South Pacific Underwater Medicine Society Journal

ISSN 0813 - 1988
ABN 29 299 823 713

Volume 34 No. 1
March 2004

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

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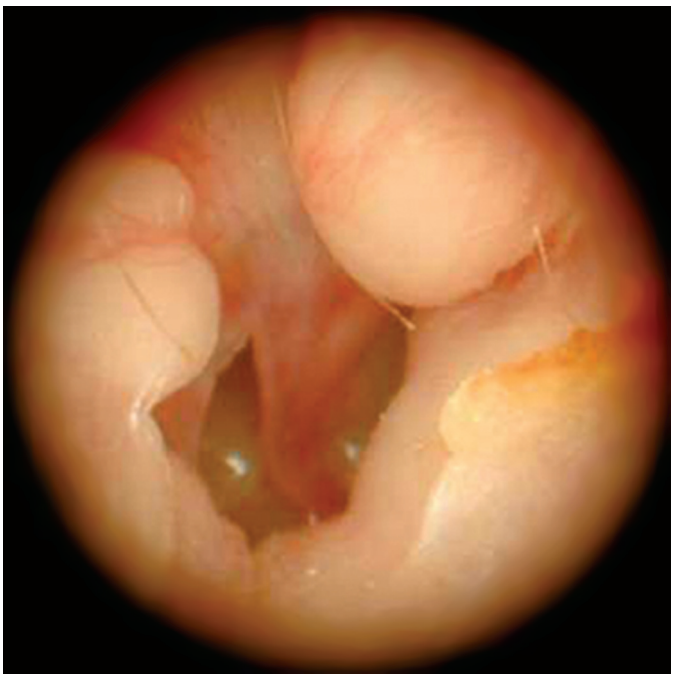

The ear and diving

Inner ear barotrauma

Inner ear decompression sickness

Carbon monoxide poisoning

Flying after diving

Diving accidents Workshop

Diving medicine in Vanuatu

Print Post Approved
PP 331758/0015

OBJECTS 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

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ADMINISTRATION

MEMBERSHIP

Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
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Further information on the Society may be obtained by writing to:
SPUMS Membership,
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The Society's financial year is January to December, the same as the Journal year.
The 2004 subscription will be Full Members A$132.00 and Associate Members A$66.00, including GST in Australia. All those outside Australia will be charged the same amounts as the GST component to partly cover the cost of having the Journal delivered to them by Air Mail. These fees must be paid in full.

The Editor's offering

The major focus of this issue is inner ear injury from diving. Whilst middle ear barotrauma is the commonest diving medical problem presenting to health workers, few see more than a handful of inner ear problems in a career. This makes Edmonds' large series of inner ear barotrauma all the more remarkable. Enthusiasm for early surgical intervention in round-window rupture has given way to a more conservative approach. Vestibular decompression sickness (DCS) is clearly not limited to deep heliox or mixed gas diving, though with the upsurge in 'tech' diving its frequency will increase. Wong and Walker make the point that many cases present out of hours when sophisticated otological investigations are unavailable, and this lends credence to the view of Klingmann et al that where there is diagnostic doubt one should do bilateral myringotomies and administer hyperbaric oxygen therapy. Doolette and Mitchell, in a paper reprinted from the *Journal of Applied Physiology,* hypothesise that in the inner ear a transient supersaturation for helium resulting in a counter-diffusion mechanism may explain the onset of DCS with breathing gas switches, and that these should be carefully scheduled on the ascent. It is evident from the papers presented here that a variety of mechanisms may lead to permanent inner ear damage from diving. A very recent paper suggests the likelihood of residual damage is greater with inner ear DCS than following barotrauma.[1]

Professor Gorman based a talk at the 2003 ASM on the paper reprinted here from *Toxicology*. The toxicity of carbon monoxide (CO), which is known to be a neurotransmitter, is a complex issue. He concludes "while the incidence and often poor outcome of CO poisoning is well recognised, and although the biology of CO is increasingly understood, the toxicology of the gas is mysterious and no data exist to establish best practice for managing poisoned patients."

There is increasing unease regarding the escalating financial and human costs of long air evacuations for hyperbaric treatment of tourists developing DCS in remote areas. One response has been the establishment, for instance by DAN and Subaquatic Safety Services, of recompression chambers in various countries around the world where diving activities are high but the local health infrastructure is minimal. As Grace points out this has its own disadvantages in that the local community then has to fund out of meagre health resources the maintenance of this facility - not cheap - and the hyperbaric care of uninsured tourists - the majority, I suspect. Combine this with real doubts about the cost-benefit of air evacuation in mild cases - also the majority - compared with conservative local medical care, and clearly we have a problem in the SEAP region that requires attention. The Phuket Workshop, in part, and that on remote management of DCS in Sydney in May are intended to address this issue. SPUMS members should always act responsibly when diving abroad and ensure they have adequate diving travel insurance.

This brings me to the UHMS and SPUMS Annual Scientific Meetings in Sydney and, immediately following, in Noumea. Both meetings are under-subscribed. Such a double opportunity to enhance knowledge and meet with international colleagues is rare. Please do give serious consideration to coming to one or both meetings. There is still time to register for what will be an exciting two weeks. If these meetings are not what you as a member want then take the time to write the Committee and tell them what activities SPUMS should be providing for its members.

Sadly, David Doolette has resigned as Education Officer. David has done an outstanding job maintaining the standard of theses for the SPUMS Diploma and helping applicants to see their research projects through to fruition. The high standards he has set have resulted in disappointment for some, but have also ensured that the Diploma has grown in stature. This has been clearly recognised by the Australian and New Zealand College of Anaesthetists in endorsing the SPUMS Diploma as the stepping stone for the College's Certificate in Diving and Hyperbaric Medicine.

If any academic member of SPUMS wishes to be considered for this extremely important role within the Society then please contact the President. We need to find a replacement urgently. In the interim, all enquiries regarding the Diploma should be addressed to the Editor, who is a current member of the Academic Board, at the journal address.

Recently, a working group reviewed the Society's rules, which had not been revised since 1999. Several proposed, mainly minor, changes for consideration at the AGM in June 2004 are posted in this issue. Most importantly, it was felt that the international standing of this journal would be enhanced by the adoption of a less parochial name. SPUMS is now an international society with nearly 20% of its membership outside of the South-East Asia Pacific region. Choosing a journal title is not easy, though many entertaining acronyms were put forward!

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Mike Davis

Front cover photos depict the ear of a surfer showing exostoses in the external canal and a traumatic perforation of the tympanic membrane courtesy of Murray Grieg, an otolaryngologist in Christchurch, and 'The Lady' from the wreck of the *President Coolidge* courtesy of Robert Grace.

We are still hoping the many SPUMS photographers will send us photos for the journal front page – hope lives eternal!

Invited editorial

Diving and inner ear damage

Carl Edmonds

Key words
Editorials, inner ear barotrauma, inner ear decompression illness, inner ear decompression sickness

The most recent and comprehensive review of the history of this subject was made by Shannon Hunter and Joe Farmer, of Duke University, Durham, North Carolina.[1] Most of my references are in that document, and I will restrict myself to a brief overview and some recent observations.

Those who do not know the past are doomed to repeat it. In the 19th century A H Smith described 'caisson disease', affecting the ear. Alt and Heller demonstrated inner ear injury during compression and decompression and identified the cause in both humans and animals. Heller described 24 cases of a Ménière-like syndrome, and noted that these cases had undergone very long exposures in caissons. Vail, in 1929, eloquently explained the pathophysiology of the effects of both barotrauma and decompression sickness (DCS) on the inner ear.

Then, somehow, all this was forgotten. World War Two saw an increase in diving, and men from the artillery became divers. Hearing loss was attributable to noise exposure and gunfire, and otologists dismissed the possibility of diving/caisson-induced inner ear damage. In the diving world until the 1970s, the consensus was that diving only induced transitory middle ear damage, called ear squeeze or reverse squeeze.

Rubenstein and Summitt, of the US Navy Experimental Diving Unit, described 10 cases of inner ear decompression sickness (IEDCS) caused mainly by very deep diving.[2] All presented with severe vertigo, some with tinnitus and sensorineural deafness. Buhlmann and Gehring, from Zurich, described another 12 cases, mainly the result of deep heliox dives.[3] Farmer and his colleagues from Duke University added another 13 cases, and the concept of IEDCS was established and defined.[4]

Few of these cases resulted from recreational dives. They tended to be very deep air or heliox dives, precipitated by gas switching to air, and free from other manifestations of DCS. These observations were substantiated when Lambertsen and Idecula, in 1975, observed vestibular lesions while breathing a heavy, slow-moving gas (air, neon) in a fast-moving gas environment (helium), this counter-diffusion of gases occurring at a constant depth.[5]

Canadian researchers demonstrated the bubble production that characterises DCS within the first few hours and its conversion into haemorrhages and exudates in the inner ear.[6] Bubble enucleation in the osteoplastic cell cavities around the inner ear increases local pressure and may disrupt into the perilymph spaces. This pathology is converted, after a month or so, into fibrosis and new bone formation around the permanently damaged inner ear.

This explains why recompression therapy must be instituted rapidly, within the first three to six hours. If the diver has surfaced, a conventional long US Navy oxygen table is employed. If IEDCS develops during decompression, reversion to the original gas mixture, immediate descent to incident depth plus 3 Ata (303 kPa), and then oxygen supplementation, is indicated.

The above observations all seem to describe discrete entities with little confusion regarding diagnosis or symptoms. There have been occasional isolated cases of air divers and caisson workers, comprehensively documented and investigated, developing DCS including inner ear symptoms, such as those described by Reisman et al.[7] Excessive exposure (duration) was customary. Unfortunately other series of alleged IEDCS cases have less verifiable diagnoses.[8] This also applies to the current presentation, in which Wong and Walker highlight the difficulty in differential diagnosis.[9]

Such difficulties do not plague the diagnosis of inner ear damage from barotrauma (IEBt). This terminology was introduced by Freeman and Edmonds, when they documented and demonstrated these injuries by pre- and post-incident audiograms.[10,11] They showed that permanent hearing loss can follow barotrauma and that the pathology included round-window fistulae (RWF). The first two corrective operations for RWF were performed, very successfully, in Sydney in 1971. The Australian workers not only demonstrated sensorineural hearing loss, but also vestibular lesions, in the first text on diving otology.[12] Because the cases were clearly diagnosable, there was little contention when the findings disputed the conventional belief that air diving had no permanent audiological effects. That original work has not only been verified by many others, but is now complemented by a recent series of 50 similar cases.[13]

Sensorineural hearing loss, possibly with loud tinnitus and/or dysacusis, should always imply a possible diagnosis of IEBt. Pure tone audiometry (PTA) is essential in assessing diagnosis and severity. Vestibular symptoms may be of any severity, and sometimes the electronystagmogram (ENG) may demonstrate damage even without symptoms.

IEBt is usually associated with middle ear barotrauma (MEBt), and forceful attempts at middle ear equalisation (ME=), usually by a Valsalva manoeuvre. This increases pressure within the intracranial fluid and through its cochlear duct to the perilymph. These explosive pressures distort the inner ear membranes, one of which is the round

window. The latter may tear, producing a fistula and leaking perilymph, or stretch, allowing effusion of perilymph into the middle ear or air into the inner ear. Haemorrhages within the inner ear are likely.[1]

The explosive pressure from an attempted Valsalva is aggravated by the under-pressure in the middle ear (MEBt of descent). Implosive pressures can develop with over-pressure within the middle ear during ascent and with movement of the ossicular chain during sudden successful Valsalvas.

Oval-window fistula is less common than RWF, except in divers with otosclerosis or previous middle ear surgery. Thus the term 'labyrinthine-window fistula' is more accurate. Both a vertical orientation of the round window, and involvement of the fissure of Hurley, have been associated with RWF.

The pathophysiology guides the treatment of IEBt. The patient is advised to cease all activities that increase intracranial pressure (ME=, lifting, exertion, nose blowing or sneezing, coughing, sexual activity, straining at stool, etc.) He is kept at bed rest with the head elevated. PTA performed daily will monitor progress, as the membranes heal and haemorrhages settle, but if there is either severe or progressive hearing loss, exploration is warranted. Vasodilators and anticoagulants, such as nicotinamide and aspirin, are contra-indicated. Surgery is required less often now that careful attention is given to conservative management.

Improvement or even cure of the sensorineural deafness is possible in the first few weeks. The vestibular symptoms will diminish (unless the perilymph leak continues) over the next few weeks or months as cerebral inhibition of vestibular function develops, or if the vestibular damage improves. ENGs are invaluable in diagnosis, localisation and assessing outcome. They can be performed in recompression chambers.

Differential diagnosis is difficult if the dive profile does not clearly indicate either MEBt or DCS (vestibular or cerebellar). A comprehensive inquiry is needed to exclude MEBt. Other symptoms of DCS may be of value. The simple statement that the diver had 'no trouble with ME=' is inadequate, and a delay between dive and onset of symptoms does not discriminate between diagnoses. In the isolated cases of air divers and caisson workers reported by Reissman et al, excessive exposure (duration) was customary.[7] Recent cases have been postulated as not requiring such exposure, the inner ear lesion being produced by DCS gas emboli passing through a patent foramen ovale.

Oxygen recompression, although theoretically hazardous with IEBt, has been employed and even recommended as a valid treatment for sudden hearing loss.[14-16]

If there is permanent damage from IEBt, most recommend that diving, free or with equipment, cease, although this is contentious. Prophylactic advice should be given regarding ME= with aviation exposure.

Australasians have no reason to be modest regarding their contribution to diving otology. Australian workers have:

- compiled prospective documentation on MEBt (Bayliss, Edmonds, Lehm and Bennett);
- discovered and treated RWF;
- demonstrated and defined the causes of hearing loss and vertigo from diving (Edmonds, Thomas, Tonkin, Freeman, Blackwood);
- described orbital barotrauma[17] and the only two large series of sinus barotraumas (Fagan, McKenzie, Edmonds);[18, 19]
- produced the first two texts on diving otology, *Otological Aspects of Diving*, in 1973;[12] and, in New Zealand, Noel Roydhouse's *Scuba Diving and the Ear Nose and Throat* in 1975;[20]
- produced the latest clinical reviews available in diving medical texts;[1, 21]
- and currently our scientists are modelling the pathophysiology of IEDCS (Doolette, Mitchell).[22]

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Original articles

Diagnostic dilemmas in inner ear decompression sickness

Robert Wong and Margaret Walker

Key words
Decompression sickness, decompression illness, ear barotrauma, inner ear, labyrinth, case reports

Abstract

(Wong R, Walker M. Diagnostic dilemmas in inner ear decompression sickness. *SPUMS J.* 2004; 34: 5-10.)
Inner ear symptoms associated with diving illness may result from barotrauma or decompression sickness. Differentiation of the underlying pathology presents a diagnostic dilemma for the diving clinician, especially if remote from otological laboratories. We present a series of such cases, some of which were likely to be due to inner ear (vestibular) decompression sickness, and others which could be confused with this diagnosis. These cases highlight the diagnostic and management dilemmas involved.
Authors' note: The term decompression sickness (DCS) was chosen in preference to decompression illness (DCI), as the latter term may lead to confusion. Inner ear DCI could mean barotrauma or DCS.

Introduction

The symptom complex of vertigo, nystagmus and nausea occurring in decompression sickness (DCS) may be caused by injury to the inner ear, the cerebellum or their connections. Cerebellar vertigo is almost always associated with other signs of cerebellar dysfunction that are obvious on clinical examination, and nausea is not usually a prominent symptom. However, DCS is an acute disease affecting vestibulo-cerebellar connections and may also have associated raised intracranial pressure.

Injury to the inner ear, however, may be the result of barotrauma (Bt) or decompression sickness (DCS), and it may be difficult to differentiate between these two pathological processes. Barotrauma-induced injury may be associated with a history of difficulty in equalising the middle ear during the dive, and with clinical signs of tympanic membrane injury and hearing loss.

Isolated inner ear decompression sickness (IEDCS) occurs more commonly in mixed-gas divers, especially at times of change in gas mixture. It has therefore become a general perception that IEDCS is rare in compressed-air diving and that most inner ear symptoms that occur are due to inner ear barotrauma. Nevertheless, Reissman et al alerted medical personnel to be aware of the possible occurrence of IEDCS among the wider population of scuba divers.[1] Subsequently, Nachum et al reported their twelve years' experience with 24 divers and concluded that IEDCS in sport diving is not as rare as previously thought.[2] IEDCS is usually associated with other signs of DCS, and in their series as many as a quarter of the divers suffering from 'serious' or 'neurological' DCS had inner ear involvement.[2] Differentiation of IEDCS and inner ear barotrauma (IEBt) is important from a therapeutic point of view because, whereas early recompression is indicated for IEDCS, recompression is relatively contra-indicated in IEBt and may worsen the condition. This paper presents eight divers from Western Australia, Tasmania and Queensland with a referral diagnosis of DCS, all arising from air diving, and in whom vertigo was a predominant symptom. The diagnostic difficulties and their management are discussed.

Case One

A 44-year-old male with 30 years' diving experience was diving alone using hookah equipment. He dived as follows:
Dive 1: 15-17 metres of seawater (msw) for 60 mins, a surface interval (SI) of 15 mins
Dive 2: 17 msw for 30 mins, SI of 5 mins
Dive 3: 17 msw for 15 mins. He surfaced with no decompression stops.

Twenty minutes after surfacing, he experienced dizziness, nausea and vomiting, especially on looking upwards. He had a tendency to fall to the right, deafness in his right ear, a generalised headache and right facial paraesthesia. He was taken to hospital where he was given oxygen, intravenous (IV) fluids, antibiotics, prochlorperazine and bed rest with head-up tilt for two days. The provisional diagnosis was IEBt, although he claimed that he had not experienced any difficulty in equalising his ears. Tympanic membranes were normal and there was no nystagmus.

The day after his dive, he developed pain in his left elbow, right shoulder, right knee, both thumbs and his back. There was little change in his symptoms over the next 24 hours. By the third day, his paraesthesia and headache had resolved. However, his dizziness, nausea and limb pains persisted and his right ear remained deaf, with no tinnitus. On the fifth day, he saw his local doctor and on his own

insistence flew to Perth on a commercial flight (lasting two hours). He claimed that his symptoms did not deteriorate en route, but later told the nurses he felt worse in transit.

On examination, there was no nystagmus nor any signs of cerebellar abnormalities. Unsteadiness prevented an assessment of the sharpened Romberg test (SRT), but he walked with a broad-based gait. Tympanic membranes were intact and mobile, and tympanogram was normal. Pre-treatment audiometry revealed a high-frequency hearing loss of greater than 50 dB at 6000 and 8000 Hz. His previous annual medical from his employment had shown a near-normal audiogram in the right ear.

He was recompressed on Royal Navy Table 62 (RN62), and had two subsequent daily hyperbaric oxygen treatments (HBOT), after which his symptoms resolved except for SRT <20 secs and subjective hearing loss in the right ear.

He was then referred to and assessed by the same ENT surgeon who had operated on him in 1997 for an 'inner ear fistula' following a diving incident. His pure tone audiogram showed 60 dB loss at 8000 Hz. A surgical exploration found no evidence of a perilymph fistula on this second occasion.

On review three months later, he still had some dizziness when he moved his head quickly, but his balance was normal and he was able to ride a motorcycle. Despite advice to the contrary, he had returned to diving.

DIAGNOSIS: Decompression sickness; neurological, vestibular and musculoskeletal

COMMENT: The presence of other symptoms of DCS (paraesthesiae, headache and limb pains) in the absence of any history of barotrauma point toward a diagnosis of IEDCS. Recompression therapy would have been indicated anyway due to the presence of the other symptoms.

Case Two

A 51-year-old, experienced diver who had been diving since he was 15, was diving off the Abrolhos Island. He dived as follows:
Dive 1: 40 msw for 30 mins, stopping at 6 msw for 10 mins to decompress, SI of 1-1.5 hours
Dive 2: 27 msw for 40 mins, surfacing without a decompression stop.

On surfacing, he felt nauseated, and claimed that his abdomen felt "bloated". This progressed to vertigo and vomiting over the next two hours. He felt weak and was unable to stand. He informed the divemaster of his symptoms and was told that he must have gastroenteritis. The following day, still feeling unwell, he was transferred from the island to Geraldton via a light aircraft. He felt worse during this air transfer. In Geraldton, a medical officer noted marked nystagmus to the right, vertigo, nausea and vomiting. Oxygen was administered and IV fluids, prochlorperazine, ondansetron, dexamethasone and salbutamol. A CT scan of his head was normal. He was transferred via the Royal Flying Doctors Service to Fremantle.

On arrival at the hyperbaric unit 30 hours after surfacing from his last dive, he was very tired and complained of vertigo especially when he attempted to sit up. He was ataxic and had nystagmus with fast beat to the right. Muscle power and tone were normal, as were sensation and reflexes. There were no cerebellar signs detected. Tympanic membranes were normal. His audiogram prior to recompression showed high-frequency loss in his right ear worse than the left. Previous audiograms had demonstrated symmetrical high-frequency hearing loss bilaterally.

He was recompressed according to RN62. After treatment, he still had vertigo, especially when his head was turned to the side. He was able to stand, but walked with a wide-based gait. Pre-treatment on the third day, he was able to do a standard Romberg test, and still had nystagmus. He received a total of nine HBOT.

Two weeks later, an ENT surgeon confirmed his pre-existing hearing abnormality. A bilateral high tone loss consistent with noise-induced injury was observed. He was ataxic with a wide-based gait. Caloric testing was said to be difficult because of a latent nystagmus that was right beating, although this changed direction when he was gazing to the left. Unfortunately, due to his disability, he was declared 'unfit' to work at his place of employment. He was referred to a neurologist who referred him to attend physiotherapy for vestibular rehabilitation.

Despite advice not to, he resumed diving seven months later, and has dived to 45 msw in Bali on numerous occasions. He says initially he had difficulty telling which way was up or down, but he claims that he is now 'OK'.

DIAGNOSIS: Vestibular decompression sickness, inner ear barotrauma

COMMENT: Although caloric testing was unsuccessful in determining the origin of this man's nystagmus, the absence of other symptoms of DCS would ordinarily point to IEBt as the cause of his symptoms. He had no history of difficult equalisation, and tympanic membranes were normal. His symptoms improved with recompression, although he retained some disability, possibly due to the delay in treatment.

Case Three

A 38-year-old diver with 10 years' experience of over 5000 dives dived to 35 msw for 45 mins on hookah. He stopped at 9 msw for 5 mins on ascent with the intention of also breathing 100% oxygen on the surface.

About 15 mins after surfacing, he felt unsteady and had difficulty standing. He went to lie down and noted he had low back pain, headache, a numb right forearm and hand, and a tingling sensation in his feet and calves. He was given oxygen to breathe from a demand regulator, but he became nauseated when he opened his eyes and vomited. He was transferred to hospital by helicopter for treatment. In transit, he was given 100% oxygen, IV fluids and metoclopramide.

On examination, he was fully conscious, clear headed and well hydrated, and although he felt generally weak he was able to move his legs freely against gravity. However, he was unable to move his head without nausea and vomiting. He did not notice any hearing loss and had no tinnitus. Neurological examination showed that all tendon jerks were brisk but equal bilaterally. Muscle tone was normal but muscle power was observed to be reduced in all groups. Light touch, position and vibration sense were all normal. Tympanic membranes appeared normal, but he appeared to be slightly deaf clinically and had unsustained lateral nystagmus. A diagnosis of IEDCS was made.

Following treatment with an RN62 his symptoms were greatly improved, with some residual dizziness when he moved his head. He was still unsteady on his feet.

The following day he felt a lot better, but still had vertigo on sitting up or with rapid eye movement. He was able to walk with assistance and was again noted to have unsustained lateral nystagmus. An audiogram revealed bilateral high-frequency hearing loss and was identical to one performed for his last diving medical examination.

By the third day he was able to eat and to walk normally, although sudden head movement still caused dizziness. An opinion was sought from an ENT specialist, who agreed with the diagnosis of IEDCS. After a total of four HBOT his nystagmus had resolved, his reflexes were normal and SRT was 60 secs.

DIAGNOSIS: Neurological decompression sickness with vestibular involvement

COMMENT: The presence of other symptoms of DCS (paraesthesia, back pain, muscular weakness) and the absence of signs of barotrauma in a provocative dive point towards IEDCS as a cause for his vestibular symptoms.

Case Four

A 43-year-old man with 28 years' diving experience dived with hookah equipment as follows:
Dive 1: 13 msw for 2.5 hr, stopping at 3 msw for 5 mins, SI approximately 45 mins
Dive 2: 10 msw for 1.5 hr, stopping at 3 msw for 5 mins, SI 1 hr
Dive 3: 10 msw for 1 hr, stopping at 3 msw for 5 mins.

He did not use a depth gauge, and admitted that his dives could have been deeper than he estimated. He had no difficulty equalising his ears at any stage of his dives.

About an hour after the last dive, as he leaned over the side of the boat to scoop up crabs, he became dizzy, unable to balance, and noticed pain and deafness in the right ear. On reaching shore, he was unable to walk due to lack of balance. He felt very tired and nauseated, and vomited once. He did not seek treatment until 24 hours later, when he presented to the regional hospital with persistent nausea and vertigo.

On examination, he was found to be unsteady with a simple Romberg test of less than 10 secs. Weber test was stated to be positive on the left. The diagnosis of 'IEBt and DCI' was made, despite his tympanic membranes being normal. He was transferred to a hyperbaric facility, arriving 29 hours after his last dive.

He was recompressed according to RN62, following which his vertigo was largely resolved and his SRT was 30 secs. Tuning fork tests were normal. His audiogram the following day showed a hearing deficit of 35 dB at 4000, 6000 and 8000 Hz of the left ear similar to his previous audiogram, due to previous noise exposure in his occupation as a mechanical fitter. He was given a follow-up treatment with Royal Navy Table 61 (RN61). Following treatment, his SRT was 60 secs. There was no nystagmus, or vertigo even when moving his head. The next day, he noticed a mild frontal headache, and some dizziness, which cleared following another HBOT. On discharge, he still had mild dizziness on rapid head movement.

DIAGNOSIS: Vestibular and neurological decompression sickness, inner ear barotrauma

COMMENT: IEBt is less likely in this case due to the lack of history of difficult equalisation, and normal tympanic membranes and normal tuning fork tests. His symptoms are predominantly vestibular in origin, and IEDCS would be a likely diagnosis.

Case Five

A 40-year-old, qualified diver with several years' experience had recommenced diving after a five-year break. He had been unwell during the preceding week with gastroenteritis, which had resolved three days earlier. He dived on a surface air supply, with no depth gauge, as follows:
Dive 1: 15 msw for 40 mins approximately, SI 90 mins
Dive 2: 28 msw for 30 mins.
Although the ascent from the second dive was controlled, he did not perform any decompression stops. He had no difficulty with equalisation during the dives.

