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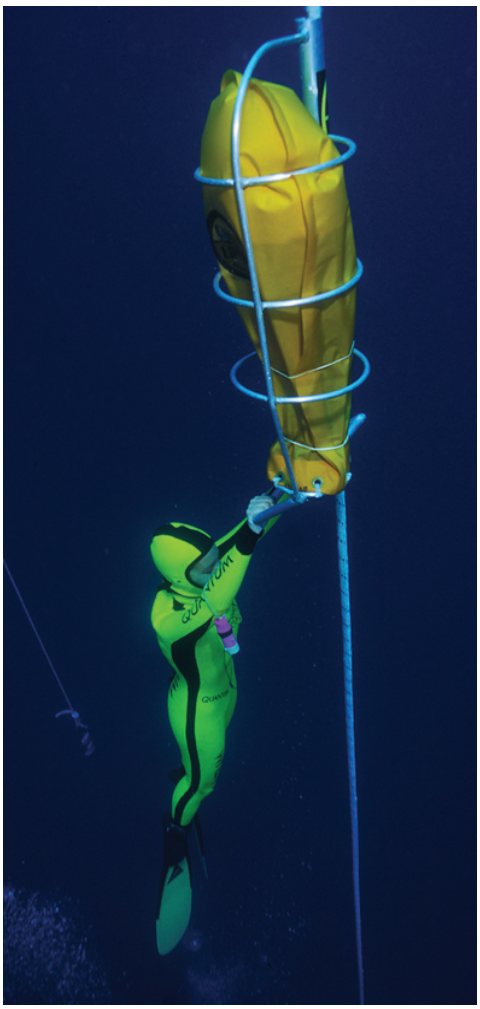

Breath-hold diving

Occupational diving health surveillance
Middle ear barotrauma
Questioning the mechanisms of hyperbaric oxygen
Travel medicine: Vaccination update
Ethical dilemmas in diving medicine: asthma

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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

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ADMINISTRATION

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The Society's financial year is January to December, the same as the Journal year.
The 2004 subscription will be Full Members A$132.00 and Associate Members A$66.00, including GST in Australia. All those outside Australia will be charged the same amounts as the GST component to partly cover the cost of having the Journal delivered to them by Air Mail. These fees must be paid in full.

The Editor's offering

My grasp of the physiology of breath-hold diving has been inadequate to understand how divers have achieved the depths of recent years. The present depth record for the variable ballast category in which the diver descends and ascends a shotline assisted by a sled and a buoyancy aid is now over 160 msw. The contraction from vital capacity of the gas volume in the lungs with compression, some collapse of the rib cage and fluid and blood shifts into the thoracic cavity seemed insufficient to explain why pulmonary haemorrhage did not occur. I was, as I suspect many readers are, unaware of the technique of 'lung packing' as described by Simpson and his colleagues in the first paper. This and the other mechanisms described now provide a new basis on which to calculate the limits to free diving. For instance, their subject could theoretically reach over 180 msw depth.

Aural barotrauma is the most common injury associated with changes in ambient pressure. Whilst generally mild and overcome by good training in ear clearing techniques, severe permanent injury may result. When treating patients in a recompression chamber it is important to minimise harm. Symptomatic aural barotrauma, mainly of a minor degree, occurs in approximately 15% of patients in the facility at which the Editor is employed, whilst Lehm and Bennett report that, if you look hard enough, nearly half of all patients will show some degree of injury following their first hyperbaric exposure. Their risk assessment confirms and expands on previous work that now allows us to identify with some confidence those patients who are likely to run into problems, combining clinical examination and modified tympanometry using portable equipment.

The move from prescriptive to discretionary medical assessment of 'fitness' for diving in recent years has not occurred generally where occupational diving is concerned. It is also not liked by the recreational diving industry for obvious reasons. The inherent problems of existing systems for employed divers based on healthcare-oriented rather than occupational health surveillance are discussed in the review article by Des Gorman. This is an important discussion paper that should be read by all doctors who perform diving medicals for working divers. Whilst much of the evidence to support his arguments has yet to be published, the rationale behind them is compelling. Time and good epidemiological research, of which there is little in the diving industry, will determine the validity or otherwise of this approach. This is a fundamental shift in philosophy to one of risk assessment and management shared by all parties concerned, doctor, diver and employer or diver training agency.

Understanding of the mechanisms of action of hyperbaric oxygen (HBO_2) at the organ or tissue level has been a slow process. Simplistic explanations in much of the literature do little to enhance this. The interaction of physics and physiology is exemplified in two interesting theoretical papers previously published by Hills and Flook. Gas-induced osmosis was a concept first proposed to explain some of the phenomena occurring as a result of inert gas switching during a series of deep experimental saturation dives at the University of Pennsylvania in the 1970s. It is here invoked to explain the oedema-reducing action of HBO_2. Hills also points out that there is minimal increase in tissue oxygen tension ($P_{tissue}0_2$) in metabolically active tissues.

Valerie Flook expands on this in her modelling of the theoretical changes in $P_{tissue}0_2$ for three tissues representing cardiac muscle, skeletal muscle and skin. Her calculations demonstrate clearly that $P_{tissue}0_2$ changes very little in highly metabolically active tissues such as the myocardium. However, even such small changes may be important for cell survival or function. This does not conflict with evidence that high oxygen tension gradients across the boundaries of hypoxic wounds are critical for fibroblast and other cell activation and migration. Neither is it likely to have relevance to the actions of HBO_2 on white cell function that are now believed to be important in treating the vascular endothelial injury caused by intravascular gas.

Trish Batchelor completes her fact-filled 'travel medicine' series with an update on vaccination. Many positive comments on her articles have been received from members, and her support of the Journal has been much appreciated.

Those who attended the Madang ASM in 2001 may be surprised that something readable has come out of the entertaining 'Pugwash' role-play session. Michael Bennett has created a complex scenario, the critical component of which is 'shopping around' behaviour for a diving medical clearance. One of the victims of a multiple diving fatality in New Zealand a few years ago may well have died at least in part as a result of such behaviour, so the story he paints is not implausible. Stephen Grant provides a legal insight into some of the issues raised by this story.

Tony Slark provides us with another entertaining autobiographical snapshot of a diving doctor's career, whilst Bill Douglas in a letter 'nails his colours to the mast' over children and diving. The items on children in the previous issue, including the Editor's offering, have generated a wide range of responses. This subject will appear again in the December issue, once the contributing authors have had an opportunity to respond. If you wish to contribute to this debate then please write to the Editor.

Michael Davis

Cover page photos:
Left – A common fur seal, *Arctocephalinae forsteri,* descending in Bligh Sound, Fiordland, New Zealand courtesy of the Editor.
Right – Yasemin Dalkilic ascending after a 120 msw breath-hold dive courtesy of Ideas in Blue/Gido Braase.
We seek photographs from members for the front page.

Original articles

Pulmonary effects of ëlung packingí by buccal pumping in an elite breath-hold diver

Graham Simpson, Janine Ferns and Sebastien Murat

Key words
Breath-hold diving, barotrauma, buccal pumping, transpulmonary pressure, lung compliance

Abstract

(Simpson G, Ferns J, Murat S. Pulmonary effects of 'lung packing' by buccal pumping in an elite breath-hold diver. *SPUMS J* 2003; 33: 122-126) Buccal pumping is a technique used by breath-hold divers to increase lung capacity above normal total lung capacity (TLC) and thus increase depth capability. Concern has been expressed that hyperinflating the lungs using the pharyngeal muscles could itself produce pulmonary barotrauma, but transpulmonary pressures after buccal pumping have not previously been measured. We studied a breath-hold diver (SM) using whole-body plethysmography and oesophageal balloon manometry. Spirometry demonstrated that vital capacity could be increased from 7.48 to 9.22 l by buccal pumping. TLC increased from 9.28 to 11.02 l, calculated by assuming a constant residual volume of 1.8 l. At normal TLC, mean maximal pulmonary relaxation pressure measured at the mouth was 8.9 cm H_2O. This rose to 86 cm H_2O following buccal pumping to 'super' TLC. Mean transpulmonary pressure (mouth pressure minus oesophageal balloon pressure) at normal TLC was 31.6 cm H_2O and at super TLC was very similar at 29.3 cm H_2O. There did not seem to be a dramatic alteration in pulmonary compliance at higher lung volumes, although this was technically difficult to measure. These data suggest that buccal pumping itself does not carry a risk of pulmonary barotrauma. We postulate that the lack of rise in transpulmonary pressure relates to increased elastic recoil of the chest wall at volumes greater than normal TLC giving a positive intrapleural pressure and preventing pulmonary over-distension. Splinting of the chest wall has been shown experimentally to reduce the risk of pulmonary barotrauma in anaesthetised animals and fresh human cadavers, probably by a similar mechanism.

Introduction

Breath-hold or 'free' diving is the oldest form of diving and has been an important commercial activity for centuries for gathering pearls, sponges, bêche de mer etc., from the ocean floor. Spear fishing, using breath-hold techniques, remains a very popular pastime. In recent decades, however, breath-hold diving has developed rapidly as a competitive sport. There are four basic disciplines: simple breath-holding for time; constant-weight (unassisted) free diving to a maximum depth possible; distance diving, where the diver covers the maximum possible distance beneath the surface in the water in one breath (with or without fins and often in a pool environment); and variable-weight (assisted) or sled diving. In this last discipline great depths are achieved by increasing the rate of descent using a heavily weighted sled on a line and often using buoyancy aids to speed the return to the surface. The depths attained by free divers using these techniques are astonishing to recreational scuba divers. The official world record has gone from about 30 m in the 1940s to the current 171 m set by Audrey Mestre in October 2002. Mestre tragically died three days after she set her record whilst attempting to break it again. The physiology of breath-hold diving has been reviewed recently by Francis.[1]

In an effort to increase maximum depths attained, some divers have developed a technique, known as 'lung packing' or 'buccal pumping' to increase their lung volume above normal total lung capacity (TLC). This technique involves inspiring to TLC, closing the glottis, and gulping a mouthful of air. The air in the mouth is compressed using oral and pharyngeal muscles and then the glottis is opened and the air forced into the lungs. The pumping movement or 'chip' is then repeated up to 50 times. Buccal pumping was developed by spear-fishing breath-hold divers in the Mediterranean many years ago and introduced to sport diving by the US Navy diver Robert Croft in the 1960s.[2]

Increasing the volume of air in the lungs above TLC carries a theoretical risk of inducing pulmonary barotrauma. Though there have been reports of the cardiovascular effects, there is limited information on the pulmonary effects of buccal pumping.[3] It has been shown that relaxation mouth pressure, reflecting intrathoracic pressure, is increased considerably by buccal pumping and this has been interpreted as suggesting a substantial risk of lung rupture.[2] However, there are no data on the effects of buccal pumping on transpulmonary pressure. We report measurements of transpulmonary pressure and lung compliance during buccal pumping performed by an elite breath-hold diver.

Case report

SM, a 34-year-old dive instructor, was originally referred following a routine occupational diving medical because of concerns about a low forced expiratory volume in one second to forced vital capacity (FEV_1/FVC) ratio. In view of his greater than normal vital capacity it was not felt that this finding was a contraindication to diving. SM is a breath-hold diver who can hold his breath in excess of eight minutes, distance fin-swim underwater over 190 m, and descend to over 150 m and over 90 m in variable-weight and constant-weight breath-hold diving categories, respectively. At his interview, SM mentioned concerns regarding the risk of barotrauma with buccal pumping and requested further investigation.

Methods

Detailed lung-function testing, including whole-body plethysmography, was performed using a Sensormedics Vmax Autobox with Vmax Vision 5.2a software including spirometry with flow-volume loops, both normally and following buccal pumping. Intrathoracic pressure at TLC with and without buccal pumping was estimated by measurement of maximal mouth pressure at full inspiration with the glottis opened and respiratory muscles relaxed. In order to measure transpulmonary pressure (mouth pressure minus intrapleural pressure) an oesophageal balloon was passed and positioned above the diaphragm. A 10 cm balloon was used on a 100 cm oesophageal catheter. The initial trials passing the balloon nasally failed as the subject could not perform buccal pumping satisfactorily because of the consequent leak of air past the soft palate. The balloon was therefore passed orally for the experiments. The balloon was initially positioned 40 cm from the teeth and then withdrawn slightly. Position was checked by monitoring catheter pressure measurements during tidal breathing, confirming negative pressure during inspiration. The balloon was inflated with 0.5 ml of air during the test. Shutter closure was regulated manually. Measurements of static pulmonary compliance and transpulmonary pressures were taken with normal breathing to TLC and following buccal pumping (to 'super' TLC). Intrathoracic pressure at super TLC was derived from transpulmonary and mouth recoil pressure.

Results

The results are summarised in Table 1. They demonstrate an increase in vital capacity of 1.74 l with buccal pumping.

TABLE 1
Spirometry, transpulmonary pressures and lung relaxation pressures at normal TLC and following buccal pumping to super TLC. TLC after buccal pumping and intrapleural pressures are derived values (see text; TLC - total lung capacity)

Parameter	Normal	Buccal pumping
FEV_1 (l) (Forced expiratory volume.sec^{-1})	5.13	5.87
FVC (l) (Forced vital capacity)	7.48	9.22
TLC (l)	9.28	11.02
Mean max. mouth pressure (mouth minus atmospheric, cmH_2O)	8.9	86
Mean transpulmonary pressure (mouth minus oesophageal, cmH_2O)	31.6	29.3
Calculated mean intrapleural pressure (cmH_20)	-22.7	+56.7

FIGURE 1
Flow (l .min^{-1}) pressure (cmH_2O) and volume (l) traces at normal TLC (total lung capacity) during pulmonary compliance and maximal relaxation pressure testing

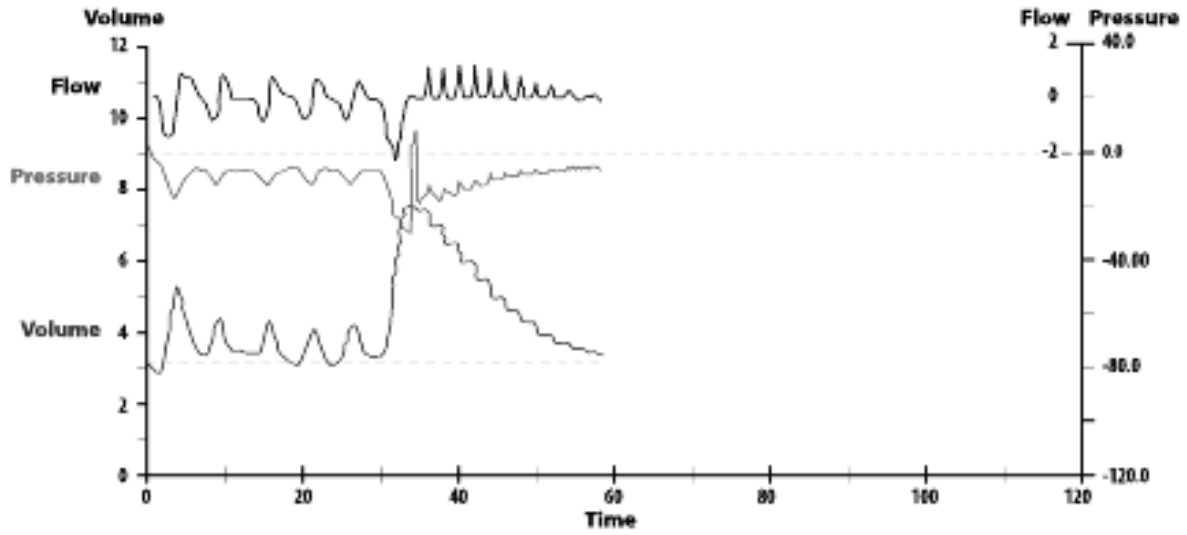
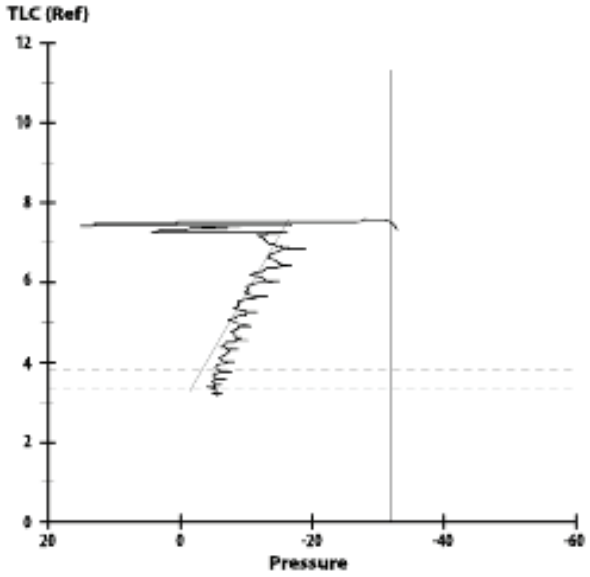


FIGURE 2
Pressure/volume curve from normal TLC showing derivation of static lung compliance (given by the slope of the diagonal line) and maximal trans-pulmonary pressure (indicated by the vertical line) (pressure - cmH_2O, volume - l)



SM found it impossible to perform buccal pumping with a mouthpiece in place so the TLC after buccal pumping is calculated by assuming that the residual volume (RV) of 1.8 l, measured during whole-body plethysmography, remains constant. Vital capacity thus is increased by 23.3% and, assuming RV is constant, TLC by 18.9%.

Figure 1 shows the flow, pressure and volume traces during a static lung compliance measurement at normal TLC. Figure 2 shows the pressure/volume relationships at normal TLC and demonstrates how static lung compliance is derived from this relationship. Maximal transpulmonary pressure is shown by the vertical line.

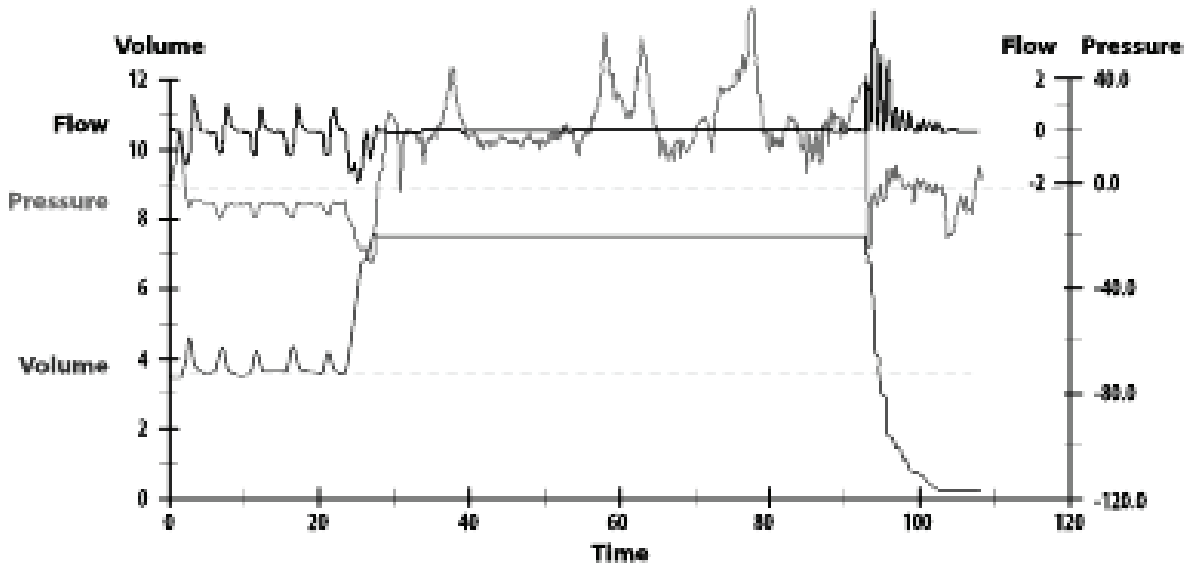
Figure 3 shows the flow, pressure and volume traces during buccal pumping with subsequent compliance and pressure measurement. It can be seen that, during the pumping manoeuvres, pressure rises in the mouth considerably and that, during the early part of the compliance measurement, flow from the lungs is rapid.

Results of mean maximal intrathoracic pressure (mouth pressure minus atmospheric pressure), mean transpulmonary pressure (mouth pressure minus oesophageal pressure) and calculated mean intrapleural pressure are shown in Table 1. It can be seen that there is no significant difference between the mean transpulmonary pressures at normal or at super TLC, though maximum mouth pressure rises considerably as does intrapleural pressure following buccal pumping.

Discussion

Barotrauma means damage caused by pressure. Increased pressure will not cause rupture of air spaces in the lung unless it is allowed to expand beyond the elastic limit of its tissues. It is, therefore, perhaps unfortunate that almost all the published literature on barotrauma concentrates on pressure changes rather than volume changes, and this includes this paper. Whether or not an airspace in a lung ruptures depends on how much it is allowed to expand and

FIGURE 3
Flow ($l.min^{-1}$) pressure (cmH_2O) and volume (l) traces after buccal pumping to super TLC (total lung capacity) during pulmonary compliance and maximal relaxation pressure testing (note high pressures in buccal pumping phase and rapid deflation on passive exhalation)



this is proportional to the pressure gradient across the walls. In the case of the whole lung, this is the transpulmonary pressure, i.e., central airway pressure minus intrapleural pressure. It is accepted that transpulmonary pressures of around 100 cm of water are sufficient to cause lung rupture in some circumstances.

Barotrauma has been reported after breath-hold ascent using scuba from a depth of around one metre, and this fits with experimental results.[4] Malhotra and Wright studied fresh, unchilled, human cadavers and showed that the intratracheal pressure at which pulmonary rupture occurred was around 75 mmHg (100 cm of water; 10 kPa).[5] They found that in cadavers whose chests and abdomens were tightly bound these pressures were very much higher. They postulated that this was because the overexpansion of the lungs was limited by the binding. The same authors produced similar findings in vivo using anaesthetised rabbits, confirming previous, similar experiments performed in the 1930s by Polak and Adams and confirmed by Schaefer et al in the 1950s using dogs.[6,7] Using these results and some arbitrary but reasonable estimates of alveolar pressure at TLC and lung compliance, the Thoracic Society of Australia and New Zealand (TSANZ) calculated that the increase in lung volume above which rupture occurs is 0.765 l.[8]

Our subject demonstrated an increase in lung volume by buccal packing that is more than double the figure calculated by the TSANZ to cause lung rupture. We have confirmed the rise in central airway pressure reported by Ornhagen et al that led them to be concerned about the danger of lung rupture.[2] However, direct measurements of transpulmonary pressure show no great increase, which can only mean that the intrapleural pressure has risen considerably during the course of buccal pumping. This would seem to be analogous in some way to the protective effects afforded by thoraco-abdominal binding in cadavers.

We would postulate that, in a young, fit, living and conscious subject, increasing the volume of air in the lungs by buccal pumping in fact hyper-pressurises rather than hyper-inflates the lungs as well as displacing blood from the pulmonary vessels, and that there is splinting of the lungs by the chest wall. This view would be supported by other data from Ornhagen et al who could show no convincing radiographic evidence of lung hyper-expansion following buccal pumping.[2]

It would seem from these results that buccal pumping is not of itself a major risk for pulmonary barotrauma occurring at the surface. Obviously, once descent occurs the risk vanishes. There are some caveats. Non-homogeneous compliance within the lung could cause problems, but the same situation should have occurred in the experimental models on which the maximum pressure figures are based. Secondly, the high pressures generated in the oropharynx and used to force air through the glottis into the lung have not been taken into account and this may be the most risky part of the manoeuvre. Many free divers in the world are performing this manoeuvre, yet there is no epidemiological evidence for or against the safety of buccal pumping. No case reports of barotrauma associated with buccal pumping have appeared.

Perhaps of equal concern are the cardiovascular effects of buccal pumping. Anecdotally, divers have reported fainting following buccal pumping, and it has been shown that the raised intrathoracic pressure associated with this manoeuvre reduces blood pressure, presumably by impeding venous return to the heart.[3]

What advantage do divers obtain by buccal pumping? For many years it was assumed that the depth limit for a breath-hold dive occurred when the total lung capacity had been compressed in accordance with Boyle's law to the lungs' residual volume. Most healthy individuals would reach this limit at a depth of 30 to 50 m. Once divers regularly started exceeding this theoretical limit, another mechanism was sought that would prevent lung squeeze. This seems to be transfer of blood into the pulmonary circulation.

Measurement of thoracic blood volume by impedance plethysmography in the 1960s showed that divers did transfer about one litre of blood to the pulmonary circulation during deep breath-hold diving.[9] Using the known lung volumes and diving records of another famous breath-hold diver, Jacques Mayol, it was possible to calculate that he too transferred approximately one litre of blood to his pulmonary circulation to enable him to achieve his record. In the case of our subject, without buccal pumping or blood transfer the maximum theoretical breath-hold diving depth is given by the formula:

$$(TLC/RV-1) \times 10 = 41 \text{ msw}$$

Assuming SM buccal pumps to the degree demonstrated in this paper he would reach residual volume at 51 msw. Currently, his maximal depth without buccal pumping is 154 msw. Assuming that this is achieved after inhaling to a TLC of 9.28 l, at this depth the thoracic gas volume would be 0.56 l. The calculated blood shift to the pulmonary circulation is 1.23 l, similar to previous findings in other divers.[10] Assuming this is the maximum amount of blood shift that he can achieve, the maximum depth he could achieve with buccal pumping would be $(11/0.56 - 1) \times 10 =$ 186 msw. That is to say, buccal pumping would be worth a theoretical 32 m of extra depth to our subject.

In the light of this sort of reward in such a competitive sport it seems unlikely that the practice of buccal pumping will disappear and further studies are indicated to confirm or refute its safety.

