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SPUMS

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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

OFFICE HOLDERS

ADMINISTRATION

MEMBERSHIP

Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
Membership application forms can be downloaded from the Society's Web Site at *http://www.SPUMS.org.au*

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

The Society's financial year is January to December, the same as the Journal year.
The 2003 subscriptions 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

Decompression illness (DCI) is a clinical diagnosis based on the diving history and physical examination of the diver. The use of various diagnostic or screening tests has been assessed over the years. These include haematological changes, complement activation, precordial doppler, somato-sensory evoked potentials and various radiological procedures as each new technology is introduced, computer tomography, magnetic resonance imaging and so on. In DCI cases with subtle or mild symptoms and signs, none of these tests are of much value. In divers with severe injuries, the diagnosis is obvious from clinical evaluation and the changes in some of these various tests, if any, tend to mirror changes in the clinical signs, and so are not particularly helpful in determining ongoing management.

The eye has often been described as a window into the brain, and this may also prove true in DCI victims. Michael Bennett's and others' work on ocular tear bubbles as a measure of decompression stress following scuba diving has looked promising. Tear bubble counts rise following various dive patterns, including single dives and multi-day repetitive diving. However, no control subjects who were not diving were used in these studies, so it was not known whether similar changes occurred in swimmers and snorkellers. Kirsteen Brown now addresses this issue in the first article, which is based on her study for the SPUMS diploma. Further research on this screening test is clearly justified.

Every reader who has travelled abroad has probably suffered at some time or another from traveller's diarrhoea (TD). One notable SPUMS ASM in Fiji had half the participants if not on their knees then at least sitting on the loo for a considerable part of the week! Continuing the theme of travel medicine from the 2002 ASM, Trish Batchelor in the latest of her articles discusses TD, which is by far the most common health problem travellers experience wherever they go, or whatever they are doing.

Two short case reports highlight other risks of tropical diving holidays. Whilst eosinophilic meningitis is an uncommon infestation in travellers, perhaps the most important lesson is the difficulty health services back in the traveller's home area have in sorting out such problems when they have limited expertise in tropical or diving medicine. The second case reminds us of one of the many classes of stinging animals in the marine environment. I hope the Diving Doctor's Diary will become a regular educational component of the journal, which clinicians can use to report interesting or unusual cases, or as a case-based educational feature.

Five inter-related papers complete the publications from the drowning workshop at the 2001 meeting in Madang. Read together, they convey well the range of topics covered, and identify some obvious areas of clinical and applied research for the future. Whilst an uncommon phenomenon, acute pulmonary oedema in divers presents pathophysiological and diagnostic challenges.

SPUMS' current policy requires all presenters at annual scientific meetings of the Society to submit manuscripts of their talks before or at the time of the meeting for publication in the Journal. There are two problems with this; members tend only to pay lip service to this requirement, making the Editor's life difficult, and some members of the Executive Committee see this 'demand' as a barrier for those who might otherwise be prepared to talk at our meetings. Clearly, the two issues are closely linked. The new Editor will continue to 'enforce' Society policy, but (he hopes!) without making himself too unpopular, until such time as the Society directs otherwise. Personally, I consider it an excellent exercise in intellectual self-discipline that speakers should approach with courage and enthusiasm! Most importantly, however, the publication of a journal worthy of indexing in the world literature would not be possible without some of this material.

In the previous issue, Simone Taylor and her colleagues reported on medications usage by experienced sport divers in the USA and Australia. In the Australian wing of that study, information was also retrieved on divers' medical history. David Taylor's recent report in *Wilderness Medicine* on these results is reproduced in this issue. Their health survey in divers has identified some important issues and unexpected findings. If our approach to pre-diving medicals is to be robust, we need better information on the health status of the current diving population. Clearly further work is needed in this area. Project Proteus (see back cover of this issue) is one approach to collecting this type of information.

The 2003 ASM will focus strongly on pre-dive medicals. Perhaps no single issue in diving medicine is so hotly debated, and everyone has their own perspective on the matter. To assist members in thinking about this important issue, the most commonly used forms of medical assessment in the Australasian region will be published with the next two issues of the journal. We start with the new 2001 revision of the Recreational Scuba Training Council Medical Statement and Guidelines. This form is used world-wide by some of the largest sport diving training agencies, and is endorsed by the Undersea and Hyperbaric Medicine Society and several prominent members of SPUMS.

It is likely that some members of the Society have been affected directly or indirectly by the Bali bombings. Our sympathies go out to those of all nationalities with friends or family who were involved in some way, and especially to the Bali community whose home has been violated by this senseless act. One direct effect of this event for SPUMS is the re-routing of the travel itinary for next year's ASM in Palau, as outlined in the Notices section.

Diving accident surveys

Accompanying this issue is the newly revised form for the Diving Incident Monitoring Study (DIMS), run by Dr Chris Acott. Please photocopy this, keep copies with your dive gear and give them to your dive buddies, local dive training agencies and boat skippers. I encourage you to use them.

DIMS is modelled closely on the Anaesthesia Incident Monitoring Study (AIMS). A similar hyperbaric (HIMS) study exists, and both of these, as well as DIMS, are run internationally from Adelaide. In a variety of undertakings, incident monitoring – the reporting of 'near misses'– has proved a useful tool in identifying patterns of events and important components of their occurrence, recognition, and management in order to avoid or mitigate adverse effects.

In diving, we have some way to go in 'closing the loop', that is, in applying the knowledge gained from surveys like DIMS and Project Stickybeak in order to reduce the risks of diving. For instance, Guy Williams in his paper states "Divers using modern equipment properly maintained should not run out of air. If divers are running out of air, and they are, there is something wrong with how they are trained." His statement is clearly supported by the international diving accident and fatality data. It presents a challenge to the sport diving training industry to look at new and innovative methods of teaching in-dive gas management to prevent out-of-air situations.

As well as DIMS, Project Stickybeak, internal agency systems such as PADI's and the international DAN database, most countries have their own, usually voluntary, reporting systems. For instance, in New Zealand, decompression sickness is a Notifiable Disease, like tuberculosis or malaria, and, by law, cases must be reported. We are also asked to report diving accidents to New Zealand Underwater, whilst Water Safety NZ maintains a national drowning database.

There are several problems with these systems. First, except for the legal requirement above, none imply statutory obligations on the part of the reporter or the data collector. Second, reporting is voluntary or the search may even be generated by the data collector. Third, effective collection and recording often depends on the enthusiasm of individuals (Walker for Project Stickybeak, Acott for DIMS, McAniff for USA data in the 1970s and 1980s). Fourth, all are incomplete due to the inherent difficulties of capturing all the data, or because the survey was only ever designed as a population sample. Fifth, there is no standardisation of data handling and, finally, the lessons learned are not often applied to diving practice and equipment design.

This is not to say that the collection of mortality and incident data is not of value. Clearly we need to know what is happening before we can remedy problems. Examples of what is possible are PADI's abandonment of bouyant free ascent training as a result of their own internal accident survey, and the introduction by the Cave Diving Association of Australia of a training and certification system to reduce the Mount Gambier sinkhole fatalities in South Australia. Perhaps it is time for the various individuals and organisations involved to pool their efforts to create a unified reporting system for diving incidents, accidents and fatalities. A SPUMS workshop would be an ideal forum.

Michael Davis

SPUMS Journal CD

The South Pacific Underwater Medicine Society has produced a CD, readable by at least Windows and Macintosh computers, containing Volumes 1 - 30 of the Society's Newsletter and Journal as Adobe.pdf documents. To read and print these documents use Adobe Acrobat Reader (version 3 or later) which can be downloaded free from the Adobe web site <www.adobe.com>. Supplies are limited.

None genuine without this label

The Index is also supplied as a downloadable tab-separated document, which can be entered into the reader's database. It is in .rtf (rich text format), Windows 97 .doc and .txt for Windows file formats. Macintosh formats are .rtf and Word for Mac 5.1.

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

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Original articles

Ocular tear film bubble counts in swimmers and snorkellers

Kirsteen E Brown, Michael H Bennett and Annabel Dominguez

Key words
Recreational diving, snorkelling, swimming, tear film bubbles, decompression illness, diagnosis

Abstract

Background: The number of ocular tear film bubbles is increased following compressed air single- and multi-day diving in both sea water and hyperbaric chambers. However, the effect of sea water immersion and shallow breath-hold diving on the tear film has not been studied.
Methods: We examined the ocular tear films of 20 volunteers before and up to 72 hours following 30 minutes' exercise swimming or snorkelling in sea water.
Results: Both groups had a baseline median bubble count per eye of 0.33 (interquartile ranges, 0–1 and 0–0.9 respectively). There was no significant rise in the median bubble count following either exercise.
Conclusions: Our findings suggest that the observed increase in ocular tear film bubble counts in recreational compressed air diving is indeed related to decompression stress, and unrelated to the salt water environment per se.

Introduction

Despite the accumulating knowledge about the process of de-nitrogenation during decompression and its role in decompression illness (DCI), there still remains no test to confirm a diagnosis of DCI. Clinicians must rely on dive profile, clinical history and examination. In 1878, Paul Bert first postulated the existence of nitrogen bubbles forming in the blood and tissues following rapid decompression. Venous gas bubbles can be detected using precordial doppler ultrasound and this technique has been extensively used to assess decompression stress and validate dive tables. Venous bubbles can be detected within minutes of surfacing from a dive, reach a peak between 1 and 3 hours and are not detectable after 5 hours.[1] Unfortunately, the transient nature of venous bubbles limits their usefulness as a diagnostic test. Not only is there poor correlation between intravascular bubble counts and the presence of clinical DCI, but the majority of divers present for assessment and treatment long after venous bubbles have dissipated.[2] In one recent report, the average time from causative dive to presentation at a hyperbaric medicine unit was greater than 24 hours.[3]

The search for a diagnostic tool has led to the investigation of the ocular tear film. Although intra-ocular bubbles after decompression were first noted by Boyle in 1670, no particular clinical significance had been attributed to bubbles in the eye. Recently, a number of reports have been published suggesting that ocular tear film bubbles may relate directly to decompression stress.[4–8] However, no data exist for swimmers and snorkellers exercising in sea water. The present study has been undertaken with individuals exposed to the marine environment, in the absence of compressed air breathing, in order to establish counts for ocular tear film bubbles following marine exposure alone.

Materials and Methods

This study was undertaken at the Hyperbaric Medicine Unit, Prince of Wales Hospital, Sydney, and was approved by the South Eastern Sydney Area Health Service Research and Ethics Committee prior to volunteer enrolment.

Poster advertisements requested experienced adult swimmers and snorkellers in general good health to volunteer for participation in the study. Volunteers were excluded if they gave any history of ocular tear film dysfunction or ophthalmic disease (excepting refractory errors), or if they had undertaken compressed air diving or breath-hold diving to a depth greater than two metres in the four days prior to the study. Diving was prohibited for three days following the in-water exercise. Twenty volunteers were recruited following an explanation of the study including the slit-lamp examination procedure.

Following recruitment, a baseline examination of the ocular tear film was made with a standard slit-lamp (SL900, Haag-Streit, Switzerland) using our established protocol.[4] The volunteer subject closed their eyes for five seconds prior to each examination. The average bubble count was obtained from three sweeps of the inferior gutter of the ocular tear film, from medial to lateral (against the tear flow, which occurs from the lacrimal gland to the lacrimal canaliculi). Only bubbles in the gutter itself were counted, as small bubbles on the eyelid are not uncommon after blinking.

Volunteers were then randomly assigned to either the swimming or snorkelling group by means of a random number generator, with 10 people in each group. The swimming or snorkelling exercise was performed in enclosed sea water (either a sea pool or sheltered bay) for 30 minutes, pace being determined by the individual swimmer. To avoid the discomfort associated with swimming in salt water, the swimmers wore goggles. Snorkellers wore half-face masks and used a standard snorkel. The snorkellers were also required to perform several shallow breath-hold dives during their snorkelling exercise to a maximum depth of three metres. A member of the research team trained in adult resuscitation supervised the in-water exercise, and a public lifeguard patrolled the swim site.

The second tear film examination was performed within four hours of the in-water exercise, and at 12, 24, 48 and 72 hours thereafter. All participants completed the six examinations.

Two studies performed with recreational divers using scuba equipment have previously been completed in the Prince of Wales Department of Diving and Hyperbaric Medicine.[4] In the first study, 42 single-day divers performed a dive to 25 metres for 25 minutes with a five minute safety stop. In study two, 11 divers underwent repetitive, multi-day diving exposures over five days. The median bubble counts at 24 hours for these two groups of divers were compared to the those at 24 hours of the swimmers and snorkellers.

STATISTICAL ANALYSIS

Volunteers were recruited as a convenience sample and no power calculation was made. As the results were not normally distributed, non-parametric methods were used (Shapiro-Wilks W test). Median tear film bubble counts per eye were obtained and compared using the Friedman test for multiple comparisons (a non-parametric equivalent of ANOVA). The Mann-Whitney U test was employed for individual comparisons between groups and time points. All calculations were made using StatsDirect statistical software, version 1.611, Iain Buchan, 2000.

Results

All swimmers and snorkellers completed the 30-minute exercise in salt water, either in a sheltered bay or sea pool.

FIGURE 1
MEDIAN BUBBLE COUNTS PER EYE IN SWIMMERS (AVERAGE OF THREE SWEEPS)
(error bars represent interquartile ranges) * significant difference from pre-swim (p <0.05)

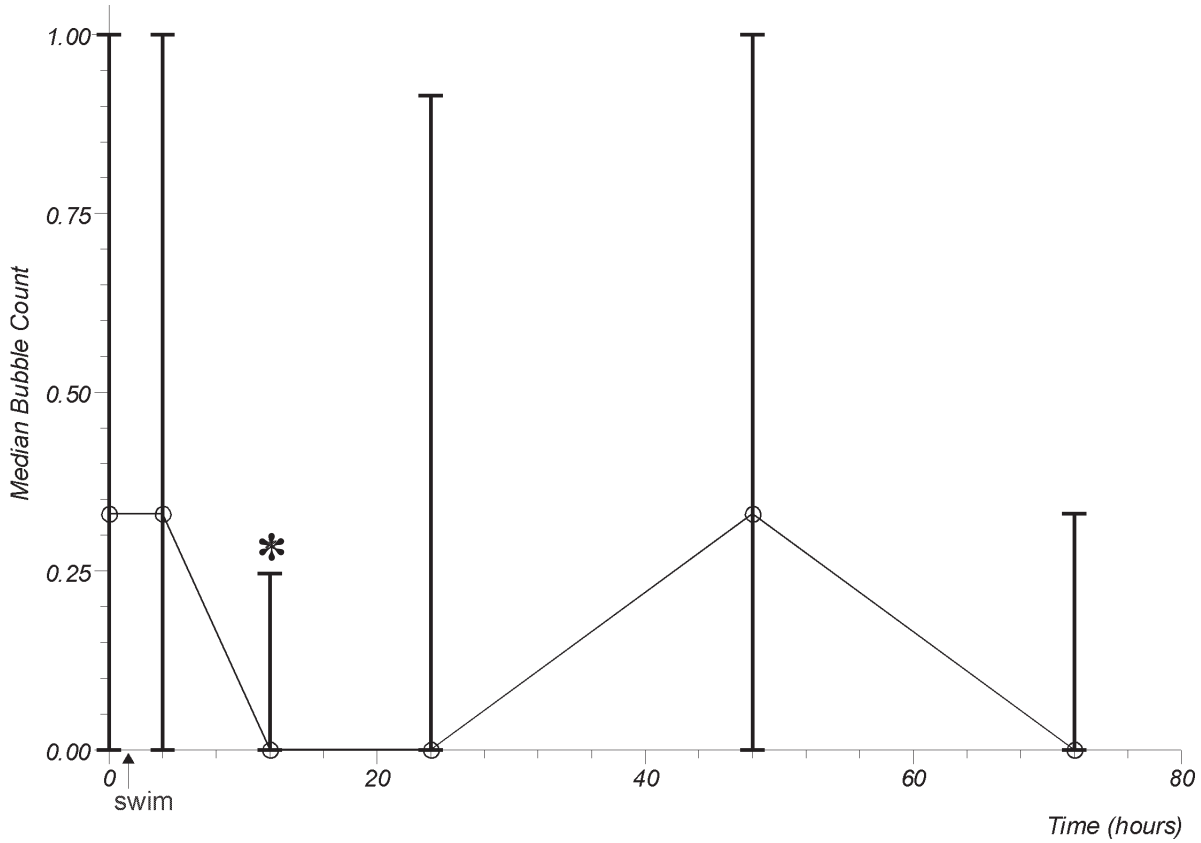
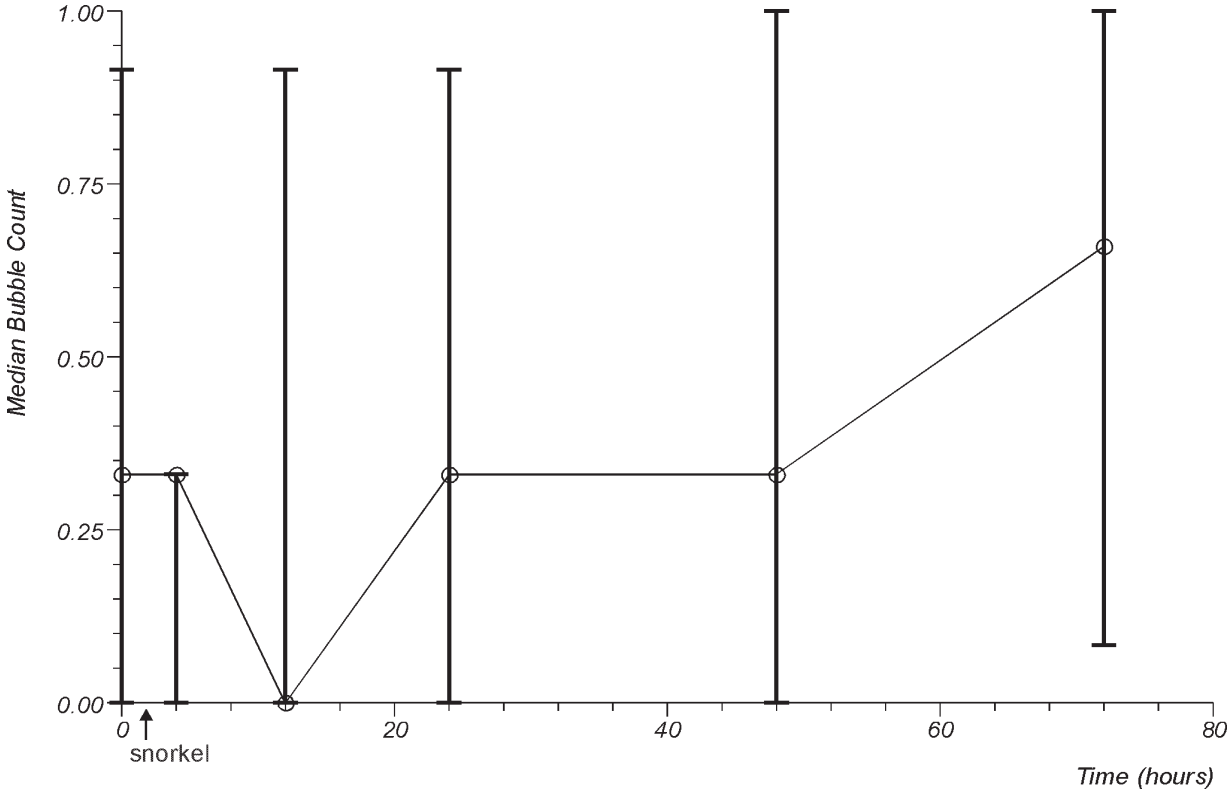


FIGURE 2
MEDIAN BUBBLE COUNTS PER EYE IN SNORKELLERS (AVERAGE OF THREE SWEEPS)
(error bars represent interquartile ranges). There were no significant differences between readings in this group



In the swimmers, the mean age was 29 years (SD 8.35 years, n = 10). The mean age of the snorkellers was 31.9 years (SD 6.88 years, n = 10). There were five female swimmers and four female snorkellers. Three people wore soft contact lenses while swimming and two while snorkelling. There was one smoker in each group.

The swimmers had a baseline median bubble count per eye of 0.33 with an interquartile range of zero to 1.0. The median bubble count at 12 hours was significantly lower than the baseline (Count: zero; $p < 0.02$, Mann-Whitney U test), but with no significant change from the baseline at all other times (Figure 1).

The snorkellers also had a baseline median bubble count per eye of 0.33 with an interquartile range of 0 to 0.915. Bubble counts did not change significantly for any examination time in this group (Friedman test, $p > 0.3$). The highest median bubble count per eye at any time (0.66) was noted in the snorkelling group at 72 hours (Figure 2).

One member of each group demonstrated unusually high counts. These two were the only subjects to ever have bubble counts of more than 3.0 per eye. The swimmer demonstrated high counts on three occasions with median bubble counts of 6.0 at 24 hours, 4.0 at 48 hours and 3.33 at 72 hours. The snorkeller had a bubble count in one eye of 4.6 prior to undertaking the in-water exercise and counts of one or greater on three further occasions.

Median bubble counts per person were also calculated. In both groups the highest median bubble count was 0.5 (range of 0 to 1.33). When median bubble counts per person were compared, no significant differences were seen for any time point (swimmers $p > 0.2$, snorkellers $p > 0.5$), or when the two groups were compared ($p > 0.2$). Only 12.5% of the total readings showed a tear film bubble count greater than one bubble per person; with 11 subjects (55%) never having an average count greater than one bubble at any time.

The median bubble counts per eye for the swimmers and snorkellers have been compared to those previously obtained by Bennett et al for single- and multi-day divers (Figure 3).[4] The time point of 24 hours was chosen to reflect the average time of first presentation at a hyperbaric unit for medical assessment.

INTER-OPERATOR VARIABILITY

Four slit-lamp operators were involved in the readings, although two investigators performed 96% of the examinations. Investigator 1 (KB) performed 56.6% of the

readings and Investigator 2 (AD) 39.2%. Both investigators counted a median of 0.33 bubbles per person.

Discussion

Extra-ocular tear film bubbles were first noted under both hard and soft contact lenses after diving and later in the eye of a hyperbaric chamber attendant not wearing contact lenses.[5] Since this observation, research data have been accumulating to indicate that extra-ocular tear film bubbles may be a measure of decompression stress.

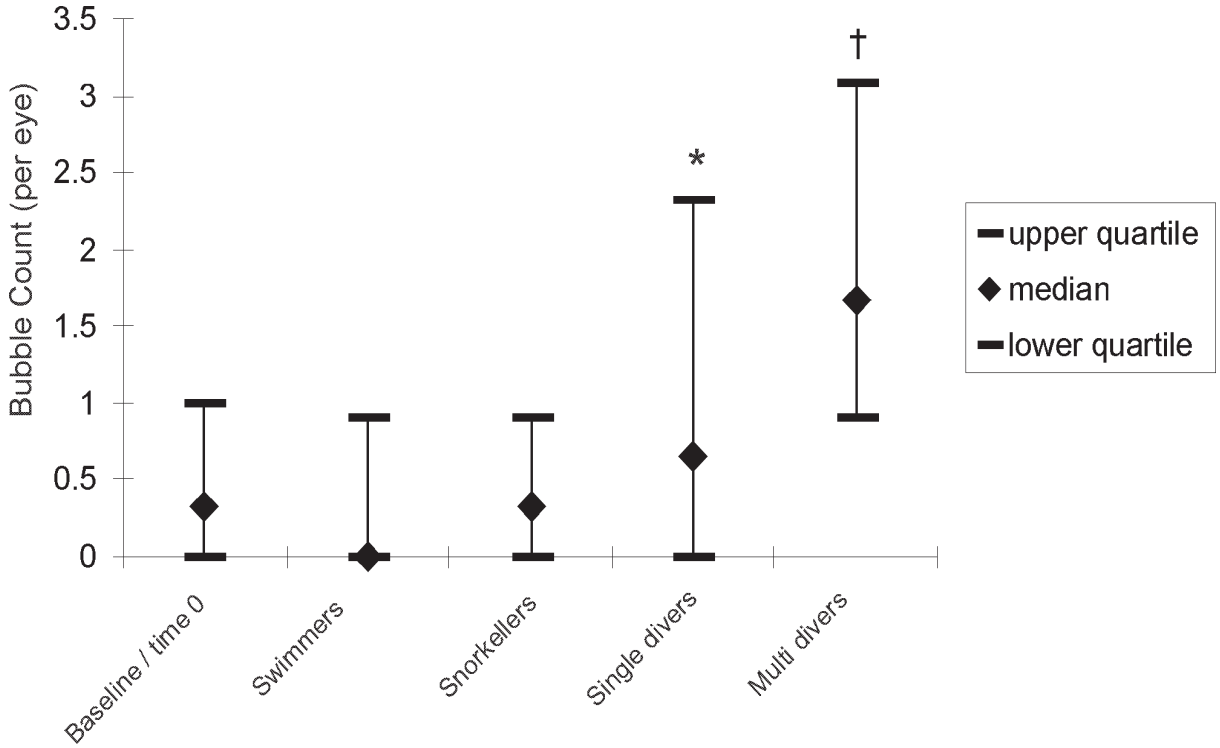
Tear film bubble counts were increased when volunteers underwent dry hyperbaric chamber air breathing dives[6,7] but not oxygen breathing dives.[8] A similar increase in ocular tear film bubble counts was noted following open water compressed air dives in the Prince of Wales studies.[4] This is not matched in the present study by swimmers and snorkellers exercising in the marine environment.

Tear film bubble counts from the swimmers and snorkellers were compared with the divers' counts at 24 hours post-exercise or dive, a time when patients commonly first present to a hyperbaric unit for medical assessment with symptoms of decompression illness.[3] Median bubble counts in the swimmers were significantly lower than both the single- and multi-day divers at 24 hours. The snorkellers were also significantly different from the multi-day divers but not the single-day divers ($p > 0.05$). The difference between the snorkellers and single-day divers may have reached significance with a larger sample.

The tear film bubble counts at baseline in this study are very similar to those in the previously reported studies in recreational divers.[4] This study, therefore, further confirms the low baseline median count and range that we can expect in the general diving population. The failure to demonstrate any significant change from baseline following swimming and snorkelling compared with diving, strengthens the case for a causal relationship between compressed air diving and increased tear film bubble counts.

Although dry chamber dives produced significant increases in bubble counts after only 15 minutes of exercise,[7] in the present study an exercise period of 30 minutes was chosen to provide a meaningful comparison with the Prince of Wales studies in compressed air divers.[4]

FIGURE 3
MEDIAN BUBBLE COUNTS AND INTERQUARTILE RANGES AT 24 HOURS FOR SWIMMERS, SNORKELLERS AND DIVERS[4]
baseline shows pooled data for median and interquartile ranges at time zero
*** significant difference from baseline and swimmers ($p < 0.02$)**
† significant difference from baseline, swimmers ($p < 0.0001$) and snorkellers ($p < 0.0001$)



The reason for the significantly lower bubble count at 12 hours in the swimmers remains unclear. It is conceivable that the wearing of goggles may have an effect on the tear film not seen with the half-face mask worn by snorkellers and divers. The snorkellers had the highest (non-significant) median count of 0.66 at 72 hours. While it is unlikely that this increase relates to gas accumulation from shallow breath-hold diving, as bubble numbers would be declining by 72 hours, it is possible that some increase may have occurred due to mask squeeze even though the dives were limited to three metres. It is more likely that both results represent the normal range and are due to random variation in small sample sizes.