On reaching the surface, he noticed an immediate headache, began to cough, and had pain in his chest and lower abdomen. He swam back to the boat, and boarded it unaided. He then recalls saying "You blokes better look after me because I'm really crook", then felt his face begin to twitch

and lost consciousness. He remained unconscious for the entire trip back to shore (approximately 90 mins) and was left sitting up in the back of the boat by his dive buddies.

On arrival at the jetty they were met by the local doctor, who noted him to be grossly cyanosed and making little respiratory effort. Blood pressure was 70 mmHg systolic. He was placed supine and was administered oxygen and IV fluid. Within about 20 minutes, he became responsive and was opening his eyes to command. He was retrieved to the nearest hyperbaric facility by air ambulance, but was troubled by vomiting during the transfer. Metoclopramide 10mg IV was given with no effect.

On arrival, he was conscious and cooperative, but constantly vomiting and severely nauseated. Pulse 108 per min, BP 123/80 mmHg, chest clear, equal air entry. There was a mottled rash over the left lower chest and abdomen, which blanched with pressure. His mental state was assessed with difficulty due to his constant vomiting and severe nausea, but appeared normal. He had nystagmus on central gaze, worse on lateral gaze to the right. Tone, power, and reflexes were normal. Sensation was grossly normal, but difficult to assess due to limited cooperation as a result of constant vomiting. Examination of the fundi showed no evidence of papilloedema. A Romberg test was not performed due to his inability to stand up. He had no joint pains. An audiogram showed no change from a previously recorded test at his workplace.

He was commenced on IV lignocaine infusion and recompressed according to RN62. His nausea ceased at 18 m but recurred after arrival at 9 m, and remained to the end of the treatment. He was commenced on prochlorperazine 10 mg orally as required for nausea. The lignocaine infusion was continued for 48 hours. He underwent a further thirteen daily HBOT. His nausea and vertigo gradually improved, and he was able to walk with assistance by the fifth day. By the seventh day his SRT was greater than 60 secs. On the ninth day, he reported altered sensation to temperature in his legs. Review by a neurologist showed a reduction in temperature sensation below dermatome level T11 and reduced pain sensation below T10. There was a gradual improvement in his sensory loss over the following weeks.

DIAGNOSIS: Neurological decompression sickness with vestibular involvement, cerebral arterial gas embolism (CAGE).

COMMENT: The hyper-acute onset of symptoms may suggest CAGE, but the dive was extremely provocative for bubble formation. Acute DCS is also likely, especially given the diverse nature of symptoms that evolved over subsequent days. The predominant symptoms at presentation were vertigo and nausea, and IEBt could have been a possible consideration. Given the history of unconsciousness there was never any doubt about the need for recompression therapy.

Case Six

A 21-year-old male with no formal diving training was diving with Case Five on hookah surface air supply, with no dive plan, depth gauge or dive computer as follows:
Dive 1: 15 msw for 40 mins, SI 40 mins
Dive 2: 28 msw for 30 mins followed by a controlled ascent to the surface, without a 'safety' or decompression stop.

His dive buddy lost consciousness on surfacing and was evacuated for urgent recompression. Despite this, and despite being personally contacted and advised to seek medical attention if he developed any unusual symptoms, he did not seek medical attention until six days later.

At presentation he complained of constant vertigo and nausea, worse in the mornings, and generalised aches and pains worst in his neck, shoulders and hips. The symptoms had developed within six hours of his dive, and had been sufficiently severe that he had been unable to attend work that week. Examination was normal except for some coarse nystagmus on far lateral gaze bilaterally, and an impaired SRT of 5 secs. There were no signs of cerebellar dysfunction. An audiogram was normal, and ENT opinion suggested IEDCS was the most likely cause of his nausea and vertigo.

He was recompressed according to RN62, and had a further five hyperbaric oxygen treatments. All symptoms completely resolved with this course of treatment.

DIAGNOSIS: Neurological decompression sickness with vestibular involvement

COMMENT: This diver had also undertaken an extremely provocative dive, and subsequently developed DCS. Vestibular symptoms were prominent, and were the reason for presentation. The complete response to recompression should be noted, especially considering the long delay to treatment.

Case Seven

This 33-year-old man with 18 months' diving experience had been diving for abalone as follows:
Dive 1: 15 msw for 120 mins, SI 15 mins
Dive 2: 10 msw for 90 mins, SI 30 mins
Dive 3: 6 msw for 120 mins.

The following day he dived to 16 m for 90 mins, with no decompression stops. About 20 minutes after surfacing, he developed dizziness, unsteadiness, vomiting, and abdominal pain. He waited until his dive buddy returned to the boat about 30 mins later, and they returned to the marina, arriving about two hours later. He was transferred by ambulance to the nearest hyperbaric unit.

On arrival, he had marked horizontal and rotatory nystagmus, worse on gaze to the right. Cranial nerve examination was otherwise normal. Muscle tone, power

and reflexes were normal, as was respiratory, cardiovascular and abdominal examination. Gait and SRT were not assessed due to severe nausea and vomiting on movement. Audiogram was unchanged from a pre-diving test.

He was treated with IV fluids and lignocaine infusion. A Doppler assessment detected high-grade intravascular bubbles (Grade 4) five hours after ascent from depth. He was recompressed on RN62, and all symptoms initially resolved at 18 m (280 kPa). Following ascent to 9 m (190 kPa), there was a gradual return of symptoms, and following treatment he reported a return of his dizziness and nausea. He was commenced on prochlorperazine and had eight further daily HBOT. His nausea and vertigo were severe for three days, necessitating inpatient treatment, then gradually resolved, and he was well at discharge.

DIAGNOSIS: Vestibular decompression sickness

COMMENT: The normal audiogram, absence of barotrauma, lack of neurological abnormalities, and the presence of severe vestibular symptoms all point to IEDCS.

Case Eight

A 25-year-old, recreational diver dived as follows:
Dive 1: 12.3 msw for 60 mins, stopping at 5 msw for 5 mins during ascent, SI 90 mins
Dive 2: 18 msw for 62 mins. During this dive, he did multiple ascents and descents between 6 msw and 18 msw. At the end of the dive, he did another safety stop, a '5 at 5'.

He had no difficulty equalising his ears during or after the dive. After his dive, he went to the gym and did a 'work-out' for about two hours and experienced some dizziness. At work the next day, he experienced true vertigo and also noticed some visual blurring and clumsiness of his hands. He also noticed that he was staggering a bit and had a 'twinge' of pain in his left shoulder, which did not persist.

He went to see his local medical practitioner, who found him to have marked diplopia on left lateral gaze, but no nystagmus. He had mild staccato dysarthria and moderately severe Rombergism with a tendency to fall to the right and backwards. He had a stamping, wide-based gait, dysdiadokinesis worse on the left, past pointing with intention tremor, and a positive heel-shin test. Muscle power and tone were normal, and there was no change in sensation or proprioception. Hearing was normal. He was recompressed on RN62 with complete recovery.

DIAGNOSIS: Cerebellar decompression sickness

COMMENT: This diver suffered one episode of vertigo, but had no nausea or nystagmus. His symptoms were predominantly cerebellar in nature, easily detected at clinical examination.

Discussion

The aetiology of inner ear DCS is not well understood. Doolette and Mitchell have proposed a three compartment model of the inner ear, comprising membranous labyrinth (vascular compartment), perilymph and endolymph.[3] In circumstances where the tissues become saturated with inert gas, such as prolonged air diving, decompressing too rapidly will exceed the ability of the labyrinthine vessels to remove inert gas as it diffuses from the two fluid compartments of the inner ear, leaving them susceptible to bubble formation. The unique fluid make-up of the inner ear therefore makes it a prime site for DCS under provocative circumstances.

IEDCS is an uncommon occurrence in air diving, but this should not lead to the assumption that IEDCS occurs only in heliox or hydrox diving and that, therefore, most inner ear problems in air diving are due to IEBt. The differential diagnosis between IEBT and IEDCS may be difficult.[4]

Farmer[5] and Nachum et al[2] suggested some distinguishing features that might be of assistance in making a diagnosis:

- In IEBt, a diver will normally report difficulty in clearing his ears at some stage.
- Symptoms of IEBt appear during the dive, whereas those of IEDCS appear after the dive.
- Other symptoms of DCS may accompany IEDCS.
- Signs of middle ear Bt may accompany IEBt but do not necessarily occur with IEDCS.
- Hyperbaric therapy will generally improve the symptoms of IEDCS, but may aggravate those of IEBt.

The distinction is not straightforward. Edmonds indicated that the delay of onset of symptoms is not necessarily a diagnostic feature in DCS, as it is not uncommon to find this in cases with perilymph fistulae.[6] Vertigo may also occur without hearing loss in both diseases. Also DCS may cause cerebellar manifestations (staggers) as well as vestibular damage, as illustrated in Case Eight.

When doubt exists, Edmonds recommends electronystagmography and iced-water caloric tests in the recompression chamber. Other useful tests include dynamic posturography, vestibulospinal response reactions to stress, and electrocochleography. Temporal bone polytomography and CT might be of value.[7] Molvaer, however, indicated that there is no conclusive test to tell whether or not a perilymph fistula is present.[8] If in doubt, recompression may be diagnostic.

Usually these cases present to the diving physician outside normal working hours, when complex neurophysiological diagnostic testing is not available. In some centres, there is a significant waiting period before neurophysiological testing can be performed, and in most cases it is not in the best interests of the diver to wait for confirmatory testing prior to recompression. Clinicians therefore have to rely on details gleaned from the history of the dive, and from

physical signs elicited on examination to make their diagnosis. As in all types of DCS, conservative treatment with bed rest, IV fluids and oxygen may result in some improvement without recompression, but the chance of long-term sequelae is greatly increased.

Classical teaching has told us not to recompress divers who may be suffering from inner ear barotrauma, but in some cases recompression may produce improvement in symptoms. Molvaer hypothesised that in IEBt, especially if there is dysfunction of the Eustachian tube, some gas from the middle ear might be forced through the perilymph fistula and enter the inner ear, either into scala tympani or scala vestibuli, depending on which window is damaged.[8] During further ascent, the inner-ear gas will expand and may damage cochlear or vestibular structures. If this hypothesis is correct, recompression may be therapeutic.

In most cases of IEDCS, divers have violated recommended dive tables. In the series reported by Nachum et al, 79% (23 cases) violated the tables.[2] In the same series, the onset of symptoms varied from immediate onset to five hours following the dive. Interestingly, one diver had five episodes of IEDCS. Ten cases (34%) had pure vestibular symptoms and all but one with vestibular symptoms had nystagmus. Four cases (14%) had cochlear symptoms alone and 15 cases (52%) had a combination of symptoms. Fifteen (52%) had isolated IEDCS, whereas 14 had additional symptoms of DCS. Thus, absence of other symptoms of DCS does not confirm that the diagnosis is IEBt. Of the 17 patients treated within six hours of symptom appearance, nine (53%) were cured. Of the 25 cases with vestibular injury and the 19 with cochlear damage, only seven (28%) and six (32%) respectively made a full recovery. Prompt treatment is therefore prudent to increase the chance of recovery, and delays in recompression whilst awaiting neurophysiological testing may not be warranted.

A recent study by Cantais et al found that a major degree of right-to-left shunt, as detected by transcranial Doppler, was associated with an increased incidence of cerebral and IEDCS, suggesting paradoxical venous gas embolism as a possible aetiology.[9] This may explain the occurrence of IEDCS in divers with minimally provocative dive profiles, and raises the question of whether we should seek a right-to-left shunt in divers presenting with DCS.

This series helps to illustrate the diagnostic dilemma faced by physicians when confronted with a diver with acute vertigo. The main decision, to recompress or not to recompress, must be carefully considered on the weight of history and symptoms and physical signs at presentation.

Conclusions

Although IEDCS is uncommon in air diving, it must be considered as a differential diagnosis for vertigo, as failure to treat IEDCS with recompression may result in permanent disability for the diver. It is sometimes difficult to distinguish between IEDCS and IEBt, but a careful history and clinical examination will often allow a distinction to be made between these two clinical entities.

Acknowledgments

The authors gratefully acknowledge Dr David Griffiths, Director of Hyperbaric Medicine,Townsville Hospital for providing Case Three, and Dr Evan Jenkins who referred Case Eight. Dr Carl Edmonds provided valuable advice.

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Inner ear barotrauma: a retrospective clinical series of 50 cases

Carl Edmonds

Key words
Inner ear barotrauma, diving, ear barotrauma, treatment

Abstract

(Edmonds C. Inner ear barotrauma: a retrospective clinical series of 50 cases. *SPUMS J.* 2004; 34: 11-4.)
A retrospective series of 50 cases of inner ear barotrauma is reviewed, as regards the diving and otological history, the clinical manifestations, basic audiometric investigations and treatments. This disorder may be predicted to some degree by a previous history of otological barotraumas, otological pathology and the use of inappropriate middle ear auto-inflation and diving techniques. The absence of tympanic membrane haemorrhage does not exclude the diagnosis, nor does a delay between the dive and the developing symptoms. The dive profile, symptomatology and investigations assist in verifying the site of the lesion and the likely damage, as well as differentiating inner ear barotrauma from various decompression sickness manifestations, including inner ear, cerebellar and cerebral. Treatment procedures and indications are reviewed.

Introduction

It is now 30 years since the term inner ear barotrauma (IEBt) was introduced and the first series described.[1,2] In the same papers, the pathology of round-window fistula (RWF) was verified surgically, and other pathologies considered.

The main reason for the initial series being accepted, against the prevailing belief that barotrauma did not cause permanent hearing loss, was the presence of pre- and post-incident pure tone audiometry (PTA).[3] This investigation was obligatory in the compulsory diving medical examination in Australia.

Similarly, the first group of cases with vestibular pathology from IEBt was presented and verified by the application of the electronystagmogram (ENG).[4] This not only allowed for the localisation of the lesion to a peripheral site in the eighth cranial nerve, as opposed to cerebral and cerebellar disease, but also indicated the degree of pathology.

This present, much larger, series was extracted in order to review and reassess the clinical observations and basic investigatory results with IEBt. This allows for a comparison with the major dysbaric differential diagnosis, inner ear decompression sickness (IEDCS).

Methodology

Fifty cases were analysed. The first 25 consecutive cases were extracted from the 1970s, and the last 25 consecutive cases from the 1990s. To ensure there was no likelihood of diagnostic confusion, the cases had to meet the following criteria:

- have occured in association with air diving (free or scuba) and result in enduring inner ear pathology, verified by PTA and/or ENG;
- have been exposed to single, shallow dives (usually less than 10 m) or with depth/duration profiles of less than half the US Navy no-decompression limits;
- and/or have a demonstrated RWF at surgery.

All were referred for assessment because of the injury to the Diving Medical Centre and/or the School of Underwater Medicine of the Royal Australian Navy. This was, therefore, a selected series.

Each case was documented using a pre-designed 'Ear Barotrauma Protocol' with a checklist for specific features of the history, clinical examination and investigations. Many were sent for further otological consultation by the same clinicians responsible for the original seminal papers on this subject.

To be designated as having a hearing loss, divers had to experience a decrement of at least a 20 dB in at least two frequencies, compared with their previous audiogram. Most losses observed were far in excess of this.

Treatment of sensorineural hearing loss was designated as successful if the audiogram returned to within 15 dB of the pre-incident audiogram. Partial success represented improvement equal to or greater than 20 dB, compared with the post-incident audiograms.

During the time period covering these diving cases, there were two other cases of IEBt from aviation exposure, which were not included. Another case, which did actually comply with the criteria, was excluded when the predominately conductive deafness was diagnosed as otosclerosis and successfully treated with surgery. This patient may also have experienced some IEBt.

Results

DEMOGRAPHY

These cases represented just under 1% of the diving problems encountered at the Diving Medical Centre. If one included all divers presenting with possible IEBt, but not meeting the above strict criteria, the prevalence was well over 1%.

Twenty six (52%) of the divers in this series were moderately experienced, having performed 6-50 dives. Thirteen (26%) were inexperienced or under training (1-5 dives) and 11 (22%) were very experienced (>50 dives). Although all were scuba divers, the incident causing injury occurred whilst free diving in five (10%). The female to male ratio was 3:7.

Presentation to a physician usually occurred in the first week (43 cases, 86%), with the remainder over the next two to three weeks. Medications prescribed included decongestants in four cases (8%) and aspirin in three (6%).

As regards hearing loss, the right side was affected in 20 (40%), the left side in 17 (34%), and three (6%) cases were bilateral.

PREVIOUS OTORHINOLOGICAL PATHOLOGY

Chronic otorhinological (ENT) pathology was present in 20 cases (40%), comprising allergic rhinitis, chronic sinusitis, previous surgery or other ear pathology, in order of decreasing frequency. This pathology was usually of a minor degree. There was a history of acute ENT pathology in 8% including infections (pharyngitis, otitis media, sinusitis). There was a past history of:

- recurring middle ear barotrauma (MEBt) of descent in 28 (56%);
- one episode of MEBt of descent in three (6%);
- MEBt of descent and ascent in three (6%);
- aviation MEBt in 12 (24%);
- IEBt in six (12%).

In performing middle ear equalisation (ME=) techniques, not one employed the recommended 'equalising ahead of the descent' technique, and only three (6%) used the feet first descent, as has been advocated.[5]

DIVE PROFILE

Although many initially claimed to have had no trouble with ME=, specific interrogation revealed that they had been obliged to undergo a slow descent (22 cases, 44%), or a 'yo-yo' (multiple interruptions of descents by short ascents) descent (22 cases, 44%) because of sluggish ME=. A few developed symptoms associated with uncontrolled rapid ascents or descents. Most of those who initially claimed no ME= problems, apart from the indicative dive profile, also had both symptoms and signs of MEBt.

CLINICAL FEATURES AND INVESTIGATIONS

In total, after more complete interrogation than simply asking if there was any problem with ME=, there was a clinical history consistent with MEBt of descent in 44 cases (88%) and five had MEBt of both descent and ascent.

Some symptoms were attributable to this associated MEBt, such as a blocking, fullness or crackling sensation in the ear (37 cases, 74%), persisting pain after the dive (11 cases, 22%) or slight epistaxis from the side of the MEBt (6 cases, 12%).

Grading of MEBt by otoscopy is traditional in diving medicine.[6] Physical signs of MEBt were not always evident by the time the subject was first examined by a diving physician, with 19 cases (38%) being Teed Grade 0. Grade 1 was evident in two (4%), Grade 2 in eight (16%), Grade 3 in 10 (20%) and Grade 4 in three (6%). The remaining eight showed some degree of haemorrhage, which was not graded at the time.

Many of those with a history of MEBt had a flattened tympanometry and sometimes a mild conductive deafness.

Inner ear symptoms attributable to cochlear damage included hearing loss (40 cases, 80%), tinnitus (43, 86%) and dysacusis (10%). The hearing loss was always confirmed as sensorineural, either high frequency at 4 kHz, 6 kHz and 8 kHz, or virtually total, involving the lower frequencies also. Four divers (8%) did not recognise their hearing loss, despite a definite high-frequency sensorineural deafness.

Vestibular symptoms occurred in 16 cases (38%) and always included vertigo. Sometimes these symptoms were mild, lasting only a day or so, but frequently they were severe, with nausea, vomiting, prostration and ataxia. Lying horizontal or sudden head movements caused aggravation in some. In four cases (8%) there was ENG evidence of pathology, despite there being no symptomatology attributed to the vestibular system.

Inner ear symptoms were noted either during the dive or immediately on ascent in most cases (31, 62%), although in 16 (32%) they developed over the next few hours and in three (6%) they developed during the night following the dive. Sometimes the vestibular symptoms were noted after the cochlear, usually when the diver tried to board the boat. In four cases (8%) the symptoms followed exertion (lifting the anchor or scuba tanks) or other causes of raised intracranial pressure (Valsalva, sneezing).

TREATMENT

Conservative treatment was administered in 33 cases (66%) with partial success evident in 21 (42%) and cure in three (6%). With conservative management, symptoms other than hearing loss were too variable to be accurately quantified.

Tinnitus tended to improve over hours, days, weeks or months, with or without treatment. Vestibular symptoms consistently improved in all cases over weeks or months. Often the only evidence of persisting vestibular pathology was from provocative ENGs.

Surgery was performed in 13 divers (26%), with nine (18%) showing improvement in hearing, two (4%) being cured and one worsened, at the time of discharge. Much more impressive was the often dramatic improvement in tinnitus and vestibular symptomatology, from the time of the operation. The longest time interval between incident and surgery was 10 weeks; surgery was performed because of the diagnosis of persistent RWF, and had excellent, immediate results.

Recompression therapy had been inexplicably undertaken in three cases, but did no evident damage, prior to more appropriate referral for surgical treatment. Drug therapy had previously been used in three cases (aspirin, nicotinic acid), without effect. Decongestants and anxiolytics were sometimes employed during symptomatic and conservative treatment. Because of the time delay, the remainder received no therapy.

Discussion

Clinical descriptions of IEBt are not available in the diving medical literature before 1972, as the disorder was not recognised until then. Thus much of the literature quoted before that time did not discriminate between decompression sickness (DCS), IEBt and non-diving aetiologies such as noise, gunfire and explosions. Thus, using this literature to support any position on the topic of inner ear problems from diving is somewhat misleading.

Since then there have been case reports and clinical series of diving otological disorders reported and reviewed, but still with some confusion regarding the manifestations of MEBt, IEBt, cerebral DCS and/or IEDCS.[6-12] The lack of specificity of diagnoses sometimes causes difficulty in extrapolating the clinical features, and differential diagnoses were based on assumptions that have not been tested.

For this reason, 50 cases of evident IEBt, uncomplicated by a decompression sickness likelihood, were extracted from the files of the Diving Medical Centre, and retrospectively assessed. Differentiation from MEBt was verified by PTA and ENG.

The strong association with MEBt was verified. Also, the frequent past history of MEBt, descent, ascent and/or aviation induced, was observed. Two aspects of MEBt should not be used to exclude the diagnosis of IEBt. The initial claim of 'no ME= problem', either in clinical notes or from the diver directly, needs to be carefully explored. It is often a euphemism used by divers, who are renowned for the use of denial as a psychological mechanism, to indicate that ME= problems were overcome successfully and did not require aborting the dive.
Unless the examiner has also specifically questioned the diver about the reasons for:

- slow descent;
- employing 'yo/yo' techniques;
- 'fullness' sensations in the ear following a dive;
- epistaxis,

it is likely that MEBt will be missed. If the diver also employs less effective ME= techniques, such as head-first descent, or waiting until middle ear pressure is felt before employing ME=, the likelihood of MEBt becomes greater.

The absence of tympanic membrane haemorrhages does not exclude MEBt and/or IEBt. It could be explained as follows:

- Haemorrhage into the tympanic membrane is not inevitable in MEBt if mucosal effusion in the middle ear or a perilymph leak dominates the pathology and contributes to ME=. This is recognised by the inclusion of Grade 0 (symptoms without membrane tympanic signs) in the Teed classification of MEBt, and haemorrhage would be less expected in very slow descents.
- MEBt manifestations often resolve within days, before otoscopy is performed by the physician.
- The most widely accepted pathophysiology of IEBt is the explosive effect of the raised intracranial pressure associated with the Valsalva manoeuvre.[11,13,14] It is the latter that is likely to be directly related to IEBt, not the MEBt per se.
- Most general otological cases of RWF have nothing to do with dysbaric barotrauma, but are related to sudden raised intracranial pressures in non-divers.

Thus, the failure to observe tympanic membrane pathology should not be used to exclude RWF.

Although the development of symptoms during the dive may be indicative of a diagnosis of IEBt, a delay between the dive and the appearance of symptoms does not exclude this diagnosis. Indeed, their development associated with après-dive activities may well be suggestive of membrane rupture as opposed to other pathologies of IEBt, such as haemorrhage.

Symptomatology of IEBt is easily confused with MEBt (which frequently co-exists), IEDCS and neurological (cerebral and cerebellar) DCS, if appropriate investigations are not performed. Sensorineural deafness, partial or total, is usually associated with tinnitus and occasionally with dysacusis. PTA, up to 8 kHz, is essential for accurate assessment, and a pre-incident PTA is immensely valuable.

ENG clarifies the existence of vestibular pathology, and its severity, as well as differentiating peripheral from central (cerebellar) lesions. It brings objectivity to the symptoms of dizziness and vertigo, and the assessment of nystagmus. For more detail on the use, versatility and value of ENG, conventional diving medical texts should be accessed.[6]

Although vomiting and ataxia are dramatic, they indicate only the acuteness and severity of the vertigo, not necessarily its origin.

Initially the tendency was to treat all cases with surgery, but it soon became evident that conservative treatment was often very effective. This included bed rest with head elevated, avoidance of all intracranial pressure increases (Valsalva manoeuvres, sneezing, nose blowing, straining at defaecation, lifting and other exertion). Repeated PTA is used to monitor progress.

Massive hearing loss or deteriorating hearing despite conservative treatment, are indications for middle ear exploration with plugging of labyrinthine windows even if the fistula cannot be visualised. Successful surgery often has immediate value in relieving symptoms. The 1970s contributed nine (18%) of the surgical cases, with the 1990s producing four (8%). Although verifying the observed trend, this is not statistically significant.

Our experience confirms that permanent inner ear damage predisposes to further damage if the provoking activity (diving) were to continue.[11] The high incidence of both MEBt and IEBt in the past medical history supports this belief. We thus advise IEBt patients with evidence of inner ear damage that not only should they discontinue scuba diving, but they should also avoid any hyperbaric exposure, such as with free diving. They are also advised regarding ideal ME= with aviation exposure.

Conclusions

1 IEBt may be predicted to some degree by a previous history of MEBt or previous IEBt, and is more likely in divers with ENT pathology and who use inappropriate ME= diving techniques.

2 The history of ME= needs to be carefully assessed, to be compared with the dive profile, and the absence of tympanic membrane hemorrhaging does not exclude the diagnosis.

3 The development of symptoms during the dive may be indicative of IEBt diagnosis, but a delay between the dive and the appearance of symptoms does not exclude this diagnosis.

4 The cause of the pathology (IEBt, IEDCS, neurological DCS) can be indicated by the dive profile and the presence of other symptoms of MEBt, IEBt or DCS. PTA and ENG are sometimes necessary to complement the clinical findings and verify the site and extent of the lesion.

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Australian standards committees

Dr David Smart was the successful applicant to replace Dr John Knight as the SPUMS representative on the Standards Australia SF-017 Occupational Diving Committee.

Dr Cathy Meehan is the current SPUMS representative on the Standards Australia CS-83 Recreational Underwater Diving Committee.

