not clear whether or not such a comparison would improve our understanding of wound oxygenation because the process of inserting invasive oximetry electrodes may actually adversely affect the modality it seeks to measure ($P_wO_2$), by inducing tissue oedema or haemorrhage.[6] Some centres recommend using a reference reading on the chest, and calculation of a regional perfusion index (RPI). A number of authors quote a bilateral perfusion index (BPI) comparing the affected limb with its contralateral partner.[15–19] Most of the clinical outcome studies evaluating the role of TCOM in patients treated with HBO have focused on the peri-wound $P_{tc}O_2$, and have not used either of these indices.[20–4]

**Figure 1**
**Schematic diagram of the polarographic electrode on the skin with oxygen diffusing through a semipermeable membrane to the electrolyte solution**

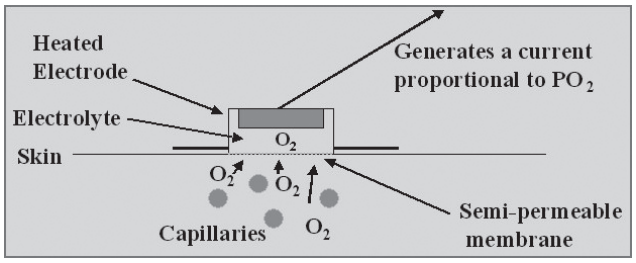



**Figure 2**
**Transcutaneous oximetry electrodes situated adjacent to a problem wound**

## DEFINING NORMAL TISSUE OXYGENATION USING TCOM

Several studies have attempted to define normal ranges for the lower limb in healthy subjects (Table 2). It has been reported that chest readings are not significantly different from leg oxygenation and that females had significantly higher $P_{tc}O_2$ in their legs than males.[25] This group also demonstrated changes in limb tissue oxygenation as limb position changed. Significant increases in foot (+18.3%) and leg (+27.2%) $P_{tc}O_2$ were observed when the leg was placed in a dependent position and similar falls noted when the legs were elevated. It has been reported that $P_{tc}O_2$ falls with age; however, these findings were not confirmed by other studies.[26–8] $P_{tc}O_2$ values tend to be consistent over time, varying on average around 10% in healthy individuals.[29] In individuals free of vascular disease, lower limb $P_{tc}O_2$ is not affected by exercise; however, in individuals with peripheral vascular disease (PVD), there is a significant fall in $P_{tc}O_2$ during and after exercise.[30,31]

## DEFINING HYPOXIA AND VALIDATING TCOM IN RELATION TO CLINICAL OUTCOMES

Early studies using TCOM demonstrated that chronic indolent soft-tissue wounds were severely hypoxic, with wound $PO_2$ levels ($P_wO_2$) ranging from 5 to 20 mmHg.[10,11] $P_{tc}O_2$ values adjacent to indolent problem wounds were < 20 mmHg compared with control values of 30–50 mmHg.[10]

We identified 56 studies involving 4,751 patients that attempted to correlate TCOM with clinical outcomes (Table 3). Twelve studies involved HBO, and will be covered later in

**Table 2**
**Normal $P_{tc}O_2$ readings in healthy individuals**

| Lead author, reference, year | Study population (n) | Average age (years) | Reference $P_{tc}O_2$ site | $P_{tc}O_2$ mmHg (± SD or range) | Comments |
|---|---|---|---|---|---|
| Dowd et al[27] 1983[1] | (205) | Unknown | Lower leg – M<br>Lower leg – F | 70<br>72 | Male/female difference not statistically significant. No significant trend $P_{tc}O_2$ vs age. |
| Dowd et al[28] 1983[2] | 'Normal' volunteers (161) | 45 | Foot<br>Leg<br>Chest (n = 91) | 67 ± 11<br>70 ± 9<br>69 ± 11 | No significant difference chest vs leg or foot. |
| Hauser et al[26] 1983 | Healthy, asymptomatic subjects (12) | Unknown | Foot | 65 ± 3 | Chest and limb $P_{tc}O_2$ fell with increasing age. |
| Dooley et al[25] 1997 | Healthy (72) | 34.2 | Foot<br>Lower leg – M<br>Lower leg – F<br>Chest | 63.0 to 67.4<br>48.8 to 56.0<br>59.3 to 64.8<br>63.4 to 68.5 | Foot and leg values increased in dependent position (+18.3% and +27.2%) and decreased in elevated position (-18.1% and -28.2%). Male/female readings significantly different |

**Table 3**
**Publications reporting clinical outcomes that have been correlated with $P_{tc}O_2$ measurements or indices derived from TCOM (*involved hyperbaric oxygen)**

| Outcome measure/Year | First author | Patient numbers | Ref |
|---|---|---|---|
| **Predicted healing** | | | |
| 1982 | Franzeck | 35 | 58 |
| 1983 | Dowd | 62 | 27 |
| 1984 | Cina | 100 | 59 |
| 1987 | Hauser | 113 | 60 |
| 1988 | Oishi | 80 | 61 |
| 1988 | Wyss | 162 | 62 |
| 1991 | Wattel* | 59 | 21 |
| 1991 | Pecoraro | 46 | 63 |
| 1992 | Campagnoli* | 28 | 48 |
| 1992 | Pinzur | 38 | 64 |
| 1994 | Conlon | 24 | 65 |
| 1995 | Yablon | 11 | 66 |
| 1996 | Bunt | 147 | 67 |
| 1996 | Bouachour* | 36 | 19 |
| 1996 | Claeys | 86 | 68 |
| 1996 | Dooley* | 60 | 49 |
| 1996 | Padberg | 204 | 39 |
| 1996 | Smith* | 26 | 56 |
| 1999 | Kalani | 50 | 69 |
| 2001 | Grolman* | 36 | 22 |
| 2002 | Strauss* | 190 | 24 |
| 2002 | Fife* | 1,144 | 23 |
| 2003 | Abidia* | 16 | 43 |
| 2003 | DeGraaff | 96 | 91 |
| **Success of procedure** | | | |
| 1986 | Rhodes | 60 | 70 |
| 1988 | Lalka | 62 | 71 |
| 1989, 1990 | Ameli | 105 | 32, 33 |
| 1991 | Stein | 127 | 34 |
| 1993 | Mathieu* | 15 | 54 |
| 1997 | Hanna | 29 | 72 |
| 2005 | Poredos | 56 | 73 |
| **Predicted severity of symptoms/disease** | | | |
| 1984 | Hauser [1] | 25 | 15 |
| 1984 | Hauser [2] | 46 | 16 |
| 1984 | Hauser [3] | 24 | 74 |
| 1984 | Byrne | 138 | 30 |
| 1984 | Wyss | 188 | 36 |
| 1988 | Kram | 32 | 75 |
| 1988 | Lalka | 62 | 71 |
| 1988 | Moosa | 22 | 76 |
| 1989, 1990 | Ameli | 105 | 32, 33 |
| 1991 | Stein | 127 | 34 |
| 1992 | Reiber | 80 | 77 |
| 1994 | Ubbink | 130 | 78 |
| 1995 | Ballard | 55 | 79 |
| **Predicted amputation +/- level** | | | |
| 1982 | Burgess | 37 | 80 |
| 1982 | White | 25 | 40 |
| 1984 | Wyss | 188 | 36 |
| 1984 | Katsamouris | 37 | 81 |
| 1985 | Harward | 101 | 82 |
| 1987 | Dowd | 101 | 83 |
| 1988 | Bongard | 26 | 84 |
| 1990 | Wattel* | 20 | 20 |
| 1992 | Reiber | 80 | 77 |
| 1992 | Chambon | 33 | 85 |
| 2005 | Zgonis* | 35 | 57 |
| **Predicted successful vascular reconstruction** | | | |
| 1988 | Kram | 32 | 75 |
| 1992 | Scheffler | 64 | 86 |
| 1997 | Ray | 41 | 87 |
| 2002 | Stalc | 57 | 88 |
| 2003 | Wagner | 34 | 89 |
| 2005 | Caselli | 43 | 90 |

the review. Study methodologies were diverse, and outcomes could be classified into five major categories:

1 prediction of healing
2 success of surgical procedure
3 predicted severity of symptoms or disease
4 predicted level of amputation
5 predicted success of vascular reconstruction.

Fifteen studies (1,137 patients) demonstrated that TCOM provided better overall predictive capability than Doppler studies measuring ankle-brachial index (ABI) and segmental pressures, or laser fluximetry across one or more of the five outcome measures (Table 4). Three studies suggested TCOM showed similar ability and one reported laser Doppler fluximetry to have better predictive capability than TCOM.[32–5]

Other indirect methods of measuring tissue oxygenation have not reliably predicted the fate of a problem wound. Arterial calcific medial sclerosis in diabetics may artificially elevate ankle blood pressures, making Doppler studies in these patients unreliable.[36] This may be one reason why TCOM has proven superior to ABI measurement in diabetics. However, over half of the authors in Table 4 studied non-diabetics or mixed populations. TCOM has also been used to evaluate the impact of local pressure or stretching on the skin. Falls in $P_{tc}O_2$ levels were observed in patients with tetraplegia with and without pressure ulcers.[37]

**Table 4**
**Studies where TCOM has demonstrated outcome prediction superior to Doppler studies of ankle-brachial index and segmental pressures, or laser fluximetry**

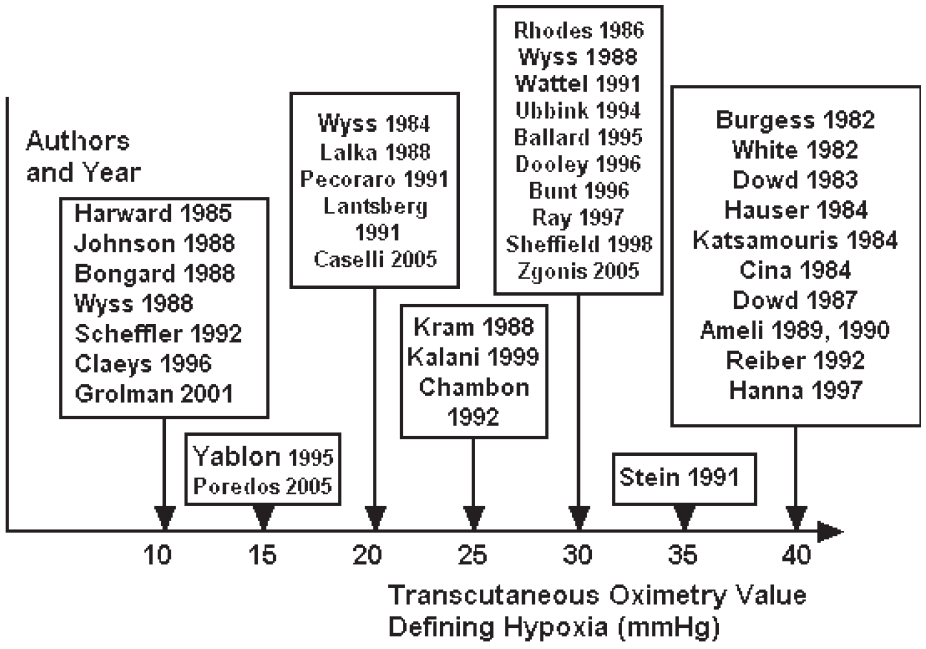
| Year | First author | Patient numbers | Reference |
|---|---|---|---|
| **Outcome measure** | | | |
| **Predicted healing** | | | |
| 1988 | Wyss | 162 | 62 |
| 1994 | Conlon | 24 | 65 |
| 1995 | Ballard | 55 | 79 |
| 1996 | Padberg | 204 | 39 |
| 1999 | Kalani | 50 | 69 |
| **Success of procedure** | | | |
| 1988 | Lalka | 62 | 71 |
| 1997 | Hanna | 29 | 72 |
| 1997 | Ray | 41 | 87 |
| **Predicted severity of symptoms/disease** | | | |
| 1984 | Hauser [1] | 25 | 15 |
| 1984 | Hauser [2] | 46 | 16 |
| 1994 | Ubbink | 130 | 78 |
| **Predicted amputation +/- level** | | | |
| 1982 | White | 25 | 40 |
| 1984 | Wyss | 188 | 36 |
| **Predicted successful vascular reconstruction** | | | |
| 1988 | Kram | 32 | 75 |
| 1992 | Scheffler | 64 | 86 |

In an experimental model, Melis demonstrated significant falls in $P_{tc}O_2$ when skin was undermined and stretched in plastic surgical procedures.[38]

Based on these data, there have been attempts to define a 'cut off' (sic) value where TCOM predicts success or otherwise of a procedure (such as amputation), or predicts healing. Sheffield wrote (1998) "*No single value can be specified 'normal' $O_2$ tension for all tissue. Rather there exists a series of gradients.*"[14] $P_{tc}O_2$ values will depend on the patient population, the technique used and site of measurement, the position of the limb and the therapeutic intervention. Study design is also important. Some authors attempted to define a $P_{tc}O_2$ value below which all patients failed the treatment regimen, whereas others attempted to define a value above which all patients have successful outcomes. Usually there was a 'grey zone' between the values determined for success or failure. This provides one explanation for the considerable variability of $P_{tc}O_2$ values defining hypoxia (Figure 3).

Calculations of sensitivity and specificity have been used to provide guidance regarding the usefulness of measuring $P_{tc}O_2$ in the above studies; however, there is considerable diversity in study design. The majority do not test the intervention of HBOT, hence the sensitivity and specificity data are not helpful to the hyperbaric clinician. In addition, these calculations provide little comfort to the patient who is in the 'statistical grey zone', where neither success nor failure is guaranteed. Padberg's group attempted to account for this variability by producing a graph of probability of healing versus $P_{tc}O_2$.[39] Their method of presenting their results provides the clinician with meaningful data to present to the patient. For a given $P_{tc}O_2$ value, there is a quotable

**Figure 3**
**Defining hypoxia using transcutaneous oximetry**



percentage chance of healing. They also showed that diabetes mellitus and renal failure increased the $P_{tc}O_2$ value required for successful healing.[39]

The clinical endpoints of the studies cited varied greatly. In Table 3, 24 studies investigated TCOM for the ability to predict healing in patients with pre-existing problem wounds, 13 studies investigated prediction of procedural or vascular reconstruction success, and 11 studies examined TCOM as a predictor of amputation level. A further 13 studies assessed the ability of TCOM to predict severity of symptoms or disease. In addition, the inclusion criteria varied across these studies, making direct comparison problematic. Some studied only diabetic patients, others peripheral vascular disease without diabetes or mixed populations.

Given the differences in methodologies and outcome measurements used in the available literature, it is not surprising that there is a broad spread of hypoxic TCOM values up to 40 mmHg. For healing to occur, 50 mmHg may be a more appropriate value.[40] This at least provides the clinician with some guidance when assessing new patients. Any treatment algorithm must allow for this 'grey zone' in definitions; it is reasonable to define wound hypoxia as $P_wO_2 \leq 40$ mmHg, with allowances up to $\leq 50$ mmHg for patients with other factors such as diabetes and renal failure.[36,39]

## THE ROLE OF TCOM IN THE ASSESSMENT OF PROBLEM WOUNDS FOR TREATMENT WITH HBOT

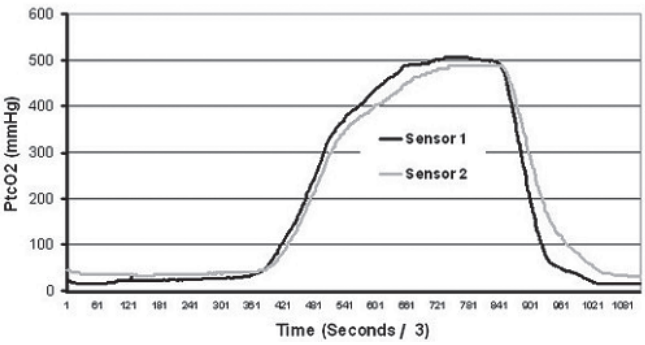
Transcutaneous oximetry has been used to predict success of a treatment intervention in Tables 3 and 4 under two broad models, based on the timing of TCOM relative to the intervention:

- TCOM *before* the treatment intervention, to measure the level of oxygenation in the patient's target tissue, and assess if this is sufficient to allow success of the treatment (e.g., amputation at a particular level, or healing of an ulcer with conservative management);
- TCOM *after* the treatment intervention to assess if the intervention caused sufficient increase in $P_{tc}O_2$ to result in successful healing.

Both models may have potential for assessing the role of TCOM in the treatment of problem wounds with HBOT.

HBOT has demonstrated some success as a treatment for refractory problem wounds and radiation-injured tissue.[41–5] As a therapeutic intervention, HBOT is unique in that $P_IO_2$ is increased during delivery of oxygen under pressure and its effect on $P_{tc}O_2$ can be measured (Figure 4). When evaluating the role of TCOM in this context, it would be expected that the treatment intervention (HBOT) would acutely increase $P_wO_2$ (Figure 4), and as healing progresses, lead to progressive improvements in surrounding tissue oxygenation. Peri-wound $P_{tc}O_2$ increases with increased $P_IO_2$, and this increase is amplified when breathing oxygen at pressures of greater than one atmosphere.[20,21,23,46–9]

**Figure 4**
**Real-time measurement of $P_{tc}O_2$ (peri-wound) in a patient breathing air, then HBO at 2.4 ATA**




| Site of measurement | Peak values of $P_{tc}O_2$ (mmHg) breathing: Room air | 100% $O_2$ | HBO 2.4 ATA |
|---|---|---|---|
| Chest | 58 | 286 | 725 |
| Sensor 1 | 33 | 40 | 507 |
| Sensor 2 | 18 | 54 | 488 |

There is a close correlation between arterial oxygen partial pressure ($P_aO_2$) and $P_{tc}O_2$ in the hyperbaric environment.[25] In some patients, physiological $P_{tc}O_2$ levels are only achievable in the hyperbaric environment (Figure 4), and the elevation of $P_wO_2$ from baseline is far greater than the proportionate increase in $P_IO_2$.[21] The patient in Figure 4 had failed to increase $P_{tc}O_2$ breathing 100% oxygen at 1 ATA, but a brisk increase occurred in HBO.

In one series, baseline $P_{tc}O_2$ levels adjacent to the hypoxic wounds when breathing air were less than 15 mmHg, and these increased over the course of multiple HBO exposures to above 30 mmHg.[10] This effect has also been observed for oedematous wounds, and was associated with healing.[49] Delayed tissue injury from radiation therapy is another example of a specific type of problem wound. In patients who had received head and neck irradiation for cancer, tissue around the oral cavity was hypoxic and the hypoxia was restored to 80% of normal values over a course of 15–30 HBO treatments.[41,50–2] The HBO-induced angiogenic response in irradiated tissue was well documented, and the authors showed that increases in $P_{tc}O_2$ were correlated with clinical success (tooth socket, bone and mucosal healing) that was sustained for up to 3 years.[41,50–2]

In diabetic patients, improvements in $P_{tc}O_2$ on the dorsum of the foot were reported at the completion of HBOT, compared with controls.[42] Similar findings were reported by another study in a recent abstract.[53] In the first study, even though there were fewer major amputations in the HBOT group compared with controls, the authors did not specifically relate the TCOM values to outcome.[42]

Thus HBOT leads to an acute change in the ambient tissue environment, with subsequent angiogenesis leading to

**Table 5. Studies using TCOM as outcome predictor in subjects receiving HBOT (PVD – peripheral vascular**

| Author | Year | Number of subjects | Study description | Threshhold $P_{tc}O_2$ value | Outcomes |
|---|---|---|---|---|---|
| Mathieu et al[18] | 1990 | 33 patients, post-traumatic upper and lower limb ischaemia 16 with and 17 without arterial injury | Use of TCOM in air, 100% oxygen and HBO 2.5 ATA to predict need for amputation. Used bilateral perfusion index (BPI) | No value determined at air, 100% $O_2$, or HBO<br>Recommended HBO if BPI > 0.2 and < 0.4; no outcome data to support this | No amputation: $P_{tc}O_2$ HBO = 425 ± 292 mmHg<br>Amputated: $P_{tc}O_2$ HBO = 114 ± 148 mmHg<br>Air and 100% $O_2$ values not significant<br>BPI < 0.4 sensitivity 100%, specificity 94%<br>BPI < 0.2 gives 100% predictive value for amputation |
| Wattel et al[20] | 1990 | 20 patients mixed PVD (9 cases) or diabetes (11 cases) | Use of TCOM in air, 100% oxygen and HBO 2.5 ATA to predict healing in patients receiving HBOT | No value determined for air or 100% $O_2$,<br>$P_{tc}O_2$ < 50 mmHg in HBO – all worsened<br>$P_{tc}O_2$ > 100 mmHg in HBO – all healed | Data not tabulated to enable sensitivity and specificity<br>Healed $P_{tc}O_2$ in HBO = 635 ± 388 mmHg<br>Not healed $P_{tc}O_2$ in HBO = 45 ± 20 mmHg (P = 0.003)<br>Rise of $P_{tc}O_2$ in HBO significant<br>100% $O_2$ values not significant |
| Wattel et al[21] | 1991 | 59 diabetic patients | Use of $P_{tc}O_2$ to predict healing in patients receiving HBOT, used absolute values and ratios relative to reference sensor on chest | Only values in HBO useful $P_{tc}O_2$ > 450 mmHg in HBO 2.5ATA included all successes | Success = HBO $P_{tc}O_2$ = 786 ± 258 mmHg<br>Failure = HBO $P_{tc}O_2$ = 323 ± 214 mmHg; P < 0.005<br>Ratios in HBO also significantly different<br>No sensitivity/specificity data |
| Campagnoli et al[48] | 1992 | 28 patients, lower limb ulcers mixed aetiology (24 diabetic) | Use of $P_{tc}O_2$ to predict healing in patients receiving HBOT, used absolute values | $P_{tc}O_2$ > 400 mmHg | Minimal statistical analysis performed<br>If $P_{tc}O_2$ > 400 mmHg achieved in HBO – all healed |
| Mathieu et al[54] | 1993 | 15 patients, post-pedicle musculocutaneous flap transplantation referred for HBOT | Predictive value $P_{tc}O_2$ in HBO for healing. Measured absolute values and flap perfusion index relative to chest 3hr post-op | $P_{tc}O_2$ > 70 mmHg in HBO 2.5ATA | $P_{tc}O_2$ > 70 mmHg in HBO predicted flap success<br>$P_{tc}O_2$ <= 35 mmHg in HBO predicted flap failure<br>No values predictive in air or 100% $O_2$<br>HBO values: failure = 12 ± 12 mmHg;<br>success 378 ± 385 mmHg (P < 0.01)<br>**7/15 total success** |
| Bouachour et al[19] | 1996 | 36 patients with crush injuries (RCT) | Measured BPI as a secondary outcome of treating crush injuries with HBO | Not assessed | HBO group more likely to achieve complete healing and less likely to receive extra surgery. BPI breathing air, measured in HBOT group improved progressively from 0.4 to 0.8. No significant change in placebo group |
| Dooley et al[49] | 1996 | 60 patients, oedematous non-healing leg wounds | Measured $P_{tc}O_2$ and predicted healing following HBO treatment | No threshold value determined | Pre-treatment $P_{tc}O_2$ correlated with degree of oedema, did not predict healing. HBO significantly reduced oedema. 20 patients lost to follow up |
| Faglia et al[42] | 1996 | 68 diabetic patients (RCT) | Assessed risk of major amputation with and without HBO. Measured change in $P_{tc}O_2$ | Not assessed | HBO group significantly less likely to receive major amputation. (Absolute Risk Reduction 26%) $P_{tc}O_2$ breathing air improved significantly in treatment group, from 5.0 ± 5.4 to 14.0 ± 11.8 mmHg, P = 0.0002 |