Two people demonstrated unusually high tear film bubble counts on three occasions. One examination was performed by Investigator 1 (KB), the others by Investigator 2 (AD) and confirmed by a third person. The two highest single readings obtained were 6 and 4.6 bubbles per eye respectively. While no formal attempt was made to identify any factors that may be associated with high counts, we noted that these readings were on volunteers who reported lack of sleep and a high alcohol intake in the preceding 12 hours. These same two volunteers were the only persons to average a bubble count greater than three bubbles per eye. The association between high bubble counts and fatigue or high ethanol intake may merit further investigation.

In order for ocular tear film bubble counts to be clinically useful in the diagnosis of DCI, the number of bubbles must be greater for divers with decompression illness and they would need to persist long enough to be detected on presentation to the evaluating physician. As noted previously, this time to presentation is rarely less than eight hours and averages over 24 hours.[3] Mekjavic demonstrated persistence of ocular tear film bubbles following chamber dives, with peak bubble numbers for up to two days but declining on the third day.[9]

The Prince of Wales Hyperbaric Unit is currently assessing the tear film of divers presenting with symptoms of DCI, and early results suggest bubble numbers are related to decompression stress. This would confirm the previous research data in which ocular tear film bubble numbers were increased following chamber dives to PADI no-decompression limits at differing depths.[6] In addition, tear film bubble formation displayed a dose-related rise with increasing duration of dives to 70 feet of sea water. Morariu also demonstrated a significant increase in tear film bubble counts following exercise in a hyperbaric chamber, which would correlate with increasing nitrogen uptake.[7] In keeping with the view that ocular bubbles are being formed in the process of de-nitrogenation, Jaki et al showed that the observed increase in bubble numbers following compressed air breathing was not matched by a similar increase after breathing 100% oxygen via a hood.[8]

This study's results show little inter-operator variability, with the two principal investigators recording a median count of 0.33 bubbles per person. Inter-operator variability will assume some importance if reliable readings are to be obtained and used as the basis for the decision to transfer to a hyperbaric facility. It may be that a technique using photography or assessing bubble volume, as suggested by Morariu should be developed to standardise results.[10]

However, our consistent results are encouraging and would allow for remote clinical use, as most emergency departments possess a slit-lamp. It may also provide a convenient method of monitoring hyperbaric exposure for attendants working in hyperbaric chambers. Although we have reported both bubble counts per person and per eye, the significant results and trends between groups were only apparent using median bubble counts per eye, suggesting this may be the more sensitive method of data analysis.

The next step is to investigate patients presenting to hyperbaric units with symptoms of decompression illness and this is now underway at the unit in the Prince of Wales Hospital. It is our hope to clearly define the place, if any, of tear film bubble counts in the quantification of decompression risk following compressed air diving, the monitoring of individuals subject to frequent compression and, finally, in the diagnosis of decompression illness.

Conclusion

Swimming in sea water and shallow breath-hold diving do not lead to an increase in ocular tear film bubble numbers. Therefore, the significant rise in tear film bubble numbers seen in recreational divers using scuba equipment is most likely to be due to decompression stress and unrelated to the salt water environment.[4] We have also confirmed the likely normal variation in the diving population.

Acknowledgements

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Declaration

The authors declare they have no financial interest in any commercial product involved in this research and received no financial assistance for the conduct of this study.

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This study by Dr Brown was accepted as part of the requirements for the Diploma of Diving and Hyperbaric Medicine of the South Pacific Underwater Medicine Society.

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The diving doctor's diary

Eosinophilic meningitis presenting as decompression illness

Michael Davis

Key words
Decompression illness, meningitis, eosinophilia, nematodes, case reports

Abstract
An experienced diver presented with suspected decompression illness (DCI) 17 days after an uncontrolled ascent from a 33 m dive carried out whilst on a tropical holiday. He had progressive symptoms of general malaise, headache, irritability, impaired mentation and facial, shoulder and leg pains and itchiness. A raised eosinophil count in blood and cerebrospinal fluid led to a diagnosis of eosinophilic meningitis rather than DCI, and this was confirmed by subsequent serology positive for the rat lung nematode worm *Angiostrongylus cantonensis*. Recovery was slow, with persistent symptoms still present at 18 months. This case illustrates the diagnostic difficulties where care givers in a traveller's home region are not versed in either diving or tropical medicine.

Introduction

Travel to tropical regions is never without health risks. When this is combined with diving activities, the development of symptoms in the traveller may present diagnostic problems, particularly where care givers in the patient's home region are not versed in either diving or tropical medicine. The following case illustrates these difficulties and the message that all post-diving symptoms are not due to decompression illness (DCI), even when the diving profile is a provocative one.

Case report

During a holiday in Tonga, a 48-year-old experienced diver undertook several dives. His final dive lasted 22 minutes to a maximum depth of 33 metres. He became low on air, panicked and made a rapid ascent to the surface despite the efforts of his buddy to restrain him. He developed a left-sided headache immediately after the dive, but was otherwise asymptomatic.

That evening he felt unwell and hot ("flu-like"). These symptoms persisted and he developed itchiness in the shoulders, right arm and both legs. He flew home to New Zealand three days after the dive, during which flight epigastric and central low thoracic back pain developed associated with bloating, anorexia and constipation unrelieved by ranitidine. He developed a rash thought to be due to this drug. A week later, he was admitted to the district hospital where he was described as anxious and depressed. Gastroscopy revealed a small gastric ulcer and he was discharged after five days on omeprazole.

Over the next few days, he developed worsening right leg pain. The severe headache persisted, associated with facial pain and tenderness and he had some photophobia. He became increasingly "jittery" and his wife reported slowed mentation and nocturnal hallucinations. He was readmitted to hospital, where neurological examination was normal, including normal fundi, and a CT scan of the brain was negative. The only abnormal investigations were GGT 102 IU.l^{-1} (normal range 0–50), ALT 91 IU.l^{-1} (normal range 0–50), eosinophilia 1.29 x 10^{9}.l^{-1} (normal range <0.51) and an abdominal ultrasound showing a "fatty liver".

At this stage, because of the provocative diving history, the patient's wife raised the possibility that his symptoms were diving related and he was referred to the regional hyperbaric centre 17 days post-dive. On admission, he had slow mentation with a Minnisota Mini-Mental Score of 26/30, mild neck stiffness, diminished pin-prick sensation over the anterior right thigh in the L2/3 distribution, a limping gait and sharpened Romberg's test of 15 seconds, though heel-toe walking was normal. There were no other neurological signs. The right knee and ankle and left wrist were slightly swollen and painful on movement, physical examination otherwise being unremarkable.

DCI was considered to be most unlikely but he was given a single short oxygen treatment (RAH Table 18.60.30) with no benefit. He was then referred to the Infectious Diseases service. MRI scan of the brain was unremarkable. Blood screen demonstrated a normal white count but with an eosinophilia of 1.8 x 10^{9} l^{-1}. A clinical diagnosis of eosinophilic meningitis was made. Lumbar puncture showed normal CSF pressure with a turbid aspirate. Globulins (CSF protein 1.63 g.l^{-1}; normal range 0.15–0.40) were moderately elevated, and white cell count was 493 x 10^{6}l^{-1} with 35% eosinophils. Gram stain was negative and there was no growth after six days.

Serology sent to the Institute of Clinical Pathology, New South Wales, was subsequently positive for *Angiostrongylus*

cantonensis, confirming the diagnosis of eosinophilic meningitis.

The patient continued to suffer severe chronic pain problems requiring referral to the regional pain centre. He gradually improved over the following year or so, but persistent upper limb girdle pain and recurrent headaches were still present at 18 months' follow up.

Discussion

Angiostrongylus cantonensis is a rat lung nematode worm with a complicated life cycle involving slugs or snails and the rodent central nervous system.[1] It may enter the food chain when the snails or rats are eaten by carnivores including fish or freshwater prawns. Human infection has been recorded throughout the western Pacific and parts of the Far East. Man is not a normal host but may become infected by eating third stage infective larvae in the normal intermediate host or other paratenic hosts that have not been correctly cooked. The incubation period ranges from 1 to 15 days.

Symptoms develop when the worm migrates across the blood-brain barrier, causing an inflammatory meningitis or meningo-encephalitis. Acute severe occipital or bitemporal headache, neck stiffness, paraesthesiae of the limbs and visual impairment are the typical symptoms. The pathological changes are caused by dead and degenerating worms and the inflammatory response to these.

This is a self-limiting infection lasting four to six weeks with a low mortality. Antihelminthic agents are of no clinical benefit and pain relief is difficult and may require opioids. Steroids are also of no proven benefit but often given in desperation as severe pain syndromes may occur requiring referral to specialist pain clinics as in this case.

DCI may be mimicked by a variety of conditions or by drug side effects. Mefloquine is not advised for use by divers travelling to malarial-risk areas because of its central nervous system side effects which may be confused with symptoms of DCI. In the past decade, we have seen a number of patients referred for suspected DCI in whom the diving activity was either coincidental or only contributory. Their diagnoses have included viral meningitis and myocarditis, atherosclerotic cerebrovascular disease, atypical or pseudoepilepsy, migraine with visual disturbances, inner ear barotrauma and musculo-skeletal injuries.

Whilst DCI cannot be excluded as a dual pathology in this case, it would seem unlikely given his subsequent course. Such diagnostic dilemmas reaffirm the need for thorough medical assessment of all divers with suspected DCI.

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Obituary

Ichiro Nashimoto, 1935–2002

Dr Nashimoto is probably not well known to the majority of SPUMS members. However, he was a long-time researcher and active medical doctor in diving, tunnel and caisson work and hyperbaric medicine in Japan and the Far East. Even back in the 1960s he was running a clinical hyperbaric facility at the Tokyo Medical and Dental University. He contributed to many publications and articles, including co-editing with Ed Lanphier two Undersea Hyperbaric Medicine Society (UHMS) workshops on decompression illness in 1987 and 1991. He was a tireless worker in trying to improve the safety of caisson workers and harbour divers including the development of decompression tables.

He is recalled by several members of UHMS as a delightful host, renowned for his "night diving" pub tours, and as a kind and jovial man.

Professor Nashimoto died in May 2002 after a short fight with liver cancer.

An unknown stinger of the deep

Vanessa Haller and Jane Bowman

Key words
Envenomation, marine animals, toxins, first aid, case reports

Abstract
An experienced diving instructor reported an unidentified sting received whilst diving in Malaysia, and her self-care for this. Despite the wide differential diagnosis, careful questioning of the diver suggested this was most likely due to a colonial hydroid. A brief review of the Order Hydroida is given. The principles of first aid care are outlined. Topical anti-inflammatory creams generally control ongoing symptoms. Protective clothing whilst diving is important but may leave the extremities and the head and neck exposed.

Case report

JB is an experienced dive instructor with thousands of logged dives. Whilst ascending from a dive on the USS Salute at Labuan, Malaysia, she felt a number of stings on her right hand and a couple on her neck. Without thinking, her reaction was to rub her hand. After leaving the water, her hand became extremely itchy and as the day wore on, she felt a severe burning sensation. Her neck itch did not bother her so much and seemed to disappear overnight. The itching and burning sensation continued throughout the night and she awoke on a number of occasions scratching. Next morning she had inflamed, tender, blister-like sores on her hands (Figure 1).

JB then used hydrocortisone cream as required, the initial application only relieving the itching for approximately one to two hours. Overall, she needed three applications. The following day the itching was less. She used two applications of tea tree oil and the itching ceased.

Ten days later, she could still see marks, especially if her hands were cold but no other after effects. JB has been stung in a similar way before, with a similar reaction each time, but usually the itching lasted much longer, at times up to two weeks. Previously, she had used other treatments such as 'Stingose', which did not appear to help.

Marine stingers

The sea is full of a huge diversity of life forms, many of which have developed weapons for hunting and/or protection. There are over 13,000 marine stingers. The differential diagnosis includes sea lice, fire coral, sea nettles, sea wasps, jellyfish and hydroids. Even the most innocuous looking sea creatures may have a hidden supply of stingers just waiting for something or someone to rub against them, eg. nudibranchs.

JB reported that she was stung whilst close to the bottom and she noted strings of fine hair-like structures floating in the water. This observation aids the diagnosis.

HYDROIDS (ORDER HYDROIDA)

Hydroids are plant-like organisms, but are in fact animals. They are related to jellyfish, sea anemones and corals but may often look like algae. There are two major sub-orders. In Anthomedusa, the polyp is not protected by the exoskeleton, which stops at the base of the polyp (gymnoblastic hydroids or Athecata). In Leptomedusa, both polyp and gonadal structures are protected by exoskeleton cups (calyptoblastic hydroids or Thecata).

Marine hydroids usually exist as colonies of animals living interconnected with specialised functions. They have a complicated life cycle with a fixed plant-like asexual generation, where they attach to all types of surfaces including rocks, kelp, crabs, and wrecks. These develop into a free-swimming jellyfish-like (corbulae) generation, where they produce the medusa stage of the hydroid. These produce eggs and sperm, which fertilize and develop into planulae and start new hydroid colonies. Some trail tentacles that normally secure them to the ocean floor. All hydroids are carnivorous filter feeders. They catch prey in the water column with the aid of grappling and stinging nematocytes. When scuba diving, exhaled bubbles can dislodge hydroids from their fixed place into the water column.

Symptoms

JB felt an initial "electric shock" and then a burning sensation. The pain and itch settled whilst in the water. During the night, she woke scratching incessantly. Next day, she noted blisters some of which were blood filled.

The initial sting gave the "electric shock" sensation, and the toxin and inflammatory reaction resulted in the burning sensation and itch. JB rubbed her hands and released more toxins from the nematocysts, hence this was the area which developed the greatest reaction and blisters. Whilst in the water, the itch settled. This could be due to the coolness of the water. When hot in bed, the urge to scratch was much greater and the inflammatory reaction was aggravated. Once this was appropriately treated, her symptoms settled. Steroid cream was more potent than previous measures used.

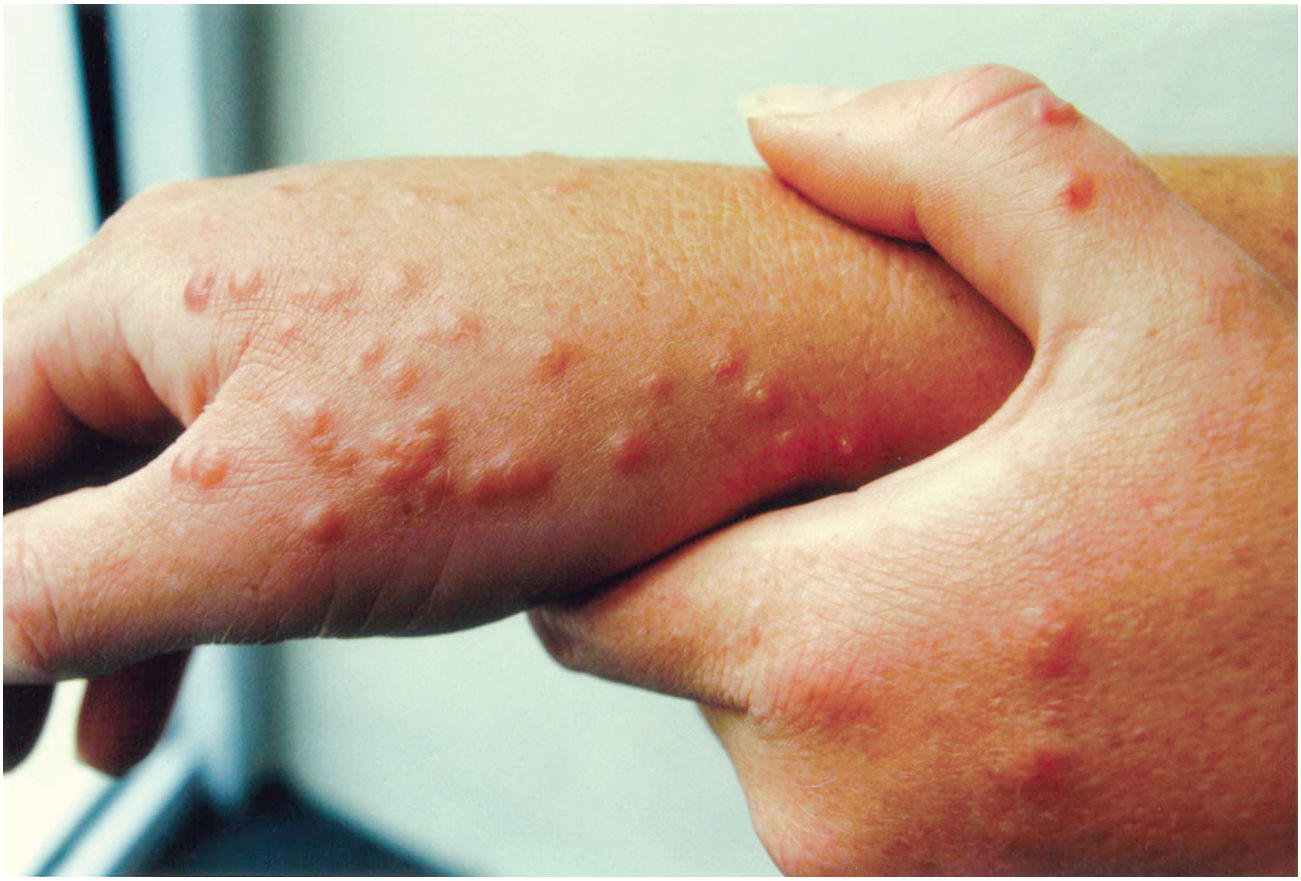

FIGURE 1. HAND LESIONS ON THE MORNING AFTER THE STING

First aid

Obviously, prevention is the first step. The best prevention is to avoid contact and avoid stingers' areas. In the diving industry, this would exclude some fantastic diving areas. It is important to avoid rubbing and scratching. If available, pour vinegar over the area for 30 seconds as this helps inactivate any remaining stinging cells. Alternatively, run cold water over the area. Cold compacts help to relieve pain and itching and also help settle the inflammatory reaction.

Topical anti-inflammatory creams are usually sufficient. JB found the steroid cream the best and this is very appropriate. I do not know of any randomised clinical studies concerning the use of tea tree oil, but there are a lot of anecdotal accounts of its anti pruritic effects.

Some victims may develop a more systemic reaction, particularly if they are subjected to repeated stinging events, and may require more supportive and systemic treatment.

Skin protection

Protective clothing whilst diving is recommended to prevent stings in areas where stingers are highly populated. This is usually in the form of some sort of wetsuit, or lycra-like suit. The minimum that is required is a couple of layers of pantyhose material. This often leaves the head, neck and hands exposed. Hoods and gloves can be worn to protect these areas. Gloves are in some areas discouraged from being worn, to try to prevent people damaging sea life including corals, hence common sense needs to prevail. If a diver is sensitive to hydroids, or is diving in a known area of high hydroid population, lycra-like gloves may be tried. Carry vinegar and containers of cold fresh water.

Recommended reading and web sites

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SPUMS Annual Scientific Meeting 2001

Drowning and near-drowning workshop: an overview

Christopher J Acott

Key words
Drowning, near drowning, pulmonary oedema. decompression illness

Abstract
Drowning is the third most common cause of accidental death in the general population and causes about one fifth of all accidental deaths in children. Alcohol is a frequent association in immersion accidents. Drowning or near drowning is not a diagnosis, the cause for the incident must be found. The ability to predict outcome from near drowning has been difficult, but the presence of spontaneous breathing, or the return of breathing, and the presence of a pulse shortly after rescue are all associated with a good outcome. All near-drowned victims may deteriorate even if they appear 'normal' on hospital admission, and therefore require a period of observation which many consider should be at least 24 hours. Hypothermia as a cerebral protection mechanism in near drowning may play little role in scuba diving. Diving as a cause of drowning has been neglected statistically, and there are only limited data on the interaction between near drowning, compressed gas diving, decompression illness and treatment. Theoretically, near drowning should decrease inert gas elimination because of induced changes in cardiovascular and pulmonary function. There are no data on the management of the near-drowned diver who may require recompression. Theoretically, recompression in the initial management of the near-drowned diver may cause deterioration in the diver's clinical condition. Due to these theoretical risks associated with recompression, the timing of recompression in the near-drowned diver may be important.

Introduction

Drowning is death due to asphyxia while immersed in a liquid medium, whilst near drowning is survival (at least temporarily) from an immersion incident. The classification used in this workshop is listed in Table 1.

Drowning ranks as the twentieth most common cause of death worldwide.[1] Drowning continues to be the third most common cause of accidental death in the general population and is more common in males than females.[1] It accounts for about one fifth of all accidental deaths in children.[2] While statistics for drowning deaths are known, there are no precise data for near-drowning incidents.[1] Some estimates put near-drowning incidents to be 500–600 times more common than drowning.[3]

Alcohol is a frequent association in immersion accidents either in the victim or, in the case of children, in supervising adults. Approximately 40% of childhood drownings occur in private swimming pools. Of note in childhood drowning is that children drown silently. In Australia, drowning is second only to motor vehicle trauma as the most common cause of accidental death.

In the majority of cases, the nature of the liquid medium does not influence the outcome. Drowning or near drowning will be the final common pathway of many problems occurring whilst in the water. One of the difficulties in studying the epidemiology of drowning fatalities has always been the tendency for pathologists to report it as "death by drowning" without any insight into the underlying cause(s). Drowning or near drowning is not a diagnosis, the cause for the incident must be found.

TABLE 1
CLASSIFICATION OF THE IMMERSION INCIDENT

Drowning:	Death due to asphyxia (with or without fluid aspiration) while immersed in a fluid
Near drowning:	Survival from an immersion incident (with or without fluid aspiration)
Secondary drowning:	Delayed onset of pulmonary oedema in a victim who appeared to have recovered from an immersion incident

Australian drowning statistics are collected by the Royal Life Saving Society.[4] In the financial year 1997/8, 326 people drowned, 52 of whom were children under the age of five. These data also show that for every child who drowned, five or six were admitted to hospital as a result of an immersion incident, and of these 5–20% had neurological sequelae. Data are available for each state and

although the numbers may vary, the percentages are approximately the same. Data for South Australia between 1995 and 1998 are shown in Table 2.[4]

Outcome

The ability to predict outcome from near drowning has been difficult, but recovery may involve varying degrees of functional disability. There is also a lack of full statistical data providing a ratio of those 'successfully resuscitated' to survivors left with permanent neurological damage.[1]Available data indicate that those factors listed in Table 3 may be associated with a poor outcome. The presence of spontaneous breathing, or the return of breathing, and the presence of a pulse shortly after rescue are all associated with a good outcome. In children, a detected motor response to pain upon arrival in hospital is associated with a good prognosis.[5]

Kries et al recently published that magnetic resonance spectroscopy was a good predictor of outcome.[6] However, their series contained only 16 patients. Szpilman published a severity classification related to mortality of near-drowned victims based on clinical data from 1831 cases gained at the site.[7] Szpilman's classification has some prognostic merit but relies on the clinical acumen of the rescuers. All but Grade 1 victims require oxygen administration. Grade 2 victims need a later careful clinical assessment. Szpilman's classification is listed in Table 4.

All near-drowned victims may deteriorate, even if they appear 'normal' on hospital admission. Some authors believe they should be observed for six hours, but others consider it more prudent to observe them for 24 hours.[1]

Drowning and scuba diving

Diving as a cause of drowning has been neglected statistically. Rarely does the drowning literature refer to diving as an important cause of drowning or near drowning. The Divers Alert Network (DAN) reported in 1997 that 63% of diving fatalities drowned.[3]

There are also limited data on the interaction between near drowning, compressed gas diving, decompression illness (DCI) and treatment (the author found one article in a Medline search). Whether near drowning delays inert gas elimination, thus predisposing a diver to DCI is unknown. Theoretically, near drowning should decrease inert gas elimination because of induced changes of cardiovascular and pulmonary function. Near drowning causes a reduction in cardiac output,[8] an increase in areas of low ventilation-perfusion ratios (shunt) in the lungs, and pulmonary hypertension.[9]

Experimentally, pulmonary hypertension causes venous sludging in the epidural plexus, promoting bubbling and venous infarction of the spinal cord.[10] However, experimental data on spinal cord DCI ignore the negative physiological effects of neurogenic paralysis on blood pressure and cardiac output, which will decrease spinal cord perfusion and increase any vascular stasis thus promoting

TABLE 2
SOUTH AUSTRALIAN DROWNING DATA 1995-1998

TOTAL	**49**
Male	76%
<5 years	14%
15–49 years	53%
At home	20%
Beach	35%
Country area	53%

TABLE 3
FACTORS ASSOCIATED WITH A POOR OUTCOME IN IMMERSION INCIDENTS

- Fixed dilated pupils
- Prolonged immersion
- Delay in effective resuscitation
- Absence of spontaneous respiration
- Severe metabolic acidosis – pH <7.1
- Asystole on admission to hospital
- Recovery of a pulse only after arrival at the hospital
- Glasgow Coma Scale (GCS) <5 on admission
- Hyperglycaemia

TABLE 4
SZPILMAN'S CLASSIFICATION[7]

GRADE	CLINICAL SIGNS	MORTALITY
Grade 1	Normal pulmonary auscultation with coughing	0%
Grade 2	Abnormal pulmonary auscultation with rales in some pulmonary field	0.6%
Grade 3	Acute pulmonary oedema without hypotension	5.2%
Grade 4	Acute pulmonary oedema with hypotension	19.4%
Grade 5	Isolated respiratory arrest	44%
Grade 6	Cardiopulmonary arrest	93%

further bubbling. Local changes in tissue perfusion (due to local effects of hypoxia, acidosis, and vasodilation) and a decreased cardiac output will also reduce tissue perfusion, and may cause prolonged tissue supersaturation, thus increasing the risk of DCI.

The pathophysiology of cerebral injury in near drowning is one of global hypoxia. Disruption of the blood-brain barrier results in cerebral oedema accompanied by a rise in intracranial pressure (ICP). Large increases in ICP are infrequent in near drowning and tend to appear after the initial resuscitation and in victims with some evidence of neuronal dysfunction. A rapid increase in ICP is an indicator of the severity of the neuronal injury and is a result of the brain injury rather than the cause of it.[5] Levels of ICP >20 mmHg and a cerebral perfusion pressure <60 mmHg are associated with a poor outcome despite aggressive therapy.[5] However, a normal ICP is not a guarantee of normal outcome. The impact of these changes on DCI is unknown.