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Predictors of middle ear barotrauma associated with hyperbaric oxygen therapy

Jan P Lehm and Michael H Bennett

Key words
Ear barotrauma, ENT, morbidity, hyperbaric research

Abstract
(Lehm JP, Bennett MH. Predictors of middle ear barotrauma associated with hyperbaric oxygen therapy. *SPUMS J* 2003; 33: 127-133)
Introduction: Middle ear barotrauma (MEBT) is a relatively common complication of hyperbaric oxygen therapy (HBO_2). Many factors have been reported to increase the risk of this complication. This study investigates risk factors for MEBT associated with the initiation of HBO_2.
Methods: Patients scheduled for elective HBO_2 were recruited over a 12-month period. Possible risk factors for MEBT on history and examination were recorded prior to the initial HBO_2. During or immediately after this initial treatment, the presence of ear symptoms or new otoscopic tympanic membrane (TM) changes were determined as evidence of MEBT.
Results: Sixty subjects contributed data during the study period. The initial HBO_2 session was associated with mild MEBT in 43% of patients and in 32% of ears. There were no cases of free blood in the middle ear or perforated TM. MEBT was positively correlated with an immobile TM on otoscopy during the Valsalva manoeuvre. Multivariate logistic regression suggests the risk of MEBT can be predicted from the results of TM otoscopy during Valsalva manoeuvre and tympanograms before and after Valsalva (dynamic tympanogram).
Conclusions: MEBT is common in patients starting HBO_2. Patients can be stratified into low-, intermediate- and high-risk groups on the basis of the combined information from otoscopic visualisation of the mobility of the TM during Valsalva and dynamic tympanograms.

Introduction

The use of hyperbaric oxygen therapy (HBO_2) for diving-related conditions and various medical conditions has been increasing in Australia in recent years.[1] During the period 1 July 2001 to 30 June 2002, more than 23,000 HBO_2 treatments were administered to 1,349 patients in Australia.[2] The use of HBO_2 is generally safe and serious side effects are rare. However, HBO_2 is not without risks, and middle ear barotrauma (MEBT), also referred to as 'middle ear squeeze' or 'barotitis media', is by far the most common complication at the initiation of a course of treatments.[3] The reported incidence of MEBT ranges from 2% in a military setting to 94% in a group of intubated patients.[4,5] The incidence of MEBT in non-intubated medical patients ranges from 10% to 82%.[6,7] We would, therefore, predict that between 130 and 1,100 patients suffer from this complication every year in Australia.

The considerable range in the reported incidence of MEBT is probably due in part to the wide manifestations of this complication and the failure to establish a standard clinical definition as to what constitutes MEBT. In mild cases, 'fullness only' may be reported, while otalgia is reported in moderate cases. Severe ear pain associated with tympanic membrane (TM) rupture occurs in the worst cases. Other symptoms include tinnitus, vertigo and conductive hearing loss.[8,9] The severity of MEBT is assessable by otoscopy. In a previous study, we found that the most useful method for evaluating MEBT was if otoscopy was performed before and after the first HBO_2. If this was not done, interpretation of any otoscopic findings after subsequent compressions was confusing as the timing of the changes could not be determined.[10] In our experience, the majority of patients who have problems equalising will suffer from MEBT during the first HBO_2.

Many actual and potential risk factors for MEBT have been discussed previously. Analysis of case series data supports the following risk factors: the presence of an artificial airway (tracheostomy or endotracheal tube); the patient having a reduced level of consciousness; abnormal Eustachian tube (ET) function on history or testing; head and neck radionecrosis; nasal and paranasal disease; age over 55 years; and female sex (Table 1).[5-7,11-14] Yet more putative risk factors are supported only by expert opinion, and include a previous history of middle ear surgery, ear infections or smoking.[9]

A number of investigations have been suggested as useful in the prediction of MEBT, but none appears to have been tested in the context of compression and HBO_2. Such investigations are largely aimed at assessment of middle ear pathology and ET function and include tympanometry, sonotubometry, tubo-tympanoaerodynamography, laser-doppler-vibrometry and ventilation capacity testing.[3,15] Of these, tympanometry using a hand-held instrument is the simplest method. The instrument can be operated by staff with minimal training and has the capacity to produce reliable tympanograms.[16,17]

TABLE 1
Risk factors for middle ear barotrauma during hyperbaric oxygen therapy

Artificial airway (endotracheal tube or tracheostomy)
Abnormal Eustachian tube function
Head and neck radionecrosis
Nasal and paranasal disease
Impaired consciousness
Age over 55 years
Female gender

Possible factors
History of middle ear surgery
History of ear infections
Smoking

In this study, we prospectively assessed multiple risk factors for the development of MEBT. We hypothesised that MEBT could be accurately predicted by the presence or absence of a small subset of possible historical or investigational factors previously considered.

Methods

The study was undertaken over a 12-month period following approval of the appropriate research and ethics committee. Adult patients presenting for elective HBO_2 were eligible for the study. Those patients who were unconscious, had existing TM rupture or middle ear ventilation tubes in situ, and those in whom the TM could not be visualised were excluded from the study.

Study participants were first assessed for risk factors for MEBT based on past medical history. The assessment was by questionnaire developed specifically for this study and included all potential risk factors identified following review of the existing literature and expert opinion. Following completion of the questionnaire, subjects were examined and otoscopy performed. To exclude evidence of pre-existing barotrauma, all subjects' TMs were graded for the presence of barotrauma according to the scheme of Edmonds et al, a modification of the Teed score (Table 2).[8,18]

Following clinical examination, the subjects were instructed how to perform effective middle ear equalisation techniques by experienced hyperbaric attendant staff. Once the ability to perform the manoeuvre was demonstrated, the mobility or otherwise of the TMs during the Valsalva manoeuvre was observed by otoscopy and recorded. Subjects were assessed as able to successfully ventilate the middle ear if the TM was seen to 'bulge' toward the observer during a Valsalva manoeuvre, and this finding was assumed to indicate a patent, functional ET.[19] This test result was then recorded as 'Mobile TM' if the TM was seen to be bulging and 'Immobile TM' if the TM did not move.

TABLE 2
The Edmonds classification of MEBT[8]

Grade 0:	Symptoms without physical signs
Grade 1:	Injection of the tympanic membrane (TM), especially along the handle of the malleus
Grade 2:	Grade 1 plus slight haemorrhage within the substance of the TM
Grade 3:	Gross haemorrhage within the substance of the TM
Grade 4:	Free blood in the middle ear, as evidenced by blueness and bulging
Grade 5:	Perforation of the TM

The subjects then underwent further assessment using a series of three tympanogram recordings for both ears using the Welch Allyn Microtymp (Welch Allyn, Inc., Skaneateles Falls, New York, USA). Tympanometry assesses TM, middle ear and ET function by interpretation of TM impedance to sound over a range of external auditory canal pressures. All tympanograms were classified as 'normal' if the maximal impedance was between -99 and +200 decapascals (daPa) and the static admittance less than 1.5 millimho (1 millimho = 10^{-8} $m^3.Pa^{-1}.s^{-1}$), an A-type tympanogram according to Jerger's system.[20] All other tympanograms were classified as 'abnormal'.

The first tympanogram was performed before any attempt to actively ventilate the middle ear ('Static Tymp' Test). The subjects were then asked to perform a Valsalva manoeuvre and requested not to talk or swallow until a second tympanogram was completed on both ears. Subjects were then asked to swallow three times and a third tympanogram performed. The presence of normal ET function and correct performance of the manoeuvre were recorded if the peak pressure became more positive following the Valsalva manoeuvre and returned to baseline after swallowing. The result of this series of tympanograms was then recorded ('Dynamic Tymp' Test).[21]

Following this series of investigations, the subjects underwent their first HBO_2 session in a multiplace chamber supervised by a hyperbaric nurse attendant. The treatment consisted of pressurisation on air to 242 kPa (2.4 Ata) over 10 minutes followed by inhalation of 100% oxygen at 242 kPa for 90 minutes. The treatment was completed by slow decompression on oxygen over 10 minutes. The compression phase could be extended if patients complained of equalisation problems. Both the nurse attendant and supervising medical team attempted corrective strategies if any such difficulties were experienced.

Patients complaining of continuing difficulties were deemed 'unable to equalise' and were removed from the chamber after decompression to atmospheric pressure. Any symptoms referrable to MEBT were recorded after specific questioning by a study investigator and included fullness in the ears,

pain, vertigo, dizziness and subjective hearing problems. A final otoscopy was performed and any barotrauma recorded using the same classification system used prior to compression. If changes of mild erythema (grade 1) were present on TM examination before HBO_2, the ear was subsequently classified only as a MEBT if there was an increase in the TM score. Finally, we recorded the successful completion or otherwise of the initial HBO_2 session.

STATISTICAL ANALYSIS

Subjects were enrolled prospectively in an opportunistic way and no power calculations were made prior to commencement of enrolment. Results were recorded and analysed both as the number of individuals affected by MEBT, and the number of ears so affected. Fisher's exact test was employed for univariate analysis, with chi-square test for trend when using data from both ears. Factors were included in a logistic regression if univariate analysis suggested a significant association ($p </= 0.1$), or previous publications suggested a significant association.

Beginning with all those factors included, we employed a backwards, stepwise elimination method to determine the most useful predictive model for MEBT. Using this method, the factor contributing least to the predictive value of the model is eliminated at each stage until such a removal significantly reduces that predictive value. Any differences between groups or association of risk factor with barotrauma were considered statistically significant when the p-value was less than or equal to 0.05.

The performance of the tests for TM and ET function (examination for TM mobility and both static and dynamic tympanometry) were examined using sensitivity and specificity for the occurrence of barotrauma, along with positive predictive values (PPV) and likelihood ratios (LR). In the context of the present study, PPV is the probability that an individual who tests positive will experience MEBT, while the LR (+ve test) estimates how much the odds of an individual with a positive test have increased from baseline risk in the study group. All calculations were made using statistical software from StatsDirect, StatsDirect Ltd., version 2.2.3, 2002.

Results

Seventy eight subjects were enrolled in the study. Eighteen were excluded due to data loss through error or inability to perform all tympanograms required for meaningful analysis. Therefore, data from 60 subjects (120 ears) were available for analysis. One of the 60 did not complete the initial HBO_2 due to problems equalising and sustained grade 1 MEBT.

The subjects were aged from 22 to 92 years (mean 59). There were 41 males and 29 females. Six had never flown and none of the remaining 54 subjects reported problems equalising their ears during the descent of an aeroplane.

TABLE 3
Univariate analysis of potential risk factors for middle ear barotrauma (MEBT)

	MEBT	No MEBT	Odds ratio	95% confidence intervals	p-value
CLINICAL RISK FACTORS					
Age >55 years	18	19	1.78	0.54 - 6.06	0.42
Female gender	10	8	2.03	0.58 - 7.25	0.26
Scuba diver	0	4	0	0 - 1.93	0.13
Head and neck surgery	10	15	0.79	0.24 - 2.52	0.79
Head and neck radiation	11	10	1.76	0.53 - 5.88	0.41
Past middle ear infection	3	2	2.09	0.22 - 26.5	0.64
Middle/inner ear surgery	5	0	infinity	1.31 - infinity	0.01
Smoker	5	6	1.11	0.23 - 5.04	> 0.99
Upper respiratory tract infection in last month	8	10	1.07	0.30 - 3.72	> 0.99
TESTS					
At least one side abnormal:					
Tympanic Membrane (TM) Mobility	12	9	2.38	0.71 - 8.11	0.17
'Static' Tympanometry	5	3	2.46	0.42 - 17.3	0.28
'Dynamic' Tympanometry	23	24	3.19	0.69 - 20.0	0.12
Both sides abnormal:					
TM Mobility	6	2	4.8	0.74 - 51.8	0.07
'Static' Tympanometry	3	0	infinity	0.56 - infinity	0.08
'Dynamic' Tympanometry	12	11	1.79	0.55 - 5.85	0.30
PATIENTS	**26**	**34**			

This potential risk factor was therefore not analysed further. Three patients had received HBO_2 on a previous occasion and one of these reported ear problems at that time; however, none of these three experienced MEBT during this study.

Twenty six subjects (43%) had MEBT after the first HBO_2 in a total of 38 of 120 ears (32%). Seven subjects sustained grade 0 MEBT, 14 had grade 1 MEBT, three had grade 2 MEBT, and two had grade 3 MEBT. In terms of single ears, 13 had grade 0 MEBT, 18 ears had grade 1, three had grade 2, and four ears had grade 3 barotrauma. No ear had evidence of grade 4 or 5 barotrauma. The presence or absence of potential risk factors is summarised in Table 3, including the results of the univariate analysis.

UNIVARIATE RISK FACTOR ANALYSIS

The results of the univariate analysis are summarised in Table 3. None of the divers sustained MEBT, while 26 of 56 (46%) 'non-divers' did so. All subjects with a history of ear surgery developed MEBT resulting in the odds ratio reaching infinity. This was the only statistically significant historical risk factor for MEBT on the univariate analysis.

Details of the characteristics of the three tests used (TM Mobility, 'Static Tymp' Test and 'Dynamic Tymp' Test) taken in isolation are given in Table 4. Figure 1 illustrates the probability for MEBT as the number of abnormal tests per patient increases. Each of the three tests is analysed separately for trend across the possible values of no abnormal ear (score 0), unilateral abnormal ear (score 1) or bilateral abnormal ears (score 2). On this analysis, only immobile TM on otoscopy is associated with a significant trend to increasing prediction of MEBT with increasing score (chi-square test for linear trend 3.88, DF = 1, p = 0.049).

TABLE 4
Comparison of Immobile Tympanic Membranes (TMs), 'Static Tymp' and 'Dynamic Tymp' tests as single predictors of middle ear barotrauma

Predictor	Immobile TMs (one or both)	Static Tymp (any side abnormal)	Dynamic Tymp
+ve predictive value*	0.57	0.63	0.49
-ve predictive value*	0.64	0.60	0.77
Sensitivity	0.46	0.19	0.89
Specificity	0.73	0.91	0.29
Likelihood ratio# (+ve test)	1.70	2.18	1.25
Likelihood ratio# (-ve test)	0.73	0.89	0.39

* Proportion of abnormal tests associated with barotrauma (+ve) and of normal tests associated with absence of barotrauma (-ve).

\# The increase in the odds of barotrauma if the test is abnormal (+ve test), or decrease in the odds of barotrauma if the test is normal (-ve test).

MULTIVARIATE ANALYSIS

Logistic regression was employed to investigate the predictive power for MEBT of several factors in combination. The risk factors 'diver' and 'ear surgery' were excluded from the regression as these have an odds ratio of zero and infinity respectively (no divers got MEBT and all subjects with a history of ear surgery got MEBT). Hence neither would contribute in a meaningful way to a multivariate model.

Beginning with a model including the factors age over 55, gender, head and neck radiation, and the results of TM Mobility, 'Static Tymp' Test and 'Dynamic Tymp' Test, we used an elimination stepwise method for determination of the best predictive model for MEBT. The best model (chi-square test for predicted likelihood ratio 7.15, DF = 2, p = 0.03), included the results of the dynamic tympanometry (normal or either ear abnormal) and the presence or absence of immobile TMs (normal or either ear abnormal) according to the equation:

$$\text{Ln}[P/1 - P] = -2.0 + 1.6 \times \text{Dynamic Tymp} + 1.2 \times \text{Immobile TMs}$$

where P is the proportional response predicted by the model, that is, if the predicted proportion of subjects with MEBT is b out of n subjects, then P = b/n.

Solving this equation for the different combination of results for the 'Dynamic Tymp' Test and TM Immobility gives the probability of an individual subject experiencing MEBT as shown in Table 5. From this table it can be seen that if the 'Dynamic Tymp' Test is normal and both TMs are mobile, the calculated probability of MEBT from the first HBO_2 is 12%; while if both predictors are abnormal, the risk of MEBT rises to 70%.

Discussion

We describe an analysis of potentially predictive factors for MEBT. Based on a review of the literature, we chose to include only clinical factors and three bedside tests designed to assess TM and ET function. All three are simple, non-invasive and easy to perform in the clinical setting. In our small sample, only three potential risk factors were sufficiently associated with MEBT on univariate analysis to be included in the initial multivariate model (history of middle ear surgery, TM Mobility Test and 'Static Tymp' Test). Of these, ear surgery was an 'all or none' predictor and could not meaningfully be included in a logistic

TABLE 5
The calculated probability of middle ear barotrauma (MEBT) from the initial hyperbaric oxygen treatment using different combinations of the results from two tests, 'Dynamic Tymp' and TM Mobility (see text for explanation; TM - tympanic membrane)

Dynamic Tymp	TM Mobility	Probability of MEBT
Normal	yes	0.12
Normal	no	0.32
Abnormal	yes	0.40
Abnormal	no	0.70

regression model. Based on our assessment of the literature, we also chose to include age, gender, previous head and neck irradiation and 'Dynamic Tymp' Test in our initial model. All of these factors have been previously identified in prospective studies as significantly associated with MEBT.[6,7,12]

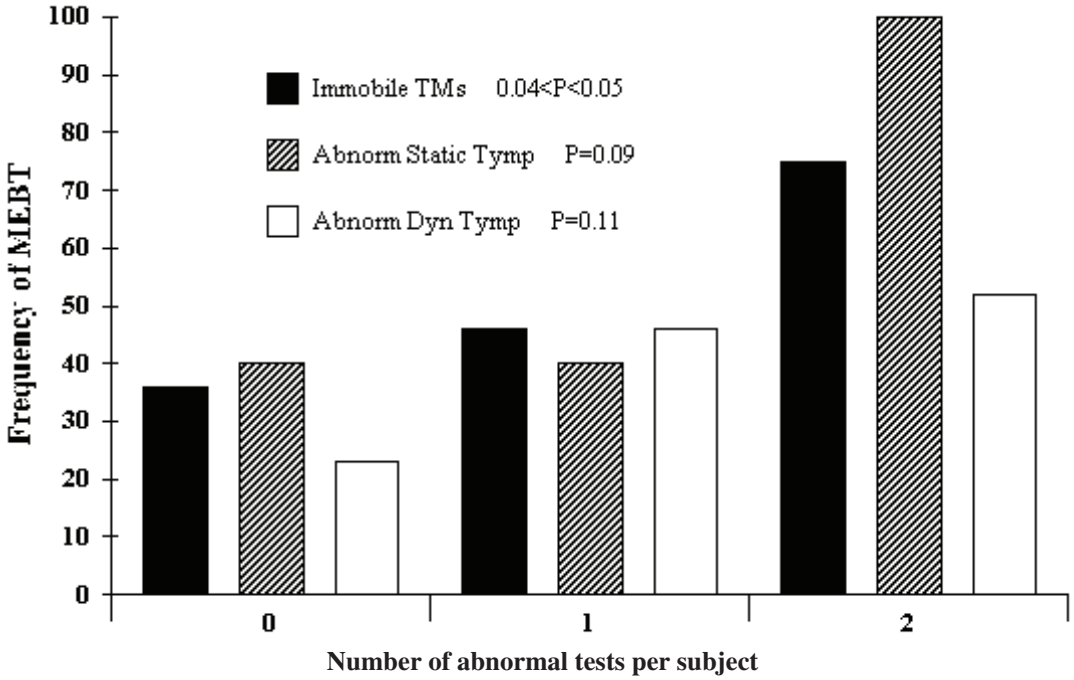
We examined subjects only in relation to their first HBO_2 session for two reasons. First, we wanted to avoid potential confusion over the significance of tests for the individual treatment under study. For any compression following the first, subjects were likely to enter the chamber with pre-existing TM abnormalities making interpretation of post-treatment tests problematic.[22] This is particularly likely following extended oxygen breathing at pressure.[19] Second, it is our clinical experience that most barotrauma occurs during the first compression.

The current study found an overall MEBT incidence of 43% of patients and in 32% of ears with the first HBO_2. None of the patients sustained the most serious grades, 4 and 5, of MEBT in this series, probably attesting to good practice and supervision by the inside attendant. The incidence in this series lies between other reported figures ranging from 10% (retrospective review) to 82% in Fernau's prospective study of 33 patients over 20 treatment sessions.[6,7]

The incidence of MEBT in any study will probably depend on vigilance in looking for this complication, the patient population and compression practice. The relatively high incidence in this study is probably a consequence of actively seeking symptoms and signs of MEBT. In our study group none of the patients who were classified as divers suffered MEBT. Intuitively, this is not an unexpected finding as this patient group is expected to be proficient at equalising middle ear pressure. In contrast, all five patients with a history of middle ear or inner ear surgery sustained MEBT with the first HBO_2. This was the only statistically significant historical risk factor. We are unaware of any other studies demonstrating ear surgery to be a predictor of MEBT, but such a finding makes pathophysiological sense.

MEBT is caused by the expansion and contraction of small volumes of air within the middle ear in accordance with Boyle's law. With reductions in ambient pressure during the 'ascent' phase of HBO_2 the gas in the middle ear will

FIGURE 1
Frequency of middle ear barotrauma (MEBT) when none (0), one (1) or both (2) ears tested abnormal for each of three clinical tests (Tympanic Membrane (TM) Mobility, 'Static Tymp' and 'Dynamic Tymp')



expand. This expanding gas generates a relatively positive middle ear pressure and opens the ET passively to equalise ambient and middle ear pressures. During compression, however, the ET is usually only capable of opening for equalisation with active manoeuvres.[23] Muscle contraction by swallowing, yawning or by the Valsalva manoeuvre is usually successful in opening the tubal lumen via the actions of the tensor and levator muscles of the palate.

These manoeuvres become increasingly difficult if the pressure gradient is allowed to increase as the ambient pressure is elevated. When the gradient reaches 80–90 mmHg (0.11–0.12 atm abs) the cartilaginous portion of the ET collapses and further attempts to equalise are futile.[24] The failure to equalise the pressure in the middle ear before this 'point of no return' during compression may be caused by poor function of the ET as a result of congenital, anatomic or pathophysiological factors or by poor technique.

Poor ET function with subsequent MEBT can be overcome by ventilation of the middle ear through the TM by the insertion of temporary tympanostomy tubes or formal grommets. However, as this is an active intervention not entirely without risks, it is desirable to identify the patients at higher risk of MEBT before deciding on this procedure. For example, several studies have demonstrated a very high risk of MEBT in unconscious patients.[5,11] We believe this risk warrants the routine insertion of ventilation tubes before HBO_2 in these patients.

In awake patients, most cases of MEBT are relatively benign and even a TM rupture is likely to heal spontaneously with little morbidity expected. However, the animal and human diving literature does describe serious middle and inner ear damage from barotrauma, including oval/round window rupture, perilymph fistula and hearing loss. Inner ear damage, although rare, could be catastrophic, with potential for lifelong hearing damage.[3,11]

Assessing ET function accurately at the bedside is not an easy task. Several previous authors have discussed the problems. Beuerlein employed visualisation of the TM during the Valsalva manoeuvre to classify the patient as an 'autoinflater' or 'noninflater' as a means of predicting MEBT.[11] We used the 'Static Tymp' Test in a previous study[10] and found that all patients with an abnormal test developed MEBT. However, if the test was normal, the result was unhelpful in predicting MEBT. The current series found a 63% positive predictive value (abnormal 'Static Tymp' Test) but confirmed the poor sensitivity of this test. Both this and our previous study of the 'Static Tymp' Test show a poor sensitivity. Static tympanometry may be better suited to detect middle ear effusions.[3]

Dynamic ET function testing can be done in several ways. The best known is the 9-step inflation/deflation test described by Bluestone and used in the previously mentioned study by Fernau where patients were categorised as 'autoinflaters' or 'noninflaters'.[7] Fernau found that this test was best at predicting subjects at increased risk of MEBT if it was performed after the first HBO_2. Our 'Dynamic Tymp' Test is a similar, but simpler, version of the same test using the Welch Allyn Microtymp instrument. In our study, this test ('Dynamic Tymp', one or both sides abnormal) has a high sensitivity (0.89) but a low specificity (0.29) for MEBT.

From our regression analysis, we introduce a novel concept of combining two tests (TM Mobility and 'Dynamic Tymp') to stratify patients for risk of MEBT. Because of the complementary combination of a low-sensitivity, high-specificity test (TM Mobility) and a high-sensitivity, low-specificity test ('Dynamic Tymp'), risk of MEBT at the first HBO_2 can be stratified into low (12%), intermediate (32–40%) and high (70%) groups.

With identification of patients at increased risk of all grades of MEBT, it may be possible to decrease patient and middle ear morbidity by intensifying patient education, maximising equalisation practice and advocating selective insertion of tympanostomy tubes. It may also prevent the rare case of serious inner ear damage. A decrease in ear morbidity will also result in fewer delays and cancellations of treatments and may also decrease the compression times for multiplace treatment sessions.

Conclusions

We propose that the combination of two simple, clinical tests can stratify patients into three risk categories for MEBT. This stratification may be useful in directing educational efforts and identifying patients at increased risk of MEBT during the first HBO_2. Further studies are required to validate this concept.

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Review article

From police to health adviser: the evolution of modern occupational health surveillance

Des F Gorman

Key words
Occupational diving, fitness to dive, health surveillance, diving industry, medicals - diving, questionnaire, standards

Abstract
(Gorman DF. From police to health adviser: the evolution of modern occupational health surveillance. *SPUMS J* 2003; 33: 134-139) Occupational health surveillance differs from healthcare-oriented surveys, and is an exercise in facilitated, informed risk management. In most situations, issues of condition importance and prevalence determine the survey composition and issues of predictive power should be the basis of survey type. This is usually not the case, as well illustrated by medical assessments of 'fitness' for diving. There is a need to introduce discretion into what has been inappropriately prescribed practice. There is also a need to emphasise functional capacity testing at the expense of medical examination and investigation.

Introduction

Occupational diving (diving) is a good model with which to demonstrate the principles of modern occupational health surveillance.