| Author | Year | Number of subjects | Study description | Threshhold $P_{tc}O_2$ value | Outcomes |
|---|---|---|---|---|---|
| Smith et al[56] | 1996 | 26 patients (16 diabetic) | Used $P_{tc}O_2$ to predict response of wounds to 40 HBO treatments using wound scores. Measured $P_{tc}O_2$ breathing air, 100% $O_2$ and in HBO at 2.4 ATA | $P_wO_2$ > 800 mmHg in HBO | Responders had higher wound $PO_2$ on 100% $O_2$ (284 vs 132 mmHg), and in HBO (1047 vs 509 mmHg) $P_wO_2$ > 800 mmHg linked to HBO response Overall response rate 9/26 |
| Lindstrom et al[55] | 1998 | 20 patients with tibial shaft fractures requiring an intramedullary nail (RCT) | Five days HBO at 2.5 ATA; improvement on $P_{tc}O_2$ or blood flow compared with air breathing | Measured flow in tibialis posterior artery and $P_{tc}O_2$ at three days after surgery | Tibialis posterior artery flow improved significantly in the HBO group but there was no significant difference in $P_{tc}O_2$ at three days after surgery Measurements not correlated with clinical outcomes |
| Grolman et al[22] | 2001 | 36 patients, critical limb ischaemia and non-healing ulcers, (24 diabetic, 9 renal failure) | Used $P_{tc}O_2$ to predict which patients benefit from HBO. Used increase in $P_{tc}O_2$ >10 mmHg breathing 100% oxygen vs air at 1 ATA as indicator of potential response | Rise in $P_{tc}O_2$ > 10 mmHg breathing 100% oxygen | Absolute value of TCOM not predictive Breathing surface oxygen, if rise in $P_{tc}O_2$ > 10 mmHg, 19/27 healed, vs 1/9 if $P_{tc}O_2$ <= 10 mmHg HBO $P_{tc}O_2$ not assessed |
| Lin et al[53] | 2001 | 29 diabetic patients (RCT) | HBO 30 treatments compared with standard care for improved healing of diabetic foot wounds, or improved indicators of vascular state | Not assessed | $P_{tc}O_2$ increased over 30 HBO treatments 36 ± 21 to 56 ± 21 mmHg; P < 0.01 Laser Doppler flow improved significantly No significant improvement in ankle-brachial index |
| Fife et al[23] | 2002 | 1,144 diabetic patients with lower extremity ulcers | Determined if pre-treatment air $P_{tc}O_2$, or in-chamber $P_{tc}O_2$ predictive of healing | $P_{tc}O_2$ in ambient air not predictive of outcome in HBO $P_{tc}O_2$ > 200 mmHg predictive of success | In-chamber $P_{tc}O_2$ > 200 mmHg best discriminator (74% reliable) $P_{tc}O_2$ < 15 mmHg air at 1 ATA and in-chamber $P_{tc}O_2$ < 400 mmHg; negative outcome 75.8% reliable, 73.3% positive predictive value |
| Strauss et al[24] | 2002 | 190 patients, foot and ankle wounds (no demographic data) | Determined $P_{tc}O_2$ values in HBO that correlate with healing wound | $P_{tc}O_2$ < 100 mmHg in HBO = less chance of response $P_{tc}O_2$ < 50 mmHg in HBO, all had amputations | $P_{tc}O_2$ > 200 mmHg in HBO associated with wound healing Sensitivity 0.8, Specificity 0.44, positive predictive value 0.88, negative predictive value 0.3, accuracy 74% Healing rate 88% if $P_{tc}O_2$ > 100 mmHg |
| Abidia et al[43] | 2003 | 16 diabetic patients RCT | Assessed role of HBOT in ischaemic diabetic lower limb ulcers | Not assessed | No difference in $P_{tc}O_2$ between pre and post treatment in either group; no difference between groups; 2 withdrew; 5/8 healed in HBO group, 1/8 healed placebo |
| Zgonis et al[57] | 2005 | 35 patients, 40 feet | Use of pre-operative $P_{tc}O_2$ to predict healing in patients receiving partial foot amputations then 20 HBO treatments | $P_{tc}O_2$ > 29 mmHg successful outcome | $P_{tc}O_2$ > 29 mmHg pre-operative = successful outcome Difficult to determine if HBO influenced outcome |

**disease, HBO – hyperbaric oxygen, TCOM – transcutaneous oximetry, RCT – randomised controlled trial)**

sustained increases in $P_{tc}O_2$ levels, demonstrable with TCOM. Therefore, in patients receiving HBOT, the pre-treatment $P_{tc}O_2$ breathing air might not correlate with outcome, because the treatment itself has potential to change the measurement. When assessing the role of TCOM as an outcome predictor in patients receiving HBOT, it would, therefore, be most appropriate to undertake TCOM during exposure to HBO and after the course of HBOT, as per the second model above. This is an important issue when interpreting outcome data for the HBOT studies that follow.

## HYPERBARIC OXYGEN AS A TREATMENT FOR PROBLEM WOUNDS – CORRELATING TCOM WITH CLINICAL OUTCOMES

Based on the above evidence, the rationale for the use of HBOT for problem wounds is:

- problem wounds are often hypoxic
- elevated $P_IO_2$ (during HBOT) has been shown to acutely correct the hypoxia around problem wounds
- HBOT has been associated with healing in problem wounds
- HBOT has been associated with sustained improvement in $P_{tc}O_2$.

Given these premises, is there evidence that HBO increases $P_{tc}O_2$ in problem wounds that is linked to improved outcomes? If the evidence exists, how might it best be applied in clinical guidelines?

Table 5 summarises the 16 studies (12 in Table 3) identified that have investigated the role of TCOM in the assessment of problem wounds receiving HBOT.[18–24,42,43,48,49,53–7] Early studies investigated if TCOM values in air, 100% oxygen and HBO, measured before treatment, were predictive of healing when HBOT was used for post-traumatic ischaemia, PVD wounds, diabetic wounds and pedicle flaps.[18,20,21,54] Pre-treatment $P_{tc}O_2$ values breathing air could not be correlated with improvements of limb oedema or wound condition that were associated with HBOT.[49]

These studies generally concluded that only $P_{tc}O_2$ measured in HBO was useful and the $P_{tc}O_2$ values measured breathing air or 100% oxygen at 1 ATA were not useful in predicting outcome. This is logical because HBO was the therapeutic intervention, and patients responding to treatment should be able to demonstrate some effect on $P_{tc}O_2$ in HBO if hypoxia is relevant in their problem wound. When the above authors attempted to define a threshold $P_{tc}O_2$ value breathing HBO, each of the studies produced a different result. A threshold value was determined for poor outcome in HBOT when $P_{tc}O_2 < 50$ mmHg.[20] Threshold values for positive outcome ranged between $P_{tc}O_2 > 70$ mmHg in pedicle flaps and > 450 mmHg in HBOT for diabetic wounds.[21,54] A similar threshold of $P_{tc}O_2 > 400$ mmHg was reported for mostly diabetic patients.[48] Two studies related their outcomes in traumatic ischaemia and crush injuries to the TCOM BPI.[18,19] A BPI < 0.2 predicted universally poor outcomes.[18] Individuals sustaining an amputation had lower $P_{tc}O_2$ values than those whose limbs were saved.[18] If patients were going to respond to HBOT, then a progressive improvement in BPI occurred during the course of treatment.[19] Other studies in diabetic patients have not linked improvements in $P_{tc}O_2$ values after HBOT with outcomes such as healing or risk of amputation.[42,53] In another study of only 16 diabetic patients there was no significant difference in $P_{tc}O_2$ values post-treatment compared with before HBOT.[43] Five days' HBOT in patients with tibial shaft fractures resulted in improved tibialis posterior artery blood flow; however, $P_{tc}O_2$ did not change.[55] The clinical significance of these findings in traumatic injuries is unclear.[55]

In a mixed population of patients receiving 40 HBO treatments for problem wounds, $P_{tc}O_2$ was measured breathing air, oxygen and HBO at 2.4 ATA prior to treatment.[56] They found that 8 out of 9 'responders' had $P_{tc}O_2 > 800$ mmHg in HBO, whereas only 1 out of 6 patients with $P_{tc}O_2 < 800$ mmHg were 'responders'. Despite demonstrating that 'responders' had higher values breathing 100% oxygen and HBO, a threshold value was not suggested. A further weakness of this study was the use of wound scores rather than wound healing to define response.

In 36 patients with critical limb ischaemia and non-healing ulcers, of whom 18 had failed revascularization and 24 were diabetic, the absolute $P_{tc}O_2$ values in 100% oxygen were not predictive of outcome but a rise in $P_{tc}O_2 > 10$ mmHg was positively associated with healing.[22]

Pre-operative $P_{tc}O_2$ as a predictor of healing was evaluated in patients receiving partial foot amputations followed by 20 HBO treatments.[57] A $P_{tc}O_2 > 29$ mmHg was predictive of healing. From the data presented, it was not possible to determine if HBOT influenced the outcome.

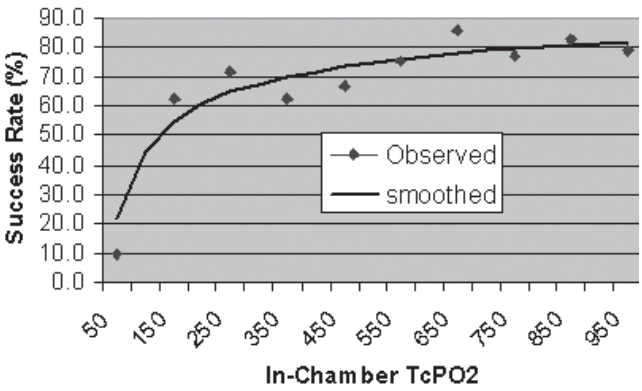
In 144 patients with problem foot wounds treated with HBO, 126 (88%) remained healed at one year if $P_{tc}O_2$ had measured > 200 mmHg during HBOT, compared with 17 out of 29 patients (59%) whose $P_{tc}O_2$ in the hyperbaric chamber was < 100 mmHg.[24] An 'intermediate responder' group with $P_{tc}O_2$ values of 100–200 mmHg in HBO also had healing rates of 88%. In their discussion, Strauss et al stated that using $P_{tc}O_2 > 200$ mmHg in HBO to define 'responders' had low specificity and negative predictive value. This is not surprising when the 'intermediate responders' had the same healing rate as the 'responders'. Their discussion was somewhat confusing when they stated that their protocol was modified to define 'intermediate responders' as $P_{tc}O_2$ values of 50–200 mmHg in HBO. From the data presented it is not possible to determine how they arrived at their decision. An overall healing rate of 83% is an exceptional result. Unfortunately, although data were collected prospectively on 159 of the 190 patients in the series, the study presented only very limited patient demographic data and statistical analysis, and the numbers of HBO treatments were not recorded, making assessment of this case series difficult.

In the largest series to date of the role of TCOM in predicting outcomes for patients receiving HBOT, 1,144 consecutive diabetic patients with lower-limb problem wounds were retrospectively studied in a multicentre 10-year review.[23] The aim was to assess if TCOM breathing air, oxygen and HBO correlated with patient outcomes. Whether absolute value of $P_{tc}O_2$ breathing oxygen at ambient pressure or the percentage increase in $P_{tc}O_2$ correlated with outcome was also assessed. Success with HBOT was defined as complete epithelialisation or partly healed (complete granulation); failure was defined as partial granulation only or requiring amputation. Sixty-eight patients had no outcome data available and nine died, leaving 1,067 for analysis. Of these, 756 had full follow-up information. TCOM was conducted at ambient pressure breathing air for 629 patients, oxygen for 499 patients, and HBO for 221 patients.[23]

At two weeks post-HBOT, 117 out of 118 patients defined as 'healed' after HBOT remained healed. At four-week follow up, 390 out of 446 patients defined as 'partial healing' after HBOT were classified as healed. Overall, 68.4% of all wounds were fully healed at follow up. The mean number of HBO treatments for success was 34. The HBOT regime was interrupted in 15.9% and these patients had significantly worse outcomes. Worse outcomes were noted with increasing grades of wound severity. No difference in outcome was noted in patients treated with HBO at 2.0 ATA versus 2.4 ATA.[23]

This study reported treatment failure as their outcome measure.[23] They found that $P_{tc}O_2$ values breathing air at ambient pressure had almost no predictive value for benefit from HBOT, apart from confirming that the wounds were hypoxic. Breathing 100% oxygen at ambient pressure, the absolute $P_{tc}O_2$ value was more reliable than the increase with oxygen challenge; however, the best reliability achieved was 69.9%. If $P_{tc}O_2 < 35$ mmHg breathing 100% oxygen, the failure rate with HBOT was 41%. In-chamber $P_{tc}O_2 < 200$ mmHg breathing HBO was the best single discriminator for failure with HBOT. Using this threshold value, the test had a reliability of 74% (positive predictive value for failure to heal 58.3%, sensitivity 23.3%, specificity 93.8%, false negatives 23.5%). For in-chamber $P_{tc}O_2 < 100$ mmHg, the likelihood of failure was 90%. A graph of HBOT success versus in-chamber $P_{tc}O_2$ re-calculated from Fife's data is shown in Figure 5 (Otto G, personal communication, June 2006). A slightly higher degree of discrimination is achieved by combining the $P_{tc}O_2$ measurements breathing air at ambient pressure with $P_{tc}O_2$ measurements breathing HBO.[23]

**Figure 5**
**Percentage of successful outcome related to $P_{tc}O_2$ breathing HBO for healing of problem wounds. Line of best fit calculated using least squares exponential regression model. Data from Fife et al, Table 7[23] (TcPO2 = transcutaneous oxygen pressure (mmHg))**




**%success = 100 – 5.73 x (TCOM)$^{-0.5046}$**

### Summary of HBO data – conclusions and recommendations

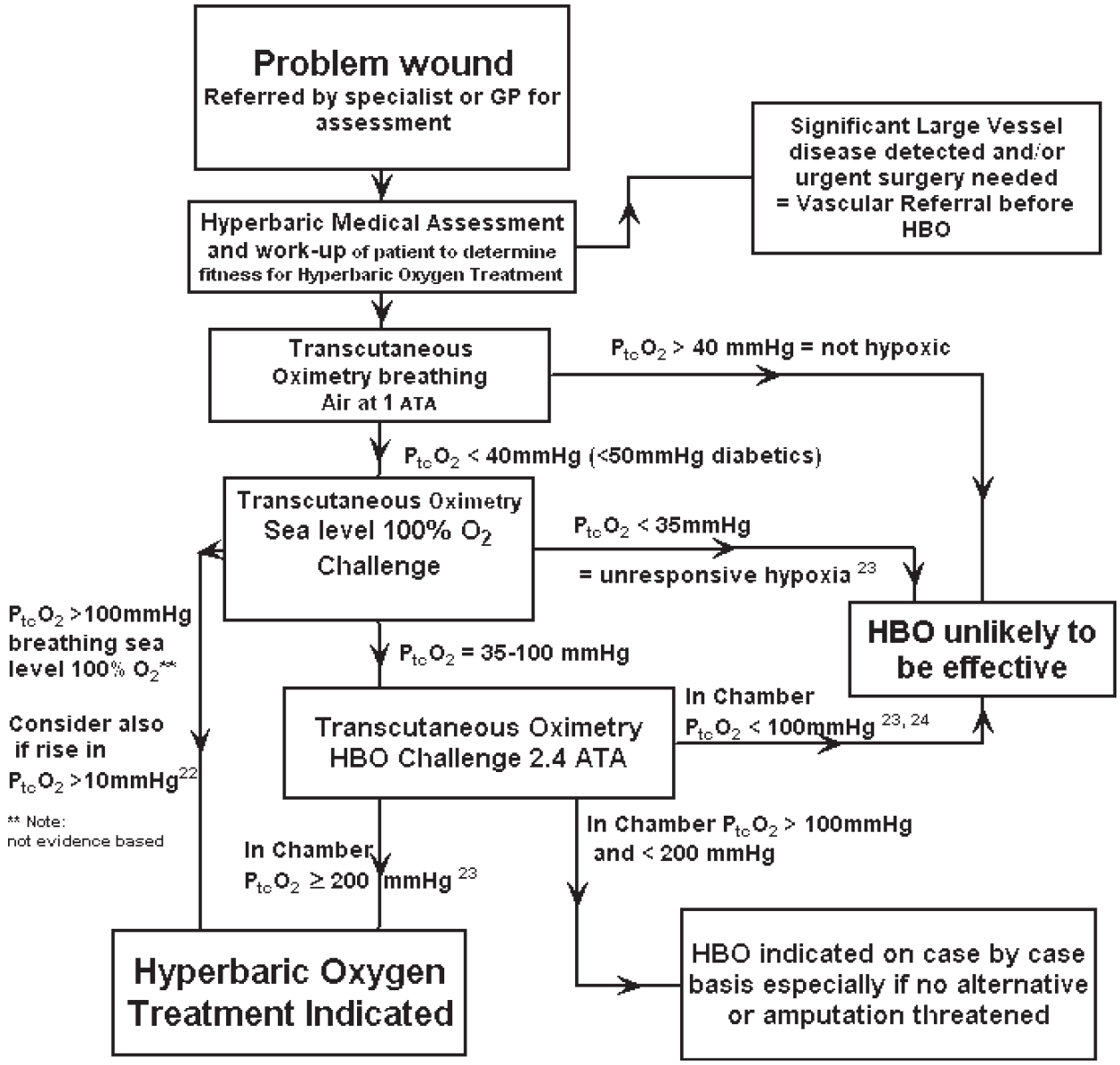
There are limitations to all of the studies reviewed. However, it is still possible to formulate available evidence from TCOM research to guide our clinical decisions in treating problem wounds with HBOT:

- TCOM produces a valid estimate of wound oxygenation, and the available literature suggests that $P_{tc}O_2$ values less than 40 mmHg provide evidence that a wound is hypoxic. A higher value of $P_{tc}O_2 < 50$ mmHg may be appropriate to define hypoxia in diabetics and patients with renal failure.
- TCOM breathing air is unable to predict benefit of subsequent HBOT.
- TCOM breathing 100% oxygen has some predictive value. If $P_{tc}O_2 < 35$ mmHg, then the failure rate is 41%, with an overall accuracy of 69%.
- If in-chamber $P_{tc}O_2 < 100$ mmHg, then the failure rate with HBOT is between 41 and 90%. In-chamber $P_{tc}O_2 < 50$ mmHg is predictive of HBOT failure.
- In-chamber $P_{tc}O_2$ measured breathing HBO is the best single predictor of response to HBOT. $P_{tc}O_2 > 200$ mmHg is a useful predictor of success (74% to 88% reliable). Earlier small studies suggest that in-chamber $P_{tc}O_2$ values should be greater than 400–450 mmHg to guarantee success.

Figure 6 presents a decision flowchart using TCOM to guide clinicians when determining suitability of patients for HBOT. Patients with significant arterial disease should first receive a vascular surgical opinion before commencing HBOT. In assessing suitability for HBOT, the initial step is to confirm that the wound is hypoxic breathing air. Fife et al recommended "*patients with normal values of $TcPO_2$ in air should not be selected for hyperbaric oxygen therapy unless there are mitigating circumstances…*"[23]

Some hyperbaric facilities do not have the capability to undertake in-chamber TCOM, hence it is reasonable to perform an oxygen challenge breathing 100% oxygen at ambient pressure. Data from Strauss et al's 'intermediate responders' suggests that if $P_{tc}O_2 > 100$ mmHg breathing HBO, then there is an 88% chance of healing.[24] By inference, if $P_{tc}O_2 > 100$ mmHg can be reached breathing 100% oxygen at ambient pressure, then it should be higher breathing HBO. However, until a threshold value breathing 100% oxygen at

**Figure 6**
**Problem wound algorithm, incorporating use of TCOM in selection process (with references)**




ambient pressure is determined that suggests benefit from HBOT, uncertainty will exist.

There is a need to validate 100% oxygen, ambient-pressure TCOM threshold values in a larger prospective study to determine if it is useful in predicting response to HBOT. In the interim, we have selected threshold values breathing 100% oxygen at ambient pressure of 35 mmHg for 'non responders' and 100 mmHg to define 'responders'.[23,24] Grolman's group suggested that a rise in $P_{tc}O_2$ > 10 mmHg breathing 100% oxygen at ambient pressure had better predictive value for HBOT success (70%) than the actual $P_{tc}O_2$ value.[22]

When evaluating TCOM in the hyperbaric environment, current studies suggest a high chance of success with HBOT if $P_{tc}O_2$ > 200 mmHg. If the $P_{tc}O_2$ < 100 mmHg, then patients are unlikely to respond to HBOT. Where $P_{tc}O_2$ = 100–200 mmHg, then HBOT should be recommended on a case-by-case basis, especially if there is no viable alternative treatment.

**Unanswered questions requiring further research**

The above conclusions and recommendations have been drawn from research that has significant limitations. Most of the studies were retrospective, non-randomised and with small numbers. Even the RCTs had small numbers and did not clearly link TCOM with clinical outcomes. A larger, prospective multicentre trial is required to further clarify the role of TCOM in assessment of problem wounds treated with HBOT. In this larger study, TCOM values (in air, 100% oxygen at ambient pressure, and HBO) measured before and after HBOT, need to be carefully correlated with clinically

significant outcomes. A number of basic validation studies are also required, for example:

- studies to correlate invasive $P_wO_2$ with $P_{tc}O_2$ values
- studies to link the measurement of $P_{tc}O_2$ breathing air, 100% oxygen and HBO, in health and disease, to determine the degree of correlation between values
- studies to validate the use of 100% oxygen at ambient pressure to predict HBO response
- studies of post-HBOT $P_{tc}O_2$ values to evaluate their correlation with clinically meaningful outcomes.

Sheffield stated in 1998 "*At this writing, $P_{tc}O_2$ is as much an art as a science*."[14] Since his review we have advanced some way towards defining the role of TCOM in the assessment of problem wounds for treatment with HBOT. There remains considerable work to be done before TCOM is firmly established with a foundation of evidence expected of modern medical procedures.

**References**


1 Niinikoski J. Hyperbaric oxygen therapy of diabetic foot ulcers, transcutaneous oximetry in clinical decision making. *Wound Rep Regen*. 2003; 11: 458-61.

2 Cianci P. Advances in the treatment of the diabetic foot: Is there a role for adjunctive hyperbaric oxygen therapy? *Wound Rep Regen*. 2004; 12: 2-10.

3 Davis JC, Hunt TK, editors. *Problem wounds: the role of oxygen*. New York: Elsevier; 1988. p. vii.

4 Clark CL Jr, Wolf R, Granger D, Taylor Z. Continuous recording of blood oxygen tensions by polarography. *J Appl Physiol*. 1953; 6: 189-93.