Hypothermia and near drowning

Near drowning and the potential protective mechanisms of hypothermia have been the subject of many reviews. An excellent review concerning this topic was published by Golden et al.[1] A hypothermic decrease in cerebral metabolism has been considered as an explanation for survival following prolonged immersion, especially in children. Profound hypothermia is protective because it reduces oxygen consumption.

However, cardiovascular and respiratory responses due to cold water immersion (severe bradycardia or catecholamine excess causing ventricular arrthymias, cold water stimulation of breathing and a decrease in breath-hold time) would most likely be fatal before the victim had time to become hypothermic.[11] In addition, studies by Sterba et al have shown that sudden immersion in cold water will increase metabolic rate.[12]

The so-called 'protective mechanism' of hypothermia, therefore, may be incorrect and misleading. Hypothermia noted in a victim who has drowned in warm water is probably an indication of cerebral death, but aggressive resuscitation should not be abandoned because of this assumption. In addition, spearfishermen, snorkellers and compressed gas divers wear protective suits that delay the onset of hypothermia (hence these swimmers and divers rarely become profoundly cold) and this would prevent the suggested protective mechanism of hypothermia.

Management

Treatment is directed towards both the primary cause and near drowning. Early institution of continuous positive airway pressure (CPAP) or mechanical ventilation (IPPV), and positive end expiratory pressure (PEEP) are often associated with a rapid correction of hypoxia.[13] PEEP will not alter the underlying pulmonary injury, but will enable oxygenation during recovery. Initially, high-inspired oxygen concentrations may be needed. Animal data have shown a return of a normal A-a gradient and P_aO_2 within 10 minutes of resuscitation using 5 cms PEEP when ventilated with an FiO_2 of 100%.[13]

Steroids,[14] diuretics and prophylactic antibiotics are not indicated in the clinical management of near drowning.[1] Antibiotics, however, may be needed if the near drowning occurred in polluted waters. The appropriate antibiotic will be determined by sputum and/or blood cultures. Animal data have failed to show any benefit from exogenous surfactant, although there are anecdotal case reports of its successful use in a nine-year-old, near-drowned victim.[15] Evidence of trauma (cervical spine injuries must never be overlooked) and sinus and ear barotrauma should be looked for. These may act as a nidus of infection later.

Management of the near-drowned recompressed diver

Resuscitation may initially retard inert gas elimination in a patient with compromised cardiovascular and respiratory systems. IPPV and PEEP are associated with a decrease in cardiac output and blood pressure due to impaired venous return, decreased ventricular filling, increased pulmonary vascular resistance, and altered configuration and compliance of the right and left ventricles, even in patients without significant pulmonary pathology.[16,17]

There are no data on the management of the near-drowned diver who may require recompression (no reference was found by the author using a Medline search). Recompression in the initial management of the near-drowned diver may cause deterioration in the diver's clinical condition. Recompression and hyperbaric oxygen therapy (HBOT) may depress inert gas elimination because of worsening pulmonary oedema when applied to a patient with decreased left ventricular function. Recompression and HBOT, by increasing systemic vascular resistance and afterload, may further decrease left ventricular contractility, cause centralisation of blood volume and an imbalance between right and left heart function that worsens pulmonary oedema in these patients.[18]

There may also be an increased risk of pulmonary barotrauma in a patient receiving IPPV and PEEP. Recompression may also increase the work of breathing in a spontaneously breathing patient in respiratory distress because of the increase in gas density with increased pressure. An increase in the work of breathing may promote carbon dioxide retention and precipitate oxygen toxicity.

Due to these theoretical risks associated with recompression, the timing of recompression in the near-drowned diver may be important. More data are needed. A summary of the management of near drowning is presented in Table 5.

TABLE 5
SUMMARY OF THE MANAGEMENT OF NEAR DROWNING

- Rescue and remove from danger
- Begin CPR as soon as possible
- Transfer to hospital
- Determine primary cause*
- Advanced resuscitation: CPAP, intubate, ventilate, high inspired oxygen, PEEP, assess circulation
- Biochemical analysis including arterial blood gas, serum electrolytes (including magnesium* and CPK*), full blood count, bleeding and clotting profile
- CXR, cervical spine* films if needed
- Look for evidence of ear and sinus barotrauma (these may become a nidus for infection later)*
- Nasogastric tube
- Bladder catheterisation
- Monitor temperature
- Invasive haemodynamic monitoring as required (Swan Ganz etc)
- Inotropes and fluids as required
- Treat primary cause
- Watch for fluid depletion (osmotic-induced diarrhoea due to swallowed sea water)*
- Treat complications as they become evident
- Consider recompression in any compressed gas (scuba) diver*

*Often forgotten

Pulmonary oedema post immersion

The prevalence of pulmonary oedema (without evidence of aspiration) during scuba diving is unknown.[19] One series quotes a rate of about two per cent of divers.[20] There are numerous diving and non-diving causes of pulmonary oedema and these are listed in Table 6. The potential impact of near drowning and pulmonary oedema on inert gas kinetics has been modelled by Doolette and Acott.[21]

A return to diving following near drowning

A return to diving following a near-drowning episode has not been discussed in the medical or diving literature. Medical suitability to return to diving would depend on the presence of neurological and respiratory sequelae and the cause of the near-drowning episode. Normal respiratory function may take up to 16 weeks to return in non smokers who have near drowned.[22]

TABLE 6
CAUSES OF PULMONARY OEDEMA POST IMMERSION

Diving (*breathhold and scuba)

Near drowning*
Aspiration (fluid and gastric contents)*
Marine envenomation*
Inhalation of contaminants (carbon monoxide, oil, bacteria from equipment)
Intra-alveolar haemorrhage (on anticoagulants)*
Neurogenic pulmonary oedema (hypoxia)*
Negative pressure pulmonary oedema (laryngospasm)*
Pulmonary oedema of immersion
Pulmonary oedema and strenuous swimming*
Pulmonary decompression illness ('chokes')
Underwater blast*
Pulmonary barotrauma of descent (BH)
Oxygen toxicity

Non diving

Myocardial infarction
Acute allergy
Chest infection
Trauma

Summary

The 2001 SPUMS workshop on drowning and near drowning in divers addressed the following issues:

The epidemiology of drowning in compressed air divers (Australian and New Zealand data)[23,24]

Whether or not there is an increased risk of decompression illness with near drowning (using a probabilistic model of inert gas kinetics)[21]

The pathophysiology of near drowning[25]

The differential diagnosis of pulmonary oedema following compressed air diving, including the salt water aspiration syndrome and immersion pulmonary oedema[26,27]

The physiological effects of IPPV and PEEP in a hyperbaric environment[28]

The use of lignocaine as adjunctive therapy in dysbaric disease[29]

A diving physician's personal view of why divers drown[30]

Near drowning and the unresponsive diver; rescue training for recreational scuba divers[31]

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The pathophysiology of drowning

Robert North

Key words
Drowning, near drowning, pathophysiology

Abstract
The effects of drowning, both fatal and non-fatal, on the different major organ systems within the body, and the process of hypoxia are reviewed. Pulmonary fluid aspiration results in an increase in low ventilation-perfusion ratio areas and decreased compliance. However, in most victims variable quantities of the liquid medium are aspirated. It is believed that in at least 10% of drowned people laryngospasm occurs, but practical evidence conflicts with the theory that the closed larynx keeps water from entering the trachea. Widespread atelectasis occurs after surfactant is denatured, leading to large shunts and perfusion mismatch. Five to twenty five per cent of patients progress to life-threatening acute respiratory failure. The effects of immersion on the cardiovascular system are an increased venous return and a central shift of blood volume, but the cardiovascular system has been stated to show remarkable stability, and the changes in blood volume are not significant enough to be life threatening. There are differences in biochemical changes in the blood with inhalation of fresh water and sea water. However, these are minimal and not clinically significant. A severe lactic acidosis develops secondary to the hypoxia. The most common renal complication is oliguria due to acute tubular necrosis. The roles of the diving reflex and hypothermia in cerebral protection are still not well understood. The overall pathophysiological processes in drowning and near drowning are complex but ultimately are determined by the degree and duration of hypoxia.

Introduction

Drowning is the most common cause of death among divers, in both those using scuba and snorkelling equipment. Something usually happens to the diver initially to predispose them to drowning, for example, an out-of-air situation or, with breath-hold diving, loss of consciousness after having hyperventilated before descent, often referred to (incorrectly) as 'shallow water blackout'.

When someone does die from drowning, it is not simply a case of water flowing into their lungs. The effects of drowning and near drowning on the different major organ systems within the body, and the process of hypoxia will be reviewed. The effects of cold water immersion, the diving reflex and hypothermia and cerebral protection will also be discussed.

Respiratory response to immersion

With vertical submersion, there is an overall increase in the work of breathing of 65%, due to the pressure of water compressing the lower body. This causes displacement of blood into the pulmonary circulation, cephalic movement of the diaphragm and minor compression of the chest wall. This is generally not a problem in healthy people.[1]

With submersion of the head comes a voluntary apnoea. The 'break point' of this voluntary action is determined mainly by the arterial carbon dioxide level. When the break point is reached, an involuntary inspiration occurs.[2]

Pulmonary fluid aspiration destroys or dilutes surfactant (salt water versus fresh water near drowning), disrupts the alveolar capillary membrane, and damages the pneumocytes causing atelectasis. This results in an increase in low ventilation-perfusion (V/Q) ratio areas and decrease in compliance.[3] Aspiration of vomitus is common.[4] However, in most victims, variable quantities of the liquid medium are aspirated. After a latent period, which can extend to 48 hours, 5–25% of patients progress to life-threatening acute respiratory failure – the so-called 'secondary drowning syndrome'.

It is commonly believed that in at least 10% of drowned people laryngospasm occurs, thus occluding the airway. This is based on the finding that 10% of drowning victims at autopsy have little or no water in their lungs.[4] However, these are autopsy findings, not necessarily proving the absence of water in the lungs at the time of drowning. Fresh water is readily absorbed from the lungs and most dry lung autopsies were from fresh water victims.[6] Also, anaesthetists have been able to intubate an asphyxiated patient after agonal relaxation of the larynx in patients with laryngospasm. Hypoxia and hypercarbia are both factors leading to laryngeal relaxation. This practical evidence conflicts with the theory that the closed larynx keeps water from entering the trachea.

Associated with laryngospasm, there is a mucous outpouring in the bronchi and bronchospasm.[2] Both of these factors may help to prevent water entry with terminal relaxation. Laryngospasm occurs more commonly in adults than children and is believed to be facilitated by other factors, for example alcohol ingestion.

Once water enters the lungs, compliance falls and pulmonary vasoconstriction and pulmonary hypertension

occur. As described earlier, widespread atelectasis occurs after surfactant is denatured, leading to large V/Q mismatch. Drowning in salt water may differ from that in fresh water, in that pulmonary oedema is facilitated by the osmotic pull from salt water ions.[2] However, this effect can also happen due to foreign material in fresh water. Finally, after the period of involuntary aspiration, there is a secondary apnoea, followed by gasping and later respiratory arrest. Death ultimately ensues from apnoeic hypoxia.[6]

Cardiovascular response

The effects of immersion on the cardiovascular system are an increased venous return and a central shift of blood volume.[7] As a result, there is an increase in cardiac output of one to two thirds. The increase in central blood volume stimulates stretch receptors leading to a diuresis to counter the apparent increased blood volume.[8]

After water enters the lungs of the 90% of drowning victims without 'dry' lungs at autopsy, the cardiovascular system has been stated to show remarkable stability.[2] Theoretically, with fresh water entering the lungs, the greater osmolality of blood draws water from the alveoli into the circulation. This leads to an increase in blood volume. Again, the greater osmolality intracellularly draws water into the cells and thus leads to haemolysis.[3] This process also leads to an increase in potassium. However, haemoglobin and haematocrit levels in victims drowned in fresh water are not significantly altered, so the theory may be incorrect. Salt water inhalation theoretically leads to osmotic flow of intravascular fluid into the alveoli, because salt water is hypertonic relative to serum, which would cause a decrease in blood volume.[9]

In both cases, the changes in blood volume are generally not significant enough to be life threatening. Although there are differences in biochemical changes in the blood with inhalation of fresh water and sea water, these are minimal and not clinically significant.[6]

A reduction in cardiac output may occur due to immersion diuresis and decreased myocardial function from hypoxia, induced electrolyte abnormalities and acidosis.[7,8] These cardiovascular changes and an increase in pulmonary vascular resistance are independent of the tonicity of the fluid aspirated and are a direct effect of hypoxia.[7]

Blood pressure changes during drowning are due to hypoxia, which initially causes marked sympathetic stimulation with tachycardia and an increase in systemic resistance, followed by myocardial failure and hypotension. Blood pressure changes may also be due to the development of arrhythmias. A variety of ECG changes are seen, varying from no change, ventricular fibrillation (VF) leading to arrest, and extreme bradycardia in cold water as hypothermia develops. Atrial and ventricular arrhythmias are a consequence of catecholamine excess, acidosis and hypoxia. In summary, any arrhythmia may develop.

Serum electrolytes are rarely abnormal, except for the acidosis and an elevated serum magnesium.[9] Near drowning in industrial fluid may, however, cause electrolyte abnormalities. Haemolysis and rhabdomyolysis may occur early or be a later complication of sepsis.[10] Hypermagnesaemia will depress myocardial function and cause peripheral vasodilation.

Hypoxia is the inadequate oxygenation of cells and is caused by apnoea as in the case of laryngeal spasm or inadequate delivery of oxygen to the lungs. A low partial pressure of oxygen is detected by the carotid and aortic bodies and the direct lack of oxygen to the respiratory centre in the brain also generates some of the following response. Hypoxia leads to vasodilatation and an increase in cardiac output.[4] Another feature of hypoxia is pulmonary hypertension. This is caused by constriction of the pulmonary artery as a response to the low partial pressure of oxygen. Subsequently, right ventricular afterload failure may develop.[7]

A severe lactic acidosis develops secondary to the hypoxia. Eventually, if hypoxia is severe the myocardium will fail. When there is a low level of oxygen and a high level of carbon dioxide in the blood, the cerebrovascular response is vasodilatation to increase cerebral blood flow. Eventually, an acidosis will develop in the brain, with subsequent cerebral oedema and raised intracranial pressure.[4] Severe hypoxia will affect brainstem centres and death will ultimately result after respiratory arrest.

In summary, there are a wide range of sometimes conflicting factors – immersion, aspiration, hypoxia, fluid and electrolyte shifts, sympathetically mediated cardiac rate and rhythm changes, the diving reflex and hypothermia – that may impact on the cardiovascular system.

Gastric and renal response

Swallowing of water occurs, particularly if the water is choppy, and is more common in colder water when swimming is more difficult due to impaired coordination. Vomiting often occurs and may lead to aspiration. The water that is swallowed also contributes to cooling of the body.[1]

The incidence of renal failure following near drowning is unknown but is certainly less than the frequency of lung, brain or heart injury. The most common complication is oliguria due to acute tubular necrosis (ATN). ATN in near drowning is thought to be a consequence of hypoxia, hypotension, haemolysis and rhabdomyolysis.[10]

Behaviour during drowning

Behaviour of drowning victims varies depending on the events leading to their drowning. A series of 100 fatalities reviewed by Edmonds, Walker and Scott[5] reported that over

half of the victims showed no change in behaviour. These 'quiet' drownings, when the victim does not struggle, are attributed to shallow water blackout, the effects of cold, alcohol/drug intoxication, or other medical conditions, for example arrhythmias or cerebral arterial gas embolism.

Animal studies have shown an initial struggle on submersion, sometimes with inhalation, followed by a suspension of movement. There is frequent swallowing and then a late, violent struggle. Finally, convulsive movements occur and exhalation.[4] Observations of humans drowning have shown the same sequence of events. Survivors of near-drowning episodes have reported a sense of panic, which is often described as a key factor in drowning but often cannot be observed by witnesses to the incident.

Diving reflex

The role of the diving reflex in drowning and near drowning is still not well understood. The reflex is mediated by stimulation of the ophthalmic division of the fifth cranial nerve.[1] It results in apnoea, peripheral vasoconstriction, shunting of blood to the cerebral and coronary circulations, and bradycardia.[2,4] The reflex occurs independently of chemoreceptor and baroreceptor influence.

The diving reflex is more pronounced in infants than in adults, enhanced by cold and anxiety, and is modified by the equipment being used by the diver.[1] For example, a diver wearing a drysuit with only the face exposed will have a more profound response than a diver without the drysuit. Fifteen per cent of people are said to show a profound diving response.[1] The diving reflex may be a significant part of why some children have been able to survive with minimal or no functional loss after prolonged periods of immersion.

Immersion in cold water

Upon immersion in cold water, there is often an initial involuntary gasp. In most cases, this appears to be controlled by the diver until the head is again clear of the surface. This may be followed by uncontrolled hyperventilation.[2] Due to the initial large inspiration, tidal breathing occurs close to total lung capacity, thus a feeling of dyspnoea occurs. The maximum breath-hold time in cold water becomes only about 10 seconds. Respiratory drive increases in water colder than 25°C, and is maximal at 10°C.[1]

It is easy to imagine in colder water, particularly if it is choppy, that aspiration will occur, as the swimmer is breathing a lot harder and is unable to hold their breath for very long. Aspiration is also more likely to occur when core body temperature has fallen to about 34°C. Impaired neuromuscular function results with cold peripheries when limb temperature is about 28°C, and with a core temperature of between 33°C and 35°C.[1]

Vasoconstriction occurs in cold water, leading to both increased arterial and venous blood pressures. This centralises blood volume, and may double cardiac output. Urine output increases by up to a third of that seen in thermoneutral water.[1] However, vasoconstriction of the scalp is less than elsewhere and so more heat is lost from the head.

Shivering occurs to produce heat but this function ceases at about 33°C. Ventricular fibrillation occurs at about 28°C core temperature and is either mediated vagally or from excessive catecholamine release. Asystole occurs between 24°C and 26°C.[1]

Hypometabolism occurs with decreased temperatures. There is 6–7% decrease in cerebral blood flow for each degree of temperature loss. Loss of consciousness occurs at about 30°C core temperature, and cerebral activity ceases at 22°C.[1] Children are affected more by cold and cool down quicker than adults, as their body surface area to mass ratio is greater and they have less fat to insulate them.

Hypothermia and cerebral protection

The true mechanism of cerebral protection by cold is unknown. It is thought to work with the mammalian diving reflex, which shunts blood to the brain after peripheral vasoconstriction, and hypometabolism due to the cold. Theoretically, it is not possible physiologically to cool an immersed person quickly enough to prevent hypoxic brain damage from occurring.[1]

The rate of change in core temperature is dependent on the water temperature, movement of the water against the skin, body surface area to mass ratio, insulation (for example fat, wetsuit or drysuit), peripheral circulation, and conditions affecting the person (for example alcohol or injury). Even with these conditions 'optimised' it is still not thought possible to cool a body rapidly enough to afford cerebral protection.[1]

Conclusions

Drowning eventually leads to death via respiratory arrest resulting from hypoxia, but before that the effects on the body involve all the major organ systems. The effects on each contribute to the responses and effects on the other systems, making the pathophysiological process quite complicated. Conditions such as cold and the diving reflex also compound the physiology of drowning. However, much has yet to be learnt about the process and of what value cold and the diving reflex may have in the survival or treatment of near-drowned patients.

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Causes of drowning in divers

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Key words
Drowning, incidents, recreational diving, training, buddies

Abstract
Drowning is the endpoint of many diving fatalities for a variety of reasons. Most divers are not well enough trained and should be encouraged to undertake more training beyond basic recreational open water diving. Unexpected equipment failures may sometimes occur. How to cope with these problems is not always adequately taught during initial training. A quarter of equipment failure incidents result in morbidity, yet a surprising number of divers have poorly maintained or serviced equipment. Running out of air is one of the most common serious diving incidents, which suggests that there is something wrong with how divers are trained. Air integrated gauges/computers, most of which have audible warnings, are useful tools for gas supply management and should be utilised more than at present. Wrist mounted displays are more readily seen than dangling consoles. The Cave Diving Association of Australia training and certification system is an example of successful self-regulation in recreational diving. Medical conditions may predispose divers to a diving incident. Increasingly, risk recognition and management is being emphasised in the medical assessment of diving candidates.

Introduction

Prevention is the best method of stopping divers drowning. Only fish are fit to dive, so humans are at risk every time they enter the water. Drowning is the endpoint of many diving fatalities for a variety of reasons. The following is an experienced scuba diver/general practitioner's personal view of this subject. I will comment on aspects of prevention, diving equipment, diving practice and medical problems that I consider relevant.

Prevention

How might we reduce the number of divers drowning? My belief is that most divers are not well enough trained in general, and the preponderance of untrained or inexperienced divers in the fatality statistics bear this out.[1,2] We should be encouraging divers to undertake more training and particularly for some of the more specialised aspects of diving such as wreck diving or cave diving. We should encourage people who want to do anything more than basic recreational open water diving to obtain the appropriate training. It really is worthwhile doing extra training, and there are many training modules available these days.

Equipment failure and maintenance

Unexpected equipment failures like blown O-rings, ruptured hoses and problems with buoyancy compensators (BCDs) may sometimes occur. How to cope with these problems is not always adequately taught during initial training.

All diving equipment sold currently includes a contents gauge. A failed gauge is perhaps the only excuse for running out of air. Unfortunately, most divers do not have their gauge examined when their diving regulator is serviced. If you have not had your gauge checked recently, then do so soon.

In a report on equipment failure problems, 27 of 105 events resulted in morbidity.[3] The same report describes strategies to minimise adverse outcomes in diving.[1] Table 1 shows how the divers were injured and the causes of their problems.

It is amazing the number of divers who have poorly maintained, poorly or never serviced diving equipment. In most parts of the world scuba cylinders have to be tested by law or they will not be filled. Perhaps there should also be a voluntary code of practice whereby regulators need a date code indicating servicing date. It could be argued that regulators, the contents gauge and the BCD should all have a mandatory service every year.

Loss of gas supply

Divers obviously need better education about how to cope with an out-of-air situation, as running out of air is one of the most common causes of diving incidents. Another option for divers who run out of air is to have more air. Obviously we cannot carry an infinite amount of air, but if one has a spare supply, it is a good idea. The concept of redundant systems is in fact nothing new in Victoria (Australia) where the dive industry has just such a voluntary code of practice in place. If you are going out on any of the charter boats and wish to dive below 30 m you must have a bail-out bottle; you either have to have your own or they will rent you one.

It is remarkable that divers run out of air as a single event. The fact is that many divers seem to conduct their dive in such a poor manner that they do not have an adequate gas supply to return to the surface. Discussions with the operators of dive charter vessels indicate that running out of air happens regularly. This suggests to me that there is something wrong with how we are training divers. I emphasise that running out of air really just should not happen.

I believe air integrated gauges/computers, most of which have audible warnings, are really useful tools for gas supply management. They alert the diver if the dive is going to be limited by the decompression tables or the air supply, provided you look at them regularly. I would encourage divers to consider them when they next purchase new equipment. The ideal place to have a gauge when diving is on the wrist because it is easy to look at. Most divers look at their wrist more often than they examine a dangling trail gauge during a dive. A wrist-mounted computer display, especially if it includes air supply, is a better and safer option than having to retrieve a contents gauge and look at it.

SHOULD DIVER-EMPTIED TANKS BE FILLED?

Perhaps the lateral thinker's approach to divers running out of air would be for the dive industry to decline to fill completely empty scuba tanks without doing an internal visual inspection and charging a fee for this service. An alternative and innovative approach was adopted by the dive operator at the 2000 SPUMS ASM, who warned divers that an empty tank signified the diver wished to purchase

TABLE 1
MORBIDITY ASSOCIATED WITH EQUIPMENT FAILURE (from Acott[1])

Morbidity	Total	C	CG	D	I	F
Decompression illness	16	6	6	2	2	-
Pulmonary barotrauma	6	-	2	-	2	2
Salt water aspiration	1	-	1	-	-	-
Near drowning	1	-	-	-	-	1
Ear barotrauma	1	-	-	1	-	
Not specified	2	-	-	-	2	-
Totals	**27**	**6**	**9**	**2**	**7**	**3**

Causes
C = Computer failure
CG = Contents gauge failure
D = Depth gauge failure
I = Inflator failure (spontaneous inflation 5, failure 2)
F = First stage failure

the cylinder, the cost of which would appear on their hotel account!

MULTIPLE CYLINDERS AND FULL FACE MASKS

As far as I know divers are not trained to use multiple cylinders in open-water training. There is no doubt that extra cylinders and extra valves and regulators increase the complexity of the diver's equipment. Multiple cylinders weigh more, and because of their bulk there is more risk of entrapment. There is more to maintain and more to go wrong. Of course, this makes diving even more expensive. Should all divers be trained to use these back-up systems as part of their basic training? I would say probably not. Should divers who are doing deep air dives or technical dives be encouraged to use a full-face mask? If they do have a fit or lose consciousness underwater, they might have a chance of surviving if they had somebody nearby.

Diving practices

ENTRAPMENT

Many people have run out of air when entrapped. For example, they have died in caves in South Australia; that is, until the Cave Diving Association of Australia (CDAA) introduced a training and certification system. The CDAA system is one of the success stories of self-regulation in recreational diving in reducing cave diving fatalities. Divers have died lost in wrecks, they have become entangled or lost and ultimately exhausted their air supply and drowned. A diver may also become virtually entrapped, in that the diver may have done a decompression dive and discovered that they cannot surface due to a decompression ceiling, although they are low on air. Unusual environmental conditions such as the powerful down-currents experienced in Palau may present similar virtual entrapment problems.

BUDDY DIVING

I am not a great proponent of buddy diving. I believe that unless your buddy is tethered to you on a line, he (or she) is not close enough to be of much use to you. If they are out of sight or at a distance then they are not going to be able to provide you with much support in the event of an incident.

Medical problems

There are a variety of medical reasons why one might drown. Epilepsy, diabetes, myocardial infarction, oxygen toxicity, carbon monoxide and other gas toxicities and hypoxia (eg. shallow water blackout) are a few. There are many more medical reasons why one might lose consciousness underwater and drown, or if lucky near drown and be rescued.

SPUMS supports making certain that divers are fully fit to dive, hopefully to detect and advise on medical conditions that may predispose prospective divers to having a diving incident. Risk recognition and management are emphasised in the current SPUMS diving medical assessment form.

Conclusions

I believe that divers need to be better trained. Divers using modern equipment properly maintained should not run out of air. If divers are running out air, and they are, there is something wrong with how they are trained. Diving training agencies need to emphasise to trainees that divers have to think about what they should be doing to maintain and enhance their own safety.

Drowning is the endpoint of many diving fatalities for a variety of different reasons. Most divers who drown or near drown do so either by running out of air or losing access to their air supply. Divers mostly run out of air through basic errors and simply not looking at their gauges. Other factors include diver error leading to panic and panic leading to diver error. Entrapment (eg. lost in a cave or wreck) may result in an out-of-air situation. Equipment failure and unconsciousness from medical causes may also result in drowning.