There is a difference between healthcare-oriented and occupational health surveillance. The former is usually predicated on the basis that early detection of and consequent intervention for disease will alter the outcome for the individual and/or society. This requires knowledge of the appropriate numbers needed to survey (NNS) and treat (NNT) and of numbers needed to harm (NNH) before the utility of a survey can be assessed. Failure to assess these numbers can result in harmful and or ineffective surveys; screening for breast cancer with mammography is an example of such a poor survey.[1]

Occupational health surveillance on the other hand is more responsive to duty-of-care responsibilities that arise from health and safety in employment legislation by the prohibition of discrimination on the basis of disability intrinsic to human rights-type legislation, and is highly influenced by privacy legislation. As a result, a health condition may be selected for survey for which there is no meaningful intervention. Generally, the process is highly dependent on selecting important and prevalent conditions, so that the survey will have high predictive power.[2]

Nevertheless, some health conditions may be included that do not exist at appropriate levels of prevalence. This highlights a difference between clinical and statistical significance. Some conditions may be unusual in such a (potential) worker cohort, but of great clinical importance, for instance epilepsy and diving, such that concerns about statistical validity are superseded by the need for detection. Some others need to be included for purposes of baseline data collection. An example of the latter is audiology, where many of those undergoing an initial medical assessment for diving had pre-existing hearing loss.[3]

The case for occupational health surveillance

When the New Zealand Department of Labour decided to introduce a standard medical assessment for all occupational divers,[4] many recreational diver instructors raised questions about the rationale for their inclusion. These questions well illustrate the generic argument about the utility of occupational health surveillance and will be addressed here in a question and answer format.

What evidence is there that assessing a person's fitness to dive affects the morbidity and mortality of recreational and occupational diving?

Notwithstanding the biological nonsense of a human being 'fit to dive', this question cannot be answered and to a large extent misses the point. The reason for the former is that no such data have been collected and, even if an attempt were made to do so, would be unlikely to confidently answer the question. Based on anecdotal data, my estimate is that the incidence of decompression illness (DCI) in Australian recreational divers is about 1/10,000 decompressions. My estimate of the fatality rate from any cause for this group would be about 1/100,000 hours of diving. It is clear that a study sufficiently powered to have an 80% likelihood of demonstrating a doubling of mortality after diving, in association with a subject disease of even high prevalence,

for instance asthma, would have to be based on more than one million hours of diving exposure.[2]

Some attempts have been made in this context; for example, asthma is said to double the risk of a lung injury after emergency ascent training from about 1:60,000 to 1:30,000 ascents,[5,6] but the statistical basis of this claim is not robust. It is also for this reason that a 'numeric wellness outcome' was used by Dr David Doolette and me to estimate the utility of decompression practice in South Australian tuna farm divers rather than the 'occurrence of DCI', which is after all an arbitrary threshold-based outcome category imposed on the spectrum of diver health status.[7]

The problem of answering any question about the utility of a health survey here to improve health outcomes after diving is also exaggerated by the following. First, the traditional, prescriptive approach to diving medical assessments has resulted in divers 'shopping around' for medical clearances and progressively greater withholding of key health information.[8] This is certainly my anecdotal experience with New Zealand occupational divers, where a change from such an approach to a more discretionary one has resulted in many long-time divers admitting to health problems that were both long-standing and previously undeclared. Second, the cause of more than 80% of all events that can or do lead to harm in any occupational setting is human error and/or violations of accepted practice, and not uncontrollable hazards and predisposing health problems.[9]

The Diving Incident Monitoring Study suggests that these conclusions can be extrapolated to diving,[10-12] and that study of the subject of diving accident causation would be better facilitated by consideration of the protection motivation theory than any survey of underlying health conditions.[13] This theory proposes that five factors predict preventive behaviour for health-at-risk issues: perceived susceptibility; perceived severity of the health consequences; perceived efficacy of taking the possible health action; perceived barriers to taking action; and self-efficacy expectancy of taking the action.

Most importantly, however, the question of the efficacy of occupational health surveillance in terms of influencing the morbidity and mortality of diving does miss the point, as such surveys should be an exercise of hazard identification, risk assessment and explanation, and of risk acceptance and/or rejection. That is, the primary obligation of occupational health surveillance is to facilitate informed choice by workers, employers, insurers and society.

What is the significance of duties of care intrinsic to health-in-employment legislation, of privacy legislation and of human rights legislation?

As implied above, such legislation is central to occupational health surveillance. A duty of care is intrinsic to health-in-employment legislation. Occupational diving is variably physically and psychologically demanding and occurs in an unpredictable, mobile, dense, irrespirable environment.[14] Although human error is primarily responsible for most diving incidents and accidents,[9] it is essential that a person's work-related health risks are known if they are to be managed adequately.[10-12] However, the issue here is how best to assess such risks and to what extent this can be done by a medical practitioner (doctor).

Privacy legislation is often cited as a reason against discretionary health surveillance, in that secondary risk takers, such as employers, government agencies, dive school instructors and insurers, are consequently excluded. This is false; privacy legislation simply requires that consent to inform other risk takers is obtained prior to the survey and that this consent be based on knowledge of the way in which the health data will be collected, analysed, stored and shared. Doctors need to develop the skill of information management.

For example, the requirement of a job may be that the workers need to be blood donors because of the remoteness of the work site. A potential worker is found to be HIV positive. The employer does not need to know such detail, and can make a decision about employment here if simply told that the potential worker is unsuitable for blood donation. It is my experience that too often doctors either breach their patient's or client's privacy by telling secondarily involved people and agencies 'everything', or prevent sensible decision making by telling them 'nothing'.

Human rights or disabled peoples legislation should have the greatest bearing on occupational health surveillance. That it does not is testimony to this 'gold mine' having yet to be discovered by the Australasian legal profession. To paraphrase, central to such legislation is the concept that someone cannot be denied employment on the basis of a disability unless that disability precludes them from being able to undertake the tasks required, and/or the person's health condition represents an unacceptable risk to them and/or those whom they are to work with, at work. All practicable, which does not mean convenient or cheap, steps should be taken to accommodate that disability.

It is clear, then, that any occupational health surveillance must be prefaced by a thorough knowledge of the functional requirements of the employment; that is, a functional rather than a strategic job description. It is also clear most jobs have not been so analysed and why a course to train doctors to assess diver fitness must be based on knowledge of the diving environment. On this basis, many standards of 'occupational fitness' are seen to be discriminatory.

For example, admission to one naval diver-training programme requires the candidate to complete a two-mile run in a set maximum time of 11 minutes. This is sexually discriminatory, as most women cannot satisfy this requirement and it is difficult to see how conventional naval

diving requires such running skills. By contrast, a standard based on having to be able to swim 400 metres using fins against a 1.5-knot current would satisfy any human rights legislation. Finally, while a functional orientation is essential, a medicalisation of the assessment is not. That is, whether or not a candidate can meet the functional requirements of a job are better assessed by functional capacity assessments (physical competency testing) than by facsimile testing in a doctor's rooms.[15]

Is there a difference between the health surveillance needs of different occupational diving groups (e.g., recreational diving instructors versus repair and construction divers)?

Although health professionals often assume the role, adoption of thresholds of acceptable risk at work is actually a societal responsibility.[16,17] In the case of diving, it is easiest to consider differential health survey thresholds and hence process by considering categories of private and public risk. The booted, helmeted construction diver has little of either because of duplicate systems, surface support, a standby diver and the absence of any dependent workers. By contrast, the free-swimming recreational diver instructor has much greater levels of both, as they have in real terms little equipment redundancy and are responsible for the health and safety of diving students and novices. Any differential standard, then, would be based on a requirement for a higher level of health reliability for those employed in the recreational diving industry. However, a common standard is preferable in most jurisdictions as divers usually are variously and not specifically employed.

In what role is the doctor engaged when undertaking an assessment of a recreational and an occupational diver's fitness for diving?

Doctors and their regulatory authorities are often unaware of the nature of their role when performing occupational health surveys.[18] Whereas the usual role in this context is that of undertaking an audit as a commissioned agent of a third party, such as an employer, government agency, insurer or diving school,[19] many doctors mistakenly believe that they have a doctor-patient relationship with the person being surveyed. This frequently leads to inappropriate advocacy behaviour; for example, both Sir John Scott and I, and a New Zealand coroner, considered this phenomenon to be a significant contributor to the dysfunction of the system used to assess medical 'fitness to fly' in pilots in New Zealand (see below).[16,17,20]

Careful attention is also needed to the semantics of any subsequent certification to ensure that the doctor's role is that of risk assessment and explanation and not that of risk acceptor. Given that an air-breathing mammal with negligible diving reflex such as *Homo sapiens* can never be literally 'fit to dive', for reasons of medico-legal prudence alone, certification should avoid such statements and conform instead to informed consent formats.[21]

Bad health surveillance

An analysis of bad health surveillance programmes is informative with respect to identifying the principles of effective health surveillance.

ROYAL AUSTRALIAN NAVY'S OBESITY SCREENING PROGRAMME

The Royal Australian Navy (RAN) had a long-standing obesity 'detection' programme, based on a variety of measures such as height, weight, skin-fold thickness, neck girth, body-mass index and even density.[22] The origins of the programme, which was expensive and produced many anomalies with respect to fitness, e.g., rugby front-row forwards, are uncertain. One anecdotal explanation was that the programme was a response to obese sailors not being able to escape from a stricken ship through small openings in the hull.

Assuming some veracity for this claim, this is a good example of 'medicalisation' of a functional issue.[15] At body temperature, human fat is fluid and consequently obese people are quite capable of negotiating small openings, much more so than those with broad skeletal and muscular shoulder girdles. The simple measure here would be an employment requirement to be able to pass through a standard opening, a process that does not require a doctor's assessment.

THE NEW ZEALAND CIVIL AVIATION AUTHORITY'S PROCESS OF ASSESSING PILOTS' FITNESS TO FLY

In the 1980s, the aviation industry in the United Kingdom and elsewhere recognised the hazards inherent in management-employee interactions and the "lethal combination of human error and a weak organizational structure".[23] In 1999, Noah (pg 242), stated

> "*Once the definition and identification of illness begin primarily to serve the needs of non-medical decision makers, such as insurer, regulatory agencies, and litigants, closer scrutiny of the diagnostic process is warranted.*"[24]

Sir John Scott and I undertook a random audit of the New Zealand Civil Aviation Authority pilot-medical files.[17] More than half of the files were flawed; most flaws were trivial but some errors certified pilots as 'fit to fly' when they should not have been so licensed. We thought that this unacceptably poor practice did not have its roots in doctor dishonesty or incompetence, but rather in system design with consequent funder capture. The system contained no systematically established external audit or rigid, mandatory confidential reporting to enhance safety through identification of problems involving individual pilots.

Not surprisingly, there had been an observable drift in practice, sustained by collegial reinforcement.[25] The pilots

funded the medical assessment system, and it quickly became responsive to their needs. Some doctors lost perspective of their primary obligation to the government and people of New Zealand. As cited briefly above, they saw their primary role as that of pilot advocacy.[16,17,20]

Concurrently, the New Zealand Medical Council's draft guidelines on 'certification' stated that a doctor's first responsibility was "to the patient".[18] This is wrong in law. Rather, in the context of certification, and particularly for a third party, such as the Department of Labour or an insurance company, it is clear that the legal responsibility of a medical practitioner is to that third party for whom they are acting as a commissioned agent.[19]

OCCUPATIONAL DIVERS IN NEW ZEALAND AND THE AUSTRALIAN AND NEW ZEALAND STANDARD AS/NZ 2299

An analysis of occupational diver assessments performed according to Australian and New Zealand Standard AS/NZ 2299 has shown a profile of low positive predictive values (PPV) and high negative predictive values (NPV), which would be far more useful in diagnosis than in health surveillance.[3,26] This observation is understandable given that this standard was based on a list of diseases considered to be either absolute or relative contraindications to diving. That is, the standard was designed to be diagnostic and not a survey of health risks at work.

In the analysis of the standard,[3] none of the questionnaire, examination and investigation items, alone or in combination, offered an acceptable balance of sensitivity and specificity. The data also 'suggested' that decisions regarding fitness to dive were not based so much on the questionnaire response, but more so on the free-text component.

It follows that if the AS/NZ 2299 questionnaire is to be used, then review of the yes/no responses, free-text clarification by the medical assessor and a subsequent clinical audit are critical components. It is also apparent that such an assessor cannot be naïve in the context of diving medicine. However, the real conclusion here is that any tool used to survey 'fitness for work' must be evaluated for consumer understanding and for both statistical reliability and reproducibility.[2] Few have been so in practice.

BREAST CANCER SCREENING BY MAMMOGRAPHY AND HIV SCREENING IN THE AUSTRALIAN MILITARY

Despite the claims of the related health-disease industry, breast cancer screening by mammography does not appear effective. The NNS to prevent a death is very high and the NNH, by way of surgery for benign masses, is low.[1] The reason for this is that in young women the incidence of benign masses is high, the incidence of malignant masses is low and the natural history of the latter is virulent. In this environment, the predictive power of almost any survey will be unacceptable.[2] The same situation of low prevalence and consequent poor predictive power explains the biopsychosocial harmfulness and low utility of the RAN's HIV screening programme. My assessment of this programme showed a positive predictive power of demonstrating infection with HIV in the first phase of the programme of only 6.7%.[2]

The evolution from prescribed to discretionary formats for occupational health surveillance

The traditional prescribed approach to determining 'occupational fitness' has actuarial roots. By prescribing thresholds of fitness and identifying those diseases considered contraindications to employment, actuaries are able to estimate risk and cost (premium). The approach was adopted by naval diving authorities,[22,27] and subsequently by Standards Australia,[26] given the appeal of an apparently consistent system and in response to increasing problems of diver morbidity and mortality. However, the sole strength of a prescribed approach is that the doctor undertaking the survey needs only to be able to interpret and administer the prescription; that is, they can be naïve.

By contrast, there are several weaknesses to such an approach. Firstly, the key risk taker is excluded from any involvement, which leads to both the shopping around behaviour and loss of veracity cited above.[8] Secondly, divers continue to dive despite medical contra-indications.[28] Thirdly, only a few health conditions, e.g., visual acuity and hearing, are able to be defined by prescription, whereas most, e.g., asthma, are not and sensible case definitions are elusive.[6]

It is not surprising, therefore, that our audit of AS/NZ 2299 has been shown to be of limited utility.[3,26] There is an inevitable need for centralised audit and for arbitration.

A discretionary approach to assessing 'occupational fitness' is to a large extent a response to the problems of prescriptions and to some degree is a predictable over-reaction. In this model of survey, the risks associated with the job for the person are identified and explained and any decision is left to the person concerned. The primary strength is that the key risk taker is central to the process, although there is a danger of excluding other risk takers as discussed above in the context of privacy legislation. The major weakness in administration is that only knowledgeable doctors can be used and the major flaw in application is that objective data usually do not exist so that most risk explanation is qualitative and not quantitative. For example, there is no diving-relevant test of cardiovascular fitness, and lung function tests are poorly predictive of risk for lung injury due to decompression barotrauma.[29]

The obvious solution is a 'halfway house', based on a kernel of community-determined, prohibited conditions. That is, a prescriptive element of conditions such as active exercise-induced asthma, epilepsy, insulin-dependent diabetes and ischaemic heart disease for divers is combined with a much larger penumbra of conditions for which discretion is exercised by the diver and secondary risk takers.

The process of modern occupational health surveillance

Modern health surveillance has three steps: the identification of relevant conditions for survey, the selection of survey tools and process and the audit of survey efficacy, with modification as necessary.

A condition here may refer to a disease, such as asthma, a treatment of such a disease, such as ß-blockade for hypertension, a state of aerobic fitness or even to an anthropometric measure. An example of the latter is the length of the thigh in a jet-fighter-pilot candidate in the context of ejection seats and the distance from the seat back to the rigid console. The key issues for selection are importance, which as discussed already is a literal and figurative functional outcome of human rights legislation, and prevalence, as demonstrated by some of the examples of poor health surveillance cited above.

I recommend the following four 'questions' are addressed to identify 'importance':

1 The effect that the condition will have on the person's ability to undertake the requisite tasks;
2 The effect that the work and work environment will have on the condition;
3 The effect that the condition will have on the 'safety' at work of the person and those with whom they work;
4 The effect that the condition will have on the likelihood of a work-related illness or injury.

Using asthma as an example shows that this condition is very important in the diving environment for several reasons. Impaired respiration will limit exercise tolerance and work performance. Many aspects of the diving environment will precipitate asthma. Asthma will increase the likelihood of drowning by impairing the diver's ability to swim ashore or back to the boat, etc; there are some weak data to this effect.[30] The safety of the diver's workmates, at least during rescues, will be compromised. The risk of pulmonary barotrauma may double, as cited above.[5,6] Similar to asthma, sickle cell disease and TB would also be selected as being important conditions in divers. However, as well demonstrated by the examples of breast cancer and HIV infection, predictive power is determined by prevalence,[2] such that in a developed European society only asthma would be selected from this group for inclusion in a survey of divers.

The selection of survey tools should be determined in the context of several factors. Unless the language of a questionnaire is tested against the subject population, it may not be intelligible to the consumers. This was my strong, anecdotal experience in developing the current questionnaire used for New Zealand occupational divers in lieu of AS/NZ 2299. It may also have a low PPV.[3,26] The yield of physical findings obtained in the absence of a suggestive history and that affect outcome of the survey is very low, and even lower for most investigations. Most relevant conditions are best tested functionally and are better not 'medicalised'.[15] Some of the key issues, for instance emotional coping skills, aquaphobia and claustrophobia in diver candidates, are impossible to test effectively in a doctor's rooms.

It is clear that a well-constructed questionnaire will be the ideal triage and that the undertaking of any physical examination and investigations should be predicated by some history suggestive of an important condition. It is too soon to assess the utility of the current New Zealand occupational diver health questionnaire. However, the recent audit of the initial assessment of these divers revealed only three independent predictors of certification outcome: a past history of asthma ($p < 0.0001$), abnormal cardiac auscultation ($p < 0.0005$) and abnormal respiratory function tests ($p < 0.0001$).[3]

Some tests do need inclusion for reasons of baseline, such as audiology given that many diver candidates were shown to have significant hearing loss before beginning their diving careers.[3] Medico-legal considerations may encourage some diver-employers to also insist on pre-employment X-rays of long bones and on psychometric testing. Unfortunately, as already stated, given the significance of cardio-respiratory fitness to survival in the ocean as a diver, there is no diving-relevant test of cardiovascular fitness and lung function tests are poorly predictive of risk for lung injury due to decompression barotrauma.[29]

Current international practice for occupational divers is to insist on annual assessment. There is no logic to this frequency and, although ageist,[31] an age-dependent frequency of assessment would be more sensible. Similarly, some tests do not require iteration; for example, the forced vital capacity, the only weakly predictive respiratory parameter for lung injury in divers,[29] does not change significantly after adolescence. The efficacy of substituting a questionnaire as a triage tool for the routine, full, annual AS/NZ 2299 [26] assessment for years 2 to 5 inclusive for every five-year cycle for New Zealand occupational divers will be tested later this year.

Summary

Occupational health surveillance is a demanding process of risk management and should conform to the principles of this discipline. Current practice generally falls well short of an acceptable standard. Diving has been used here to illustrate the modern principles of such surveys.

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The world as it is

Ethical dilemmas and diving medicine: asthma and recreational diving ñ a hypothetical case report

Michael H Bennett and Stephen Grant

Key words
Asthma, diving medical, recreational diving, ethics, legal and insurance

Abstract
(Bennett MH, Grant S. Ethical dilemmas and diving medicine: asthma and recreational diving - a hypothetical case report. *SPUMS J* 2003; 33: 140-145) A hypothetical case report is presented involving two diving medical practitioners, two dive training candidates and an unusual chain of events. This explores a number of ethical problems that might arise for doctors who perform diving medical examinations. Any resemblance of actual persons to characters depicted in this account is entirely coincidental.

Introduction

An hypothetical case report is presented concerning two diving medical practitioners, two dive training candidates and an unusual chain of events. This scenario was originally developed for live presentation at the 2001 SPUMS meeting in Madang, Papua New Guinea, in order to illustrate and explore a number of ethical problems that might arise for those of us who perform diving medical examinations. It is entirely a work of fiction, and any resemblance of actual persons to characters depicted is entirely coincidental.

This report is presented as a series of situations with questions that might be worth considering at each step. I suggest you pause at each set of questions and consider what your course of action might be in the same situation. There are no correct answers, but a summary of suggestions from the floor during the original presentation is given at the end of the presentation of events. Many thanks to all those who contributed, particularly our panel members Guy Williams, Robyn Walker, Drew Richardson, Barbara Trytko, Simon Mitchell and Hamish Turnbull.

Part 1: The initial consultations

Dr W is a general practitioner and a member of SPUMS with a long-standing interest in diving medicine. He is an active scuba diver himself, and works diligently to remain well informed of the medical aspects of this activity. In February 2001, he was consulted by a 30-year-old male (SB) who wished to undertake recreational scuba diving. Dr W asked him to complete the SPUMS diving medical questionnaire prior to formal interview and examination.

On presenting his form, Dr W noted that SB had given a positive response to the question concerning asthma. Further questioning revealed that SB had been diagnosed asthmatic at the age of seven, following a visit to his local hospital emergency department. He frequently used bronchodilators when younger, particularly in association with upper respiratory tract infections and exercise. His episodes of wheezing had gradually subsided and he had required no specific treatment since he was 16. He specifically denied any wheezing, nocturnal coughing or other symptoms since that time and had frequently exercised strenuously with no sign of bronchospasm.

Physical examination was unremarkable. Ventilatory function tests (VFTs) performed in the surgery were: forced vital capacity (FVC) 4.15 l (101% normal), forced expiratory volume in 1 second (FEV_1) 3.70 l (103% normal), FEV1/FVC 89%, peak expiratory flow (PEF) 480 $l.min^{-1}$ (99% normal) and forced expiratory flow between 25% and 75% of FVC (FEF_{25-75}) 95% of predicted.

Meanwhile, across the road Dr B, a general practitioner with similar interests and abilities, is also seeing a recreational diving candidate. In this case the candidate is a 32-year-old female (UB) who has similarly indicated a history of asthma on her questionnaire. She is otherwise well, participates in active sports almost daily, and a month before this consultation she had successfully completed the Sydney half-marathon run.

In this case, further questioning revealed a history of bronchospasm since childhood with frequent use of beta-agonist inhalers. UB reported with some enthusiasm, however, that all has been pretty well since starting on regular steroid inhalers some four years previously following a brief admission to an intensive care unit for control of bronchospasm (formal mechanical ventilation not required). Since that time, she has used Ventolin only occasionally, perhaps once every six weeks or so. She does not become wheezy with exercise, but may do so when it is cold.

Physical examination revealed no specific abnormality, in particular no signs of bronchospasm. VFTs could not be performed as Dr B's vitalograph was undergoing its regular six-monthly service. The machine would, however, be available again in an hour or so.

QUESTIONS:

1. *What further investigations (if any) would you like to perform for each of these candidates?*
2. *What specialist referral would you make, (if any) for these candidates?*
3. *What would be your advice to these two candidates?*
4. *What is the medicolegal position regarding these medicals?*

Dr B decided that UB was unfit for recreational diving given her clinical history and was not willing to sign the diving medical certificate as requested by the training agency. UB was very unhappy with this decision given her general level of fitness and stormed out of the office. She refused to pay for the consultation.

As she left the office, UB noticed the surgery across the road and, armed with her new knowledge of diving medicine, decided to seek a further consultation. She saw Dr W, but this time failed to reveal her history of asthma. Her examination proceeded smoothly and she was certified to undertake scuba training.

Part 2: Getting away from it all

Three months later the two good friends, Drs B and W, arrived on board the *Black Pig* for their live-aboard diving holiday around the islands of Papua New Guinea. They were greeted by their host, Captain Pugwash, their divemaster, Charlie Hook, and introduced to their fellow-divers for the week. Somewhat to their dismay, they found both SB and UB on board and ready for the tropical diving honeymoon they had planned now both were fully certified 'open water' divers. Dr W was somewhat embarrassed to be living and diving with two of his patients for the week, while Dr B was quite distressed to see UB on board. He saw a dilemma and retreated to the cabin he shared with Dr W to think the situation over.

QUESTIONS:

5. *Should Dr B discuss the situation with Dr W?*
6. *Should Dr B discuss the situation with UB?*
7. *Should Dr B discuss the situation with the crew?*

Dr B first decided to discuss the situation with his colleague and friend, Dr W. Dr W shared his concern, particularly given that UB would appear to have deliberately concealed from him important information that would bear directly on her fitness to dive. They decide that Dr B should approach UB and articulate their concerns and advice.

UB appeared unabashed by the dilemmas that so disturbed Dr B. Specifically, when asked about her responses to questions about her asthma, she replied that she was confused by the first questionnaire and more appropriate with her responses on the second. In any case, she pointed out that she understood her disease better than Drs B or W, and had done 10 dives since her dive medical without problems. She had been able to swim to the boat without getting breathless, had not had any wheeze at all and ensured this by always taking Ventolin before a dive. This holiday was so important to her and her husband that she could not see why Dr B would want to spoil everything for them now. She specifically instructed Dr B not to tell her partner or any of the crew about her condition.

In fact, SB and UB were travelling with two friends on this diving holiday of a lifetime, one of whom was James Suckit, a solicitor from the Sydney firm Suckit and See. UB decided she should confide in James and seek some advice from him concerning her legal situation should Dr B inform the divemaster of the situation. She told James that she had been advised by Dr B that she was not fit for diving, but had disagreed with this assessment and sought an opinion with Dr W. Dr W had cleared her to dive, but she was concerned that Dr B may tell the divemaster that she should not be diving.

QUESTIONS:

8. *What further action should Dr B take, if any?*
9. *What would be reasonable advice from Mr Suckit?*
10. *Does the candidate signature on the diving medical form allow a practitioner to divulge information to others?*
11. *Would you dive as a buddy with UB?*

Part 3: The first dive

The first planned dive was a descent onto a wall. Maximum depth was to be about 30 metres, with the bottom at about 60 metres. There was a 3 knot current running along the reef, so the dive planned was a drift, and the visibility was unfortunately quite poor, 4 to 5 m, following a recent storm.