Articles reprinted from other journals

A biophysical basis for inner ear decompression sickness

David J Doolette and Simon J Mitchell

Key words
Decompression sickness, labyrinth, nitrogen pharmacokinetics, helium pharmacokinetics, diving

Abstract

(Doolette DJ, Mitchell SJ. A biophysical basis for inner ear decompression sickness. *J Appl Physiol.* 2003; 94: 2145-50.) Isolated inner ear decompression sickness (DCS) is recognised in deep diving involving breathing of helium-oxygen mixtures, particularly where breathing gas is switched to a nitrogen-rich mixture during decompression. The biophysical basis for this selective vulnerability of the inner ear to DCS has not been established. A compartmental model of inert gas kinetics in the human inner ear was constructed from anatomical and physiological parameters described in the literature and used to simulate inert gas tensions in the inner ear during deep dives and breathing gas substitutions that have been reported to cause inner ear DCS. The model predicts considerable supersaturation, and therefore possible bubble formation, during the initial phase of a conventional decompression. Counter-diffusion of helium and nitrogen from the perilymph may produce supersaturation in the membranous labyrinth and endolymph after switching to a nitrogen-rich breathing mixture even without decompression. Conventional decompression algorithms may result in inadequate decompression for the inner ear for deep dives. Breathing gas switches should be scheduled deep or shallow to avoid the period of maximum supersaturation resulting from decompression.

Introduction

Injury to the vestibulo-cochlear apparatus ("inner ear") during compressed gas diving may be caused by barotrauma, acoustic trauma, or by decompression sickness (DCS). Retrospective reviews of DCS cases in both military[9] and recreational divers[16] suggest that 26% of patients suffering "serious" or "neurological" DCS exhibit evidence of vestibulo-cochlear involvement. These presentations are significant since residual deficits in balance and in hearing are common despite recompression treatment, and the window of opportunity for optimal treatment appears relatively short.[3]

The putative cause of inner ear decompression sickness (IEDCS) is bubble formation initiated when the tension (concentration/solubility) of dissolved gas exceeds ambient pressure within the inner ear during ascent. However, beyond this presumed involvement of bubbles there is uncertainty regarding their precise location and effects, or the circumstances under which they are likely to form. Early descriptions of IEDCS following shallow dives often treated ear problems as being of secondary importance to the other central nervous system manifestations of DCS that were almost invariably present. However, the development of deeper diving techniques involving breathing of helium-oxygen gas mixtures has been associated with the occurrence of "pure" or "isolated" IEDCS,[7] especially where switches to air or other nitrogen-rich breathing gas mixtures are made to accelerate decompression.[3] Indeed, isobaric switches of inspired gas, made while the diver is held at a constant ambient pressure, have precipitated symptoms of IEDCS in the absence of any decompression.[10]

This selective vulnerability of the inner ear to DCS under these conditions has been attributed to substantial transfer of highly diffusible helium into the inner ear from the middle ear gas space by diffusion across the round window membrane.[7,10] Gas uptake from the middle ear, in addition to that absorbed from the blood, may predispose the inner ear to bubble formation during decompression. Bubble formation might be enhanced or even initiated by elevated inner ear gas tension resulting from "counter-diffusion" of different gas species of different diffusivity from the blood and middle ear.

We are not aware of any attempts to more formally define the biophysical mechanisms of IEDCS, yet this is an issue of increasing contemporary relevance. Whereas many of the occupational diving tasks that necessitated deep mixed gas diving have now been devolved to remote operated vehicles, there is a new cohort of so-called "technical" recreational divers who are indulging in deep mixed gas "bounce" (short duration, non-saturation) dives in growing numbers. Not surprisingly, reports of isolated IEDCS are starting to emerge from this group.

We present here an illustrative case of isolated IEDCS occurring in a recreational technical diver undertaking a mixed gas dive. We then present a physiological model describing gas kinetics in the inner ear that demonstrates a basis for selective vulnerability of the inner ear to DCS in

mixed gas diving. Finally, on the basis of this model, we outline some precautions that may be taken in deep mixed gas bounce diving in order to avoid precipitating IEDCS.

Case report

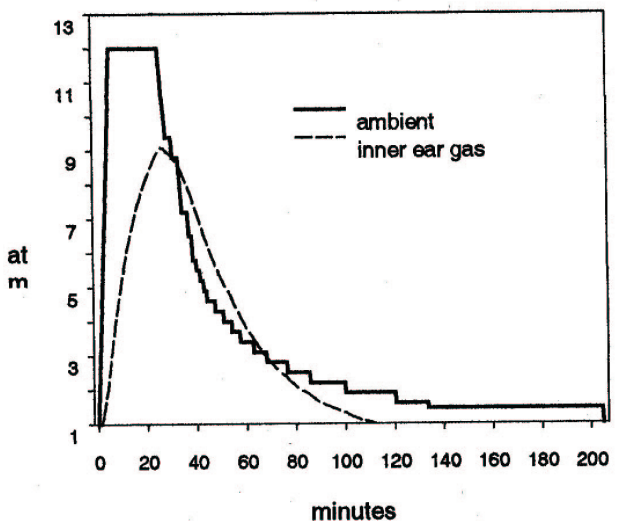
A 33 year old male recreational technical diver who had previously made more than 1000 uneventful dives undertook a wreck dive to 110 msw (1201 kPa), 11.9 atmospheres absolute (atm abs), for 25 minutes using a Buddy Inspiration closed circuit rebreathing SCUBA (A.P. Valves, Helston, Cornwall, U.K.). The diluent gas for the descent, bottom time and the first part of the ascent was trimix comprising 8% oxygen / 60% helium / 32% nitrogen. The diluent was changed to air at 30 msw during the ascent, and the breathing loop was flushed with 100% oxygen at the 4.5 msw stop. The PO_2 set point was 1.3 atm throughout the dive except at the 4.5 msw stop. The dive plan is shown in Figure 1, and the diver did not deviate from this plan. The decompression was controlled using a VR3 diver carried computer (Delta P Technology, Poole, Dorset, U.K.) in closed circuit mixed gas mode. These devices operate on the Proplanner decompression algorithm software (Delta P Technology, Poole, Dorset, U.K.).

The descent, bottom time and the first part of the ascent were uncomplicated. In particular, there were no problems with middle ear equalisation. There were no ascent rate violations and no decompression stops were omitted. Shortly after arrival at the 9 msw stop the diver began to experience rotational vertigo. This occurred in short bursts at first, but soon progressed to become persistent. The PO_2 in the rebreather loop at the time was 1.3 atm according to all 3 oxygen sensors. Breathing 50% oxygen / 50% nitrogen using an open circuit SCUBA did not alleviate the problem, and the decompression was continued on the rebreather. The diver became profoundly nauseated and could not keep his eyes open because of the vertigo. He had to alternate between open circuit SCUBA and the rebreather in order to vomit. Despite these debilitating symptoms, he was able to complete the planned decompression stops.

The diver was retrieved onto the boat, given 100% oxygen by demand valve regulator, and evacuated to the nearest recompression chamber where he arrived approximately 4 hours after the onset of symptoms. At this time his vertigo had settled substantially but he remained nauseated. It was still possible to elicit nystagmus on positional testing and he was incapable of performing the Sharpened Romberg Test. An air conduction audiogram revealed normal and symmetrical hearing. There were no other symptoms or signs of DCS or middle ear barotrauma. This appeared to be a case of isolated inner ear (vestibular) DCS.

The diver was recompressed to 4 atm abs and treated according to the protocol specified by the Royal New Zealand Navy Table 1A, which is a modification of the US Navy Air Treatment Table 1A (22) whereby the patient breathes 50% oxygen / 50% helium until decompressed to 2.8 atm abs and breathes 100% oxygen thereafter. This elicited a substantial improvement, but there was persistent mild dysequilibrium for several days after. The diver received 3 once daily follow up treatments consisting of 100% oxygen breathing for 60 minutes at 2.8 atm abs and throughout a 30 minute decompression to 1 atm abs. He has recovered completely (including normalisation of his Sharpened Romberg Test) and has made an uneventful return to diving.

Figure 1. Pressure-time schedule for the case report mixed gas rebreather technical dive (ambient pressure in atm abs, solid line) that resulted in isolated IEDCS. The predicted inner ear vascular compartment (membranous labyrinth) dissolved gas tension (atm) is shown as the dashed line. Dissolved gas tension includes model predictions of nitrogen and helium tensions plus a fixed value (0.19 atm) representing metabolic gas tensions ($P_{tisO_2}+P_{tisCO_2}+P_{H_2O}$)



Methods

PHYSIOLOGICAL MODEL OF INNER EAR INERT GAS KINETICS

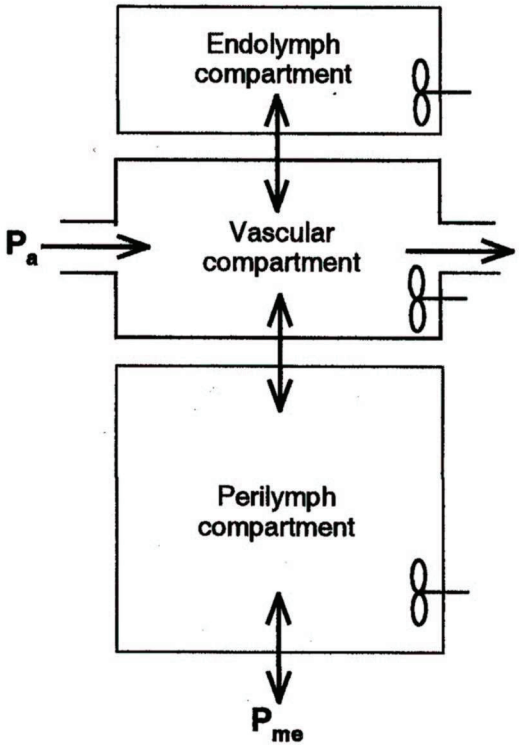
A physiological compartmental model of inert gas kinetics in the human inner ear (Figure 2) was constructed from parameters in the literature (Table 1). Three well-stirred compartments represented the membranous labyrinth, the perilymph, and the endolymph. Inner ear uptake and wash out of inert gas occurred via perfusion of the membranous labyrinth (vascular compartment) which, unlike the endosteum, is densely vascular.[14] Additionally, inert gases diffused between the middle ear gas space and perilymph across the round window membrane, whereas the oval window membrane was considered occluded by the ossicles. Within the inner ear, inert gas diffused between the vascular compartment and each labyrinthine fluid compartment. Diffusion-limiting membranes of zero volume notionally

Table 1. Model parameters

Parameter	Description	Value	Reference
V_{end}	Endolymph compartment volume	0.0388 mL	(8)
V_{per}	Perilymph compartment volume	0.166 mL	(8)
V_{vas}	Vascular compartment volume	0.070 mL	*
Q	Inner ear blood flow	0.00036 mL x s^{-1}	*
A_{vas}	Vascular compartment surface area	3 cm^2	*
A_{rw}	Round window surface area	0.022 cm^2	(18)
h_{end}	Diffusion distance	0.025 cm	*
h_{per}	Diffusion distance	0.033 cm	*
h_{rw}	Diffusion distance	0.4 cm	*
S_{He}	Helium solubility	0.01 mL x mL^{-1} x atm^{-1}	(12)
S_{N_2}	Nitrogen solubility	0.015 mL x mL^{-1} x atm^{-1}	(12)
D_{He}	Helium diffusivity	3.94 x 10^{-5} cm^2 x s^{-1}	(12)
D_{N_2}	Nitrogen diffusivity	1.3 x 10^{-5} cm^2 x s^{-1}	(12)

* Derived values, see text.

Figure 2.
Physiological compartmental model of inner ear dissolved inert gas kinetics. Boxes with propellers indicated the well-stirred endolymph, vascular (membranous labyrinth), and perilymph compartments. Single headed arrows indicate the direction of flows of inert gases by perfusion with blood. Double headed arrows indicate the directions of flows of inert gas by diffusion. P_a (arterial blood inert gas tensions) and P_{me} (middle ear inert gas tensions) represent the model inputs



separated the compartments and the middle ear; the flows of inert gases across these membranes (Table 2) were formulated to reflect a slab approximation to the diffusion geometry (A/h, Table 1) of the labyrinthine fluid compartments. Diffusivities and solubilities of nitrogen and helium in blood, tissue, and labyrinthine fluids were characteristic of aqueous tissues. This compartmental approximation of diffusion does not consider transient inert gas tension gradients within the tissues represented by the compartments but provides an effective first order approximation to the volume averaged inert gas tensions.[20] Model input comprised arterial inert gas tensions and middle ear inert gas partial pressures, both being considered to equilibrate instantly with inspired inert gas partial pressures adjusted for water vapour pressure at 37°C.

Not all parameter values for the human inner ear model were available in the literature and some were estimated or scaled from animal data. Blood flow was scaled up from measurements made on guinea pig inner ear[17] which is one tenth the volume of the human inner ear.[8,21] Vascular compartment volume was derived from blood flow using values of tissue perfusion (mL.100 g^{-1}.min^{-1}) for rat cochlea and vestibular sensory organs.[13] Doubling or halving these two parameters resulted in only small quantitative but not qualitative differences in model simulations. The diffusion geometry (A/h, Table 1) of the labyrinthine fluid compartments was based on modelling the endolymph and perilymph volumes as being contained in concentric tubes of 0.025 cm (h_{end}) and 0.058 cm (h_{per}+h_{end}) radius respectively, separated by the membranous labyrinth tissue which has a surface area of A_{vas} on each face. The diffusion distance from the round window (h_{rw}) was based on the radius of a sphere of inner ear volume (V_{end} + V_{per} + V_{vas}).

The model was written as the following ordinary differential equations for each inert gas:

Table 2. Compartment time constants and flows (see Table 1)

	Time constant Formula	Value s	Flow Formula	Value mL x s^{-1} x atm^{-1}
Vascular-endolymph	h_{end}^2/D_{N_2}	48	D_{N_2} x S_{N_2} x A_{vas}/h_{end}	2.34 x 10^{-5}
	h_{end}^2/D_{He}	16	D_{He} x S_{He} x A_{vas}/h_{end}	4.73 x 10^{-5}
Vascular-perilymph	h_{per}^2/D_{N_2}	84	D_{N_2} x S_{N_2} x A_{vas}/h_{per}	1.77 x 10^{-5}
	h_{per}^2/D_{He}	28	D_{He} x S_{He} x A_{vas}/h_{per}	3.58 x 10^{-5}
Middle ear-perilymph	$(h_{rw}$ x $V_{ie})/(D_{N_2}$ x $A_{rw})$	3.83 x 10^5	D_{N_2} x S_{N_2} x A_{rw}/h_{rw}	1.07 x 10^{-8}
	$(h_{rw}$ x $V_{ie})/(D_{He}$ x $A_{rw})$	1.26 x 10^5	D_{He} x S_{He} x A_{rw}/h_{rw}	2.17 x 10^{-8}
Vascular perfusion	V_{vas}/Q	194	Q x S_{N_2}	5.40 x 10^{-6}
			Q x S_{He}	3.6 x 10^{-6}
Inner ear perfusion	V_{ie}/Q	761		

$$S \times V_{vas} \times \frac{dP_{vas}}{dt} = Q \times S \times (P_a - P_{vas}) - PSA_{per} \times (P_{vas} - P_{per}) - PSA_{end} \times (P_{vas} - P_{end})$$

$$S \times V_{per} \times \frac{dP_{per}}{dt} = PSA_{per} \times (P_{vas} - P_{per}) - PSA_{rw} \times (P_{per} - P_{me})$$

$$S \times V_{end} \times \frac{dP_{end}}{dt} = PSA_{end} \times (P_{vas} - P_{end})$$

Where P_{vas}, P_{per}, P_{end}, and P_a are the vascular, perilymph, endolymph, and arterial blood inert gas tensions, P_{me} is the middle ear inert gas partial pressure, PSA = D x S x A/h are the relevant flow values given in Table 2, and all other abbreviations are defined in Table 1. Simulations were performed using Scientist for Windows [computer program] (version 2.01. Salt Lake City (UT): Micromath Inc.; 1995).

VALIDATION OF THE MODEL

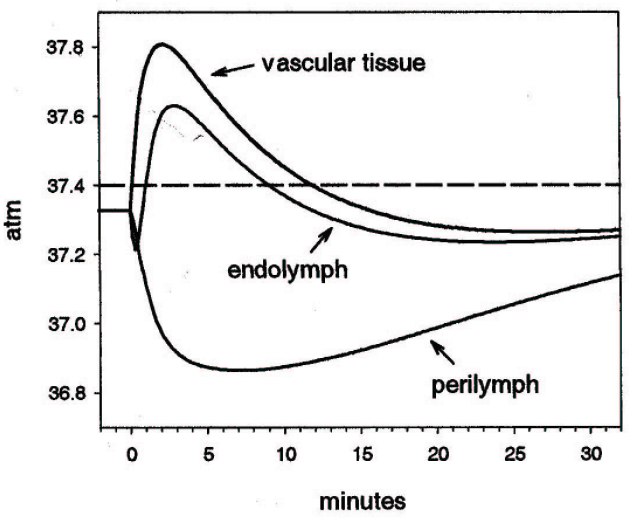
There are no measurements of inner ear inert gas kinetics to validate the model against; however, oxygen, which has similar diffusivity to nitrogen, has been measured in the labyrinthine fluids using polarographic electrodes. One such report gives the initial slope of the decline of oxygen tension in the guinea pig cochlea perilymph and endolymph during anoxia.[19] Assuming zero vascular compartment oxygen tension, these data can be used to calculate approximate time constants = initial tension/(-initial slope) for diffusion of oxygen from the guinea pig endolymph of 30 s and from the perilymph of 44 s; these are of the same order as the derived nitrogen time constants in the present human model.

Results

SIMULATIONS USING THE INNER EAR MODEL

Figure 3 shows a simulation of the gas tensions in the inner ear model compartments during an isobaric gas switch reported to result in DCS.[10] After a full day residence at 37.4 atm abs breathing the chamber atmosphere of 0.21 atm oxygen, balance helium, with no change in ambient pressure, the subject began breathing 0.21 atm oxygen, 10 atm nitrogen, balance helium through a mask, during which severe nausea, vomiting and vertigo developed. The breathing gas switch results in a transient elevation of vascular compartment and endolymph gas tensions above ambient pressure; such supersaturation is required for bubble formation. The peak vascular compartment gas tension was 37.8 atm (0.4 atm supersaturation) occurring at 2 minutes after the gas switch.

Figure 3. Model simulation of an isobaric breathing gas switch from helium-oxygen to helium-nitrogen-oxygen that resulted in IEDCS as described in results. Dissolved gas tensions (atm) in the three model compartments are shown as solid lines. The horizontal dashed line indicates the ambient pressure (atm abs)



In Figure 3 and other simulations of isobaric breathing gas switches after blood:tissue equilibrium, the peak change in inner ear vascular compartment gas tension from equilibrium was approximately 4.8% of the change in arterial nitrogen tension, expressed in terms of inspired gas for a switch from breathing gas 1 to 2 as $0.048 \times (P_{IN_2\text{-}2}-P_{IN_2\text{-}1}) \times (1-P_{H_2O}/P_{amb})$, where P_{amb} is ambient pressure and P_{H_2O} is saturated water vapour pressure at 37°C. At equilibrium the total tissue gas tension equals dry inspired inert gas partial pressure $(P_{amb}-P_{IO_2}) \times (1-P_{H_2O}/P_{amb})$ plus the tissue metabolic gas tensions $(P_{tisO_2}+P_{tisCO_2}+P_{H_2O})$. For an isobaric switch peak supersaturation (total tissue gas tension-P_{amb}) is:

$$(0.048 \times (P_{IN_2\text{-}2}-P_{IN_2\text{-}1})-P_{IO_2}) \times (1-P_{H_2O}/P_{amb})+P_{tisO_2}+P_{tisCO_2}$$

The peak supersaturation depends on the magnitude of the inert gas substitution and the inspired oxygen tension since tissue oxygen tension is relatively independent of inspired oxygen partial pressures used in diving.

The transient increase in gas tension following a substitution of nitrogen for helium in breathing gas results from the difference in gas transfer between compartments (Table 2). The flow of nitrogen into the vascular compartment via the arterial blood exceeds wash-out of helium in venous effluent. The transfer of helium into the vascular compartment by diffusion from the perilymph and endolymph exceeds the counter-diffusion of nitrogen in the opposite direction and temporarily exceeds the wash-out of helium in the blood.

Examination of the flows and time constants in Table 2 indicates that transfer of nitrogen and helium across the round window is negligible in comparison to the other transport processes. The time constant is related to the flow by V x S/flow and by definition 99% equilibration by this process alone occurs in 4.6 time constants. Whereas the round window is only 70 µm thick[5] and likely freely permeable to inert gases, owing to the large diffusion distances through the inner ear fluid spaces, transfer of gas via this route had little effect on the inner ear compartment gas tensions over the time course of the present simulations. The time constants for transfer of inert gases between the vascular compartment and the labyrinthine fluids are sufficiently large that considerable inert gas tension gradients across endolymph and perilymph may persist over the time course of the present simulations.

Although a multi-compartmental model, the gas kinetics of the entire inner ear for longer time periods can be approximated from the overall perfusion time constant, equivalent to a half time of 8.8 min (ln2 x TC). Similar kinetics are illustrated in Figure 1 which shows, in addition to the depth/time profile of the dive reported in the case history, the inner ear gas tensions simulated according to the present model. This simulation indicates considerable supersaturation during the early (deep) phase of decompression. For clarity at this scale, only the vascular compartment is illustrated. This half time is quite slow in comparison to a well-perfused tissue such as brain, which would have a half time of 1.7 min (assuming brain blood perfusion of 40 mL.100 g^{-1}.min^{-1}). The magnitude of counter-diffusion effect during the switch from trimix to air diluent at 4 atm abs (51 minutes in Figure 1) is modest because the partial pressure of nitrogen substituted for helium is small, and is manifest only as a transient slowing of gas wash-out, not evident at this scale in Figure 1. However the breathing gas switch from trimix to air diluent occurred while the inner ear vascular compartment was already supersaturated (1.09 atm) due to decompression and shortly following the time of maximum supersaturation (1.7 atm) at 44 minutes and 4.6 atm abs.

Discussion

The case described here typifies the type of technical recreational diving that is becoming increasingly widespread. It also illustrates the potential for isolated IEDCS to occur in a seemingly random fashion during an otherwise uneventful deep technical dive that has gone according to plan. As we will discuss below, this case and others like it suggest that some decompression algorithms for deep technical dives, particularly those incorporating switches to nitrogen-rich breathing gas, may need to be modified in order to avoid such events. It certainly seems possible that isolated IEDCS will become more common as technical diving grows in popularity.

IEDCS is a poorly defined clinical entity and is difficult to study epidemiologically. One of the principal difficulties is that several distinct pathological processes may produce the same symptoms and there is no gold standard test for distinguishing between them. In particular, there is frequent difficulty distinguishing between isolated IEDCS and inner ear barotrauma (barotraumas are tissue damage caused by compression or expansion of adjacent gas spaces). Clinicians must rely on interpretations of the circumstances of the dive as well as on the features of the illness itself in order to come to a diagnosis, but ambiguity is common. It is an important distinction since the DCS patient requires recompression whereas recompression is relatively contraindicated in inner ear barotrauma.[16] Distinguishing the source of vestibulo-cochlear symptoms in DCS patients with obvious widespread neurological involvement is also difficult, since it is possible that central rather than end-organ lesions may be involved. Clinically this distinction is less important since all DCS patients require recompression, but such cases complicate epidemiological analyses of IEDCS.

Notwithstanding these difficulties, both the case presented here and those in the series published by Farmer et al (1976) provide reasonable evidence that end organ IEDCS exists as an isolated pathological entity. None of these cases experienced any difficulty with middle ear equalisation or

exhibited any evidence of middle ear barotrauma; nor were there any other neurological symptoms suggestive of a central nervous system involvement. Moreover, since those discrete cerebral and cerebellar areas responsible for vestibulo-cochlear function exhibit no unique characteristics that would influence gas kinetics, it seems unlikely that symptomatic bubble formation would manifest only in those areas. Isolated inner ear damage is a more plausible explanation for these cases. Inner ear damage has been demonstrated by in vivo studies in guinea pigs[15] and squirrel monkeys[11] subjected to decompression.

Although isolated IEDCS has been described following relatively shallow air diving[16] it appears to be more commonly associated with deep helium-oxygen diving.[2,4] IEDCS onset has been described either early during the initial rapid phase of decompression from very deep dives (150 to 300 msw) while still breathing helium-oxygen, or following a switch to air breathing at shallower depths.[2,4]

IEDCS may be associated with deep diving because a considerable gas burden must be acquired to cause symptoms. The model proposed here suggests an inner ear half time (8.8 min) that allows considerable gas uptake during a deep dive and considerable supersaturation, and potentially bubble formation, in all inner ear compartments during the initial rapid phases of decompression. Many decompression algorithms, including the ZH-L16 [1] that forms the basis of many technical diving decompression algorithms including the Proplanner used in the present case report, control decompression according to the rule $P_{tis} < W \times P_{amb} + Z$ where P_{tis} is the total inert gas tension in a compartment and W and Z are constants. Although such rules can be empirically derived without biophysical assumptions, they have also been interpreted as describing a critical volume of released gas (bubbles) causing DCS.[6] In this case, by mass balance of the amount of inert gas in the compartment immediately prior to and following just critical bubble formation, $W = 1 + V_c/S$ and $Z = W \times (P_e + P_{st} - P_{tisO_2} - P_{tisCO_2} - P_{H_2O})$, where V_c is the critical volume of gas, S is the solubility of inert gas in the tissue, P_e and P_{st} are pressures opposing bubble formation due to tissue deformation and surface tension, respectively. Isolated IEDCS would selectively occur following deep dives and earlier than expected during decompression if the constant W were small and Z were large in comparison to the compartments responsible for other symptoms of DCS.[6] It seems plausible that V_c (and therefore W) might be small in the inner ear, which, as a sensitive transducer of mechanical energy, may be disrupted by small volumes of bubbles. Furthermore, P_e (and therefore Z) may be large since the inner ear is composed primarily of incompressible liquid within a non-distensible bony capsule with only small canal outlets.

The association of IEDCS with helium-oxygen diving in the absence of breathing gas switches may be coincidental since helium-oxygen breathing is standard practice for deep diving. However, IEDCS is clearly associated with switching breathing gas from helium to nitrogen rich mixtures,[4,10] and the present model suggests such gas substitution may cause bubble formation in the membranous labyrinth and endolymph. The model implicates transient supersaturation resulting from a counter-diffusion mechanism in which helium transfer from the perilymph to the other compartments temporarily exceeds the wash-out of helium in the venous blood. This transient mechanism is analogous to the steady-state "counter perfusion" mechanism proposed by Hills (1977). The anatomical requirements for this phenomenon are a large, diffusion-limited source of helium adjacent to a tissue with a relatively small blood supply and are provided by the perilymph surrounded by the smaller volume of vascular membranous labyrinth. Conversely, theoretical treatment of a more typical tissue arrangement such as muscle capillary unit suggest a transient under-saturation of tissue could result from switching breathing gas from helium to nitrogen rich mixtures.[23] It is on similar premise that such breathing gas switches are used to accelerate decompression.