I have made various suggestions with regard to equipment such as redundant systems, and mandatory servicing. I think some of them would be best brought about through voluntary action by the dive industry.

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Immersion pulmonary oedema

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Key words
Pulmonary oedema, pulmonary edema, diving, scuba, immersion, cold, beta blockade

Abstract
Pulmonary oedema of immersion is a rare complication of scuba diving. A case and brief discussion of the differential diagnosis and pathophysiology are presented. Onset of symptoms typically occurs during the dive, and is characterized by dyspnoea, cough and frothy sputum. Spontaneous recovery frequently occurs once the diver leaves the water. If investigated, the findings are of hypoxaemia and chest X-ray changes typical of pulmonary oedema. The differential diagnosis includes decompression illness, pulmonary barotrauma, near drowning, salt water aspiration syndrome and non-diving causes of pulmonary oedema. The condition may be precipitated by the increase in pulmonary capillary hydrostatic pressure that occurs on immersion, coupled with the imposition of a pressure gradient between mouth pressures and hydrostatic pressure at the chest in the upright diver, and the imposition of more negative intra-alveolar pressures during breathing of a denser gas or from a poorly tuned regulator. Divers with heart disease, hypertension, or taking negative inotropic agents may be at greater risk.

Introduction

Diving physicians are sometimes presented with patients complaining of isolated respiratory symptoms and signs arising during or immediately after diving. A diagnosis that is frequently overlooked in this setting is immersion pulmonary oedema. It is important to consider this problem because recompression is not indicated in its treatment. Recognition may prevent a costly and unnecessary evacuation to a hyperbaric facility. Immersion pulmonary oedema is a rare condition but case reports and series have steadily appeared over the past 10 years. The pathophysiology is poorly understood. Dr Joanne Grindlay and I recently reviewed the problem, and this presentation is a synopsis of the important points made in that paper.[1]

Case report[1]

A 31-year-old female medical practitioner who had no relevant past history and was on no medications was undertaking the second pool session of a PADI open water diving course. The first pool session had been conducted two nights previously and she had experienced no problems. The pool session in question involved a number of skills including taking the regulator in and out of her mouth underwater, and she experienced no problems with any of the skills being practised. The session lasted 90 minutes and was conducted in a chlorinated pool, four metres in depth, water temperature 20°C. She was wearing reasonable thermal protection, a two-piece 5 mm wetsuit with a hood but no gloves.

Immediately after the end of the session, which involved quite a few ascents and descents throughout the evening, she noticed that she was short of breath. She developed a wheezy cough productive of some watery sputum which was slightly pink and she became cyanotic. This observation was made by medical colleagues who were doing the diving course with her. She self-presented at an emergency department with progressive shortness of breath. On admission, respiratory rate was 20 breaths per minute with widespread crepitations on auscultation. Oxygen saturation was 88% on room air. Blood gases were recorded while she was on oxygen with an inspiratory fraction of 50%. P_aO_2 was 63 mmHg, oxygen saturation 92%, and P_aCO_2 and acid-base state normal. Chest X-ray showed an interstitial pulmonary oedema pattern.

She was treated with 30 minutes continuous positive airway pressure by mask and given 20 mg frusemide intravenously. She made a very rapid symptomatic recovery and was discharged. For about two weeks afterwards she felt slightly dyspnoeic on exertion but at rest she was asymptomatic.

A number of investigations were carried out. Her dive equipment was examined and was found to be in working order. An electrocardiogram and echocardiogram were normal with no evidence of a patent foramen ovale or valvular heart disease. A stress test, histamine and hypertonic saline challenge tests, and finally a high resolution inspiratory and expiratory thoracic helical CT scan, were all normal. She returned to diving, completing her course and at last follow up had done about 50 dives in a wide range of conditions without incident.

Discussion

The main feature of this case was acute pulmonary oedema with no other symptoms and no latent period after diving. The initial diagnosis made at the time of presentation in the emergency department was decompression illness (DCI), notwithstanding such a non-provocative dive profile.

DCI due to venous bubble formation from dissolved nitrogen may cause dyspnoea and possibly pulmonary oedema, but to the author's knowledge such manifestations are unreported after such unprovocative diving. Moreover, pulmonary manifestations of DCI are usually accompanied or followed by other, more specific, neurological and musculoskeletal symptoms.[2] Therefore, the diagnosis of DCI in this case seems implausible.

Pulmonary barotrauma may certainly occur in a swimming pool situation, but is not known to present as pulmonary oedema. More typically, pulmonary barotrauma presents either with neurological or cardiac symptoms and signs from cerebral arterial gas embolism, or with evidence of pneumothorax and/or mediastinal and subcutaneous emphysema. None of these were present.

Near drowning produces pulmonary oedema of short latency. However, clinically apparent near drowning requires a history of aspiration, which is usually brought on by panic, a malfunctioning regulator, or something else. There were no such events in this case and it seems unlikely that a sufficient occult aspiration of water could have occurred without this informed diver noticing.

Salt water aspiration syndrome is a condition attributed to aspiration of small amounts of water through a diving regulator.[3] The classical symptoms and signs of cough, fever, constitutional malaise and sometimes patchy consolidation on chest X-ray (but not of a pulmonary oedema pattern) were not present in this woman. Moreover, this syndrome is reported as having a long latent period, and does not typically onset during the dive as is often the case in immersion pulmonary oedema.

Exercise-induced pulmonary oedema is an obscure disorder of uncertain relevance to humans, but it may have contributed to several cases reported recently in fin swimmers exercising hard for long periods.[4] Pulmonary capillary fragility is increased by catecholamine release during exercise in other species, eg. horses, but it is not commonly seen in humans. This patient was not exercising vigorously.

Non-diving causes of pulmonary oedema or of symptoms suggestive of pulmonary oedema include asthma, myocardial infarction, trauma, allergy, anaphylaxis and exposures to toxic gas. None of these conditions appeared relevant to this case.

In the absence of other adequate explanations, it was presumed that this woman had suffered immersion-induced pulmonary oedema. This condition is probably a form of acute onset left ventricular failure, but its pathophysiology is not definitively described. There are probably several important components. Firstly, it is known that immersion itself, and the peripheral vasoconstriction associated with either immersion or exposure to cold, both cause a simultaneous increase in cardiac pre-load and after-load as blood volume is centralised and peripheral resistance increases. Not surprisingly, there is an increase in mean pulmonary artery pressure, and therefore pulmonary capillary hydrostatic pressure.

Secondly, immersion of a diver in an upright position results in the basal lung tissue being exposed to a hydrostatic pressure approximately 15–20 cm greater than that of the airway pressure at the mouth (which equilibrates with alveolar pressure during breathing). This differential is volume-compensated by engorgement of the pulmonary blood vessels, which may predispose to capillary stress failure.

Thirdly, it seems likely that the negative intra-alveolar pressures generated during inspiration due to airways resistance are greater during diving because turbulent flow is more likely when breathing a denser gas. Finally, and in relation to the latter point, if the diver is breathing from a poorly tuned regulator that requires increased negative pressure to activate gas flow, this will also enhance negative intra-alveolar pressure.

It is plausible that this combination of increased pulmonary capillary hydrostatic pressure, pulmonary vessel distension, and exaggerated negative cyclical intra-alveolar pressure promotes the transudation of fluid through the capillary walls into the alveoli. This sequence of events would be more likely in a 'predisposed' person whose myocardial response to increased load is impaired. Interestingly, most cases seen by the author recovered spontaneously over a period of hours once the diver was removed from the water. The case described here was one of the few that received active treatment.

A number of contributory or risk factors have been proposed for this condition. The earliest descriptions suggested that this was a cold immersion induced phenomenon.[5] Cold water immersion produces a greater degree of vasoconstriction and a consequent increase in pre-load and after-load that has been confirmed experimentally. However, other reported cases and our own unpublished experience indicate that extreme cold is clearly not a prerequisite for pulmonary oedema of immersion.[6]

Hypertension and coronary artery disease have been reported in some cases, but in others there have been no identifiable predisposing factors whatsoever, as in the present case. All of the 11 cases reported by Wilmshurst et al (1982) had an abnormal peripheral resistance with a high resting forearm vascular resistance and an exaggerated vascular response to cold exposure.[7] However, none of the three cases reported by Pons exhibited these features.[8]

Another potential contributory factor is beta blockade or medication with negative inotropic agents. Of six cases seen by the author, three were on beta blockers.[1] Hampson and

Dunford reported six patients, one of whom was on beta blockers and one on a calcium-channel blocker.[6] Medication with these agents might simply be an epi-phenomenon, reflecting the presence of hypertension or other cardiovascular disease. Nevertheless, it makes sense that a negative inotrope might predispose a swimmer or diver to cardiac decompensation in the presence of a sudden increase in pre-load and after-load.

Over-hydration has been reported as a potential risk factor.[4] Eight subjects in a strenuous military swim developed pulmonary oedema after drinking five litres of water prior to the swim to avoid dehydration. The heavy exercise in this group was proposed as another risk factor (see above).

What advice should be given to someone about diving after an episode like this? The simplest advice would be to never dive again, as one could not guarantee this would not happen again even if no predisposing factors could be identified. This may become a risk/benefit decision for the diver to make after being counselled about the issues. Certainly, if investigation identified an obvious risk factor advice not to dive is clearly indicated. Appropriate counselling would include advice to wear good thermal protection, including a dry suit, boots, gloves and hood in cooler waters, avoidance of over-hydration and overexertion, and use of a well tuned regulator. Other advice would include diving from a platform where oxygen is immediately available at the surface, and not at some remote location.

Summary

The syndrome of sudden onset pulmonary oedema may occur in divers of all ages and levels of fitness but may be more likely in older divers, those with cardiac disease or hypertension, and those using negative inotropic agents or who are over-hydrated. With respect to the dive, it can occur in all temperatures of water but the risk is probably higher in colder water and with vigorous exertion. Onset of pulmonary oedema may occur any time during or soon after the dive. It does not appear to be a phenomenon associated with decompression, since almost all cases develop symptoms during the period at depth. It is characterised by dyspnoea and cough productive of white or pink frothy sputum. Rapid spontaneous recovery occurs after leaving the water in many cases. It appears that divers may return safely to diving after such an event but this should only occur after careful assessment for predisposing factors and appropriate counselling. This is a rare phenomenon whose pathophysiology is not clearly established.

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Audience participation

Davis, New Zealand: The incidence of secondary drowning has been raised. The figures quoted by Dr North were 5% from John Pearn's work in Australia on children,[1] and 25% from Modell's work in Florida.[2] It may be anywhere between these. There are only three useful definitions in the drowning literature as far as I am concerned; drowning which is death by immersion, near drowning which is survival from an immersion incident and secondary drowning which is a syndrome of pulmonary oedema as described here by Dr Mitchell. This latter definition has now gone out of favour, but secondary drowning may occur after a variable latent period of up to 24 hours following a near-drowning episode. It does not appear to be dependent on the severity of the initiating event. In other words it can be a life-threatening complication following a minor aspiration incident. I suspect that is what the case described suffered from. Pulmonary oedema following an immersion incident usually responds extremely rapidly and effectively to continuous positive airway pressure or to mechanical ventilation combined with positive end expiratory pressure.

On another subject, comment was made on buddy pairs dying. In the New Zealand series, there were three multiple drowning incidents, two buddy pairs and three out of a group of seven divers – the so called French Pass Incident.[3] SPUMS will hear more about this accident once the legal processes are complete, which could take several years. In this tragic diving accident, seven scuba divers were sucked to a depth of over 90 meters by strong currents. Amazingly, four of them survived, but three did not.

Mitchell, Brisbane: Apropos your comment about my misdiagnosis, I just wonder if you have ever seen a case of secondary drowning with florid symptoms that occurred in a victim who had absolutely no awareness of aspirating anything. (**Davis:** No) Secondly, I am not aware of any case of secondary drowning that occured with no latency at all. Of course the aspiration could have occurred early in the evening and there would have been a latent period but there was no awareness of any aspiration. Therefore, I do not agree with you.

Bennett, Sydney: You mentioned the question of returning to diving and the fact that at least some of the people you know returned to diving with no more events. Have you actually treated someone multiple times for emergent pulmonary oedema?

Mitchell: No I have not. None of the cases that I am aware of that went back to diving have suffered it again. However, two of them who had a single event, recovered and went diving again before they saw me, and developed the same problem again within days of the first episode. Clearly, they had not fully recovered and something was still predisposing them to the second event. No one who has had it, seen me, heard my advice, had some time off and then gone diving again, has had another event to my knowledge.

Bennett: My other question is, would you comment on the reason for being given 50% oxygen on arrival at the emergency department. When were you involved and what would your advice have been?

Mitchell: I was not involved acutely. I was contacted after one of the consultants suggested she might need recompression therapy, which I did not believe she needed. She had an adequate arterial O_2 on 50%, but I do not know why they chose that. If I had been managing her in the first aid setting, I would have put her on 100% oxygen but whether that would have been necessary I am not sure. Have we any emergency physicians here who might be able to cast any light on it? (No audience response.)

Brogan, Perth: It does not matter what you do with your gear, you can still run into trouble. I had mine serviced about three weeks before I came. I did about 10 dives before I left Australia. On the third dive here I had a first stage problem, total loss of air at 40 feet, and had to take off for the surface. So you can always be unlucky!

Bennett: A number of speakers have made the comment that they cannot understand how with modern dive training methods there could possibly be an out-of-air situation. Let me tell you a true story from about a week ago. This involved an experienced dive buddy pair, both of them in excess of 600 dives, and buddied up for most of that time. One of the divers had a progressive leak in the high pressure hose which became more and more obvious until one of the crew heard it, and drew her attention to it. The decision was made by the diver to just remove that hose and block the plug so now she had no contents gauge. About three dives later, the other diver, the one who actually had a contents gauge, noticed early in the dive it was a little difficult to breath. Looking at his contents gauge for the first time, it read zero, so he swam over to his buddy who did not have a contents gauge and pointed to his contents gauge and indicated it was a bit difficult for him to obtain air. The buddy's response was to tap the gauge and indicate it must be broken.

They then took off further down the reef, and at about 20 metres it was really very difficult for him to breathe. So he indicated to his buddy that he needed to breathe on her octopus. They successfully managed this manoeuvre without any problems, except, just as he was getting himself settled, she saw something of great interest lower down the reef and took off like a rocket, leaving him with only the rubber mouthpiece in his mouth. Now, he said, he was slightly concerned because he was not sure just how far away she was going. He managed to catch up with her and put what was left of the octopus in his mouth, in the process of which he dropped the mouthpiece he was holding. She then saw this and took off after it. By now she was at about 35 metres. He recovered from that, followed her down, and breathed on the octopus without the aid of a mouthpiece.

They then made their way up to about 20 meters and completed a pleasant 40 minute dive, both of them breathing from a tank with no contents gauge, having already realized that at least one of their tanks had not been filled prior to the dive. The most amazing thing about this story is their reaction when we all got back on the boat, which was to wonder why anybody thought this was a difficult problem because they felt they had dealt with it very well; it was a perfectly safe dive as far as they were concerned!

Thomas, Sydney: Dr Mitchell, I presume the cases you describe had normal electrolytes and renal function? Has anyone looked at renal function acutely in these patients and whether they produce normal amounts of urine, or whether they perhaps are concentrating? I would assume that if they have normal electrolytes then that does tend to make the diagnosis of either neardrowning or salt water aspiration unlikely.

Mitchell: Electrolytes were done on the case I presented and they were normal, but nothing more formal in terms of renal function tests was done. I would have to go back to

the literature to tell you whether or not they were done on some of the other reported cases. In most of the cases that I have they were not done. Many of them I saw in the context of their seeking an opinion on what happened and what they should do about it in future, so I was not involved in the acute management. There was one whom we admitted, who had a full blood count and renal function tests, which were normal, but I could not tell you about the other cases. As to whether or not all salt water aspirations result in abnormal renal function tests I am not sure about that. I was of the opinion that they did not necessarily.

Haller, Melbourne: With all these people running out of air, I was just wondering whether anyone has done any trials or whether anyone would know the physiology of how long someone would last using the air out of their mask? If you run out of air, could you breathe in and out of your nose to get to the surface using the air in your mask as a form of emergency air source?

Bennett: There is not enough volume in it to get past your dead space is one answer, I think. But it would help you feel more comfortable on the ascent essentially. Not a bad idea.

Jones, New Zealand: There was a technique widely touted 25 years ago of breathing from your buoyancy compensator (BC). But this was killed by the description of Key West scuba divers disease, which was Legionella basmaneii. It was recommended that you flush out your BC between dives with hypochlorite to sterilise it, just in case you needed to breathe from it again.

Bradley, Central Coast, NSW: Just to comment on safety. Often on these conferences we place reliance on our dive masters who in some cases may have a lot less experience than us, which comes back to having a good dive plan regardless of what dive masters are saying to us. A case in point occurred in Layang Layang two years ago, where the directions of the dive master were taken, when in fact it was quite an unsafe practice that was being proposed. In this case, the current picked up during the dive to a considerable rate. Against the strong current, the dive master was trying to home everyone back into a particular spot that we had dived several times before, whereas the safest practice was to go with the current and do our deco in mid-water, away from the reef. Half the team actually did that, while the other half struggled valiantly to stay on the reef. We all ended up doing mid-water deco.

Knight, Melbourne: One thing to remember is that as you rise in the water, the pressure differential between your tank and the second stage rises and you can get some air out of the tank. Divers should keep their regulators in their mouth if they run out of air and try to breath in and out on the ascent. If they did not have enough air to inflate their compensator, dropping the weight belt would make them buoyant. Encouraging this technique markedly reduced the incidence of injury and death from rapid ascents in one of the Canadian National Parks some decades ago.

Bennett: How many of you have run stone motherless cold out of air whilst diving? (Several members of the audience raise their hands.) So there are a few people amongst this group. None of you are diving tomorrow!

Carney, New Zealand: Dr Mitchell, are the people with pulmonary oedema that you have treated all similar in type or are they men and women of all ages?

Mitchell: That is one of the surprising things. It seems to be able to affect anybody but typically they're older, perhaps less fit, possibly with some sort of cardiac problem like hypertension or another predisposition. It is probably safest to just see it as something that can happen to anybody.

Robyn Walker, Sydney: We have had one clearance diver candidate on a four-hour surface swim to Manly and back, done in winter fully dressed in appropriate wetsuit, gloves and hood, pulled from the water halfway back in frank pulmonary oedema. Similar cases have been reported in swimmers as you described.[4] He was given oxygen and frusemide and made an uneventful recovery overnight. Intensive cardio-respiratory work up was normal and he was permitted to continue diving. On a repeat swim, he went into pulmonary oedema for the second time. He was superbly fit in all other respects and we found no other abnormality with him, but the Navy could not risk him every time he did a long surface swim going into pulmonary oedema.

Bennett: He presumably did the same level of exercise but in different settings and he was okay then?

Walker: Yes. Navy divers have a high physical activity load, and in part of his acceptance test he would have done a two-week course where we estimate they need about 10,000 calories a day just to get them through the level of physical activity that they do on that course. So he was superbly fit in other respects and, in fact, had no problem diving in water of the same temperature as on the long surface swim.

Davis: This is an interesting phenomenon. Because of my long-distance sea swimming activities when I was younger, I took a special interest in the physiological work on English Channel swimmers who spend many hours swimming in cold water.[5] I do not recall from that literature this phenomenon being reported at all, and this puzzles me. Dr Mitchell, do you remember whether in the Israeli swimmers paper, they were using fins, mask and snorkel or was it just ordinary swimming? In other words, were they like Dr Walker's case, or not? I wonder whether this is an issue of using swimming aids and protective suits that creates the difference.

Mitchell: I think they were using swimming aids, and were diver trainees like Dr Walker's diver.

Davis: It would be worth reviewing the old long-distance swimming physiology work to see whether it is documented anywhere. It is the use of swimming aids in one form or another that perhaps creates a different respiratory and cardiovascular workload which someone swimming without aids in cold water for long periods does not experience. Another interesting aspect is that many of these swimmers were not youngsters; Gerry Forsberg broke the two-way English Channel record in his late forties, for instance.

von Neullen, Holland: Dr Mitchell, could a contaminated air source be a cause of pulmonary oedema, and have you ever seen such cases?

Mitchell: You are absolutely right. If you inhale irritants or noxious substances you can definitely suffer pulmonary oedema. The air in this patient's cylinder was tested and no contaminants were found. So it was not the problem in her case. I personally have not seen any diver suffer pulmonary oedema because of contaminants in the breathing mix but I believe such cases have been reported in the literature.

Thomas: Do you know if they routinely test for oxides of nitrogen in the air? (**Mitchell:** I do not think so.) These are described as a cause of pulmonary oedema. Her air was probably tested for carbon monoxide and hydrocarbons as it was a diving situation. Presumably though, you would expect a large number of people to be affected if the system was contaminated.

Deborah Yates, Sydney: It is highly unlikely that they would have been able to test for oxides of nitrogen because it is difficult to get hold of the equipment. We have recently seen some cases in blast furnace workers, but we had considerable difficulty measuring levels. Quite variable levels were seen (paper in preparation).

Thomas: If you measure the levels of nitric oxide in hospital compressed air they vary with the urban pollution. We have recorded up to about 300 parts per billion in our hospital air. This is probably not important, but you do not know what else is being compressed out of the atmosphere to give to the patients. Oxides of nitrogen have been described as being present in ventilator circuits in intensive care units.

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Salt water aspiration syndrome

Simon Mitchell

Key words
Salt water aspiration syndrome, near drowning, diving, scuba diving

Abstract
A "salt water aspiration syndrome" has been described in scuba divers. It is characterised by early cough, a latent period of one to 15 hours, followed by a productive cough, retrosternal chest pain, dyspnoea, shivering, nausea, vomiting and constitutional symptoms such as malaise and fever. It could be a mild form of near drowning, a reaction to the inhalation of irritants or micro-organisms, or due to occult nebulisation of salt water.

Salt water aspiration syndrome (SWAS) was first described by Carl Edmonds.[1] He described 30 cases in military divers characteriaed by an early post-dive cough and then, after a latency which might be one to 15 hours, a productive cough, dyspnoea, shivering, nausea, vomiting and various constitutional symptoms such as malaise and fever. One of the defining symptoms was retrosternal pain, which he reported as being present in 90% of divers. Typical physical findings were lung crepitations and patchy consolidation on the chest X-ray in 50% of patients. The cases reported all recovered over 24 hours with treatment on 100% oxygen and little else. Since then, there have been various anecdotal

case reports, including from South Australian abalone divers (Acott C, personal communication) who believe they suffer SWAS from time to time.

The pathophysiology of SWAS is probably similar to that of near drowning, but at a lower level of severity. For example, the presence of salt water in the alveoli might dilute or destroy pulmonary surfactant, thus causing atelectasis. This, in turn, would cause a cough and possibly dyspnoea, and the patchy changes that are seen on chest X-ray. These radiological changes differ from the typical pulmonary oedema pattern most commonly seen in near-drowning cases. Hypertonic saline (sea water) and the various irritants, particulate matter or marine organisms contained in it, may elicit an inflammatory response. This might further contribute to alveolar collapse and also might explain the fever and malaise. The delay in developing such a response could account for the latent period described by Edmonds. Hypertonic salt water may also exhibit an osmotic attraction of fluid into the alveolar space as described in this issue by North.[2]

Given the obvious similarities with near drowning, what then are the differences between SWAS and near drowning? It may simply be a matter of nebulised water volume. Near drowning is considered to involve the aspiration of moderate amounts of water into the lungs and does not typically involve the systemic symptoms with a long latency described by Edmonds. Salt water aspiration syndrome probably involves more subtle or even occult aspiration of which the diver might not be aware. Such aspiration might occur in those situations typically associated with near drowning, such as panic at the surface, out of air situations, loss of the regulator and any unconsciousness when immersed.

Subtle or occult aspiration might occur via nebulisation of salt water with a leaky regulator diaphragm, a leaky exhaust valve or mouthpiece tears. Such equipment problems do arise, and most experienced divers have noticed the slightly wetter feeling of breathing air under those circumstances. Whether or not this results in an aspiration syndrome is not clear. The nebulisation of droplets to traverse the airways down into the respiratory tissue is an exacting science. Nevertheless, it seems plausible that a regulator nebulising water particles across a wide range of sizes will produce some of the right size to pass into the distal airways.

Another issue that arises is whether pathogenic organisms might be involved. Dr Jones alluded to this in the discussion with respect to Key West scuba divers disease (page 204, this issue). An alternative infectious theory has been proposed by Bradley and others.[3] They described a similar syndrome, although typically with a longer latency and less shortness of breath, that was attributed to contamination with various organisms, most commonly *Pseudomonas spp.*, and their endotoxins. These were isolated from the breathing circuits of diving equipment. One of the cases reported died due to *Pseudomonas* infection.

Salt water aspiration syndrome may be a heterogeneous collection of problems with different causes but similar manifestations. It could be a mild form of near drowning, a reaction to the inhalation of irritants or, indeed, be due to occult nebulisation of salt water. It is conceivable that this was true even for the cohort of divers described originally by Edmonds.[1] This author has only very rarely seen cases where the classic pattern of SWAS was followed. This may be because of spontaneous recovery and under-reporting; in other words, these divers might often not come to the attention of diving physicians.

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Audience participation:

Knight, Melbourne: At the time Carl Edmonds described this syndrome, the Royal Australian Navy was using the Porpoise Regulator. During the divers' training they were expected to disassemble and reassemble it while holding their breath. I have had salt water aspiration twice. Once was on the diving medical officers course. After buddy breathing in a salt water swimming pool, three of us developed symptoms overnight. We all improved on oxygen. The next time was in Thailand. There was a lot of particulate matter in the sea and my regulator had a faulty exhaust flap valve. My own view of the aetiology is that the cause is primarily particulate matter in the water, especially plankton, combined with a wet regulator.

Thomas, Sydney: In subjects who have inhaled 4.5% saline in challenge tests, we have demonstrated an airway neutrophilic infiltrate at 18-24 hours. This can be associated with an increase in airway reactivity. In addition, salt water would induce a mild peripheral inflammation and restrictive lung pattern. Thus, this challenge would be similar to the process experienced by those with salt water aspiration, who probably also have a neutrophilic inflammatory lung infiltrate. It can be very mild and disappear within 24 hours.

SPUMS Annual Scientific Meeting 2002

Traveller's diarrhoea

Trish Batchelor

Key words
Travel medicine, traveller's diarrhoea, tourism

Abstract
Every year more than 30 million people from developed countries travel to less developed countries for business, pleasure or adventure. Numerous studies have shown that even on trips of short duration, up to 50% of these travellers will experience some form of illness whilst abroad, or on their return home. Many of these travellers will present to physicians on their return home, hence an understanding of common travel health problems is an essential aspect of many fields of medicine, particularly general practice, infectious diseases and emergency medicine. The most common health problem to affect travellers is diarrhoea. Traveller's diarrhoea is predominantly bacterial in origin and causes significant morbidity. Despite a variety of preventative measures that can be put in place, most travellers are non-compliant with these. However, this type of advice may not necessarily provide protection anyway. An aggressive approach to treatment utilising antibiotics is commonly recommended both in adults and children. Maintenance of hydration is also important.