SB and UB had done 10 ocean dives since qualifying as open water divers, and were very keen to get in the water. James and his partner were supposedly much more experienced, and claimed to have done a Nitrox course, but James had left both his C-card and nitrox card at home. They requested nitrox for their dive as it "would allow them to go deeper with safety". James seemed surprisingly unfamiliar with his equipment given these claims and

required a lot of assistance from his buddy and legal partner, Jane See, before he was ready for the water. He requested two weight belts to carry his 'usual' complement of lead shot, and was clearly very negatively buoyant as he prepared for entry.

QUESTIONS:

12. Should the divemaster allow SB and UB to make the dive?
13. Should the divemaster allow James and Jane make the dive?
14. What is the advantage of nitrox mixtures?
15. What would you do in this situation if you were Dr B?

Unfortunately, there was a bit of trouble on the first dive. James was pretty clumsy in the water and, as he flailed about at 15 metres, he kicked UB's regulator out of her mouth. UB, not particularly experienced or comfortable in the water, had trouble replacing it, inhaled a little water and made a rapid swimming ascent to the surface. She felt anxious and a little wheezy at the surface but regained the boat and made for her cabin. One of the crew, concerned at her early appearance, followed her to the cabin where he saw her using a Ventolin inhaler. Now very concerned for her safety, he found a quiet moment to report what he had seen to the divemaster, Charlie Hook, when he returned to the boat. UB, however, seemed to recover well and was bright and cheerful that evening as they contemplated better diving the next day.

QUESTIONS:

16. Should the divemaster confront UB?
17. Should the divemaster involve the doctors on board?
18. If so, with or without the knowledge of UB?

Part 4. The last dive

The second day passed uneventfully with three pleasant, relatively shallow dives in clear, calm conditions. Charlie had been reassured by UB that she was fine and not an asthmatic, rather she was an occasional Ventolin user. He had agreed to her request not to involve the doctors.

On the third day, the party planned a 40-metre dive in the morning to the wreck of a B-52 bomber and with considerable anticipation they made their preparations. Unfortunately, their dive did not proceed smoothly. At 20 metres on the ascent, James ran out of air and attempted to wrench Jane's regulator from her mouth. She went on to her octopus, but within a few breaths, she too was out of air and they both made a grab for UB's octopus. There is some confusion over what happened next, but there was widespread panic in the water and SB was rendered unconscious from a blow from James' flailing fist. He was recovered at 30 metres by Charlie and brought to the surface, where he regained consciousness. Meanwhile, Jane and James safely reached the surface using the two doctors' octopus supplies.

UB was clearly distressed by witnessing what she assumed to be the demise of her newly acquired husband and it seems she made a panic ascent to the surface. She was found unconscious at the surface and did not respond to vigorous resuscitation by the crew and physicians present. She was pronounced dead by Dr B 45 minutes after being located on the surface.

QUESTIONS:

19. What is the most likely reason for UB's death?
20. Specifically, is asthma likely, or relatively unlikely, to have contributed to her death?
21. If there is any blame to be apportioned for her death, where do you think that blame lies?

Part 5: Summary of responses from the floor of the meeting

1. Most physicians felt that SB was certifiable as fit for dive training, although there was some discussion as to whether he deserved a provocation test with hypertonic saline and/or a chest X-ray. The 14-year symptom-free period and normal spirometry (particularly FEF_{25-75}) were accepted by many to be sufficient evidence to certify without further testing. The great majority agreed that UB was unfit to undertake dive training, and that a provocation test would not alter this decision one way or the other.

2. Probably no specialist referral was required for SB. UB could be referred if she desired and was not convinced that her condition was not compatible with scuba diving. Sophisticated testing would be very likely to confirm a risk of bronchospasm and therefore a significant risk of harm associated with diving.

3. Most agreed they would advise SB to beware of diving with any signs of wheeze and to seek medical advice if he had any return of asthma symptoms. All agreed UB should be counselled fully on the nature of the potential problems she faced and given a clear explication of the risks involved with her scuba diving. She should probably be given the opportunity for an informed second or specialist opinion should she desire further advice.

4. A medical certification for fitness to undertake training for scuba is not a legal requirement in Australian states other than Queensland, or in the USA or New Zealand. All the usual obligations will apply once an evaluation has been

undertaken, however. In general, reputable instruction agencies in Australia will not accept for training any candidate who has not been 'passed' by a medical practitioner as fit. There is a considerable grey area in relation to the increasingly common practice of advising in writing of specific risks for an individual candidate, as opposed to a black and white yes/no fitness declaration.

5. Such a discussion is very likely to constitute a breach of confidentiality if Dr B has been given no specific consent for it. It is not clear how this position changes if he is made aware that his friend Dr W has also seen UB. Nevertheless, a majority of the participants did not feel it was unethical or unwise for Dr B to discuss the consultation he had undertaken with his colleague Dr W. It should be noted that the candidate signs a statement that allows the physician to "supply information in regard to my medical fitness to dive to the diving instructor". As the instructor is not aboard, this would not seem to bear on the situation.

6. The consensus was that it would be appropriate for Dr B to approach UB for a private discussion of the situation. There were widely different opinions on the most appropriate things to say, however! Some felt it was appropriate only to re-emphasise the potential dangers of diving with asthma, while others were more assertive. Some suggested the doctor should make it clear that he was not prepared to dive with UB, and that he intended to make it known to the crew and other divers that he would not do so – but without specifying to them why he had made this decision. Nobody was prepared to divulge the medical details to the others on board.

7. See above. Some physicians felt they had a duty to the crew and divers to make them aware that a potential buddy might be more of a liability than an asset; however, no-one clearly asserted that they would inform third parties without the consent of UB herself.

8 and 9. The group did not feel the response of UB would alter their decisions as described above in (6).

10. The consent to divulge details to others is limited as discussed in (5) above. Certainly, clear instructions not to divulge this information to others, as in this case, compelled Dr B not to do so. Some noted that there would be a point at which not to do so would constitute danger to others and may equally compel Dr B to reveal the medical information. All agreed this was not a pleasant prospect.

11. All participants agreed they would not dive as the designated buddy to UB under these circumstances, and that to do so may constitute, at least in part, an endorsement of her fitness dive under these circumstances.

12. Most participants did not feel the dive as planned was suitable for these two relatively inexperienced divers. It was noted that a competent and professional divemaster would have already assessed the abilities of the divers on his boat and planned a dive more appropriate to those abilities. This is a most inappropriate first dive for a group unfamiliar to the divemaster and each other. It was suggested that in any marginal situation the divemaster would plan to accompany the novices on their first dive to further assess their abilities in the water. Gentle persuasion and suggestion was likely to achieve acceptable results in most situations, rather than a more aggressive and censuring approach.

13. All agreed that the attitude of this couple would set off alarm bells for the divemaster. In particular, the statement concerning nitrox would be worrying in the extreme and demonstrated a basic misunderstanding of the nature and purpose of this mix. The statements in (12) above are equally valid for this couple.

14. Nitrox mixtures are probably more usefully known as 'oxygen enriched air' for the purposes of scuba diving. They contain more oxygen than air and, consequently, for any given depth they provide a lower nitrogen load and consequent risk of decompression illness. This property has allowed the development of tables permitting increasingly longer bottom times for mixtures with an increasing proportion of oxygen. The critical corollary, however, is that with increasing oxygen proportions in the mix, the maximum safe depth at which the mixture can be breathed is reduced due to the risk of oxygen toxicity. Breathing nitrox 40 (40% oxygen, 60% nitrogen), for example, would result in a PO_2 of 1.6 Ata (162 kPa) at 30 m depth, and 1.4 Ata (141 kPa) at only 25 m. Nitrox breathing does not extend the safe depth capability compared with air.

15. See (12) and (13) above for discussion of some of the issues raised.

16. This is very difficult for the divemaster, who must act with tact and discretion to achieve the best outcome for all. The divemaster cannot be expected to assess the medical condition of UB and must act on her assurances that she does not have a medical contra-indication to diving. While he may have suspicions, the group felt he was not in a position to prevent UB from further diving unless he had confidence in his diagnosis.

17 and 18. The group felt that there was no contract for confidentiality between UB and the divemaster in this regard and that the divemaster could discuss his concerns with the doctors if he wished. The doctors would be expected to state their prior involvement with the subject but could not discuss her case specifically. They could respond in a general way concerning their opinion on diving and Ventolin use. Many considered the divemaster should inform UB of his intention to consult the doctors.

19. Most likely to be either pulmonary barotrauma and cerebral arterial gas embolism (CAGE) secondary to a panic ascent while breath-holding, or panic and subsequent

drowning.

20. It is not clear from the information given whether asthma would have contributed to this fatality. It is certainly possible for an individual with normal lungs to suffer barotrauma and CAGE in this situation. Aspiration followed by bronchospasm during the rapid ascent might contribute to barotrauma occurring, but it should be stressed that there is no evidence that such events are more or less likely in asthmatics than others. We simply do not have the data and work at present from biological plausibility.

21. This question was not addressed during the discussion at the ASM. In the author's (MB) opinion in this situation, UB is principally responsible for the circumstances that led to her scuba diving when the incident occurred. James Suckit and Jane See could be viewed as partially responsible for her death by causing her to panic and make her uncontrolled ascent. The responsibility of the divemaster is to the safe conduct of the dive and some might question the wisdom of the planned dive in the circumstances described. It should be noted, however, that the divemaster in this situation is not performing as an instructor and does not have that relationship with the divers on this boat.

Part 6: Legal commentary on issues raised by the hypothetical case report (the 'Pugwash' scenario)

- The Pugwash scenario describes a complicated sequence of events in which certain medical practitioners (also scuba divers) are asked to certify patients as fit to undertake scuba diving.
- SB is certified by Dr W as being fit for recreational scuba diving. The medical basis on which that certification is given is not a matter for legal opinion.
- UB is refused certification by Dr B, but subsequently, armed with the information gleaned from the first examination, she obtains certification from Dr W.

What is the medico-legal position regarding SB's treatment by Dr W, and UB's treatment by Dr B?

- SB's consultation is unremarkable, from a legal perspective. Whether or not SB was fit for diving is a medical question to be determined on relevant medical evidence.
- Similarly, the consultation between Dr B and UB is a medical matter.
- Nonetheless, the High Court in Australia has held that medical opinion is not determinative of the scope of a doctor's duty of care. A court will make its own decision, informed by relevant medical expert evidence, if necessary, as to what is the standard of care owed by a medical practitioner.
- Later Drs B and W meet SB and UB on board ship while on a diving holiday. This presents Dr B with a dilemma.

Should Dr B discuss the situation with Dr W?

- Dr B's consultation and the information provided to him during the course of that consultation are confidential to UB under doctor/patient privilege. It is not appropriate for Dr B to discuss UB's consultation with him with anybody else without the consent of his patient. The test for determining whether privilege has been waived is whether the patient's action is inconsistent with the maintenance of the privilege.
- It should be noted that there is some scope to argue that the doctor/patient privilege can be partially waived if disclosure would substantially benefit a general duty to society. A frequently used example is where a medical practitioner becomes aware of the patient committing a serious offence.
- Notwithstanding the fact that there is no duty on medical practitioners to disclose privileged information, case law suggests that a medical practitioner is prohibited from providing false or misleading information. In light of this, if Dr B is asked whether UB had seen him in relation to obtaining a diving medical certificate, he should ensure that whilst not disclosing specifics of the examination, he does not provide information contrary to his diagnosis of UB.

Should Dr B discuss the situation with UB?

- Dr B should discuss the issue with UB. While Dr B may have discharged his obligations as a consulting doctor to UB in the refusal of certification, that may not be the end of his obligation. The trend in liability law (including the law relating to medical practitioners and their duties) is towards the imposition of positive obligations on persons with responsibilities to their clients. If Dr B fails to act, UB may at a later time assert that he had breached his duty to her.
- A medical practitioner's duty extends not only to "the examination and diagnosis" of the patient, but also includes the "treatment of the patient and the provision of information in an appropriate case". Whilst it is arguable that Dr B's duty would extend to the given circumstances (he had already provided UB with a diagnosis), it would be unwise for Dr B not to speak to UB. At the very least, such discourse may be seen as a way for Dr B to confirm that UB understood his diagnosis and its implications.

Should Dr B discuss the situation with the crew?

- Dr B's obligation of confidentiality to UB prevents him from discussing the matter with the crew.
- Dr B discusses the matter with UB and she indicates that she will not refrain from diving. Dr B, bound by his duty of confidentiality, cannot take the matter further.
- UB seeks advice from James Suckitt, a solicitor. Reasonable advice from Mr Suckitt would be that she

should refrain from diving and that she places herself and other people at risk by undertaking diving when she has not been certified fit to dive after full disclosure of her medical condition.

Does the candidate's signature on the diving medical form allow a practitioner to divulge information to others?

- A practitioner may only divulge information to others to the extent permitted by the form.

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Doctor to the divers

Tony Slark

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(Slark AG. Doctor to the divers. *SPUMS J* 2003; 33: 145-148)

HMNZS Pukaki was returning from the Christmas Island hydrogen bomb tests in 1957 and we called at the northern Cook Island of Manihiki. There was no anchorage and the ship just steamed up and down the reef whilst the lucky members of the crew were taken ashore over the reef edge by longboats. I was asked to see one of the islanders in the village on the other side of the lagoon. He was a pearl shell diver. A couple of months earlier he had been hauled from the water after a heavy day's shell gathering, and had been unable to walk since. 'Taravana' they said. Paraplegia, this new doctor thought, but my training had not included anything of underwater medicine. In fact, the subject did not exist outside naval diving manuals. Sadly, I had not much to offer on this most isolated of Pacific islands. However, after our return to Auckland the Director of Naval Medical Services handed me the RN Manual, saying "Read this, my boy, you are to be the doctor to the divers."

A diving operation was to be undertaken in deep water off the coast of Great Barrier Island, to calibrate a submarine detection apparatus. The diving tender, an old tug called *Manawanui*, was to be accompanied by the oceanographic research vessel *HMNZS Tui* which would carry a recompression chamber capable of simulated depths of 100 ft. At our base in the dockyard a larger chamber with a 300 ft depth capability was installed for training and treatment.

The operation required the divers to move a steel arm following surface instructions at a depth of 250 ft. On one dive, after achieving this with some difficulty, the lead diver was back on the surface after a prolonged decompression on air and oxygen. We were all relaxing, time for a 'tot', but the diver said "No thanks, I don't feel like it." He had a preoccupied look. Then I saw him rubbing his left shoulder. Yes, it was hurting, he said. In the chamber at its maximum 100 ft level he was completely better, so he was decompressed in accordance with the treatment schedule. Fortunately, he did not get bent again.

After this I decided that I had better get to know more about the whole business of diving so I asked to do the basic diving courses. The first was the 'shallow water' course in which we learnt how to use oxygen rebreathing apparatus to do ship's bottom searches. As a diving system it was anything but foolproof, and could and did go wrong in many different ways. At that time, I was introduced to the service principle that being called "Sir!" did not afford protection from forthright verbal attacks on one's capabilities by the instructors if their advice was ignored. Those of us who performed a satisfactory bottom search at night and passed were given a special treat. We were able to have a dive in less murky water with an open circuit air breathing set. And so I was introduced to a new and growing sport.

Straight after getting married and my joining the Royal New Zealand Navy, Eileen and I had sailed for New Zealand (NZ) from England in June 1956. Ambitions to travel, have a nautical field of work, and to marry, all seemed to combine nicely. It was not long after our arrival in the Pacific, therefore, that the events above occurred. In 1960, I returned to England for postgraduate study, for which I decided to do Occupational Medicine. On the completion of this I was allowed to go to the Royal Naval Physiological Laboratory (RNPL) for six months before returning to NZ – a splendid opportunity. At that time, Peter Bennett was at RNPL working on nitrogen narcosis, and Val Hempleman was formulating helium diving tables. My mentor was the Senior Medical Officer, Eric MacKay. Also there in 1961 was Rex Grey from the Royal Australian Navy, who was putting his efforts into formulating a reference base and library for the recently established School of Underwater Medicine.

I decided to see what had happened to all the 'bent' divers for whom I could find records. Although I could find only 137 case records, this was enough to show that the treatment tables had a dubious scientific background, and a rather uncertain practical effectiveness. My survey also indicated that prolonged oxygen administration at the shallower depths and a continuous leak away of pressure instead of stages could be of benefit. Unfortunately, I was unable myself to pursue these ideas because I had to return to NZ and other duties. Since then, of course, these principles have become standard practice as a result of others' research.

At the end of this time I was able to go to Norway on *HMS Reclaim*, where the helium tables designed by Val Hemplemen were to undergo trials in the sheltered deep waters of Sjorfiord, near Bergen. We had some serious cases of decompression sickness (DCS), which showed again that the diagnosis was often not as plain and well defined as the diving manuals would have one believe. It was also clear that severe fatigue and general malaise could be the precursors of more serious problems. This provides a considerable difficulty as it is obviously impossible to recompress every tired diver who feels a bit 'crook', and there is no way of measuring such symptoms.

Back in New Zealand, dive shops opened to cater for the increasing number of amateur sporting divers, and the New Zealand Underwater Association provided a national body for the increasing number of dive clubs. I was asked to become its medical adviser and we initiated a scheme of medical examinations for recreational diving candidates. I also became consultant to the Department of Labour and a similar programme was introduced for the working diver.

Our naval recompression chamber was the only treatment facility in the country, and its availability to the civilian population was fully accepted by the Navy. Fortunately, the actual number of cases requiring chamber treatments remained relatively few, though always very well publicised in the media. The civilian cases all presented after a considerable delay, because of diagnosis and evacuation difficulties, and very few were what could be classified as 'simple' limb-pain bends.

One weekend we had a call about a badly bent diver being brought in by boat from around Kawau. The chamber was prepared and the staff called in. I went to meet the boat. The diver had been dead since he had been retrieved from the water, drowned, but the skipper hadn't liked to say so over the radio! He had a dry suit on, with a small right-angled tear on the shoulder, doubtless torn on the edge of the crayfish hole he had not been able to get himself out of. I was always surprised at how little the divers who came to us seemed to know of the details of their diving schedules. Depths and times were vague; one even said to me "Oh you'll have to ask my buddy, he always checks that."

After giving a talk to one club on the hazards of diving, a member of the audience said to me "We often dive to 200 ft, but we never get nitrogen narcosis." With the cooperation of the naval diving team we arranged for them to have a dry recompression chamber (RCC) dive to 150 fsw. They all disregarded the tests we had given them, and relaxed into hilarity generated in part by the 'narks' and also the unexpected 'Donald Duck' voice quality at pressure. "Letsh have a shong," was the call as they decided to forgo the simple arithmetical task that I had requested. We managed to do this for quite a number of local clubs on Saturday mornings, always with the same result. The party was always brought to an abrupt halt by the cold fog in the chamber produced by the decompression.

I had completed my commitment to the Navy in 1964 and had entered general practice in Devonport so was well placed to continue as their consultant in underwater medicine. For this decade, our navy maintained its deep diving capability with annual training during the summer in Mercury Bay. The *Manawanui* was based in Whitianga for this period, and set out early every morning for calm and deep water. The operation entailed gradually working up to dives of 300 ft with traditional 'hard hat' and surface-supplied air apparatus. It was my very pleasant naval reserve duty to go with them as their medical officer. Considering that the diving was at the very margin of safety we were fortunate that only one diver suffered DCS. He was evacuated by helicopter to Devonport. The limb pain quickly responded to treatment in the RCC and he continued his career as a naval diver without further trouble.

The *Wahine*, a Cook Strait ferry, sank in the entrance to Wellington Harbour in a storm in April 1968 with the loss of many lives. This began a prolonged salvage and diving operation. Initially, this was mostly in shallow water, about 40 ft, and was DCS free. Then the wreck shifted into deeper water and broke up. The diving was then deeper and harder, to cut the wreck into moveable sections. The first 'bend' was the senior diver of the team in March 1970. He recognised his limb pains immediately and travelled up to us in Auckland by a normal commercial flight, which hurt him even more. He was quite aggressively blunt about not having any general medical examinations. Initial treatment with the new Goodman and Workman oxygen tables did not relieve his symptoms, but after a short while at 165 ft he was better. He also had a marked limp, apparently present for many years. An X-ray showed almost complete destruction of his left hip from osteonecrosis, and there was widespread involvement of other bones. I was surprised that it was not more painful. The following week another diver came to us with the same symptoms. He had dived 3 1/2 hours at 65 ft, without decompressing. Again the oxygen tables did not relieve his pain but higher pressure did.

I was asked to review safety procedures. It was apparent that monitoring of the dive schedules was difficult and unsatisfactory so, amongst other measures, I suggested the use of a 'decompression meter'. This simple tool was often misused and has been much maligned. From then on to the completion of the salvage, 10,800 hours of diving were

undertaken without further DCS. Later, the same procedures were used with success for the salvage of the *Seawise Princess* (ex Queen Elizabeth) in Hong Kong Harbour.

The annual calendar of the diving world in New Zealand included a 'convention' at Mayor Island on Labour weekend. In 1971 it proved to be a disaster. The first bend occurred on the Saturday afternoon. He was evacuated to Devonport and in spite of a neurological element in his decompression sickness he was successfully treated with a fairly short table. The chamber was again in use the next day for a diver who had been diving to 160 ft and was semi-conscious and virtually quadriplegic. His response to treatment, which included prolonged oxygen at 2 Ata (202 kPa) was disappointing. He was still in the chamber when a third diver arrived on Monday evening.

After a dive to 250 ft this diver had surfaced virtually out of air, and had called across the water for another bottle (his normal practice!), but been unlucky because his support vessel had started to sink. He said that he thought that he might have a touch of the bends, then lapsed into coma. However, when he got to us he was conscious again and complained only of being very tired and weak. We had managed to get our reserve chamber into action, though this did not have any oxygen capability, nor did it have a man lock. However, we had a Viet Nam veteran as an attendant for him and pushing the chamber beyond its proper limit we got him to 165 ft. All to no avail sadly – he became more sleepy, then unconscious and unrouseable, and died. Post-mortem examination failed to reveal any intravascular gas or provide us with an explanation; however, in retrospect there is little doubt that he was suffering from disseminated intravascular coagulation. At that time this was only just becoming recognised as a final pathway in many serious disease processes.

Until this time in New Zealand I think we had been fairly lucky with the number of cases of severe DCS that had occurred. The Poor Knights has accessible and relatively protected cliff faces that run down to a sandy bottom at around 150 ft. In contrast, the Bay of Plenty is potentially a more hazardous area as the popular sites are reefs that do not reach up to the surface. This requires an open water swim from reef top to surface and support vessel. Getting back to the surface to see only a distant mast top when one is at the peak of a swell certainly provokes anxiety. The timing of the convention at the beginning of the season when people tended to be out of practice was also ill-judged, and it has not been repeated.

A trust was set up in the Bay of Plenty to provide a chamber as a memorial to the diver who died. Eventually they managed to get one, but it was not possible to get it integrated into the hospital system. The trust underestimated the ancillary support that was necessary, and the chamber was never used. However, a similar fund-raising exercise in the South Island was more successful, culminating in 1979 with the commissioning in Christchurch of the first civilian hospital-based multi-place RCC in NZ. The voluntary staff of the unit were trained by the RNZN diving team and myself both at *HMNZS Philomel* and in the new Christchurch facilities.

The naval pattern of management, with a chamber at the diving site, was essentially to treat first and ask questions afterwards. Our chamber was at the dockside at *HMNZS Philomel*, Devonport, on the north shore of Auckland Harbour, at a distance from other medical facilities. This was less than ideal for the civilian sports divers who often had a long journey to get to us, and whose problem might not require recompression. For instance, it was not uncommon to have cases of water inhalation evacuated to us as the 'bends'. Wherever possible I tried to get a chest X-ray, electrocardiogram and blood screening done, speedily of course, before starting a recompression.

The chamber was fitted with a large oxygen cylinder to which we fitted a full face mask with a regulator on the side. This enabled the comfortable breathing of 100% oxygen, after the decompression schedule had got back to 2 Ata (202 kPa). We could also use the shorter 'minimal pressure oxygen recompression' Goodman and Workman tables. There were also hints from conferences and papers that other methods of management, including heparin, dextran, and steroids, could be of benefit. Unfortunately, by the nature of things these reports could incorporate only small numbers of patients and were therefore insufficiently conclusive to be integrated into our treatment protocols.

A fit young diver who, I believe, made a considerable sum illegally diving for crayfish was evacuated to us after a dive to 110 fsw for 25 minutes. Shortly after surfacing he felt that the right side of his face and left side of his body were paralysed. However, by the time that he arrived his symptoms had resolved and he was not recompressed. Three months later he came back after a dive of 80 ft for 20 minutes, with partial paralysis of the right arm and leg and other minor neurological signs. He was successfully recompressed this time using the oxygen table. I advised him that he was particularly 'bends prone' and that he should give up diving. But his plan was to dive again, far distant from fisheries inspectors…and medical aid. I offered him a trial simulated dive in the RCC, to which he agreed. We decided on 140 ft for 20 minutes with decompression according to the RN tables. The profile was chosen because it provided considerable decompression stress, but was one which he could have managed himself in open water.

I accompanied him as attendant and the dive proceeded uneventfully, but shortly after saying at the 20 ft stop "I don't think this will prove anything," he said "I feel crook, and I have a pain at the back of my neck." At the 10 ft stop he complained of double vision and started to mumble, soon becoming unconscious. He had a macular purple-red rash over his trunk and arms, his left hand was tightly clenched and pupils fixed, dilated and deviated to the right. Then his left arm and hand started jerking. He was immediately

FIGURE 1. Tony Slark in November 2000

recompressed to 60 ft on oxygen and within five minutes he was fully conscious, the rash had gone and he was without neurological signs. I felt rather better too! "What happened?" he asked. "Oh, nothing much," I replied, "I'll tell you all about it when we get you out of here." He took my advice and has never dived again.