5 Silver IA. Some observations on the cerebral cortex with an ultra micro membrane covered oxygen electrode. *Med Electr Biol Eng*. 1965; 3: 377-87.

6 Hunt T. A new method of determining tissue oxygen tension. *Lancet*. 1964; 13: 1370-1.

7 Huch R, Lubbers DW, Huch A. Quantitative continuous measurement of partial oxygen pressure on the skin of adults and newborn babies. *Pflugers Arch*. 1972; 337: 185-98.

8 Hopf HW. Development of subcutaneous wound oxygen measurement in humans: contributions of Thomas K Hunt, MD. *Wound Rep Regen*. 2003; 11: 424-30.

9 Huch R, Huch A. Fetal and maternal $P_{tc}O_2$ monitoring. *Crit Care Med*. 1981; 9: 694-7.

10 Sheffield PJ, Workman WT. Transcutaneous tissue oxygen monitoring in patients undergoing hyperbaric oxygen therapy. In: Huch R, Huch A, editors. *Continuous transcutaneous blood gas monitoring*. New York: Marcel Dekker; 1983. p. 655-60.

11 Sheffield PJ, Workman WT. Non-invasive tissue oxygen measurements in patients administered normobaric and hyperbaric oxygen by mask. *Hyperbaric Oxygen Rev*. 1985; 6: 47-62.

12 Medicare Services Advisory Committee. Application 1054. *Hyperbaric oxygen therapy for the treatment of refractory non-healing wounds in non-diabetic patients and soft tissue radiation injury*. Commonwealth of Australia; May 2003.

13 Fronek A. *Transcutaneous monitoring of $PO_2$ in the assessment of peripheral vascular disease*. Radiometer Medical A/S Denmark; 1997.

14 Sheffield PJ. Measuring tissue oxygen tension: a review. *Undersea Hyperb Med*. 1998; 25: 179-88.

15 Hauser CJ, Klein SR, Mehringer M, Appel P, Shoemaker WC. Assessment of perfusion in the diabetic foot by regional transcutaneous oximetry. *Diabetes*. 1984; 33: 527-31.

16 Hauser CJ, Klein SR, Mehringer M, Appel P, Shoemaker WC. Superiority of transcutaneous oximetry in noninvasive vascular diagnosis in patients with diabetes. *Arch Surg*. 1984; 119: 690-4.

17 Kram HB, Shoemaker WC. Diagnosis of major peripheral arterial trauma by transcutaneous oxygen monitoring. *Am J Surg*. 1984; 147: 776-80.

18 Mathieu D, Wattel F, Boucachour G, Billard V, Defoin JF. Post-traumatic limb ischaemia: prediction of final outcome by transcutaneous oxygen measurements in hyperbaric oxygen. *J Trauma*. 1990; 30: 307-14.

19 Bouachour G, Croner P, Gouello JP, Toulemonde JL, Talho A, Alquier P. Hyperbaric oxygen therapy in the management of crush injuries: A randomized double-blind placebo controlled clinical trial. *J Trauma*. 1996; 41: 333-9.

20 Wattel F, Mathieu D, Coget J-M, Billard V. Hyperbaric oxygen therapy in chronic vascular wound management. *Angiology*. 1990; 41: 59-65.

21 Wattel FE, Mathieu MD, Fossati P, Neviere RR, Coget JM. Hyperbaric oxygen in the treatment of diabetic foot lesions. Search for healing predictive factors. *J Hyperb Med*. 1991; 6: 263-8.

22 Grolman RE, Wilkerson DK, Taylor J, Allinson P, Zatina MA. Transcutaneous oxygen measurements predict a beneficial response to hyperbaric oxygen therapy in patients with non-healing wounds and critical limb ischaemia. *Am Surg*. 2001; 67: 1072-80.

23 Fife CE, Buyukecakir C, Otto G, Sheffield P, Mader J, Pontani B, Harrist R. The predictive value of transcutaneous oxygen tension measurement in diabetic lower extremity ulcers treated with hyperbaric oxygen therapy: a retrospective analysis of 1144 patients. *Wound Rep Regen*. 2002; 10: 198-207.

24 Strauss MB, Bryant BJ, Hart GB. Transcutaneous oxygen measurements under hyperbaric oxygen conditions as a predictor for healing of problem wounds. *Foot Ankle Int*. 2002; 23: 933-7.

25 Dooley J, King G, Slade B. Establishment of reference pressure of transcutaneous oxygen for the comparative evaluation of problem wounds. *Undersea Hyperb Med*. 1997; 24: 235-44.

26 Hauser CJ, Shoemaker WC. Use of transcutaneous $PO_2$ regional perfusion index for quantity tissue perfusion in peripheral vascular disease. *Ann Surg*. 1983; 197: 337-43.

27 Dowd GS, Linge K, Bentley G. The effect of age and

sex of normal volunteers upon the transcutaneous oxygen tension in the lower limb. *Clin Phys Physiol Meas*. 1983; 4: 65-8.
28 Dowd GSE, Linge K, Bentley G. Measurement of transcutaneous oxygen pressure in normal and ischaemic skin. *J Bone Joint Surg*. 1983; 65B: 79-83.
29 Coleman LS, Dowd GS, Bentley G. Reproducibility of $tcPO_2$ measurements in normal volunteers. *Clin Phys Physiol Meas*. 1986; 7: 259-63.
30 Byrne P, Provan JL, Ameli FM, Jones DP. The use of transcutaneous oxygen tension measurements in the diagnosis of peripheral vascular insufficiency. *Ann Surg*. 1984; 200: 159-65.
31 Holdich TA, Reddy,PJ, Walker RT, Dormandy JA. Transcutaneous oxygen tension during exercise in patients with claudication. *BMJ*. 1986; 292: 1625-8.
32 Ameli FM, Stein M, Provan JL, Aro L, St Louis EL. Comparison between transcutaneous oximetry and ankle-brachial pressure ratio in predicting runoff and outcome in patients who undergo aortobifemoral bypass. *Can J Surg*. 1989; 32: 428-32.
33 Ameli FM, Stein M, Provan JL, Aro L, Prosser R. Predictors of surgical outcome in patients undergoing aortobifemoral bypass reconstruction. *J Cardiovasc Surg (Torino)*. 1990; 31: 333-9.
34 Stein M, Ameli MF, Gray R, Elliott D, Grosman H, Aro L. Angiographic assessment of arterial outflow: predictive value of a new classification system. *J Vasc Interv Radiol*. 1991; 2: 365-70.
35 Mars M, McKune A, Robbs JV. A comparison of laser Doppler fluximetry and transcutaneous oxygen pressure measurement in the dysvascular patient requiring amputation. *Eur J Vasc Endovasc Surg*. 1998; 16: 53-8.
36 Wyss CR, Matsen FA, Simmons CW, Burgess EM. Transcutaneous oxygen measurements on limbs of diabetic and non-diabetic patients with arterial vascular disease. *Surgery*. 1984; 95: 339-45.
37 Liu MH, Grimm DR, Teodorescu V, Kronowitz SJ, Baumann WA. Transcutaneous oxygen tension in subjects with tetraplegia with and without pressure ulcers: a preliminary report. *J Rehabil Res Dev*. 1999; 36: 202-6.
38 Melis P, Noorlander ML, van der Kleij AJ, van Noorden CJ, van der Horst CM. Oxygenation and microcirculation during skin stretching in undermined and non-undermined skin. *Plast Reconstr Surg.* 2003; 112: 1295-301.
39 Padberg FT, Back TL, Thompson PN, Hobson RW 2nd. Transcutaneous oxygen ($TcPO_2$) estimates probability of healing in the ischaemic extremity. *J Surg Res*. 1996; 60: 365-9.
40 White RA, Nolan L, Harley D, Long J, Klein S, et al. Non-invasive evaluation of peripheral vascular disease using transcutaneous oxygen tension. *Am J Surg*. 1982; 144: 68-75.
41 Marx RE, Johnson RD, Kline SN. Prevention of osteoradionecrosis: a randomized prospective clinical trial of hyperbaric oxygen versus penicillin. *J Am Dent Assoc*. 1985; 3: 49-54.
42 Faglia E, Favales F, Aldeghi A, Calia P, Quarantiello A, et al. Adjunctive systemic hyperbaric oxygen therapy in treatment of severe prevalently ischemic diabetic foot ulcer. *Diabetes Care*. 1996; 19: 1338-43.
43 Abidia A, Laden G, Kuhan G, Johnson BF, Wilkinson AR, et al. Role of hyperbaric oxygen therapy in ischaemic diabetic lower-extremity ulcers: a double blind randomized controlled trial. *Eur J Vasc Endovasc Surg*. 2003; 25: 513-8.
44 Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen for chronic wounds. *Cochrane Database of Systematic Reviews.* 2004; 2: CD004123.
45 Bennett MH, Feldmeier J, Hampson N, Smee R, Milross C. Hyperbaric oxygen therapy for late radiation tissue injury. *Cochrane Database of Systematic Reviews.* 2005; 3: CD005005.
46 Johnson WC, Grant HI, Baldwin J, Hamilton JV, Dion JM. Supplemental oxygen and dependent positioning as adjunctive measures to improve forefoot tissue oxygenation. *Arch Surg*. 1988; 123: 1227-30.
47 Bongard O, Bounameaux H, Fagrell B. Effect of oxygen inhalation on skin microcirculation in patients with peripheral arterial occlusive disease. *Circulation*. 1992; 86: 878-86.
48 Campagnoli P, Orani G, Sala G, Meazza D, Sacchi C, et al. Prognostic value of $TcPO_2$ during hyperbaric oxygen therapy. *J Hyperb Med*. 1992; 7: 223-7.
49 Dooley J, Schirmer J, Slade B, Folden B. Use of transcutaneous pressure of oxygen in the evaluation of oedematous wounds. *Undersea Hyperb Med*. 1996; 23: 167-74.
50 Beehner MR, Marx RE. Hyperbaric oxygen induced angiogenesis and fibroplasia in human irradiated tissues. In: *Proc 65th Meeting of the Am Dent Assoc Oral Maxillofac Surg*. 1983. p. 78-9.
51 Thorn JJ, Kallevave F, Westergaard P, Hansen EH, Gottrup F. The effect of hyperbaric oxygen on irradiated oral tissues: transmucosal oxygen tension measurements. *J Oral Maxillofac Surg*. 1997; 55: 1103-7.
52 Marx RE, Ehler WJ, Tayapongsak P, Pierce LW. Relationship of oxygen dose to angiogenesis induction in irradiated tissue. *Am J Surg*. 1990; 160: 519-24.
53 Lin TF, Chen SB, Niu KC. The effects of hyperbaric oxygen therapy in treatment of early diabetic foot. *Undersea Hyperb Med*. 2001; 28(Suppl): 67.
54 Mathieu D, Neviere R, Pellerin P, Patenotre P, Wattel F. Pedicle musculocutaneous flap transplantation: Prediction of final outcome by transcutaneous oxygen measurements in hyperbaric oxygen. *Plast Reconstr Surg*. 1993; 91: 329-34.
55 Lindstrom T, Gullichsen K, Lertola K, Niinikoski J. Effects of hyperbaric oxygen on perfusion perameters and transcutaneous oxygen measurements in patients with intramedullary nailed tibial shaft fractures. *Undersea Hyperb Med*. 1998; 25: 87-91.
56 Smith BM, Desvigne LD, Slade JB, Dooley JW, Warren

DC. Transcutaneous oxygen measurements predict healing of leg wounds with hyperbaric therapy. *Wound Rep Regen*. 1996; 4: 224-9.

57 Zgonis T, Garbalosa JC, Burns P, Vidt L, Lowery C. A retrospective study of patients with diabetes mellitus after partial foot amputation and hyperbaric oxygen treatment. *J Foot Ankle Surg*. 2005; 44: 276-80.

58 Franzeck UK, Talke P, Bernstein EP, Goldbranson FL, Fronek A. Transcutaneous $PO_2$ measurements in health and peripheral arterial occlusive disease. *Surgery*. 1982; 91: 156-63.

59 Cina C, Katsamouris A, Megerman J, Brewster DC, Strayhorn EC, et al. Utility of transcutaneous oxygen tension measurements in peripheral arterial occlusive disease. *J Vasc Surg*. 1984; 1: 362-71.

60 Hauser CJ. Tissue salvage by mapping of skin surface transcutaneous oxygen tension index. *Arch Surg*. 1987; 122: 1128-30.

61 Oishi CS, Fronek A, Golbranson FL. The role of non-invasive vascular studies in determining levels of amputation. *J Bone Joint Surg*. 1988; 70A: 1520-30.

62 Wyss CR, Harrington RM, Burgess EM, Matsen FA. Transcutaneous oxygen tension as a predictor of success after an amputation. *J Bone Joint Surg*. 1988; 70A: 203-7.

63 Pecoraro RE Ahroni JH, Boyko EJ, Stensel VL. Chronology and determinants of tissue repair in diabetic lower extremity ulcers. *Diabetes*. 1991; 40: 1305-13.

64 Pinzur MS. Transcutaneous oxygen as a predictor of healing in amputations of the foot and the ankle. *Foot Ankle Int*. 1992; 13: 271-2.

65 Conlon KC, Sclafani L, DiResta GR, Brennan MF. Comparison of transcutaneous oximetry and laser Doppler flowmetry as non-invasive predictors of wound healing after excision of extremity soft-tissue sarcomas. *Surgery*. 1994; 115: 335-40.

66 Yablon SA, Novick ES, Jain SS, Graves DE. Postoperative transcutaneous oxygen measurement in the prediction of delayed wound healing and prosthetic fitting among amputees during rehabilitation. A pilot study. *Am J Phys Med Rehabil*. 1995; 74: 193-8.

67 Bunt TJ, Holloway GA. $TcPO_2$ as an accurate predictor of therapy in limb salvage. *Ann Vasc Surg*. 1996; 10: 224-7.

68 Claeys LG, Horsch S. Transcutaneous oxygen pressure as predictive parameter for ulcer healing in endstage vascular patients treated with spinal cord stimulation. *Int Angiol*. 1996; 15: 344-9.

69 Kalani M, Brismar K, Fagrell B, Jorneskog G. Transcutaneous oxygen tension and toe blood pressure as predictors for outcome of diabetic foot ulcer. *Diabetes Care*. 1999; 22: 147-51.

70 Rhodes GR, Skudder P (Jr). Salvage of ischaemic diabetic feet. Role of transcutaneous oxygen mapping and multiple configurations of in situ bypass. *Am J Surg*. 1986; 152: 165-71.

71 Lalka SG, Malone JM, Anderson GG, Hagaman RM, McIntyre KE, Bernhard VM. Transcutaneous oxygen and carbon dioxide pressure monitoring to determine limb ischaemia and to predict surgical outcome. *J Vasc Surg*. 1988; 7: 507-14.

72 Hanna GP, Fujise K, Kjellgren O, Feld S, Fife C, et al. Infrapopliteal transcatheter interventions for limb salvage in diabetic patients: importance of aggressive interventional approach and role of transcutaneous oximetry. *J Am Coll Cardiol*. 1997; 30: 664-9.

73 Poredos P, Rakovec S, Guzic-Salobir B. Determination of amputation level in ischaemic limbs using $tcPO_2$ measurement. *Vasa J Vasc Dis*. 2005; 34: 108-12.

74 Hauser CJ, Appel P, Shoemaker WC. Pathophysiologic classification of peripheral vascular disease by positional changes in regional transcutaneous oxygen tension. *Surgery*. 1984; 95: 689-93.

75 Kram HB, Appel PL, Shoemaker WC. Comparison of transcutaneous oximetry, vascular hemodynamic measurements, angiography, and clinical findings to predict the success of peripheral vascular reconstruction. *Am J Surg*. 1988; 155: 551-8.

76 Moosa HH, Peitzman AB, Makaroun MS, Webster MW, Steed DL. Transcutaneous oxygen measurements in lower extremity ischaemia: effects of position, oxygen inhalation and arterial reconstruction. *Surgery*. 1988; 2: 193-8.

77 Reiber GE, Pecoraro RE, Koepsell TD. Risk factors for amputation in patients with diabetes mellitus. A case control study. *Ann Intern Med*. 1992; 117: 97-105.

78 Ubbink DT, Jacobs MJHM, Tangelder GJ, Slaaf DW, Reneman RS. The usefulness of capillary microscopy, transcutaneous oximetry and laser Doppler fluximetry in the assessment of the severity of lower limb ischaemia. *Int J Microcirc*. 1994; 14: 34-44.

79 Ballard JL, Eke CC, Bunt TJ, Killeen JD. A prospective evaluation of transcutaneous oxygen measurements in the management of diabetic foot problems. *J Vasc Surg*. 1995; 22: 485-92.

80 Burgess EM, Matsen FA, Wyss CR, Simmons CW. Segmental transcutaneous measurements of $PO_2$ in patients requiring below the knee amputation for peripheral vascular insufficiency. *J Bone Joint Surg*. 1982; 64A: 378-82.

81 Katsamouris A, Brewster DC, Megerman J, Cina C, Darling RC, Abbott WM. Transcutaneous oxygen tension in selection of amputation level. *Am J Surg*. 1984; 147: 510-7.

82 Harward TRS, Volny J, Golbranson F, Bernstein EF, Fronek A. Oxygen inhalation-induced transcutaneous $PO_2$ changes as a predictor of amputation level. *J Vasc Surg*. 1985; 2: 220-7.

83 Dowd GS. Predicting stump healing following amputation for peripheral vascular disease using the transcutaneous oxygen monitor. *Ann R Coll Surg Engl*. 1987; 69: 31-5.

84 Bongard O, Krähenbuhl B. Predicting amputation in severe ischaemia. *J Bone Joint Surg*. 1988; 70B: 465-7.

85 Chambon JP, Desmyttere J, Grard C, Alsberghe MC,

Devulder B, Quandalle P. Study of static transcutaneous oximetry before amputation in patients with arteritis. *J Chir (Paris)*. 1992; 129: 352-6.

86 Scheffler A, Rieger H. A comparative analysis of transcutaneous oximetry ($tcPO_2$) during oxygen inhalation and leg dependency in severe peripheral arterial occlusive disease. *J Vasc Surg*. 1992; 16: 218-24.

87 Ray SA, Buckenham TM, Bellie A-M, Taylor RS, Dormandy JA. The predictive value of laser Doppler fluximetry and transcutaneous oximetry for clinical outcome in patients undergoing revascularization for severe leg ischaemia. *Eur J Vasc Endovasc Surg*. 1997; 13: 54-9.

88 Stalc M, Poredos P. The usefulness of transcutaneous oximetry in assessing the success of percutaneous transluminal angioplasty. *Eur J Vasc Endovasc Surg*. 2002; 24: 528-32.

89 Wagner HJ, Schmitz R, Alfke H, Klose KJ. Influence of percutaneous transluminal angioplasty on transcutaneous oxygen pressure in patients with peripheral arterial obstructive disease. *Radiology*. 2003; 22: 791-7.

90 Caselli A, Latini V, Lapenna A, Di Carlo S, Pirozzi F, et al. Transcutaneous oxygen tension monitoring after successful revascularization in diabetic patients with ischaemic foot ulcers. *Diabetic Med*. 2005; 22: 460-5.

91 DeGraaff JC, Ubbink DT, Legemate DA, Tijssen JGP, Jacobs MJHM. Evaluation of toe pressure and transcutaneous oxygen measurements in management of chronic leg ischaemia: A diagnostic randomized clinical trial. *J Vasc Surg*. 2003; 38: 528-34.

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# The state of oxygen-enriched air (nitrox)

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**Key words**
Diving, scuba diving, enriched air - nitrox

**Abstract**

(Lang MA. The state of oxygen-enriched air (nitrox). *Diving and Hyperbaric Medicine*. 2006; 36: 87-93.)
The evolution of the use of oxygen-enriched air (nitrox) in diving can be traced to its origin in 1874, its use in the scientific diving community in 1979, and its introduction on a global scale to the recreational diving community in 1985. As with any emerging technology that has found a broader market appeal, controversies invariably arise. In 2000, the DAN Nitrox Workshop reviewed physiological issues as they pertain to recreational nitrox use such as carbon dioxide retention, oxygen toxicity potential, and narcosis, and nitrox decompression sickness (DCS) incidence rates compared to air. In collaboration with the recreational diving industry, nitrox equipment engineering considerations vis-à-vis the 40%-oxygen-cleaning rule, nitrox training, and operational data were also reviewed. To provide an update on the state of nitrox, the workshop information is synthesized and additional new certification and operational data reviewed for 2000–2005.

## Introduction

The use of oxygen-enriched air (nitrox) has been a mainstream recreational diving mode since it was first introduced to sport divers in 1985 by former NOAA Deputy Diving Officer Dick Rutkowski. The mainstream recreational diving training associations (PADI, NAUI, and SSI) now support nitrox training programmes in addition to their traditional open-circuit compressed-air scuba programmes. The training organisations that focus on technical diving (IANTD, ANDI, and TDI) have amassed several additional years of experience in providing nitrox training to the recreational diving community.

In 2000, the DAN Nitrox Workshop[1] was prioritised as a diving safety project of interest to the diving industry by means of Divers Alert Network financial support. As with any emerging technology that has found a broader market appeal, controversies invariably arise. Ignorance, myths, and misconceptions often fuel opposite views. A critical interdisciplinary examination of the current issues surrounding nitrox was in order to disseminate credible diving safety information. This forum was provided to objectively evaluate the available operational and physiological data relating to the use of nitrox, and discuss risk management, equipment, and training parameters.

An approximation of the magnitude of nitrox consumption was essential. This seemed achievable by our ability to provide a denominator of nitrox divers and nitrox dives, as a sub-set of the overall level of recreational diving activity. Many other discussions of nitrox-related topics flowed from these numbers, i.e., comparable incidence rates of decompression sickness (DCS) in nitrox and compressed-air use, and growth of nitrox training and equipment sales.

Physiological issues such as carbon dioxide retention and oxygen toxicity were also in need of critical examination. Nitrox training and equipment issues were discussed to comprehensively address risk management and legal considerations regarding its use. The recreational diver is the ultimate beneficiary of our improved collective knowledge of the state of the art of nitrox diving in 2000. The intermediary beneficiaries of this information are the providers and manufacturers of nitrox products (instructors, equipment manufacturers, dive stores, and nitrox dispensers).

## The history of nitrox

Hamilton discussed the evolutionary history of nitrox diving along the following timeline:[2]

**1874** H Fleuss probably made the first nitrox dive with a rebreather.

**1947** C Lambertsen published the first nitrox paper.[3]

**1955** E Lanphier described the use of nitrogen-oxygen mixtures in diving and the equivalent air depth method for using a standard air table with an enriched air mix.[4]

**1960s** A Galerne used on-line blenders for commercial diving.

**1979** M Wells developed NOAA nitrox and equivalent air depth (EAD) tables were published in the NOAA diving manual for scientific diving.

**1985** D Rutkowski developed a nitrox training programme for recreational diving.

**1989** The Harbor Branch Oceanographic Institution Nitrox Workshop addressed the following issues and rationale:[5]
- oxygen limits
- decompression and the EAD
- nitrox mixing

- terminology: the term 'nitrox', borrowed from habitat diving, implies that nitrogen is the advantage. The US Navy now prefers 'oxygen-nitrogen'. New NOAA designations are $NN_{32}$ and $NN_{36}$. $EAN_x$ was agreed upon, with 'x' being the percentage of oxygen ($O_2$). The correct term proposed was 'oxygen-enriched air'.