Introduction

> "Health is infinite and expansive in mode and reaches out to be filled with the fullness of the world, whereas disease is finite and reductive in mode and endeavors to reduce the world to itself."
>
> Oliver Sacks

Each year, more than 30 million people from industrialized countries travel to a developing country for business or leisure.[1] On doing so, they potentially expose themselves to sudden changes in climate, altitude, microbial flora and traffic conditions. These factors, combined with stress and fatigue may easily result in illness or injury whilst abroad. In fact, numerous studies have shown that the rate of illness in travellers varies from 36–75%.[2-4] The key factors determining the health risks to which a traveller will be exposed, include the destination, duration, accommodation, standards of food hygiene in the destination, and the individual traveller's risk-taking behaviour.[5]

In all epidemiological studies of travellers' health problems, gastrointestinal disorders are the most common complaint; primarily traveller's diarrhoea (TD). One can compare data collected from long-term Peace Corp workers,[3] leisure travellers,[1] Royal Geographic Society expedition members,[6] and members of a Mount Everest mountaineering expedition,[7] and find that gastrointestinal complaints dominate in all groups (Figures 1 and 2).

Interestingly, respiratory problems are consistently the second most common problem in travellers and account for around 25% of complaints in all groups. There is, however, a significant difference in the less common complaints when one compares the groups. As one might expect, the Everest mountaineers have a much higher rate of environmental problems such as altitude sickness.[7] Royal Geographic Society expeditioners had the highest rate of accidents[6] and long-term Peace Corp workers had significantly more skin and psychological problems than other groups.[3] This paper will focus on the most common of traveller's complaints – traveller's diarrhoea.

Epidemiology

Diarrhoea is the most common health problem faced by travellers. Depending on the destination, up to 50% of travellers can expect to have an episode of TD on a two-week trip. TD is currently defined as more than four loose stools in 24 hours, or more than three loose stools in 24 hours plus at least one of the following complaints: nausea, vomiting, abdominal cramps, fever, faecal urgency, abdominal pain, tenesmus, bloody or mucous stools.[8] Three general geographic zones of risk have been identified. Low risk areas (<10%) include Australasia, North America, Central and North Europe, and Japan. Intermediate risk areas (15–20%) include Russia, the Caribbean, Israel, Eastern and Southern Europe, South Africa and China, whilst high-risk areas (20–50%) include the developing countries of Africa, South and Central America and Asia.[9] In very high-risk areas this risk persists for a prolonged period. A study of long term expatriates in Nepal demonstrated a 50% per month risk of an episode of diarrhoea that persisted for at least the first two years of their stay.[10]

Whilst TD is often considered a trivial illness by doctors in developed countries, it can in fact be debilitating and usually occurs in unfamiliar surroundings and often with limited access to reliable medical care. Nearly 40% of those who

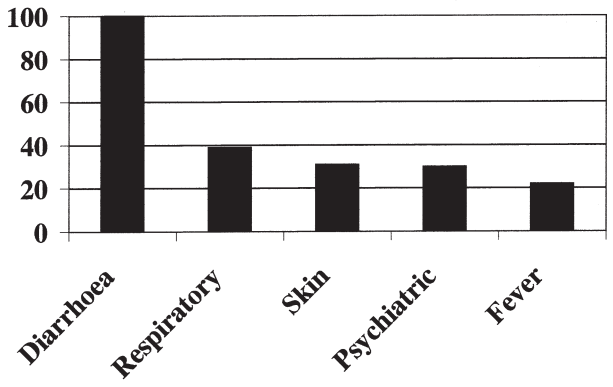

FIGURE 1
HEALTH EVENTS PER 100 PEACE CORP VOLUNTEERS PER YEAR

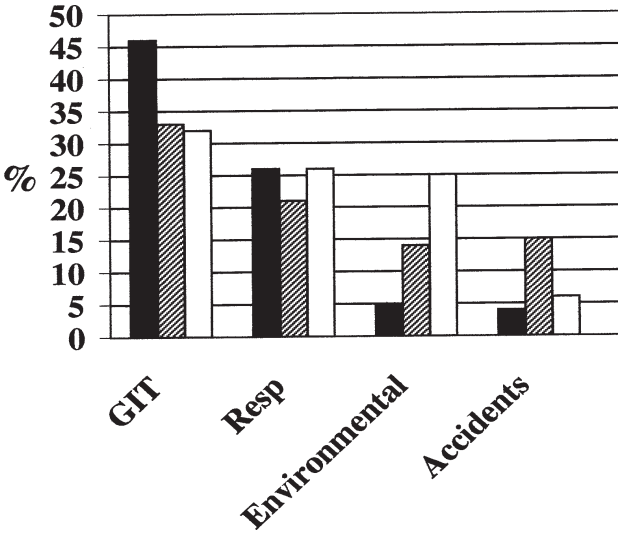

FIGURE 2
HEALTH EVENTS IN THREE TRAVEL GROUPS
USA Tourists - black
Royal Geographic Society Expedition - hatched
Mount Everest Climbers - open
(GIT - gastrointestinal tract; Resp - respiratory)

develop TD are forced to modify their activities (often at great expense), at least 20% are confined to bed for a day or more, and 1% are hospitalized.[9]

Greg Child, one of the world's top mountaineers, describes his experience of TD in Pakistan thus:

> "The next phase of the expedition is ambitious – climb Rakaposhi, a huge mountain reaching into sky and cloud. But ambition has left, replaced by unshakeable illness in us all. A combination of dehydration and dysentery flattens me, and I end up back in Karambad, in hospital for the day, and I'm prostrate for a week. It was a small thing that entered our gut, bacterial in dimension, but devastating out of all proportion to its size."[11]

Apart from the obvious discomfort of an episode of TD, complications can occur such as chronic diarrhoea (in 3% of individuals), Reiters syndrome, problems resulting from the impaired absorption of regular medications and there is the risk of dehydration in susceptible individuals such as children, the elderly, the pregnant, and diabetics.

Prevention

Prevention of TD has traditionally centered on patient education and avoidance of 'risky food and water'. In general, contaminated food is more commonly a cause for diarrhoea than contaminated water. However, there are numerous pathogens that may be spread by contaminated water, so travellers should still be advised to avoid untreated water. Boiling is the most effective method of making contaminated water safe to drink. It needs only be brought to the boil, even at altitude.[12] Halogens such as iodine and chlorine are useful against all but the parasites Cyclospora and Cryptosporidium – travellers to areas with these pathogens (eg. Nepal from May – September) should be aware that using halogens alone will provide inadequate protection. Bottled water may not be reliable – carbonated drinks are safer and the seal should be checked on any bottled drink.

Travellers are always advised to avoid salads, uncooked food or unpeeled fruit. This is best summarised as, "Cook it, peel it, boil it or forget it".[13] Does this advice work and are travellers compliant? A Swiss study of short-term travellers to moderate- and high-risk destinations showed that only 2% of people adhered consistently to these recommendations. Within three days of arrival in their destination, 53% had consumed ice in their drinks and 73% had eaten salads or raw vegetables.[13] Clearly, compliance is a problem, however following the recommendations may not necessarily provide protection anyway.

A study on diarrhoea in long-term expatriates in Nepal, a highly endemic environment, showed that eating out in restaurants was the dominant risk factor for developing diarrhoea, independent of the type of food consumed.[10] The authors state,

> "This study suggests that travellers should be informed about the ubiquitous exposure to pathogens and the fact that the dominant risk factors for diarrhoea are difficult to modify (younger age, duration of stay, seasonality, and eating meals in restaurants)."[10]

This would not suggest that it is wise to eat with impunity overseas, but to be aware that despite one's best efforts, illness may occur. There are some foods known to be of particular risk, shellfish being the most important of these.

Aetiology

The dominant aetiology of TD is bacterial, with enterotoxigenic Eschericia coli (ETEC) being the dominant pathogen in most studies. Table 1 shows accumulated aetiology data from a number of studies.[14-16] The aetiology does vary significantly, however, from country to country. For example, data being collected currently in a travel medicine centre in Nepal, shows the dominant pathogen is *Campylobacter*, with ETEC following closely behind (Pandey P, Cave W, The CIWEC Clinic Kathmandu, personal communication). Because of this dominant bacterial aetiology, in the absence of good medical and diagnostic facilities, travellers are advised to self-treat using algorithms and medical kits prepared at home.[17]

Children with TD

Children appear to be even more susceptible to TD than adults. The approach to TD in children has traditionally been the same as if they were not travelling. However, attitudes to management are now changing. Although there are only limited data on TD in children, one Swiss study showed that children between the ages of three months and three years had a high incidence of diarrhoea and tended to have both severe and prolonged disease. In children of this age group, TD persisted for a median of 18 days and an average of 30 days.[18]

It may be difficult to establish whether a child has diarrhoea. In 1985, the National Institute of Health Consensus Report defined TD in children as characterised by a twofold or greater increase in frequency of unformed stools.[19] Increasingly, it is being recommended that if children have TD that fits the current criteria that they should be treated, like adults, with antibiotics (see below).

TABLE 1
RATES OF PATHOGENS ISOLATED IN TRAVELLER'S DIARRHOEA
(The rates for *Cyclospora*, *Cryptosporidium* and viral infections are unknown)

Pathogen	Average rate (%)	Range
ETEC*	50	30–75
EAEC**	15	5–30
Shigella	10	0–30
Campylobacter	<5	0–15
Salmonella	<5	0–10
Giardia	<5	0–10
E. histolytica	<3	0–5
None	20	10–40

* Enterotoxigenic *E. coli*
** Enteroadherent *E.coli*

Treatment

TD is usually a self-limiting condition, but because it often occurs in areas with unfamiliar or inadequate medical facilities it is potentially very disruptive. For this reason, travellers are advised regarding empiric treatment if no reputable facility is available. Management of traveller's diarrhoea comprises hydration, symptomatic relief and definitive treatment. In all cases, travellers are advised to use a rehydration solution such as Gastrolyte to avoid dehydration and the problem of requiring intravenous fluids in a potentially unsterile medical environment.

If the TD is mild, ie. fewer than three motions in 24 hours and without other symptoms, either rehydration alone, or the addition of Loperamide if the diarrhoea is inconvenient, are recommended. If the criteria for TD are met, initial treatment is with a fluoroquinolone such as Norfloxacin (400 mg BD for three days) or Ciprofloxacin (500 mg BD for three days).[19,20] If this treatment does not resolve the problem, the traveller should seek medical advice to exclude a fluoroquinolone-resistant bacterial diarrhoea or parasitic cause for their symptoms.

Azithromycin, at a dose of 500 mg daily for three days is the drug of choice for fluoroquinolone-resistant *Campylobacter*.[20] Unfortunately, it can be difficult to find medical facilities abroad with adequate laboratory support to provide this information. For high-risk destinations and activities such as trekking in the Himalaya, Tinidazole may be trialed for presumed *Giardia* infection.

In children, the drugs of choice are also Ciprofloxacin and Azithromycin. In Australia and New Zealand, Ciprofloxacin is not generally used in children. However, in many countries it is used in prolonged courses for children with chronic conditions such as cystic fibrosis with no apparent adverse effects.[21] Azithromycin is an excellent alternative, which has been shown to be particularly effective against fluroquinolone-resistant *Campylobacter* and *Shigella spp*.[20,22,23] However, it is not available in paediatric formulations in Australia or New Zealand.

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Medical kits for travellers

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Key words
Medical kits, travel medicine, tourism

Abstract
As health problems are so common in travellers, and reliable medical facilities are often unavailable in less developed countries, travel medicine specialists advocate the use of personal medical kits. These kits are designed to allow the traveller to self-treat common minor conditions when they are in the not uncommon situation of having no access to reliable medical care. Another concern in less developed countries is the proliferation of fake or substandard medications.

Millions of people each year leave their homes to travel, many of them to less developed areas of the world. Multiple studies have shown an illness rate of 50% for travellers on a short trip to less developed countries. It is clear that with such a high rate of illness abroad, traveller's medical kits are an important aspect of pre-travel preparation. In many less developed countries it is difficult, if not impossible, to access reliable medical care. This is particularly true if one is in a remote location undertaking adventure activities such as trekking, rafting, diving, etc.

Additionally, there is a thriving trade in both fake and substandard medications in many parts of the developing world. The World Health Organization estimates that 15%

of the world's drugs are substandard. Several recent studies show the problem is widespread. In an examination of one of the newer malaria treatments, Artesunate, samples from a number of countries in South East Asia were analysed and it was found that 38% of samples had no active ingredient.[1] A 1999 study of 242 Cambodian pharmacies found that 71% sold fake Artesunate and 60% sold fake Mefloquine.[2] In Nigeria, 48% of samples of 27 different drugs were found to be substandard.[3]

Travellers' medical kits should be designed to provide medications and instructions for the treatment of the most common conditions to be expected for that individual-based on their activities, season, duration and style of travel, access to reliable medical care and past medical history. A kit for a one-week stay in a diving resort in Thailand will be vastly different from that prepared for a group of six trekking through a remote area of the Indian Himalaya for one month, or a group of white-water kayakers in Costa Rica.

Table 1 shows medications that may be included in a medical kit for travel to a remote area in a less developed country. For a shorter trip to a less remote area, a medical kit is more likely to focus on the two most common health problems in travellers: diarrhoea and respiratory infections. Environmental ailments such as altitude sickness are an increasing problem as access to previously remote areas of the world opens up to more and more people.[4]

Travellers should be aware that the majority of ailments they may experience overseas cannot be prevented by vaccination. They should understand the behavioral adjustments they should make, but they should also understand the limitations of such methods of prevention, not to mention the problems with compliance. For this reason, all travellers should have a medical kit to take with them, prepared by their personal doctor or a travel medicine specialist. The extent of this kit will depend upon the individual characteristics of the traveller and their trip.

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TABLE 1
MEDICATIONS FOR PERSONAL MEDICAL KIT SUGGESTIONS
(ordinary first aid and anti-malarials not included)

CIRCUMSTANCES	MEDICATIONS RECOMMENDED
Gastrointestinal	Gastrolyte, Loperamide, Buscopan, Prochlorperazine, Coloxyl, Mylanta, Norfloxacin (or Ciprofloxacin or Azithromycin or Cotrimoxazole), Tinidazole
Respiratory	Paracetamol, Ibuprofen, Pseudoephedrine or decongestant nasal spray, Promethazine, Roxithromycin (or Amoxicillin/Clavulanate), throat lozenges
Skin	Steroid cream, Cephalexin, Betadine
Female	Diflucan, BHCG test kit, Postinor, Ural
Environmental	Sunscreen, DEET-containing insect repellant, condoms
Altitude	Acetazolamide
Climbing	Nifedipine, Dexamethasone, Codeine, Rectinol
Diving/surfing	Doxycycline, Ciprofloxacin, Bactroban, Soframycin drops, Pseudoephedrine or decongestant nasal spray, anti-motion sickness agent, normal saline for irrigation, wound irrigation syringe

Reprints from other journals

Experienced, recreational scuba divers in Australia continue to dive despite medical contra-indications

David McD Taylor, Kevin S O'Toole and Christopher M Ryan

Key words
Scuba, diving, medical examination, medical conditions and problems

Abstract

Objective: In Australia, a medical examination is required prior to undertaking a scuba diving course in order to screen for contra-indications to diving. No further medical screening is required, yet divers may develop diseases during their diving careers. This study aimed to survey experienced recreational scuba divers to determine the prevalence of diseases contra-indicated in diving.
Methods: A cross-sectional, postal survey of divers belonging to scuba diving clubs across Australia.
Results: 346 divers returned completed questionnaires. 254 (73.4%) were male and 258 (74.6%) were aged 31–60 years. The mean years of diving was 10.6 ± 9.18 years and the mean number of dives undertaken was 414 ± 740 dives. 162 (46.8%) divers were overweight, 45 (13.0%) divers required regular medication and 39 (11.3%) divers smoked. 36 (10.4%) divers reported a past or present history of asthma and the same number reported hypertension or coronary heart disease. 86 (24.9%) divers reported past or present psychological symptoms. 42 (12.1%) divers reported hearing difficulties and 81 (23.5%) divers reported past or present tinnitus. Two divers had a past history of epilepsy, two had a history of pneumothorax and one diver was diabetic.
Conclusions: Experienced, recreational scuba divers continue to dive despite medical contra-indications. This raises the questions: Did the divers fail to disclose these conditions at the initial examination or did these conditions develop subsequently? Is the risk associated with these conditions clinically significant or should screening examinations be undertaken at regular intervals? The high prevalence of hearing difficulties and tinnitus may be the result of aural barotrauma and requires further research.

Introduction

Scuba diving is a popular but high-risk sport.[1,2] In Australia, medical practitioners screen diving candidates for medical conditions thought to increase the risks of diving.[1,3] Today, standards for fitness to scuba dive are described in the Australian Standards[4] and guidelines for medical examinations have been published.[1,2,5]

Traditionally, candidates with medical conditions contra-indicated in diving (eg. asthma, diabetes, epilepsy) have been excluded from the sport via the medical examination. However, the screening medical examination may not detect such conditions[6] and practitioner's determination of fitness to dive may be inconsistent.[7] Also, anecdotal and some published evidence suggests that divers with contra-indicated conditions tend to remain covert and may falsify their medical history in order to dive.[6,8]

In Australia, data on the prevalence of contra-indicated conditions among divers are scant. In particular, the fitness to dive of experienced recreational divers is not known. Importantly, this group may have been screened and certified as fit at the beginning of their diving careers but may have developed contra-indications that have subsequently placed them at increased risk.

This study aimed to examine the health of experienced, recreational scuba divers in Australia and to determine the prevalence of medical conditions which preclude diving within this group. These data will assist in evaluating the effectiveness of the initial medical screening process, the need for repeated medical examinations and the safety of these conditions among experienced, recreational divers.

Methods

The study was a cross-sectional survey of active, experienced scuba divers belonging to scuba diving clubs across Australia. A list of clubs, published on the "Diving in Australia" internet website,[9] was used to identify sources of potential subjects. E-mail or telephone contact with each club's contact person (president or secretary) was attempted. Forty eight clubs were contactable and all agreed to participate in the study.

In June 2000, each club's contact person was mailed a variable number of study questionnaires depending on the

TABLE 1
GENDER AND AGE OF THE SUBJECTS (n = 346, percentages in parentheses)

	Subject age group (years)						
	18–20	**21–30**	**31–40**	**41–50**	**51–60**	**61+**	**Totals**
Male	5 (1.5)	44 (12.7)	85 (24.6)	69 (19.9)	46 (13.3)	5 (1.5)	254 (73.4)
Female	5 (1.5)	28 (8.1)	35 (10.1)	16 (4.6)	7 (2.0)	1 (0.3)	92 (26.6)
Totals	10 (2.9)	72 (20.8)	120 (34.7)	85 (24.6)	53 (15.3)	6 (1.7)	346 (100)

number of active members. These were distributed at the next club meeting, completed immediately and returned by the contact person in a stamped, addressed envelope. Two months after the initial mailing, clubs that had not returned questionnaires were mailed a reminder letter. This was repeated in one further month's time, when necessary.

The study questionnaire was anonymous and designed by the authors. The first section collected demographic data on the divers including diving experience. The second section consisted of a list of medical conditions, each with the potential to interfere with a diver's activity and potentially compromise safety. This list was derived from published sources of information relating to medical fitness to dive.[1,3-5] The divers were asked to record when they had ever experienced each of the conditions. The possible responses were: "never", "in the past" and "now and I dive with it". Only one response was permitted for each condition.

Body Mass Index (BMI) was calculated by the investigators and based upon reported weight and height values. The following classifications were used: BMI <20 (underweight), 20–24.9 (normal), 25–29.9 (overweight), 30–34.9 (obese) and ≥35 (morbid obesity).

Most responses were analysed descriptively. Mean values are reported with standard deviations. Comparison of proportions utilised the Yate's corrected chi-square test with EpiCalc, version 1.02 (2000) software.

The study was authorised by the Institutional Review Board (Ethics Committee) of the University of Pittsburgh, PA, United States of America.

Results

Of the 48 clubs that initially agreed to participate, 29 clubs (60.4%) ultimately returned questionnaires. (New South Wales 10, Victoria 7, Western Australia 4, Northern Territory 3, South Australia 2, Tasmania 2, Queensland 1.) A total of 346 questionnaires were returned.

The divers' gender and age are described in Table 1. Almost three quarters were male and most (59.3%) were middle-aged (31–50 years). As expected, most were experienced scuba divers. The mean "years of diving" was 10.6 ± 9.18 years (median 7.5) and the mean "number of dives done" was 414 ± 740.82 dives (median 200).

Table 2 describes factors associated with the divers' socioeconomic status. Most divers were university graduates or postgraduates (206 divers, 59.6%) and were in white collar or professional employment (208 divers, 60.1%).

Table 3 describes the divers' risk factors for illness. Almost one half were classified as overweight or greater (162 divers, 46.8%). Importantly, eight divers (2.3%) were morbidly obese. Most divers admitted to a small (=5 drinks per week) or moderate alcohol intake (6–10 drinks per week) with only 42 divers (12.1%) consuming more than 15 drinks per week. The number of smokers in the group was relatively small and approximately the same number reported illicit drug use (marijuana, amphetamines, LSD). Only 53 divers

TABLE 2
SOCIOECONOMIC STATUS OF SUBJECTS (%)

Highest level of education completed		
Primary school	8	(2.3)
High school	131	(37.9)
University	157	(45.4)
Postgraduate	49	(14.2)
No response	1	(0.3)
Total	346	(100)
Employment status		
Unemployed	24	(6.9)
Blue collar/trade	104	(30.1)
White collar/clerical	53	(15.3)
Professional	155	(44.8)
No response	10	(2.9)
Total	346	(100)

TABLE 3
RISK FACTORS FOR ILLNESS AMONG THE DIVERS, n = 346 (%)
(* marijuana 28, amphetamines 3, LSD 2, not specified 4
OCP - oral contraceptive pill)

The body mass index (BMI):		
Underweight (<20)	9	(2.6)
Normal (20–24.9)	157	(45.4)
Overweight (25–29.9)	118	(34.1)
Obese (30–34.9)	36	(10.4)
Morbid obesity (=35)	8	(2.3)
No response	18	(5.2)
Number of alcoholic drinks per week:		
None	28	(8.1)
<1	52	(15.0)
1-5	120	(34.7)
6-10	62	(17.9)
11-15	40	(11.6)
15+	42	(12.1)
No response	2	(0.6)
Smoking status:		
Non smoker	294	(85.0)
Smoker	39	(11.3)
No response	13	(3.8)
Illicit drug use:		
Not used	303	(87.6)
Used*	37	(10.7)
No response	6	(1.7)
Daily medication use:		
OCP only	8	(2.3)
OCP plus other medication	1	(0.3)
Other medication	44	(12.7)

reported taking daily medication.

Table 4 describes the medical conditions, experienced by some divers, generally regarded as relative or absolute contra-indications to diving. Respiratory and upper respiratory diseases predominated. Seasonal allergies were common with almost half of all divers having either a history or active disease. Importantly, 28 divers (8.1%) had a history of asthma or chronic obstructive airways disease and an additional 10 divers (2.9%) were current sufferers. As expected, pressure equalising problems were common, with 50 divers (14.5%) continuing to dive despite this difficulty. Tympanic membrane rupture was not uncommon. However, as with the two cases of round or oval window rupture, it is not known if these injuries were caused by barotrauma whilst scuba diving. Also of importance is the finding that two divers reported a history of pneumothorax.

A history of neurological conditions was reasonably common although prevalent illness was confined mainly to recurrent headaches (19 divers, 5.5%) and migraine (13 divers, 3.8%). No diver reported suffering from epilepsy at the time of the survey although two divers had a past history of this condition. Single divers reported a history of brain tumour, TIAs and brain aneurysm, and five divers reported a history of significant head trauma. Thirteen divers (4.8%) reported having suffered from decompression illness. Cardiac conditions were relatively uncommon and mainly confined to hypertension (12 divers, 3.5%). It is possible that some divers who reported a history of hypertension were, in fact, being treated for this condition. Importantly, three divers reported a history of angina. The nature of the cardiac arrythmias reported is unknown. Many divers reported a history of psychiatric illness and, importantly, 14 divers (4.0%) continued to dive with either claustrophobia or anxiety. Only one diver suffered from diabetes at the time of the survey.

Table 5 describes medical conditions, experienced by some divers, not generally regarded as relative or absolute contra-indications to diving. However, these conditions may interfere with a diver's activity or may be aggravated by diving. Tinnitus was surprisingly common with almost one quarter of all divers having a history or presently suffering from this condition. Hearing loss was also reported more than expected with 42 divers (12.1%) reporting this condition.

As expected, many medical conditions listed on the questionnaire had not been experienced by any of the divers. These included myaesthenia gravis, stroke, multiple sclerosis, paraplegia, trigeminal neuralgia, congestive cardiac failure, mitral valve prolapse, cardiac valve stenosis, pulmonary blebs or cysts, emphysema, lung surgery, ulcerative colitis, kidney failure or transplant, osteonecrosis, limb amputation, haemophilia, leukaemia and sickle cell disease.

Discussion

The response rate of participating dive clubs was rather poor. However, the divers in this study are likely to represent experienced, recreational divers belonging to dive clubs in Australia. Each of the States and Territories was represented in approximate proportion to its population, with the exception of Queensland which was under-represented. This is of importance as much of the diving in Australia is undertaken in Queensland. The finding that most divers were male, middle-aged, and had logged a considerable number of dives over many years, is consistent with a United States (US) study of experienced, recreational divers.[10] The high socioeconomic class of most divers is consistent with their ability to continue participation in this relatively expensive sport.