From the early 1970s I was able as preceptor to offer final-year medical students an 'elective' in underwater medicine. This was a most rewarding experience. For three months during the summer I was able to take them diving frequently, and we had informal seminars every week. Their part of the bargain was to keep a journal and to write a referenced essay on one of the countless questions that had come up in our discussions. Some of these essays have been published in this journal. The programme was well received at the Medical School, and several of the students have remained in contact and have become my very good friends.

We had occasional requests from local hospitals to give various unfortunate patients who had gas gangrene treatments in our chamber. We treated four with success. An 84-year-old man was declined as he was moribund by the time he got to us. He just survived the return journey to the hospital by ambulance. A six-year-old girl whose knee wound had become infected with *Clostridium perfringens* got better after only two treatments. I have always been surprised that we had so few requests for our services.

At one time several patients suffering from multiple sclerosis (MS) sought chamber treatments. This was not easy as it was certainly on the fringe of orthodoxy and the rules for admission to the Naval Hospital did not extend to non-service personnel suffering from general medical conditions. However, we did manage to slip in a few. Only one benefited. Her circulation in both lower legs was compromised by long years in a wheel chair. Feet that were cold and blue became pink and warm, but it was regrettably a short-term benefit. Jersey, in the Channel Isles, had a recompression chamber donated specifically for the treatment of MS, which I was able to visit, but I have not seen an analysis of their results.

Developments in diving and hyperbaric medicine have been slow in New Zealand. After many years of struggle using enthusiastic volunteers, the Christchurch facility gradually developed. Their chamber has now been replaced by a state-of-the-art facility, which I was invited to formally open in November 2000 (Figure 1), and a full hyperbaric medicine service provided. The RNZN also now has an excellent unit adjacent to the Naval Hospital, which has provided a comprehensive service since December 1990. All naval medical officers can expect proper training in this and all aspects of marine medicine.

My one regret is that I could not spend more time with this fascinating subject. The only opportunity would have been to switch to the Royal Navy. Although this might have been possible it would have meant leaving New Zealand for good, and I was not prepared to do this. With the necessity to earn a normal medical living here, I could devote only a portion of my time to underwater medicine . However, for many years I was able to keep up my association with the RNPL with visits and at conferences. The subject provided excellent justification for interesting travel, and meeting interesting people. Now there are good textbooks on the subject, the Undersea and Hyperbaric Medicine Society has provided an academic foundation for keeping abreast of developments, and this work is augmented by the SPUMS, which, always great for comradeship and the annual scientific meetings, has this excellent journal. No longer does the flutter-clatter of a helicopter going past my veranda on a fine Sunday afternoon mean a night at the RCC.

Dr Anthony George Slark, MB, BS(Lond), DPH, DIH, DObst, FAPHM, MRCGP, MFOM, finished his naval career as Director General, Defence Medical Services, New Zealand. Tony is a Past President of the Society.
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The database of randomised controlled trials in hyperbaric medicine developed by Dr Michael Bennett and colleagues at the Prince of Wales Diving and Hyperbaric Medicine Unit is at:

<www.hboevidence.com>

SPUMS Annual Scientific Meeting 2002

Vaccines for travel ñ an update

Trish Batchelor

Key words
Travel medicine,vaccination, infectious diseases, review article

Abstract
(Batchelor T. Vaccines for travel – an update. *SPUMS J* 2003; 33: 149-156) Many vaccine-preventable diseases that have been eradicated from developed nations remain a significant health problem in the less developed countries of the world. Making reasoned vaccination recommendations to a traveller requires a sound knowledge of the epidemiology of the diseases in the destination country and should take into account individual factors such as length of trip, specific activities, underlying health conditions and style of trip. As a guideline, vaccines can be categorised as either 'routine', 'required' or 'recommended'. It is important that travellers and their advising doctors are not lulled into a false sense of security that all health problems related to travel will be prevented by undertaking a vaccination programme before travelling.

Introduction

Many vaccine-preventable diseases are but textbook entities for young doctors working in the developed world today. Unfortunately, this is not yet the case in many of the world's developing nations. When travellers from the developed world visit less developed countries they are potentially exposed to many pathogens to which they are immunologically naive. Vaccinations provide important protection against a number of serious illnesses still prevalent in many parts of the world.

Making decisions about vaccine recommendations for an individual traveller requires the consideration of a number of factors, including the individual's health status; season of travel; style of travel; duration of travel; activities; cost issues and the individual's attitude to risk. Cost-benefit analysis of travel vaccines on a community basis shows that the cost of preventing travel-related disease is high e.g., US$30,000 to prevent one case of hepatitis A.[1] In countries such as Australia and New Zealand it is up to the individual to decide if they are prepared to pay in order to prevent or limit their risk of contracting various diseases.

In view of the potentially high cost to the individual it is imperative that recommendations are based on a sound knowledge of the epidemiology of the disease in the destination country and a reasoned analysis of the risk and benefit for each individual. The rational prescription of vaccines will balance risk, benefit and cost to provide individual recommendations. There is a risk that undertaking a pre-travel vaccination programme will induce complacency and the traveller must be aware that vaccines will not protect them against the most common of travellers' illnesses – diarrhoea, respiratory infections (other than influenza and pneumococcal pneumonia), altitude-related problems, injuries and skin infections.

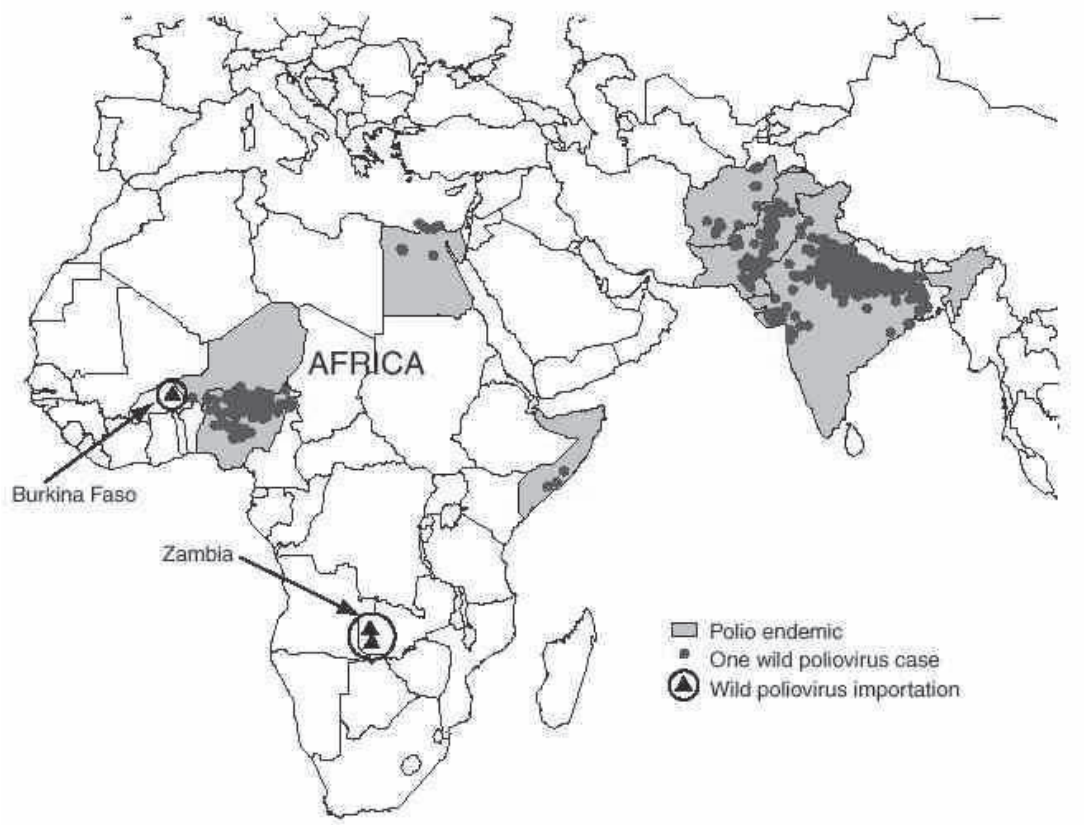
Routine vaccinations

Travel is an excellent opportunity to review the status of an individual's routine vaccinations. The anti-vaccination movement is relatively strong in Australia and New Zealand. Practitioners will not uncommonly be faced with parents planning a trip to a less developed country with completely unimmunised children. This requires great tact and excellent communication skills. With an explanation of the concept of herd immunity and the lack thereof in less developed countries, many parents will reconsider their stance and opt to have their children vaccinated against at least some vaccine-preventable diseases. The availability of inactivated polio vaccine and acellular pertussis vaccine has been helpful in allaying the fears of side effects related to the older versions of these two vaccines. In an ideal world no parent would take their unvaccinated child to a less developed country. After a non-judgemental and information-sharing consultation, it is hoped many parents will understand the different level of risk involved in travel as opposed to being safely at home.

POLIOMYELITIS

There are three different strains of the polio virus capable of causing disease. The virus, which principally affects motor and autonomic neurones, is spread via both the faecal-oral and respiratory routes. Infection can vary from inapparent to severe paralysis and death. In 1988 the Assembly of the World Health Organization (WHO) resolved to eradicate polio from the world by the year 2000. Whilst this goal has not yet been achieved, significant progress has been made and as of 2003 fewer than 10 countries are still reporting polio (Figure 1).[2] The National Health and Medical Research Council (NHMRC) of Australia continues to recommend a booster of oral polio vaccine (OPV) to all travellers. However, authorities such

FIGURE 1. LOCATION OF LABORATORY-CONFIRMED POLIOMYELITIS IN COUNTRIES WHERE POLIO IS ENDEMIC, 2002 (as of 9 April 2003; from ref 2)



as WHO and the Centers for Disease Control (CDC) recommend boosters only for those travellers who are visiting endemic countries and who have not previously received a single adult booster.

National guidelines differ regarding the use of OPV in preference to inactivated polio vaccine (IPV). There is a small risk of vaccine-associated paralysis after the administration of OPV. Countries such as the United States and New Zealand now routinely administer IPV for at least the first two of the childhood series of polio vaccinations in order to decrease the risk of this complication. The risk of paralysis reduces significantly with subsequent doses of OPV and in a previously vaccinated adult recipient is estimated to be between 1 in 2.5 million and 1 in 5 million.[3,4] Travellers should be warned of this small risk of paralysis and be offered the choice of OPV or IPV in countries where both options are available. If the traveller has no past history of vaccination they should receive a primary course of IPV.

TETANUS, DIPHTHERIA AND PERTUSSIS

This vaccine combining adult diphtheria and tetanus (ADT) should be administered if the traveller has not had a booster within the last 10 years. Current recommendations within Australia are for a course of five injections before the age of 19 and then a single booster at the age of 50. Travellers are, however, still recommended to have had a booster within the previous 10 years, primarily to avoid the necessity of making contact with the local healthcare system should they sustain a minor, but still potentially tetanus-prone, wound. *Clostridium tetani*, the causative organism of tetanus, remains ubiquitous throughout the world. Cases are still reported even in the industrialised world in unvaccinated children and older adults with waning immunity. A 1993 survey in Sydney showed only 52% of adults aged over 50 had adequate tetanus antibody titres.[5]

A new vaccine combining ADT and acellular pertussis (Boostrix) is now available in Australia and New Zealand. Pertussis remains a significant problem throughout the world as immunity acquired from childhood vaccination wanes in early adult life. Children under the age of six months who have not yet completed their primary vaccination schedule are at the highest risk of severe disease. There is evidence that adults carrying the disease can be important sources of transmission to this susceptible group.[6]

Unfortunately, the duration of immunity provided by the pertussis component of Boostrix is short (2–5 years) compared with the protection provided by the other components of the vaccine. It is therefore difficult to identify the ideal timing for administration of the vaccine. Theoretically, the ideal time to administer Boostrix would be during pregnancy, as this would help reduce the pool of adults who could potentially infect very young, unvaccinated children. However, there are inadequate safety data on the vaccine in pregnancy. Others have suggested that

administration in late adolescence would be most appropriate.[7] Currently, it is recommended for healthcare workers in close contact with young children and those considering childbearing who require an ADT booster. Medical aid workers should also be offered this vaccine.

MEASLES, MUMPS AND RUBELLA

Measles-mumps-rubella vaccination (MMR) is often overlooked in the pre-travel consultation. Of particular concern is measles, a paramyxovirus, easily spread via respiratory droplets. Complications of measles include acute encephalitis (2–10/10,000 cases) and delayed subacute sclerosing panencephalitis (1/25,000 cases). In the years between 1976 and 1995 measles caused more deaths in Australia than diphtheria, tetanus, pertussis and polio combined,[8] and the disease remains common in many developing countries. Outbreaks in Australia have been linked to travellers. A booster of MMR is recommended to all travellers born after 1966 who do not have evidence of having had two vaccines in the past.

VARICELLA

Varicella vaccine was licensed for general use in the US in 1995 and by 1996 the CDC had recommended that all healthy individuals over the age of one with no history of the disease should be immunised. Varicella vaccine is not funded in Australia and New Zealand despite its administration being recommended as 'best practice'. Pending new guidelines for varicella in Australia include a routine vaccination to children at the age of 18 months, with a catch-up dose between the ages of 10 and 13 for those previously unimmunised. Over the age of 14, two vaccines administered at least six weeks apart are required.

The uptake of varicella vaccine has not been adequate and this is most likely based on the perception that varicella is a mild disease. Whilst this is true of the majority of cases, complications such as pneumonia, encephalitis, cerebellar ataxia and necrotising fasciitis do occur. Congenital varicella can cause a severe syndrome resulting in skin, eye, limb and brain damage.[9] In Australia there are 240,000 cases of varicella resulting in 1,200 hospitalisations and an average of 4.2 deaths annually.[10]

A remembered history of chickenpox correlates highly with positive serology. Those who have no recall of the disease should be offered serology and, if negative, recommended vaccination. As a live vaccine, it is contra-indicated in pregnancy and the immunocompromised.

INFLUENZA

The pre-travel consultation is an excellent opportunity to ensure that high-risk individuals have received the current influenza vaccine. Influenza is a common disease in travellers and any traveller who wishes to reduce their risk of infection should receive the vaccine. The outbreak of severe acute respiratory syndrome (SARS) this year provides another good reason to vaccinate; as the symptoms of influenza are indistinguishable from SARS, reducing the chance of contracting influenza will also reduce the risk of being assessed as a potential SARS patient. Large outbreaks of influenza have occurred on cruise ships and at other large gatherings.[11] Cruise-ship passengers can also be responsible for introducing influenza into host countries; for example, Vanuatu suffered a large flu outbreak two years ago due to infected cruise-ship visitors.

Each year, in October, the WHO releases the composition of the flu vaccine for the approaching northern-hemisphere winter. In most years the southern-hemisphere vaccine has offered adequate protection; however, this should be confirmed annually.

PNEUMONIA

Pneumococcal vaccine (23-valent) is a routine vaccination for individuals over the age of 65, asplenic individuals and the immunosuppressed. It is expected that the updated version of the Australian Immunisation Guidelines will also recommend pneumococcal vaccine to smokers. Again, the pre-travel consultation is a good opportunity to ensure at-risk individuals are up to date. Pneumonia is a common cause of morbidity in elderly travellers. The new conjugated vaccine (Prevnar) may be appropriate for young children in expatriate families going to live in developing countries.

HEPATITIS B

Childhood immunisation against hepatitis B is slowly becoming routine throughout the world. In relation to travellers, the WHO states,

> *"the vaccine should be considered for virtually all travellers to highly endemic areas".*[12]

The hepatitis B virus is transmitted from person to person via the body fluids of an infected individual. Potential exposures for travellers include sexual contact; medical intervention using unsterile equipment (injections, dental work etc.); skin-perforating cosmetic procedures (tattooing, piercing, etc.); helping an injured person who is bleeding; accidents during sporting activities; and sharing personal items such as razors. A recent survey of 9,000 British travellers showed that approximately 10% of travellers had received high-risk exposures to the virus and approximately 70% had potential exposures whilst abroad. Only 16.9% of these travellers had been vaccinated against hepatitis B.[13] The incidence rate of hepatitis B in travellers has been estimated at 60/100,000 per month for symptomatic disease and 360/100,000 for asymptomatic disease in Asia. In Latin America and Africa the incidence has been estimated at 20/100,000 for symptomatic and 60/100,000 for asymptomatic disease.[14] In one region of the United Kingdom, travellers accounted for 12% of reported cases between 1990 and 1994.[15] It is estimated that there are 350 million carriers worldwide, with carriage rates as high as

15% in some Asian and central-African countries.

The incubation period of hepatitis B is 45 to 180 days and the period of infectivity extends from some weeks prior to the development of symptoms until the end of the acute illness. Acute illness is indistinguishable from other forms of hepatitis. After the acute infection between 1% and 12% of adults will become carriers or develop chronic infection. If the disease is acquired in the neonatal period this rate approaches 90%. Chronic active hepatitis develops in over 25% of carriers and some 15–25% of these individuals will develop hepatocellular carcinoma or cirrhosis.

The standard vaccination schedule for adults is three injections administered at 0, 1 and 6 months. If protection against hepatitis A is also required, the combined vaccine (Twinrix) can be utilised. A rapid schedule is also approved and this comprises three injections at Day 0, 7 and 21 with a booster at one year. This schedule is valuable if the traveller has less than one month until their departure, or they are likely to be lost to the six-month follow-up vaccination recall. There is good evidence that individuals who respond adequately to the primary vaccination series do not require booster doses.[16]

Current NHMRC guidelines for hepatitis B vaccination recommend boosters for those at high risk of exposure e.g., healthcare workers, or immunocompromised individuals. In these individuals, booster doses should be given when antibody titres drop below protective levels. Routine antibody testing after vaccination is not recommended by most authorities but it may be considered on an individual basis. Those who are at higher risk of not seroconverting (e.g., the immunosuppressed) or at high risk of exposure should be offered serology six to 12 weeks post-vaccination. Non-responders should be investigated for carriage status and if negative can receive three more vaccines at one- to two-month intervals. There is no value in administering more than six doses of the vaccine to non-responders.

Vaccines recommended to all travellers to areas with less than adequate food and water hygiene

HEPATITIS A

Hepatitis A virus is spread via contaminated food and water and remains highly endemic in many developing countries. In non-immune travellers the risk of contracting the disease varies from 3/1,000 per month in low-risk travellers (urban and resort-style travel) to 20/1,000 per month in those who are more 'off the beaten track'.[17]

The incubation period of hepatitis A is 15–50 days and infectivity is highest from the latter part of the incubation period until several days after the onset of jaundice. Symptoms are indistinguishable from other forms of hepatitis – fever, malaise, nausea, right upper quadrant pain and the onset of jaundice. There is no specific treatment available for hepatitis A, and fulminant hepatitis occurs in 3–4% of infected individuals over the age of 40. The disease is usually asymptomatic or very mild in young children, but increases in severity with increasing age.

Several inactivated vaccines are available on the world market, offering comparable levels of protection. If required, they can be interchanged for booster immunisations. The standard schedule is two injections at an interval of six to 12 months. Strong amnestic responses to delayed boosters have been proven. Therefore, there is no need to recommence the course of vaccination even if several years have elapsed since the initial vaccine was administered.

There are no clear guidelines on the timing of booster doses, with mathematical models predicting protective antibodies for over 25 years. Antibody testing post-vaccination is not recommended as seroconversion rates approach 100%, except in the immunocompromised. Some individuals, however, may benefit from pre-vaccination antibody testing in order to avoid the cost of this expensive vaccine. Such individuals include those who grew up in an endemic country, those with a history of childhood jaundice, and travellers over 60 years of age, as they are more likely to have been previously infected and will therefore have lifelong immunity. If time permits they should be offered antibody testing prior to vaccination.

TYPHOID

Typhoid fever, otherwise known as enteric fever, is transmitted by the bacterium *Salmonella typhi*. This food- and water-borne disease remains common throughout the developing world. The annual incidence is estimated at over 16 million cases, resulting in 600,000 deaths. Countries noted to be of particular risk to travellers include India, Nepal, Peru and Indonesia. Immigrants returning to their home countries to visit family and friends are at particular risk of typhoid as they often presume they have immunity and eat and drink without taking any special precautions.

After an incubation period of one to two weeks, typhoid presents as a slowly rising fever accompanied by headache, malaise and sometimes non-productive cough. Varying degrees of abdominal pain, diarrhoea or constipation may be present. As the disease progresses the patient looks increasingly toxic, and without treatment the case fatality rate approaches 10%. Of great concern is the increasing level of quinolone-resistant typhoid being reported, particularly in India and Nepal.[18]

All travellers to countries with poor food- and water-hygiene standards should be vaccinated. There are two vaccines currently available – the Vi polysaccharide injection and the Ty21a oral capsules. The injectable Vi vaccine is generally preferred as patient compliance with the capsules is notoriously poor. The injectable vaccine provides around 70% protection for two to three years. If travel to a highly endemic country is undertaken more than two years after vaccination a booster is recommended. Neither of these

vaccines protect against paratyphoid.

Compulsory vaccines

YELLOW FEVER

Yellow fever is a flavivirus transmitted by mosquitoes, with a distribution limited to the African continent and parts of South America. Outbreaks of yellow fever have increased in frequency and severity over the past 20 years and the WHO estimates that around 300,000 infections, resulting in 20,000 deaths, occur in endemic areas every year. Travellers are required to show proof of vaccination within the last 10 years when entering most countries within the endemic zone, and may be required to show proof when entering non-infected countries within six days of visiting a country in the endemic zone. The vaccination can only be administered at WHO-approved vaccination centres, and these centres should carefully examine an individual's itinerary to determine if the vaccine is required.

The yellow fever virus causes a biphasic illness. Initially, general symptoms of fever, myalgias, chills, anorexia and nausea dominate. About 15% of patients progress to a second phase after a few days, characterised by resurgence of fever, jaundice, abdominal pain and haemorrhagic manifestations. The mortality rate in this group of patients is around 50%.

In recent years, concern has been raised over the potential adverse effects of the yellow fever vaccine. The CDC published a report that highlighted an association between severe adverse events and age over 75.[19] Elderly travellers should be warned of the potential risk of the vaccine and it should only be administered if the risk of being exposed to the virus outweighs the risk associated with the vaccine. For most travellers, however, the risk of severe adverse events is extremely low, and it should be administered if travel is to be undertaken to an endemic area.

Yellow fever vaccine is a single injection and provides at least 10 years' protection. It is a live vaccine and is therefore contra-indicated in pregnant women (unless they are at significant risk), the immunosuppressed, those with egg allergies and children under the age of nine months.

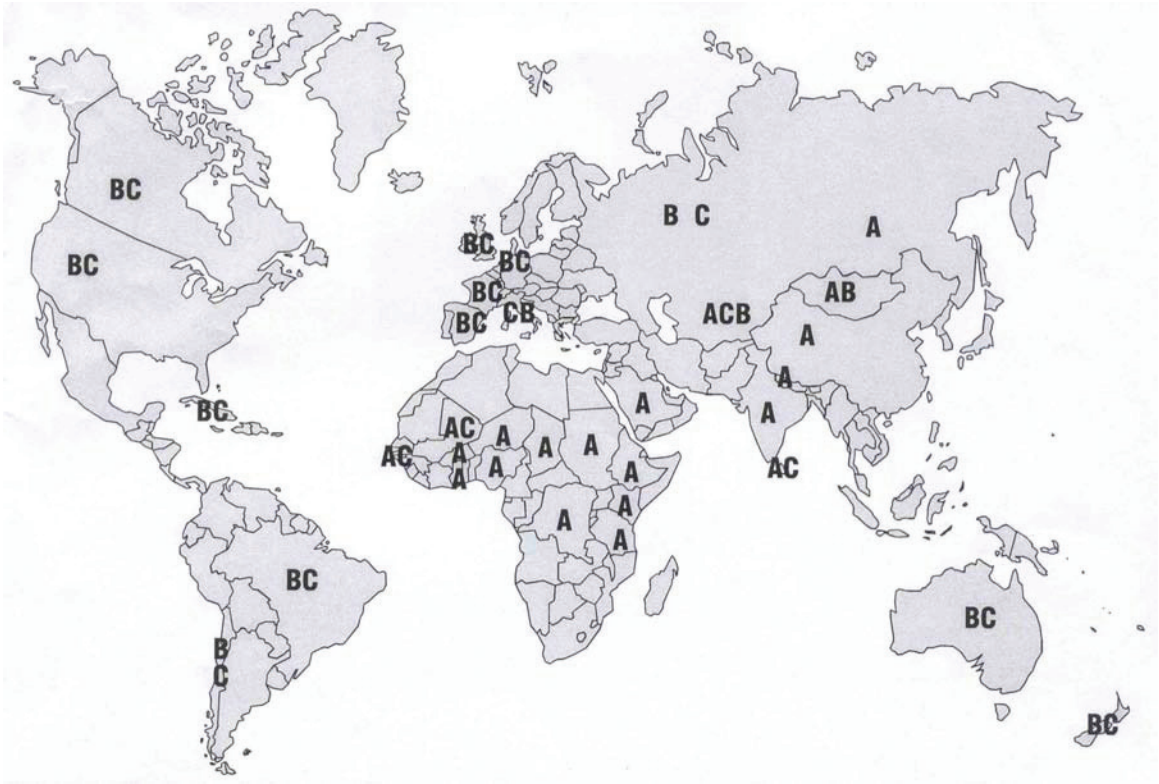
Vaccines recommended for long-term travellers or those undertaking specific activities

MENINGITIS

There are at least 13 serotypes of *Neisseria meningitidis*; however, more than 90% of clinical disease worldwide is caused by groups A, B and C (Figure 2).[20] Meningococcal disease is spread via aerosol drops or secretions. Invasive disease remains a medical emergency, with case fatality rates at around 10% even in developed countries.