**1991** 'Not Invented Here' went to work. Bennett, Bove, and *SkinDiver Magazine* all took stands against nitrox use by recreational divers.[6,7]

**1992** The Scuba Diving Resources Group (a committee of the Outdoor Recreation Coalition of America) organised a nitrox workshop in response to nitrox agencies and products being denied access to the Diving Equipment and Marketing Association (DEMA) trade show in Houston.[8] The workshop resulted in the following endorsements:
- the EAD principle
- the NOAA limits for oxygen exposure (but lower limits were encouraged)
- the use of normal DCS treatment procedures for air diving after nitrox dives (the $O_2$ exposure of recreational nitrox dives should not affect treatment)
- pending testing, mixes up to 40% $O_2$ could be used in equipment suitable for air provided equipment was clean and $O_2$-compatible lubricants were used
- dry nitrox would not corrode cylinders and other gear appreciably faster than air
- air for mixing should be 'oil free'
- cylinders used for nitrox should be compatible with $O_2$
- mixes should be analysed properly before use, and
- mixing in standard cylinders by adding $O_2$ and topping with air is considered unsafe.

**1993** The aquaCorps TEK93 conference took place in San Francisco. A measurable and attainable air quality standard was set by nitrox industry leaders at 0.1 mg/m$^3$ oil.

**1993** The Canadian Forces issued EAD tables, based on the standard air tables, with an upper $O_2$ limit of 1.5 ATA $PO_2$ and depth and time limits more stringent than the air tables.[9]

**1996** PADI takes the plunge, nitrox has arrived. NAUI, SSI, and even BSAC have nitrox programmes.[10] The diving media have become supportive of nitrox.

**1999** A survey by RW Hamilton for the US Navy showed 100,000s of (not well-documented) open-circuit nitrox dives. Commercial diving does not use nitrox much, but it has become fashionable among recreational divers. The DCS incidence record is good, and nitrox dive computers are readily available.

**1999** The Occupational Safety and Health Administration (OSHA) was petitioned by PADI and Oceanic in 1995 on behalf of Dixie Divers, Inc. for a recreational nitrox variance for scuba instructors from commercial diving regulations that was approved for:
- $PO_2$ of 1.4 ATA and a maximum 40 per cent nitrox mix
- 130 feet maximum depth and dives within the no-stop limits
- a stand-by diver, and
- diving within one hour of a chamber.

**2001** NOAA diving manual includes a chapter as stand-alone course guide for nitrox diving.

## The physiology of nitrox

### DECOMPRESSION

Nitrox improves decompression, which is based on the fraction of nitrogen ($N_2$) only. Therefore, more $O_2$ and less $N_2$ is better. Nitrox allows for greater bottom times for no-stop dives. Decompression dives (with required stops) using an enriched-air mix will result in a total decompression time shorter than that required with air. When nitrox is breathed and air decompression tables are used, the decompression times are not affected, but the dives are considered more conservative. This benefit can apply to repetitive dives, flying after diving, and diving at altitude.

### OXYGEN TOXICITY

Convulsions from central nervous system (CNS) toxicity can occur without warning and likely lead to loss of the mouthpiece and subsequent drowning. Warning signs and symptoms, if they do occur, include: visual disturbances (including tunnel vision); tinnitus; nausea; twitching or muscle spasms (especially in the face); irritability, restlessness, euphoria or anxiety; and dizziness. Thus, the diver's exposure to high levels of oxygen must be managed by time limits at maximum $PO_2$ (Table 1.) Standardised recreational nitrox depth limits are 110 feet of sea water (fsw) ($EAN_{36}$) and 130 fsw ($EAN_{32}$). Pulmonary or whole-body oxygen toxicity is monitored by oxygen toxicity units (OTU) or units pulmonary toxicity dose (UPTD). Because of the length of exposure time required to elevate oxygen

**Table 1**
**NOAA oxygen exposure limits[12]**

| $PO_2$ (ATA) | Maximum single dive (mins) | Maximum 24 hrs (mins) |
|---|---|---|
| 1.60 | 45 | 150 |
| 1.50 | 120 | 180 |
| 1.40 | 150 | 180 |
| 1.30 | 180 | 210 |
| 1.20 | 210 | 240 |
| 1.10 | 240 | 270 |
| 1.00 | 300 | 300 |

**Table 2**
**$PO_2$ limits adopted by the Israeli Navy**

| Degree of retention | End-tidal $CO_2$ (torr) | Mixed-expired $CO_2$ (torr) | Maximum $PO_2$ (ATA) |
|---|---|---|---|
| None | <50 | <41 | 1.6 |
| Moderate | 50–55 | 41–45 | 1.4 |
| Extreme | >55 | >45 | 1.2 |

levels, the onset of CNS effects is unlikely to occur in recreational diving applications. Whole-body symptoms include primarily pulmonary effects (coughing, chest pain, and a reduction in vital capacity) and more diffuse symptoms (paraesthesiae, numbness of fingertips and toes, headache, dizziness, nausea, and a reduction in aerobic capacity).

## NARCOSIS

Nitrogen narcosis in oxygen-enriched air diving is not a real issue. However, $O_2$ can be as narcotic as nitrogen[13] but nitrox diving is not efficient at depths where narcosis becomes prominent.

## $CO_2$ RETENTION

$CO_2$ build-up is not an issue for recreational nitrox mixes, but may be a hazard in the deeper range of nitrox diving.[14] It causes a reduced ventilatory response, such that breathing a dense mix while exercising can lead to unconsciousness. Headaches are a symptom of hypercapnia, caused by dilation of the arterial vessels in the brain. Kerem et al discuss the Israeli Navy experience with pure $O_2$ rebreathers,which shows victims of CNS $O_2$ toxicity to be both retainers and late detectors of build-up of inspired $CO_2$/malfunctioning absorbers. [15] For higher-risk, extreme $CO_2$ retainers, more conservative $PO_2$ limits were adopted by the Israeli Navy (Table 2).

## NITROX EFFECTS

The late Jon Hardy initiated a study of human function to test nitrox as a product in 1999.[16] Does diving with nitrox as the breathing gas cause:

- less nitrogen narcosis?
- less fatigue?
- less gas consumption?
- better thermal balance?
- less decompression stress?

Initial results showed no variation in gas consumption between air and nitrox under similar conditions. Difficulty was acknowledged in experimentally designing a study to objectively measure fatigue, decompression stress, and thermal balance. Unfortunately, testing of the reduced nitrogen narcosis of nitrox was not completed by Hardy. More recently, in a double-blinded, randomised controlled study, 11 divers carried out dives breathing either air or $EAN_{36}$ at 18 msw in a dry chamber for 40-minute bottom times.[17] Divers were assessed before and after two exercise periods during the dive. These chamber exposures produced no measurable difference in fatigue, attention levels, or ability to concentrate.

**Table 3. Manufacturers' nitrox equipment recommendations (modified from Oliver[18])**

| Company | Maximum $fO_2$ authorised (%) | | | |
|---|---|---|---|---|
| | 23.5 | <41 | <51 | 100 |
| Apeks | | 1 | | |
| Aqua-Lung | | 1 | | |
| Atomic | | 1 | | |
| Beuchat | | | 2 | |
| Cressi-Sub | x | | | |
| Dacor (parent company policy) | | 2 | | |
| Dive-Rite | | 2 | | |
| Genesis | | 4 | | |
| International Divers Inc. | | 1 | | |
| Kirby-Morgan | | | 1 | |
| Mares America | | 2 | | |
| Oceanic | | | 2 | |
| OMS | | | | 1 |
| Sherwood Scuba | | 4 | | |
| Scubapro | | 1 | | 2,4 |
| Thermo valve | | 2 | | |
| Zeagle (policy reevaluated) | | 3 | | 4 |

**Key code - Enriched Air Nitrox (EAN) Sep 00**

x Maximum limit. EAN not recommended.
1 All models are factory-prepared for EAN using $O_2$ compatible materials.
2 Designated models factory-prepared for EAN using $O_2$ compatible materials.
3 Standard air components declared acceptable. Viton o-rings available.
4 Conversion components available for installation by technician qualified to prepare for $O_2$ service.

## Nitrox equipment

Oliver summarised the findings and conclusions of the DEMA Manufacturers Committee on oxygen-enriched air and provided manufacturers' recommendations on nitrox and equipment use.[18]

Two major manufacturers (Scubapro and Aqualung) issued technical bulletins in 2001 on the use of their equipment with nitrox:

SCUBAPRO ENGINEERING BULLETIN #271 (05 SEPTEMBER 2001)

- All Scubapro regulators sold after October 2000 are approved for use with nitrox up to 40% $O_2$ and for an operating pressure not to exceed 3300 psi. The regulators can be used with gases under the restrictions listed above straight out of the box. Specific models are listed by Scubapro.
- For use with gases (other than air) falling outside of the range detailed above (i.e., 40+% $O_2$, 3300+ psi), the only approved regulator is the MK20 (brass version only) after appropriate cleaning and installation of the nitrox kit, when the operational limit becomes 100% $O_2$ to 3500 psi.

AQUALUNG AND APEKS REGULATORS

- New Aqualung and Apeks regulators are now EAN compatible up to 40% $O_2$ right out of the box. See <www.aqualung.com>, technical library–nitrox compatibility and converting existing regulators to $EAN_{40}$ use.
- Owner's responsibility is to maintain cleanliness of the regulator and cleaning procedures (note switches from air to nitrox). Second-stage cleaning prevents cross-contamination.
- Difference in the regulators is in the manufacturing process (i.e., a regulator 'safe' room). Hyperfiltered air (condensed hydrocarbons < 0.1 mg/m$^3$) is used for testing, as are some oxygen-compatible components.

Table 3 shows updated manufacturers' recommendations.

**Nitrox training and operational data**

Table 4 lists the nitrox training requirements for the recreational and scientific diving communities.

Table 5 presents updated (until 2005) nitrox instructor and diver certification information since the original data published in 2001. For reference, Table 6 shows total numbers of entry-level open-water scuba certifications as collected by DEMA for 2000–2005.[19] Finally, Table 7 is likewise updated for available nitrox and air exposures and cases of DCS.

Vann concluded that laboratory and open-water experience suggests that nitrox diving may be practised with low risks of DCS and $O_2$ toxicity.[20] From DAN data on mixed-gas diving dating from 1990 for diving fatalities, from 1995 for diving injuries and from 1997 for safe dives:

- a higher proportion of safe divers used nitrox than of divers who were injured or died
- nitrox divers were older than air divers
- over 60% of nitrox divers who dived safely had specialty training
- safe nitrox diving was most common aboard charter boats and there were no air or nitrox fatalities from liveaboards
- nitrox divers who dived safely dived fewer dives over more days than did air divers
- in general, nitrox divers dived deeper than air divers, regardless of whether they dived safely, were injured, or died
- for either air or nitrox, injured divers and diving fatalities had higher proportions of rapid ascent and running out of gas than did safe divers
- maximum $PO_2$ was above 1.3 ATA for half of the 74 injured nitrox divers
- while the incidence of $O_2$ toxicity during nitrox diving is unknown, convulsions and/or unconsciousness were reported for three divers who had a maximum $PO_2$ of 1.4, 1.6, and 1.9 ATA respectively
- careful depth control is important to avoid excessively high $PO_2$ during nitrox diving.

**Table 4**
**Recreational and science/government training requirements**

| | IANTD | ANDI | TDI | PADI | NAUI | SSI | NOAA | NASA | AAUS | UNCW |
|---|---|---|---|---|---|---|---|---|---|---|
| **Max $PO_2$ limit (ATA)** | 1.6 | 1.6 | 1.6 | 1.4 | 1.4 | 1.6 | 1.6 | 1.6 | 1.6 | 1.6 |
| **$O_2$ content range (%)** | 22-40 | 22-50 | 22-40 | 22-40 | 22-40 | 22-40 | 32 & 36 | 46 | 22-40 | 28-40 |
| **$O_2$ cleaning (%)** | >40 | >21 | >40 | Mfr | >40 | >40 | >40 | >23 | n/a | >40 |
| **$O_2$ limits (ATA)** | all agencies NOAA | | | | | | | | | |
| **OTU/UPTD** | 300/day | n/a | n/a | n/a | 350/day | NOAA | Repex | 415/day | Repex | Repex |
| **Mix analysis accuracy** | all agencies ± 1% | | | | | | | | | |
| **$EAN_x$ table/DC** | T/DC | T/DC | T/DC | T/DC | T/DC | T/DC | T/DC | T | T/DC | T/DC |
| **Agency tables** | Y | Y | NOAA | Y | Y | Y | NOAA | USN | NOAA | USN |
| **Table model** | B-Pi$N_2$ | B-Pi$N_2$ | USN-EAD | Rogers-RDP | RGBM-USN | USN-EAD | USN99-EAD | USN-EAD | mUSN99-EAD | mUSN99-EAD |
| **Encourage DC** | Y | Y | Y | Y | Y | Y | N | n/a | n/a | n/a |
| **Prerequisites** | none | none | OW | OW | none | OW | n/a | n/a | n/a | n/a |

(B – Buhlmann; DC – dive computer; EAD – equivalent air depth; m – modified; Mfr – manufacturer's recommendation; OW – open water certification; RGBM – reduced gradient bubble model; Rogers-RDP – recreational dive planner; USN – US Navy; USN99 – US Navy 1999 dive tables (unpublished))

**Table 5**
**Available nitrox diver certification data up to November 2000 as reported by organisation representatives at the 2001 workshop[1] on oxygen-enriched air diving and then thereafter**

| Period: | | Until November 2000 | | November 2000 to November 2005 | | |
|---|---|---|---|---|---|---|
| **Level:** | **From** | **Divers** | **Instructors** | **Instructors** | **Divers** | **Region** |
| NAUI* | 1992– | 4,472 | 878 | 10,221 | 92,859 | Worldwide |
| PADI | 1996– | 46,788 | 7,274 | 24,817 | 223,932 | Worldwide |
| SSI | 1996– | 1570 | 605 | 1,500 | 12,417 | USA |
| IANTD | 1991– | 64,378 | 8,140 | 6,140 | 89,049 | Worldwide |
| TDI | 1994– | 66,206 | 12,823 | 8,758 | 51,592 | Worldwide |
| ANDI | 1989– | 49,118 | 3,196 | 5,350 | 81,200 | Worldwide |
| UNCW | 1986– | 803 | n/a | 8 | 523 | USA |
| NOAA | 1981– | 139 | n/a | n/a | 323 | USA |
| NASA | 1996– | 384 | 8 | n/a | n/a | USA |
| AAUS | 1987– | n/a | n/a | n/a | n/a | USA |
| **TOTAL** | | **233,858** | **32,924** | **56,794** | **551,895** | |

(*NAUI instructor number increase (2000–2005) results from their authorisation to teach nitrox in addition to compressed-air scuba; n/a – data either not tracked organisationally, or not available; nitrox certifications for divers participating in Aggressor and Sea Hunter fleet courses are included in the totals of the training agencies)

## Discussion and conclusions

The DAN nitrox workshop concluded the following in 2000 for entry-level, recreational, open-circuit nitrox diving:

- no evidence was presented that showed an increased risk of DCS with the use of oxygen-enriched air (nitrox) versus compressed air
- a maximum $PO_2$ of 1.6 ATA was accepted based on the history of nitrox use and scientific studies
- routine $CO_2$ retention screening is not necessary
- $O_2$ analysers should use a controlled-flow sampling device
- $O_2$ analysis of the breathing gas should be performed by the blender and/or dispenser and verified by the end user
- training agencies recognise the effectiveness of dive computers
- there is no need to track whole-body exposure to $O_2$ (OTU/UPTD)
- use of the 'CNS oxygen clock' concept, based on NOAA $O_2$ exposure limits should be taught. However, it should be noted that CNS oxygen toxicity could occur suddenly and unexpectedly
- no evidence was presented, based on history of use, to show an unreasonable risk of fire or ignition when using up to 40% nitrox with standard scuba equipment. The level of risk is related to specific equipment configurations and the user should rely on the manufacturer's recommendations.

Additional data collected for 2000–2005, while insufficient for statistical purposes (due to some data categories not being tracked organisationally and therefore remaining unknown), serve to show several trends. The certification numbers of nitrox instructors and divers has approximately doubled, and there does not appear to be a commensurate doubling of nitrox DCS incidence rates. However, comparisons of DCS probabilities between compressed air and nitrox remain tenuous at best. Yet, over one million more nitrox dives (from fill data) were done in the last five years than in the history of its use until November 2000. Liveaboard diving operations report almost exclusive nitrox and dive-computer use aboard their vessels. Due to their operations at remote dive locations and given the nature of their captive diver audiences (i.e., adequate time for reporting of DCS symptoms prior to returning to port), one would expect any significant DCS rates from nitrox diving to be readily apparent.

The Diving Equipment and Marketing Association reported almost one million entry-level open-water scuba certifications and the nitrox training organisations reported over 500,000 nitrox certifications. The relationship or overlap between nitrox and open-water certifications cannot be defined at this point in time due to data collection criteria. The maximum $PO_2$ limit of 1.6 ATA continues to be used

**Table 6**
**Numbers of entry-level, open-water scuba certifications as reported by DEMA (2005) based on records from NAUI, PADI, SDI, and SSI[19]**

| Year | No. certifications |
|---|---|
| 2000 | 185,714 |
| 2001 | 198,241 |
| 2002 | 183,394 |
| 2003 | 173,476 |
| 2004 | 173,225 |
| 2005 (Jan–June) | 74,758 |
| **Total** | **988,808** |

**Table 7**
**Available nitrox and air dive data for occurrence of decompression sickness (DCS) as reported at the 2001 workshop[1] and then thereafter**

| Period: | Until November 2000 | | | | November 2000 to November 2005 | | | |
|---|---|---|---|---|---|---|---|---|
| | Nitrox fills | DCS | Air fills | DCS | Nitrox fills | DCS | Air fills | DCS |
| NAUI | 17,604 | 0 | n/a | n/a | 3,242,309 | n/a | n/a | n/a |
| PADI | n/a | 17 | n/a | n/a | n/a | n/a | n/a | n/a |
| SSI | n/a | 0 | n/a | n/a | n/a | n/a | n/a | n/a |
| IANTD | 1,411,266 | 0 | n/a | n/a | n/a | n/a | n/a | n/a |
| TDI | n/a | 0 | n/a | n/a | n/a | n/a | n/a | n/a |
| ANDI | 967,450 | 0 | n/a | n/a | n/a | n/a | n/a | n/a |
| Ocean Divers | 26,000 | n/a | 235,504 | n/a | 34,000 | 0 | n/a | n/a |
| UNCW | 23,407 | 5 | 21,201 | n/a | 13,365 | 0 | 18,911 | 1 |
| NOAA | 4,894 | 1 | 156,697 | 22 | 15,618 | 2 | 64,757 | 18 |
| NASA | 34,651 | 0 | n/a | n/a | 45,635 | n/a | 0 | 0 |
| AAUS | 18,461 | 1 | 442,679 | 27 | 52,325 | 3 | 518,695 | 14 |
| Aggressors | 33,778 | 1 | n/a | 11 | 127,759 | n/a | n/a | n/a |
| Sea Hunter | 30,400 | 0 | n/a | n/a | 130,600 | 0 | 15,000 | 0 |
| **TOTAL** | **2,567,911** | **25** | **856,081** | **60** | **3,661,611** | **5** | **617,363** | **33** |

with no documented ill effects. No further issues have arisen from manufacturers with respect to their equipment being used with nitrox without incidental exposure to oxygen content above 40%.

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**References**

1 Lang MA, editor. *Proceedings of the DAN Nitrox Workshop, November 3-4, 2000*. Durham, NC: Divers Alert Network; 2001. p. 197.
2 Hamilton RW. A brief history of diving with oxygen-enriched air using open-circuit scuba apparatus. In: Lang MA, editor. *Proceedings of the DAN Nitrox Workshop, November 3-4, 2000*. Durham, NC: Divers Alert Network; 2001. p. 5-18.
3 Lambertsen CJ. Problems of shallow water diving. Report based on experiences of operational swimmers at the Office of Strategic Services. *Occup Med*. 1947; 3: 230.
4 Lanphier EH. Use of nitrogen-oxygen mixtures in diving. In: Goff LG, editor. *Underwater physiology. Proceedings of the Underwater Physiology Symposium*. Publication no. 377. Washington, DC: National Research Council, National Academy of Sciences; 1955.
5 Hamilton RW, Crosson DJ, Hulbert AW, editors. *Harbor Branch Oceanographic Institution Workshop on Enriched-Air Nitrox Diving*. Report 89-1. Rockville, MD: NOAA National Undersea Research Program; 1989. p. 74.
6 Bennett PB. Nitrox for recreational diving? *DAN Alert Diver*. Nov/Dec 1991. p. 5-6.
7 Bove AA. Nitrox: for sport diving? *Pressure*. 1992; 21: 1.
8 Hamilton RW. *Evaluating enriched air ("nitrox") diving technology*. Boulder, CO: Scuba Diving Resources Group; 1992. p. 22.
9 DCIEM. Open-circuit nitrogen-oxygen diving procedures: Appendix D. In: *DCIEM diving manual*. DCIEM 86-R-35. North York, ON: Defence and Civil Institute of Environmental Medicine; 1993
10 British Sub-Aqua Club. The BSAC nitrox decompression tables for 21% air, 32% and 36% oxygen mixes. South Wirral, Cheshire, UK: British Sub-Aqua Club; 1995.
11 OSHA. Grant of permanent variance to Dixie Divers. Federal Register 64: 71242-71261. 1999.
12 NOAA. *NOAA diving manual: Diving for science and technology*. 4th ed. Silver Spring, MD: NOAA Office of Undersea Research, US Department of Commerce; 2001.
13 Linnarsson D, Ostlund A, Sporrong A, Lind F, Hamilton RW. Does oxygen contribute to the narcotic action of hyperbaric air? In: Sterk W, Geeraedts L, editors. *Proceedings of the XVIth Meeting of the European Undersea Biomedical Society*. Amsterdam: Foundation for Hyperbaric Medicine; 1990.
14 Lanphier EH, Bookspan J. Is nitrox really the way to go? In: Naraki N, Taya Y, Mohri M, editors. *Proceedings*

*of the 13th meeting of the UJNR Panel on Diving Physiology, 1995 Oct 23-25; Miura, Kanagawa, Japan*. Yokosuka, Japan: Japan Marine Science and Technology Center; 1996.
15 Kerem DH, Arieli R, Daskalovic Y, Shupak A, Eynan M. Nitrox and $CO_2$ retention. In: Lang MA, editor. *Proceedings of the DAN Nitrox Workshop, November 3-4, 2000*. Durham, NC: Divers Alert Network; 2001. p. 81-6.
16 Hardy J. Testing nitrox as a product. In: Lang MA, editor. *Proceedings of the DAN Nitrox Workshop, November 3-4, 2000*. Durham, NC: Divers Alert Network; 2001. p. 98-9.
17 Harris RJD, Doolette DJ, Wilkinson DC, Williams DJ. Measurement of fatigue following 18 msw dry chamber dives breathing air or enriched air nitrox. *Undersea Hyp Med*. 2003; 30: 285-91.
18 Oliver WN. Nitrox and recreational diving equipment compatibility: preliminary findings by DEMA Manufacturers Committee. In: Lang MA, editor. *Proceedings of the DAN Nitrox Workshop, November 3-4, 2000*. Durham, NC: Divers Alert Network; 2001. p. 139-44.
19 DEMA. Entry-level certified diver count [Certification census 2005]. San Diego, CA: Diving Equipment and Marketing Association; 2005. Accessed 21 May 2006. Available from: <http://www.dema.org/associations/1017/files/CertCensus2005-Q1-Q21.ppt>
20 Vann RD. 2001. Nitrox diving data review. In: Lang MA, editor. *Proceedings of the DAN Nitrox Workshop, November 3-4, 2000*. Durham, NC: Divers Alert Network; 2001. p. 19-29.