Although almost half of the divers were overweight, this

TABLE 4
REPORTED MEDICAL CONDITIONS THAT ARE RELATIVE AND ABSOLUTE CONTRA-INDICATIONS TO DIVING (n=346, percentages in parentheses, DCI - decompression illness)

Disease system	Past history of disease			Presently suffers from disease		
	Male	Female	Total	Male	Female	Total
Respiratory						
Seasonal allergies	54 (15.6)	18 (5.2)	72 (20.8)	54 (15.6)	25 (7.2)	79 (22.8)
Asthma	21 (6.1)	6 (1.7)	27 (7.8)	5 (1.5)	4 (1.2)	9 (2.6)
Chronic obstructive airways disease	-	1 (0.3)	1 (0.3)	1 (0.3)	-	1 (0.3)
Spontaneous pneumothorax	-	1 (0.3)	1 (0.3)	-	-	-
Traumatic pneumothorax	1 (0.3)	-	1 (0.3)	-	-	-
Eye, ear, nose and throat						
Equalising problems ears/sinuses	68 (19.7)	22 (6.4)	90 (26.0)	31 (9.0)	19 (5.5)	50 (14.5)
Chronic sinusitis	9 (2.6)	1 (0.3)	10 (2.9)	7 (2.0)	5 (1.5)	12 (3.5)
Diving within 12 months eye surgery	5 (1.5)	2 (0.6)	7 (2.0)	2 (0.6)	-	2 (0.6)
Chronic otitis media	10 (2.9)	2 (0.6)	12 (3.5)	1 (0.3)	-	1 (0.3)
Eardrum rupture	17 (4.9)	4 (1.2)	21 (6.1)	-	-	-
Inner ear surgery	5 (1.5)	-	5 (1.5)	-	-	-
Round or oval window rupture	2 (0.6)	-	2 (0.6)	-	-	-
Penetrating eye injury	1 (0.3)	-	1 (0.3)	-	-	-
Neurological						
Recurrent headaches	36 (10.4)	14 (4.1)	50 (14.5)	10 (2.9)	9 (2.6)	19 (5.5)
Migraine headaches	38 (11.0)	18 (5.2)	56 (16.2)	5 (1.5)	8 (2.3)	13 (3.8)
Brain/spinal cord disease or trauma	5 (1.5)	-	5 (1.5)	3 (0.9)	-	3 (0.9)
Spinal cord injury (temporary)	5 (1.5)	-	5 (1.5)	2 (0.6)	-	2 (0.6)
DCI (with nerve damage)	10 (3.9)	2 (0.6)	12 (3.5)	1 (0.3)	-	1 (0.3)
Brain tumour	-	1 (0.3)	1 (0.3)	-	1 (0.3)	1 (0.3)
Epilepsy	1 (0.3)	1 (0.3)	2 (0.6)	-	-	-
Head trauma (unconscious >24 hours)	4 (1.2)	1 (0.3)	5 (1.5)	-	-	-
Transient ischaemic attack	1 (0.3)	-	1 (0.3)	-	-	-
Brain aneurysm	1 (0.3)	-	1 (0.3)	-	-	-
Meniere's disease	2 (0.6)	-	2 (0.6)	-	-	-
Cardiac						
Hypertension	18 (5.2)	3 (0.9)	21 (6.1)	10 (2.9)	2 (0.6)	12 (3.5)
Cardiac arrythmia	3 (0.9)	-	3 (0.9)	3 (0.9)	-	3 (0.9)
Patent foramen ovale	-	1 (0.3)	1 (0.3)	2 (0.6)	-	2 (0.6)
Coronary heart disease	1 (0.3)	-	1 (0.3)	2 (0.6)	-	2 (0.6)
Cardiac valve incompetence	-	-	-	1 (0.3)	-	1 (0.3)
Angina	3 (0.9)	-	3 (0.9)	-	-	-
Myocardial infarction	1 (0.3)	-	1 (0.3)	-	-	-
Psychiatric						
Claustrophobia	7 (2.0)	5 (1.5)	12 (3.5)	5 (1.5)	3 (0.9)	8 (2.3)
Anxiety	17 (4.9)	5 (1.5)	22 (6.4)	3 (0.9)	3 (0.9)	6 (1.7)
Depression	20 (5.8)	9 (2.6)	29 (8.4)	1 (0.3)	1 (0.3)	2 (0.6)
Drug or alcohol abuse	9 (2.6)	-	9 (2.6)	2 (0.6)	-	2 (0.6)
Panic disorder	6 (1.7)	1 (0.3)	7 (2.0)	-	-	-
Schizophrenia	3 (0.9)	-	3 (0.9)	-	-	-
Miscellaneous						
Joint surgery or trauma	17 (4.9)	3 (0.9)	20 (5.8)	8 (2.3)	3 (0.9)	11 (3.2)
Diabetes	-	-	-	1 (0.3)	-	1 (0.3)
Bleeding gastric or duodenal ulcer	5 (1.5)	-	5 (1.5)	1 (0.3)	-	1 (0.3)

TABLE 5
REPORTED MEDICAL CONDITIONS OTHER THAN RELATIVE AND ABSOLUTE CONTRA-INDICATIONS TO DIVING
(n=346, percentages in parentheses, TMJ - temperomandibular joint)

	Past history of disease			Presently suffers from disease		
Disease state	**Male**	**Female**	**Total**	**Male**	**Female**	**Total**
Tinnitus	31 (9.0)	11 (3.2)	42 (12.1)	33 (9.5)	6 (1.7)	39 (11.3)
Hearing loss	8 (2.3)	5 (1.5)	13 (3.8)	26 (7.5)	3 (0.9)	29 (8.4)
Chronic back pain	12 (3.5)	2 (0.6)	14 (4.1)	10 (2.9)	4 (1.2)	14 (4.1)
Arthritis	2 (0.6)	-	2 (0.6)	10 (2.9)	2 (0.6)	12 (3.5)
Excessive ear wax	48 (13.9)	5 (1.5)	53 (15.3)	8 (2.3)	2 (0.6)	10 (2.9)
Severe gastric reflux	17 (4.9)	2 (0.6)	19 (5.5)	8 (2.3)	2 (0.6)	10 (2.9)
Herniated spinal disc	9 (2.6)	-	9 (2.6)	6 (1.7)	1 (0.3)	7 (2.0)
Anaemia	1 (0.3)	4 (1.2)	5 (1.5)	3 (0.9)	2 (0.6)	5 (1.5)
Scoliosis	2 (0.6)	2 (0.6)	4 (1.2)	2 (0.6)	1 (0.3)	3 (0.9)
Bronchitis	47 (13.6)	24 (6.9)	71 (20.5)	3 (0.9)	-	3 (0.9)
Functional bowel disease	1 (0.3)	-	1 (0.3)	2 (0.6)	-	2 (0.6)
TMJ dysfunction	-	-	-	2 (0.6)	-	2 (0.6)
Crohn's disease	1 (0.3)	-	1 (0.3)	1 (0.3)	-	1 (0.3)
Pneumonia	9 (2.6)	8 (2.3)	17 (4.9)	-	-	-
Bowel obstruction	6 (1.7)	-	6 (1.7)	-	-	-
Hepatitis	3 (0.9)	-	3 (0.9)	-	-	-
Inguinal hernia	2 (0.6)	-	2 (0.6)	-	-	-
Aseptic bone necrosis	1 (0.3)	-	1 (0.3)	-	-	-
Polycythaemia	1 (0.3)	-	1 (0.3)	-	-	-

proportion did not differ significantly ($p > 0.05$) from that of the general Australian population (45%)[11] and is consistent with a report on other experienced divers.[10] This finding has important implications. It may reflect a level of physical fitness that is suboptimal for a sport that can require considerable exertion, particularly in the event of an emergency. Also, obesity can further restrict the mobility of divers already encumbered with scuba gear. These factors may be particularly relevant to the eight morbidly obese divers. Finally, it has been demonstrated that obesity is a risk factor for decompression sickness.[1]

Alcohol can impact directly upon diving safety in two ways. Clearly, diving whilst under the influence of alcohol is likely to increase the risk several fold. Also, moderate or heavy alcohol intake can lead to dehydration and, in turn, an increased risk of decompression sickness.[1,3] The alcohol consumption of the divers in this study is not remarkable but the findings give no indication of the proximity of consumption to diving.

As expected, the proportion of smokers was relatively small and significantly less ($p < 0.01$) than that of the Australian population (24%).[11] This finding is encouraging as many tobacco-related respiratory diseases, including asthma and chronic obstructive airways disease, have long been considered contra-indications to diving.[1] A small proportion of divers did report illicit drug use although, like alcohol, the proximity of drug use to diving is not known. Although only 45 divers (13.0%) required daily medication for chronic disease, many others are likely to require medication for episodic illness, for example, asthma, allergies and migraine.

Traditionally, there have been good theoretical reasons why many medical conditions have been contra-indicated in diving. Firstly, the sub-aquatic environment is inherently dangerous and any condition that might reduce the physical or mental capacity of the diver may increase these dangers significantly. Such conditions include migraine, epilepsy, seasonal allergy, asthma, cardiac disease, obesity, diabetes, hernias and some psychiatric states.[1-3,12,13] Secondly, some conditions are associated with an increased risk of barotrauma associated with changes in ambient pressure. Such conditions include seasonal allergies, asthma and obstructive airway diseases, pneumothorax, equalisation difficulties, chronic sinusitis, middle and inner ear disease or surgery, and ocular surgery.[1,3,13] Thirdly, some conditions are associated with an increased risk of decompression illness; these include obesity, diabetes, and central nervous system and connective tissue scarring.[1,3,5] This study demonstrates that many conditions within these three categories are quite prevalent among experienced divers.

Few other studies have examined the prevalence of chronic disease among scuba divers. A recent Australian study by

Cresp et al[6] examined the health status of recently qualified divers. As expected, their subjects had a similar gender ratio (72% male), were younger (median age 24 years), leaner (24.0% overweight or greater) and inexperienced (76% had done fewer than 20 dives). Compared with the present study, Cresp et al found a greater prevalence of asthma and obstructive airways disease (10.3%) and hypertension (4.2%) but a similar prevalence of epilepsy (1.0%), diabetes (0.2%), ischaemic heart disease (0.2%) and smoking (11.6%). The authors noted that a number of divers commented on how easy it was to avoid the detection of medical conditions during the diving medical examination. Hansen et al reported higher prevalence of hypertension (9.7%) and active asthma (4.2%) in their large study of experienced divers in the US.[10]

In this study, the prevalence of medical contra-indications to diving is of concern. It is possible that some divers failed to disclose their conditions during the medical screening examination.[6] If confirmed, this possibility would challenge the worth of the traditional screening process as an instrument for the determination of fitness to dive. A second possibility is that some divers develop contra-indicated conditions as they age yet continue to dive through ignorance or neglect. In Australia, there is no requirement in sport diving for repeat medical assessment for fitness to dive. This raises the question of whether medical assessment should be undertaken at regular intervals throughout a diver's career, as part of a re-certification process.[14]

The above discussion is based on the premise that the theoretical risks of diving with medical contra-indications are clinically significant. This may not be the case. Indeed, some studies have challenged traditional theory by suggesting that the risk associated with some conditions, including asthma and diabetes, may not be as great as originally thought.[8,13,15,16] Furthermore, the available evidence shows that diving fatalities as a direct result of medical conditions are rare. An analysis of the deaths of 286 fatalities in the United Kingdom and the US between 1990 and 1994 showed that health factors, as possible contributory causes, accounted for 2% and 5.1% of deaths, respectively.[16] Australian figures appear to be higher. Of the 46 scuba diving deaths in Australia between 1993 and 1997 inclusive, eight cases (17.4%) were thought to have a possible medical contributing factor. Presumed cardiac events were considered the most common contributing medical conditions. These diving deaths included two cases of planned suicide.[17-21]

In recent years, the risks associated with many medical conditions have been re-evaluated.[5] This has led to policy shift among professional bodies, notably the South Pacific Underwater Medicine Society (SPUMS), regarding candidate fitness to dive[5,22,23] and a shift away from the prescription-based approach to diving fitness towards a more discretionary one.[3,5,7] This would involve the medical practitioner in a risk assessment rather than a regulatory role.[5] SPUMS has proposed a draft policy statement for consideration by Standards Australia which states "A medical practitioner's statement of the compatibility of a candidate's health and recreational diving must include both an acknowledgment of "health risk" and an acceptance of liability by the candidate." Should a candidate then choose to ignore the advice given, the advising physician should not be subsequently liable.[5] In any event, a shift toward this discretionary approach might encourage the disclosure of existing conditions and promote a more informed discussion about the risks involved.

In this study, few divers reported other conditions that might interfere with their diving activity or may be aggravated by diving. However, the reported prevalence of tinnitus and hearing difficulties is greater than expected and greater than the prevalence reported by Hansen et al[10] of 8.1% and 5.1%, respectively. It is conceivable that the repeated, even subclinical, aural barotrauma often experienced over a long diving career might have an aetiological role in these conditions. The findings suggest that further research into the possible association between scuba diving and chronic middle and inner ear disease is required.

This study has several limitations. The sample size was small relative to the estimated number of experienced, recreational scuba divers in Australia. Also, recruitment of subjects from dive clubs only, the under-representation of Queensland clubs (and total Queensland divers) and the club response rate may have introduced selection bias. Although the questionnaire was anonymous, the results are subject to prevarication and recall bias. Finally, as a screening study examining many medical conditions, precise definitions of each condition could not be provided to the divers. This may have limited the accuracy of some responses.

This study indicates that further research is required in order to evaluate more clearly the risks associated with some traditionally contra-indicated medical conditions. This undertaking may allow previously denied candidates the opportunity to participate in the sport with reasonable safety. If such research does confirm clinically significant theoretical risks then repeated re-assessment of fitness to dive should be considered. It is recommended that the evolution of the traditional screening examination continues and that a greater emphasis be placed upon risk assessment and candidate education. Such an evolution may require a revision of training procedures for medical practitioners planning to undertake diving medical examinations.

Conclusion

Many experienced recreational scuba divers continue to dive with medical conditions. This raises the question of non-disclosure at the initial medical screening examination or subsequent disease development. The findings may indicate either that the risks of some conditions are not of clinical

significance, or that repeated screening assessments are indicated. Research to assess more clearly the risks of traditionally contra-indicated conditions is required and an evolution of medical screening towards a risk assessment process is recommended.

Acknowledgments

The authors would like to thank the Pittsburgh Emergency Medicine Foundation for their financial support of this project. This Foundation played no part in the design or execution of this study and in no way controlled or influenced the final manuscript.

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Lidocaine in the treatment of decompression illness: a review of the literature

S J Mitchell

Key words
Lidocaine, lignocaine, decompression illness, arterial gas embolism, neuro-protection, recompression

Abstract

While recompression and hyperbaric oxygen administration remain the mainstays of treatment for decompression illness (DCI), drugs that might improve outcomes or prove beneficial in first aid management have been sought. There has been much interest in lidocaine, a sodium channel-blocking agent used clinically as an antiarrhythmic and local anesthetic. The relevant literature is reviewed. Lidocaine is neuro-protective in cerebral arterial gas embolism (CAGE) in vivo, and in a variety of in vivo and in vitro models of ischemic brain injury. There has been limited in vivo investigation of efficacy in DCI where bubbles have formed from dissolved nitrogen. Mechanisms of neuro-protection by lidocaine include deceleration of ischemic ion fluxes across the neuronal cell membrane and prevention of the consequent neurotoxic events. In addition, lidocaine lowers neuronal metabolism, exerts advantageous effects on cerebral hemo-dynamics, and is a potent anti-inflammatory. There is one randomized double blind study that demonstrates improved neuropsychological outcomes in cardiac surgery patients receiving lidocaine. Clinical evidence of efficacy in DCI is limited to anecdotal reports. Expeditious administration of lidocaine is justified in cases of unequivocal CAGE. Speculative use may be justified in severe neurologic DCI after patient counseling and consent.

Reprinted with kind permission from *Undersea Hyperb Med* 2001; 28: 165-174

S Mitchell was based at the Wesley Centre for Hyperbaric Medicine, Brisbane, Australia at the time of writing.
A summary of this paper was presented by Dr Mitchell at the SPUMS ASM, 2001, Madang, PNG.

Editor's comment: Dr Mitchell reviewed the literature on lignocaine in the treatment of decompression illness (DCI) for his SPUMS Diploma thesis seven years ago.[1] Since then, our understanding of the neuro-protective actions of lignocaine has progressed, not least as a result of Mitchell's own work; the randomised study mentioned in the abstract.[2] The present, comprehensive review, for which there is not space here to republish in full, brings up to date the body of knowledge underlying his final conclusions. The Slark Hyperbaric Unit, Auckland, commenced a randomised study on lignocaine in DCI some years ago, which has not been completed to date.[3] Likewise, James Francis has described how setting up an international multi-centre trial proved impossible.[4] The role of lignocaine in diving DCI, therefore, is not proven clinically, and nothing is known of its potential in altitude DCI, eg. in extra-vehicular space activities.

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Treatment of multiple sclerosis with hyperbaric oxygen therapy

M Bennett and R Heard

Key words
Multiple sclerosis, hyperbaric oxygen, treatment

Absract

Despite considerable research effort, there is little controlled evidence that a course of hyperbaric oxygen therapy (HBO_2T) results in any benefit for patients with multiple sclerosis (MS). The great majority of randomized trials involved investigating a course of 20 treatments at pressures between 1.75 and 2.5 atm abs daily for 60–120 min over 4 wk against a placebo regimen. None has tested the efficacy of HBO_2T against alternative current best practice. A systematic review of this randomized evidence suggests there is no significant benefit from the administration of HBO_2T (Improved EDSS after HBO_2T: OR = 2.02, 95% CI 0.63–6.43. Improved sphincter function: OR = 1.3, 95% CI 0.8–2.11). On average, 42 patients would need to be treated before we could expect one individual to benefit with an improved disability status score; however, we cannot be confident that the number we would need to treat is less than infinite (NNT = 42, 95% CI 15 to infinity). There is some case for further investigation of possible therapeutic effects in selected sub-groups of patients and for the response to prolonged courses of HBO_2T at more modest pressures; however, the case is not strong. At this time, we cannot recommend the routine treatment of MS with HBO_2T.

Reprinted with kind permission from *Undersea Hyperb Med* 2001; 28:117-122

M Bennett and R Heard are based at the Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, University of New South Wales, Sydney, Australia.

Editor's comment: Michael Bennett has created a very useful database of randomised trials of hyperbaric oxygen, both in animal research and clinical practice. This is regularly updated. Each reference is followed by a critical review in an evidence base style .[1] This may be found on the UHMS web site <http://www.uhms.org> or is available at <http://www.hboevidence.com/>. Members are recommended to read Bennett's earlier SPUMS Journal article on evidence based journal reading.[1]

Reference

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Effects of water immersion on pulmonary function in asthmatics

J J Leddy, A Roberts, J Moalem, T Curry and C E G Lundgren

Key words
Diving, asthma, pulmonary function, exercise, closing volume, pulmonary barotrauma

Abstract

Immersion induces air trapping in the lungs, as does asthma. Consequently, when using diving apparatus, asthmatics may face greater risk than non-asthmatics of pulmonary barotrauma (PBT) during ascent. We studied the pulmonary airflows and closing capacities (CC = closing volume + residual volume) in subjects with exercise-induced asthma (A, n = 12) and in healthy controls (C, n = 11) under four conditions: dry and immersed, both before and after exercise (treadmill running, non-immersed). Immersed, both C and A had significant and equivalent reductions in vital capacity, FEV_1, FEV_1/FVC, and $FEF_{25\%-75\%}$. Post-exercise and immersed, pulmonary airflows deteriorated further in A but were better in C: FEV_1 (A, 3.6 ± 0.8 liter vs. 3.3 ± 0.8 liter, p = 0.001; C, 3.9 ± 0.5 liter vs. 4.1 ± 0.6 liter, p = 0.006), $FEF_{25\%-75\%}$ (A, 3.5 ± 1.0 liter · s^{-1} vs. 3.0 ± 0.8 liter · s^{-1}. p < 0.05; C, 4.0 ± 0.9 liter · s^{-1} vs. 4.3 ± 0.9 liter · s^{-1}, p <0.05). Therefore, in contrast to C, A subjects had reduced pulmonary airflows during immersion after exercise. Furthermore, A subjects more often had no closing volume phase IV when immersed after exercise than C (p = 0.005). Interpreting the absence of phase IV as indicative of more air trapping in the asthmatics during immersion after exercise would be consistent with the reductions in airflow.

Reprinted with kind permission from *Undersea Hyperbaric Med* 2001; 28: 75-82

The authors are at the Center for Research and Education in Special Environments, Department of Physiology and Biophysics, and the Sports Medicine Institute, State University of New York at Buffalo, Buffalo, New York.

Commentary on Leddy et al by Paul Thomas

This interesting study tries to tease out what happens to exercise-induced asthmatic subjects during immersion, and compares the results with the pre-exercise values, and with normal control subjects. After immersion, they demonstrate a reduction in all lung volumes, including FEV_1 in both groups. After exercise, there is a fall in FEV_1 in the asthmatic group, which is part of the inclusion criteria for participation. A complex technique is used for estimating the point during expiration at which many airways close, but the data are only interpretable in half of the asthmatic subjects, thus this measurement did not show any significant changes. The authors had postulated that this measurement could be used to show that premature airway closure might occur during immersion, and that this would be more pronounced in asthma.

The study confirms that lung volumes are reduced by immersion as has been documented previously.[1] Immersion therefore reduces lung volume, which in turn allows airways to narrow and collapse in both subject groups. They are unable to demonstrate whether this leads to gas trapping, but speculate that this might be the case. What we need is a better method of measuring gas trapping in these asthmatic divers, if it occurs.

In subjects with mild obstruction secondary to smoking, when measuring gas volumes before and after hyperbaric oxygen therapy, we were unable to show a difference by body plethysmography.[2] The inaccessibility of the water medium not only means that diving is for the determined, but that physiologists find it troublesome to perform their measurements too. Given the 'head-out' immersion, the findings of this study are applicable to swimming at the surface. Should all asthmatics be advised not to swim? Not from these data!

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SPUMS notices

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 documentary 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's address is:
Dr David Doolette, Department of Anaesthesia and Intensive Care, University of Adelaide,
Adelaide, South Australia 5005, Australia.
Phone: +61-8-8303-6382.
Fax: +61-8-8303-3909.
E-mail: <David.Doolette@adelaide.edu.au>.

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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SPUMS membership dues

The Treasurer reports that there has been confusion amongst some members with regard to the GST component of the membership dues. Varying proportions or all of the GST component have been deducted by some in the mistaken belief they do not have to pay this.

Please note the 2003 membership fees of Aus$132 (full) and $66 (associate) are inclusive of GST and are payable ***in full*** by ***all*** members. Whilst members outside Australia are not required to pay Australian GST, the same total membership fee applies to offset ***in part only*** the additional costs of air mailing their copies of the journal.

If members believe they are eligible to claim the GST component then that is their responsibility with the IRD. Chasing up part payments adds cost and considerable time to the work of the Treasurer and the Administration Officer. Your cooperation in this would be appreciated.

Any enquiries regarding this or other membership matters should be addressed to the Administration Officer, Steve Goble, e-mail <stevegoble@bigpond.com.au>.

SPUMS Executive Committee Meeting 18 May 2002

The minutes of this meeting were published before they had been ratified. They were confirmed as a true record at the committee meeting in Sydney, 25 October, 2002.
Moved: R Walker, seconded: G Williams, carried.

Members should note that the minutes of society annual general meetings and ANZHMG annual meetings are published prior to the succeeding year's meeting and are therefore not ratified at the time of publication. This is done to keep members informed within a sensible time frame.

South Pacific Underwater Medicine Society

31st Annual Scientific Meeting

Date:16 to 26 May 2003
Venue: Palau Pacific Resort, Palau, Micronesia

Theme: "Risk, diving and the pre-dive medical"

Workshop: "Designing a pre-diving medical for the 21st Century"

Principal guest speaker: Professor Des Gorman

Those wishing to present papers are asked to contact:

Dr Michael Bennett
Dept of Diving and Hyperbaric Medicine
Prince of Wales Hospital
High Street
Randwick, NSW 2031
Work: 02-9382-3883
Mobile: 0411-483-491
Fax: 02-9382-3882
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The deadline for abstract submission is 16 February 2003

Intending speakers are reminded that it is SPUMS policy that they must provide the convener with the printed text of their paper and the paper in electronic format before their presentation.

Those wishing more information about the ASM can contact the convener:

Dr Catherine Meehan
McLeod St Medical
67 McLeod St
Cairns, QLD 4870
Work: 07-4052-1583
Mobile: 0417-783-653
Fax: 07-4052-1930
E-mail: <cmeehan@ozemail.com.au>

Important Notice to all potential delegates, ASM 2003

Due to Australian Government terrorist warnings in Manila, the Committee has decided to recommend travel to Palau via Guam.

ITINERARY SPECIALLY PREPARED FOR SPUMS by ALLWAYS TRAVEL (Effective: 29/10/2002)

THURSDAY 15 MAY
Arrive Cairns from various ports

FRIDAY 16 MAY
12.10 AM Depart Cairns on Continental Air CO903
4.45 AM Arrive Guam
Transfer to Pacific Island Club, Guam
Located in Tumon Bay overlooking the beach, this resort has a huge choice of water activities plus a number of international standard restaurants. Some of the activities include archery, tennis and squash.
Day use – twin share – transfers

FRIDAY 16 MAY
1830 Depart Guam on Continental Airlines CO 953
1940 Arrive Kuror, Palau
Transfer to Palau Pacific Resort,
PO Box 308, Koror, Palau
Phone: 680-488-2591
Nestled among 64 acres of tropical gardens by the beach, this resort offers superb accommodation, along with extensive recreational facilities. Facilities include snorkelling, windsurfing, catamaran sailing, tennis and scuba diving (shop located on site).
Gardenview twin share - transfers

SATURDAY 24 MAY
2.25 PM Depart Kuror on Continental Air CO 894
10.55 PM Arrive Guam
Pacific Island Club, Guam
Twin share – transfers

SUNDAY 25 MAY
6.30 PM Depart Guam on Continental Air CO 902
11.10 PM Arrive Cairns
Transfer to Holiday Inn, Cairns
Centrally located, close to all down-town amenities.
Twin share - transfers

MONDAY 26 MAY
Connections back to Australian and other ports

For further information and pricing, contact:
ALLWAYS DIVE EXPEDITIONS
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Toll Free: 1800-338-239 or 1800-777-857

ANZHMG Annual General Meeting

Rydges Hotel, Christchurch
Thursday 29 August 2002, 0900 hours

1 Present
David Smart (Chairman), David Wilkinson (Secretary), Mike Davis, Ian Millar, Greg Emerson, Brian Spain, Hamish Holland, Harry Oxer, Barb Trytko, Neil Hampson

2 Apologies
Bob Wong, Martin Hodgson, David Griffiths, Richard Harris, Margaret Walker, Sarah Sharkey, Simon Mitchell, Mike Bennett, Jan Lehm

3 Minutes of previous meeting
Minutes of the previous annual general meeting held in Fremantle in September 2001 were accepted as an accurate record of events.

4 Business arising
No issues identified.

5 Chairman's address
David Smart paid tribute to those involved in what has been a busy year. As a result of the MSAC issue, there currently exist several documents which constitute comprehensive and current reviews (soft-tissue radiation injury, non-diabetic hypoxic wounds and transcutaneous oximetry). Discussion resolved that these documents are too valuable to leave just with Medicare and that they should be published in appropriate journals. The need to measure outcome was highlighted, in particular an outcome measure of wounds. It was noted some wound assessment scales are currently in use in various units and that for units with appropriate data, consideration should be given to publishing their results. Mike Davis made comment that he has a modified wound scoring system that is easy to use and may be of interest. Neil Hampson, as current president of the UHMS, made the observation that the UHMS has been involved with similar issues related to accepted indications.
Action: Mike Davis to circulate wound scoring system to all units for consideration.