A recent case-control study in the US showed a number of factors to be associated with an increased risk of invasive disease: active and passive smoking; recent respiratory illness; corticosteroid use; new residence; new school; and

FIGURE 2. Distribution of predominant *N. meningitidis* serogroups (A, B, C), 1996-1997[20]



household crowding.[21] Other studies have identified certain behaviours as risk factors during outbreaks: smoking; visits to crowded, poorly ventilated bars; and intimate kissing.[22]

It is difficult to give clear guidelines on who should be vaccinated against meningitis, as the disease rate in travellers is extremely low. When assessing an individual's risk, factors that should be taken into account include their destination, the nature and duration of their exposure, the age of the traveller and the state of their underlying health. However, all travellers to the 'meningitis belt' in sub-Saharan Africa should be vaccinated, as should visitors to any countries with increased disease activity.

According to the WHO,

> *"higher risk exists for long-term travellers and those who will be in close contact with the local population through accommodation, public transport and work".*[23]

Those at greatest risk are children under the age of five years, with a secondary peak occurring in the 15–24 age group. Asplenic travellers should also be vaccinated.

One should be aware of the worldwide distribution of serotypes if one is to prescribe rationally (Figure 2).[22] Serogroup A is most commonly implicated in endemic and epidemic disease in Africa; groups A and C predominate in Asia; and groups B and C predominate in most of the developed world. Thus, vaccinating a traveller to sub-Saharan Africa with group C vaccine is clearly inappropriate.

JAPANESE B ENCEPHALITIS

Japanese b encephalitis (JBE) is caused by a flavivirus transmitted by *Culex sp.* mosquitoes, which breed in rural areas, particularly rice paddies, and are outdoor, night-time feeders. The disease was previously only found in Asia; however, it has now spread as far west as Papua New Guinea, and as far south as Cape York. There are two broad epidemiological patterns. In the more temperate northern parts of Asia, such as Nepal, northern India and Japan, the disease occurs in epidemics during the warmer summer months, generally May to October. In more southern tropical areas, cases occur sporadically throughout the year with a peak early in the rainy season.

JBE is common in the local Asian population. Serological surveys have shown that in endemic areas the majority of the population have been infected by early adulthood. However, only between 1 in 250 and 1 in 1,000 of infected persons develop clinical disease. This still results in over 50,000 clinical cases reported annually.

JBE remains a rare disease in travellers. The CDC reviewed cases of JBE in travellers in 1993 and concluded that the overall risk to travellers was in the order of one per million per week. This estimate, however, included a denominator dominated by short-term travellers to low-risk areas. When trying to estimate a risk for travellers to high-risk areas during a high-risk season they provided a rough estimate of between 1 in 5,000 and 1 in 20,000 per week.[24] Whilst JBE is rare, it is a very serious disease, with a 30% mortality rate and around 50% of survivors suffering permanent neurological sequelae. There is no specific treatment.

Vaccination involves administering a series of three injections of the Biken vaccine at Day 0, 7 and 30. If there is limited time it can also be given as an accelerated schedule at Day 0, 7 and 14. Both regimens result in almost 100% seroconversion, but a lower antibody titre is reached with the rapid schedule. The optimal timing of booster doses has not been adequately studied and current recommendations are to boost after two to three years if exposure continues.

Concerns have been raised over the incidence of adverse events following administration of this vaccine. Millions of doses had been administered in Asia without apparent problems before it was used extensively in travellers. In the early 1990s a number of severe adverse reactions were reported from Denmark, Australia and Canada. These included angio-oedema and urticaria that often occurred some days after the administration of the vaccine. Since then, such reactions have occurred only sporadically, and three batches of vaccine were linked to the markedly increased rate of adverse events during that period.

Anaphylaxis has never been reported to occur after the administration of the Biken vaccine. Further, detailed surveillance since the early 1990s has shown that the rate of adverse events due to the vaccine is no different to the rates reported for many other vaccines.[25] In view of the small possibility of a delayed hypersensitivity reaction it is still advised that travellers have access to medical care for the ten days following vaccination.

Vaccination is recommended for the following groups:

- expatriates living in endemic countries;
- travellers spending time in rural areas during the transmission season, particularly in rice-growing areas;
- backpackers and cyclists who are doing long trips with uncertain itineraries;
- military personnel in endemic areas;
- long-term workers or visitors to rural areas visiting during the transmission season.

The recommendations of the WHO and CDC are rather open and suggest vaccination for those spending more than two weeks in rural areas during the transmission season.

RABIES

Rabies virus is serotype 1 of 7 serotypes of the genus *Lyssavirus*. It is transmitted by the bite, scratch or lick of an infected animal. The disease remains a major public health problem in much of the developing world, with over

50,000 deaths occurring annually, at least half of which occur in India. Millions of post-exposure treatment doses are given annually. The risk of a traveller developing rabies is low; however, the risk of being bitten by a potentially rabid animal is relatively high, 1 in 1,000 travellers per month in one epidemiological study.[26] In terms of vaccine-preventable diseases this risk was exceeded only by the risk of contracting hepatitis A. However, rabies pre-exposure vaccination is often neglected in the pre-travel consultation.

Rabies causes an invariably fatal encephalitis. The incubation period ranges from 20–90 days, although incubation periods of many years have been recorded. The initial symptoms of rabies are non-specific and last around 10 days before encephalitis intervenes. At this stage symptoms such as aerophobia, hydrophobia, disorientation, and hyperactivity develop and are accompanied by signs of autonomic instability such as hyperventilation, hypersalivation and hyperthermia. Gradual deterioration over a period of up to two weeks results in coma, or death from cardiac or respiratory failure.

Rabies is widespread, but is not found in the Pacific Islands, or some Caribbean islands. Up-to-date information on geographic distribution can be found on the WHO web site.

The major advantage of pre-exposure rabies vaccination is that it simplifies the post-exposure treatment regimen required should a traveller be exposed. If an unvaccinated traveller is bitten, full rabies post-exposure prophylaxis (PEP) is required. This consists of the administration of human rabies immunoglobulin (HRIG), ideally within 48 hours of the exposure, and a series of five vaccinations over the course of one month. HRIG is in short supply throughout the world and is unobtainable in most developing countries.

Even in a country like Thailand, which is considered a 'model developing country' in regards to rabies control, one survey showed that the supply of HRIG was a problem in over 50% of the country's hospitals.[27] If a traveller who has received pre-travel vaccination is bitten, they require only two injections of vaccine over the course of three days and no HRIG. Vaccine is widely available throughout most of the developing world. Thus post-exposure treatment is vastly simplified, and the potential stress involved in trying to obtain adequate PEP is greatly reduced.

Rabies pre-exposure vaccination involves a series of three injections ideally administered at Day 0, 7 and 28. Rapid schedules can be administered, but they may result in lower antibody levels. The standard dose is 1 ml intramuscular; however, the vaccine can also be given via the intradermal route. This offers a cost saving to the traveller but should only be done by an experienced practitioner, and with the proviso that antibody levels are checked 2–6 weeks post-vaccination, as seroconversion rates are not 100%.[28] For practical purposes intradermal vaccination should be limited to the travel-medicine-clinic setting and intramuscular vaccination is always preferred if the budget allows. A booster is recommended after one year and thence based on antibody levels.

Vaccination is recommended to all travellers spending more than three months in endemic areas and those who are more specifically at risk if doing shorter trips, e.g., children, cyclists, cavers, animal handlers and naturalists.

TUBERCULOSIS

It is estimated that one third of the world's population is infected with tuberculosis and as the human immunodeficiency virus (HIV) epidemic spreads so the tuberculosis situation worsens.

Tuberculosis is a subacute or chronic granulomatous disease caused by *Mycobacterium tuberculosis* that may affect any organ or system in the body. Depending on the infecting dose and an individual's natural resistance the untreated infection may heal completely or with varying degrees of scarring, undergo remission leaving a latent focus which may become reactivated or may progress to active disease.

The risk of tuberculosis infection in travellers has been studied only recently. The most relevant study was a multi-centre, prospective cohort study of 1,072 BCG-naive immunocompetent individuals visiting highly endemic countries (disease rate >100/100,000) for between three and 12 months.[29] The overall incidence rates were 3.5/1,000 person months for latent infections and 0.6/1,000 for active disease. However, healthcare workers had a significantly higher rate of latent infection at 7.9/1,000 per person month. The authors concluded that

> *"the risk of M. tuberculosis infection in long-term travellers to high-endemicity countries, even if not engaged in health care work, is substantial and of similar magnitude to the average risk for the local population. BCG vaccination or pre and post travel tuberculin skin testing of high risk travellers should be considered."*[29]

There are others who argue that in view of the problems associated with Mantoux testing – lack of specificity and sensitivity as well as compliance issues –it is not worth routinely testing travellers. Rather, they suggest awaiting the rare case of development of clinically overt disease and then treating appropriately.[30]

There are no clear guidelines as to the role of BCG, or pre- and post-travel skin testing in adult travellers. It is, however, generally agreed that children under the age of five spending more than three months in a highly endemic environment should receive the BCG vaccine. In adults the role of BCG remains unclear and most travel medicine practitioners will take either of the above-mentioned approaches, pre- and post-travel skin testing, or waiting for overt disease. An experienced travel-medicine practitioner should be consulted for advice regarding risk management of tuberculosis in the long-term traveller.

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Articles reprinted from other journals

A role for oxygen-induced osmosis in hyperbaric oxygen therapy

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Key words
Reprinted from, hyperbaric oxygen, gas-induced osmosis

Summary

(Hills BA. A role for oxygen-induced osmosis in hyperbaric oxygen therapy. *Medical Hypothesis* 1999; 52(3): 259-263) The principles of gas-induced osmosis, demonstrated in the 1970s, have been applied to the very large steady-state gradients of O_2 arising between arterial blood and hypoxic tissue during hyperbaric oxygen (HBO) therapy to produce a fluid 'pump' in the desired direction for resolving accompanying oedema. Thus, in soft-tissue injuries, an oxygen-induced fluid pump would break the vicious cycle between ischaemia, hypoxia and oedema at the point of *oedema* rather than *hypoxia*, as hitherto assumed. This osmotic mechanism enables the successes of HBO therapy in hypoxic disorders to be reconciled with early failures in such areas as hyperbaric radiotherapy, where substitution of O_2 for N_2 in inspired air was clearly not reflected at the tissue level. This argument also applies to the success of HBO in treating air embolism and decompression sickness over simple compression. The oxygen pump would seem to offer a more plausible explanation for the success of HBO therapy than theories based upon O_2 delivery by the circulation, especially when considering cardiovascular reflexes to elevated PaO_2 and the marginal increase in blood O_2 content upon switching to HBO from normobaric oxygen breathing.

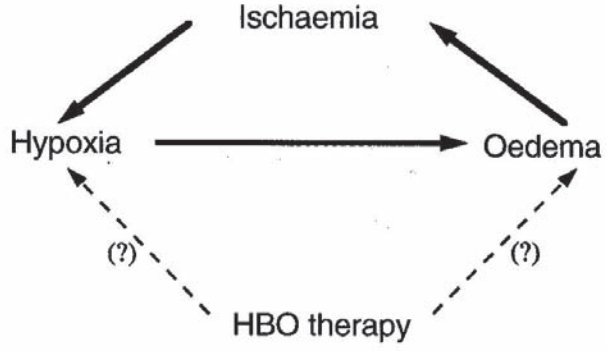
Introduction

In making a 'careful comeback',[1] hyperbaric oxygen (HBO) therapy has now become established as a recognized clinical modality with a proven efficacy for a limited number of disorders.[2] It can no longer be called the 'Maverick of Modern Medicine' as it was dubbed in the 1970s, following the disappointing results of hyperbaric radiotherapy[3] despite impressive in vitro scientific studies predicting success.[4] Failure to substantiate a number of extravagant claims made at the time in treating a host of other diseases exacerbated the scepticism. There were also compelling physiological arguments[5] explaining why large increases in arterial PO_2 do not necessarily elevate tissue PO_2 to any significant degree. In those days, the 'believers' emphasized arterial values of PO_2 while 'disbelievers', including this investigator,[5] regarded venous values as offering a better reflection of tissue oxygenation on the grounds that venous blood leaves in gas-equilibration with the tissue. So why does HBO work in some disorders and not in others?

There is no doubt that the use of hyperbaric oxygen is the treatment of choice in resolving air embolism or any disorder related to an unwanted gas phase in tissue.[2] Not only does the additional hydrostatic pressure reduce the volume of that gas, increasing the tension gradient of the inert gas controlling its rate of resolution, but the substitution of O_2 for N_2 in the breathing mixture is *not* reflected at the tissue level.[6] The resulting 'inherent unsaturation' of the tissue is transformed into a larger O_2 gradient,[7] later termed an 'oxygen window' for removing the inert gas and resolving the gas phase as a whole. The above examples of the application of HBO are consistent with each other in demonstrating the failure of HBO to affect the tissue level of O_2 significantly, so why should it be so successful in disorders related to hypoxia? Typical of these diseases is soft-tissue trauma, for which quite remarkable rates of resolving the resulting compartment syndrome have been claimed.[8–10] In these injuries; oedema forms rapidly with rapid onset of ischaemia as blood perfusion is curtailed by the compartment effect with extravascular tissue pressure rising, particularly in muscle encased in a tough fascial sheath of low compliance. The resulting vicious cycle of hypoxia, ischaemia and oedema is depicted in Figure 1.

It is generally assumed that the problem is resolved by HBO by oxygen simply diffusing into the tissue to break the vicious cycle at the point of hypoxia. However, if tissue oxygen tensions are increased as marginally as indicated

FIGURE 1
Shows the vicious cycle between hypoxia, ischaemia and oedema characteristic of soft-tissue injury



by the other disorders discussed above, viz. air embolism and hyperbaric radiotherapy, why should HBO do so in hypoxic injury, so what other mechanism could be acting to promote its effect?

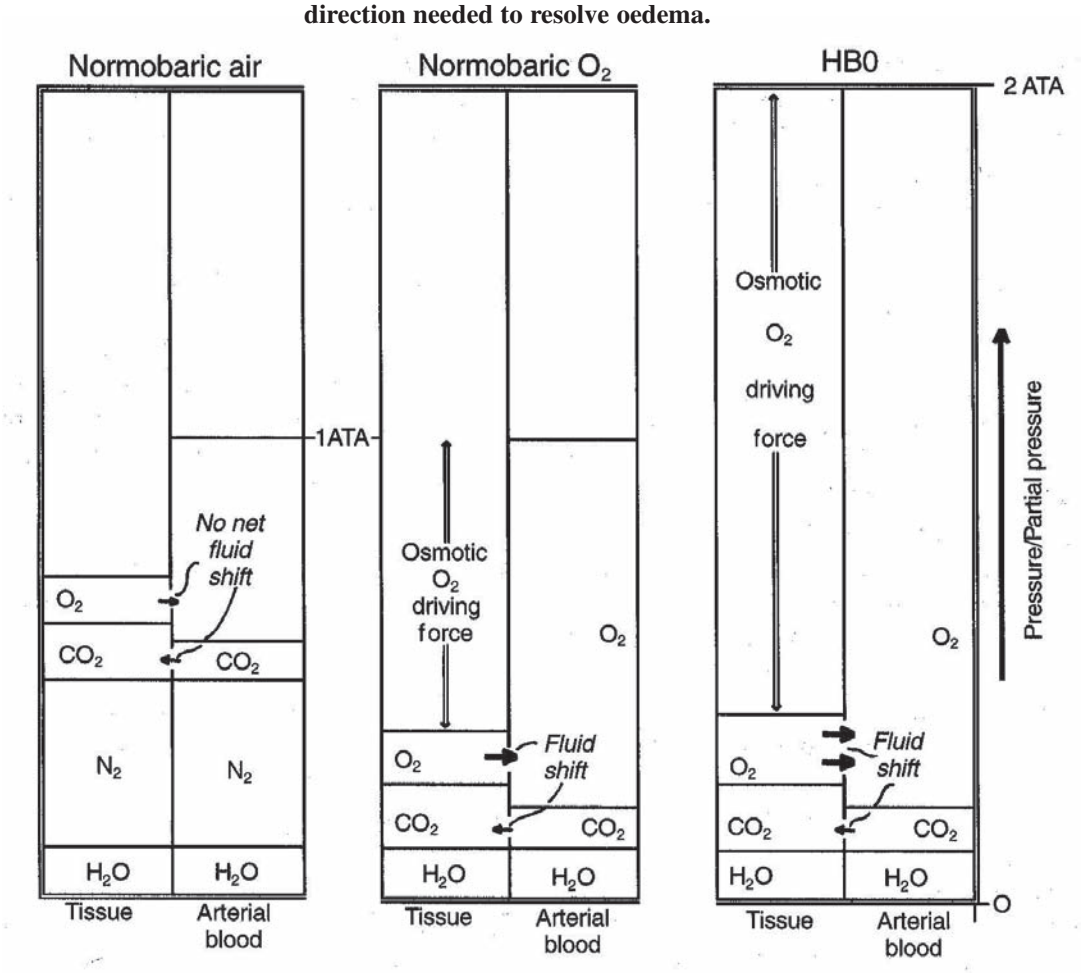
GAS-INDUCED OSMOSIS

In the early 1970s, there was considerable interest in a phenomenon termed 'gas-induced osmosis',[11] which was discovered by observing unexplained fluid shifts that occurred in deep-sea divers as they compressed, decompressed and switched inert gases. This mechanism[12] can also explain the haemoconcentration that occurs during decompression[13,14] and haemodilution upon voluntary hyperventilation.[15] The phenomenon was demonstrated for inert gases in vitro using both natural[11] and synthetic membranes,[16] by which gases in solution can exert an osmotic pressure like any other solute of comparable molecular size. This mechanism was tested in vivo using lungs separately ventilated at the same PIO_2 by means of a Carlan's tube so as to maintain *steady-state* gradients. The heavier gas steadily 'pulled' fluid away from the lighter inert gas.[17]

OXYGEN

A similar steady-state situation arises with the metabolic gases with a permanent gradient of CO_2 'pulling' fluid in the opposite direction to that of O_2. These forces are roughly balanced under normobaric air-breathing with a typical respiratory exchange ratio of 0.8 (see Fig. 2).[18] At least, when applied to the lungs for air breathing under normobaric conditions, there is a slight osmotic gradient pulling water *out* of the air spaces in helping to resolve any oedema. Upon elevating the PIO_2, however, the O_2 concentration in the fluid lining to the alveolus increases, increasing the oxygen gradient across the pulmonary membrane without a corresponding change in CO_2, nor one in the PO_2 of venous blood perfusing the lung. Thus, as PIO_2 is elevated, a point is reached at which the net osmotic 'pull' induced by the gas gradient changes direction and starts to 'pull' fluid *into* the air spaces. It may be fortuitous, but this switch-over level just happens to coincide with the PIO_2 for the onset of pulmonary oxygen toxicity,[18] which is characterized by increased perivascular filtration and lymph flow.

FIGURE 2. Illustrates the concentration differences of various gases present between arterial blood and tissue and how these can generate osmotic forces for shifting fluid in the directions indicated by the arrows. Note how the net force is negligible during air breathing but is very much greater with HBO and, moreover, acts in the direction needed to resolve oedema.



SOFT TISSUES

If oxygen tension gradients can shift fluid as indicated above, it raises the possibility that they can do the same in traumatized tissue. Arterial blood with a PaO_2 of 2 Atm (202 kPa) will now have an appreciable osmotic 'pull', especially in the arterioles before the oxygen diffuses into the tissue where its metabolic consumption provides the 'sink' for this gas. This large O_2 gradient will now 'pull' water out of the traumatized tissue, resolving the oedema and restoring blood perfusion. It can be argued that *any* increase in blood flow is desirable, whether the blood contains a little more oxygen or not.

QUANTITATIVE ASPECTS

The following equation has been derived[17] to describe the osmotic gradient ($\Delta\Pi$) in terms of the gradient in gas partial pressure (ΔP) as:

$$\Delta\Pi/\Delta P = \alpha\sigma\,(T/273)^1$$

where α is the Bunsen coefficient (solubility) of the gas, T is the absolute temperature and σ is the Staverman reflection coefficient describing the 'leakiness' of the membrane to the solute. For large molecules such as albumen, or high-molecular-weight dextran used as an infusion solution in trauma, $\sigma = 1$, i.e., no leaks, whereas gases are much less efficient with $\sigma \approx 0.05$.[11,12] For oxygen ($\alpha = 0.024$ml O_2.m1 H_2O^{-1}) at 37°C ($T = 310$°A) at 2 ATA ($\Delta P = 1520$ mmHg) to give $\Delta\Pi = 2.1$ mmHg. This may seem small but, relative to an oncotic pressure of 25 mmHg for plasma, this represents a fluid shift of 8.4%. Such a shift in resolving oedema could greatly reduce extravascular pressure, restoring blood perfusion, alleviating the hypoxia and so breaking the vicious cycle whereby hypoxia and oedema are mutually exacerbating. In trauma, the oncotic pressure is likely to be reduced as injured membranes become 'leaky' and so the fluid shift could be greater than the 8.4% estimated above. The driving forces are depicted in Figure 2.

OPPOSING FORCES

The osmotic pressure induced by an elevated oxygen gradient is quite unlike any other in that it is permanent because O_2 is being continually consumed metabolically in the traumatized tissue and, therefore, only oxygen can be used to induce this continuous fluid 'pump' resulting from that gradient. The fluid shift will not be opposed by ionic gradients represented by the osmolarity of the tissue because mobile ions and other small molecules will permeate membranes and eventually follow water, redistributing to contribute no osmotic gradient. The osmotic pressure derived from plasma colloids will be supporting the oxygen gradient, although it is essentially the reduction of this oncotic fluid 'pull' caused by membrane damage which was responsible for oedema forming in the first place. In some ways, the oxygen osmotic gradient can be regarded as an adjunct to the colloid osmotic pressure in resolving oedema until that pressure can be re-established and blood perfusion restored.

Hypothesis

The foregoing arguments lead to the hypothesis that a steady-state gradient of oxygen arising between its source in arterial blood and its site of metabolic consumption in tissue can 'pump' fluid up the concentration gradient just like any other solute of comparable molecular size. Under normal conditions this osmotic 'pull' is approximately balanced by that of CO_2 diffusing in the opposite direction. However the O_2 *gradient* pumping water can be greatly increased if the tissue is hypoxic and PaO_2 is elevated by the subject breathing oxygen at several atmospheres pressure. Thus, in HBO therapy, oxygen-induced osmosis could be a potent force in resolving the oedema which occurs in soft-tissue injury and break the vicious cycle between hypoxia, ischaemia and oedema at the point of *oedema* – see Figure 1.

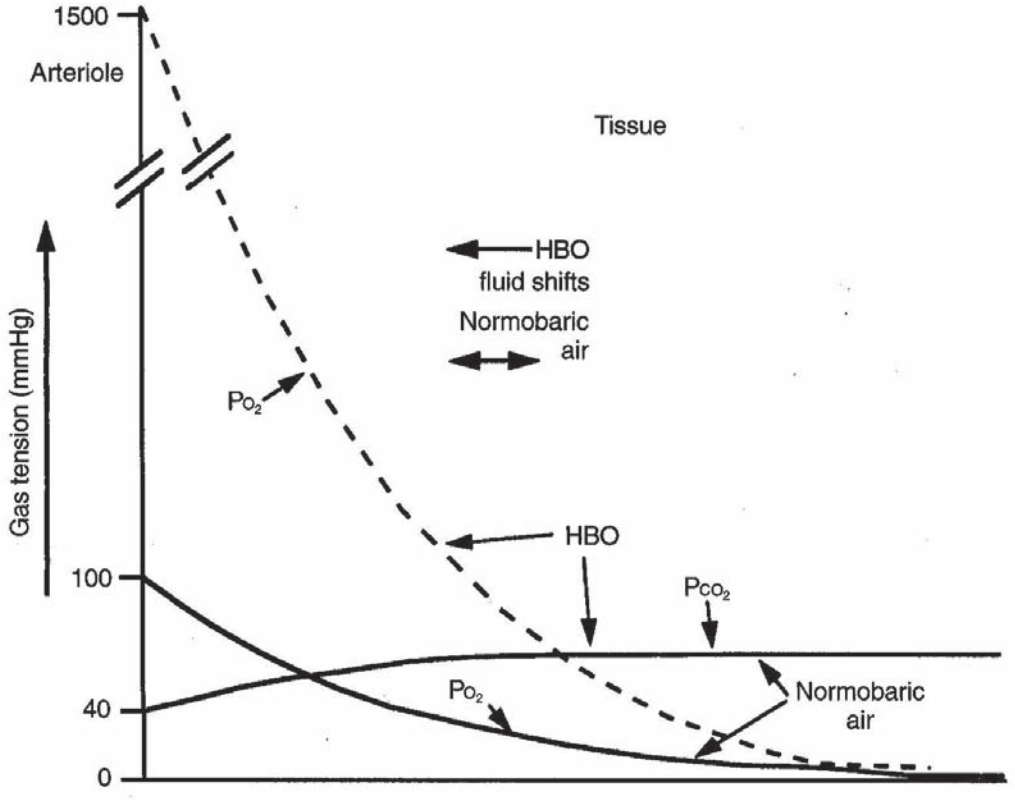
Discussion

The 'oxygen pump' offers an intriguing mechanism by which the O_2 tension gradients induced by HBO can shift fluid. Moreover, the shift is in the desired direction of resolving the oedema – see Figures 2 and 3 – thus posing the concept that, in soft-tissue injuries, the vicious cycle (Figure 1) is broken at the point of 'oedema' rather than 'hypoxia' as generally assumed. An increase of 10-fold in the P_IO_2 and, hence, in the 'fluid shift' in switching from air breathing to O_2 at 2 ATA (Figure 2) would seem more likely to benefit the tissue than a few percent increase in delivered oxygen before allowing for decreased cardiac output and reflex vasoconstriction.[19] Ironically perhaps, the low PO_2 resulting from the tissue hypoxia has a beneficial aspect by increasing the O_2 gradient, thus *promoting* the fluid shift – at least until the circulation is restored and hypoxia resolved by reducing ischaemia.