The present model suggests diffusion of middle ear gas across the round window is negligible although previously proposed mechanisms implicated steady state counter-diffusion of atmospheric helium and blood nitrogen across the round window and tympanic membranes.[7,10] This previous proposal appears to have been made by analogy with the production of dermal lesions and continuous production of venous gas emboli by steady state counter-diffusion across the skin in humans or animals exposed to a helium atmosphere while breathing a different inert gas without decompression.[10]

The present model of dissolved inert gas kinetics is not a complete model of inner ear decompression; for instance it does not explain the 50-minute delay between predicted maximum supersaturation and onset of symptoms of IEDCS in the present case report. This delay may be related to the dynamics of bubble growth due to gas influx and expansion during decompression or biological processes induced by bubbles that are not considered in the present model. Nevertheless, the present physiological model can be used to deduce some qualitative implications for scheduling of technical deep diving decompression. First, the use of some decompression algorithms to plan very deep bounce dives (arbitrarily in excess of 100 msw) may result in inadequate decompression for the inner ear and may require, for instance, adjustment of W and Z constants for compartments with half times near 8.8 minutes as described. This would typically result in deeper decompression stops. Second, breathing gas switches from helium-rich to nitrogen-rich mixtures will produce an over-saturation in the inner ear that increases with depth and decreasing inspired oxygen partial pressure. Such breathing gas switches should be carefully scheduled either deep (with due consideration to nitrogen narcosis) or shallow to avoid the period of maximum supersaturation resulting from the decompression

(Figure 1). Switches should also be made whilst breathing the largest inspired oxygen partial pressure that can be safely tolerated with due consideration to oxygen toxicity.

Acknowledgments

This work was supported in part by a Jean B Reid Research Associateship, Health Science Research Committee, the University of Adelaide.

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Embolic inner ear decompression illness: correlation with a right-to-left shunt

Christoph Klingmann, Peter John Benton, Peter Arthur Ringleb, Michael Knauth

Abstract
(Klingmann C, Benton PJ, Ringleb PA, Knauth M. ***Laryngoscope***. 2003; 113: 1356-61.)
Objectives/Hypothesis: Inner ear decompression illness is thought to be a rare phenomenon in recreational divers, isolated signs and symptoms of inner ear dysfunction usually being attributed to inner ear barotrauma.
Study Design: We present 11 cases of inner ear dysfunction in nine divers with inner ear decompression illness.
Results: All nine divers had significant right-to-left shunt as diagnosed by transcranial Doppler sonography.
Conclusions: The authors thought that mechanism of causation in these cases may have been intravascular bubble emboli and that inner ear decompression illness may be more common among recreational divers than currently recognized. Failure to treat inner ear decompression illness with recompression therapy can result in permanent disability. Because the differential diagnosis between inner ear barotrauma and inner ear decompression illness can be impossible, the authors suggested that divers who present with inner ear symptoms following a dive should have recompression immediately after having undergone bilateral paracentesis.

Key words
Inner ear decompression illness, inner ear barotrauma, patent foramen ovale, right-to-left shunt, hyperbaric oxygen therapy

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Right-to-left shunt and risk of decompression illness with cochleovestibular and cerebral symptoms in divers: case control study in 101 consecutive dive accidents

Cantais E, Louge P, Suppini A, Foster P, Palmier B. *Crit Care Med*. 2003; 31: 84-8.

This group from the Naval Hospital in Toulon, France, investigated the role of right-to-left shunts using transcranial Doppler ultrasonography in a group of 101 consecutive divers referred to their hyperbaric centre with symptoms of decompression illness (DCI). They compared them with a control group of 101 healthy divers who had not suffered DCI. They defined the shunt as major if the number of high-intensity transient signals in the middle cerebral artery was more than 20.

Of the 101 divers presenting with DCI, a right-to-left shunt was detected in over half, whereas only 25 (a quarter) of the divers in the control group had a right-to-left shunt. This overall difference was not significant at the 5% level. However, when a shunt was detected, it was major in 12 of the 25 divers in the control group, but in 49 of the 59 divers with DCI ($p < 0.001$).

Looking at different sub-groups within the DCI group, they found that the proportion of major right-to-left shunts was 24 out of 34 divers who had cochleo-vestibular symptoms ($p < 0.0001$). In addition, 13 of 21 divers with cerebral DCI ($p < 0.0001$), ten of 31 with spinal DCI ($p < 0.01$) and two compressed air workers with caisson sickness had major right-to-left shunts.

Based on these results, they concluded that major right-to-left shunts were associated with an increased incidence of cochleo-vestibular and cerebral DCI, suggesting paradoxical embolism as a potential mechanism.

This study and the others reported in this issue, suggest that, as in other anatomical areas of the nervous system, several different mechanisms may result in decompression injury to the inner ear.

SPUMS Annual Scientific Meeting 2002

A review of diving and hyperbaric medicine in Vanuatu

Robert F Grace

Key words
Recreational diving, resort diving, tourism, decompression illness, hyperbaric facilities, travel medicine

Abstract

(Grace RF. A review of diving and hyperbaric medicine in Vanuatu. *SPUMS J.* 2004; 34: 23-6.)
Many of the world's great dive sites are located in developing countries. Vanuatu is a typical example. It is a small, developing island nation comprising 84 separate islands spanning 1000 km in the South West Pacific. Amongst these islands are fabulous coral reefs, prolific marine life and world-famous wrecks. Combine these with warm, tropical waters and Vanuatu is a paradise for scuba divers. The author has provided medical supervision for the hyperbaric chamber and been the focus for diving-related medical issues in Vanuatu since 1999. This article details experiences in diving and hyperbaric medicine in Vanuatu, including a summary of the treatment of 19 divers with decompression illness. It also highlights some of the difficulties encountered and some of the health and safety issues to be considered when visiting developing island nations.

Introduction

Vanuatu is a popular dive-holiday destination. One of Vanuatu's best-known dive sites is the wreck of the *President Coolidge*, a deep wreck dive at Luganville, in the north of the country. The *Coolidge* was alleged to be the location of a high incidence of decompression illness. In 1999 the travel-insurance industry approached the local dive operators to discuss withdrawing insurance for divers coming to Vanuatu. The cost of flying bent divers to Australia by pressurised aircraft was proving exorbitant. The insurance industry suggested that if the dive operators could obtain a recompression chamber to treat divers in-country they would continue to provide insurance. As a result of this dialogue a chamber was procured, funded by subscriptions levied on the dive operators.

The chamber was located in Luganville close to the *Coolidge*. This created a number of logistical problems. In particular, there was an absence of medical officers with experience or interest to supervise the chamber's operations. As a result the author began to supervise the chamber from Port Vila with an expatriate nurse experienced in hyperbaric medicine providing on-site assessment. This arrangement was far from ideal. Subsequently the managers in Luganville were no longer able to run the chamber. It also became apparent that as many divers were getting decompression illness (DCI) in Port Vila in the south as at Luganville in the North. Therefore the chamber was relocated to the capital, Port Vila.

Decompression illness (DCI) in Vanuatu

In the past four years, 37 divers have sought advice regarding the possibility of suffering from DCI. Of these, 29 (78%) were thought to have DCI. The treatment obtained by each of these divers is listed in Table 1. Note that four were flown offshore for treatment; one due to weather difficulties, and the others when the chamber was non-operational. Four divers refused any treatment.

The following information refers to the 19 divers who were treated in the chamber in Vanuatu. The mean age of the divers was 37.2 years, range 24–52. Ten were female, of whom three were menstruating at the time of their injury. Only two divers were smokers. Gastroenteritis was reported in three (16%) divers, raising the possibility of dehydration as a contributing factor. Demographic and diving details are shown in Table 2.

Table 1. Treatment received by 29 divers developing decompression illness in Vanuatu

	Number	Percentage
Hyperbaric treatment in Vanuatu chamber	19	65
Evacuation by pressurised aircraft for overseas treatment	4	14
Normobaric oxygen and intravenous fluids	2	7
Refused treatment	2	7
Refused treatment and took commercial flights home	2	7
Total	**29**	**100**

The dive histories for these 19 divers showed a fairly consistent pattern of repetitive and 'deep' diving, often with reverse profiles. The divers performed a mean of 1.8 dives per day in the period leading up to the episode of DCI. The dives undertaken were to a mean maximum depth of 32.3 meters sea water (msw). The mean depth of dives in Port Vila was 27.5 msw, while in Luganville it was 39.5 msw, reflecting the depth of the *Coolidge*. All dives were with open-circuit scuba; 16 using air and three using Nitrox mixtures. One diver performed 45 dives in 18 days, waited 20 hours before flying and developed knee pain at altitude. It is interesting to note that three divers had out-of-air situations that resulted in DCI. In all but one of the 19 cases the buddy diver suffered no illness.

One diver had a classic case of cerebral arterial gas embolism (CAGE) with loss of air supply at depth, followed by an uncontrolled ascent and loss of consciousness on the surface. The others were cases of predominantly static neuro-cutaneous or musculo-cutaneous DCI. Shoulder pain, tingling in the distal limbs, and rash were the most common manifestations of DCI (Table 3).

All the divers, including the CAGE victim, were treated with an initial Royal Navy Table 62. This was usually completed by two follow-up treatments using a Table 18:60:30 (1.5 hr, maximum pressure 280 kPa), range 0-4 follow ups. One exception was a follow-up RN Table 61. With only one exception no diver required additional recompression treatment outside of Vanuatu. The one diver who had further treatment re-presented to an Australian unit complaining of minor, residual knee pain and received a further nine treatments.

Discussion of DCI cases

It is estimated from levied funds that somewhere between 8,000 and 12,000 scuba dives are undertaken each year in Vanuatu. Twenty nine divers experiencing DCI represents an incidence of approximately 0.07%. This may be an underestimate as some divers will not report symptoms. The 2002 report on Project Dive Exploration indicated that 32,000 dives resulted in 11 divers being recompressed, equivalent to an incidence of about 0.03%.[1] The Vanuatu rate is slightly higher and this may reflect the presence of the *President Coolidge*. The incidence is also higher than those in some other holiday dive destinations such as Thailand, where an incidence of 0.02% has been reported.[2]

The presence of gastroenteritis in three divers raises the possibility of dehydration as a contributing factor. The frequency of gastroenteritis in DAN's 2002 review is only 4%.[1] Repetitive diving and dehydration are known to be risk factors for decompression illness. The diving patterns reported, with repetitive and deep diving, are not surprising. Most divers coming to Vanuatu for a dive holiday undertake a large number of dives in a short space of time.

Table 2. Features of divers and dives resulting in decompression illness

		Number
Medications	Nil	6
	Oestrogens or OCP	5
	Anti-malarials	4
	Anti-histamines	2
	Anti-inflammatories	1
	Diuretics	1
Health problems	Nil	10
	Gastroenteritis	3
	Hypertension	2
	Arthritis	2
	Asthma	1
Dive planning	Dive computer	17
	Tables	3
	Followed other diver	1
	No gauges at all	1
Purpose of dive	Recreational	17
	Student	1
	Work (freeing anchor)	1
Problems during dive	Nil	13
	Rapid ascent, out of air	3
	Cold	2
	Sensation of air deficit	1
	Heavy exertion and out of air	1

OCP = oral contraceptive pill

Table 3. Symptoms of decompression illness reported by 19 divers in Vanuatu

Symptom	Number	Percentage
Shoulder pain	11	58
Tingling in distal limbs or digits	11	58
Rash	10	53
Headache	6	32
Muscle pains	5	26
Decreasing mental function	4	21
Knee pain	4	21
Vertigo	3	16
Nausea	3	16
Shortness of breath	3	16
Blurred vision	3	16
Tingling in face	2	11

DAN's 2002 report lists pain as the most common symptom of DCI, present in 40% of their patients, followed by paraesthesia.[1] A report from Turkey also lists the shoulder as the most common site for musculoskeletal pain in recreational divers with DCI.[3] Our data are similar.

Most of the divers were keen to return home at the earliest possible opportunity and often waited only a couple of days

before flying. As a result, whenever possible, even if divers feel very good after their first couple of treatments we try to give them a further follow up. In comparison, DAN's 2002 review of recreational scuba diving injuries reports over 50% of injured divers received only one hyperbaric treatment.[1] It is interesting to note that 4 (14%) of the 29 divers presenting with DCI either refused treatment or chose to ignore advice and flew home on commercial flights. At least one of these divers developed worsening of symptoms during the flight and required oxygen.

A surprising number of divers do not carry insurance. This is a problem as treatment is expensive. It is still substantially cheaper to be treated in Vanuatu than pay for a pressurised aircraft to come and retrieve you. For divers with mild symptoms and no insurance this is a difficult decision. Many divers feel compelled to return home for a multiplicity of reasons rather than stay and be treated and are therefore inclined to risk travelling. It is difficult to be emphatic about the long-term consequences of a mild episode of DCI. Until more is known this issue will be a recurring one in remote dive locations.

Compared with most hyperbaric facilities the chamber in Vanuatu is a small operation. It comprises one multi-place chamber operated on an as-needed basis. In Vanuatu, we are often surprised by the number of follow-up treatments given to some patients overseas. One cannot help but wonder whether the high number of vested interests in these large facilities leads to a tendency to provide more treatments than might objectively be required, particularly when good, hard evidence seems to be lacking.

A number of full-time hyperbaric professionals are scathing of small, isolated chambers in remote locations. It is true there are many problems with their operation. However, in the event of serious DCI they can provide rapid treatment well in advance of the time of any retrieval aircraft. The best times to aircraft-on-the-ground in Vanuatu are in the order of 14 hours. The author is quite firmly of the opinion that if he should surface after a dive with ascending paralysis he wishes to be placed in the local chamber immediately. In Port Vila this would take about an hour in an emergency.

It is also interesting to note that in DAN's 2002 survey of 200 chambers in the United States 30% had not treated any dive-related injuries.[1] Thus, a small chamber like ours is able to offer comparatively greater experience in dealing with divers than some larger units. Further, as so many divers are travelling without insurance they are far more likely to be able to cover the costs of direct in-country recompression out of their own pocket than the cost of a pressurised aircraft and its team from Australia. The latter is at least an order of magnitude more expensive. From an economic viewpoint, if remote chambers contribute to keeping the cost of travel insurance down perhaps we are all better off in the long run?

Fitness to dive

Many people come to Vanuatu on holiday without thoughts of diving. When they arrive, resort courses and other easy opportunities to learn to scuba dive mean that a steady stream of tourists have questions regarding their fitness to dive. The most common issue is asthma. There are no facilities to perform respirometry or saline challenges in Vanuatu. These are tests that most diving medical standards require.[4] Australian Standard AS 4005.1-2000 states that pulmonary function tests shall be conducted.[5] We are unable to comply with this standard. Vanuatu, like most developing countries with good-quality diving, has no standards of its own on fitness to dive.[6]

Thus, the author takes a conservative approach. The difficulty arises in so called 'asthmatics' who have been free of asthma since childhood, without the need for medication for many years. These individuals have the dangers of diving with asthma explained to them. They are then left to make their own decision as to their fitness or otherwise to dive.

Other presentations for assessment have included individuals with ischaemic heart disease, diabetes and epilepsy. If at all possible anyone even vaguely considering undertaking a dive course when on holiday should seek medical advice prior to leaving their country of origin.

Miscellaneous problems

Ear problems are the most common group of non-DCI complications to arise following scuba diving. Three cases of significant ear barotrauma have presented, including bilateral tympanic membrane perforations and a possible round-window injury that was referred to Australia. Otitis externa is very common in Vanuatu. There is an audiogram facility at Vila Central Hospital. Sub-conjunctival haemorrhage, sea urchin injuries and stonefish stings are among other miscellaneous problems encountered. Questions regarding the safety of medication, particularly anti-malarials are also common. Hypoxic syncope of ascent and near drowning in a deep-water free diver required helicopter evacuation from a remote island. Two other near drownings requiring hospital admission and a case of acute myocardial infarction in a diver prior to descent are some of the other cases of diving medical interest.

Diving-related fatalities

There were six water-related fatalities during this time. One tourist drowned whilst snorkelling. This individual was resuscitated by bystanders but later declared braindead after transfer overseas. Another tourist suffered a fatal cardiac arrest on the beach immediately after snorkelling. A local snorkeller drowned while fishing, probably as a result of breath-hold diving and hypoxic syncope of ascent. Two tourists known to be poor swimmers drowned in shallow

water. One scuba diver with a history of depression may have committed suicide by diving alone at night under the influence of alcohol.

The 'real risks' on your dive holiday

Despite the emphasis on diving-related problems, people on dive holidays are far more likely to develop problems above the water than below it. Motor vehicle accidents, particularly involving motor scooters, and other trauma are a common problem. Tourists, including divers, fall off their scooter and sustain a range of different injuries far more commonly than suffering any diving-related problem. This is exacerbated in Vanuatu by the fact that we drive on the right-hand side of the road, not the left as in Australia and New Zealand where most of the tourists originate. Alcohol may play a role in the incidence of accidents. One diver sustained a life-threatening injury falling from a balcony and was lucky to survive.

There is an ever-present risk of malaria. Gastroenteritis or traveller's diarrhoea is common. There is also the risk of viral infections when exposed to a large number of new people e.g., in a hotel group. All these account for a much larger number of health-related problems than does diving per se.

Medical services in Vanuatu

Many tourists do not have a real understanding of the limited capabilities of the health services in remote locations such as Vanuatu. To be seriously ill or injured far from home is of major concern.

Vila Central Hospital is the major referral hospital for Vanuatu. Out of hours there are no medical staff in the hospital, they are available only on call. What medical staff there are come from many different cultural backgrounds. They have different standards of training and different languages all contributing to the potential for problems. Communication between staff is difficult. The hospital switchboard is ad hoc and unreliable. Out of hours the hospital switchboard will be unable to get an international telephone connection. Simple things such as blood tests and X-rays are unreliable. There is no CT scanning facility, etc. Until recently there was no hot water. Periodically the hospital runs out of basic drugs. Everything seems to take a long time, and so on.

In travelling from the airport to your hotel and then to your dive boat, it is easy to forget how low the standards of infrastructure are in locations such as Vanuatu. When diving in remote locations in the developing world, diver/tourists must put their dive site into the wider geographical context and accept this as part of the risk. Divers should carry comprehensive travel insurance at all times and ensure that their insurance covers them for their planned activities. As divers seek ever more remote locations it is worth noting that all the insurance in the world will not be able to get you out of some places, despite what the brochure says.

Vanuatu is a beautiful place to visit and a beautiful place to dive. Diving and dive medicine in Vanuatu has its challenges but those divers and dive doctors who accept these challenges, whilst taking appropriate precautions, will not be disappointed.

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SPUMS still needs a volunteer from the membership to take an ongoing interest in redeveloping and maintaining the SPUMS web site with the aid of a web site professional with whom negotiations are currently in progress.

Computer literacy is essential. Some web site development experience would be desirable.

Any expressions of interest should be directed to the SPUMS President, the Secretary or the Journal Office.

Flying after diving DAN Workshop 2002 - an overview

Drew Richardson

Key words
Flying (and diving), meetings, DAN - Divers Alert Network

Abstract

(Richardson D. Flying after diving DAN Workshop 2002 - an overview. *SPUMS J.* 2004; 34: 27.)
In May 2002, Divers Alert Network hosted a one-day workshop to review the state of knowledge on flying after diving, and to discuss whether there was a need for new flying-after-diving guidelines for recreational divers. After single no-decompression dives a minimum pre-flight surface interval of 12 hours is suggested. After multiple no-decompression dives per day or multiple days of diving a minimum pre-flight surface interval of 18 hours is suggested. For dives requiring decompression stops, there is little experimental or published evidence on which to base a recommendation. For decompression diving, a pre-flight surface interval substantially longer than 18 hours appears prudent.

In May 2002, Divers Alert Network, (DAN) hosted a one-day workshop in Durham, North Carolina, USA, to review the state of knowledge on flying after diving, and to discuss whether there was a need for new flying-after-diving (FAD) guidelines for recreational divers. Forty individuals representing the recreational diving industry, government agencies and DAN attended the workshop. Paul Sheffield, previous organiser of an Undersea Hyperbaric Medical Society workshop on FAD in 1989, served as Chair.

Discussions during the morning sessions began with a review and overview by Richard Vann and Paul Sheffield of the history of FAD guidelines and the development of FAD guidelines over time. These presentations were followed by Ed Flynn's review of the 1999 US Navy procedures for ascent to altitude after diving. Richard Vann presented 'Diving at the limits: chamber trials of flying after diving', the results of recent studies concerning the risk of decompression sickness in FAD. Data presented were derived from experiments conducted with dry and resting human subjects in a hyperbaric chamber environment. The study generated 802 FAD exposures and resulted in two main conclusions, and one relevant observation. It first concluded that decompression sickness (DCS) decreased significantly with increasing pre-flight surface interval (PFSI) ($p = 0.018$). The second conclusion was that repetitive dives required significantly longer PFSIs for low DCS ($p = 0.018$). The relevant observation was that no DCS occurred in 52 trials of a 17-hour PFSI.

Following Richard Vann, Jake Freiberger of DAN presented a retrospective analysis of flying after diving attempting to answer the question "Does the PFSI influence the risk of DCS after repetitive, multi-day, open-water recreational diving?" Dr Freiberger also presented an economic model for risk assessment in determining FAD intervals. His papers were followed by Ed Thalmann, who described project goals for a proposed test utilising existing data and new data to develop a decompression model capable of computing risk of DCS for altitude exposures following air dives designed for the USN. Neal Pollock of Duke University, North Carolina, presented data from a study of military free-fall parachuting after diving for Special Forces applications. These trials examined flying at 25,000 feet after diving. Dr Pollock also reviewed NASA flying-after-diving procedures developed in preparation for the Hubble Mission in the 1990s.

In the afternoon, discussions centred on whether changes to the existing FAD guidelines were warranted by the data presented in the morning, and, if so, what those changes should be. The workshop concluded that changes were justified.

The consensus guidelines were as follows.
A. Dives within no-decompression limits:
- Single no-decompression dive: A minimum pre-flight surface interval of 12 hours is suggested.
- Multiple dives per day or multiple days of diving: A minimum pre-flight surface interval of 18 hours is suggested.

B. Dives requiring decompression stops:
There is little experimental or published evidence on which to base a recommendation for decompression diving. A pre-flight surface interval substantially longer than 18 hours appears prudent.

DAN expects to publish the workshop proceedings this year and to submit to the *Undersea and Hyperbaric Medicine Journal* a paper reporting on the experimental trials described briefly above.

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SPUMS Annual Scientific Meeting 2003

The clinical toxicology of carbon monoxide

Des Gorman, Alison Drewry, Yi Lin Huang, Chris Sames

Abstract

(Gorman D, Drewry A, Huang YL, Sames C. The clinical toxicology of carbon monoxide. *Toxicology.* 2003; 187: 25-38.) Carbon monoxide (CO) is a dangerous exogenous poison and an essential endogenous neurotransmitter. This gas when inhaled has an anaesthetic effect, which is poorly understood, but which may be fatal if compensatory mechanisms are exhausted, if cardiac oxygen (O_2) needs exceed myocardial oxygenation and/or if apnoea or asphyxia onsets. Although there is considerable evidence that hypoxia occurs late in CO poisoning, both the treatment of acutely poisoned people and environmental exposure limits are largely based on a hypoxic theory of toxicity. The significance of recent demonstrations of increased endogenous CO and NO production in neurons of animals exposed to exogenous CO, and of a related sequestration of leucocytes along the endothelium and subsequent diapedesis is also not fully understood, but may in part explain both acute and delayed deleterious effects of a CO exposure. Delayed brain injuries due to a CO exposure may be preventable by hyperbaric O_2. However, the ideal dose of O_2 in this context, if any, is unknown and other potential treatments need to be tested.

Keywords
Carbon monoxide; clinical toxicology; exogenous poison

Abbreviations
APP, Beta Alzheimer's precursor protein; BBF, Brain blood flow; CO, Carbon monoxide; COHb, Carboxyhaemoglobin; DNS, Delayed neurological sequelae; GCS, Glascow coma scale; GFAP, Glial fibrillary acid protein; Hb, Haemoglobin; HBO, Hyperbaric oxygen; H and E, Haemotoxylin and Eosin; HO, Haeme oxygenase; Hx, History; kPa, kilopascals; LOC, Loss of consciousness; NBO, Normobaric oxygen; NLOC, No loss of consciousness; NMDA, *N*-methyl-D-aspartate; NNH, Number needed to harm; NNT, Number needed to treat; NO, Nitric oxide; NOS, Nitric oxide synthetase; O_2, Oxygen; OHb, Oxyhaemoglobin; I, Partial pressure of inspired oxygen; PMNL, Polymorphonuclear leucocyte; RCT, Randomised controlled study; Rx, Treatment; TAF, Thionine and acid fusion; UPTD, Units of pulmonary toxic dose

1. Introduction

A review of the clinical toxicology of carbon monoxide (CO) is justifiable for many reasons, and especially because this ubiquitous, colourless, non-irritant, odourless environmental gas is often lethal when inspired, but at the same time it is an endogenous neurotransmitter (Barinaga, 1993; Gorman and Runciman, 1991; Haley, 1998; Runciman and Gorman, 1993; Verma et al., 1993). Carbon monoxide is the most common lethal poison in every community yet studied, and accounts for more hospitalisations (50% attempted suicides and 33% occupational exposures in Australasia) than all other non-prescribed poisons combined (South Australian unpublished coronial data). Despite some form of treatment, more than 10% of survivors are left with a presumed brain injury (Juurlink et al., 2000; Myers et al., 1985). The onset of these injuries may be delayed for several days after the exposure. The assumed toxic mechanism of hypoxia secondary to hypoxaemia does not, by itself, explain much of the published in vivo and clinical data (Gorman et al., 2001; Gorman et al., 2002; Langston et al., 1996; Ludbrook et al., 1992b; Mayesky et al., 1995; Meilin et al., 1996; Meyer-Witting et al., 1991; Thom et al., 1997). Nevertheless, the treatment of CO poisoned patients and environmental exposure limits are based on this theoretical toxicity (Juurlink et al., 2000; Kindwall, 1994).

This review of the hypotheses proposed to explain the toxicity of CO will be divided into hypoxic and cellular theories. This will be followed by a brief review of the management of people poisoned with CO in the context of evidence based best practice.

2. The hypoxic theory of carbon monoxide toxicity

Haldane (1896) proposed a hypoxic basis for CO toxicity in 1896. He argued that hypoxia would arise from the hypoxaemia that occurs when carboxyhaemoglobin (COHb) forms; this is still the most widely accepted explanation of CO toxicity (Kindwall, 1994). Thirty-one years later, his son reported an experiment, which demonstrated that CO had a mortal toxicity that did not appear to be related to haemoglobin (Hb) (Haldane, 1927).

There are also other strong in vivo and clinical arguments against a paramount hypoxic hypothesis for CO toxicity.