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# Work in progress

## The outcome of chronic wounds following hyperbaric oxygen therapy: a prospective cohort study – the first year interim report

Glen C Hawkins, Michael H Bennett, Annelies E van der Hulst

**Key words**
Chronic wounds, hyperbaric oxygen therapy, research

**Abstract**

(Hawkins GC, Bennett MH, van der Hulst AE. The outcome of chronic wounds following hyperbaric oxygen therapy: a prospective cohort study – the first year interim report. *Diving and Hyperbaric Medicine*. 2006; 36: 94-98.)
**Introduction:** The treatment of chronic wounds is a major health cost. This study is an ongoing prospective cohort looking at the effects of hyperbaric oxygen therapy (HBOT) on the healing of chronic wounds.
**Methods:** Data are being collected from patients presenting to hyperbaric facilities in Australia with chronic (> 3 months' duration) non-irradiated wounds, including details of aetiology, wound characteristics and possible predictors of wound healing. Participants are being enrolled whether or not a decision was made to treat with HBOT. Assessments are performed at the end of the course of HBOT and at one, six and 12 months after hyperbaric treatment. The aim is to quantify the proportion healed and to identify any significant predictors for wound healing.
**Results:** There are 110 participants included in this analysis with 88 receiving HBOT. Excluding the miscellaneous aetiologies, at the end of treatment 52.3% of patients had a 'good' outcome to the wound, increasing to 64.1%, 91.7% and 78.2% at one, six and 12 months respectively. Logistic regression for participants with diabetic wounds suggests that wound area, chronicity and transcutaneous oxygen readings on room air combine to produce a statistically significant model for prediction of wound healing at one month after treatment.
**Conclusions:** This ongoing cohort study suggests that HBOT is highly associated with the healing of chronic wounds in the patients in this study. The wound area at presentation, the duration of the wound and the transcutaneous oxygen pressure on air may predict the likelihood of a chronic wound in diabetic patients healing by one month after treatment.

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### Introduction

Chronic wounds are defined as an interruption in the continuity of the skin where conventional treatment has not achieved healing within a reasonable time (e.g., 3 months).[1] Such wounds are an increasing burden on healthcare systems throughout the world. Studies have shown a prevalence in hospital patients of up to 24%, and 2% of the general population have some form of chronic wound at any one time.[2–5] This creates a significant financial burden on funding agencies with costs exceeding several billion dollars per year.[6–8] There are compelling reasons to deliver treatment modalities that are cost effective to individuals with such wounds.

Treatment regimens for chronic wounds are multi-modal but have been traditionally of two types: specific treatments designed to reduce the effect of the underlying disease (such as tight glycaemic control in diabetics and compression bandages in venous insufficiency), and wound environment optimisation dressings (e.g., hydrocolloid gels and antibacterial impregnated dressings).

The rationale for adjunctive hyperbaric oxygen therapy (HBOT) in chronic wound care is the premise that the underlying problem in many of these wounds is hypoxia. While acute wounds require low oxygen tensions, low pH and a high lactate load to initiate angiogenesis and wound healing,[9,10] later phases of healing are critically dependent on oxygen, e.g., fibroblastic collagen deposition and macrophage bacteriocidal activity.[11–13] It has been suggested that the stimulus for healing is a rapid drop in the partial pressure of oxygen from surrounding healthy tissue to the wound. In chronic wounds there is a much more gradual drop across the wound margin and this may inhibit healing significantly.[14]

The Medicare Services Advisory Committee (MSAC) was established in 1997 to advise the Australian Minister for Health and Ageing on the safety and cost effectiveness of new medical technologies and procedures, and to make recommendations for funding under the Medicare Benefits Scheme.[15] One such review was initiated into the provision of HBOT, and in 2001 MSAC recommended that a properly conducted prospective trial should be undertaken on the treatment of chronic non-irradiated wounds with HBOT. This report presents the first results of a prospective cohort of patients enrolled since June 2004.

## Methods

All hyperbaric facilities in Australia and New Zealand were invited to participate in the study. There are currently 13 such chambers treating patients for chronic wounds. Three facilities (Prince of Wales Hospital, Sydney (POW), Wesley Hospital, Brisbane (WES) and Royal Hobart Hospital, Hobart (HOB)) have been able to start in the first year and three other facilities are currently awaiting ethics approval or the conditions of their approval have not permitted submission of data in the first year. No enrolments were undertaken prior to obtaining approval from the relevant local ethics committee. Data were collected on each patient by each facility and an identifying number was included in the data collection sheet that allowed each centre to follow individual patients' progress through the four reporting stages. At the collection centre (POW) each individual patient was given a code number to identify the enrolling centre they were from and order of enrolment. Analysis was performed on the POW code numbered datasheets entered into a computer database.

### PATIENT SELECTION

All patients referred to a hyperbaric facility for assessment of one or more chronic wounds (present for more than three months) are eligible for inclusion, regardless of prior therapy. Patients considered unsuitable for HBOT due to the presence of a contra-indication, inadequate prior therapy or anticipated lack of response are therefore also eligible for enrolment. Acute (including extensive debridement within three months) wounds and those due to irradiation tissue injury are excluded from the study. The study authors did not determine assessments or impose HBOT schedules on the study centres as no definitive treatment schedule has been shown to be better than any other. We also feel that this allows the study to reflect 'true practice' in the hyperbaric field, with the variety of equipment available in each centre also influencing clinical practice.

### DATA COLLECTION

A standardised datasheet was developed that recorded demographic data, possible factors contributing to poor wound healing, treatment up to the date of assessment, subsequent hyperbaric treatment (if performed) and outcome immediately following HBOT as well as at one month, six months, and twelve months after HBOT.

Data were collected on a Filemaker Pro™ database (v7, Filemaker Inc, Santa Clara, California). Each patient was given a designated identification number for tracking through the four assessment times. Each facility is responsible for data collection on the subjects enrolled at that facility. Units are being encouraged to use all means at their disposal to locate missing subjects, including direct contact and through their local medical services and family members. Each unit was reminded at the appropriate times when a patient was due for re-assessment.

**Table 1**
**Clinical outcome scores for wounds**

| Clinical description | Category | Outcome class | Outcome |
|---|---|---|---|
| Deceased | 1 | No benefit | BAD |
| Nil benefit ± major amputation | 2 | | |
| Minimal benefit + minor amputation | 3 | Some benefit | |
| Improved + minor amputation | 4 | | |
| Substantially healed | 5 | Healed | GOOD |
| Healed | 6 | | |

### OUTCOMES

Outcomes are scored on a six-point scale originally developed by Dr Harry Oxer (Davis FM, personal communication, 2003) at Fremantle Hospital Hyperbaric Medicine Unit. However, for this interim assessment we categorised all outcomes as either 'good' or 'bad' as shown in Table 1. We have specifically placed amputations of any sort into the 'bad' outcome category because this seems an appropriately conservative approach to assessing the effectiveness of HBOT. While an amputation may indicate a good outcome (e.g., saved limb but lost toes) or poor outcome (e.g., superficial foot ulcer but lost toe) there may be no clear indication which is the case for any individual patient. In addition, any amputation will alter the location and dynamics of the wound – essentially converting a chronic wound into an acute surgical wound. We planned an annual analysis for reporting back to the contributing units.

### WOUND CATEGORIES

Wounds were allocated to one of four main aetiological categories for analysis – diabetic (DM), peripheral vascular disease (PVD), venous disease and miscellaneous (including vasculitic and auto-immune diseases). Because the miscellaneous group contains highly diverse aetiologies in very small numbers, no analysis of the fate of this group has been undertaken in this interim report. Similarly, this report does not compare the chance of a good outcome with and without HBOT because of the small numbers in the non-HBOT group.

### STATISTICS

No sample size calculations were performed for this study, as it is an ongoing opportunistic cohort study. We performed a descriptive statistical analysis and a backward stepwise

**Table 2**
**Number of patients and mean ages (with range) of all patients enrolled in the study (whether they had HBOT or not) by aetiology**

| Aetiology | Number | Average age years (range) | % total wounds |
|---|---|---|---|
| DM | 46 | 66.4 (42–89) | 41.8% |
| PVD | 27 | 73.9 (37–91) | 24.5% |
| Venous | 18 | 69.2 (43–87) | 16.4% |
| Miscellaneous | 19 | 61.7 (11–83) | 17.3% |
| **Total** | **110** | **67.8 (11–91)** | **100%** |

(DM – diabetes mellitus; PVD – peripheral vascular disease; venous – venous insufficiency)

logistic regression analysis on each aetiological group for factors that may predict wound outcome. This was done in order to develop a predictive model for wound healing after HBOT. All calculations were performed using StatsDirect v2.4.5 (StatsDirect Ltd., StatsDirect statistical software. <http://www.statsdirect.com> England: 2002).

## Results

There were 110 patients enrolled in the study of whom 88 received hyperbaric oxygen treatment. Sixty-seven (61%) were males and 43 (39%) females. The group receiving hyperbaric oxygen had 54 (61%) males with an average age of 67.2 years. The group that did not receive hyperbaric oxygen had 13 (59%) males with an average age of 70.4 years. The breakdown by aetiology is shown in Table 2.

### HYPERBARIC TREATMENT

The average number of treatments for the patients receiving HBOT was 24.4 (range 1–70). The average number of treatments, for each aetiology, is given in Table 3, while the overall frequency distribution is shown in Figure 1.

### OUTCOME DATA

Figure 2 shows the percentage of people in each aetiological group with a 'good' outcome (Scores 5 and 6, Table 1). Overall, immediately after the HBOT course, 52.3% of all aetiological groups combined had a 'good' outcome and this proportion increased to 64.1%, 91.7% and 78.2% at one, six and twelve months respectively. These data suggest that diabetic wounds improve most rapidly following HBOT, with venous wounds catching up at one month and arterial wounds at six months. At the time of the final draft of this interim report, we have follow-up data on 60% of those enrolled, and 43% at one year. Because these data sets are substantially incomplete, they will be reported in future annual analyses.

Because of the small numbers enrolled in the study, regression analysis for outcome was possible only for those patients with diabetes mellitus. We performed univariate analysis and a logistic regression for 'good' outcome at the end of HBOT and at one month after HBOT with the potential predictors being gender, duration of wound (months), wound area ($cm^2$), transcutaneous partial pressure of oxgyen ($P_{tc}O_2$) in air (mmHg), and $P_{tc}O_2$ on 100% oxygen at 1 ATA for 10 minutes (mmHg). Neither at the end of HBOT nor at one month follow up were there any significant predictive factors identified on univariate analysis. Stepwise logistic regression for healing at the end of the HBOT course did not produce a useful model. However, analysis at one month follow up suggested the following model was predictive of healing:

$$\text{Log (OR)} = 2.30 - (0.09*\text{TWA}) - (0.11*\text{DUR}) + (0.06*P_{tc}O_2)$$

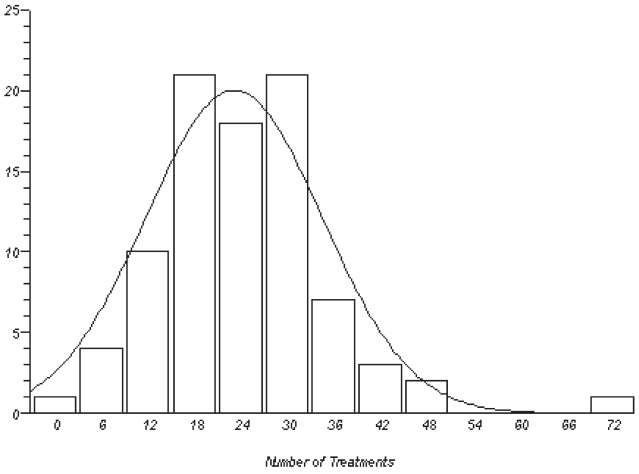
where OR = odds ratio, TWA = total wound area in $cm^2$, DUR = duration of wound in months and $P_{tc}O_2$ = transcutaneous partial pressure of oxygen on air at 1 ATA in mmHg.

**Table 3**
**Number and treatment averages of those patients who had HBOT by aetiology**

| Aetiology | Number of patients | Mean number of treatments (SD) |
|---|---|---|
| DM | 40 | 23.4 (10.2) |
| PVD | 20 | 24.7 (8.46) |
| Venous | 13 | 24.2 (9.49) |
| Miscellaneous | 15 | 27.0 (14.63) |
| **Total** | **88** | **24.4 (10.53)** |

(DM – diabetes mellitus; PVD – peripheral vascular disease; venous – venous insufficiency)

**Figure 1**
**Frequency distribution (with curve of best fit) of number of treatments for patients receiving HBOT**



This model suggests that, at presentation, wound healing is negatively impacted by increased wound area, duration of wound and a lower $P_{tc}O_2$ in air. For example, using this model we would predict that for a wound 7 $cm^2$ in area and of 10 months' duration with a resting $P_{tc}O_2$ (in air) of 30 mmHg, the odds of healing at one month after completion of a course of HBOT are nearly 11 to one (odds ratio 10.7).

**Discussion**

This study suggests that we can expect 50% of chronic wounds to heal by the end of a course of HBOT and up to 90% of wounds to be healed at six-month follow up. These wounds have all persisted for at least three months at presentation despite comprehensive wound care, and we believe this represents a real and important clinical benefit. Although 50% may not seem a particularly large proportion, given the population of Australia (20,404,617)[16] and an assumed prevalence for chronic wounds of 1%, this represents over 100,000 people who could potentially have a good outcome from HBOT.

Hyperbaric facilities have been treating chronic wounds for several years but there has been very little high-quality clinical research evidence to demonstrate the effectiveness of HBOT. A recent Cochrane meta-analysis on the efficacy of HBOT for chronic wounds included four randomised controlled trials (RCTs).[1] Three of these studies enrolled diabetic foot-ulcer patients and one enrolled patients with venous ulcers. There were no RCTs on the effects of HBOT on arterial ulcers. These RCTs suggest that there was a benefit in having HBOT for diabetic and venous ulcers but a larger, multi-centre study is required.

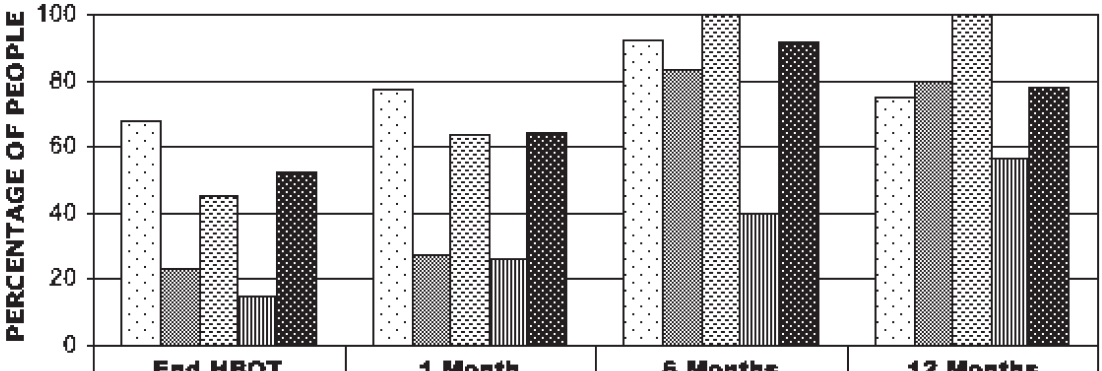
Because of the small number of patients enrolled in this study who did not receive HBOT (n = 22), we have not reported the fate of ulcers in that subgroup of patients in this analysis. While the reasons they were thought unsuitable for HBOT were not always clear, we hypothesise most of them had normoxic $P_{tc}O_2$ levels, failed to adequately respond to oxygen challenge with an increase in $P_{tc}O_2$ or declined to undergo therapy. We intend to more fully address this group in our next report.

There are differences in response to HBOT between aetiologies. Diabetic wounds have a faster resolution (higher percentage of those with a good outcome at the end of treatment) and appear to have a higher chance of a good outcome for the first few months after treatment. The estimated difference narrows rapidly and at six months there is very little between the three main aetiologies.

Logistic regression suggests that even with this small data set, we are able to show that features such as total wound area, duration of wound and $P_{tc}O_2$ at the wound site breathing air at 1 ATA are significant predictors of the proportion of wounds that will heal. We hope that as the data set grows, the regression model will become increasingly predictive of those wounds that can be expected to heal. This would have useful clinical applications for the selection of candidates for HBOT.

There are several limitations to the interpretation and applicability of this study. First among them is the loss of data as the study progresses. This is largely due to inability to contact some patients for follow up, despite considerable efforts to do so. Currently 56.6% of patients' data are lost

**Figure 2**
**Number of people with 'good' outcome after HBOT as a percentage of total people receiving HBOT**




| | End HBOT | 1 Month | 6 Months | 12 Months |
|---|---|---|---|---|
| DIABETES | 67.6 | 77.4 | 92 | 75 |
| PVD | 23.5 | 27.3 | 83.3 | 80 |
| VENOUS | 45.5 | 63.6 | 100 | 100 |
| DATA MISSING | 15.1 | 26.4 | 40 | 56.6 |
| ALL WOUNDS | 52.3 | 64.1 | 91.7 | 78.2 |

ASSESSMENT TIME

at the 12-month assessment time reflecting difficulties in following up patients out to this time period. We have attempted to address this with better patient tracking and by co-ordinating the follow up of patients with active reminders to the collection centres involved. Some apparent loss of data is in fact due to significant numbers of participants who have not yet reached the final assessment time.

Another significant limitation for this cohort study is the relatively small number of participants who had chronic wounds but did not receive HBOT. Financial considerations have made it impractical to improve the methodology of this study by the active recruitment of a comparison cohort of participants for whom hyperbaric referral has not been considered. Such a study is beyond our means at this time but remains highly desirable.

In conclusion, we have reported the first 110 patients of an ongoing prospective study. Our results suggest that a clinically important proportion of patients can expect a good outcome by one month after the completion of hyperbaric therapy. We continue to collect data prospectively and hope to generate a useful predictive model by which to identify those patients in whom HBOT is appropriate. We believe that this study is important in helping to better define the role of hyperbaric oxygen in these patients.

**Acknowledgements**

The authors wish to thank the staff at Royal Hobart Hospital, Wesley Hospital and Prince of Wales Hospital for the collection of the wound care data.

**References**

1 Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. *Cochrane Database of Systematic Reviews*. 2004 (2): CD004123.
2 Stausberg J, Kroger K, Maier I, Schneider H, Niebel W, Interdisciplinary Decubitus Project. Pressure ulcers in secondary care: incidence, prevalence, and relevance. *Adv Skin Wound Care*. 2005; 18: 140-5.
3 Lahmann NA, Halfens RJ, Dassen T. Prevalence of pressure ulcers in Germany. *J Clin Nurs*. 2005; 14: 165-72.
4 Chauhan VS, Goel S, Kumar P, Srivastava S, Shukla VK. The prevalence of pressure ulcers in hospitalised patients in a university hospital in India. *J Wound Care*. 2005; 14: 36-7.
5 Graham ID, Harrison MB, Nelson EA, Lorimer K, Fisher A. Prevalence of lower-limb ulceration: a systematic review of prevalence studies. *Adv Skin Wound Care*. 2003; 16: 305-16.
6 Albert S. Cost-effective management of recalcitrant diabetic foot ulcers. *Clin Podiatr Med Surg*. 2002; 19: 483-91.
7 Bennett G, Dealey C, Posnett J. The cost of pressure ulcers in the UK. [see comment]. *Age Ageing*. 2004; 33: 230-5.
8 Gordois A, Scuffham P, Shearer A, Oglesby A, Tobian JA. The health care costs of diabetic peripheral neuropathy in the US. *Diabetes Care*. 2003; 26: 1790-5.
9 Knighton DR, Hunt TK, Scheuenstuhl H, Halliday BJ, Werb Z, Banda MJ. Oxygen tension regulates the expression of angiogenesis factor by macrophages. *Science*. 1983; 221: 1283-5.
10 Jensen JA, Hunt TK, Scheuenstuhl H, Banda MJ. Effect of lactate, pyruvate, and pH on secretion of angiogenesis and mitogenesis factors by macrophages. *Lab Invest*. 1986; 54: 574-8.
11 Hunt TK, Pai MP. The effect of varying ambient oxygen tensions on wound metabolism and collagen synthesis. *Surg Gynecol Obstet*. 1972; 135: 561-7.
12 Niinikoski J, Grislis G, Hunt TK. Respiratory gas tensions and collagen in infected wounds. *Ann Surg*. 1972; 175: 588-93.
13 Hohn DC, MacKay RD, Halliday B, Hunt TK. Effect of $O_2$ tension on microbicidal function of leukocytes in wounds and in vitro. *Surg Forum*. 1976; 27: 18-20.
14 Davis JC, Hunt TK, editors. *Problem wounds: the role of oxygen*. New York: Elsevier Science Publishing Co.; 1988.
15 Committee MSA. MSAC-About Us. 2005 7/10/2003 [cited 2005 28/8/2005]; Available from: <http://www.msac.gov.au/bckgrd.htm>
16 Australian Bureau of Statistics: Projected population. 2005 [cited 2005 18/9/2005]; Available from: <www.abs.gov.au>

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*This paper is based on Dr Hawkins' thesis for his SPUMS Diploma, awarded earlier this year.*

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# The world as it is

## British Sub-Aqua Club (BSAC) diving incidents report 2005

Compiled by Brian Cumming, diving safety and incidents advisor
<http://www.bsac.org/techserv/increp05/intro.htm>

**Summary of the 2005 report prepared by Colin Wilson**

British Sub-Aqua Club (BSAC) for many years have been collecting data on diving incidents and publishing a yearly report. It is produced in the interest of promoting dive safety and contains details of UK sports diving incidents occurring amongst divers from all affiliations plus incidents occurring worldwide involving BSAC members.

The 2005 report follows the same format as previous years with sections on fatalities, decompression incidents, injury/illness, boating and surface incidents and a couple of sections relating to technique and equipment problems. It also contains links to the reports of the previous eight years for those interested in further reading. If you wish to read it at your leisure rather than on-line the complete document is available in pdf format.