It could be argued that a membrane rendered 'leaky' by injury would also be 'leaky' to the oxygen gradient and would therefore compromise the 'oxygen pump' proposed above. There are two responses to this criticism. Firstly, injury is unlikely to be homogeneous such that adequate membrane integrity could be maintained to allow the 'oxygen pump' to function in some areas whereas colloid has leaked in others. Secondly, it is a moot point whether any membrane is actually needed in the case of a *steady-state* gas gradient, the injury producing a viscous fluid milieu across which gas gradients will be established as depicted in Figure 3. Osmosis is almost invariably studied by physical chemists in *equilibrium* situations[20] by balancing an osmotic pressure against a hydrostatic pressure. Osmosis is essentially a manifestation of the drive for solutions to become uniform in concentration. Just as diffusion represents the movement of *solute* molecules to reduce concentration gradients, so osmosis represents the movement of *solvent* molecules to achieve the same end thermodynamically. This movement would occur whether a membrane is present or not, and so it can be argued that a membrane is only needed when it is necessary to manifest

FIGURE 3

Depicts the large gradient in the concentration of oxygen in physical solution between an injured (hypoxic) tissue and blood in an arteriole or at the arterial end of a capillary during HBO therapy. This gradient will cause an osmotic shift of fluid towards blood, i.e. oxygen-induced osmosis. Note how the hypoxia provides a further 'sink' for O_2, thus enhancing this 'oxygen pump' shifting fluid in the desired direction for resolving the oedema.



an osmotic pressure as a hydrostatic pressure, as occurs in standard equilibrium studies of osmosis. The physical chemistry of osmosis under non-equilibrium conditions more relevant to physiological situations has been discussed in more detail by Hammel & Schölander.[20] If this thermodynamic argument is correct, it would mean that the osmotic gradient induced by HBO could be appreciably larger than the $\Delta\Pi$ value of 2.1 mmHg calculated above where the limitation was imposed by the value of $\sigma = 0.05$ for a membrane that is very leaky by virtue of the molecular size of the solute (O_2). Any opposing effect of nitrogen will be transient, lasting only until that gas is washed out; while any resulting drop in efficacy will be reversed, and the benefit recouped as the N_2 re-enters tissue upon return to air breathing.

The 'oxygen pump' mechanism expounded above does not exclude the conventional assumption that HBO simply delivers more O_2 to resolve the hypoxia. Both will act together to resolve the problem. However the 'oxygen pump' also offers an explanation for the failure of HBO in non-hypoxic situations, such as hyperbaric radiotherapy[3] and its effectiveness in resolving air embolism and decompression sickness[2] where a rise of PO_2 reciprocating the fall in PN_2 at the bubble site would defeat the object of the exercise. Hence the osmotic induction of fluid shifts by HBO could offer a simple physiological mechanism by which to reconcile the efficacy of HBO in hypoxic situations with earlier experience of this modality and, hopefully, one that will persuade other 'disbelievers' like myself to return to the fold.

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Tissue oxygen levels during hyperbaric oxygen breathing

Valerie Flook

Key words
Reprinted from, hyperbaric oxygen, oxygen

Abstract
(Flook V. Tissue oxygen levels during hyperbaric oxygen breathing. *European J Underwater Hyperbaric Med* 2001; 2(2): 41-48) The benefits of hyperbaric oxygen therapy are difficult to evaluate not least because of the difficulty of constructing a dose-response curve. Oxygen levels within the body are determined by so many different factors that, throughout the body as a whole, tissue oxygen partial pressures could range from close to arterial to almost zero. It is difficult to make measurements at the target site and published data show a very wide range of values. However, it is relatively simple to calculate the likely range of oxygen partial pressures in the capillaries and to calculate the effect of factors such as blood flow changes on these. This allows a qualitative estimate of the likely effects of such factors. This paper presents the results of calculations of capillary oxygen levels for three tissue types representing high, medium and low oxygen extraction tissues. Changes in blood flow, changes in shunt levels and in blood oxygen capacity have been considered. The results show that for high oxygen extraction tissues in particular the benefits of increased inspired oxygen may be surprisingly small. A tissue extracting as much oxygen as does the heart may have only a 2 kPa increase in end capillary oxygen partial pressure when inspired oxygen is changed from normal to 300 kPa. Reduced blood flow, increased venous admixture, reduced oxygen capacity all reduce capillary oxygen levels and reduce the benefit of hyperbaric oxygen.

Introduction

Hyperbaric oxygen therapy is used in an attempt to increase oxygen levels throughout the tissues in the hope that effective oxygen levels will be reached in the target tissues. In order to properly evaluate any therapeutic technique it is necessary to have a measure of the dose delivered in order to define a dose-response relationship, ideally at the target organ. This is very difficult to achieve for hyperbaric oxygen because of both local and general, temporal and spatial variations in supply and usage. Not only is oxygen freely distributed beyond the circulatory system but, being a metabolic gas, it is consumed to a variable extent in different parts of the body. Even with a constant overall oxygen consumption, the rate of extraction of oxygen from the bloodstream depends on the blood flow per unit volume of tissue, which is different for different tissues and may vary within a tissue. A change in oxygen level in arterial blood might itself be expected to change blood flow in some parts of the body. A further complication arises from the fact that oxygen partial pressures within tissue depend, not only on oxygen requirement and blood flow, but also on the distance of the particular bit of tissue from the nearest capillary.

Increased oxygen levels within the capillaries result not only in a higher partial pressure of oxygen in the tissue close by the capillary, but also in an increase in the distance that oxygen can diffuse to reach cells remote from the capillary. This is well described in Wattel et al.[1] Movement of gas requires a driving force, a partial pressure gradient, and this means not only to the cell but from the cell wall to the metabolising mitochondria. There is a range of oxygen levels along a capillary and ideally the blood should reach the entrance to the venule (end-capillary) with enough oxygen to allow diffusion to the furthest cells served by that capillary and on into those cells. It is difficult to make realistic calculations of oxygen levels beyond the capillaries because cells are situated at different distances from the capillary; some cells may use oxygen from several nearby capillaries; the relative contribution of each capillary may change as, for example, the pattern of patent capillaries changes. These concepts are discussed in Lambertsen and can be summarised in a quotation from that work.[2]

> *"Because each organ, its many different tissue components and even minute units of* a *single tissue have different relations of blood flow and metabolism, oxygen pressure gradients vary considerably from one discrete locus to another."*

The purpose of the present paper is not to achieve the impossible and make quantitative predictions for tissue oxygen levels but rather to use a quantitative approach to give an appreciation, in qualitative terms, of the wide discrepancy between the level of oxygen inspired into the lungs and levels which can be achieved in tissues. In some circumstances an increase in inspired oxygen of 101 kPa may give an increase in the target tissue oxygen of only 1 or 2 kPa.

It is widely accepted that for any gas the partial pressure of the gas in the venous blood draining a tissue is equal to that of the average tissue partial pressure and there is no obvious reason to abandon that hypothesis. This gives the possibility of measuring PO_2 in the venous blood draining the tissue as a means of determining dose. However, for

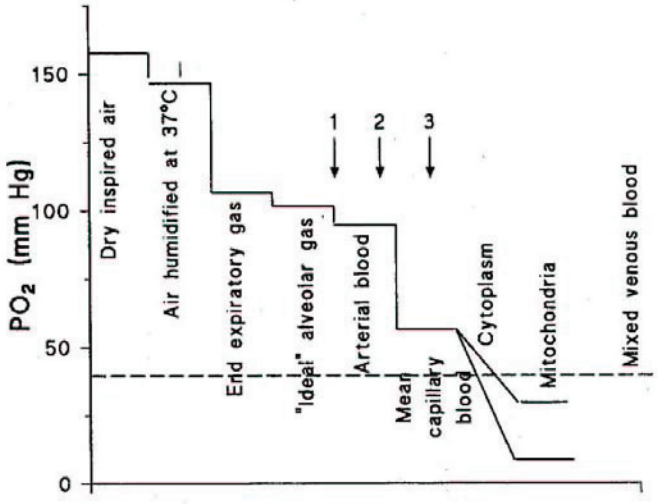
some of the applications of hyperbaric oxygen, e.g., wound healing, it would be more useful to have some idea of the local oxygen levels. There are several reports in the literature describing transcutaneous oxygen pressure measurements close to wounds.[3-5] Some take the pragmatic approach of trying to determine a threshold level of periwound $P_{tc}O_2$ during hyperbaric oxygen therapy, which can be used prognostically to indicate whether or not to continue with treatments.[1] The fact that so many things influence oxygen levels means that even carefully controlled measurements under ideal conditions in normal subjects result in a wide range of values.[6] This being so there is some value in making an attempt to calculate likely tissue levels.

A theoretical approach always includes assumptions, some are introduced to simplify a very complex situation, some to cover lack of detailed knowledge. Assumptions always weaken the value of the calculated results but the consequences of each assumption, and of variation in the magnitude of parameter values, can be evaluated. Evaluating the consequences of assumptions in itself always adds to the understanding of the relative significance of the various factors which play a role. This paper presents the results of such a theoretical determination of tissue oxygen levels. It effectively follows on from, and extends the work of Lambertsen.[2] To some extent the actual numbers resulting from the calculations are not the main objective. We should expect the useful conclusions to be qualitative rather than strictly quantitative.

As an introduction to the overall picture, Figure 1 shows the range of oxygen partial pressures, from the gas inspired into the lungs down through the 'oxygen cascade' to the cells, cytoplasm and mitochondria. Capillary oxygen partial pressure is shown as a single mean value that is somewhere between arteriolar and venular levels. Beyond that, in the cells and mitochondria, a range of values is shown to indicate the kind of variation that exists within a tissue. Mixed venous blood is shown as a dashed line to indicate that the partial pressure of oxygen is variable and will depend on levels within the tissues.

The change in oxygen partial pressure from arteriole to venule is the main subject of this paper. The three arrows marked on Figure 1 identify points down the oxygen cascade at which the delivery of oxygen to the tissues can be influenced. Arrow #1 marks the point at which levels of venous admixture, or shunt, affects delivery by increasing alveolar to arterial oxygen partial pressure difference. Arrow #2 marks the site of influence of changes in tissue blood flow which influences the arterial to venous oxygen difference. Arrow #3 marks the position at which the ability to extract oxygen from the blood becomes important and for present purposes this relates to the oxygen carrying capacity of the haemoglobin. These represent three possible causes of hypoxia during normal air breathing at 101 kPa. The traditional presentation of the oxygen cascade usually shows five possible causes of hypoxia. The two extra to the three considered in this paper are a reduction in alveolar ventilation which causes a reduction in arterial oxygen and at the level of the cell, a toxic inability to utilise the oxygen, histotoxic hypoxia. These are not dealt with here.

FIGURE 1
The oxygen cascade from inspired gas to mitochondria



The calculations

The basic calculations have been made using the 'normal' i.e., textbook values for oxygen extraction per unit volume of blood and assuming that this is extracted at a constant rate along the capillary. Thus 25% of the way along the capillary 25% of the oxygen which will be taken from that blood has been removed. Calculations have been made at ten points along the capillary.

Three representative tissues have been studied, skin, muscle and heart. These have been chosen to give a range of values for oxygen extraction which spans most of the body tissues for most conditions. Values for blood flow, oxygen consumption and oxygen extraction are presented in Table 1. Skin is a low oxygen extraction tissue. Heart, which is a continuously working tissue, is a high extraction tissue.

TABLE 1
Values for blood flow, oxygen consumption and exraction for skin, muscle and heart tissue

Tissue type	Skin	Muscle	Heart
Blood flow $ml.100gm^{-1}.min^{-1}$	13	6	84
Oxygen Consumption $ml.100gm^{-1}.min^{-1}$	0.33	3.3	9.7
Oxygen Extraction $ml.l^{-1}$	25	55	115.5

Obviously real oxygen extraction varies. For example, working muscle may have a higher oxygen extraction than resting muscle depending on the extent to which the increase in blood flow matches the increase in metabolic activity, whilst skin, when responding to a thermal stress, may change the percentage of blood flow which is shunt and therefore change oxygen extraction. The brain has a very labile blood flow with little change in oxygen consumption. However, the range of oxygen extraction values likely in brain tissue is within the range of the tissues studied here and has been dealt with in Lambertsen.[2] The viscera and the carotid bodies have a lower oxygen extraction than skin and therefore will, in general, have higher oxygen levels than those presented here.

The oxyhaemoglobin dissociation curve which has been used for the calculations is that for human blood presented by Gomez which derives from the experimental data of Dill.[7,8] To the volume of oxygen carried bound to haemoglobin has been added dissolved oxygen at a rate of 24 $ml.l^{-1}$ for each 101 kPa pressure. No attempt has been made to adjust for the change in carbon dioxide partial pressure and in pH as the blood changes from arterial to venous during movement down the capillary.

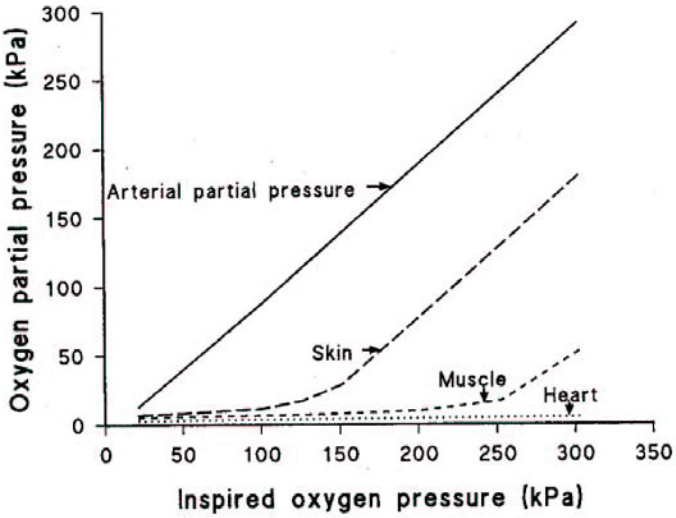
The calculations have been made for inspired oxygen partial pressures 21.2, 50.5, 101, 151.5, 202, 252.5 and 303 kPa, i.e., from normobaric air up to 3 atmospheres pure oxygen. Arterial oxygen pressures have been calculated on the basis that oxygen, nitrogen, carbon dioxide and water vapour make up the total of dissolved gases and that mixed arterial oxygen pressures equate with alveolar. Thus the effect of normal physiological shunt has been ignored largely because increased oxygen itself gives rise to a very variable increase in shunt as will be discussed later. The effect of the normal level of physiological shunt can be derived from the results of the effect of increased levels of shunt.

Calculations have been made for the changes identified by the arrows on Figure 1:

- increased venous admixture or shunt blood which causes an increased alveolar to arterial oxygen difference, usually with a decrease in arterial oxygen levels
- reduced tissue blood flow which causes a greater arterial to venous oxygen difference
- a reduction in the level of functioning haemoglobin which reduces the volume of oxygen carried to the tissue

The effect of venous admixture has been calculated by replacing normal arterial oxygen concentrations with those from the average arterial oxygen partial pressures given in Clark and Lambertsen.[8] Arterial oxygen partial pressure for inspired oxygen 303 kPa was interpolated from the values for 202 and 354 kPa given in that paper. The oxygen extraction values given in Table 1 were then subtracted from these new values for arterial oxygen content in the same way as for normal conditions, extraction was assumed to be linear along the length of the capillary.

FIGURE 2
The relationship between inspired oxygen partial pressure and end capillary oxygen pressure



The effect of a 20% reduction in blood flow was calculated by using appropriately adjusted values for oxygen extraction to give an oxygen extraction, $ml.l^{-1}$ of blood, equal to 20% above normal for each tissue and working from the normal arterial oxygen content.

The effect of a 50% reduction in haemoglobin was accounted for by halving the volume of oxygen carried by haemoglobin and subtracting the normal oxygen extraction from the new total oxygen concentration.

Results

Figure 2 shows the relationship between inspired oxygen partial pressure and end-capillary oxygen pressure for the three tissue types. Arterial oxygen partial pressure is also shown for comparison. The higher the oxygen extraction the greater the difference between arterial and tissue oxygen pressures. The shapes of the curves representing end-capillary oxygen pressure in the tissues reflect the shape of the oxy-haemoglobin dissociation curve. At higher inspired oxygen levels tissues such as skin draw all the metabolic oxygen from dissolved oxygen without unloading any of the haemoglobin. Under these conditions the relationship between inspired and end-capillary oxygen is linear and parallel to the arterial oxygen partial pressure. The tissue has full benefit from the increased oxygen; an increase in arterial oxygen gives the same increase in tissue oxygen. At lower inspired oxygen levels metabolic oxygen is drawn partly from dissolved gas and partly from dissociated gas. The curves approximate to linear again when, at the end of the capillary, metabolic requirements are entirely met by dissociating oxygen from haemoglobin. From this figure it is obvious that, for a high extraction tissue such as the heart, at the end of the capillary dissociated oxygen is being used no matter how high the inspired oxygen levels, the benefit from increased inspired oxygen is small.

FIGURE 3
Capillary oxygen partial pressures for three tissues

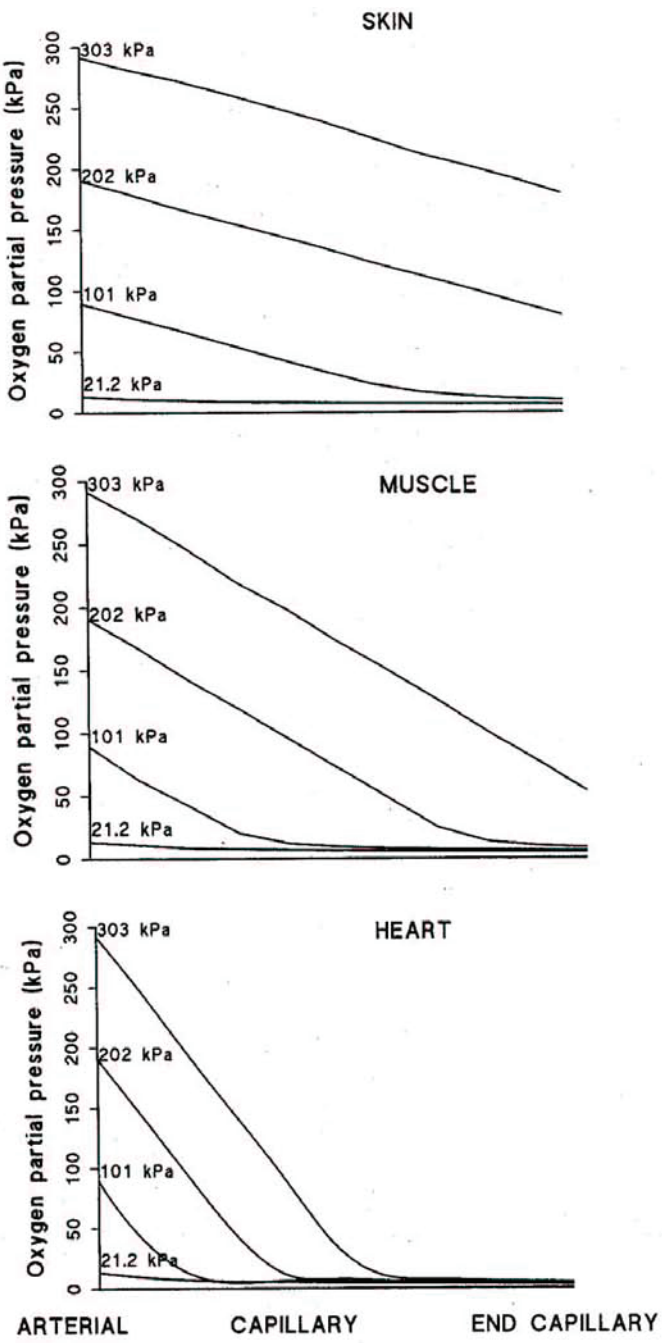


FIGURE 4
Capillary oxygen when venous admixture is present

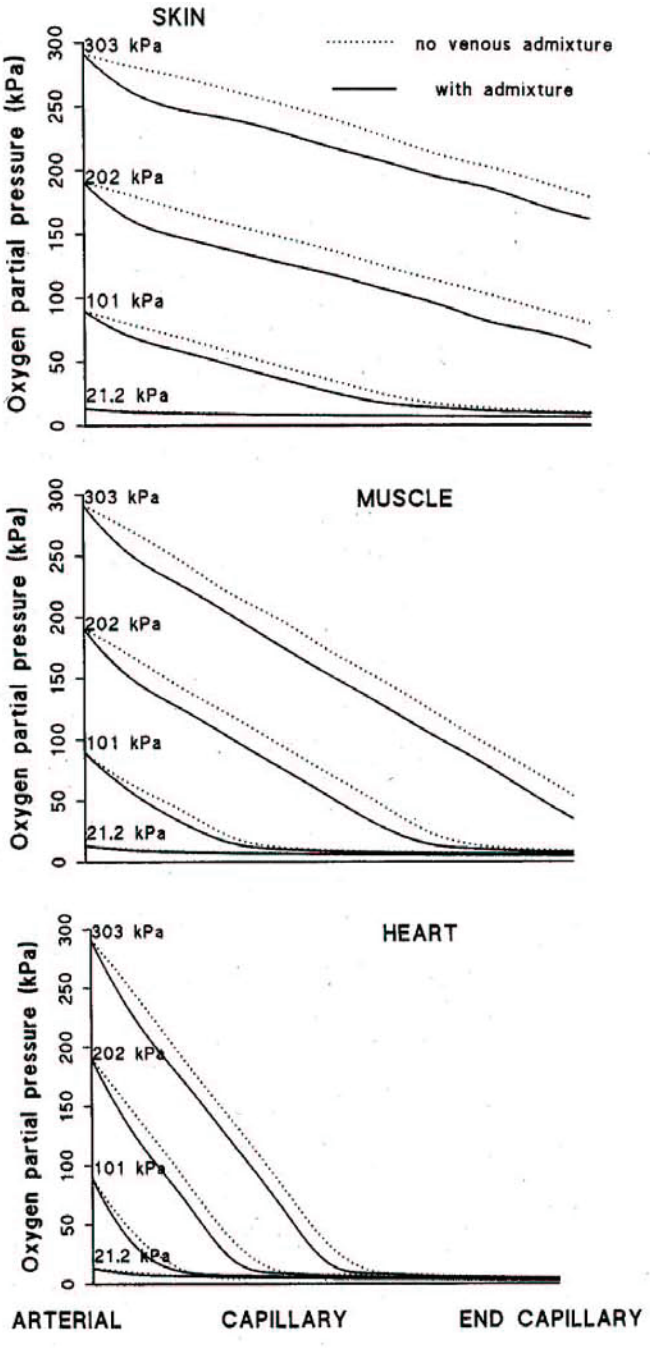


Figure 3 shows the oxygen partial pressure at each point along the capillary, from the arterial to the venous end, for all four levels of inspired oxygen. The linear sections of the lines are where the metabolic oxygen is drawn from dissolved gas, the horizontal sections of the lines are where dissociated oxygen supplies all the metabolic oxygen.

The vertical distance between the lines is a measure of the benefit derived from increasing inspired oxygen. Thus at the arterial end of the capillary, all tissues derive full benefit from increased inspired oxygen. At the venous end high extraction tissues such as the heart derive very little benefit. The higher the oxygen extraction the greater the proportion of tissue which derives less benefit.

Although it is not the purpose of this work to give the impression that this approach can give realistic numbers for tissue oxygen pressures, some of the number can be startling. For example breathing 303 kPa oxygen increases end capillary oxygen pressures in the heart only from 2.85 kPa (for air breathing) to 4.9 kPa.