Figure 1.
Left and right cerebral hemispheric blood flow (H_2-clearance) versus oxyhaemoglobin concentration in rabbits made hypoxaemic by either dilution of inhaled air with nitrogen or carbon monoxide (Ludbrook et al., 1992b)

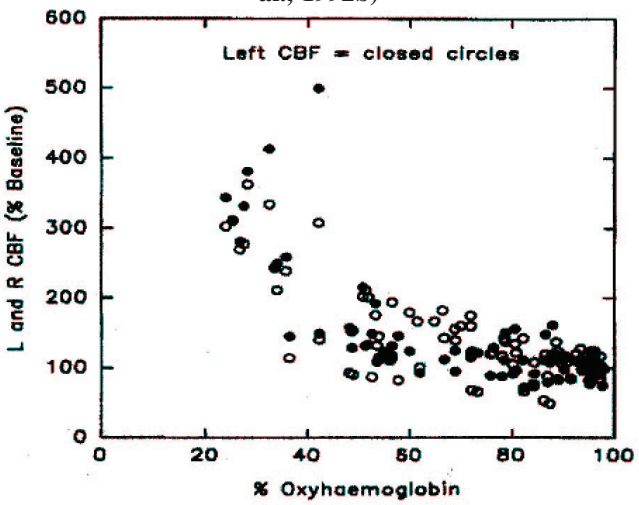
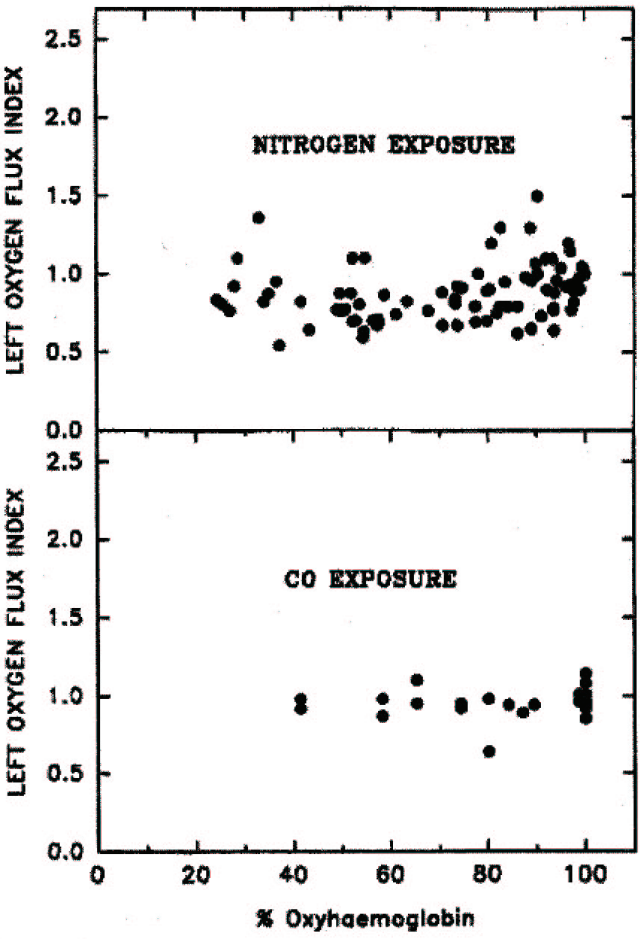


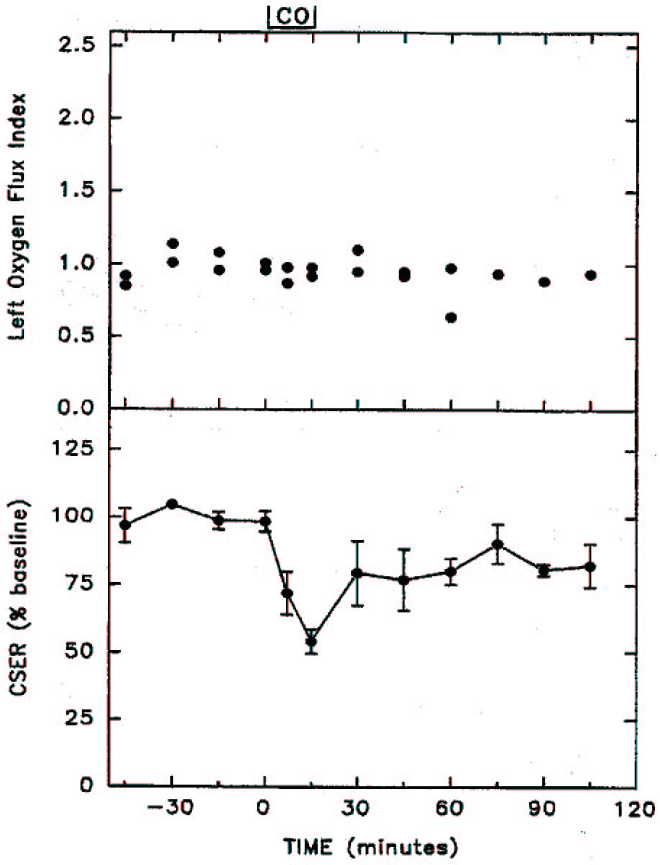
Figure 2.
Left cerebral hemispheric oxygen flux (blood flow x oxygen content) versus oxyhaemoglobin concentration in rabbits made hypoxaemic by either dilution of inhaled air with nitrogen or carbon monoxide (Ludbrook et al., 1992b)



In vivo experiments in many different animal models and clinical studies have produced variable results, from isolated white matter injury to discrete apoptosis, to diffuse cortical atrophy (Gale et al., 1999; Ginsberg et al., 1974; Gorman et al., 2001; Okeda et al., 1981; Piantadosi et al., 1997; Prockop and Naidu, 1999; Thom et al., 2000). The extent to which these results are compatible with a hypoxic injury is variable and in some animal studies the effect of CO has been shown to be independent of COHb formation and/or hypoxia (Ludbrook et al., 1992b; Meilin et al., 1996; Meilin et al., 1998; Thom et al., 1997). Although plasma lactate levels do increase in animals exposed to CO (Penney and Chen, 1996), the lactate does not appear to originate in the brain (Langston et al., 1996). In addition, increased plasma levels of oxidised proteins seen in rats after a CO exposure are neither directly related to hypoxic stress from COHb formation nor significantly influenced by circulating platelets or polymorphonuclear leucocytes (PMNL), but are reduced by nitric oxide synthetase (NOS) blockade (Thom et al., 1997).

Studies in anaesthetised rabbits and in awake sheep have shown that hypoxia cannot explain the early anaesthetic or cortical evoked response suppressant effects of CO (Gorman et al., 2001; Gorman et al., 2002; Langston et al., 1996; Ludbrook et al., 1992b; Meyer-Witting et al., 1991). Hypoxaemia, whether caused by an inert diluent or CO, causes an increase in heart rate, cardiac output and brain blood flow (BBF) that maintains oxygen (O_2) delivery to the brain (see Figs. 1 and 2). As cited above, this increase in BBF occurs independently of COHb formation and/or tissue hypoxia (Mayesky et al., 1995; Meilin et al., 1996). However, it does appear to be regulated and not a simple vasodilatory response to CO and/or nitric oxide (NO) as O_2 delivery to the brain is near-perfectly maintained in rabbits and sheep exposed to CO (see Figs. 1 and 2) (Langston et al., 1996; Ludbrook et al., 1992b). Red blood cells are released into the circulation, presumably from the spleen, to further increase O_2 delivery (Gorman et al., 2001). Although O_2 dissociates from Hb at lower O_2 tensions in the presence of COHb, cerebral arterio-venous O_2 extraction and BBF data were used to show that the uptake of O_2 by the brain during a narcotic CO exposure in previously awake sheep was adequate for normal function (Langston et al., 1996). Nevertheless, despite this maintenance of O_2 delivery and uptake, and in the absence of any biochemical markers of neuronal hypoxia, evoked responses are inhibited in anaesthetised rabbits and awake sheep are narcotised both apparently and electrophysiologically (see Fig. 3). Hypoxia is only seen in these animals when COHb levels are very high (>70%) and the cardiovascular and cerebrovascular homeostatic response is overwhelmed (see Fig. 1). The BBF autoregulatory response to hypoxaemia in general and to CO specifically cannot only be overwhelmed by decreasing oxyhaemoglobin concentration (OHb) (Langston et al., 1996), but also by sufficiently high doses of CO to cause

Figure 3.
Left cerebral hemispheric oxygen flux (blood flow x oxygen content) and the voltage amplitude of a cortical somatosensory evoked response (CSER) before, during and after a 15 min exposure to carbon monoxide in air (Ludbrook et al., 1992b)



episodes of spontaneous brain depolarisation (Mayesky et al., 1995) and by induced cortical spreading depression (Meilin et al., 1998). The autoregulation is also lost in aged rats (Mendelman et al., 2000). Such an aging effect, along with the increasingly compromised cardiovascular and cerebrovascular function that is seen with increasing age and that will limit any BBF autoregulation, may explain the observation that age is a risk factor for poor outcome in CO poisoned people (Weaver et al., 2002). The significance of this autoregulation in CO poisoning underpins the concern that we have about the in vivo confusion of the effect of CO alone and that of an interaction of CO and ischaemia. It is our opinion that this is a common feature of many in vivo models of CO poisoning. To illustrate our concern, brain lesions seen in sheep after an exposure to CO are more concentrated ipsilaterally in the hemisphere that is on the same side of the body as any carotid artery cannulation (Gorman et al., 2001). Some caution is needed in the interpretation of the in vivo data for the following reasons:

- First, anaesthesia has been usual in these studies and this will affect cerebrovascular behaviour and obscure the behavioural effects of the poison (Gorman et al., 2001; Gorman et al., 2002; Ludbrook et al., 1992a).
- Second, as cited above, many studies have probably caused a combined CO-ischaemic injury and such an interactive insult is very different from that of CO alone (Gorman et al., 2001). This is not surprising given the maintenance of O_2 delivery to the brain, which occurs during and after an exposure to CO as a consequence of a sequential increase in heart rate, cardiac output and BBF, and that has been reported in rats, anaesthetised rabbits and awake sheep (see Figs. 1 and 2) (Gorman et al., 2001; Gorman et al., 2002; Langston et al., 1996; Ludbrook et al., 1992b; Mayesky et al., 1995; Meilin et al., 1996, 1998; Meyer-Witting et al., 1991; Penney and Chen, 1996). It is likely that this protective BBF autoregulatory effect is mediated by NO (Meilin et al., 1996).
- Third, there has been a wide variety in the employed species, CO administration protocols and outcome measures.

It is also difficult to reconcile clinical observations of CO poisoned patients with a paramount hypoxic toxicity (Gorman et al., 1992; Gorman and Runciman, 1991; Juurlink et al., 2000; Myers et al., 1985; Runciman and Gorman, 1993). The quality of health outcomes does not correlate well with the measured COHb levels. Poisoned patients usually have normal blood gases, are normotensive and have few if any systemic markers of hypoxia. Radiological and pathological findings are often not well explained by hypoxia alone.

However, as is the case for the in vivo studies, caution is needed in interpretation (Gorman et al., 1992; Gorman and Runciman, 1991; Juurlink et al., 2000; Myers et al., 1985; Runciman and Gorman, 1993). Most studies are not controlled. Those controlled studies have been small and most have been variously but significantly flawed (see Table 1). Almost half of all poisonings are suicide attempts. Other poisons are often used simultaneously and long-term recovery is complicated by the original mental health disorder. Many patients have a super-imposed hypoxic injury due to asphyxia and/or apnoea. The time from exposure to treatment varies and O_2 is used to a variable extent in the acute resuscitation. Pre-morbid neuropsychometric data do not exist, and it is probable that most outcome surveys have over-estimated the extent of any brain injury and have mistaken co-morbidities for such injury.

3. The cellular theories of carbon monoxide toxicity

As cited above, JBS Haldane (1927) demonstrated that CO had a mortal toxicity that appeared to be independent of Hb. Subsequently, proposed toxic mechanisms to account for this observation include binding to mitochondrial cytochromes, myoglobin, and to non-specific absorption onto catalyst surfaces (Gorman and Runciman, 1991; Piantadosi, 1987; Runciman and Gorman, 1993).

However, relevant cellular enzymes such as cytochrome c ($a–a_3$) have a greater affinity for O_2 than CO, in contrast to Hb, such that limited CO–cytochrome binding may occur in the absence of tissue hypoxia (Piantadosi, 1987). Even allowing for the usually low levels of mitochondrial O_2, this argument has been used to discount significant intracellular CO poisoning; but, for reasons discussed below, this disregard may be inappropriate. Thom has proposed one hypothesis that may explain the delayed effects of CO poisoning (Ischiropoulos et al., 1996; Thom, 1993; Thom et al., 1994; Thom et al., 1997; Thom et al., 1999; Thom et al., 2001). He hypothesised that CO activates PMNL's, which diapedese and cause brain lipid peroxidation. Further, he noted that CO affects platelet scavenging of NO, which in turn will interfere with PMNL binding to endothelial cells, such that the diapedesis will be delayed until after the CO is withdrawn. That is, this phenomenon can explain the delayed but not the anaesthetic effects of CO. The process of PMNL diapedesis and brain lipid peroxidation is inhibited by 303 kPa of O_2, but not at 101 kPa. This might explain some of the clinical reports that delayed brain injury is prevented by hyperbaric O_2 (HBO) (Myers et al., 1985; Runciman and Gorman, 1993; Weaver et al., 2002). In the most recent controlled randomised clinical study reported, and using an intention to treat analysis, the numbers needed to treat (NNT) for HBO versus normobaric O_2 (NBO), to prevent such a delayed effect 6 weeks, 6 months and 1 year after the exposure were 4.76, 5.88 and 6.66, respectively (Weaver et al., 2002). Such data are in conflict with an Australian study that showed a negative NNT of 21 1-month after treatment with HBO (Scheinkestel et al., 1999) and with a French controlled study that suggested only short-term benefit for HBO in the context of preventing delayed neuropsychiatric sequelae (Mathieu et al., 1996); the NNT in the latter study increased from 18 at 3 months, to 33 at 6 months and to 143 at 12 months. It is most noteworthy that all the controlled studies published to date, with the probable exception of the most recent (Weaver et al., 2002), have been sufficiently flawed to make their utility low (see Table 1) (Juurlink et al., 2000).

Since Thom's original observations (Thom, 1993; Thom et al., 1994), more data has been published in support of and to clarify this mechanism of delayed injury. First, CO has been shown to induce both neuronal and glial NOS and haeme oxygenase (HO) and hence to increase intracellular levels of both NO and CO (Gorman et al., 2002; Ischiropoulos et al., 1996; Thom et al., 1999). Second, CO not only increases NO levels, but also is responsible for the potent oxidant, peroxynitrite, being deposited in vascular walls and throughout the brain parenchyma; this is probably secondary to PMNL sequestration in the microvasculature, and is seen together with xanthine oxidase formation and brain lipid peroxidation, all of which are prevented to some extent by NOS blockade (Ischiropoulos et al., 1996; Thom et al., 1999; Thom et al., 2001). Third, these latter effects are not influenced significantly by neutropenia or thrombocytopenia (Thom et al., 1999). Fourth, the CO-induced PMNL adherence to endothelium seen in rats, which becomes apparent about 18 h after the exposure and hence some time after the blood brain barrier is first seen to be damaged (Thom et al., 1999), is induced by qualitative changes in platelet activating factor (Thom et al., 2001). The failure of a pilot study in sheep to show benefit for lignocaine in CO poisoned sheep is surprising in this context (Gorman et al., in press; Mitchell, 2001).

Other researchers have argued that CO has direct psychiatric effects by deranging dopaminergic and serotonergic neural function (Hiramatsu et al., 1994; Muraoka et al., 1998). This is unaffected by *N*-methyl-D-aspartate (NMDA) receptor ion channel complex blockade (Hiramatsu et al., 1994), although the latter largely prevents CO-induced hippocampal cortical injury in rats and mice (Ishimaru et al., 1992; Penney and Chen, 1996). Such hippocampal neurodegeneration and the perhaps related learning deficits in CO poisoned mice are variously affected by neuroactive steroids (Tangui et al., 2000).

The final major cellular theory relates to the role of CO as an endogenous neurotransmitter (Barinaga, 1993; Haley, 1998; Verma et al., 1993). Some neurons are rich in HO and some neural tract function is dependent on CO production. The suggestion then is that injury by inhaled CO may be caused by an excess of neural functions normally regulated by CO and/or by agonist antagonism of neuronal function normally under NO regulation (Kostoglou-Athanassiou et al., 1998).

Narcotic doses (1% in air) of CO administered to awake sheep for up to 2 h, induces anaesthesia (Gorman et al., 2001, 2002). The histological and immunochemical outcome in the brains of these sheep were compared 5–14 days later with control sheep, which were not exposed to CO but were matched for process in every other way. The CO exposed sheep showed a statistically significant increase in periventricular white matter infarcts, with glial activation about these infarcts and some axonal dysfunction (see Fig. 4). Very surprisingly, there was no evidence of neuronal death or apoptosis, but clear evidence of neuronal and glial activation of HO-1 and -2 and NOS-1 and -2, but not of NOS-3, which is endothelial (see Fig. 4).

While much of the last of these observations cannot be currently explained other than as a stress response (Sharma et al., 1998), to some degree the significance of any neuronal induction of HO and NOS is apparent. Most importantly, the cellular theories of CO poisoning need to be re-visited, as intracellular NO and presumably CO will rise significantly (Ischiropoulos et al., 1996; Meilin et al., 1996; Thom et al., 1997, 1999). As cited above, the cellular theories have been discounted to date on the basis of the relative affinities of O_2 and CO to mitochondrial cytochromes, which favours O_2 binding (Piantadosi, 1987). This is clearly not the case for guanylyl cyclase, which binds either CO or NO to produce cyclic-GMP (Barinaga, 1993; Haley, 1998; Verma et al., 1993). The potential for cellular

dysfunction then is apparent.

It is even possible that the anaesthetic effect of CO is relatively harmless as is the case for most other general anaesthetics. The sheep studies cited above would support this argument (Gorman et al., 2001). The difference between these sheep and other in vivo models and people poisoned with CO may be explained variously by the following:

- First, the sheep preparation avoided any ischaemia and other disruption of cerebrovascular behaviour and did not combine the CO exposure with an anaesthetic, which is the case for most other in vivo work. Nevertheless, a greater concentration of white matter infarcts was still found in the brain hemisphere ipsilateral to the carotid artery cannulated for monitoring. One sheep showed impaired BBF after surgical preparation and when that sheep was exposed to CO, the resulting brain injury was much worse than for any other sheep studied.
- Second, there may be species differences in the manner in which BBF is maintained and/or in which red cells are released into the circulation. However, such effects have been reported in rats, rabbits and sheep (Gorman et al., 2001, 2002; Ischiropoulos et al., 1996; Langston et al., 1996; Ludbrook et al., 1992b; Mayesky et al., 1995; Meilin et al., 1996; Meilin et al., 1998; Meyer-Witting et al., 1991; Penney and Chen, 1996) and similar results have been cited in cats and monkeys (Ginsberg and Myers, 1974; Okeda et al., 1981).
- Third, the sheep airway was maintained during the CO-induced anaesthesia.

Our hypothesis here is that acute brain injury in CO exposed people may largely arise from hypoxia due to either asphyxia and apnoea (during what is essentially an uncontrolled general anaesthetic exposure) and aspiration of vomitus, or to failure of the cerebrovascular compensation for the CO-induced hypoxaemia. The latter will be accelerated by myocardial disease and ischaemic heart disease, by cerebrovascular arteriosclerosis, which will impair reactivity, and compounded by the use of other brain active substances such as tri-cyclic antidepressants and alcohol as part of a suicide attempt and by concurrent exposure to fume such as hydrogen cyanide in a house fire. We also believe that Thom's PMNL theory (Ischiropoulos et al., 1996; Thom, 1993; Thom et al., 1994, 1997, 1999, 2001) at least in part explains any delayed neural injury; a similar mechanism has been shown to account for brain injury after arterial air embolism (Helps and Gorman, 1991). This has therapeutic implications (Mitchell, 2001). However, we are cognisant of the strong relationship between recovery after a head injury and pre-morbid factors such as depression, emotional coping skills and work satisfaction (Kushner, 1998), and hence are uncertain as to the extent to which the delayed neuropsychological sequelae of CO are due to brain injury and the extent to which they are a reaction to an acute threat to health and hospitalisation, with consequent illness beliefs (Pilowsky, 1997). It is also noteworthy that many CO exposures in first world countries are suicide attempts (unpublished South Australian Coronial data). A control group of non-CO exposed acutely hospitalised patients, both for and not for suicide attempts is clearly necessary in any longitudinal study of CO poisonings.

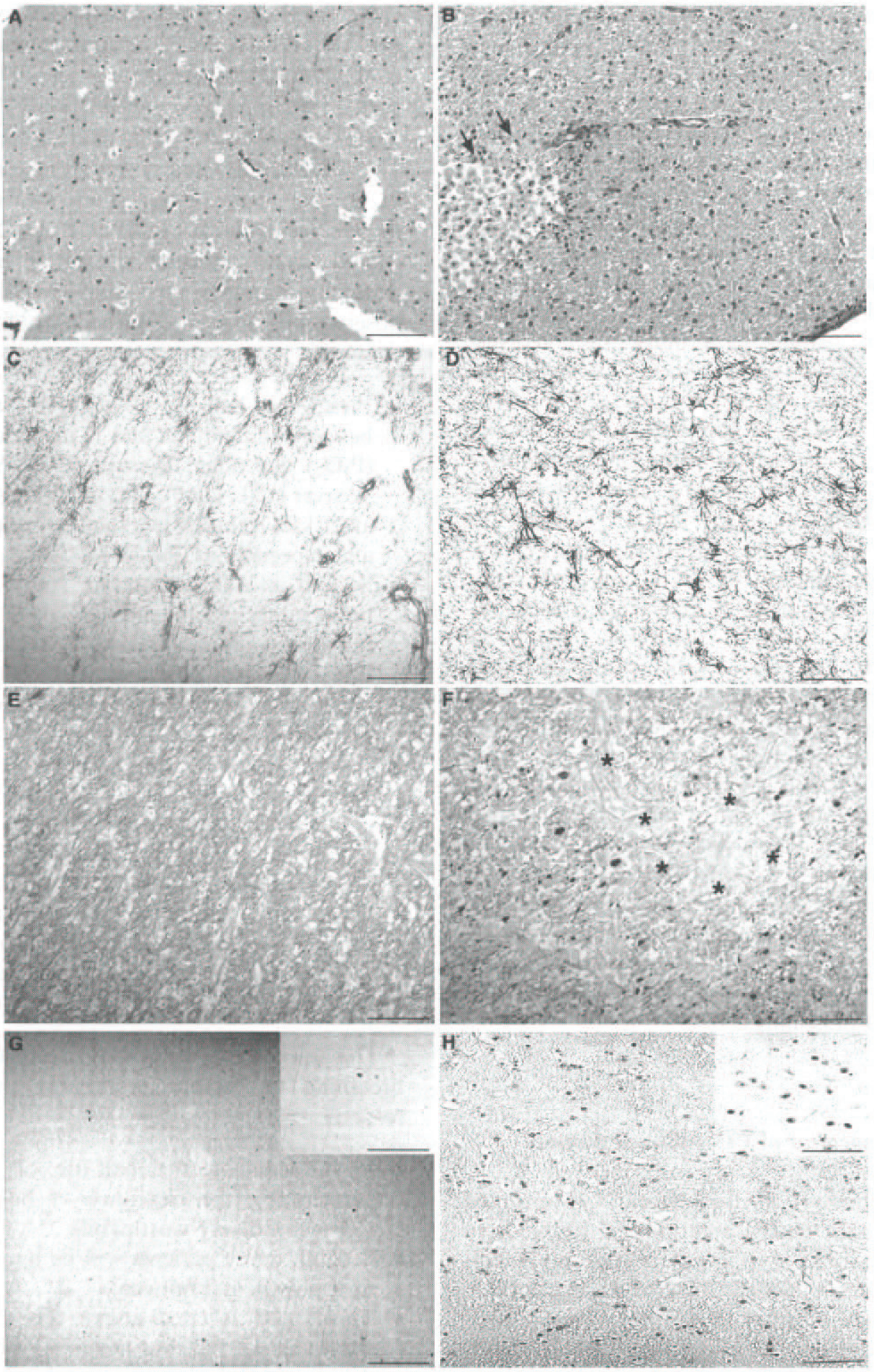
4. The management of CO poisoned patients

Most attention in the treatment of CO poisoned people has been on the breathing of 100% O_2 to reduce the half-life of COHb (Gorman and Runciman, 1991; Juurlink et al., 2000; Myers et al., 1985; Runciman and Gorman, 1993). However, COHb is not toxic in itself (Goldbaum et al., 1975; Orellano et al., 1976), and brain hypoxia is probably not a feature of CO poisoning until either cardiovascular homeostasis is exhausted (see Fig. 1) and/or asphyxia or apnoea onsets (Gorman et al., 2001, 2002; Langston et al., 1996; Ludbrook et al., 1992b; Mayesky et al., 1995; Meilin et al., 1996, 1998; Meyer-Witting et al., 1991; Penney and Chen, 1996). Not surprisingly then, an ideal dose of O_2, if it exists, has neither been identified by these clinical studies (Juurlink et al., 2000) nor can it be derived from other clinical situations and/or from in vitro and in vivo data. The following three observations illustrate the reason for the latter comment:

- First, the half-life of COHb varies widely between individuals and is inspired O_2 tension (P_IO_2) dependent (Sasaki, 1975; Smart, 2002; Weaver et al., 2000), varying from several hours at 21 kPa to much less than an hour at 101 kPa and especially at 282 kPa.
- Second, in a large German study of traumatic brain injury (Holbach and Caroli, 1974), brain function and metabolism was enhanced at a P_IO_2 between 101 and 151 kPa, but were progressively worse between 202 and 252 kPa.

Figure 4.

Representative samples of CO-induced white matter damage and HO-1 immunochemistry in the sheep brain 5 days post CO exposure (control sheep panels on the left) (Gorman et al., 2001; Gorman et al., in press). (A–B) H and E staining showing normal brain white matter (A) and peri-ventricular white matter micro-infarction (arrows) and gliosis after a CO exposure (B). (C–D) GFAP immunostaining of glial activity showing greater activity (about the infarcts cited above) in brain white matter after a CO exposure (D). (E–F) beta-APP immunostaining showing positive stained and hence damaged axons (about the infarcts cited above) in brain white matter after a CO exposure (F). (G–H) HO-1 immunostaining showing a greater frequency of cells demonstrating HO-1 activity in brain white matter (H) and cortex (insert) after a CO exposure. Scale bar: 1 cm = 25 um.


A
B
C
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E
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H

- Third, Thom (1993) has shown that the delayed PMNL diapedesis and subsequent lipid peroxidation that is seen in rats after a CO exposure, is inhibited if they breathe a P_IO_2 of 303 kPa, but not at 101 kPa. Clearly, two points on a dose continuum do not demonstrate the nature of the relevant dose response relationship and the extrapolation of these rodent data directly to the human experience should be cautious.