The data are collected from a variety of sources including reports submitted by BSAC members, the Maritime and Coastguard Agency (MCGA), the Royal National Lifeboat Institution (RNLI) and press reports. There are details of over 440 incidents listed in the appropriate sections of this report with a varying degree of detail depending on the data source. There is the well-recognised problem of trying to equate incidents with prevalence. The problem, as always, is knowing the denominator. To help towards this, the number of BSAC members in relation to fatalities is provided at the end of the report.

**Fatalities**

There are 17 recorded fatalities in this time period, being in line with the average of 17.8 per year from the last 10 years, though thankfully fewer than the 25 of 2004. In 2005 there were two incidents of double fatalities. There is an interesting analysis of these cases in which, as expected, there were usually a number of contributory factors leading to the fatality. A summary of these factors is given below.

- Two cases are thought to have involved myocardial infarctions whilst diving.
- In three cases, divers were negatively buoyant and sank, with two of these being in difficulty on the surface.
- Four cases involved unplanned separation. In two of these cases three divers were diving together.
- One case involved a hose separation, leading to running out of air.
- Four cases involved intentional solo diving (24%). Two of these cases involved deep dives, one to 60 msw and the other to 68 msw.
- Two cases involved running out of air supply. One was due to a diver going back into the water after the dive to retrieve lost equipment.
- Three cases involved rebreathers.

There is a further look into accumulated BSAC data of a total of 138 fatalities since 1998, which shows that 13% of all these fatalities occurred while solo diving. This is a considerably higher figure than the fraction of solo diving actually taking place.

**Decompression illness**

There are 110 recorded reports of decompression illness (DCI), amounting to 120 cases. When all causal factors are excluded, analysis shows that around 50% are due to simple loss of buoyancy control with divers being unable to correctly control their rate of ascent. This is attributed to poor training and lack of skill. As with the fatalities, some cases involved more than one cause.

- 40 involved rapid ascents (33%)
- 30 involved diving deeper than 30 msw (25%)
- 28 involved repetitive diving (23%)
- 19 involved missed decompression stops (16%)

**Other problems**

Brian Cumming does highlight the continuing rise in incidents associated with abnormal ascents. This year's report contains 98 ascent-related incidents, though these did not all end in DCI. Eighty-one reports involved rapid ascents and there were 28 reports of missing decompression stops (with some relating to both). As ascent problems appear at the root of a large number of DCI cases, dive clubs should address as a priority what appears to reflect poor training and lack of skills of their members in an effort to reduce these. How many times have we had to treat a case of DCI because the diver was practising some rusty technique and got it wrong?

The individual reports of incidents provide small vignettes of the individual cases. For example:

*"June 2005. A diver conducted a dive on a wreck to a maximum of 68 m[sw] using a rebreather. He dived alone although others from his party were in the water at the same time. He was last seen hovering motionless above the*

*wreck. He failed to surface and an extensive search involving aircraft was made. His body has not been recovered."*

*"July 2005. Two divers conducted a dive to a depth of 37 m[sw]. One of the divers deployed a delayed SMB [surface marker buoy] to make their ascent. The reel jammed and dragged the diver upwards. He let go of the reel but was unable to stop the ascent. His buddy held on to him until 10 m[sw]. The buoyant diver rose straight to the surface. The other diver completed a safety stop at 3 m[sw] and then ascended to the surface. Ten minutes after surfacing the diver who made the rapid ascent noticed pain in his wrist and lower arm. He was placed on oxygen and the Coastguard alerted. Both divers were airlifted to a recompression facility. The diver with symptoms was recompressed and symptoms resolved."*

Although reading through the incidents becomes a bit repetitive, they display the regular well-recognised themes leading to and causing problems. The overview makes interesting reading with sensible analysis of their reports, being constrained by the details supplied to BSAC. I applaud Brian's endeavours in collecting and presenting his data, both of this and previous years, and recommend both divers and dive physicians read it.

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# SPUMS Annual Scientific Meeting 2005

## Delayed treatment of decompression illness: factors affecting treatment outcome, a retrospective review of 51 cases [Abstract]

Nathan Tweed, Peter HJ Mueller and Elliott Singer

**Key words**
Decompression illness, decompression sickness, hyperbaric oxygen therapy, treatment, flying (and diving), morbidity

(Tweed N, Mueller PHJ, Singer E. Delayed treatment of decompression illness: factors affecting treatment outcome, a retrospective review of 51 cases. *Diving and Hyperbaric Medicine*. 2006; 36: 100. **Abstract only**)

Between 25 July 2003 and 24 July 2004 an audit was conducted to describe a group of patients presenting to London Hyperbaric Medicine (LHM), Whipps Cross University Hospital, with symptoms of decompression illness (DCI). Since presentations at this inland facility are likely to be delayed it is of interest whether they differ in symptomatology or outcome from more typical cases of DCI. The records of all divers presenting during that time were reviewed; a total of 86 divers. Thirty-five divers were excluded, 28 did not have a diagnosis of DCI and seven had received their first hyperbaric treatment elsewhere. The remaining 51 divers with a diagnosis of DCI had been assessed using the Institute of Naval Medicine diver assessment form, and this was used together with referral letters to record information related to diver training, dive profiles prior to injury, history and examination results, treatment(s) given and outcome. Patients seen at LHM reported more symptoms than those in data from the Diver Alert Network.[1] Furthermore, there was a significant positive correlation between the number of hours from the onset of symptoms and the number of treatments required for an optimal outcome ($P = 0.01$). Patients who flew between their last dive and the onset of treatment did not require any more treatments on average than those who did not. However, divers seen at LHM are a self-selected group, and, therefore, it is hard to interpret the importance of this latter observation.

**Reference**

1 Elliott DH, Moon RE. Manifestations of the decompression disorders. In: Bennett PB, Elliott DH, editors. *The physiology and medicine of diving*. 4th ed. London: WB Saunders; 1993. Table 17.5. p. 493.

*Nathan Tweed, MA(Cantab), was a medical student attached to London Hyperbaric Medicine, Whipps Cross University Hospital, Leytonstone, London E11 1NR, England*
***E-mail:*** *<nathan.tweed@gmail.com>*
*Peter HJ Mueller was Medical Director, London Hyperbaric Medicine, at the time of this audit, and*
*Elliott Singer is a general practitioner, hyperbaric physician at LHM and vice-chairperson of the British Hyperbaric Association.*

***Paper presented by Dr Mueller at the 2005 SPUMS ASM, The Maldives.***

# Critical appraisal

## Hyperbaric oxygen therapy significantly reduced pain and swelling and increased wrist range of movement in patients with early CRPS

**Clinical bottom line**

1 There was a significant difference in favour of HBOT for pain, oedema and wrist flexion at day 45 after the start of the trial.
2 Functional significance for the patients is unknown.
3 No patient in either arm progressed to the atrophic stage of complex regional pain syndrome (CRPS).

**Citation**

Kiralp MZ, Yildiz S, Vural D, Kesin I, Ay H, Dursun H. Effectiveness of hyperbaric oxygen therapy in the treatment of complex regional pain syndrome. *J Int Med Res*. 2004; 32: 258-62.

**Lead author's e-mail:** <mkiralp@hotmail.com>

**Three-part clinical question**

For patients with early post-traumatic CRPS, does hyperbaric oxygenation result in a reduction in the severity of symptoms and signs of the syndrome?

**Search terms**

Hyperbaric oxygenation, complex regional pain syndrome, reflex sympathetic dystrophy

**The study**

Double-blind, concealed and randomised controlled trial with intention to treat.

**The study patients**

Diagnosed with CRPS about 1.5 months after trauma to upper limb and had not received prior treatment for CRPS.

CONTROL GROUP

(N = 34; 34 analysed) Air at 2.4 ATA for 90 minutes daily for 15 sessions over three weeks, plus paracetamol 500 mg three times daily.

EXPERIMENTAL GROUP

(N = 37; 37 analysed) As above, but breathing 100% oxygen at 2.4 ATA.

**The evidence**

See Table 1.

**Comments**

1 All patients were probably young and relatively fit at entry because the trial was recruited at a military hospital.
2 The HBOT was given prior to other therapy and immediately on diagnosis (1.5 months after injury). This trial therefore measures response of CRPS at an early stage.
3 We have assumed all patients had CRPS affecting the hand.
4 There is no measure of the functional significance of these changes. It is not clear what the significance of 2 units of the VAS is, for example.
5 Patients did appear well matched for severity prior to therapy.
6 We have assumed no losses to follow up, but not specifically stated in the paper.
7 No mention of any confounders considered except age.

**Appraised by**

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*Appraised Wednesday 21 December 2005*

**Key words**

Critical appraisal, hyperbaric oxygen therapy, pain, vasoconstriction

**Table 1**
**Major outcomes in randomised study of hyperbaric oxygen for early complex regional pain syndrome**

| Outcome at day 45 | Air group mean (SD) | HBOT group mean (SD) | Difference | 95% CI |
|---|---|---|---|---|
| Visual analogue scale for pain (0 to 10 scale) | 5.61 (1.37) | 3.72 (1.42) | 1.89 | 1.23 to 2.55 |
| Wrist flexion (degrees) | 44.55 (16.11) | 59.86 (17.38) | -15.31 | -23.26 to -7.36 |
| Wrist circumference (cm) | 18.2 (0.72) | 16.98 (0.71) | 1.22 | 0.88 to 1.56 |

# Articles reprinted from other journals

## Adjuvant hyperbaric oxygen therapy ($HBO_2$) for treatment of necrotizing fasciitis reduces mortality and amputation rate [Abstract]

SJ Escobar, JB Slade Jr, TK Hunt, P Cianci

**Objective:** A retrospective analysis of 42 patients with necrotizing soft tissue infections treated with adjunctive $HBO_2$ to ascertain efficacy and safety. Overall mortality was 11.9% and morbidity 5%.
**Summary background data:** Necrotizing soft tissue infections have historically high rates of mortality and morbidity, including amputation. Common misconceptions that prevent widespread use of adjunctive $HBO_2$ for this diagnosis include delays to surgery, increased morbidity, and significant complications.
**Methods:** Forty- two consecutive patients (average age 56.1) with necrotizing fasciitis presenting to a major referral centre were treated with adjunctive $HBO_2$ as part of an aggressive programme of surgery, antibiotics, and critical care. Involved areas included the lower abdomen (15 patients), thigh and perineum (9 patients), flank (4 patients), lower leg (3 patients), and arm, shoulder, and axilla (2 patients). Co-morbidities included diabetes mellitus, chronic renal failure, intravenous drug abuse, peripheral vascular disease, and malignancy.
**Results:** Mortality was 11.9% (5 patients). Both amputations (a finger and a penis), occurred prior to transport to our facility. The average number of surgical debridements was 2.8 per patient; 1.25 performed prior to the start of $HBO_2$. The infectious process was controlled after an average of 7 $HBO_2$ treatments were administered to ensure successful wound closure. Complications consisted of only mild ear barotrauma in 3 patients (7%), and confinement anxiety in 17 (41%) but did not prevent treatment.
**Conclusion:** Compared to national reports of outcomes with "standard" regimens for necrotizing fasciitis, our experience with $HBO_2$, adjunctive to comprehensive and aggressive management, demonstrates reduced mortality (34% vs 11.9%), and morbidity (amputations 50% vs 0%). The treatments were safe and no delays to surgery or interference with standard therapy could be attributed to $HBO_2$.

*Department of Hyperbaric Medicine, Doctors Medical Center, San Pablo, CA, and Department of Surgery, University of California, San Francisco, CA*

**Reprinted with kind permission from Escobar SJ, Jr Slade JB, Hunt TK, Cianci P. Adjuvant hyperbaric oxygen therapy ($HBO_2$) for treatment of necrotizing fasciitis reduces mortality and amputation rate. *Undersea Hyperb Med*. 2005; 32: 437-43.**

**Key words**
Reprinted from, hyperbaric oxygen therapy, necrotising infections, infectious diseases, abstracts

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## Human factors in decompression sickness in compressed air workers in the United Kingdom 1986–2000 [Abstract]

Andrew P Colvin

**Aims:** To determine if personal characteristics (individual or human factors) in compressed air workers can be associated with repetitive episodes of decompression sickness (DCS). To determine the distribution of repetitive decompression sickness in the compressed air workforce in the United Kingdom for the period 1986–2000.

**Design:** A case control study of UK compressed air workers in which subjects with repetitive DCS during a single compressed air project were matched to two control groups. An analysis of the Health and Safety Executive (HSE) Decompression Database 1986–2000 was undertaken to examine the relative contribution of those workers with repetitive DCS to the overall number of DCS episodes during the study period and to determine if repetitive DCS could be associated with any particular contract characteristics (not all compressed air contracts during the study period contained workers with multiple episodes of DCS).

**Study population and setting:** All compressed air workers in the HSE Decompression Database during the period 1986–2000 were identified.

**Subjects:** The "cases" were 62 compressed air workers selected from the study population with 2 or more episodes of decompression sickness confirmed during a single compressed air contract. Two separate groups of control subjects were identified from the study population, i.e., those with no episodes of DCS (zero bend controls) and those with a single episode of DCS during a single compressed air contract (single bend controls). All controls were matched to the cases by compressed air contract, equivalent pressure exposure and occupation.

**Methodology:** Personal history and clinical examination findings were collated on all subjects from the contemporaneous clinical records of the pre-employment or initial compressed air medical examination held by the Contract Medical Adviser. Non-personal data (i.e., pressure/exposure data including reported DCS episodes) was collated from the HSE Decompression Database as reported by the Compressed Air Contractor to the Health and Safety Executive. A total of 62 cases, 71 single bend controls and 130 zero bend controls were included in the study. Fifty-two fully matched sets consisting of 52 cases matched with two zero bend controls and one single bend control were the basis for initial analysis. Subsequently comparisons between cases and control groups were undertaken using a variety of statistical analysis, the most important being conditional logistic regression.

**Results:** 4% of the workforce in the HSE Decompression Database 1986–2000 suffered 50% of the episodes of DCS requiring therapeutic recompression. No significant differences were found between those with multiple bends (cases), single bends or zero bends when analysing the various personal characteristics assessed during the initial or pre-employment compressed air medical examination. There is some evidence to suggest that a survivor or "healthy worker" effect is operating in the compressed air workforce with regard to susceptibility to DCS. Those contracts likely to produce repetitive DCS in some workers often had an average pressure greater than 1 bar gauge/201 kPa absolute, greater number of compressed air workers being exposed and longer shift lengths (greater than 6 hours at pressures below 1.5 bar gauge/251 kPa absolute or greater than 4 hours at pressures over 1.5 bar gauge/251 kPa absolute).

**Conclusions:** This study did not find any differences in the personal characteristics of compressed air workers with multiple, single or zero episodes of DCS when matched by contract, occupation and pressure exposure. This negative finding contradicts long-standing associations with age and obesity often reported in the literature based on historical and often anecdotal reports. "Bend prone" compressed air workers do exist but this study suggests that the history and clinical examination (and tests) undertaken at the routine pre-employment compressed air medical examination will not reveal them. If individual or human factors causing, or associated with, susceptibility to DCS could be identified then a significant reduction in episodes of DCS in the compressed air workforce could occur. This study suggests that different methodology or clinical examination will be required to achieve this however.

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**Reprinted with the kind permission of HMSO from Colvin AP. Research Report RR 171 Human factors in decompression sickness in compressed air workers in the United Kingdom 1986–2000. Sudbury, Suffolk: HSE; 2003. ISBN 0 7176 2771 3**

**Key words**
Reprinted from, decompression sickness, decompression illness, working in compressed air, risk factors, occupational health, tunnelling, medical database, abstracts

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# Further investigation of possible musculoskeletal and cognitive deficit due to welding in divers identified in the ELTHI diving study [Executive summary]

Jennie I Macdiarmid, John AS Ross, Sean Semple, Liesl M Osman, Stephen J Watt, John R Crawford

**Introduction:** Within a welding environment there is potential for exposure to various chemical and physical hazards. Welding generates toxic gases and high levels of fine particles containing a complex variety of metals, varying with the type of welding and material used. Welding has been associated with a number of acute and chronic health related hazards, including respiratory, cognitive, musculoskeletal and hearing complaints. The results of the ELTHI diving study support these findings, showing that an occupational history of welding is associated with an increased prevalence of cognitive, musculoskeletal and hearing complaints. There were, however, insufficient numbers of non-diver welders in the ELTHI

diving study to determine whether diver welders were more at risk than non-diver welders. It is proposed that there could be an interaction between diving and welding, with divers being at greater risk to adverse health effects since many of them weld at pressure in small, poorly ventilated welding habitats.

**Aim:** To determine if the health impact of diving and welding is greater than welding alone, with particular interest in the effect on cognitive and musculoskeletal symptom complaint.

**Methods:** The study consisted of a postal questionnaire survey. The 5 page questionnaire assessed welding and diving exposure, symptom complaints, diagnosed medical conditions, lifestyle history, cognitive failure (cognitive failure questionnaire (CFQ)) and health related quality of life (SF-12).

**Study population:** Three age matched study groups were targeted in the survey: diver welders, non-diver welders and non-diver non-welders. Non-divers were men who had worked offshore but never as a diver. Diver welders and non-diver non-welders were selected from men who had previously participated in the ELTHI diving study. Due to an insufficient number of non-diver welders in the ELTHI diving study, a new sample of non-diver welders was recruited through CAPITA Health Solutions. These were men who had had an offshore medical examination between 1990–2002, stated their occupation as a welder and had not dived. 361 diver welders, 352 non-diver welders and 503 non-diver non-welders were traced and sent a questionnaire. Non-responders were sent a maximum of 3 questionnaires at 4 week intervals.

**Re-analysis of the ELTHI diving study data:** In addition to the questionnaire survey, data from the ELTHI diving study were re-analysed to include only divers. Using hierarchical logistic regression analysis, diving experience was related to symptoms complaint, adjusting for lifestyle factors, welding, diving accidents and chemical exposures. A separate model was used for each complaint, which were 'forgetfulness or loss of concentration', 'joint pain or muscle stiffness', 'back or neck pain' and 'impaired hearing'.

**Results:** The questionnaire survey had a response rate of 48%, with response being higher among those who had previously completed the ELTHI diving study (53%) than newly recruited non-diver welders (30%). 182 diver welders, 108 non-diver welders and 252 non-diver non-welders were included in the analysis.

**Cognitive complaint:** The prevalence of complaint of 'forgetfulness or loss of concentration' was significantly higher among diver welders (33%) than both non-diver welders (10%) and non-diver non-welders (14%). Consistent with this, diver welders scored significantly higher on the CFQ than both non-diving groups, reflecting greater cognitive symptomatology. Non-diver welders and non-diver non-welders did not differ significantly on either of these measures of cognitive complaint.

**Musculoskeletal complaint:** As observed for cognitive complaint, the complaint of 'joint pain or muscle stiffness' and 'back or neck pain' was significantly more common among diver welders than both non-diver welders and non-diver non-welders.

The only other symptom to differ between the groups was 'impaired hearing', which was more common in diver welders (29%) and non-diver welders (32%), than non-diver non-welders (12%). Physical and mental health related quality of life did not differ significantly between the 3 study groups.

**Welding and diving exposure related to symptom complaint:** Divers tended to have shorter welding careers and had been exposed to less welding fume than non-divers. The divers, on average, had been exposed to the equivalent of 11 years of welding fume at a concentration of $5 mg.m^{-3}$ for 240 days per year, while in non-divers equivalent exposure was 36 years. Divers were more likely than non-divers to have welded aluminium (29% vs 8%). Despite differences between diver and non-diver welders in welding experience, there were no convincing correlations between welding exposure and symptom complaint within this population. Divers who welded at pressure had longer diving careers and greater welding fume exposure than divers who had not welded at pressure, but they were no more likely to complain of cognitive or musculoskeletal symptoms.

**Re-analysis of the ELTHI diving study data:** Further analysis of the ELTHI diving study data in divers, revealed other factors, such as diving accidents and chemical exposures, were related to cognitive, musculoskeletal and hearing complaint. Neurological decompression illness, exposure to contaminated gas while diving, exposure to petrochemicals and work as a welder were associated with the complaint of 'forgetfulness or loss of concentration'. 'Back or neck pain' was associated with contaminated gas, exposure to petrochemicals and neurological decompression illness. 'Joint pain or muscle stiffness' was associated with pain only decompression illness, exposure to contaminated gas and exposure to petrochemicals. 'Impaired hearing' was associated with exposure to contaminated gas and noise. With respect to diving techniques, after adjusting

for these other factors, only mixed gas bounce diving was associated with reported 'forgetfulness or loss of concentration' and reported 'joint pain or muscle stiffness', and saturation diving related to reported 'impaired hearing'.

**Summary:** The main finding of the study confirms the previous observations that the health impact of welding and diving is greater than that of welding alone. Cognitive and musculoskeletal complaints were found to be more common in diver welders than non-diver welders. Welding, therefore, should be taken into consideration when assessing the long term health impact of diving at work. In addition, further analysis of the ELTHI diving study indicates that in addition to welding, other work related factors, such as accidents and chemical exposures, are associated with the complaints studied. Further investigation is needed to determine a causal link. If a causal link were established with chemical exposures then greater focus would need to be placed on occupational hygiene and interventions introduced to control possible toxic exposures within the diving sector.

*Departments of Environmental and Occupational Medicine, Medicine & Therapeutics, and Psychology, University of Aberdeen, UK*

**Reprinted with the kind permission of HMSO from Macdiarmid JI, Ross JAS, Semple S, Osman LM, Watt SJ, Crawford JR. Research Report RR 390 Further investigation of possible musculoskeletal and cognitive deficit due to welding in divers identified in the ELTHI diving study. Sudbury, Suffolk: HSE; 2005. ISBN 0 7176 6169 5**

**Key words**
Reprinted from, occupational diving, health surveillance, questionnaire, diving industry, morbidity, epidemiology, abstracts

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# Missed diagnoses: Benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo (BPPV) is probably the most common cause of vertigo. Estimates of incidence range from 11–64 per 100,000 per year, increasing by around 38% per decade of life; it is twice as common in women as men.[1] It is believed to be caused by free-floating particles (canaliths) leaving the macula and entering one of the semicircular canals. Vertigo is typically triggered by movement, and can be intense, but short-lived. A study of patients referred to a neurology department found that the most common misdiagnoses were unspecified dizziness/vertigo, transient ischaemic attacks, cervicogenic vertigo and psychogenic dizziness/vertigo.[2] Diagnosis of BPPV is usually made with a procedure known as the Dix-Hallpike manoeuvre. The sensitivity of the test is not 100%, so if symptoms persist re-testing is recommended. BPPV is a treatable disease – most successfully by repositioning treatments which, in experienced hands, have a success rate of more than 95%. While, after three months, around 84% of cases resolve naturally, treatment is still worthwhile. Although described as benign, it can be severely disabling – a large number of working days and productivity are lost, and it is a common cause of falls in the elderly. Some patients may suffer for many years, particularly if this diagnosis is missed.

For a more detailed article on BPPV, see <www.emedicine.com/ent/topic761.htm> or <www.dizziness-and-balance.com/disorders/bppv/bppv.html>.

**References**

1 Hilton M, Pinder D. The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. *Cochrane Database Syst Rev*. 2004; (2) CD003162.
2 Gordon CR, Zur O, Furas R, Kott E, Gadoth N. Pitfalls in the diagnosis of benign paroxysmal positional vertigo. *Harefuah*. 2000; 138: 1024-7, 1087.