6 MSAC report
David Smart summarised the status of the MSAC review of Hyperbaric Oxygen Treatment. The ANZHMG continues to dispute the findings of the report and we await a process to address our concerns. The AMA have been helpful in this regard. It appears the much-vaunted UK review of the initial report has sunk without trace. We await the next move with suspicion. Strategies for our own response were discussed.

7 ANZHMG/SIG list of approved indications for HBO_2 - No change to current list.

8 Introductory Course in Hyperbaric Medicine – April 2002, The Alfred Healthcare Group
Ian Millar reported the ANZHMG/SIG course was successfully run, and preparations are under way for the next course – 17–28 March 2003.
Dr Millar reminded the meeting the initial plan was for the course to be run for two years before moving on to another institution. Invitation was made for other institutions to consider this opportunity.

9 ANZCA-SIG certificate
In the absence of Dr Wong, a progress report was tabled. Acceptance of a plan for the certificate is progressing. Foundation Certification will probably close on 30th June 2003. An announcement is expected in a forthcoming College Bulletin.

10 AS4774 and Standards – scientific standard
Ian Millar, as representative to Standards Australia, reported on the development of different standards for different types of occupational exposure. Inconsistencies in the requirement for a RCC at the dive site were discussed – it was suggested Standards Australia could consider approaching SPUMS about running a workshop to establish uniform criteria for requiring a RCC. It was noted that most people were not aware of the call for public comment on Standards Australia draft documents – it was suggested such calls related to documents of relevance should be advertised in the SPUMS Journal. Mike Davis noted that the previous standard applying to hyperbaric facilities (AS/NZS 2299.1) covered both Australia and New Zealand whereas the current standard (AS 4774) has only Australian acceptance. Dr Davis offered to ask about achieving New Zealand recognition.
Action: Mike Davis to enquire about New Zealand recognition for AS 4774. Ian Millar to bring suggestion of SPUMS workshop to Standards Australia.

11 Hyperbaric Facility accreditation
Nothing to report.

12 SPUMS Journal
Mike Davis formally presented himself as the new editor of the SPUMS Journal. Contributions are enthusiastically encouraged but an appropriate standard will be expected. Readers are advised to watch out for a revamped format as Dr Davis seeks to strengthen the scientific status. A hyperbaric medicine section is envisaged. Following a contribution from SPUMS/ANZHMG member Dr Stephan Neff, it is anticipated that the journal will be compatible with "EndNote" software. A report on the UHMS meeting was requested.
Action: Ian Millar graciously volunteered a report on the San Diego UHMS meeting.

13 Minimum data set
There was agreement that the data collected from each unit and printed in the HTNA ASM proceedings should be reviewed and revised as required. The ANZHMG must work

in collaboration with the HTNA, and ensure the data collected are relevant for its purpose. Suggested areas for modification include format of list of indications, outcome measures and aural barotrauma.
Action: David Smart to circulate a discussion paper regarding the minimum data set by end March 2003.

14 UHMS Sydney 2004
Arrangements are proceeding for the UHMS ASM to be held in Sydney at the end of May 2004. Congratulations to Sydney for taking on this task.

15 HTNA Hobart 2003
Being held in Hobart in the last week in August at the Hotel Grand Chancellor. At this point, invited speakers appear to be David Elliott and Valerie Flook.
Action: By all – put this in your diary now.

16 Other business
Therapeutic Goods Association – The question was raised as to the status of hyperbaric chambers and whether they constitute Registered Medical Devices. This needs to be clarified.
Action: Ian Millar to obtain more detail and circulate, because the issue has been raised with the Alfred Hyperbaric Service.

Meeting closed at 1200 hours

The ANZHMG executive (Drs Smart and Wilkinson) attended the end of the HTNA AGM. Dr Smart addressed the HTNA about the need to work collaboratively – this was an opportunity to share information and avoid confusion. Issues discussed included Medicare, Australian Standards and outcome measures.

Dr David Wilkinson
Honorary Secretary ANZHMG

SPUMS members' news

Michael Bennett and Simon Mitchell, SPUMS Committee Members, were elected respectively as vice-president and committee member-at-large of the Undersea and Hyperbaric Medicine Society at the UHMS annual meeting in La Jolla in June.

Members are invited to keep the editor up to date with their own or others' activities, honours, etc. that they think fellow members of SPUMS might be interested to hear about.

Letter to the Editor

Breath-hold diving – an update

Dear Editor

With reference to my recent letter to the Journal concerning Jacques Mayol and in particular the Editor's comments,[1] together with the article by James Francis on breath-hold diving in the same edition,[2] I am now writing to advise that there is yet another breath-hold diving record to be reported.

Stuart Wavell, writing in the News Review section of *The Sunday Times* in the UK on 25 August 2002, reported that 29-year-old Tanya Streeter had the previous week dived to a depth of 525ft (160m), off the Turks and Caicos Islands, whilst standing on a weighted sled. Her return to surface was also assisted, in that she used a lift bag, which she had to disconnect from the sled and inflate by opening a valve. Such an assisted breath-hold dive falls into the "no limits" category and she apparently holds records in a total of six free diving categories.

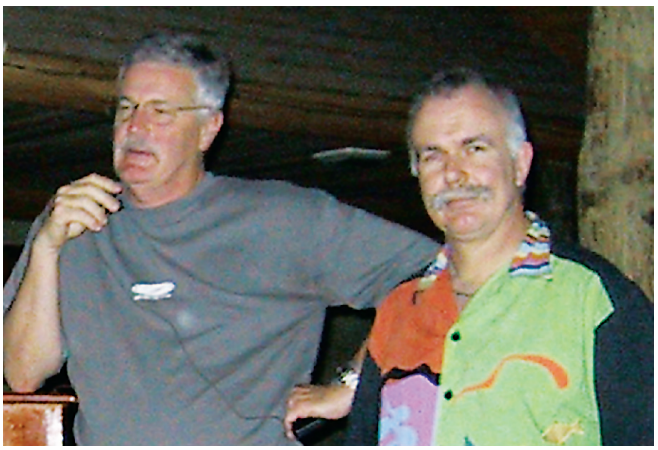
Wavell also reported that Streeter started free diving in 1997, and broke the world record for "constant-ballast" diving in fresh water the following year. Her dive to 57 m in 2 min 10 sec made her the first woman to set a world free diving record. In this category, I understand the unassisted breath-hold dive is made using fins during the descent and ascent.

It seems that the breath-hold diving fraternity are developing their competitions into quite a fast-moving business. Streeter estimates that without sponsorship, her latest record would have cost US$75,000–85,000 for boats, a safety crew and accommodation for the judges. When asked whether she was worried about her record being beaten, she replied, "No, I would give it six weeks!"

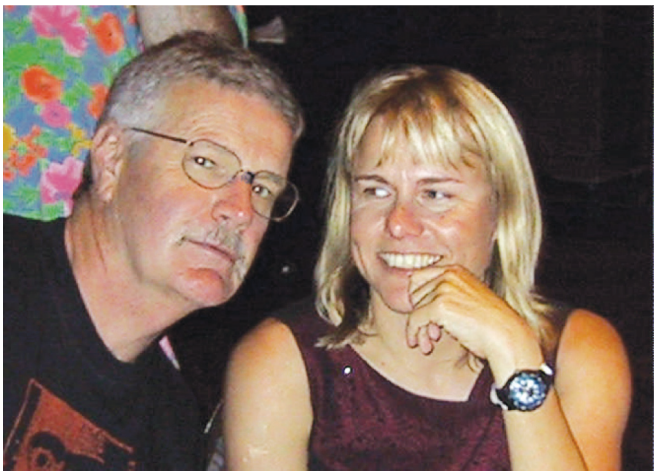
Nigel McKie
Helston, Cornwall, United Kingdom
E-mail: <nigelmckie@helston.fsbusiness.co.uk>

References

1 McKie NIP. Jacques Mayol. *SPUMS J* 2002; 32: 25-26
2 Francis TJR. Breathhold Diving. *SPUMS J* 2002; 32: 31-35



Drs Chris Acott and Guy Williams
Dr Trish Batchelor with Dr Acott



SPUMS ASM, Port Vila, Vanuatu, May 2002

Dr Roybn Walker and Dr John Knight
Adrienne McKeonne, Allways Travel

Seasonal greetings

The SPUMS Journal editorial team, Michael Davis and Sarah Webb, would like to thank all contributors to the Journal for their efforts in 2002 and to wish all SPUMS members a healthy and happy 2003. Good diving!

The world as it is

R·S·T·C
Recreational Scuba Training Council

UNDERSEA & HYPERBARIC MEDICAL SOCIETY

MEDICAL STATEMENT

Participant Record (Confidential Information)

Please read carefully before signing.

This is a statement in which you are informed of some potential risks involved in scuba diving and of the conduct required of you during the scuba training program. Your signature on this statement is required for you to participate in the scuba training program offered

by_____and
Instructor

_____located in the
Facility

city of_____, state/province of _____.

Read this statement prior to signing it. You must complete this Medical Statement, which includes the medical questionnaire section, to enroll in the scuba training program. If you are a minor, you must have this Statement signed by a parent or guardian.

Diving is an exciting and demanding activity. When performed correctly, applying correct techniques, it is relatively safe. When established safety procedures are not followed, however, there are increased risks.

To scuba dive safely, you should not be extremely overweight or out of condition. Diving can be strenuous under certain conditions. Your respiratory and circulatory systems must be in good health. All body air spaces must be normal and healthy. A person with coronary disease, a current cold or congestion, epilepsy, a severe medical problem or who is under the influence of alcohol or drugs should not dive. If you have asthma, heart disease, other chronic medical conditions or you are taking medications on a regular basis, you should consult your doctor and the instructor before participating in this program, and on a regular basis thereafter upon completion. You will also learn from the instructor the important safety rules regarding breathing and equalization while scuba diving. Improper use of scuba equipment can result in serious injury. You must be thoroughly instructed in its use under direct supervision of a qualified instructor to use it safely.

If you have any additional questions regarding this Medical Statement or the Medical Questionnaire section, review them with your instructor before signing.

Divers Medical Questionnaire

To the Participant:

The purpose of this Medical Questionnaire is to find out if you should be examined by your doctor before participating in recreational diver training. A positive response to a question does not necessarily disqualify you from diving. A positive response means that there is a preexisting condition that may affect your safety while diving and you must seek the advice of your physician prior to engaging in dive activities.

Please answer the following questions on your past or present medical history with a **YES** or **NO**. If you are not sure, answer **YES**. If any of these items apply to you, we must request that you consult with a physician prior to participating in scuba diving. Your instructor will supply you with an RSTC Medical Statement and Guidelines for Recreational Scuba Diver's Physical Examination to take to your physician.

_____ Could you be pregnant, or are you attempting to become pregnant?

_____ Are you presently taking prescription medications? (with the exception of birth control or anti-malarial)

_____ Are you over 45 years of age and can answer YES to one or more of the following?
- currently smoke a pipe, cigars or cigarettes
- have a high cholesterol level
- have a family history of heart attack or stroke
- are currently receiving medical care
- high blood pressure
- diabetes mellitus, even if controlled by diet alone

Have you ever had or do you currently have...

_____ Asthma, or wheezing with breathing, or wheezing with exercise?

_____ Frequent or severe attacks of hayfever or allergy?

_____ Frequent colds, sinusitis or bronchitis?

_____ Any form of lung disease?

_____ Pneumothorax (collapsed lung)?

_____ Other chest disease or chest surgery?

_____ Behavioral health, mental or psychological problems (Panic attack, fear of closed or open spaces)?

_____ Epilepsy, seizures, convulsions or take medications to prevent them?

_____ Recurring complicated migraine headaches or take medications to prevent them?

_____ Blackouts or fainting (full/partial loss of consciousness)?

_____ Frequent or severe suffering from motion sickness (seasick, carsick, etc.)?

_____ Dysentery or dehydration requiring medical intervention?

_____ Any dive accidents or decompression sickness?

_____ Inability to perform moderate exercise (example: walk 1.6 km/one mile within 12 mins.)?

_____ Head injury with loss of consciousness in the past five years?

_____ Recurrent back problems?

_____ Back or spinal surgery?

_____ Diabetes?

_____ Back, arm or leg problems following surgery, injury or fracture?

_____ High blood pressure or take medicine to control blood pressure?

_____ Heart disease?

_____ Heart attack?

_____ Angina, heart surgery or blood vessel surgery?

_____ Sinus surgery?

_____ Ear disease or surgery, hearing loss or problems with balance?

_____ Recurrent ear problems?

_____ Bleeding or other blood disorders?

_____ Hernia?

_____ Ulcers or ulcer surgery ?

_____ A colostomy or ileostomy?

_____ Recreational drug use or treatment for, or alcoholism in the past five years?

The information I have provided about my medical history is accurate to the best of my knowledge. ***I agree to accept responsibility for omissions regarding my failure to disclose any existing or past health condition.***

Signature	Date	Signature of Parent or Guardian	Date

STUDENT

Please print legibly.

Name_____ Birth Date _____ Age _____
First Initial Last Day/Month/Year

Mailing Address _____

City_____ State/Province/Region _____

Country _____ Zip/Postal Code _____

Home Phone ( )_____ Business Phone ( )_____

Email _____ FAX_____

Name and address of your family physician

Physician _____ Clinic/Hospital _____

Address_____

Date of last physical examination _____

Name of examiner_____ Clinic/Hospital_____

Address _____

Phone ( )_____ Email _____

Were you ever required to have a physical for diving? ☐ Yes ☐ No If so, when?_____

PHYSICIAN

This person applying for training or is presently certified to engage in scuba (self-contained underwater breathing apparatus) diving. Your opinion of the applicant's medical fitness for scuba diving is requested. There are guidelines attached for your information and reference.

Physician's Impression

☐ I find no medical conditions that I consider incompatible with diving.

☐ I am unable to recommend this individual for diving.

Remarks _____

_____

_____

_____ Date _____
Physician's Signature or Legal Representative of Medical Practitioner Day/Month/Year

Physician_____ Clinic/Hospital_____

Address_____

Phone ( )_____ Email _____

Guidelines for Recreational Scuba Diver's Physical Examination

Instructions to the Physician:

Recreational **SCUBA** (Self-Contained Underwater Breathing Apparatus) can provide recreational divers with an enjoyable sport safer than many other activities. The risk of diving is increased by certain physical conditions, which the relationship to diving may not be readily obvious. Thus, it is important to screen divers for such conditions.

The **RECREATIONAL SCUBA DIVER'S PHYSICAL EXAMINATION** focuses on conditions that may put a diver at increased risk for decompression sickness, pulmonary overinflation syndrome with subsequent arterial gas embolization and other conditions such as loss of consciousness, which could lead to drowning. Additionally, the diver must be able to withstand some degree of cold stress, the physiological effects of immersion and the optical effects of water and have sufficient physical and mental reserves to deal with possible emergencies.

The history, review of systems and physical examination should include as a minimum the points listed below. The list of conditions that might adversely affect the diver is not all-inclusive, but contains the most commonly encountered medical problems. The brief introductions should serve as an alert to the nature of the risk posed by each medical problem.

The potential diver and his or her physician must weigh the pleasures to be had by diving against an increased risk of death or injury due to the individual's medical condition. As with any recreational activity, there are no data for diving enabling the calculation of an accurate mathematical probability of injury. Experience and physiological principles only permit a qualitative assessment of relative risk.

For the purposes of this document, **Severe Risk** implies that an individual is believed to be at substantially elevated risk of decompression sickness, pulmonary or otic barotrauma or altered consciousness with subsequent drowning, compared with the general population. The consultants involved in drafting this document would generally discourage a student with such medical problems from diving. **Relative Risk** refers to a moderate increase in risk, which in some instances may be acceptable. To make a decision as to whether diving is contraindicated for this category of medical problems, physicians must base their judgement on an assessment of the individual patient. Some medical problems which may preclude diving are **temporary** in nature or responsive to treatment, allowing the student to dive safely after they have resolved.

Diagnostic studies and specialty consultations should be obtained as indicated to determine the diver's status. A list of references is included to aid in clarifying issues that arise. Physicians and other medical professionals of the Divers Alert Network (DAN) associated with Duke University Health System are available for consultation by phone +1 919 684 2948 during normal business hours. For emergency calls, 24 hours 7 days a week, call +1 919 684 8111 or +1 919 684 4DAN (collect). Related organizations exist in other parts of the world – DAN Europe in Italy +39 039 605 7858, DAN S.E.A.P. in Australia +61 3 9886 9166 and Divers Emergency Service (DES) in Australia +61 8 8212 9242, DAN Japan +81 33590 6501 and DAN Southern Africa +27 11 242 0380. There are also a number of informative websites offering similar advice.

NEUROLOGICAL

Neurological abnormalities affecting a diver's ability to perform exercise should be assessed according to the degree of compromise. Some diving physicians feel that conditions in which there can be a waxing and waning of neurological symptoms and signs, such as migraine or demyelinating disease, contraindicate diving because an exacerbation or attack of the preexisting disease (e.g.: a migraine with aura) may be difficult to distinguish from neurological decompression sickness. A history of head injury resulting in unconsciousness should be evaluated for risk of seizure.

Relative Risk Conditions

- **Complicated Migraine Headaches whose symptoms or severity impair motor or cognitive function, neurologic manifestations**
- **History of Head Injury with sequelae other than seizure**
- **Herniated Nucleus Pulposus**
- **Intracranial Tumor or Aneurysm**
- **Peripheral Neuropathy**
- **Multiple Sclerosis**
- **Trigeminal Neuralgia**
- **History of spinal cord or brain injury**

Temporary Risk Condition

History of cerebral gas embolism without residual where pulmonary air trapping has been excluded and for which there is a satisfactory explanation and some reason to believe that the probability of recurrence is low.

Severe Risk Conditions

Any abnormalities where there is a significant probability of unconsciousness, hence putting the diver at increased risk of drowning. Divers with spinal cord or brain abnormalities where perfusion is impaired may be at increased risk of decompression sickness.

Some conditions are as follows:

- **History of seizures other than childhood febrile seizures**
- **History of Transient Ischemic Attack (TIA) or Cerebrovascular Accident (CVA)**
- **History of Serious (Central Nervous System, Cerebral or Inner Ear) Decompression Sickness with residual deficits**

CARDIOVASCULAR SYSTEMS

Relative Risk Conditions

The diagnoses listed below potentially render the diver unable to meet the exertional performance requirements likely to be encountered in recreational diving. These conditions may lead the diver to experience cardiac ischemia and its consequences. Formalized stress testing is encouraged if there is any doubt regarding physical performance capability. The suggested minimum criteria for stress testing in such cases is at least 13 METS.* Failure to meet the exercise criteria would be of significant concern. Conditioning and retesting may make later qualification possible. Immersion in water causes a redistribution of blood from the periphery into the central compartment, an effect that is greatest in cold water. The marked increase in cardiac preload during immersion can precipitate pulmonary edema in patients with impaired left ventricular function or significant valvular disease. The effects of immersion can mostly be gauged by an assessment of the diver's performance while swimming on the surface. A large proportion of scuba diving deaths in North America are due to coronary artery disease. Before being approved to scuba dive, individuals older than 40 years are recommended to undergo risk assessment for coronary artery disease. Formal exercise testing may be needed to assess the risk.

* METS is a term used to describe the metabolic cost. The MET at rest is one, two METS is two times the resting level, three METS is three times the resting level, and so on. The resting energy cost (net oxygen requirement) is thus standardized. (Exercise Physiology; Clark, Prentice Hall, 1975.)

Relative Risk Conditions

- **History of Coronary Artery Bypass Grafting (CABG)**
- **Percutaneous Balloon Angioplasty (PCTA) or Coronary Artery Disease (CAD)**
- **History of Myocardial Infarction**
- **Congestive Heart Failure**
- **Hypertension**
- **History of dysrythmias requiring medication for suppression**
- **Valvular Regurgitation**

Pacemakers

The pathologic process that necessitated should be addressed regarding the diver's fitness to dive. In those instances where the problem necessitating pacing does not preclude diving, will the diver be able to meet the performance criteria?

*** NOTE: Pacemakers must be certified by the manufacturer as able to withstand the pressure changes involved in recreational diving.**

Severe Risks

Venous emboli, commonly produced during decompression, may cross major intracardiac right-to-left shunts and enter the cerebral or spinal cord circulations causing neurological decompression illness. Hypertrophic cardiomyopathy and valvular stenosis may lead to the sudden onset of unconsciousness during exercise.

PULMONARY

Any process or lesion that impedes airflow from the lungs places the diver at risk for pulmonary overinflation with alveolar rupture and the possibility of cerebral air embolization. Many interstitial diseases predispose to spontaneous pneumothorax: Asthma (reactive airway disease), Chronic Obstructive Pulmonary Disease (COPD), cystic or cavitating lung diseases may all cause air trapping. The 1996 Undersea and Hyperbaric Medical Society (UHMS) consensus on diving and asthma indicates that for the risk of pulmonary barotrauma and decompression illness to be acceptably low, the asthmatic diver should be asymptomatic and have normal spirometry before and after an exercise test. Inhalation challenge tests (e.g.: using histamine, hypertonic saline or methacholine) are not sufficiently standardized to be interpreted in the context of scuba diving.

A pneumothorax that occurs or reoccurs while diving may be catastrophic. As the diver ascends, air trapped in the cavity expands and could produce a tension pneumothorax.

In addition to the risk of pulmonary barotrauma, respiratory disease due to either structural disorders of the lung or chest wall or neuromuscular disease may impair exercise performance. Structural disorders of the chest or abdominal wall (e.g.: prune belly), or neuromuscular disorders, may impair cough, which could be life threatening if water is aspirated. Respiratory limitation due to disease is compounded by the combined effects of immersion (causing a restrictive deficit) and the increase in gas density, which increases in proportion to the ambient pressure (causing increased airway resistance). Formal exercise testing may be helpful.

Relative Risk Conditions

- **History of Asthma or Reactive Airway Disease (RAD)***
- **History of Exercise Induced Bronchospasm (EIB)***
- **History of solid, cystic or cavitating lesion***
- **Pneumothorax secondary to:**
  - **-Thoracic Surgery**
  - **-Trauma or Pleural Penetration***
  - **-Previous Overinflation Injury***
- **Obesity**
- **History of Immersion Pulmonary Edema Restrictive Disease***
- **Interstitial lung disease: May increase the risk of pneumothorax**

* Spirometry should be normal before and after exercise

Active Reactive Airway Disease, Active Asthma, Exercise Induced Bronchospasm, Chronic Obstructive Pulmonary Disease or history of same with abnormal PFTs or a positive exercise challenge are concerns for diving.

Severe Risk Conditions

- **History of spontaneous pneumothorax.** Individuals who have experienced spontaneous pneumothorax should avoid diving, even after a surgical procedure designed to prevent recurrence (such as pleurodesis). Surgical procedures either do not correct the underlying lung abnormality (e.g.: pleurodesis, apical pleurectomy) or may not totally correct it (e.g.: resection of blebs or bullae).
- **Impaired exercise performance due to respiratory disease.**

GASTROINTESTINAL

Temporary Risks

As with other organ systems and disease states, a process which chronically debilitates the diver may impair exercise performance. Additionally, dive activities may take place in areas remote from medical care. The possibility of acute recurrences of disability or lethal symptoms must be considered.

Temporary Risk Conditions

- **Peptic Ulcer Disease associated with pyloric obstruction or severe reflux**
- **Unrepaired hernias of the abdominal wall large enough to contain bowel within the hernia sac could incarcerate.**

Relative Risk Conditions

- **Inflammatory Bowel Disease**
- **Functional Bowel Disorders**

Severe Risks

Altered anatomical relationships secondary to surgery or malformations that lead to gas trapping may cause serious problems. Gas trapped in a hollow viscous expands as the divers surfaces and can lead to rupture or, in the case of the upper GI tract, emesis. Emesis underwater may lead to drowning.

Severe Risk Conditions

- **Gastric outlet obstruction of a degree sufficient to produce recurrent vomiting**
- **Chronic or recurrent small bowel obstruction**
- **Severe gastroesophageal reflux**
- **Achalasia**
- **Paraesophageal Hernia**

ORTHOPAEDIC

Relative impairment of mobility, particularly in a boat or ashore with equipment weighing up to 18 kgs/40 pounds must be assessed. Orthopaedic conditions of a degree sufficient to impair exercise performance may increase the risk.

Relative Risk Conditions

- **Amputation**
- **Scoliosis must also assess impact on respiratory function and exercise performance.**
- **Aseptic Necrosis possible risk of progression due to effects of decompression (evaluate the underlying medical**

cause of decompression may accelerate/escalate the progression).

Temporary Risk Conditions

- **Back pain**

HEMATOLOGICAL

Abnormalities resulting in altered rheological properties may theoretically increase the risk of decompression sickness. Bleeding disorders could worsen the effects of otic or sinus barotrauma, and exacerbate the injury associated with inner ear or spinal cord decompression sickness. Spontaneous bleeding into the joints (e.g.: in hemophilia) may be difficult to distinguish from decompression illness.

Relative Risk Conditions

- **Sickle Cell Disease**
- **Polycythemia Vera**
- **Leukemia**
- **Hemophilia/Impaired Coagulation**

METABOLIC AND ENDOCRINOLOGICAL

With the exception of diabetes mellitus, states of altered hormonal or metabolic function should be assessed according to their impact on the individual's ability to tolerate the moderate exercise requirement and environmental stress of sport diving. Obesity may predispose the individual to decompression sickness, can impair exercise tolerance and is a risk factor for coronary artery disease.

Relative Risk Conditions

- **Hormonal Excess or Deficiency**
- **Obesity**
- **Renal Insufficiency**

Severe Risk Conditions

The potentially rapid change in level of consciousness associated with hypoglycemia in diabetics on insulin therapy or certain oral hypoglycemic medications can result in drowning. Diving is therefore generally contraindicated, unless associated with a specialized program that addresses these issues.

Pregnancy: The effect of venous emboli formed during decompression on the fetus has not been thoroughly investigated. Diving is therefore not recommended during any stage of pregnancy or for women actively seeking to become pregnant.

BEHAVIORAL HEALTH

Behavioral: The diver's mental capacity and emotional make-up are important to safe diving. The student diver must have sufficient learning abilities to grasp information presented to him by his instructors, be able to safely plan and execute his own dives and react to changes around him in the underwater environment. The student's motivation to learn and his ability to deal with potentially dangerous situations are also crucial to safe scuba diving.

Relative Risk Conditions

- **Developmental delay**
- **History of drug or alcohol abuse**
- **History of previous psychotic episodes**
- **Use of psychotropic medications**

Severe Risk Conditions

- **Inappropriate motivation to dive – solely to please spouse, partner or family member, to prove oneself in the face of personal fears**
- **Claustrophobia and agoraphobia**
- **Active psychosis**
- **History of untreated panic disorder**
- **Drug or alcohol abuse**

OTOLARYNGOLOGICAL

Equalisation of pressure must take place during ascent and descent between ambient water pressure and the external auditory canal, middle ear and paranasal sinuses. Failure of this to occur results at least in pain and in the worst case rupture of the occluded space with disabling and possible lethal consequences.