This uncertainty about dose is not to argue that breathing O_2 is not useful for the following reasons:

- First, reducing the half-life of COHb and accelerating the clearance of body stores of CO is intuitively worthwhile.
- Second, many patients will be hypoxic because of asphyxia and apnoea.
- Third, and as cited above, Thom (1993) has shown that HBO, but not NBO will inhibit the delayed PMNL diapedesis and subsequent lipid peroxidation that occur after a CO exposure.

The latter is consistent with the data presented by a Salt Lake City research group (Weaver et al., 2002), in which and again as cited above, the NNT for HBO over NBO in preventing delayed neural sequelae both 6 weeks and 1 year after the exposure was 4.76 and 6.66, respectively. However, a Cochrane Collaboration Review of earlier controlled randomised studies could not show any sustained benefit for HBO in this context (Juurlink et al., 2000). Several conclusions can be made from this Cochrane review:

- First, the controlled studies reported are, with the probable exception of the Salt Lake City study (Weaver et al., 2002), flawed and of low utility (Ducasse et al., 1995; Mathieu et al., 1996; Raphael et al., 1989; Scheinkestel et al., 1999; Thom et al., 1995). We have summarised the studies in Table 1.
- Second, the Cochrane review itself is internally inconsistent. For example, the Australian study (Scheinkestel et al., 1999) was given a Jadad rating of 5 by the Cochrane reviewers (Jadad et al., 1996), despite their recognition of a statistical approach that made consequent error inevitable.
- Third, the Cochrane reviewers did not cite what is arguably the most impressive clinical study published to date (Myers et al., 1985), which while it was not randomised and has many features that are not well described, had a biased control group that was selected by a clinical triage of such factors as COHb levels, and clinical and neuropsychometric status. This study is also summarised in Table 1 and shows not only an apparent strong advantage for HBO over NBO, but also that conventional triage techniques do not identify those patients likely to develop delayed neural sequelae. The latter may even support the argument cited above that these sequelae may be largely independent of any acute CO-induced brain injury per se.
- Fourth, the studies have used such different doses of HBO and NBO that any compilation of data is impossible. We have used an established biological marker of O_2 dose (Bardin and Lambertson, 1970) to quantify both HBO and NBO doses in Table 1. The concern here is that some physicians have elected to rely on NBO to treat CO poisoned patients, as a consequence of the Australian study (Scheinkestel et al., 1999), but are using a dose of NBO very dissimilar to that used by the researchers.
- Fifth, only the Australian (Scheinkestel et al., 1999) and the Salt Lake City (Weaver et al., 2002) studies used a sham treated control group, although the first of these studies also lost more than half of all subjects to follow up. The explanation offered by the authors that this was accounted for by doubling the number of patients recruited is not a satisfactory response.

In conclusion, while we do not agree with some aspects of the Cochrane review (Juurlink et al., 2000), we do agree with their conclusions. It follows that data do not exist to establish evidence-based best practice. This is also true for patient selection. In contrast to the high risk factors identified in the Salt Lake City study (any one of the following: age >50 years; COHb >25%; a history of loss of consciousness; evidence of metabolic acidosis) (Weaver et al., 2002), a similar triage failed to identify those likely to suffer delayed neuropsychological sequelae in the biased control group study cited above (Myers et al., 1985). Medico-legal issues may even predominate in clinical decision-making and the latter may pay attention to this biased control group study data (Myers et al., 1985).

Also as cited above, the observation that CO causes PMNL diapedesis, that this may explain the CO-induced delayed brain injury and that this is preventable in vivo by HBO but not NBO (Thom, 1993) has therapeutic implications, not only for HBO but also for chemical interventions. A similar mechanism of brain injury occurs in cerebral arterial gas embolism (Helps and Gorman, 1991). Such emboli are probably responsible for much of the brain injury commonly seen after open-heart surgery and a lignocaine infusion has been shown to significantly reduce the frequency and severity of such injury in cardiac surgical patients (Mitchell et al., 1999 and Wang et al., 2002). The rationale for lignocaine in this context is well established both in vitro and in vivo (Mitchell, 2001). However, and as cited above, a pilot study in sheep suggests that the interaction of lignocaine and CO may increase the likelihood of white matter infarcts, such that other potentially therapeutic agents need to be tested (Gorman et al., in press).

In the interim, while the incidence and often poor outcome of CO poisoning is well recognised, and although the biology of CO is increasingly understood, the toxicology of the gas is mysterious and no data exist to establish best practice for managing poisoned patients. The potential for research is clear and urgently needed.

Table 1
Comparison of randomised controlled studies of HBO versus NBO in CO Poisoning

Authors	Study design	Protocol	Comments	NNT[a]
Raphael et al. (1989)	Non-blinded RCT Four groups: two with, two without loss of consciousness (LOC)	HBO at 202 kPa	Non-blinded Many included with very mild poisoning	LOC 50
	LOC control (N = 127) HBO/NBO 203 kPa, 540 UPTD	HBO 2 h, NBO 4 h	Both LOC groups had HBO	NLOC 50
	LOC (N = 125) HBO/NBO/HBO 203 kPa, 840 UPTD	HBO 2 h/NBO 4 h/HBO 2 h		
	NLOC control (N – 148) NBO 101 kPa, 360 UPTD	NBO 6		
	NLOC (N = 159) HBO/NBO 203 kPa, 540 UPTD	HBO 2 h, NBO 4 h		
	Delay to treatment < 12 h (av. 6 h) Follow-up at 1 month			
Thom et al. (1995)	Non-blinded RCT	HBO 282 kPa for 30 min then 2.0ATA for 90 min preceded by NBO, av. 2.1 h NBO for av. 4.2 h	Excluded if Hx of loss of consciousness or ECG changes Non-blinded	4
	HBO (N = 30) 284 kPa, av. 338 UPTD NBO (N = 30) 101 kPa, av. 252 UPTD			
	Delay to treatment < 6 h (av. 2 h) Follow-up at 1 and 3 months			
Ducasse et al. (1995)	Single-blinded RCT	HBOT 252 kPa 2 h, then NBO 100% 4 h then 50% 6 h	Small sample	2, 2 h post Rx
	HBO (N = 13) 253 kPa, 620 UPTD NBO (N = 13) 101 kPa, 360 UPTD	NBO 100% 6 h then 50% 6 h	Patients/staff not blinded to treatment No long-term follow-up Surrogate outcomes rather than DNS measured	3, 12 h post Rx
	Delay to treatment < 2 h Follow-up at 2 and 12 h		GCS < 12 excluded	
Mathieu et al. (1996)	Non-blinded RCT HBO (N = 299) 253 kPa, 285 UPTD	HBOT 2.5ATA 90 min NBO 100% 12 h	Interim report only No details of outcome measures or dropout rate	33 at 1 months 18 at 3 months
	NBO (N = 276) 101 kPa, 720 UPTD		No sham treatment No details of randomising or blinding	33 at 6 months 143 at 12 months
	Delay to treatment < 12 h Follow-up at 1, 3, 6, and 12 months			

Table 1 (continued)
Comparison of randomised controlled studies of HBO versus NBO in CO Poisoning

Authors	Study design	Protocol	Comments	NNT[a]
Scheinkestel et al. (1999)	Double-blinded RCT	HBOT 282 kPa 60 min x 3 over 72 h, NBO (av. 60%) between treatments. This repeated by 28% of group	Unusually high oxygen doses	-17 post-Rx
	HBO (N = 104) 284 kPa, 1879 UPTD	NBO (100%) 100 min in chamber x 3 over 72 h with 60% NBO between treatments. This repeated by 15% of group	Only 46% follow-up at 1 month	-21 at 1 months
	NBO (N = 87) Sham in multiplace 101 kPa, 1423 UPTD		t-Scores calculated on age/education-based norms (but unknown pre-morbid state)	
	Three days of oxygen via non-occlusive mask between HBO/Sham HBO treatments		Jadad score 5/5, but Cochrane review states 'high likelihood of spurious statistical significance'	
	Delay to treatment av. 7.1 h Follow-up post treatment and at 1 month			
Weaver et al. (2002)	Double-blinded RCT	HBOT 303 kPa 50 mint then 202kPa 60 min at 0 h, followed by 202kPa 2 hr x 2 at 6 and 12 h 'NBO' 116 kPa 145 min at 0 h, then 116 kPa air for 2 h x 2 at 6 and 12 h	Small sample size	5 at 6 weeks 6 at 6 months 7 at 12 months
	HBO (N = 76) 303 kPa, 941 UPTD			
	NBO (N = 76) Sham in monoplace 116 kPa, 174 UPTD			
	Delay to treatment < 23 h Follow-up post treatment and at 2 and 6 weeks			
Myers et al. (1985)	HBO (N = 131) 283 kPa, 164 UPTD	HBOT 283 kPa 46 min	Not randomised or blinded	8
	NBO (N = 82) 101 kPa, 240 + UPTD Delay to treatment av. 30 min Follow-up at 1–21 days post treatment and 6–12 months	NBO 100% 4 h (or more)	Biased control group	

Accurate figures for NNH (number needed to harm) could not be calculated from the available data.
[a] NNT, number needed to treat.

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Professor Gorman presented a paper based on this review at the SPUMS Annual Scientific Meeting, Palau, May, 2003.

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Hyperbaric chamber explodes in Polokwane, South Africa

Frans J Cronje

Extracts from media statement by Frans J Cronje, MB ChB (Pret), President-Elect Southern African Undersea and Hyperbaric Medical Association (SAUHMA)

We deeply regret to inform you of a double fatality related to the explosion of a hyperbaric chamber in South Africa. Two physicians, who were brothers, were killed and a family member seriously injured when a pressure vessel exploded on their private property in Polokwane, Limpopo, on 30 January 2004. The chamber was of unknown construction and applied for personal use. One of the physicians involved had been a registrar at the Wilhelmina Gastehuis in Amsterdam at the time of Prof. Ite Boerema, the veritable pioneer of clinical hyperbaric medicine, and had been involved in the original hyperbaric programme in 1960.

While tragic and completely avoidable, this event shows that hyperbaric therapy can be extremely dangerous in the hands of lay people; even those who may understand the principles of the therapy, but are untrained to provide it. South Africa and many other countries have all suffered the consequences of the deceptive simplicity and aura of alternative medicine attributed to the therapy.

Those unwilling to practise it [hyperbaric medicine] professionally and scientifically, have shunned the precautions and guidelines offered by the various international scientific and medical organisations [and] have rendered the public not only vulnerable to financial exploitation, but also to physical harm.

Hyperbaric oxygen therapy is not an 'alternative' therapy. It has a legitimate place within the realm of mainstream, evidence-based medicine. Accordingly, it has specific uses and risks that need to be managed by those appropriately trained to do so. When provided in certified pressure vessels that are designed and manufactured for the specific purpose and operated by healthcare professionals with formal training, hyperbaric oxygen therapy is both effective and safe.

The Southern African Undersea and Hyperbaric Medical Association (SAUHMA), a Special Interest Group of the South African Medical Society (SAMA) and affiliate of the Undersea and Hyperbaric Medical Society (UHMS) since 1991, has pioneered and fostered the development of the scientifically-based use of hyperbaric oxygen therapy in South Africa. SAUHMA has been a world leader in the development of the Risk Assessment Guide for hyperbaric chambers; the publication of the SABS standards for clinical hyperbaric chambers; and the accreditation of training for healthcare professionals. We hope that this incident will not suffer the additional tragedy of going unheeded and thereby possibly being repeated.

The world as it is

Triage and management of diving accidents: the Phuket Workshop, November 2003

David Elliott, Chairperson

Key words
Accidents, diving, first aid, decompression illness, decompression sickness

Abstract

(Elliott D. Triage and management of diving accidents: the Phuket Workshop, November 2003. *SPUMS J.* 2004; 34: 40-5.)
A five-day medical workshop was held in Phuket in November 2003, sponsored by the international Subaquatic Safety Services Recompression Network. Intentionally, it was an agenda-based workshop providing a wide review with a particular focus on triage, the indications for aero-medical evacuation, action to follow an incomplete response to initial recompression, the role of subsequent repetitive recompressions and flying home after neurological decompression illness. This summary represents the views of an international group of diving doctors experienced in recompression management, and may be a useful and thought-provoking starting point on a number of important issues. Many of these will be revisited at the Undersea and Hyperbaric Medical Society Workshop in Sydney in May 2004.

Introduction

A five-day medical workshop was held in Phuket, Thailand, in November 2003, sponsored by the international Subaquatic Safety Services (SSS) Recompression Network. The first session was well attended by the diving professionals of the island, and Mauricio Moreno, President of SSS, introduced three invited lecturers. Dr Chris Acott offered a 'doctored' scuba set for inspection and reviewed the Adelaide DIMS project; Dr Frans Cronje presented the unique, South African view of marine animal injuries. Professor Alf Brubakk, Norway, reviewed the decompression procedures of the native fishermen of the Galapagos Islands.

The next four sessions, chaired by David Elliott, had some prepared contributions by the delegates present and much discussion, but no formal papers. Intentionally, it was an agenda-based workshop and had contributions on each of several well-recognised problems. It was a wide review with a particular focus on:

- triage and the indications for aero-medical evacuation;
- action to follow an incomplete response to initial recompression;
- the role of subsequent repetitive recompressions;
- flying home after neurological decompression illness.

The issues raised and any conclusions drawn are summarised below, mainly as a series of bullet points for consideration. Many of these will be revisited at the Undersea and Hyperbaric Medical Society (UHMS) Workshop in Sydney in May 2004. The Phuket Workshop summary, which represents the views of a number of diving doctors experienced in recompression management, may be a useful and thought-provoking starting point on a number of important issues for this upcoming meeting.

1. Omitted decompression

What is recommended for asymptomatic omitted decompression in recreational divers with or without a chamber on site?

A. CHAMBER AVAILABLE

- Omitted decompression time < 30 min: USNavy Treatment Table 5 (USN5)
- Omitted decompression time > 30 min: USNavy Treatment Table 6 (USN6)
- If surface interval < 3-5 min: consider surface decompression table (SurD) if no potential complications that might be associated with the cause of premature surfacing, and if all other circumstances are appropriate
- If there are additional risks then SurD with a 6-hour 'bend watch'
- If more than 1 hour post-dive with no symptoms or signs, USN5 with bend watch
- Diver's computer failure: assess the hazard individually

B. CHAMBER REMOTE

- Surface O_2; oral fluids; 6-hour bend watch
- If no O_2 available: rest; oral fluids; no diving; expect trouble and plan accordingly
- Some working and military divers may use their formal procedure for immediate in-water stops, but this is not recommended for use by inexperienced divers

2. Early reporting of possible decompression illness

Encourage early reporting and reduce denial of decompression illness (DCI). This will probably improve outcomes.

Is there a need for better instruction for entry-level divers and instructors on what to do when unexpected manifestations are discovered? Concensus view - Yes

Is present first aid training for diving accidents adequate for dive leaders? Concensus view - No

IMPROVING AWARENESS

It was noted by SSS Thailand that their programme of informal education given by hyperbaric medicine staff for local dive leaders appears to be effective in reducing delays before reporting.

OVERCOMING DENIAL (if it is not organic)

Prevention

Education of entry-level divers and instructors that DCI is often 'no-fault'. Consider the effects of:
- social stigma or inappropriate reproach;
- potential cost of treatment if no insurance and in terms of delay;
- a diver's fear of losing their job.

Management

Education is needed regarding the hazards of non-treatment. Education of employers is also needed, and avoidance of 'black marks' from clients accruing to diving contractors for 'no-fault' recompressions. Early treatment should be considered as an acceptable safety procedure that prevents serious residua.

Table 1.
Closed- vs open-circuit oxygen equipment for conscious divers

Advantages	Disadvantages
OPEN CIRCUIT	
- Simplicity	- Short duration
- Conscious and unconscious divers	- Fire hazard
- No CO_2 retention	
- Versatile	
CLOSED CIRCUIT	
- Duration	- Breathing resistance
- Gas warming	- No flush-through possible
	- Additional training

3. First aid and local evacuation

NB Surface oxygen dosage is usually unknown.
- How common is relapse after discontinuing oxygen?
- How does this concern influence the need to recompress a now symptom-free diver?

CLOSED- VS OPEN-CIRCUIT OXYGEN EQUIPMENT

The advantages and disadvantages of closed- and open-circuit oxygen resuscitation equipment are summarised in Table 1.

DETERIORATION DURING OR RELAPSE AFTER SURFACE O_2

- How common is continued deterioration? 12% of divers were still deteriorating on arrival at chamber in spite of surface O_2.[1-3]
- How important is relapse? If symptoms and signs have resolved, consider the index of severity of the original manifestations and, unless it was trivial, give preventive recompression.

So, by unanimous consensus, unless symptoms were very mild, the diver must be recompressed. There are potentially serious medicolegal implications if this is not done.

4. Diagnosis and prognosis

In the prognosis of neurological DCI outcome, risk factors are currently non-quantifiable. However, a few indicators of severity are:
- physical signs are more important (>) than symptoms alone;
- progression / relapse > static / resolving symptoms and signs;
- working divers (e.g., inshore, Scotland) > amateurs;
- depth and decompression non-compliance;
- spinal cord injury > cerebral

5. Triage of mild and severe cases - key issues

WHO DECIDES ON TRIAGE?

- Buddy or instructor
- Dive guide or shop
- Chamber doctor
- A central hotline operator with 24-hour competent medical advice

URGENCY VS ACCEPTABLE DELAY

This is determined by:
- risk factors for deterioration;
- severity of the illness (chamber outcome is dependent on the diver's condition when first recompressed, and this in turn may be affected by delay).

REMOTE ASSESSMENT

There is a need to define the essential components of on-site assessment for any level of medical expertise. It is difficult to standardise neurological and psychological assessment when language skills may jeopardise history taking. Training is required to provide effective and valid assessment.

Difficulties with remote neurology include:
- minimum requirements;
- standardisation of examination;
- need for recumbent patient to stand for certain examinations e.g., gait or Romberg test, that during the early phases may cause adverse bubble redistribution.

So adapt:
- USN neurological examination form; or
- DAN Neurocheck.

DAN Neurocheck summary:
- Standardised and relevant
- Report generation
- Data collection and research tool
- DAN considering as global project (Cronje, South Africa)

Neurocheck challenges:
- Standardised clinical assessment perceived as research orientated and more than needed for management
- DCI dilemma:
  - inadequate classification system (but not necessary for triage?)
  - non-standardised clinical data
- May be too difficult for some locations where accidents happen
- Training required
- Computer (PC / PDA) support required

DCI EVALUATION ALGORITHM

What it is:
- Guide for hotline operators trained and expected to manage DCI cases remotely
- Guide to resource allocation:
  - type and speed of evacuation
  - destination
- System intended to optimise medical evacuation and minimise residua

What it is not:
- Replacement for clinical judgment or experience
- Guide to the DCI treatment per se
- Intended to eliminate necessary indications for recompression or evacuation

Value of a triage algorithm:
- Consistent (standardised) response
- Minimises variability related to experience
- Requires valid medical information
- Clarifies key issues
- Requires diving medical competency for its application
- Cost effective
- Reviews resource allocation
- Caveat: need for regional individualisation

6. Planning indications for aeromedical evacuation

- Severity (needs competent assessment)
- Stability
- Prognostic factors related to risk of a range of possible untreated outcomes
- Appropriate care
- Critical care / pressure requirements

7. In-water recompression and monoplace chamber on site

IN-WATER O_2 RECOMPRESSION

The theoretical basis for very shallow air compression in early cases is currently being reviewed (Brubakk, Norway). This consideration is confined to O_2 recompression:
- Are the equipment, training and other resources needed for an in-water O_2 recompression well defined?
- What is the ideal pO_2 (circa 1.6 bar)?
- Consider the pearl-diver experience from Western Australia.

WHEN TO USE PRE-PLANNED IN-WATER O_2 RECOMPRESSION?

- > 16 hrs prior to evacuation
- Sufficient O_2 supply
- Cooperative and fully conscious diver
- Harness or seat
- Diver tender
- Thermal protection provisions
- Full-face mask; communications
- No flush-through potential
- Controlled ascent

AVAILABLE CATEGORIES OF MONOPLACE CHAMBERS

- Clinical, fixed monoplace (for hyperbaric oxygen and diver treatments); hospital based
- Naval monoplace for SurD; not primarily for treatment
- Transportable monoplace chamber:
  1. Planned to be at dive site for USN6, e.g., remote scientific expeditions. For risk assessment and residual risk acceptance before departure
  2. Brought to site after incident for evacuation and possible lock-in to multiplace

- Transportable two-person chamber (brought to site after incident for evacuation)

8. Audit of recompression chamber (RCC) physical resources

RISK ASSESSMENT GUIDE AND MANAGEMENT EVALUATION

These are in relation to the operational environment.

LOCAL RESOURCES FOR RCC

Staff:
- Adequacy and needed scope of training

Equipment:
- Mixed gas treatment capability
- Life support capability
- Saturation treatment capability

9. Recompression options

Recompression and the importance of complete recovery before a return to work were discussed. Considerations include:
- If no immediate response on USN6, what options are currently being used?
- Some of these options need special resources. How available are these?
- If there is incomplete recovery on surfacing, what is the role of repetitive recompressions?

DIFFICULT TREATMENTS

Many different algorithms are in use around the world, all with apparent success. Tables, other than USN6, used by Phuket participants include:
- USN5; repeated
- USN Table 7
- USN Table 4 (e.g., if no O_2 available)
- USN Table 6A1M
- Comex30, Comex50 and other Comex tables
- Royal New Zealand Navy Table 1A (ex USN Table 1A with 50/50 HeO_2, then O_2)
- Saturation tables (various)
- Royal Navy Table 71 (ex-SETT schedule)

Other tables discussed but not used by any of the participants included the 'Hawaii Spike' (220 ft air excursion before USN6).

Evidence to support table selection is needed. There is also a need for standardisation. USN6, Comex30 and repetitive recompressions are commonly used, but there are many local differences in the depth/time and gas profiles. Any comparative analysis of results should note the various versions: e.g., USA; Cayman tables; several versions of Comex30, etc. Changes are mostly small but need to be specified.

NEED FOR EXPERIENCE AND SPECIAL RESOURCES

- Do not recompress beyond 18 m without being able to manage the complications and having full life support capabilities.
- First, discuss with others experienced in this field. If you have not done it before, do not jump in but rather review the protocol with care.
- Remember to consider the chamber attendant(s).

TREATMENT OF SURFACE-ORIENTED DEEP MIXED GAS DIVER

- Initially, follow algorithms for air and Nitrox divers.
- If unresponsive: Was the diver still on helium on, or just before, arrival at the surface?
  NB Beware of deterioration due to counter-diffusion during recompression if this is on air and consider need to switch gases.
- There is no evidence of effectiveness of any particular treatment compared with others (especially if surfaced from > 150 msw depth).

POST-RECOMPRESSION DYSEXECUTIVE SYNDROME

Although a medicolegal reality, its existence is unproven.
- Seems to follow some degree of cerebral DCI by several days; often not diagnosed at time of recompression; possible analogy to post-concussion effects.
- Delayed neuropsychological sequelae: cognitive dysfunction (memory, mood, decision making).
- Best defence: give full appropriate recompression with associated treatments using accepted procedures every time.

10. Management after surfacing: repetitive recompressions

HOW DO WE MANAGE RESIDUA?

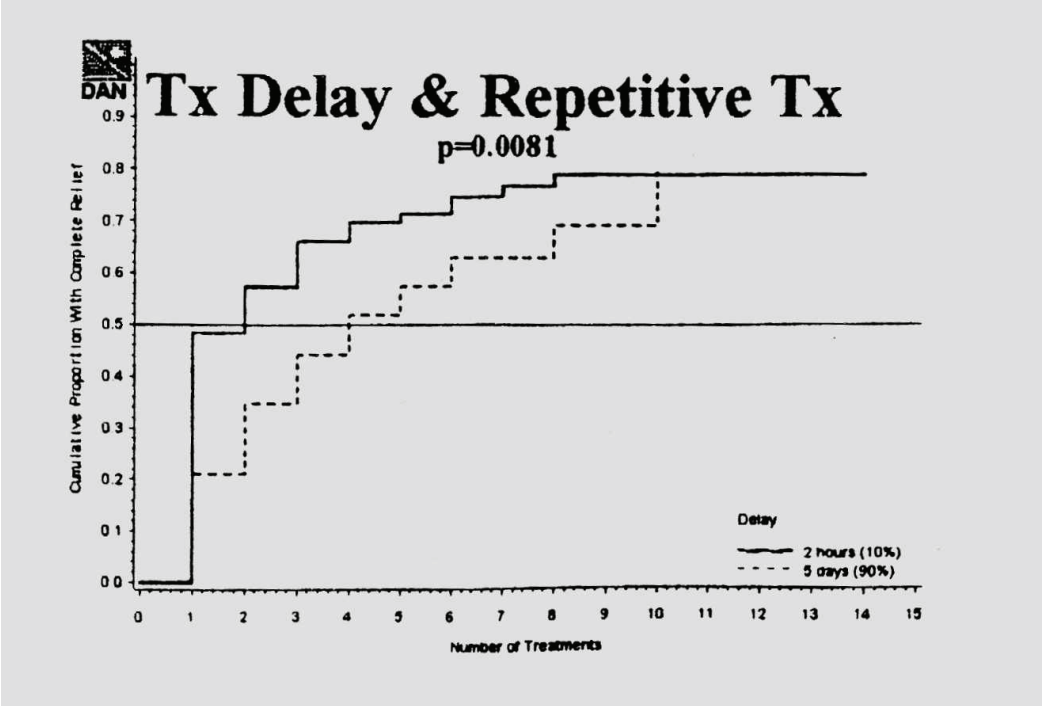
Review of 10 recompression centres in Europe and Australasia:
- No treatment
- Repetitive Comex12
- Repetitive 15 m x 90 min (USN Table9)
- Repetitive 18.60.30
- Repetitive 18 m USN5
- Repetitive 18 m USN6

All until a plateau is achieved.

REPETITIVE RECOMPRESSIONS

The tables used need to be standardised if not in compliance with already published or navy treatment tables. Repetitive recompressions can compensate for delay and may achieve full recovery in 80% of divers after both 2-hour and 5-day delays (Figure 1).[1]

Figure 1. The relationship between treatment delay and the number of repeat treatments needed to reach a clinical plateau[1]



11. Flying home after neurological DCI

PLANNING AND MANAGEMENT

No residua: three procedures are in common use (prospective study needed):

- 72-hour wait
- Prophylactic treatment; USN5s (or USN6; 18.60.30) plus 24- to 72-hour wait
- Three- to six-week wait (problem: insurers should pay hotel and some do, others do not)

Stable residua: Same approach is used, but the wait begins only after achieving a plateau symptomatically from repetitive recompressions.
Unstable residua: No flying or medical evacuation (without pressurisation).