**Reprinted with the kind permission of the Medical Protection Society from *UK Casebook*. 2005; 13(4): 25.**

**Key words**
Reprinted from, vertigo, inner ear

**Editor's comment:**
This item is reprinted here to remind diving physicians of this differential diagnosis in the vertiginous diver.

# 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 Meeting held in Melbourne on 27 November 2005

**Opened:** 0910 hr

**Present:** Drs C Acott (President), R Walker (Immediate Past-President), S Sharkey (Secretary), G Williams (Public Officer), D Smart (ANZHMG Representative), M Davis (Editor)

**Apologies:** Drs C Lee, D Vote (Committee Members)

**1 Minutes of the previous meeting**
Moved that the minutes be accepted as a true record with some minor amendments. Proposed Dr D Smart, seconded Dr C Acott, carried.

**2 Matters arising from the previous minutes**
2.1 Review of roles and functions of SPUMS Administrator is addressed at agenda item 8.9
2.2 The Treasurer confirmed that the finalised accounts from the 2004 ASM demonstrate a profit of $4205.58 after refunds. No further action required.
2.3 The Committee is satisfied that the discrepancies previously identified in the 2005 ASM financial reports have now been rectified and identified as errors in data recording as reported by the Treasurer. The Committee discussed whether new processes needed to be

implemented in order to avoid this problem in the future. The Committee determined that the current documented instructions to the Convenor are clear with respect of their responsibilities in management and reporting of ASM accounts to the Committee. ACTION: Dr Walker to distribute the current version of convenor instructions to the committee members.

2.4 Dr Sharkey reported on current progress in auditing and mustering SPUMS equipment. It is expected obsolete items that are no longer required will be offered for purchase by members of the Society. ACTION: ongoing.

2.5 SPUMS Committee overseas representatives have been sent an email to confirm their continuing availability. Responses awaited.

2.6 In response to their request the ANZCA SIG has been offered a position on the SPUMS Education Board for authorisation of the SPUMS Diploma award to ANZCA certificate candidates. A response is awaited.

2.7 Treasurer's position: Dr Williams volunteered to assume the role of Treasurer given Dr Andrew Patterson's resignation. The Committee accepted this offer. The Committee acknowledges the significant contribution made by Dr Patterson in his role as Treasurer. In particular is the recognition that he moved the organisation forward by introducing contemporary accounting practices and bringing discipline to the processes. Despite requests from the committee members, Dr Patterson is not currently willing to stay on the Committee.

**3 Annual Scientific Meeting**

3.1 The proposed programme for the 2006 ASM was further discussed and progressed.

3.2 Dr Davis advised that a formal inspection of the site for the 2007 meeting had recently been conducted. Full details to be promulgated at the next committee meeting.

3.3 Dr Acott reported that the options for the 2008 ASM to be held in PNG are being progressed.

**4 Treasurer's report**

4.1 The Treasurer reported that the Society is in a positive financial position. There are no anticipated large expenses in the immediate term.

4.2 The Treasurer raised an issue with respect of claims for expenses made by members without sufficient evidence or justification, including some made after the closure of accounts for the relevant financial year. The Committee stipulated its position that all claims for expenses are to be made in a timely manner and with the appropriate level of supporting documentation. Further, it was agreed that claims that were not adequately substantiated could not be approved for reimbursement. The Treasurer is required to apply these requirements in addressing all requests for reimbursement. Requests for reimbursement that do not comply with the requirements are to be forwarded to the President for further action.

**5 Journal report**

5.1 The web journal component was discussed. It was agreed that the whole journal would be made available to members of the Society, and that the public would have access to the front cover, contents and Editor's comments sections.

**6 Education Officer's report**

6.1 One new application for a Diploma by an ANZCA SIG candidate has been received.

6.2 The SPUMS Diploma master list is to be maintained by the Education Officer. All amendments to this list are to be forwarded to the Education Officer.

**7 Correspondence**

7.1 Unsolicited correspondence from an on-shore convention centre was received and shared with the Committee as a possible venue for future ASMs.

**8 Other business**

8.1 Dr Williams (Public Officer) reported that Consumer Affairs have enforced two new constitutional amendments on SPUMS that in effect apply now. These changes will be promulgated in the March edition of the Journal for consideration of the motion at the June 2006 AGM.

8.2 The required practice of maintaining and publishing meeting minutes was discussed. It was resolved that the Secretary will retain the minutes in a Minute Book and that the key resolutions would be published in the Journal as an "extract" of the minutes. ACTION: Dr Walker to draft a motion to accept the 'model' rules regarding publishing of minutes.

8.3 Strategies for increasing membership were discussed. The website will be a key tool. Suggested links are to be advised to Dr Walker. Promotion of the Society to junior MOs is also important. Distribution of handouts and membership forms to MOs on courses was suggested. Dr Davis suggested the Editorial Assistant would produce a pdf handout/brochure for easy printing off the website.

8.4 Dr Walker provided an update on the SPUMS website. The website manager functions were discussed. It was agreed that two committee members should share in the task of managing the "front-end" of the website. Drs Walker and Smart agreed to perform these roles. The SPUMS Administrator will continue to perform the functions required to manage the "back-end" of the site. SQUIZ have been asked to provide a proposal package for support costs and arrangements. The need to back up data on the website was recognised.

8.5 SPUMS Journal costs were discussed. It was recognised that associate memberships are being provided at a financial loss to the Society. An increase in fees is recommended in line with inflation, in recognition of increased expenditure on SPUMS website and retaining a buffer in order to have the freedom to commit to new investments. The Committee agreed that an increase in fees should be recommended. ACTION: Dr Patterson to

draft motion for consideration at next AGM.

8.6 SPUMS Committee communication issues were discussed. Some suggestions for improvement were made.

8.7 The pros and cons of advertising within the Journal were discussed. The revenue benefit vs the inadvertent endorsement of products was considered. No resolution on this issue was made.

8.8 New membership category for retired members was proposed and agreed. ACTION: Dr Walker to draft a motion regarding new membership category of retired members.

8.9 The Administrator's roles and functions were discussed and agreed. The Committee agreed that there are significant merits in establishing a contractual agreement with the SPUMS Administrator. Consideration needs to given to whether the same needs to be done for the SPUMS Editor. ACTION: Dr Sharkey to progress drafting of a contractor agreement.

8.10 The welcome letter for new members was discussed and agreed it should be reviewed. ACTION: Dr Acott.

**9 The next meeting is proposed for 12 March 2006 (0900 SA time) by teleconference.**

Closed: 1500 hr

## ANZCA Certificate in Diving and Hyperbaric Medicine

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

Eligibility criteria are:

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

2 Completion of training courses in Diving Medicine and in Hyperbaric Medicine of at least 4 weeks' total duration. (e.g., ANZHMG course at Prince of Wales Hospital Sydney, and Royal Adelaide Hospital or *HMAS Penguin* diving medical officers course).

3 **EITHER:**

a. Completion of the Diploma of the South Pacific Underwater Medicine Society, including 6 months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS.

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

**OR:**

c. Completion of 18 months' full-time experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.

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

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

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

All details are available on the ANZCA website at <www.anzca.edu.au/edutraining/DHM/index.htm>

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

## ANZCA awards Certificates of Diving and Hyperbaric Medicine

Successful candidates awarded the Certificate of Diving and Hyperbaric Medicine by examination in 2004 and 2005 were:

| | |
|---|---|
| David Edward DAVIES | (WA) |
| Glen C HAWKINS | (NSW) |
| Robert LONG | (QLD) |
| Robert John TURNER | (NSW) |
| Darren Lindsay WOLFERS | (NSW) |

There is no exam scheduled for 2006.

# Letters to the Editor

## Bronchial hyperresponsiveness, spirometry and diving

Dear Editor,

As a respiratory physiologist in the lung function laboratory of a major teaching hospital I have been impressed in the last 10 years by the number of young people, with a past history of asthma and normal spirometry who have moderate to severe bronchial hyperresponsiveness (BHR) to hyperosmolar (4.5%) sodium chloride or hyperpnea with dry air. Some of these data were summarised in our earlier paper in this journal.[1] These stimuli act indirectly by releasing inflammatory mediators to cause contraction of bronchial smooth muscle and the airways to narrow. BHR to 'indirect' stimuli is consistent with currently active asthma and this type of BHR responds to treatment with inhaled steroids. BHR to 'indirect' stimuli is particularly relevant to the activities associated with diving such as exercise and accidental aspiration of salt water.

We have reported BHR, within the asthmatic range, to bronchial provocation with a hyperosmolar aerosol (4.5% saline) in 17% of 180 potential scuba divers who had a past history of asthma, no current symptoms, and normal spirometry and had been declared fit to dive following a medical examination.[2] Many of those subjects recognised their positive response as a symptom of their previous asthma, and stated they would cease their intention to dive with scuba and would seek treatment as a result of the test. Following this report sports doctors appeared to change their practice and referred people with a past history of asthma for testing *before* rather than *after* a full medical was performed. The findings of that report remain important because the change in referral practice means we will not have the opportunity again to evaluate BHR in people with a past history of asthma found otherwise medically fit to dive. It is of interest that exercise-induced asthma (EIA) has also been shown to be frequent in defence force recruits with a history of childhood asthma and no symptoms.[3]

We also reported BHR to both dry air and to the hyperosmolar aerosol mannitol in a group of elite athletes unselected for respiratory symptoms and with excellent lung function.[4] Importantly in this same cohort there was a low sensitivity (36%) to detect BHR using provocation with the pharmacological stimulus methacholine, which acts directly on smooth muscle receptors to cause contraction.[5] This apparent paradox (pharmacological agents are widely thought to be more sensitive than other stimuli in identification of BHR) has also been reported in school children with EIA and normal spirometry but negative to provocation with histamine.[6] The difference in sensitivity to the stimuli may relate to the higher potency of the mediators released in response to exercise and hyperosmolar aerosols compared with histamine and methacholine. Thus only one hundredth of the concentration of the prostaglandin $D_2$ and one thousandth of the concentration of leukotriene $E_4$ is required to provoke the same degree of airway narrowing as histamine and methacholine. Of interest was that 40% of the children with EIA did not have a clinical diagnosis of asthma suggesting responsiveness to exercise is an early sign and may precede symptomatic asthma.[6]

What can we learn from these findings? First, there is a high percentage of people with a past history of asthma, no current symptoms or current use of medication and with normal spirometry, who have BHR to hyperosmolar aerosols and dry air hyperpnea. Second, normal values for spirometry neither predict nor exclude bronchial hyperresponsivness to these stimuli. Third, a normal response to bronchial provocation with a pharmacological agent neither predicts nor excludes BHR to hyperosmolar aerosols or dry air. Fourth, there are people with airway narrowing provoked by exercise who do not have other symptoms of asthma. Finally, we have learnt that many people with mild asthma and few symptoms or those well controlled on treatment do not demonstrate BHR to these stimuli.[7]

If there is a move towards assessment of risk of problems with diving for those with a current or past history of asthma then it would seem sensible to exclude BHR to the stimuli the diver will encounter. In cases where a person does not have normal spirometry an acute response (12% increase in $FEV_1$) to a bronchodilator can reveal BHR consistent with asthma. For those with normal spirometry medical practitioners in Australia now have available to them a bronchial provocation test kit that uses a dry powder of mannitol and has regulatory approval (Aridol™, Pharmaxis Ltd, Frenchs Forest NSW).[7] The mannitol test has been under development for more than a decade and can be used to identify BHR to exercise, dry air and hypertonic saline.

**References**


1 Anderson SD, Wong R, Bennett M, Beckert L. Summary of knowledge and thinking about asthma and diving since 1993. *Diving and Hyperbaric Medicine*. 2006; 36: 12-22.

2 Anderson SD, Brannan J, Trevillion L, Young IH. Lung function and bronchial provocation tests for intending divers with a history of asthma. *SPUMS J*. 1995; 25: 233-48.

3 Sinclair DG, Sims MM, Hoad NA, Winfield CR. Exercise-induced airway narrowing in army recruits with a history of childhood asthma. *Eur Respir J*. 1995; 8: 1314-7.

4 Holzer K, Anderson SD, Chan H-K, Douglass J. Mannitol as a challenge test to identify exercise-induced bronchoconstriction in elite athletes. *Am J Respir Crit Care Med*. 2003; 167: 534-47.

5 Holzer K, Anderson SD, Douglass J. Exercise in elite summer athletes: challenges for diagnosis. *J Allergy Clin Immunol*. 2002; 110: 374-80.

6 Haby MM, Anderson SD, Peat JK, Mellis CM, Toelle

BG, Woolcock AJ. An exercise challenge protocol for epidemiological studies of asthma in children: comparison with histamine challenge. *Eur Respir J*. 1994; 7: 43-9.
7 Brannan JD, Anderson SD, Perry CP, Freed-Martens R, Lassig AR, Charlton B. The safety and efficacy of inhaled dry powder mannitol as a bronchial provocation test for airway hyperresponsiveness: a phase 3 comparison study with hypertonic (4.5%) saline. *Respiratory Research*. 2005; 6: No.144.

**Conflict of interest**

Dr Anderson is the inventor named on the patent that covers the mannitol test. The patent is owned by her employer, the Central Sydney Area Health Service, who has licensed the commercial rights to Pharmaxis Ltd. Dr Anderson owns shares (but no options) in Pharmaxis Ltd that she purchased herself and, in the future, would benefit from royalties paid to her employer.

*Sandra D Anderson PhD, DSc, Principal Hospital Scientist, Department of Respiratory Medicine, Royal Prince Alfred Hospital, Sydney, Australia*
***E-mail:*** *<sandya@med.usyd.edu.au>*

**Key words**
Letters (to the Editor), fitness to dive, respiratory, asthma, pulmonary function

## A successor for Project Stickybeak

Dear Editor,

You appear to be unaware that I have been trying to obtain someone to help run Project Stickybeak and ultimately take over – but without success. The only two candidates John Lippmann could suggest soon lost interest, possibly when they realised the amount of work involved, especially the problems of finessing coroners and other government agencies into giving support.

There has never been active interest and involvement in the collection of 'fatality information' from members of the SPUMS Committee, although you have continued to publish the provisional reports and 'advertised' the project in the Journal.

You should be aware that I have many easily accessible data on file that are not included in the provisional reports. Also, basic data are available to anyone interested at the National Library.

Had anyone been interested they would logically have contacted me. However, I have created, and continue to add to, this database, regardless of the present lack of interest by diving authorities (lay and medical) in this 'rough trade' type of research into the improvement of diver safety.

You will recollect that some time ago I contacted you concerning a plan to hold a workshop to discuss the influence of health factors on diver safety. If you intend to proceed with this idea you will find my databank makes it easy to identify such cases in the fatalities report files. You may even manage to extract data from others.

*Douglas Walker*
*Project Stickybeak*

**Key words**
Letters (to the Editor), diving accidents, diving deaths, research

**Editor's comment:**

Since 1972, Dr Walker has created a huge database of case histories of diving fatalities, the most recent report for which appears in this issue. Despite his comments above, the Society strongly supports this work, and greatly appreciates Douglas's tenacity and hard work.

To ensure that Project Stickybeak continues, a long-term commitment from a member or members of the Society to take up the reins from Douglas, possibly with the aid of DAN-SEAP, is required.

It has been suggested that there should be a medical practitioner who can establish a close working relationship with the Police and Coroners in each coastal State of Australia to collect and collate the information for that State and forward these data to an overall coordinator who would prepare the Project Stickybeak reports. This was discussed at the Fiji ASM in June and several individuals expressed an interest in helping to continue this invaluable project. Those interested should make themselves known either to the President, Chris Acott, or the Editor.

A similar process has been established in New Zealand with Water Safety NZ, the NZU Accident Recorder, Dr Lynn Taylor and the Editor.

## Fitness standards for beach lifeguards

Dear Editor,

*Occupational Medicine* has published two papers by Reilly et al on occupational fitness standards for beach lifeguarding that you might like to cite for your readers.[1,2] Its website is <www.occmed.oxfordjournals.org>.

These papers beg the question as to what standard of fitness holders of the RLSS Bronze Medallion should attain and maintain bearing in mind the need not 'to beat the aqualung' whilst undertaking subaquatic rescue, let alone 'beat the bubbles' to the surface. There is a plethora of other considerations concerning physical capacities of rescued and

rescuer divers once the surface has been reached – but at the end of the day if you can't face the risk, don't dive!

**References**


1 Reilly T, Wooler A, Tipton M. Occupational fitness standards for beach lifeguards. Phase 1: the physiological demands of beach lifeguarding. *Occup Med (Lond)*. 2006; 56: 6-11.

2 Reilly T, Iggleden C, Gennser M, Tipton M. Occupational fitness standards for beach lifeguards. Phase 2: the development of an easily administered fitness test. *Occup Med (Lond)*. 2006; 56: 12-7.



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**Key words**
Letters (to the Editor), health, standards, rescue


## Maintenance of Professional Standards

Dear Editor,

Here are the details of the Maintenance of Professional Standards (MOPS) points approved by the Australian and New Zealand College of Anaesthetists (ANZCA) for the SPUMS 34th ASM, Fiji, 6–10 June 2006.

- Meeting sessions: 12 CME points, under Code 111.
- Hypothetical sessions (8 June 2006): 6 QA points, under Code 211.
- Airway workshops sessions (9 June 2006): 8 CME and 4 QA points, under Code 700.


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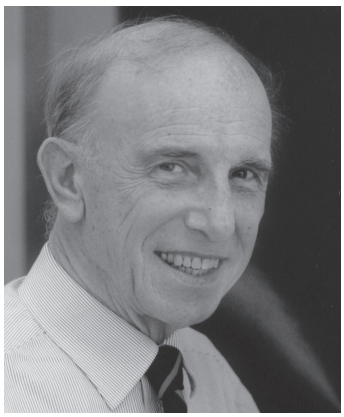
**Key words**
Letters (to the Editor), meetings, MOPS


# Obituary

## Brian Andrew Hills



Physiologist
Born: Cardiff, Wales,
19 March 1934
Died: Brisbane,
13 January 2006

Brian Andrew Hills was the head of the Golden Casket Paediatric Research Laboratory at the Mater Medical Research Institute in Brisbane for 10 years, where he studied surfactant and its role in health conditions such as sudden infant death syndrome (SIDS), asthma and glue ear. It was a research interest seemingly far removed from his engineering training, which offered a new insight to biological puzzles.

An outstanding student, he was awarded a full scholarship to Cambridge University in 1952, where he obtained a Bachelor of Arts in Physical Sciences and a Masters in Chemistry. After university, he worked as a chemical engineer in Malaya and New Zealand before entering academia in 1963 as a senior lecturer in Chemical Engineering at the University of Adelaide. He also embarked on a doctoral thesis investigating the prevention of bubbles in nylon 'melts' during the spinning of the fibres. However, it wasn't long before a chance encounter inspired him to change his thesis topic and launched him into the first of his two research interests.

Sitting next to a physiologist in the staff room one day, he became engaged in a discussion of how to prevent bubbles forming in divers. Recognising the similarities between this problem and that at the heart of his thesis, he was fascinated with the biological bubbles and soon switched the bubble topic of his thesis from nylon melts to decompression sickness, or 'the bends'.

After becoming embroiled in Hugh LeMessurier's study of the diving habits of the pearl divers of the Torres Straits and Broome, Brian embarked on a complete re-evaluation of the literature, both medical and non-medical, and practices of decompression. His thesis, published in 1966, advocated the use of adding a 'deep stop' to a diver's ascent, rather than the conventional, longer stop in shallower waters. He proposed an entirely new method of determining decompression schedules, and most of today's decompression schedules pay homage at least in part to Brian's studies. Unfortunately, the mathematical complexity of his diffusion-based model resulted in much of this work being overshadowed by the simpler, traditional methodology adopted by the US Navy.

Over the next five years, at Brown and Duke Universities in the USA, he pursued his interest in the behaviour of gases in the body leading to decompression sickness. In 1975, he was awarded a higher doctorate from the University of Adelaide for his research on decompression sickness, and in 1977 published a book on the topic.

After a brief return to England at London University, he spent three years as Professor of Physiology at the University of Texas Medical School at Galveston, before joining UTMS at Houston after a year's sabbatical at the University of Dundee, Scotland. He was a consultant to several diving companies, as well as a consultant to NASA, advising on the gaseous environment to be used in the shuttle and space station.

While at Duke University, he had discovered that the bubbles formed in many tissues throughout the body by decompression sickness were coated by surfactant. His discovery was significant since surfactant had previously been studied only in the lung. From the late 1970s, his research focused increasingly on surfactant.

Once again, he called on his training as a chemical engineer to explore the mysteries of the human body. Recognising the similarity between biological and industrial surfactants, which are used as lubricants, release (anti-stick) agents, protective barriers and corrosion inhibitors, he set about locating surfactant in areas of the body where these physical properties would be beneficial. He became a world authority on surfactant, which was recognised by Cambridge University with a higher doctorate in 1981. In 1988, his book *The biology of surfactant* was published.

He often joked that his wife, Audrey, had never forgiven him for their leaving Australia in the 1960s. It was perhaps with the desire to one day retire in Australia that he took up the position of Professor and Head of the Department of Physiology at the University of New England in Armidale, NSW, in 1986. Here, he sought to develop treatments for health conditions that his research indicated were associated with a deficiency of surfactant, such as osteoarthritis and peptic ulcer.

In the early 1990s, while still at UNE, he collaborated with Dr Brent Masters, who was then at the Mater Children's Hospital, to demonstrate differences between lung surfactant from healthy infants and those who experienced prolonged apnoeic episodes.

In 1994, Brian became the head of the Golden Casket-funded Paediatric Research Laboratory, the first laboratory at the Mater Medical Research Institute. His major project in the early years was SIDS. He demonstrated abnormal properties of lung surfactant from infants who had died of SIDS, which had potential as a test at birth for SIDS risk.

His research later focused on asthma, which he believed was associated with a deficiency in a protective coating of surfactant lining the airways. A clinical trial sponsored by a British pharmaceutical company showed promise in using synthetic surfactant to prevent asthma. The same surfactant also showed encouraging results in treating otitis media (glue ear). He also developed an artificial lubricant which was trialled in patients with osteoarthritis.

Outside of work, he was a keen sailor, and on weekends he could often be found enjoying a sail on Moreton Bay. However, he was never happier than when he was in the laboratory, extolling the virtues of surfactant. His greatest wish was to see the commercial development of one of his surfactant treatments, as confirmation of his theory of the mechanism of surfactant. Although further clinical trials are on the agenda, his wish will not be fulfilled.

Over the last few years Brian had his interest in decompression reawakened by correspondence and visits from technical divers keen to learn about his earlier work. Always keen to teach, Brian welcomed and corresponded vigorously with these divers despite his deteriorating health.

He had battled cancer for many years, and retired two years ago due to his declining health. He died at Mt Olivet Hospital, where he had been admitted in December. He is survived by his wife Audrey and children Yvette and Graham.