The inner ear is fluid filled and therefore noncompressible. The flexible interfaces between the middle and inner ear, the round and oval windows are, however, subject to pressure changes. Previously ruptured but healed round or oval window membranes are at increased risk of rupture due to failure to equalise pressure or due to marked overpressurisation during vigorous or explosive Valsalva manoeuvres.

The larynx and pharynx must be free of an obstruction to airflow. The laryngeal and epiglotic structure must function normally to prevent aspiration.

Mandibular and maxillary function must be capable of allowing the patient to hold a scuba mouthpiece. Individuals who have had mid-face fractures may be prone to barotrauma and rupture of the air filled cavities involved.

Relative Risk Conditions

- **Recurrent otitis externa**
- **Significant obstruction of external auditory canal**
- **History of significant cold injury to pinna**
- **Eustachian tube dysfunction**
- **Recurrent otitis media or sinusitis**
- **History of TM perforation**
- **History of tympanoplasty**
- **History of mastoidectomy**
- **Significant conductive or sensorineural hearing impairment**
- **Facial nerve paralysis not associated with barotrauma**
- **Full prosthedontic devices**
- **History of mid-face fracture**
- **Unhealed oral surgery sites**
- **History of head and/or neck therapeutic radiation**
- **History of temperomandibular joint dysfunction**
- **History of round window rupture**

Severe Risk Conditions

- **Monomeric TM**
- **Open TM perforation**
- **Tube myringotomy**
- **History of stapedectomy**
- **History of ossicular chain surgery**
- **History of inner ear surgery**
- **Facial nerve paralysis secondary to barotrauma**
- **Inner ear disease other than presbycusis**
- **Uncorrected upper airway obstruction**
- **Laryngectomy or status post partial laryngectomy**
- **Tracheostomy**
- **Uncorrected laryngocele**
- **History of vestibular decompression sickness**

Book reviews

Mastering rebreathers

Jeffrey E Bozanic

320 pages, hard cover
ISBN: 0-941332-96-9
Flagstaff, Arizona: Best Publishing Company, 2002
Available from Best Publishing Company, P O Box 30100, Flagstaff, Arizona 86003-0100, USA.
Ph (+1) 928 527 1005; Fax: (+1) 928 526 0370
Email: <divebooks@bestpub.com>
Copies can be ordered online at www.bestpub.com
Price: US$29.95, postage and packing extra

My general impression of this book was that it is well researched and covers a comprehensive array of topics from the history of rebreathers, through explanations of the physics and physiology, to practical techniques and procedures. Each chapter is well structured, with set learning objectives that can be evaluated by self-test questions. The text is broken into short, easy to read sections, supported by numerous colour photographs and diagrams, and interspersed with many practical tables and charts. From the safety perspective, the inherent risks and responsibilities assumed by a rebreather diver are clearly established from page one, and safety points are emphasised throughout with real diving scenarios.

The introduction differs in style from the rest of the book and uses cartoon characters to establish the overall advantages of rebreather diving through the holiday exploits of Mary (a rebreather diver) and Marvin (traditional scuba). This light-hearted approach was, no doubt, designed to engage the reader but if you find them a little irritating you'll be pleased to know they don't pop up elsewhere.

The second chapter takes the reader on an interesting journey through time, from early concepts of men diving underwater with pig skin bladders in 900BC to the development of the first rebreather in 1878. Initially, this chapter seemed a little disjointed but on re-reading I decided it gave a good picture of the key milestones in diving history.

Chapter three is where the book starts to become informative and educational. The various types of rebreathers are explained in short paragraphs, written in easy to comprehend language and supported by many colour photographs and clear diagrams (although the rebreathers are grouped in a slightly different way to that of most other textbooks on the subject). One strength of this chapter is that the functional characteristics of rebreather design are related to their practical application and this successfully stimulates the reader to select a rebreather based on the type of diving he/she will be doing.

Wherever diving physics and physiology fit on the reader's scale of interest, both chapters give clear explanations of complex issues and use everyday concepts to make that all important link between 'theory' and 'so what', and practical aspects of rebreather function. Excellent quick reference charts are provided.

For those who really need to know all the ins and outs of their rebreather, there is an excellent in-depth chapter on the pros and cons of different scrubber canister design, counterlung location, and hose and mouthpiece design. For those who prefer a more top-line approach, you would be well informed by just reading the section relevant to a specific model of rebreather.

The sections on pre- and post-dive procedures leave the reader in no doubt that rebreather diving should not be undertaken lightly and provoke the scuba diver into re-thinking some of their practices and indeed un-learning some of those which may be hazardous when diving with a rebreather. Bozanic reinforces the importance of basic skills to cope with most of the potential 'pilot errors' that can occur. Throughout the book, the emphasis is on safety and the need for proper training courses, keeping up to date and skills practise.

What is missing? One glaring omission is that the book hardly even mentions the most widely used recreational closed circuit rebreather, the Buddy Inspiration by AP Valves. Unlike the other less common rebreathers, there are no check-list appendices or descriptive text relevant to the Inspiration, and nor is it or AP Valves listed in the 'Rebreather supporters' advertisement section or the list of useful website addresses. The reader cannot help but wonder if, sadly, diving politics may have influenced this obvious omission. Surely, any recently written book about rebreathers would be expected to include detailed reference to the most commonly dived recreational closed circuit rebreather?

In summary, this is a very practical guide providing a clear overview of the types of rebreathers, and stimulates the reader to consider many factors before deciding which type to buy. This book would help the experienced recreational diver about to venture into the new world of rebreather diving, as it serves as a valuable source of information to assist in deciding which rebreather to buy and also as a reference manual to complement the chosen rebreather training course. It would also serve as a reference book for anyone involved in working with rebreather divers.

Lynn Taylor

Key words
Book reviews, technical diving, equipment

Hyperbaric nursing

Valerie Larson-Lohr, Helen C Norvell (Eds)

400 pages, hard cover
ISBN: 1-930536-00-3, 2002
Flagstaff, Arizona: Best Publishing Company, 2002
Available from Best Publishing Company, P O Box 30100, Flagstaff, Arizona 86003-0100, USA.
Ph (+1) 928 527 1005; Fax: (+1) 928 526 0370
Email: <divebooks@bestpub.com>
Copies can be ordered online at www.bestpub.com
Price: US$73.00, postage and packing extra

This is a long-awaited hyperbaric nursing text, which is part of the progression of this developing specialty, and fills an obvious gap in this area. The book is aimed at the hyperbaric nurse, covering the issues faced nursing a patient in the hyperbaric environment. In this aim, it has clearly succeeded. The overall layout is logical and easy to follow. Photos and diagrams are appropriate to the text and clearly reproduced. The book is divided into ten sections each written by various authors.

The first section gives a brief history of the Baromedical Nurses Association. Section two takes up one quarter of the book, focusing on the legislative climate that affects nursing in the United States. The first chapter of this section covers in detail documentation and how it relates to nursing as a whole, such as legal requirements and policies affecting documentation. It then goes on to cover elements of sound documentation and the differing methods of achieving this. Some readers would find this section rather heavy going.

Chapter two of this section deals firstly with generic standards of nursing practice, then finally (!) addresses the problems related to nursing the hyperbaric patient and nursing interventions. This is set out as the 'nursing diagnosis' and care plan format. Most nurses are familiar with this approach of identifying the potential problem, desired outcome, actions and evaluation. The authors have gleaned documentation forms from various hyperbaric units for assessment and ongoing care. These are clear, differing in styles and would be very helpful in assessing or developing a unit's individual documentation.

Section three thoroughly covers patient assessment and considerations for hyperbaric treatment. It gives practical advice about equipment used in the hyperbaric environment, and caring for the critically ill patient in both multiplace and monoplace chambers.

The indications for hyperbaric oxygen (HBO_2) are covered briefly but succinctly. Wound classification, documentation and tracking are well covered, assuming a previous knowledge on the reader's part. What this book doesn't cover is actual wound care. Wound care is a specialty on its own for which there are numerous other texts available.

The following sections not only look at nursing the hyperbaric patient but also staffing issues, the running of hyperbaric facility safety programmes, and training of personnel. Nursing research and international professional governing bodies are discussed. Regulatory agencies that impact on hyperbaric nursing are given a whole chapter, but this is only pertinent to the United States. The book ends with contributions from nurse leaders in the international hyperbaric field, each of whom provides a brief description of hyperbaric nursing in their country.

There is no in-depth coverage of the physics related to hyperbaric therapy or the clinical conditions treated with HBO_2 as these are well documented in other texts. There is much American nursing speak, but the essence can easily be gleaned from it. Compared to the USA, Australian and New Zealand nurses have different legislative environments impacting on nursing practice. The bottom line is that, in hyperbaric units around the world, the issues to be addressed and the patients to be cared for are much the same.

This is a valuable text, aimed successfully at the international hyperbaric nursing community. In our unit it has already proved itself to be 'nurse friendly'. Whilst trying to review the book, two of the nursing staff in my unit kept seconding it for their own use working for the post graduate certificate in hyperbaric nursing of the University of Adelaide! Every hyperbaric medicine unit should have a copy in their library and many hyperbaric nurses will want their own personal copy for regular reference.

Marj van der Linden

Key words
Book reviews, nursing, hyperbaric oxygen, treatment, textbook

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SPUMS Journal Index, Volume 32

The Index for 2002, volume 32, of the SPUMS Journal will be printed with the first issue of volume 33, 2003.

We apologise for the delay. In our efforts to bring this issue back in line with the normal printing and distribution timetable following the transfer of the Journal Office to Christchurch, it has not proved possible to have this ready in time.

Homo delphinus, the dolphin within man

Jacques Mayol

Seventh edition
Hard cover, 300 colour illustrations, 398 pages
ISBN 1-9286649-03-3, 2000
Florida, USA, Inelson-Gnocchi Publisher
Price: US$95
Available from good bookshops or <http://www.idelson-gnocchi.com>

This book is now a fitting tribute following the tragic death of Jacques Mayol in 2001. Jacques, famous for his breath-hold exploits, relates his amazing story and leads the reader into an understanding of his philosophies and what drove the man to do what he did.

Dolphins were the lifelong companions of Jacques Mayol. The main text of the book explores this link between Jacques and the dolphins that he had known. The objective of this story is to explore his concept of the dolphin within man, 'Homo delphinus', and the words present a strong argument for the reader to consider the close link between man and dolphin.

The story of the movie "The Big Blue" that made Jacques famous to the non-diving world and the real-life and movie "rivalries" between the apnoea record holders of the 1970s and 80s is related. The mammalian diving reflex and apnoea in many animals is well explained, not just dolphins and man, but incredibly includes a small group of diving breath-hold monkeys of Japan. The physics and physiology of breath-hold diving is covered, and the history of man's breath-hold diving exploits. Greek sponge divers, Pacific islanders and of course the Ama of Japan all have good time given to their cultures and techniques.

The photographs, from many talented artists, and including old black and white family photographs from his early life, are fantastic. There are some wonderful paintings, and, in the main, these are in large format full colour. The style of the book is more 'coffee table', but the words must not be ignored, as is often the case with these beautiful large books.

When you first open the book, you are naturally drawn to the images, but what a great read it is, though its size does make it a bit hard to read in bed! This book, not surprisingly, has been a bestseller in Europe and Japan, only recently being published in English, with the latest material being added for this edition to ensure it is right up to date.

Bob Ramsay

Key words
Book reviews, breath-hold diving, general interest

Obituaries

Jacques Mayol, 1927–2001

Dolphins were the lifelong companions of Jacques Mayol. He first saw dolphins when he was seven years old in the Red Sea from the deck of a steamer en route from China to France. He was born in Shanghai to French parents, but spent most of his life at his spiritual home in Elba, Italy. Jacques, during his life, also spent time in South Caicos, Turks and Caicos Islands WBI, and in Japan.

During the 1950s, when he worked at an aquarium in Florida, he developed an especially close relationship with a female dolphin called Crown. He used to swim with her out of hours, even though this was against aquarium rules. This practice of swimming with dolphins continued throughout his life. To quote his publisher, Maurizio Russo, "He was just one with the ocean."

Jacques Mayol achieved fame as an elite apnoea diver and went on to become the first man to hold his breath long enough to descend to 100 metres under water. His fame reached legendary status when the record-breaking dives became part of a long-running rivalry between Jacques Mayol and Italian diver, Enzo Maiorca. This rivalry became the inspiration for the 1988 film "Le Grand Bleu" (The Big Blue), which Jacques co-wrote. The movie was extremely successful across Europe, but was less successful in the USA. It is more remarkable that these extreme dives were conducted when he was 56 years old.

On hearing the news of his friend's death in tragic circumstances, Stephen McCulloch, Director of the Harbor Branch Oceanographic Institution in Florida, said he hoped Mayol would be remembered more for the wonderful things he had done rather than how his life ended. French President, Jacques Chirac, also gave homage to Jacques' memory in a message in "Le Monde".

May we leave you with these words from the Master of Apnoea.

> "For me, the sea is like a lover: the more it caresses me and the more I embrace it, the more we exchange our affections and the greater my desire to explore it deeply. Whether on the surface or at depth, I feel myself melt into the wide-open arms of our 'original' mother, the ocean. Each time I dive into the sea it cradles me and I emerge like a newborn baby."

Bob Ramsay
President, Historical Diving Society SEAP,
January 2002

This obituary is based on the one that appears on the Historical Diving Society USA's web site, <www.hds.org>

Philippe Tailliez, 1905–2002

Extracts from articles by Leslie Leaney, President of the Historical Diving Society-USA, and John C Fine, Liason Officer of the United Nations Environment Programme, marine biologist and author.

The full items, including quotes from Fine's lengthy friendship with Tailliez, his wife Josie and their close friend, Andre Galerne, are to be found on the net at <http://www.hds.org/tailliez.html>

It is with great sadness that we report the passing of Capitane de Vaisseau, Philippe Tailliez, on 26 September, 2002, in France. Philippe Tailliez was born on June 15, 1905, in the French coastal town of North Malo on Dunkerque beach. His father entered the French Naval College in 1893 and travelled the world from Indochina to Tahiti. It was the stories his father told young Philippe of enchanted Polynesian islands, about native pearl divers and spear fishermen, that excited the young boy's imagination. The family lived on the Atlantic coast, and Philippe explored the ports and tributaries in a small sailing canoe.

In 1924, Tailliez entered the French Naval College and became a career naval officer. By this time, he had earned a reputation as a champion swimmer. A diver since the 1930's, it was Tailliez who introduced Jacques Yves Cousteau to the sport of goggle fishing in 1936. Tailliez's first encounter with Cousteau occurred when the young ensign was assigned to his ship, the Condorcet, anchored in Toulon, France. Cousteau had seriously injured his shoulder and chest in a car accident. After the hospital confinement, Cousteau needed exercise and he and Philippe swam together. Through his spearfishing, Tailliez was soon to make the acquaintance of yet a third person, Frederic Dumas. Two years later he introduced Cousteau to Frederic Dumas and together the trio became the famed "Les Mousqemers" of the French Mediterranean coast.

In 1945, he became the first commanding officer of the Group d'Etudes et de Recharches Sous-Marines, GERS. This was the first military group to employ the Cousteau-Gagnan Aqua-Lung and in 1949, under Tailliez's command, it produced the book *Plongee en scaphandre*. This was the world's first diving manual to cover the use of the Aqua-Lung and included repeat dive tables developed by GERS. It was later revised and translated into English. His 1954 book, *Plongees sans cable*, recorded his early diving adventures and was published in English with the title *To hidden depths*. During his time as commander of the GERS. he participated in the original sea trials of deep ocean diving bathyscaphs for the French Navy.

His involvement with diving and the sea continued throughout the last half of the twentieth century. At ECOMAIR, the acronym for the Toulon based Environmental Commission for which he served as vice-president, Tailliez continued to run against the current. He battled for the environment at the same time earning the admiration and love of those that respected his dedication to the sea. Retired in 1960, after 36 years' service in the French Navy, Tailliez spent full days working as a volunteer for ECOMAIR and GRAN, an underwater archeology organization with offices in the Navy Arsenal in Toulon.

Tailliez was also one of the organisers of the first underwater natural park in France. Since its inception by decree of October 1963, he conducted 39 campaigns at Port Cros. With students and biologists, they studied the ecology and environment in this protected area near Toulon. John Fine, in his interview, says "Philippe Tailliez at 94 remains a dreamer. A poet whose adventures and experience span the breadth of modern ocean exploration, invention and discovery. He is a pioneer, a man who dreams as Jules Verne dreamt, of the sea as "A vast reservoir of nature"." Capitaine Tailliez was the recipient of numerous international awards during his long and distinguished life.

Errata

In *SPUMS J* 2002; 32: 172-173, several sentences were lost from Sandy Inglis' book review of *Oxygen First Aid* by John Lippmann.

The paragraphs concerned should have read:

Oxygen therapy is pivotal in the initial therapy of any acutely ill or injured patient. This book is targeted at all pre-hospital oxygen providers and anyone providing first aid care. The book evolves logically from initial chapters on respiratory and cardiovascular physiology to an outline of circulatory failure and shock. It then reminds the reader of the well known resuscitation algorithms of maintenance of airway, breathing and circulation with specific attention to expired air resuscitation (EAR) and cardiopulmonary resuscitation (CPR).

After this, it progresses to the specifics of oxygen therapy and outlines the benefits of breathing elevated oxygen concentrations in specific situations with particular attention to diving injuries. It then highlights the adverse effects of oxygen therapy.

Jim Marwood's review of *Stars beneath the sea* by Trevor Norton, printed in the last edition of the SPUMS Journal (*SPUMSJ* 2002;32:171) was previously published as a 'Letter to the Editor'.

The Editor apologises for this oversight and for giving Professor Norton rather too much publicity!

ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS UNDERWATER MEDICINE COURSE 2003

Dates: 24 November to 05 December 2003
Venue: HMAS Penguin
The Medical Officer's Underwater Medicine Course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving related illnesses.

Cost: $1578.00 (tbc)

For further information and application forms contact;
The Officer in Charge
Submarine & Underwater Medicine Unit
HMAS PENGUIN
Middle Head Rd
Mosman 2088 NSW

Phone: +61 -2-9960-0572
Fax: +61-2-9960-4435
E-mail: <Sarah.Sharkey@defence.gov.au>

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE COURSES 2003

Medical Officers Course

July/August 2003			
Basic	21/7/03	to	25/7/03
Advanced	28/7/03	to	1/8/03
October/November 2003			
Basic	27/10/03	to	31/10/03
Advanced	3/11/03	to	7/11/03

Cost
Basic Diving Medicine Course: $825.00
Advanced: $825.00

DMT Full Course
February/March 2003 3 weeks, 17/2/03 to 7/3/03
October 2003 3 weeks, 13/10/03 to 31/10/03

DMT Refresher Course
July/August 2003 2 weeks, 21/7/03 to 1/8/03
October 2003 1 week, 20/10/03 to 24/10/03

For further information or to enrol contact:

The Director, Hyperbaric Medicine Unit
Royal Adelaide Hospital, North Terrace
South Australia 5000.
Phone: +61-8-8222-5116
Fax: +61-8-8232-4207

DIVING AND HYPERBARIC MEDICINE INTRODUCTORY COURSE
The Alfred Hospital, Melbourne
17 to 28 March 2003

Applications are invited for a two week, full time course aimed at doctors interested in the fields of therapeutic diving and hyperbaric medicine. This includes referring clinicians who wish to gain more knowledge about the field as well as doctors who are involved or may become involved in the operation and supervision of hyperbaric medicine facilities.

The course is jointly presented by the Australian and New Zealand College of Anaesthetists Special Interest Group in Diving and Hyperbaric Medicine and the Australian and New Zealand Hyperbaric Medicine Group
Course Director: Dr Ian Miller

The course has been offered at Prince of Wales Hospital, Sydney in 2000 and 2001 and at The Alfred in 2002. The course faculty includes speakers from most of Australia's major hyperbaric units. Significant practical work is included and attendees are strongly encouraged to experience pressurisation in The Alfred's state-of-the-art chambers. A comprehensive set of course notes will be provided.

Course fee: A$1,500 (+GST for Australian registrants)
Credits: Fulfils course requirements for SPUMS Diploma and the forthcoming ANZCA SIG Certificate.
Accredited, via UHMS, for 70 US CME points.

Enquiries to:
Ms Elmarie Celestial or Dr Ian Millar
The Alfred Hyperbaric Service
Phone: +61-3-9276-2269
Fax: +61-3-9276-3052
E-mail: <hyperbaric@alfred.org.au>

DIVING MEDICAL CENTRE COURSE FOR GPs ON DIVING MEDICAL EXAMINATIONS FOR RECREATIONAL DIVERS

This intensive course runs over three days and has been approved by SPUMS to teach doctors to examine divers to AS 4005.1 standards.

The course has been approved by the RACGP QA&CPD program for a total of 120 CPD points (Group 1).

Dates: Good Friday, 18 April to evening of Easter Sunday, 20 April, 2003

Venue: Sea World Nara Resort, Gold Coast, Queensland

Contact: Dr Bob Thomas, Brisbane
Phone: +61-7-3376-1056

Undersea and Hyperbaric Medical Society

36th Annual Scientific Meeting

Dates: 19 to 21 June, 2003
Venue: Hilton, Quebec City, Canada
Contact: Don Chandler, UHMS,
10531 Metropolitan Avenue, Kingston,
Maryland 20895, USA
E-mail: <uhms@uhms.org>
UHMS web site: http://www.uhms.org

37th Annual Scientific Meeting

Preliminary Notice
Dates: 24 to 27 May, 2004
Venue: Four Seasons Hotel, Circular Quay, Sydney
Contact: International Conferences & Events (ICE)
E-mail: <uhms@iceaustralia.com>
ASM web site: http://www.iceaustralia.com/uhms2004

European Undersea Baromedical Society

2003 Scientific Meeting

Dates: 27 to 31 August, 2003
Venue: University of Copenhagen
The Panum Institute
Blegdamsvej 3 C
2200 Copenhagen N
Denmark
Contact: EUBS 2003, c/o Department of Anaesthesiology
Centre of Hyperbaric Medicine, Righospitalet
Blegdamsvej 9, DK-2100, Copenhagen, Denmark
Phone: +45-3-5454-3467
E-mail: <hbo@rh.dk>
EUBS congress web site: http://www.hbo.dk
EUBS web site: http://www.eubs.org

Preliminary notice: 2004 meeting will be held in France

Medical assessment of fitness for diving

Dates: 9 to 14 February, 2003
Venue: Plaza Resort, Bonaire, Netherlands Antilles
Director: David Elliott, MD
CME credits: req. 24 hours Cat 1 AMA PRA
Contact: Karen Reeves, 7 Lyncroft Gardens,
Ewell, Surrey KT17 1YR, UK
Phone: +44-208-393-3318
Fax: +44-208-786-7036
E-mail: <Karen@biomedseminars.demon.co.uk>

ANZ College of Anaesthetists Annual Scientific Meeting 2003

Dates: 3 to 7 May, 2003
Venue: Hobart, Tasmania
The Hyperbaric Medicine Special Interest Group will have a session at the ASM on diving and hyperbaric medicine.
Contact: <Robert.Wong@health.wa.gov.au>

HTNA Annual Meeting 2003

Dates: 27 to 30 August, 2003
Venue: Hotel Grand Chancellor, Hobart, Tasmania
Contact: Corry van den Broek
Email: <corry.vandenbroek@dhhs.tas.gov.au>

Pacific Rim Medico-Legal Conference 2003

Dates: 27 September to 4 October, 2003
Venue: Heron Island, Great Barrier Reef, Australia
Contact: Lorenzo Boccabella
Email: boccabella@qldbar.asn.au

International Congress "Diving in the armed forces today"

Dates: 23 to 27 April, 2003
Venue: Cavtat (Dubrovnic), Croatia
Contact: Cdr Nadan M Petri,
2100 Split, IPM,
P O Box 196 (HRM),
Croatia
E-mail: <nadan.petri@morh.hr>

VIII International Meeting on High Pressure Biology

Dates: 2 to 5 June, 2003
Venue: Moscow, Russia
Contact: Ludmila Buravkova
E-mail: <buravkova@imbp.ru>
Web site: http://www.imbp.ru

INSTRUCTIONS TO AUTHORS

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:
The Editor, SPUMS Journal, C/o Office 137, 2nd Floor, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand. E-mail <spumsj@cdhb.govt.nz>

Requirements for Manuscripts
Documents are acceptable on disc or by e-mail. The preferred format is Word 6 for Windows. Illustrations and tables should **NOT** be embedded in the wordprocessor document, only their position indicated. **All tables are to be tab-separated text columns rather than using the tables option or other software, and saved as separate files.** Illustrations should be separate documents in TIFF or EPS format, clearly marked with the format used. References should be in the correct format, shown below. Two printed copies of all text, tables and illustrations should be forwarded as well.

The printed copies 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 format in the Journal. All pages should be numbered. No part of the abstract, text, references or legends to figures should be underlined. Measurements are to be in SI units (mm Hg are acceptable for blood pressure measurements) and normal ranges should be included. All tables should be double spaced on separate sheets of paper. **No vertical or horizontal rules are to be used.**

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. Legends should be less than 40 words, and indicate magnification.

Abbreviations should only be used after appearing in brackets after the complete expression, e.g. decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is 3,000 words or less. Inclusion of more than five authors requires justification. Original articles should include a title page, giving the title of the paper and the first names and surnames of the authors, an abstract of no more than 250 words and the text be subdivided into Introduction, Methods, Results, Discussion, Acknowledgements and References. Case reports should not exceed 1,500 words, with a maximum of 10 references. After the references, the authors should provide their initials and surnames, their qualifications, and the positions held when doing the work being reported. One author should be identified as correspondent for the Editor and for readers of the Journal. The full current postal address of each author, with the telephone, facsimile numbers and e-mail address of the corresponding author, should be supplied with the contribution. More than 30 references per major article will require justification. Accuracy of the references is the responsibility of authors. Acknowledgments should be brief. Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references which should be limited to five per letter).

References
The Journal reference style is the "Vancouver" style, printed in the Medical Journal of Australia, February 15, 1988; 148: 189-194. 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. Examples of the format for quoting journals and books are given below.

1 Anderson T. RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15: 19-22
2 Lippmann J, Bugg S. *The diving emergency handbook*. Melbourne: JL Publications, 1985

There should be no full stops after the reference numbers. There should be a space after the semi-colon following the year and another after the colon before the page number and no full stop after the page numbers. Titles of quoted books and journals should be in italics.

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 DES number 1-800-088-200 can only be used in Australia

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

The DES numbers in both countries 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
P.O. Box 120, Narrabeen, N.S.W. 2101.

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 any incident occurring in your dive party, but do not identify anyone. 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.

To obtain or to return Diving Incident Report forms write to:
DIMS,
30 Park Avenue, Rosslyn Park, South Australia 5072, Australia.

PROJECT PROTEUS

The aim of this investigation is to establish a data base 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 dependant 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 data base.

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

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

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