MANAGEMENT ON DISCHARGE FROM RECOMPRESSION CENTRE

Following symptomatic relief of neurological DCI, what is the role of pre-flight

- treatment tables;
- repetitive recompressions;
- surface O_2

in the prevention of relapse?

CAUSES OF IN-FLIGHT / POST-FLIGHT RELAPSE

- Residual bubble growth (with further endothelial, platelet and leucocyte activation)
- Ischaemic penumbra
- Hypovolaemia

IN-FLIGHT OXYGEN

The goal is to maintain ground-level pO_2. There is a need to consider closed- vs open-O_2 delivery systems on aircraft? (see also para 3.) , and the acceptability of closed circuit to the various aviation agencies (FAA, CAA, airlines, etc).

12. Prospective data and follow up for analysis

HOW DO WE LEARN FROM OUR EXPERIENCE?

- Collect data, analyse and publish results
- Prospective, not retrospective studies
- Must define specific questions that the data should answer

Assessment of outcome immediately after treatment is available but how important is longer-term follow up after discharge? Problems of longer-term follow up include:

- questionnaires (by post, e-mail or telephone call) lead to low response rates and unvalidated data?
- appointments for an examination: who pays?

Conclusions from Workshop

These preliminary conclusions, above and below, are subject to examination of the records of the meeting and many will be reviewed at the UHMS Workshop to be held in Sydney, May 2004.

Urgent attention needs to be given to those items in italics:

- Omitted decompression and early reporting
- First aid (surface O_2) and local evacuation
- *Diagnosis and prognosis based on remote communication*
- *Triage of mild and severe cases*
- Aero-medical evacuation planning and criteria
- The role of in-water recompression and of a monoplace already on site
- Chamber and resources audit
- *Options if no immediate response to 18 m recompression*
- *Effectiveness of repetitive recompressions*
- *Protocol for flying home after neurological DCI*
- Collection of prospective data and follow up relating to specific questions

References

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3 Vann RD, Bute BP, Uguccioni DM, Smith LR. Figure 42. Prognostic factors in DCI in recreational divers. In: Moon RE, Sheffield PJ, editors. *Treatment of decompression illness, 45th UHMS Workshop*. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996.

Professor David H Elliott, DPhil(Oxon), FRCP, FFOM, OBE, is a life member of SPUMS and has been a guest speaker at a number of SPUMS Annual Scientific Meetings.
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Workshop participants

Australia
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Edward Bishop*
Michael Lavender*
Robert Long*
Bob Wong*

Canada
Michael Bourke

England and Scotland
Tim Callaghan
Margaret Clamp
David Elliott
Caroline Sheehan*
Graham Purdy*
John Ross*
Fiona Sharp*
Pippa Vickery

France
Christophe Dey*

Malaysia
Thomas Cheu*

Norway
Alf Brubakk*
Olaf Eftedal*

Saudi Arabia
John Luby*

Singapore
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South Africa
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Thailand
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Ljubisa Majic*
Wattana Pornkulwat*
Dulyakit Vittayahanapom*
Siriporn Vittayahanapom*

USA
Lorre Henderson
Klaus Torp*

*with responsibilities for recompression

SPUMS Journal CD

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

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

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

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

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 a medically qualified financial member of the Society.
2 The candidate must supply evidence of satisfactory completion of examined course(s) in Diving and Hyperbaric Medicine at an approved institution.
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 research proposal in a standard format for approval by the Education Officer before commencing their research project.
5 The candidate must produce, to the satisfaction of the Education Officer, 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. Preference will be given to reports of original basic or clinical research. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice (available at http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm). 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 Education Officer reserves the right to modify any of these requirements from time to time.

The Education Officer, Dr David Doolette, has recently resigned. Until a new Education Officer is appointed, all enquiries regarding the SPUMS Diploma should be addressed to Dr Michael Davis who, with Professor Des Gorman, is one of the remaining members of the Academic Board. Contact:

Dr Michael Davis, FRCA, FANZCA, MD,
SPUMS Journal,
C/o Office 137, 2nd Floor,
Christchurch Hospital, Private Bag 4710,
Christchurch, New Zealand.
E-mail: *<spumsj@cdhb.govt.nz>*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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ANZ College of Anaesthetists Certificate in Diving & Hyperbaric Medicine Examination

Applicants who did not meet the criteria for the award of the Foundation Certificate in Diving & Hyperbaric Medicine of the ANZ College of Anaesthetists by 30 June 2003 are advised that they may be eligible to present for the examination for the Certificate.

The criteria for examination are:

1 possession of a specialist qualification registrable in Australia/New Zealand
2 possession of DipDHM
3 minimum of 6 months' experience in anaesthesia
4 at least 12 months' *full-time equivalent* experience in diving and hyperbaric medicine in a hyperbaric department (accredited or to be accredited by ANZCA)
5 currently working in diving and hyperbaric medicine

Trainee registration (one off fee): $300.00
Examination fee: $500.00
Annual fee for certificate holders: $100.00
The interim regulation expires on 31 December 2004, after which time all candidates must meet the requirement of the SIG Workbook and the requisite training time.

Intending candidates are requested to contact
Ms Helen Morris at ANZCA for further information:
The Australian and New Zealand College of Anaesthetists
'Ulimaroa', 630 St Kilda Road
Melbourne, Victoria 3004, Australia
Phone: *+61-(0)3-9510-6299*

SPUMS diplomates

1974
G Bayliss
C Edmonds
R Gray
J How
C Lowry
A Slark
R Thomas
I Unsworth

1975
C Acott
J Chuan
G Dawson
J Knight

1976
C Macfarlane
S Maheson
H Mahdi

1978
J Anderson

1979
A Gebbie
G Lovell
B McKenzie

1980
N Barnes
B Turner

1982
M Hodgson

1987
C Dillon

1989
T Anderson
G Barry
R Capps
P Chapman-Smith
N Cooper
D Davies
M Davis
M Fraundorfer
J Gilligan
D Gorman
D Griffiths
C Kenny
D Kerr
P Laverick
M Loxton
B McCartney

1989 contd...
I Millar
R Moffitt
W Murtha
J Orton
M Osborne
H Oxer
A Robertson
P Robinson
R Schedlich
C Scheinkestel
R Stevens
C Strack
P Sullivan
A Sutherland
D Tuxen
A Veale
R Webb
J Williamson
R Wong
T Wong

1990
J Monigatti

1991
V Callanan
T Fallowfield

1992
P Mark

1995
C Butler
B Fitzgerald
T Francis
G Jelinek
S Mitchell
A Rasheed
D Smart
M Walker
R Walker
M Weinmann

1996
L Wheen

1997
M Kluger
M Skinner

1998
G Emerson
C Lee

2000
A Holley
M Bennett
D Vote
L Ekanayake
P Whyte

2001
A Waring
D Williams

2002
K Brown
R McKay

2003
J Leverment
J Lehm
B Trytko
D Wilkinson
R Harris

Minutes of the SPUMS Executive Committee Meeting held at the Prince of Wales Hyperbaric Unit, Sydney, on 25 October 2003

Opened: 1015 hr
Present: Drs R Walker (President), G Williams (Immediate Past-President), C Meehan (Secretary), M Davis (Editor), D Doolette (Education Officer), M Bennett, D Walker, S Mitchell (Committee Members), D Smart (ANZHMG Representative), A Patterson (coopted)
Dr Smart left the meeting 1330hr
Dr Patterson arrived 1100hr
Apologies: Dr B Trytko (Treasurer)

1 Minutes of the previous meeting (5 August 2003)
Moved that the minutes be accepted as a true record, after minor corrections.
Proposed Dr M Davis, seconded Dr D Doolette, carried.

2 Matters arising from the minutes

2.1 Improving our Internet cost effectiveness.
Awaiting confirmation of costs to have addresses using domain name. Update on costs for secure online payments – still pending. Dr R Walker will follow up with Dr B Trytko. Dr M Davis will follow up a lead in New Zealand who may be interested in web site development.

2.2 Update from Editor, Dr M Davis.
Lack of manuscripts from 2003 conference is an ongoing issue and causes problems. The official guest speaker does this as part of his contract. Other presenters need to endeavor to do this in a reasonable time frame.
Budget for 2004 needs to be updated.
Update on the status of the Journal office: the relationship with New Zealand Medical Association continues to be a positive one.
General feedback from Committee was that the first year of colour in the Journal was a success.
Request to purchase Endnote – approved.
Update on cost of binding one full set of journals.

2.3 Revision of Society rules.
The name of the Journal will change to 'Diving and Hyperbaric Medicine: The Journal of the South Pacific Underwater Medicine Society'.
Notification (of nominations, motions, etc) times to be adjusted in line with Journal issue publication dates.
The SPUMS Constitution needs updating. Dr R Walker, Dr M Davis and the Public Officer will

look at the Constitution and recommend changes. A call for nominations for Treasurer will go out with the December Journal.

2.4 Update on UHMS in Sydney by Dr M Bennett. All is going to schedule. There will be some additional advertising regarding the conference in the SPUMS Journal.

2.5 Update from Education officer, Dr D Doolette. There are an increasing number of candidates and associated workload. Dr M Davis will publish a list of the diploma recipients in the Journal.

2.6 Update from ANZHMG, Dr D Smart.
Medicare items discussion.
The course will be held at the Prince of Wales Hospital in March 2004.
The ANZHMG Committee will meet at the HTNA meeting in Sydney.
National database of problem wounds will be discussed.

3 Annual Scientific Meetings

3.1 2003 Palau update.
The financial report was presented and despite the reduced attendees all expenses were adequately covered. There was discussion regarding the need to update the conveners' manual in order to clarify some issues that were of concern in Palau. There was also discussion regarding whether the SPUMS liability insurance covers problems that may occur at the ASM venue.

3.2 2004 ASM update given by Dr Williams.
The conference will be held at Le Meridien, Noumea. There was some discussion as to whether PowerPoint should be upgraded to the latest version. There was also some discussion as to the need in future of updating the data projector.

3.3 2005 ASM.
The convener is Dr C Meehan. Borneo Divers, Mabul Island Resort, was suggested as a venue. However there seemed to be some concerns regarding the safety of nearby waters, and so travel to these areas is not recommended at present. Further research will go into looking at the facilities at Walindi, PNG. Other suggestions were Tahiti, and the Maldives.

3.4 NZ has been mentioned for a future ASM.

4 Treasurer's report

Update on candidates for Treasurer position. Dr Andrew Patterson joined the meeting. He has officially agreed to stand in until the AGM. Nominations for the position will be called for.

5 Correspondence

5.1 Dr John Knight has written to advise that he wishes to step down from the position of SPUMS representative on the Occupational Diving Committee. A replacement is sought. It has been suggested that a notice be placed in the next Journal regarding this. The Committee will then decide on a suitable candidate.

5.2 E-mail from Larry 'Harris' Taylor, PhD, Diving Safety Coordinator, U of MI <http://www.mindspring.com/~divegeek>. This seems to be an offer to contribute and maintain (validate once a month) a list of valid web links. Dr C Meehan will find out further details.

6 Other business

6.1 General discussion on SPUMS policy on training of hyperbaric/diving physicians led by Dr Doolette.

6.2 University of Auckland PGDipMedSc – Diving and Hyperbaric Medicine (Dr M Davis).
Dr Davis gave an overview of the web-based academic course. All three members of the Academic Board involved with this, with Dr Davis as Course Director. The course is planned to commence in part in the second semester of 2004, and will run in full from 2005.

6.3 Dr M Davis has offered to resign from Academic Board at the end of 2004 if the Committee considers conflict of interest with above requires this.

6.4 CME points – clarification of position of various colleges is being sought.

Closed: 1500hr

Notice of Annual General Meeting of SPUMS to be held at Le Meridien Hotel, Noumea, 5 June, 2004

Agenda

Apologies:

Minutes of the previous meeting:
Unratified minutes of the previous meeting will be posted on the meeting notice board and appeared in the *SPUMS J.* 2003; 33: 171.

Matters arising from the minutes:

Annual reports:
President's report
Secretary's report
Education Officer's report
Presidents' Committee report

Annual Financial Statement and Treasurer's Report:
Proposal regarding subscription fees for 2005.

Election of office bearers:
Nominations have been called for the position of Treasurer.

Appointment of the Auditor:
Proposal regarding appointment of auditor for 2004.

Business of which notice has been given:
1. Changes to the SPUMS Statement of Purposes and Rules. The following motions have been proposed.

Motion 1
Purpose 4
Existing: To promote communication between members of the Association and to publish a newsletter for the Association.
Proposed amendment: Replace the word *newsletter* with *journal* to read

> To promote communication between members of the Association and to publish a journal for the Association.

Explanation: A journal rather than a newsletter has been published for many years.

Proposed Dr R Walker, seconded Dr M Davis

Motion 2
Rule 12 (a)
Existing: The Secretary of the Association shall at least 42 days before the date fixed for the holding of any annual general meeting of the Association and at least 14 days before the fixed date for the holding of any special general meeting of the Association, cause to be sent to each member of the Association at his address appearing in the register of members, a notice by prepaid post stating the place, date and time of the meeting and a description of the purpose and a summary of the business to be transacted at the meeting.
Proposed amendment: Replace the number *42* with *21* and *his address* with *their address* to read

> The Secretary of the Association shall at least 21 days before the date fixed for the holding of any annual general meeting of the Association and at least 14 days before the fixed date for the holding of any special general meeting of the Association, cause to be sent to each member of the Association at their address appearing in the register of members, a notice by prepaid post stating the place, date and time of the meeting and a description of the purpose and a summary of the business to be transacted at the meeting.

Explanation: This brings the timing of notice of the Annual General Meeting in line with the publication of the March issue of the Journal which does not usually reach members until some time in April.

Proposed Dr C Meehan, seconded Dr M Davis

Motion 3
Rule 22 (a) & (c)
Notice of motion published in *SPUMS J*. 2003; 4: 226.

Proposed Dr M Davis, seconded Dr J Knight

Motion 4
Rule 26
Existing: The Secretary of the Association shall keep minutes of the resolutions and proceedings of each General Meeting and each Committee Meeting in books provided for that purpose together with a record of the names of persons present at the Committee Meetings. A copy of the minutes of the proceedings of all meetings of the Committee shall be incorporated in the next edition of the Journal for the information of all members of the Association.
Proposed amendments: Delete the words *in books provided for that purpose*, and add the word *ratified* before *copy of the minutes* to read

> The Secretary of the Association shall keep minutes of the resolutions and proceedings of each General Meeting and each Committee Meeting together with a record of the names of persons present at the Committee Meetings. A ratified copy of the minutes of the proceedings of all meetings of the Committee shall be incorporated in the next edition of the Journal for the information of all members of the Association.

Explanation: Specific books have not been kept for many years, the record of all meetings being the same as that published in the Journal. On occasions, minutes of Committee Meetings have been published in the Journal prior to their being ratified by the Committee.

Proposed Dr C Meehan, seconded Dr R Walker

Motion 5
Rule 28 (b)
Existing: Any cheque, draft, bill of exchange, promissory note or other negotiable instrument may be signed by the Treasurer.
Proposed amendment: Delete the words *by the Treasurer* and replace by the words *jointly by at least two approved signatories* to read

> Any cheque, draft, bill of exchange, promissory note or other negotiable instrument may be signed jointly by at least two approved signatories.

Explanation: Having all payments by a professional society countersigned by at least two appointed persons is customary practice and enhances financial security for the society.

Proposed Dr R Walker, seconded Dr A Patterson

Motion 6
Rule 29
Existing: This standing committee will be composed of life or ordinary members who have served at least one year as the President of the Society. The Committee will meet at the Annual Scientific Meeting of the Society, at the members' expense, and at the same time as the Executive Committee at one other time during the year, at the Society's expense. The Presidents' Committee will also be able to conduct telephone conferences. Chairmanship of the Committee will be the responsibility of the Immediate Past-

President and minutes will be kept by members in rotation. The Presidents' Committee will answer directly to the current Society President and be responsible for the development of actual and draft Society policy on issues identified by the Society. The Presidents' Committee will report its activities in the Society Journal and provide an annual report to the Society at the Annual Scientific Meeting.

Proposed amendment: In the second sentence replace the word *will* with *may*, and also after the word *meet* insert the phrase *as authorised by the Executive Committee* to read

> This standing committee will be composed of life or ordinary members who have served at least one year as the President of the Society. The Committee may meet as authorised by the Executive Committee at the Annual Scientific Meeting of the Society, at the members' expense, and at the same time as the Executive Committee at one other time during the year, at the Society's expense. The Presidents' Committee will also be able to conduct telephone conferences. Chairmanship of the Committee will be the responsibility of the Immediate Past-President and minutes will be kept by members in rotation. The Presidents' Committee will answer directly to the current Society President and be responsible for the development of actual and draft Society policy on issues identified by the Society. The Presidents' Committee will report its activities in the Society Journal and provide an annual report to the Society at the Annual Scientific Meeting.

Explanation: Since it was formed at the ASM in New Zealand in 1998, the Presidents' Committee has met only once and no reports have been submitted to the Society. A majority of the present Executive Committee felt that, despite this, the Committee should be retained as it may have a potential role for the Society in the future.

Proposed Dr G Williams, seconded Dr R Walker

Motion 7
Rule 39

Existing: A Journal to be known as the "SPUMS Journal" or other such name as may be adopted by the Association in general meeting, shall be produced and distributed to all members of the Association. Income from subscriptions may be applied by the Committee for such publication.

Proposed amendment: Replace the words *the "SPUMS Journal"* with the words *"Diving and Hyperbaric Medicine: The Journal of the South Pacific Underwater Medicine Society"* to read

> A Journal to be known as "Diving and Hyperbaric Medicine: The Journal of the South Pacific Underwater Medicine Society" or other such name as may be adopted by the Association in general meeting, shall be produced and distributed to all members of the Association. Income from subscriptions may be applied by the Committee for such publication.

Explanation: The present Executive Committee is of the view that the international reputation of the Society's Journal will be enhanced by a change in name, and that this is important for its long-term viability.

Proposed Dr M Davis, seconded Dr R Walker

Motion 8
Rule 40

Existing: The Committee may coopt or appoint such persons to an editorial board for the SPUMS Journal as it deems necessary to assist the Editor. The Editor shall be responsible for its publication and shall be Chairman of the Editorial Board. The Editor shall have regard to the view, if any, expressed by the majority of the Committee as to editorial policy and publishing of material.

Proposed amendment: Subject to adoption of the motion amending Rule 39, replace the words *the SPUMS Journal* with *Diving and Hyperbaric Medicine: The Journal of the South Pacific Underwater Medicine Society* to read

> The Committee may coopt or appoint such persons to an editorial board for the journal Diving and Hyperbaric Medicine: The Journal of the South Pacific Underwater Medicine Society as it deems necessary to assist the Editor. The Editor shall be responsible for its publication and shall be Chairman of the Editorial Board. The Editor shall have regard to the view, if any, expressed by the majority of the Committee as to editorial policy and publishing of material.

Explanation: This brings Rule 40 in line with the proposed change to the name of the Journal in Rule 39, provided such name change is approved by the membership.

Proposed Dr M Davis, seconded Dr R Walker

2. Notice of motion

"That the Committee instruct the organisers of future Annual Scientific Meetings of the Society to ensure separation of costing of the Meeting so that, in future, costs of travel and /or accommodation to be paid to an Agency appointed to serve the members shall be set out, and paid-for, separately from the costs of the Meeting, and that, where access to diving or other activities may be limited, allocation shall be made in the order of conference registration, rather than given preferentially to members booking accommodation via the Travel Agency."

Proposed Dr J Marwood, seconded Dr G Long

3. **Matters regarding the appointed conference travel agents**

Dr H Turnbull (notice given on 26 February 2004).

Letters to the Editor

Recreational diving fatality statistics

Dear Editor,

In my commentary on children and scuba diving,[1] I made mention of Monaghan's fatality statistics for recreational scuba diving, indicating 16.7–18 deaths per 100,000 divers.[2,3] He is not alone in this assessment, demonstrating a significant death rate well in excess of that sometimes promoted by the recreational diving industry.

The British Sub-Aqua Club have averaged 15 deaths per 100,000 divers over the last two decades and also during the year 2000.[4] Japanese data indicate 17.5 per 100,000.[5] PADI, in its Australian manual, states that there is no specific trend up or down in scuba deaths, but that PADI certified divers, thereby excluding those under training, have a death rate of 2.1 per 10,000 (21 per 100,000).[6]

The statistics can be made to look better than they are by over-inflating the denominator and multiple counting of those divers with multiple certifications. The diving industry has total control over the supply of certification numbers and has been forced to revise downwards these denominators in the past. To overcome this effect, investigators now measure the death rate per dive, as was also done by Monaghan. The rationale for this was recently argued in this journal.[7]

The death rate per dive has been found to be 3.0 (West coast of USA), 2.05 (Canada) and 2.9 (UK) per 100,000 dives.[7–9] If one assumes that the average diver dives 10 times per year (less than the figure of 15 usually used), the death rate per diver is between 20 and 30 per 100,000.

Perhaps Monaghan was too conservative. Scuba is a beautiful but hazardous sport.

Carl Edmonds
Manly, NSW, 2095, Australia
E-mail: *<puddle@bigpond.net.au>*

References

1 Edmonds C. Children and diving: a review of SPUMS articles. *SPUMS J.* 2003; 33: 206-11.
2 Monaghan R. Australian diving death rates comparison with USA and Japan. *SPUMS J.* 1989; 19: 24-5.
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5 Ikeda T, Ashida H. Is recreational diving safe? *Undersea Hyperb Med.*. 2000; 27(suppl): 138.
6 Morgan J. In: *PADI Diving Accident Management in Australia*. Australia: PADI, ISBN 09588055 0 4; 1988. p. 53.
7 Ladd G, Stephan V, Stevens L. The Abacus Project: establishing the risk of recreational scuba death and decompression illness. *SPUMS J.* 2002; 32: 124-9.
8 Hattori T. Review of diving casualties on the Monterey peninsula. Personal communication and update on *NAUI Proceedings of 8th International Conference on Underwater Education.* 1976; 146-50.
9 Hart AJ, White SA, Conboy PJ, et al. Open water scuba diving accidents in Leicester. *J Accid Emerg Med.* 1999; 16: 198-200.

Key words
Letters (to the Editor), deaths, recreational diving

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The Poetry Doctor

John Parker

Many Society members will know Dr John Parker from his books on diving medical assessment. The Editor, following Dr Wilkinson's effort in the last issue, asked John if he would contribute regularly a poem on diving. In return, he has asked that I draw readers' attention to his web site <www.thepoetrydoctor.com>.

Mind Bends

When I go diving I feel I'm reviving
By finding such wonder and peace.
I flee my life's stresses and recurring duresses
In this wonderful, watery release.
Whilst exhaling my bubbles, I blow out my troubles
To surface and leave me alone,
Whilst I stay descended, suspended, up-ended
Relaxed in a worryless zone.
I drift with the flow, my thoughts deep in tow,
Freed to roam in the stillness,
But will my thoughtful descent cause my mind to get bent
And suffer "contemplation illness"?

Book reviews

Divers in time: Australia's untold history

Jeff Maynard

160 pages, soft cover
Yarraville, Victoria: Glenmore Productions; 2003
Available from Glenmore Productions, 16 Hood Street, Yarraville, Victoria 3013, Australia
Phone: (+61) 3 9687 2109; **Fax:** (+61) 3 9687 4368
E-mail: <info@glenmoreproductions.com.au>
Copies can be ordered online at:
<www.glenmoreproductions.com.au>
Price: AU$40.00, postage and packing extra

This very readable, well-researched history of the early days of commercial and recreational diving in Australian waters reveals some of the human story of those not-so-long-ago times. The largely forgotten but important Australian input into the development of modern diving is chiefly remembered now by the graves of the pearl divers. Few know of the work of Ted Eldred, who bypassed the Gagnan/Cousteau patents on the twin-hose 'Aqualung'® by inventing the single-hose regulator we accept today, or that the name 'scuba' was thought up to avoid the (copyright) title 'Aqualung'®. In true but unfortunate Australian inventors' tradition, Eldred never gained credit or fortune from his work. Neither did Victor Brege, whose imprisonment by the Japanese allowed his patent to expire, although his mask was widely used.

There are interesting asides that tell much of the author's interest in his subject and about some of the players. One such is the brief mention that the Siebe Gorman Company destroyed all the records of both the Deane brothers and the Heinke Company after they were subsumed into it.

The development of diving has involved brave men facing dangers none understood, commercial interests with no apparent feeling of responsibility for their employees, and inventors of diving suits whose ignorance of the basic physical factors affecting divers failed to inhibit their efforts. One can note the sad case of John de Noury diving in Clifford's "Invention of the age" suit. The pearl-diving industry story includes the deaths of the first (English) hard-hat divers associated with the mix of 'White Australia' and lesser expense of employing Japanese divers, and the later triumph of technology politics, which enabled hookah divers to beat the latter in pearl-shell collection.

There is much, much more in this book, such as the Royal Australian Navy and Police divers and those very special men who dived in World War Two. It is hoped that the author will sometime make available the tales he has undoubtedly collected from the many 'old timers' he talked to in preparation for writing this book. It is hoped also that people are preserving not only the old equipment but old diving magazines. To truly understand history we need the words of the forgotten contemporaries as well as the ones with monuments in their memory.

Douglas Walker
E-mail: *<diverhealth@hotmail.com>*

Key words
Book reviews, history, scuba, general interest

Investigating recreational and commercial diving accidents

Steven Barsky and Tom Neuman

236 pages, softcover
ISBN 0-9674305-3-4
California: Hammerhead Press, Ventura; 2003
Available from Hammerhead Press & Hammerhead Video, 2419 E. Harbor Blvd. #149, Ventura, California 93001, USA
Phone: (+1) 805 985 4644; **Fax:** (+1) 805 382 6755
E-mail: <info@hammerheadpress.com>
Copies can be ordered online at <www.hammerheadpress.com>
Price: A$48.95, postage

The back cover reads "Investigating Recreational and Commercial Diving Accidents is a must read for anyone who investigates recreational, technical, professional or commercial diving accidents...It is an important reference for anyone who is professionally employed in the diving business..." So much promised, not much delivered.

It is well presented with good black-and-white illustrations and contains an index and bibliography. However, each chapter is not referenced and the bibliography short, with only 27 references, 25 from the United States. The content of the book is confusing. Is it about investigating diving fatalities, diving-related morbidity, decompression sickness incidents or other issues involved with diving? One cannot be sure. For example, there are highlighted, boxed areas scattered throughout the book entitled "Risk recognition for divers and dive professionals" containing witticisms concerning diving safety issues but not about the investigation of diving accidents. Chapter 10 concerning non-diving accidents (relating to chambers, compressors etc) is relevant, but it also includes sexual harassment issues; hardly a diving accident!

This book must have been written for the American market as it uses the Imperial system of measurement and refers, albeit briefly, to the regulations governing recreational,

scientific and commercial diving in the United States only. There is a small section on the most common diving accident sites, all in the United States with the exception of Cozumel, in Mexico, a popular recreational diving area for American divers.