*Yvette Hills and Andrew Fock*

**Key words**
Obituary, decompression, decompression sickness, surfactant

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# A personal view of Brian Hills' contribution to decompression theory and practice

David Doolette

It was with great sadness that I learnt of Brian Hills' death in January this year. I often felt I was following in his footsteps: I likewise earned my PhD at the University of Adelaide (although some 30 years later), continued work there on decompression theory, and the very small but useful collection of relevant literature in the library clearly spanned Brian's tenure. Brian's early decompression work was done with Hugh LeMessurier's aeromedicine group in the Department of Physiology, and I was able to learn some anecdotes from a contemporary. One task requested by the Air Force was the practical issue of what would happen to a thermos of hot coffee during an explosive loss of cabin pressure – a question they answered with messy results in the group's man-rated hypobaric chamber.

A serious student of decompression theory soon finds that much of the work on the science of decompression is difficult to source, being in technical reports, PhD theses, out-of-print books, or never published. Brian Hills, however, made a prodigious contribution to the mainstream scientific

literature. A Medline author search returns 186 articles spanning the years from 1967 to 2006, of which the first 15 years comprise his significant, often iconoclastic, contribution to decompression theory. I have read, re-read, and evaluated much of that. I met Brian only once but that was a fascinating two hours of conversation.

The title of his paper 'Limited supersaturation versus phase equilibration in predicting the occurrence of decompression sickness' sums up the central premise of Brian's PhD thesis and his book *Decompression sickness: the biophysical basis of prevention and treatment*.[1–3] Since the original work of Haldane and colleagues,[4] a simplifying assumption of the methods used to control decompression is that the body tissues can become supersaturated with dissolved gas during decompression without forming bubbles. Although intended only for mathematical simplicity, this assumption entrenched the erroneous concept that bubbles do not form in the body during safe decompression.

Brian's 'thermodynamic decompression model' was amongst the first in which decompression is controlled by the amount of gas that comes out of solution and forms bubbles. In this model, pain-only decompression sickness (DCS) is controlled by a single tissue in which gas uptake is diffusion-limited and where dense bubble formation during decompression causes 'phase equilibration' of gas partial pressures between the dissolved and free gases (another simplifying assumption). The driving force for gas elimination in a phase-equilibrated tissue is what Brian described as the inherent unsaturation (named the partial pressure vacancy or, more commonly, the oxygen window by others) whereby oxygen consumed in tissue is replaced by fewer molecules of more soluble carbon dioxide. The thermodynamic model was used to explain the empirically derived decompression regimens of the Torres Strait Island pearl divers, which involved deeper decompression stops and shorter overall decompression times than prevailing navy decompression schedules. This deep skew of decompression stops is a feature of all modern bubble decompression models.

The breadth of his other contributions to decompression science is too great to fully cover here. For instance he developed two early decompression computers (a thermal analogue and pneumatic analogue), a method to detect tissue bubbles using electrical impedance, and a new animal model for DCS (the kangaroo rat). He conducted theoretical and experimental investigations of bubble nucleation, inert gas uptake and washout, isobaric gas counterdiffusion, acclimation to DCS, and much more. Although some of his ideas are inconsistent with modern thought, the thermodynamic decompression model was a seminal work that profoundly influenced subsequent decompression model development.

*David Doolette, PhD, Research Physiologist, US Navy Experimental Diving Unit and Assistant Research Professor, Duke Medical School, North Carolina, USA*

**References**

1 Hills BA. Limited supersaturation versus phase equilibration in predicting the occurrence of decompression sickness. *Clin Sci*. 1970; 38: 251-67.
2 Hills BA. *A thermodynamic and kinetic approach to decompression sickness*. Adelaide: Library Board of South Australia; 1966.
3 Hills BA. *Decompression sickness: the biophysical basis of prevention and treatment*. Chichester, UK: John Wiley & Sons; 1977.
4 Haldane JS, Boycott AE, Damnant GCC. The prevention of compressed air illness. *J Hyg*. 1908; 8: 342.

**Key words**
Obituary, decompression, decompression sickness

# Book reviews

## Under water to get out of the rain

### Trevor Norton

400 pages, hardback
ISBN 0-71263-884-9
London: Random House; 2005
**Price:** GBP 12.99

I have a strong affinity for Trevor Norton and his writings. Though we have never met, his and my diving history have been on parallel paths from our first mask and snorkel with a caged ping-pong ball in the same summer, he in the North and I on the south coast of England. Television changed both our lives as teenagers as we watched *Diving to adventure*, admired Hans Hass but fell in love with the beautiful Lotte. We both learnt to dive in the early 1960s and I dived with some of the people he mentions. Our first university diving expeditions, he to the Canaries and I to Cyprus, were in 1963. We were both involved, and did not know it till I read this book, in preparations for *Kraken*, a British living-under-the-sea project in the 1970s that never materialised. Whilst I was conducting diver-performance experiments off Oban, Trevor was diving the same west coastline of Scotland, etc.

This autobiography is his third book written for a wide audience, the previous two both reviewed enthusiastically in this journal.[1,2] The tone is set in the first few pages, when Norton describes his first seaside holiday as a young boy to Scarborough, launching into an entertaining history of this spa and the British love affair with the seaside. Trevor has what one critic described as a "novelist's sensibility and eye for detail", and his writing throughout remains witty and entertaining. Moments in his life, both personal and professional, are merely stepping-off points from which to cover a huge, often unexpected variety of topics from monsters to Ama divers, filter feeders to marine mammals, Steinbeck to seaweed sex, a concentration camp on the Isle of Man to algal blooms and whisky distilleries on Jura to American Imperialism in the Philippines.

His eye is that of the natural-born observer that he surely is as a professor of marine biology. It is as though he is saying "Well, that's enough about me, what about these people and events or that aspect of the marine world, now isn't that far more exciting and fun!" It is this love of life and the world we live in that transcends his book, and is further enlivened by his wife Wyn's delightful drawings. Inevitably the book ends on a note of caution as the final chapters enumerate humanity's pollution, overuse and damage to the oceans as Norton has seen and experienced them in his professional work around the globe in collaboration with many other marine scientists. The trouble is, he says, that we take the sea for granted. This is unashamedly a poignant love story. "*Once you have a taste for the ocean, the intoxication lasts a lifetime*." Indeed it does, Trevor.

**References**

1 Norton T. *Stars beneath the sea: the extraordinary lives of the pioneers of diving*. London: Century; 1999. Reviewed in *SPUMS J*. 2000; 30: 90-1.
2 Norton T. *Reflections on a summer sea*. London: Arrow books; 2002. Reviewed in *SPUMS J*. 2003; 33: 57-8.

*Michael Davis*
*Editor, Diving and Hyperbaric Medicine*

**Key words**
Book reviews, autobiography, biology, ecology, diving research, diving scholars, history, general interest

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## Deeper into diving, 2nd edition

### John Lippmann and Simon Mitchell

512 pages, soft cover
ISBN 0-9752290-1-X
Melbourne: JL Publications; 2005
Available from: <www.submarinerpublications.com>
**Price:** A$89 (within Australia inc GST); A$98 (outside Australia), airmail postage included

John Lippmann of *Oxygen first aid* fame and Simon Mitchell of everything-about-diving fame, combine for this the second edition of *Deeper into diving*. The beautiful cover photograph, by David Doubilet, of Chandelier Cave in Palau sets the scene for what is a comprehensive review of the physics, physiology and pathology of diving as well as easy-to-read technical chapters. The title refers not only to a more detailed and in-depth review of diving in general, but also to the technical chapters which advise on mixed-gas and rebreather diving allowing greater depths to be attained. The acknowledgements read like a 'who's who' of diving medicine and provide a solid base for the book. It should be suitable not only for recreational and commercial divers, but technical divers and medical professionals as well.

The book is divided into four sections. Section one deals with the physical and physiological aspects of diving, outlining the pathogenesis of decompression illness (DCI), its clinical manifestations and prevention. It includes interesting chapters on carbon monoxide and diving, heat loss and oxygen. The oxygen chapter looks like it has largely been lifted from John's previous book, *Oxygen first aid*, but is nonetheless clear and informative. In recognition of the male-dominated diving world, women get their own chapter with special attention to the pregnant diver and to whether women are at increased risk of DCI. The next section is dedicated to decompression systems, reviewing the various dive tables and the theories behind them. Of particular use to the technical divers is a chapter on decompression software. Dive 'computers' have come a long way since their introduction in the 1960s and this short chapter provides an excellent review.

Section three reviews altitude issues, both flying after diving and diving at altitude. Finally, section four reviews technical and occupational diving with excellent chapters on nitrox and mixed-gas diving.

Each chapter is well referenced and cartoons, tables and photographs illustrate the book. Unfortunately the photographs are not in colour, which lessens their impact. Most chapters conclude with a useful summary box and the decompression chapters have MCQ-type questionnaires.

All in all the book is a good read and, I think, ideally suited to the recreational sport diver who is keen to up the ante somewhat and venture beyond so-called 'no decompression' diving to go *Deeper into diving*.

*Sandy Inglis*
*Emergency Medicine Specialist, Christchurch Hospital*

**Key words**
Book reviews, scuba diving, decompression, general interest

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# Diving in high-risk environments, 3rd edition

## Steven M Barsky

226 pages, soft cover
ISBN 0-9674305-1-8
Santa Barbara, CA: Hammerhead Press; 1999
Available from Hammerhead Press & Hammerhead Video, 2419 E. Harbor Blvd #149, Ventura, CA 93001, USA
**Phone:** +1-805-985-4644; Fax: +1-805-382-6755
**E-mail:** <www.books@hammerheadpress.com>
**Website:** <www.hammerheadpress.com>
**Price:** US$31.95 plus postage and packing

Steve Barsky has been a prolific writer in sport and commercial diving with several books and many articles to his name. *Diving in high-risk environments*, now in its third edition, was first published in 1989 and is regarded as the standard text for diving in contaminated environments. It starts with a warning, "*Diving in contaminated water is very hazardous and is not recommended*." Unfortunately sometimes there is no choice. Whilst many situations can be approached effectively from first principles, it is useful to have a resource that summarises the range of environmental problems that an employed diver might face and that helps to understand the range of tools and diving techniques that are available to support such diving operations. This book is aimed at the commercial diver and as such is an excellent reference, but it is also of value to any diving physician whose advice is periodically sought from a health and safety perspective. Barsky's obvious knowledge of the subject gained from his experience as a commercial diver, development engineer (of life-support and protective equipment) and search-and-rescue diver gives him the ability to convey technical information in a clear and easy-to-read text. From the perspective of commercial divers, the text points out that they often work in environments of which they have little knowledge – "if it looks clear, it's okay" is not always OK!

The contaminants that may affect the diver and his equipment are extremely varied, and do damage at such different rates that it is essential they be identified beforehand and the diver 'dressed in' accordingly. Also, systems to get the diver into and safely out of the water to a decontamination zone need to be planned and in place before the dive. Several case histories illustrate the problems that can arise when this is not done. Every hazard varies with concentration, exposure time, equipment compatibility and other chemicals that may have altered the physical make-up of the diving equipment at some earlier date. After the dive a data sheet with the chemical/compound strengths and approximate dilutions, exposure times, type of protection and decontamination used, along with the data regarding any breakdown or chemical permeation of the gear during exposure, should be attached to the dive log sheet. All this information should be available to medical personnel in the event of medical attention being required.

Following a brief introduction, there is a good summary of the range of chemical, biological and radioactive contaminants that may be encountered. A source of emergency advice in the USA (readers in other countries should establish these sources for themselves) is provided and the levels of protection recommended by the Environmental Protection Agency summarised. The next four chapters outline scuba and surface-support diving equipment, dive planning and techniques. For diving physicians, these four chapters also provide an excellent overview of commercial diving equipment and techniques in general.

The next chapter deals briefly with the principles of decontamination, and is followed by a chapter devoted specifically to diving in potable water. The final chapter focuses on equipment maintenance. A series of appendices includes 12 tables of test results for various contaminants and items of diving equipment. There is a glossary of terms, a good bibliography and a useful index. The illustrations and photos are of a high quality and these and the various tables are all relevant to the text they accompany. Overall, this is a useful, well-written and excellently presented reference book. Since this edition is now some seven years old, one anticipates that a new edition is likely soon.

*Trevor Carson, hyperbaric technologist, commercial diver*
*Michael Davis, Editor, Diving and Hyperbaric Medicine*

**Key words**
Book reviews, occupational diving, occupational health, environment, exogenous poison, toxins, infectious diseases, irradiation, risk management

# Deep water gold

## Keith Gordon

240 pages, soft cover
ISBN 0-473-10056-8
Whangarei, New Zealand: SeaROV Technologies Ltd; 2005
Available from SeaRov Technologies Ltd, PO Box 1094, Whangarei, New Zealand
**Fax:** +64-(0)9-434-4164
**E-mail:** <searov@xtra.co.nz>
Copies can be ordered online at <www.deepquest.co.nz>
**Price:** NZ$39.95 plus postage and packing

It is unusual for an adventure diving book to be reviewed in the Journal. However, there are several reasons why Keith Gordon's story of *RMS Niagara* merits this. It is a story combining the history of a famous ship linked closely to Australia and New Zealand, wartime action, gold and diving techniques spanning over half a century. The small handful of technical divers to have dived this wreck includes a SPUMS member, whose dive resulted in a new understanding of the potential mechanisms of inner ear decompression sickness, suffered on the ascent by his dive buddy.[1]

*RMS Niagara*, launched at Clydebank, Glasgow in 1913 for the Union Steam Ship Company, was known as the 'Queen of the Pacific', and was regarded as one of the finest ships afloat. A 13,415 tonne steamship capable of carrying 667 passengers, 237 crew and over 3,000 tonnes of cargo at an average speed of 16 knots, she plied the Pacific route between Vancouver, Auckland and Sydney for over 25 years. She could easily outrun the German raiders in the Pacific during World War One, but was not without her moments of infamy. History has often, and almost certainly unjustly, blamed the *Niagara* for the introduction of Spanish flu into New Zealand in 1918. Isolated by oceans, Australia and New Zealand were susceptible to epidemics introduced by crew and passengers on ships arriving from afar, necessitating strict quarantine measures, and often housing arrivals in appalling conditions in quarantine stations.

In June 1940, the armed merchant German cruiser *Orion* laid 228 mines across the approaches to the port of Auckland. *HMS Achilles* and *Hector* and the *Niagara* were in port at the time. In fact, the two warships returned to port at night whilst the mine laying was in progress, neither combatant being aware of the other's close proximity. A little over 24 hours later, the *Niagara* hit one of *Orion's* mines and sank about 200 km north of Auckland settling on a sandy bottom at a depth of 396 feet of sea water.

Quite apart from the fame of the *Niagara*, it was part of her cargo on the day that she sank that fired the imagination of a generation – gold. In her strongroom, packed in 295 wooden boxes, were 590 gold bars weighing over eight tons and worth in 1941 over £2.5 million. Gordon links the past with the present in this excellently written story of the *Niagara*, the German raid, the epic gold salvage ventures and the more recent exploration of this famous deep water wreck.

The story is presented in four parts. The first is devoted to the *Orion* and its mine-laying raid, and to the history of the *Niagara* and its sinking without loss of life. The second section is devoted to the major gold salvage projects, the first of which occurred in 1941, with the chief diver, a famous Australian diver "Johnno" Johnstone, working in a one-atmosphere diving bell and directing a surface-operated grab. All but 35 gold bars were recovered. Further salvage attempts were made in the 1950s, 30 more bars being recovered; the remaining five bars, now worth approximately $US900,000, are still on the wreck. The descriptions of the diving and salvage techniques used make fascinating reading.

The third section describes Gordon's fascination, from a boyhood in Christchurch diving with Wade Doak and Kelly Tarlton, with the sea, diving and the wreck of the *Niagara*. From the late 1980s, Gordon explored the wreck using remote operating vehicles, but it was not till 1999 that the wreck was first dived using ambient pressure as opposed to one-atmosphere techniques. Since then only a small handful of technical divers have dived the wreck, resulting in at least three cases of decompression sickness, including the one with an inner ear decompression injury (above).

The final short section traces Gordon's historical research in Britain and Germany. Many of the files on the *Niagara* are still inaccessible on national security grounds, some not for release until 2061! Keith's excitement in visiting the archives of the Bank of England and Guildhall, the National Archives in London and the Institute of Shipping and Naval Information in Hamburg is palpable.

This is an excellently written, fascinating historical text. It is illustrated with about 100 photographs and paintings and the typographic errors are minor and acceptably few. The text is well presented, there are several short appendices, a glossary of terms and a useful bibliography and index. Gordon avoids many of the journalistic pitfalls so often present, and to this reader irritating, in diving books, most particularly their repetition and tendency to eulogise the diving participants. This is a matter-of-fact but nonetheless exciting narrative, as the back cover says "*a true adventure story to hold the interest of all*."

*Michael Davis,*
*Editor, Diving and Hyperbaric Medicine*

**Reference**

1 Doolette DJ, Mitchell SJ. Biophysical basis for inner ear decompression sickness. *J Appl Physiol*. 2003; 94: 2145-50.

**Key words**
Book reviews, wreck diving, salvage, deep diving, history, general interest

# The DAN guide to dive medical frequently asked questions (FAQs)

## Divers Alert Network

232 pages, soft cover
ISBN 1-930536-18-6
Durham, NC: Divers Alert Network; 2003
Available from Divers Alert Network, The Peter Bennett Center, 6 West Colony Place, Durham, NC 27705 USA
Order online at <www.diversalertnetwork.org>
**Price:** US$12.95

This handy, reference-style soft cover published by DAN appears to be targeted mainly at the recreational diver or instructor. It professes to answer the most frequently asked queries relating to dive medicals received by DAN and reprints many articles previously published in *Alert diver*.

Organised into sixteen alphabetical sections, it covers fitness-for-diving issues in a systemic way, as well as including a general section allaying a variety of concerns from mask squeeze to the risk of Alzheimers associated with the use of aluminium tanks. A Q&A format is used for the introduction to each chapter and frequent case histories are used to illustrate points.

This book does not purport to being a diving medicine text. It is not referenced but does have a useful index. I found it accessible and to contain a considerable amount of useful information. With numerous authors contributing sections the standard is a little variable and the Q&A style is not one that I personally enjoy. However, despite these reservations I could envisage this book being a useful addition to the shelves of any dive shop or the personal library of a recreational diver. At US$12.95 it would seem good value.

*Dennis Boon von Ochssée*
*Department of Hyperbaric Medicine, Christchurch Hospital*

**Key words**
Book reviews, fitness to dive, DAN - Divers Alert Network

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**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>

# The poetry doctor

## The wreck

It looks so sad standing lone and desolate,
A reflection of an age gone by,
Superstructure sagging with underhanging weight,
Skin encrusted, tarnished by salt and sky.
It creaks and groans with any shift or slack,
All moving parts stiff with lack of use,
Blurred portholes of eyes stare quizzically back,
How does one sink into such self abuse?
Will this decline continue is the fear
For before the mirror it looks hardly alive?
Thank God it's SPUMS conference time of the year,
An annual reason to get moving and dive.

I wrote this with plans to get to the Fiji conference. Instead I find myself on an emergency mission with MSF in northern Nigeria in the midst of a meningitis epidemic. It is another way to blow away the cobwebs and appreciate how wonderful life is, but I assure you the SPUMS conference will be far more comfortable.

John Parker
<www.thepoetrydoctor.com>

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# SPUMS Annual Scientific Meeting 2007

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

**Guest Speaker**
Neal Pollock, PhD, Duke University

**Theme**
*From mountain high to ocean deep*
*The physiological challenges of extreme environments*

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

This will be an outstanding meeting in a beautiful area of New Zealand with superb diving at one of the world's finest sub-tropical diving sites – The Poor Knights Islands. You will need a wetsuit, but don't let that put you off!

Full registration details and a Call for Papers will accompany the September issue of the Journal.

**Preliminary enquiries to:**
Associate Professor Michael Davis
PO Box 35, Tai Tapu 7645, New Zealand
**E-mail:** <michael.davis@auckland.ac.nz>
**Phone:** +64-(0)3-329-6857
**Fax:** +64-(0)3-329-6810

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## 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:
**E-mail:** <TSV-HTNA2006@health.qld.gov.au>
**Phone:** +61-(0)7-4796-2080
**Fax:** +61-(0)7-4796-2082

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## 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>

## 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

The Sultanate of Oman lies at the crossroads of Africa, Europe and Asia. Muscat '06 is an opportunity to attend a congress of the highest quality and to visit an exotic country with a unique heritage, renowned for its hospitality and safety for you and your family.
The congress is accredited by the School of Medicine of the University of Auckland for the postgraduate programmes in diving and hyperbaric medicine.

**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>

THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND HEALTH SCIENCES

**Postgraduate Programme in Medical Science – Diving and Hyperbaric Medicine**

Enquiries are now being accepted from registered medical practitioners for the 2007 Postgraduate Diploma in Diving and Hyperbaric Medicine. During 2006, 17 physicians are undertaking the programme.

**Teaching faculty:** Des Gorman, Mike Davis, Simon Mitchell, Chris Acott, Drew Richardson, Bill Baber and others.

**Overview:** The Diploma is a distance-learning programme, available internationally without a resident component in Auckland. However, attendance at a recognised short course in diving medicine is a prerequisite. Graduates will be able to practise effective clinical diving medicine in a primary-care setting or to embark on clinical practice within a hyperbaric medicine environment.

For further information, including fees, please contact the Course coordinator:
Upendra Wickramarachichi
**Phone:** +64-(0)9-373-7599, extn 83058
**Fax:** +64-(0)9-373-7006
**E-mail:** <u.wicks@auckland.ac.nz>

To find out more about our courses and your options for entry level (PG Diploma in Medical Science or Master of Medical Science) please submit an online **Expression of Interest** in this subject at the website:
<www.health.auckland.ac.nz>
by clicking on the Quicklink to Postgraduate Study.

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**2007 ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS' UNDERWATER MEDICINE COURSE**

**Dates:** 09 to 20 July 2007
**Venue:** HMAS Penguin, Sydney
**Cost:** $1833.00 (tbc)

The Medical Officers' Underwater Medicine Course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses.
**For information and application forms contact:**
The Officer in Charge, Submarine & Underwater Medicine Unit, HMAS PENGUIN,
Middle Head Road, Mosman, 2088 NSW, Australia
**Phone:** +61-(0)2-9960-0572
**Fax:** +61-(0)2-9960-4435
**E-mail:** <Sarah.Sharkey@defence.gov.au>

**ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE COURSES 2006**
**Medical Officers Course October 2006**
Basic 16/10/06 to 20/10/06

**DMT Full Course**
**October 2006** 3 weeks, 09/10/06 to 27/10/06

**DMT Refresher Course**
**December 2006** 04/12/06 to 08/12/06

**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>

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**UNDERSEA and HYPERBARIC MEDICAL SOCIETY**
**Annual Scientific Meeting 2007**
**Preliminary Announcement**

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

**For additional information:**
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**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>

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

**Dates:** 12 to 15 April 2007
**Venue:** Sanur Paradise Plaza Hotel, Bali, Indonesia
**Registration details and further information contact:**
**E-mail:** <secretary@ahdma.com>
**Website:** <www.ahdma.com>

# 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 other software 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.**
**Enquiries to: <diverhealth@hotmail.com>**

## DIVING INCIDENT MONITORING STUDY (DIMS)

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

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

## DIVING-RELATED FATALITIES RESOURCE

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

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

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

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