Scattered throughout the book are unsubstantiated statements such as: "The loss or failure of a mask should not be a cause for panic for the properly trained diver" (page 38), or "...a properly trained or supervised diver should never die due to entanglement in kelp" (page 136). These statements are not valid as accidents do happen no matter how well the diver is trained. Incident reporting and selected cases from 'Project Stickybeak' illustrate this.

Case histories are used to emphasise points, some are good, others not so, such as the case history (page 23) discussing a diver on the antidepressant venlafaxine (Efexor) that fails to mention the adverse reaction of loss of consciousness. There is also confusion in one case history between decompression and recompression (page 91).

Chapters 3 and 4, concerning the physiology (and pathophysiology) of diving and diving equipment and their relationship to diving accidents, are inadequate. Hypothermia (page 79) and drowning (page 85) are particularly poorly done. There is no mention of cognitive processes slowing with hypothermia or an inability to do anything due to shivering or perhaps muscle rigidity. Hypothermia is often regarded as the 'insidious killer' in diving, and this is not mentioned. Drowning could have been summarised by emphasising that drowning is not a diagnosis – a cause needs to be found.

Chapter 5 entitled "Talents and characteristics of the dive accident investigator" could best be described as 'padding'. Chapter 6 entitled "The tools of the dive accident investigator" discusses laptop computers, scanners, digital cameras (versus film), personal organisers, colour or inkjet printers and voice recorders. These topics are dealt with better in any computer or other media magazine. Chapters 13 and 14, concerning the preparation of a report or legal testimony and commercial diving accidents, were informative, but don't buy the book for these two chapters alone. One very obvious omission is that there are no suggested checklists or 'aides-mémoire', which are essential for any accident investigation.

I cannot recommend this book.

Chris Acott
Hyperbaric Medicine Unit, Royal Adelaide Hospital

Key words
Book reviews, investigations, diving, accidents

NOAA diving manual: diving for science and technology, fourth edition

James T Joiner (ed)

668 pages, hardback
ISBN 0-941332-70-5
Flagstaff, Arizona: Best Publishing Company, 2001
Available from Best Publishing Company, P O Box 30100, Flagstaff, Arizona 86003-0100, USA.
Phone: (+1) 928 527 1055; **Fax:** (+1) 928 526 0370
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Amongst the missions for the National Oceanic and Atmospheric Administration (NOAA) is the conservation and management of the oceans, the United States's coasts and the Great Lakes. NOAA has the largest diving complement of any civil federal agency in the US, and probably the world, with more than 300 men and women. This number does not include all those civilian scientists, engineers and technicians who dive under the auspices of NOAA-sponsored research grants.

Whilst living in the USA in the mid 1970s I was fortunate to obtain a copy of the first edition of the NOAA Diving Manual, published in 1977, and for many years used it as a guide to diving activities and a resource for teaching. In preparing the fourth edition, the editor writes, the foremost objectives were to provide guidance on safe diving practices under varying experimental and environmental conditions, using different breathing gases and systems, and to introduce the myriad scientific and technical developments that have occurred since the last manual update a decade earlier. Because of the multidisciplinary nature of diving, over a hundred people and organisations from many fields either contributed to or reviewed sections of the manual. Whilst aimed principally at NOAA's divers, the manual is also intended to be useful for other working and recreational divers, and it achieves its goals well.

Weighing in at over 3 kg, this glossy, large-format publication is certainly not one to stuff in your dive bag or in the cuddy-cabin of a runabout, but would accompany most dive teams into the field as an important resource. There are 21 chapters and 10 appendices. Each chapter or section is divided into numbered subsections for each topic, and these contents are laid out on a separate page at the beginning of each chapter. This format, combined with a comprehensive index, make consulting the manual for specific items easy. For instance, diving from a ship whilst under way is found in section 12.17.4 (in two subsections on equipment and communications), on pages 12-31 to 12-34 of the chapter on "Diving under special conditions".

The first five chapters on history, physics and physiology,

air diving, decompression and diving support equipment are similar to many scuba diving manuals. The physics chapter, in particular, is succinct and well illustrated. I was less sure of the physiology section, particularly some of the diagrams. Deciding what physiology to teach and how to present it to lay divers is in my opinion never easy. Diving scientists, in particular, pick up on and are often frustrated by simplistic explanations consisting of half-truths, but there are plenty of physiology and medicine references in the bibliography for them to follow up. Oxygen toxicity appears in detail both here and in the later chapter on nitrox diving.

The chapters devoted to surface-supplied diving, nitrox and more advanced mixed-gas and oxygen diving are comprehensive, but that on rebreathers only brief. This mixture of general information and very detailed procedural instructions occurs elsewhere as well, and probably reflects those techniques commonplace within NOAA's dive teams at the time of publication (2001). Interestingly, NOAA uses an oxygen partial pressure limit of 1.6 atmospheres absolute rather than 1.4 as is widely used in recreational diving.

Of particular interest were the chapters on procedures for scientific and working dives, and on diving under special conditions. There is a complete chapter on diving in polluted waters. Since NOAA has always been involved in habitat diving, it is not surprising that there are two chapters on seafloor habitats, including decompression schedules from saturation. At the time of publication the habitat *Aquarius 2000* was still in operation in the Florida Keys. The final three chapters are devoted to hazardous aquatic animals, emergency medical care and accident management.

The appendices include field neurological assessment and recompression treatment tables, the NOAA no-decompression air and nitrox dive tables and the full US Navy Standard Air Decompression Table 1999. Finally, there is a useful glossary of terms and an extensive bibliography containing several hundred references.

No single book that tries to cover so much ground can satisfy all divers' needs but this one combines useful general knowledge with considerable detail of those techniques in wide use within NOAA. Being an American publication all measurements are in imperial units, but most have the metric equivalent alongside. The many illustrations, graphs and tables are mostly of high quality. However, the relevance of some photos was dubious whilst others were of out-of-date equipment.

This is a non-partisan diving manual with a broad sweep of information on the diversity of marine environments, tools and methods for working divers. The NOAA diving manual remains an excellent publication.

Michael Davis, Editor, SPUMS Journal

Key words
Book reviews, occupational diving, textbook

Hyperbaric Medicine 2003

Dick Clarke, Principal Moderator

The Ninth Annual Advanced Symposium, April 24-26, 2003, Columbia, S. Carolina
National Baromedical Services, 5 Richmond Medical Park, Columbia, SC 29203, USA.
Five four-hour sessions on DVD or Video (NTSC or PAL)
Price: US$80 per DVD or Video; US$400.00 for complete set, or combined DVD and Video set US$650.00

The Undersea and Hyperbaric Medical Society, Palmetto Health, Richland, and the University of South Carolina (USC) School of Medicine were joint sponsors of the Ninth Annual Advanced Symposium. The entire meeting was digitally recorded and CME credits approved for US-registered hyperbaric physicians, nurses and technicians.

Out of respect, the course was dedicated to Dr Jon Mader who sadly died earlier this year.

The course coordinator was Mr Dick Clarke, well known to anyone familiar with the international hyperbaric scene, an inspirational man with seemingly limitless energy. During the introduction, one speaker referred to Mr Clarke's input to the course as reflecting his "passion" and "style". Mr Clarke would undoubtedly reply that it is not about himself, but about the many people who have contributed.

The talks covered the entire field of diving and hyperbaric medicine. The faculty sparkled like a jewel, boasting a list of world leaders, and many seemed genuinely honoured to be asked to participate. The quality of the talks was extremely high. I feel I must make a comment on computer-generated talks. What looks good on a computer screen is not always the same when projected in an auditorium. There were examples of font size too small for easy reading and inappropriate choice of colour contrasts.

On a production note, two cameras at the rear of the auditorium captured the digital recording. The resolution of the image on the big screen is not perfect and lends further weight to my comments about computer-generated talks. Obviously production and editing have been kept to a minimum for cost purposes. Maybe consideration could be given to some enhancement, such as directly importing the computer image onto the recording, or indexing the talks to allow easier navigation around the DVDs.

Interspersed between the talks were a further four hours of recording, mainly dedicated to question-and-answer sessions, with several brief 'spotlights' thrown on people in the audience who have made significant contributions to the field of hyperbaric medicine. The fact that they are still turning up to such events is a tribute to the course and to their own lifelong commitment. This is a positive contribution that recognises a grand history of the evolution of scientific thought and serves to build a sense of fraternity.

The symposium has been divided into five sessions. Session 1 is a detailed review of the basic sciences as our knowledge currently exists on how hyperbaric oxygen (HBO_2) works at the cellular level. Session 2 describes several specific clinical topics. Sessions 3, 4 and 5 are evidence-based analyses. Each speaker was given an approved indication from the list supported by the UHMS and asked to summarise that topic, review the literature and assess the data using some classification for level of evidence. The fact that there are some 25 different classification systems does not facilitate the evidence-based process; fortunately only a few different systems were used.

Session 1 Basic science (165 min)

Dr Jon Buras – Mechanisms of HBO_2 in sepsis (45 min)
Investigation of the mechanisms of sepsis represents a new direction for this emergency medicine physician and basic science researcher. This topic is of great interest to the general medical research field as well as those in the hyperbaric field. Millions of dollars must have been spent on designing drugs to interrupt a process that is not yet fully understood. Dr Buras presented an excellent summary of the processes we believe are involved in sepsis. Conclusions were difficult to draw from a host of animal studies using HBO_2 due to conflicting results. Unfortunately the evidence is limited to in vitro and animal studies.

Dr Wende Reenstra Buras – Mechanisms of HBO_2 in wound healing (30 min)
A fundamental hyperbaric topic with a review of wound-healing processes. The talk also summarised the in vitro work reported by these two researchers of growing stature. Discussed were evidence of cell dysfunction associated with diabetes and age, growth-factor-receptor upregulation with HBO_2, and the observation that intracellular signalling is measurably altered after only 15 minutes' exposure to HBO_2.

Dr Joseph Boykin – Nitric oxide: endothelial dysfunction (25 min)
This plastic and reconstructive surgeon is an eminent researcher and authority in the wound-healing field. Nitric oxide (NO) appears to be a modulator of activity at several points in the repair process. Dr Boykin described the need to balance superoxide production, scavenging mechanisms and NO production. Endothelial dysfunction, which has been a hot topic in the cardiac field, was implicated in chronic wounds. Current research was discussed, seeking an index of NO bioactivity and using it in the clinical decision-making process.

Dr Bruce Cameron – When leucocytes go bad (30 min)
Dr Cameron is a researcher from the same group as Dr Thom and gave a talk the content of which was at the cutting edge of knowledge. A level of leucocyte activity appropriate for health can be overstimulated by a variety of events resulting in damage and dysfunction. Cellular processes that mediate these events were summarised and points at which HBO_2 appears to influence the process were described. We are talking cellular and molecular biology. Certainly not for the faint-hearted, the less-experienced hyperbaricist will probably find this talk hard going.

Dr Frans Cronje – Pathophysiology of DCI (35 min)
Dr Cronje is a well-respected South African physician. This was a thorough, slick talk suitable for the newest recruit to diving medicine be they doctor, nurse or technician, but still offering interest to those with more experience.

Session 2 Clinical updates (145 min)

Mr Dick Clarke – Hyperbaric literature update (55 min)
Mr Clarke performed an extensive review of the literature for recently published work covering the entire field of hyperbaric medicine. He identified 766 papers, focusing on about 70 of these. The talk was succinct, insightful and engaging. As commented by the moderator, Mr Clarke condensed two months of reading into one hour.

Dr Dirk Bakker – Treatment of advanced neuroblastomas (30 min)
There is renewed interest in the use of HBO_2 as a radiosensitiser, following on from work reported in the 1970s but subsequently put to one side. Neuroblastomas were discussed and Dr Bakker reported some of his own work involving HBO_2 and other adjunctive therapies.

Dr James Holm – Hyperbaric risk management (40 min)
This is a topic we might rather not hear about but which is important. The talk was a comprehensive discussion of medicine and the law, covering both general medicine and hyperbaric issues. It was specific to the USA but there are many obvious similarities to medicolegal concerns worldwide. Comfortingly, some strategies were offered to improve the situation with a risk management programme.

Dr Cuauhtemoc Sanchez – Optimising patient sedation (20 min)
Dr Sanchez is a well-known, engaging personality on the international hyperbaric scene. His institution treats many acute cases and the need to place a patient who is early in the course of their disease into a monoplace chamber frequently necessitates sedation. This is a difficult clinical task to perform safely and Dr Sanchez reported good results with use of a bispectral index (BIS) monitor, which records a frontal electroencephalograph signal and transforms it into a linear scale, from 0-99. He reported its use in conscious sedation and for intubated, ventilated patients.

Session 3 Evidence-based analysis of hyperbaric indications (130 min)

Dr Gosta Granstrom – Osteoradionecrosis (60 min)
Dr Granstrom, from Sweden, is a widely published researcher and surgeon in the field of radiation-affected bone. This topic could be considered one of the best-defended indications for HBO_2 from an evidence-based perspective. Considerable time was devoted to outlining

his search strategy and describing two different systems for classification of the level of evidence. Each paper was given a code from the two classification systems and if not completely familiar with the classification systems, reference during the talk to only the code could lead to some confusion. This is only a minor criticism of a thorough analysis. In particular I noted his opinion that assessment of outcome following radiation to bone can only be made after long-term follow up, with seven to eight years being the minimum suggested time frame.

Dr John Fieldmeier – Soft-tissue radionecrosis (50 min)
From another leader in the field, this was an easy-to-listen-to, comprehensive review. A range of sites may be affected by radiation injury; differences in anatomy and function preclude simply adding all the results together, meaning the evidence for any one particular site was limited. Emphasis was on head and neck and bladder sites. The evidence ranged from controlled human studies to case reports. While the gold standard randomised controlled trials (RCTs) do not exist, confidence was expressed by the consistently positive nature of the results. Dr Fieldmeier's experience reinforces the need for an adequate number of HBO_2 treatments to achieve good outcome. Near its end, this talk was cut off in midsentence at the end of the disc.

Dr James Williams – Soft-tissue radionecrosis in gynaecological sites (20 min)
Dr Williams is a gynaecologist with special interest in gynaecological oncology. His brief but succinct talk focused only on soft-tissue sites in the gynaecological domain. There was limited evidence to report.

Session 4 Evidence-based analysis of hyperbaric indications (185 min)

Dr Richard Moon – Decompression sickness and CAGE (65 min)
Using the depth of the DAN database, Dr Moon described the rationale and evidence for oxygen use in first aid and hyperbaric oxygen treatment. He comprehensively covered adjunctive therapies with levels of evidence for each.

Dr Bob Warriner – Problem wound healing (55 min)
This talk dealt mainly with diabetes, which is not surprising as most published hyperbaric studies have been on diabetic wounds. The same weight of evidence does not exist for non-diabetic wounds despite the assumed similarity in pathophysiology: that of local tissue hypoxia. Evidence for transcutaneous oximetry was discussed and how it might be used, although it was recognised that hypoxia has not been adequately validated as an indication for HBO_2. Technology assessments performed by other agencies and government bodies were examined. Dr Warriner runs a very successful wound centre in Conroe, Texas, and considerable practical advice on 'how we do it' complemented his talk.

Dr Harold Friedman – Grafts and flaps (45 min)
This experienced plastic surgeon from the USC School of Medicine explained the different flaps and grafts used in a way that was both detailed and understandable. Emphasis was given to his pet technique of composite grafts (grafts containing soft tissue and cartilage) suggesting HBO_2 may be of particular benefit. Almost all of the evidence was derived from animal studies with very few human data. Unfortunately this was an accurate representation of the literature. He gave valuable insights into the surgical approach to this topic, and provided interesting explanations of therapies other than HBO_2.

Dr Frank Voss – Chronic refractory osteomyelitis (CRO) (20 min)
Dr Voss is an orthopaedic surgeon with USC School of Medicine, not a practising hyperbaric physician. His perspective as a surgeon added great value in a well-planned talk, beginning with the basics and working up. It was sad to realise that the evidence for any form of management of CRO is based almost entirely on case series - one controlled human study using HBO_2 was given short consideration due to a perception of inadequate surgical debridement. Many clinical case series were presented, with and without use of HBO_2, and other adjunctive therapies were discussed. His suggestions as to directions for the future were thoughtful and appreciated.

Session 5 Evidence-based analysis of hyperbaric indications (200 min)

Dr Ben Slade – Acute thermal burns (45 min)
Dr Slade now works at the same institution as Dr Paul Cianci. San Pablo, California, is notable as an institution where surgeons do actually consider HBO_2 in the clinical management of acute thermal burns. Burden of disease and mechanism of injury were discussed. Many animal studies were presented, almost all supportive of HBO_2. Unfortunately the many different surrogate outcomes reported in the results made it difficult to draw any meaningful conclusions. The local protocol for burns was described, in which HBO_2 is used on clinical grounds.

Dr Dirk Bakker – Gas gangrene (40 min)
Dr Bakker is a distinguished surgeon from the Netherlands whose name has become synonymous with his topic. Aetiology was reviewed, along with mechanisms of pathogenesis and a discussion of the many toxins involved. The rest of the talk was an opportunity to learn from his accumulated clinical series spanning 40 years. It was not a formal evidence review. No RCTs exist, although Dr Bakker related his personal experience with applications to several ethics committees to perform an RCT, which were rejected by them all as unethical - in 1984! This talk contained many clinical 'pearls'.

Dr J Jeffrey Brown – Necrotising soft-tissue infections (40 min)
Dr Brown is a general surgeon and hyperbaric physician. After a brief historical review he summarised the basic sciences, pointing out problems with nomenclature and how

to classify this condition. He described a number of mechanisms by which HBO_2 may be utilised as an adjunctive therapy, and considered their relative importance. A review of the literature produced a plethora of case series and four relatively recent controlled studies. The data are not entirely clear but similar to those for the other treatment modalities utilised for this condition. Determination of prognosis is limited by what appears to be unpredictably different rates of progression of disease. He offered an interesting theory to explain these data.

Dr Lindell Weaver – Carbon monoxide poisoning (45 min)
Dr Weaver is another luminary in the field of hyperbaric medicine. He discussed burden of disease, aetiology and pathophysiology – at least what we know at present. He provided evidence that outcome from poisoning and development of neurological sequelae is still unpredictable. The bulk of the talk was a detailed explanation of Dr Weaver's recent RCT.[1]

Dr Lisardo Garcia-Covarrubias – Crush injury: acute ischaemias (30 min)
Dr Garcia is a surgical resident and hyperbaric physician. He discussed how the Advanced Trauma Life Support (ATLS) programme outlined a standard of care for arterial and skeletal extremity injury; this did not mention HBO_2. The clinical evidence was discussed, detailing the one RCT for HBO_2. No consensus could be found and this indication continues to be one for further research.

General comments

Overall, seeing this material has been very enjoyable and I have learnt much. I did feel as if I was there, live. An advantage of this format compared with actually being there is that the talks can be listened to repeatedly. The information will become dated, but no more quickly than a textbook. I was impressed by the wealth of information generated by research into HBO_2, but can see much work is still required to convince the general clinician of today. Challenging our beliefs by thinking critically about the information presented at this symposium may yet lead us towards the truth. To not undertake such a challenge may, as only the Americans can express, leave us 'well behind the power curve'.

For those not active in hyperbaric practice but with an interest, there is much to be gained by viewing this symposium. For practising hyperbaric physicians, it is a responsibility to keep knowledge up to date. If you cannot get to the Tenth Advanced Symposium in April 2004 (which will cost more) this is certainly a rewarding alternative.

Dr David Wilkinson
With specific input from Dr Piers Robertson, Dr Ed Bishop and Dr Michael Heytman.
Hyperbaric Medicine Unit, Royal Adelaide Hospital.

Reference

1 Weaver LK, Hopkins RO, Chan KJ et al. Hyperbaric oxygen for acute carbon monoxide poisoning. *New Engl J Med*. 2002; 347; 1057-66.

Key words
Video reviews, hyperbaric oxygen, meetings

SPUMS 2004
Annual Scientific Meeting
Noumea – New Caledonia

Venue - Le Meridien Noumea
30 May – 6 June 2004
(meeting to run 1 – 5 June inclusive)

Themes
Marine stingers and marine envenomation
Modelling decompression tables

Guest Speakers
***Dr Peter Fenner**, AM, James Cook University, Queensland*
***Dr David Doolette**, PhD, Adelaide University, South Australia*

Convener
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P O Box 190, Red Hill South, VIC 3937, AUSTRALIA
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For further information or to enrol contact:
The Director, Hyperbaric Medicine Unit,
Royal Adelaide Hospital, North Terrace,
South Australia 5000.
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207

WORKSHOP ON REMOTE MANAGEMENT OF MILD DCI
MAY 25, 26, 2004, SYDNEY, AUSTRALIA

The management of DCI in remote locations where hyperbaric facilities are not available is complicated by the need for costly and logistically demanding evacuations. There is a growing body of expert opinion that mild or marginal cases may be as well served by local treatment with surface oxygen, fluids, and drugs followed by non-emergent evacuation. These issues will be discussed during this UHMS Workshop by a body of experts with the objective of developing consensus guidelines for managing mild DCI in remote locations.

Co-chairs Drs. Simon Mitchell and Richard Vann. Co-editors Dr. David Doolette and Chris Wachholz, R.N.

Continuing Medical Education Units have been applied for in the US and Australia. Attendance fees for the two-day workshop is AUS $350.

For further information contact the Undersea and Hyperbaric Medical Society (UHMS). Phone +301-942-2980; e-mail uhms@uhms.org www.uhms.org or ww.iceaustralia.com/uhms2004/.

JAMES COOK UNIVERSITY AND ANTON BREINL CENTRE FOR PUBLIC HEALTH AND TROPICAL MEDICINE

Tropical marine and diving medicine (TM5508:03)
Location: Townsville
Availability: Semester 2, External subject with compulsory Limited Attendance Residential
(18 October - 22 October 2004)
Staff: Associate Professors G Gordon and P Leggat
Cost: $Aust 1,260.00
Overview: This subject, conducted in association with the Townsville Hospital, presents the basic principles of underwater and hyperbaric medicine over a five-day residential programme. A series of lectures, demonstrations and practical sessions highlight the taking of a diving history, performing a diving medical examination, decompression sickness, management of near drowning, hypothermia, sinus and ear barotrauma, diving equipment, gases, physiology, diving hazards and diving techniques. A hyperbaric unit is located at the Townsville Hospital. Optional dives may be conducted.

For further information or to enrol contact:
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James Cook University,
Townsville, Queensland 4811,
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E-mail: <sphtm-studentofficer@jcu.edu.au>

EUROPEAN UNDERWATER AND BAROMEDICAL SOCIETY
30TH ANNUAL SCIENTIFIC MEETING

Dates: September 15 to19, 2004
Venue: Ajaccio, Corsica
Convenor: Dr Bruno Grandjean
Congress organisation: Atout Corse
1, rue Saint Roch,
20000 Ajaccio, France
Phone: +33-(0)495-225293
Fax: +33-(0)495-511040
E-mail: <eubs2004@wanadoo.fr>
Web site: <www.eubs.org>

10TH INTERNATIONAL CONFERENCE ON EMERGENCY MEDICINE (ICEM 2004)

Dates: 6 to 10 June, 2004 (follows SPUMS ASM)
Venue: Cairns Convention Centre, Queensland
Web site: <www.icem2004.im.com.au>
Contact: Conference Secretariat, Intermedia Convention and Event Management, P O Box 1280, Milton, Queensland 4064, Australia
Phone: +61-(0)7-3858-5535
Fax: +61-(0)7-3858-5510
E-mail: <icem2004@im.com.au>

THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND HEALTH SCIENCES

Postgraduate Diploma Medical Science – Diving and Hyperbaric Medicine

Applications are now being accepted from registered medical practitioners for the Postgraduate Diploma Medical Science – Diving and Hyperbaric Medicine in the Faculty of Medical and Health Sciences, the University of Auckland. The Diploma can be completed in one year or spread over two years part time. Three of the six papers listed below will become available in the second semester of 2004, whilst the remainder will come on line during 2005.

This Diploma is designed as a distance learning programme, available internationally and without a resident component in Auckland. However, some courses include attendance at a recognised residential course in diving and hyperbaric medicine available within Australasia or attachment to a hyperbaric medicine unit. 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.

The paper titles are:
- Health surveillance of divers and hyperbaric workers
- Physiology and medicine of diving *
- Hyperbaric medicine
- Clinical diving and hyperbaric practice
- Essay in diving and hyperbaric medicine *
- Project in diving and hyperbaric medicine *

(* courses available from 2nd semester 2004)

For further information, including fees, please contact the Course Coordinator: Jessica Rorich
Phone: +64-(0)9-373-7599, extn. 88489
Fax: +64-(0)9-308-2379
E-mail: <occmed@auckland.ac.nz>
Full information on courses and admission regulations is available in the University of Auckland Calendar or at <http://www.auckland.ac.nz>

MEDICAL AND LEGAL CONFERENCES 2004

Australian Medico-legal Conference
Dates: 8 to 15 August 2004
Venue: Perisher Blue, NSW

Pacific Rim Medico-legal Conference
Dates: 25 September to 2 October 2004
Venue: Heron Island, Great Barrier Reef
Conference Director: Lorenzo Boccabella
E-mail: <conference@barweb.com.au>
Web site: <barweb.com.au>

We are now an endorsed provider of CME for the Royal Australian College of General Practitioners.

Instructions to authors

(Revised March 2004)

The *SPUMS Journal* welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to the *SPUMS Journal*, 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:
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Requirements for manuscripts
Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Word 97 for Windows. If submitted as a paper version, two printed copies of all text, tables and illustrations should be mailed. 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.

Paper versions and electronic files should be double spaced, using both upper and lower case, on one side only of A4 paper. Headings should conform to the current format in the *SPUMS Journal*. All pages should be numbered. Underlining should not be used. Measurements are to be in SI units (mm Hg 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 reviews. 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 be less than 40 words in length.

Illustrations, figures and tables should not be embedded in the wordprocessor document, only their position indicated. All 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 on separate sheets of paper. No vertical or horizontal borders are to be used. Illustrations and figures should be separate documents in JPEG or TIFF format. Please note that our firewall has a maximum size of 5Mbytes for incoming files or messages with attachments.

Photographs should be glossy, black-and-white or colour. Slides should be converted to photographs before being sent. Colour reproduction is available only when it is essential for clinical purposes and may be at the authors' expense. Indicate magnification for photomicrographs.

Abbreviations should only be used 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.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples of the format for quoting journals and books 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.

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

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DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

The DES numbers are generously supported by DAN-SEAP

PROJECT STICKYBEAK

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. Information, all of which is treated as being **CONFIDENTIAL** in regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organisation to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence) to:
Dr D Walker
PO Box 120, Narrabeen, NSW 2101, Australia.

DIVING INCIDENT MONITORING STUDY (DIMS)

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

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

PROJECT PROTEUS

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

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

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

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