Figures 4 to 6 show the same information as Figure 3 but for altered conditions. The dotted lines indicate the lines

FIGURE 5
Capillary oxygen partial pressure when blood flow is reduced

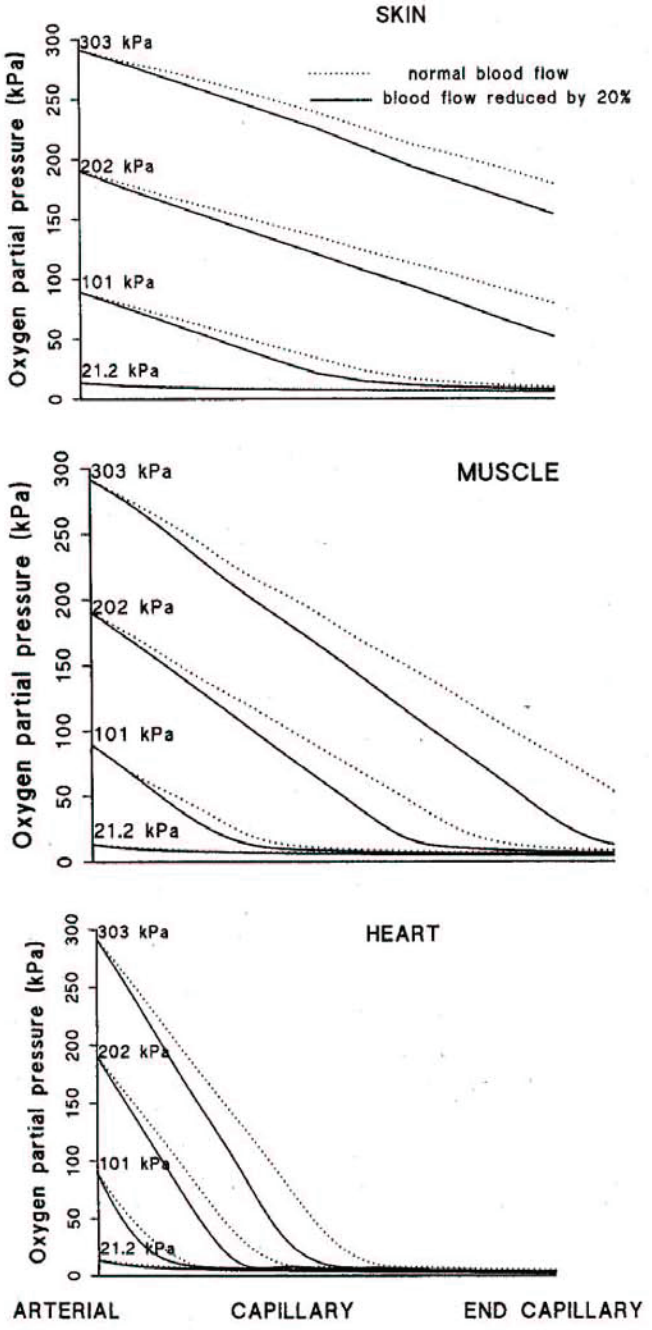
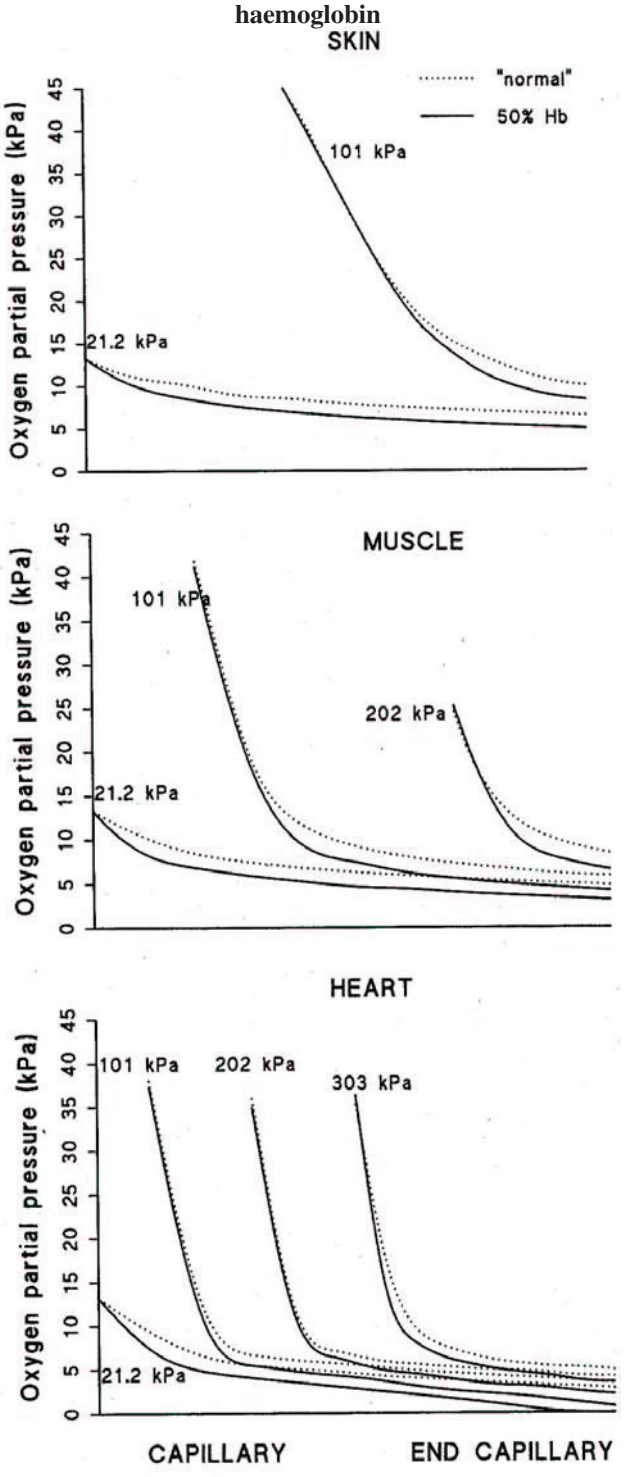


FIGURE 6
Capillary oxygen partial pressure with reduced haemoglobin



from Figure 3, i.e., normal conditions. Figure 4 shows the effect of venous admixture, Figure 5 the effect of reduced tissue blood flow and Figure 6 the effect of reduced haemoglobin levels. Figure 6 presents, on an expanded scale, the venous end of the curves because, of course, a reduction in haemoglobin has an effect only where the metabolic oxygen is drawn from haemoglobin, i.e., at the lower oxygen levels, the end of the capillary.

Discussion

It is important, when presenting theoretical simulations of any physiological function to stress the limitations of this approach. When simulating events in something as complex as the body so much of the information necessary to make a realistic simulation is missing. It is not possible to simulate reality. Despite the fact that simulations produce quantitative results the most important use one can make of the results is essentially qualitative; to use the results to aid understanding of the underlying mechanisms and their inter-relationships.

One of the major assumptions in these calculations was that the oxygen extraction from the blood was linear along the length of the capillary; 25% of the oxygen to be removed was removed at a distance 25% along the capillary. This is in fact somewhat unlikely given that the largest partial pressure gradients available to move the oxygen exist at the arterial end of the capillary. The effect of a proportionally greater extraction at the entrance to the capillary would be to lower end-capillary oxygen levels. In fact the proportion of the capillary which might be considered to be at 'low' oxygen would be greater and the end capillary oxygen pressures would be lower than calculated.

The results presented here show clearly that the greater the oxygen extraction in a tissue the greater the proportion of that tissue existing at 'low' oxygen tensions, drawing metabolic oxygen from haemoglobin. The benefit derived from increased inspired oxygen may be very small for tissues with high oxygen extraction. The figures presented in this paper relate to conditions close to the capillary but in all tissues there will be more remote cells for which the true oxygen partial pressure lies at some point below the lines shown on the figures. It must also be remembered that there is no sense in which cells with a high partial pressure of oxygen can compensate for those which have a low partial pressure.

The level of venous admixture used in the calculations was taken from Clark and Lambertsen who measured alveolar and arterial oxygen partial pressure in 6 normal subjects breathing air at 101 kPa or oxygen at pressures up to 353.5 kPa.[9] By taking the average arterial oxygen values from this paper we have considered levels of shunt which can arise, which probably will arise, in any patient during hyperbaric oxygen therapy. The effect of the shunt is to reduce the end-capillary oxygen partial pressure by something of the order of 0.2 kPa. Much more important is the fact that the shunt has resulted in a greater proportion of the tissue with 'low' oxygen levels and this effect will be compounded for cells remote from the capillary.

The reduction in blood flow was taken as 20% with no distinction made as to whether this is a local flow reduction or the result of a reduced cardiac output. As described in Lambertsen breathing oxygen above 101 kPa causes a reduction in cardiac output of about 15% with 303 kPa oxygen.[2] More recent work by Berry et al, using conscious dogs, reports a 20% reduction in cardiac output with 202 kPa oxygen.[10] These authors also present evidence for a reduction in blood flow to muscle and skin which may be a mechanism for maintaining blood flow to the organs, including coronary blood flow, despite the reduction in cardiac output.

Local blood flow changes are likely to be a reduction in flow arising from the high oxygen in the arterioles and pre-capillary sphincters where blood flow through the capillary beds is regulated. This shows up as an increase in systemic vascular resistance, 30% in 202 kPa oxygen as reported by Berry et al.[10] Vasoconstriction in response to increased levels of oxygen can be seen as an immediate first line of defence against the toxic effects of oxygen. Low oxygen levels close to the venous end of the capillaries may have little influence on blood flow.

Little is known about actual changes in peripheral blood flow, but a 20% reduction in flow resulting from a combination of reduced cardiac output and vasoconstriction seems not impossibly large for a normal reaction in a normal person. An ill patient with already reduced cardiac output might be expected to have even more tissue at 'low' oxygen levels than those shown in Figure 4.

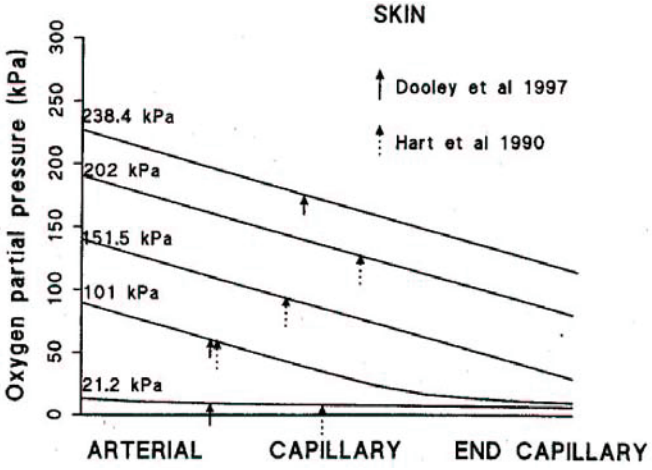
A reduction in effective haemoglobin can relate either to the anaemic patient or to a patient with carbon monoxide poisoning. The effect, on cell oxygen levels, of a reduced amount of haemoglobin is manifest only towards the venous end of the capillary where metabolic oxygen is drawn from haemoglobin. Because there is no effect at the arterial end, the proportion of tissue which has 'low' oxygen levels is not affected by the amount of haemoglobin available for oxygen transport. However, the magnitude of the effect can be quite large. For the conditions presented here the reduction in end-capillary oxygen partial pressure is almost 3 kPa for the heart when there is a 50% loss of haemoglobin. Reversal of carbon monoxide poisoning is now known to involve more than simple removal of the carbon monoxide from the body and it is interesting that there is apparently no convincing evidence that inspired oxygen at 303 kPa is any better than 101 kPa.[11] The effect of increased oxygen on brain tissue is complex and the reader is referred to Lambertsen for a more detailed description.[2]

At high oxygen levels, where only dissolved oxygen is used for metabolism, the fully saturated haemoglobin interferes with the carriage of carbon dioxide as carbamino compounds, the Haldane effect, and where cell oxygen levels are high so too will be carbon dioxide levels. Whether this results in increased or decreased brain blood flow depends on the overlying effect of hyperoxygenation on alveolar ventilation. According to Lambertsen, Lambertsen et al, and Kety and Schmidt in normal man hyperoxygenation of the brain leads to moderate contraction of brain vessels.[2,12,13] According to the work of Berry et al carotid artery blood flow decreased by 18% with 202 kPa oxygen.[10]

Moderate vasoconstriction of the brain vessels will result in a higher oxygen extraction. If we assume that the brain has an oxygen extraction similar to the heart, calculations would suggest that the difference breathing 101 kPa and 303 kPa, at the venous end of the capillary, may be as little as 2.8 kPa.

Although it is stressed that mathematical models cannot reproduce reality, it is perhaps prudent to compare predictions made here with such measurements as have

FIGURE 7
Comparison of predictions with measured transcutaneous oxygen partial pressures



been made. So far most of the measurements have been of transcutaneous oxygen partial pressure using a heated Clark polarographic electrode, heated in an attempt to increase local blood flow sufficiently to allow the electrode to 'see' arterialized capillary blood. There are two studies of transcutaneous oxygen partial pressure in normal subjects, Hart et al and Dooley et al.[5,6] Figure 7 shows arrows marking the average values for transcutaneous oxygen pressures on the chest from these studies, together with the results of the mathematical calculations for the same inspired oxygen pressures as given to the subjects. The measured values all fit towards the arterial end of the calculated values with a suggestion that, for inspired oxygen above 101 kPa, measured values fit slightly closer to the venous end which is what one would expect if the skin had increasing vasoconstriction as arterial oxygen increased, thus reducing the effect of the heated electrode.

In conclusion the main message from this work is given in Figures 2 and 3: the benefit from increased inspired oxygen is very small for some tissue types. The higher the oxygen extraction the lower the oxygen drops within the tissue and the greater the proportion of tissue existing at the low oxygen levels and this fact may be more important than the actual end-capillary oxygen levels.

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Flook V. Tissue oxygen levels during hyperbaric oxygen breathing. Reprinted with kind permission from *European Journal of Underwater and Hyperbaric Medicine* 2001; 2(2): 41-48

The major part of this paper was presented as an invited lecture at the EUBS Annual Meeting in Istanbul in 1994.

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates

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

1 The candidate must be a medically qualified financial member of the Society.
2 The candidate must supply evidence of satisfactory completion of examined course(s) in Diving and Hyperbaric Medicine at an approved institution.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written research proposal in a standard format for approval by the Education Officer before commencing their research project.
5 The candidate must produce, to the satisfaction of the Education Officer, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

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

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

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

The Education Officer reserves the right to modify any of these requirements from time to time.

The Education Officer's address is:
Dr David Doolette, Department of Anaesthesia and Intensive Care, University of Adelaide,
Adelaide, South Australia 5005, Australia.
Phone: +61-(0)8-8303-6382.
Fax: +61-(0)8-8303-3909.
E-mail: <David.Doolette@adelaide.edu.au>

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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Minutes of the SPUMS Executive Committee Meeting held in Palau on 24 May 2003

Opened: 1115 hr

Present: Drs G Williams (Immediate Past-President), C Meehan (Secretary), B Trytko (Treasurer), M Davis (Editor), M Bennett (Committee Member)

Apologies: Drs R Walker (President), D Doolette (Education Officer), S Mitchell, D Walker (Committee Members), D Smart (ANZHMG Representative)

1 Minutes of the previous meeting (26 October 2002)
Moved that the minutes be accepted as a true record. Proposed Dr M Davis, seconded Dr G Williams, carried.

2 Matters arising from the minutes

2.1 Update on the SPUMS membership given by SPUMS administrator, Steve Goble. There are currently 807 paid-up members. There was some discussion regarding cost of Internet access. Dr G Williams suggested <www.surf.net.au> as a more cost-effective option.

2.2 Update on NZ Chapter closure given by Dr M Davis. The chapter no longer exists and the moneys have been transferred into a new account.

2.3 Update from the Editor.

2.3.1 The expenses associated with producing

the SPUMS Journal were discussed.

2.3.2 The committee were happy with the new look of the Journal and with the colour additions.

2.3.3 There was discussion regarding the presentations at the ASM and the need for the papers of these presentations to be given to Dr M Davis at the time of the meeting for publication.

2.3.4 There was further discussion regarding a name change for the SPUMS Journal. One popular suggestion was 'Diving and Hyperbaric Medicine, the Journal of the South Pacific Underwater Medicine Society'.

2.3.5 SPUMS Notices, ASM flyer, committee nominations, etc, need to be reviewed regularly by Secretary/President/Treasurer. Deadlines for entry into the journal are as follows:
End of January for the March journal
End of April for the June journal
End of July for the September journal
End of October for the December journal

2.3.6 Relationship to the EUBS Journal was discussed. Dr M Davis will have a formal meeting with Dr Peter Meuller, the Editor, to discuss the Journal.

2.3.7 The journal office needs to purchase Adobe Photoshop pro, academic version.

2.4 Update on UHMS in Sydney given by Dr M Bennett. All is in order. There are some discussions regarding the flights following the meeting to the SPUMS ASM in Noumea. SPUMS is sponsoring the Kronheim lecturer, Mr John Foley, who is a member of the Australian Skeptics Society.

2.5 Dr M Davis gave an update provided by the Education Officer, Dr D Doolette.

3 Annual Scientific Meetings

3.1 2003 Palau. All is progressing well. There is concern regarding the lower number of registrants, and some potential loss of money for this conference. The extent of this will not be known till all moneys have come in and expenses been paid. The exchange rate is favourable at present, which will benefit the Society. There was some confusion about who was responsible for payment for the attendance at the social events of the Allways Travel representatives, Mr Geoff Skinner and Ms Adrienne McKeone. It appears that in the past, SPUMS has paid the cost of their attendance. In the SPUMS Convener booklet there is provision for VIP guests to be invited to the social events at SPUMS' expense. In view of this, and to avoid further misunderstandings, the Allways Travel representatives were officially invited to attend at our expense. Unfortunately, this additional cost had not been factored into the registration fee. In future, there should be discussion regarding any invited guests to the social events prior to setting the registration fee in order to cover the additional costs. Details of provision of FOCs from the resort, airline, and dive operators will be sought, along with their proposed allocation. These will be presented with the final figures.

3.2 2004 Noumea: Dr G Williams gave an update of this. He is still finalising speakers. As some of the attendants will come after the UHMS, it is suggested that Allways Travel have a separated land content cost.

3.3 2005: Dr C Meehan has been nominated to convene. The venue has not been decided on.

3.4 NZ has been mentioned as a venue for a future ASM.

4 Treasurer's report

The Treasurer has recommended that the membership subscription rate remains the same.

5 Correspondence

5.1 Letter re Australian Standard Workshop. There needed to be further clarification regarding this from Dr Walker. It was suggested that if there was to be a meeting that it take place at the time of the face-to-face committee meeting rather than the HTNA meeting in Hobart in August.

5.2 Letter Brian Casey and Dr Walker response was discussed. It was reinforced that the pre- and post-conference trips were independent of SPUMS. In future, there will be no advertising of the pre- or post-conference trips in the SPUMS conference brochure. Information regarding the above will need to be sought directly from Allways Dive Expedition, or other travel providers.

5.3 Letter Aubrey Seknow was read and discussed.

5.4 Letter Malcolm Le May was discussed.

5.5 Letter occupational diving work proposal, Department of Industrial Relations. This needs to be followed up.

5.6 Scuba doo enquiry was discussed.

6 Other business

There was no other business.

Closed: 1330 hr

Minutes of the Annual General Meeting held in Palau on 22 May 2003

Present: All members attending the Annual Scientific Meeting

Apologies: Drs D Doolette, G Long (Jnr), S Mitchell, D Smart, D Walker

Opened: 1835 hr

1 Minutes of the previous meeting
Minutes of the previous meeting have been posted on the notice board.
Motion that the minutes be taken as read and are an accurate record. Proposed Dr D Vote, seconded Dr M Davis, carried.

2 Matters arising from the minutes
Dr H Turnbull gave notice that correct protocol had not been followed at the last AGM in May 2002, when no vote was taken after presenting the motion item 8.1 on the agenda, 'Changes to the SPUMS constitution', and that the constitution, page 12 rule 13 had not been followed, 'A question arising at a general meeting of the Association shall be determined on a show of hands unless a poll is demanded. A declaration by the Chariman that a resolution has been carried or lost, and an entry to that effect in the Minute Book of the Association is evidence of the fact.' In view of this, notice is given that the motion (see *SPUMS J* 2002; 32: 20-21) will be presented again at the AGM 2004.

3 Annual reports received
3.1 President's report
3.2 Secretary's report
3.3 Education Officer's report (verbal only)

4 Annual financial statements
Motion that the financial statements be accepted.
Proposed Dr M Bennett, seconded J Lippmann, carried.

5 Subscription fees for the coming year to remain the same
Proposed Dr M Davis, seconded Dr G Thompson, carried.

6 Election of office bearers
Nominations have been called for the position of Treasurer. No nominations have been received. Dr B Trytko will stand in until a suitable replacement can be found.

7 Appointment of the Auditor: Mr David Porter
Proposed Dr B Trytko, seconded Dr M Bennett, carried.

8 Business of which notice has been given
8.1 That the travel arrangements for all SPUMS ASMs be put out to tender and that all tenders successful or not should be able to be appraised by the membership. (see *SPUMS J* 2003; 33: 51)
Proposed Dr Hamish Turnbull, seconded Dr Gareth Long.
Speakers for the motion: Dr H Turnbull, Dr D Gorman, Dr B Trytko, Mr J Lippmann, Dr M Bennett.
Speaker against the motion: Dr G Leslie.
A show of hands indicated 9 for and 17 against the motion. The motion was lost.

Closed: 1921 hr

Presidentís report 2003

I would firstly like to take the opportunity to apologise for my absence from the Annual Scientific Meeting. It is the first meeting I have missed in a number of years as a result of both work commitments and recent surgery, which have prevented me from travelling. I certainly intend to be present in Noumea in 2004 and thank Guy Williams for deputising for me this week.

Two years ago we had not foreseen the events of 11 September 2001, the Iraq conflict or the appearance of severe acute respiratory syndrome. All have conspired against the international travel industry and once again our number of attendees at the ASM is smaller than in previous years. However, the Society continues to place importance on the Annual Scientific Meeting as a forum for the advancement of diving medical knowledge and a venue to critically examine our practices in the light of international trends and developing research.

The theme of this year's meeting, 'Risk, diving and the pre-diving medical', and the workshop 'Designing a pre-diving medical for the 21st Century' certainly attest to our commitment to remain at the cutting edge of diving medicine and to be in a position to provide relevant advice to statutory bodies on request. I trust the discussion at the ASM has been robust and look forward to the publication of the outcomes in upcoming journals.

It is now some 12 months since Mike Davis assumed the Journal Editor's position and his personal touches are readily evident. The Journal cover design has changed with the addition of colour photography; however, most importantly the scientific content remains high and relevant. It remains my belief that this is the world's premier diving medicine journal and I would encourage all prospective

authors to take the time to submit those papers that we all never seem to get around to finishing.

The SPUMS Diploma has been awarded to a number of individuals throughout the year and I congratulate those diplomates who received their awards here in Palau.

Barb Trytko has submitted her resignation as Treasurer and I thank both Barb and Tim for the many hours of work they have put in over the last two years. The Treasurer's position is onerous and most members do not have the visibility of the amount of effort needed to keep the Australian Tax Office and the auditor happy. The Committee needs new blood and whilst positions like the Treasurer are often considered too hard, the Society cannot continue to function without input from its members. I would recommend to all the value of volunteering a little time to the running of the Society and would be happy to discuss further with anyone considering this in the future.

Next year the Undersea and Hyperbaric Medicine Society (UHMS) meeting is being held in Sydney from 26 to 29 May (Wednesday to Saturday) and the SPUMS meeting has been scheduled for the following week in Noumea. It is hoped that a number of the delegates from the UHMS will consider extending their trip to take in the SPUMS meeting on their way home. I would also encourage our own members to attend the UHMS meeting, which has a bilateral focus on both diving and hyperbaric medicine. It is a great time to interact with our colleagues from all over the world at relatively low cost to us.

In conclusion, I would like to publicly thank all the Committee Members for their time, enthusiasm and commitment over the past 12 months and look forward to catching up again in Noumea in 2004.

Robyn Walker
President, SPUMS

Audit report to the members of the South Pacific Underwater Medicine Society

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of The South Pacific Underwater Medicine Society for the year ended 31 December 2002 report that the accompanying Income and Expenditure and Balance Sheet have been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the year then ended.

Dated 13 June 2003
Suite 304, 20 Bungan Road
Mona Vale, New South Wales 2103

David Porter
Chartered Accountant

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY BALANCE SHEET AS AT 31 DECEMBER 2002

	2002	2001
MEMBERS' FUNDS		
Balance at 1 January 2002	118,788	109,208
Surplus/(Deficiency) for year	(3,932)	9,580
	$114,856	$118,788
represented by:		
CURRENT ASSETS		
ANZ Bank ASM Account	9,212	9,287
ANZ Access Cheque Account	8,085	17,431
ANZ VZ Plus	92,825	89,707
Accounts receivable	1,235	-
Sundry loan	1,456	1,456
GST recoverable	2,043	907
NET ASSETS	$114,856	$118,788

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Mona Vale 2103.
Dated 13 June 2003

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY STATEMENT OF INCOME AND EXPENDITURE FOR THE YEAR ENDED 31 DECEMBER 2002

	2002	2001
INCOME		
Subscriptions & Registrations	90,122	99,519
Interest	3,135	3,519
Advertising & Journal sales	2,045	180
ASM 2002	34,070	28,900
Sundry Income	1,094	121
	$130,466	$132,239
EXPENSES		
ASM costs	45,741	36,499
Secretarial wages	13,532	12,816
Stationery & Printing	612	5,948
Journal	18,288	28,287
Postage & Facsimile	3,883	2,446
Conferences & Telephone	8,819	3,514
Computer Equipment	10,732	2,095
Miscellaneous/Subscriptions	1,755	1,449
Bank Charges	5,764	4,687
Audit	1,600	3,600
Editors honorarium	18,263	16,310
ASM 2002 costs	-	313
Insurance	5,409	4,695
	$134,398	$122,659
SURPLUS/(DEFICIENCY) FOR THE YEAR	$(3,932)	$9,580

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Mona Vale 2103.
Dated 13 June 2003

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY MOVEMENTS ON BANK BALANCES FOR THE YEAR ENDED 31 DECEMBER 2002

		2002	2001
OPENING BALANCES			
ANZ bank	- ASM account	9,287	7,402
	- Access Cheque account	17,431	35,632
	- VZ Plus	89,707	61,585
		116,425	104,619
add, **RECEIPTS**		130,466	132,828
		246,891	237,447
less, **PAYMENTS**		136,769	121,002
CLOSING BALANCES			
ANZ bank	- ASM account	9,212	9,287
	- Access Cheque account	8,085	17,431
	- VZ Plus	92,825	89,707
		$110,122	$116,425

NOTE
Receipts and Payments above include Balance Sheet items (totalling $2,371) which are not included in the Income and Expenditure statement.

Treasurerís report 2003

The last financial year for the Society has been less positive than in the past due to a number of factors, including losses from the 2002 Annual Scientific Meeting in Vanuatu, increased journal running costs and a slight decrease in membership. Despite this, we have not yet had to draw on the investment income and at present are still operating at a reasonable surplus.

As of 24 April 2003 our current accounts stand at:

General operating account	$53,729.06
V2 Plus investment account	$94,614.12
ASM 1995 operating account	$9,212.52
Total	$157,555.70

Audit of the accounts for 2002 is due to be completed at the end of May and will be published in the SPUMS Journal.

Barbara Trytko, Treasurer, SPUMS

Secretaryís report 2003

At present the SPUMS membership is 807 members. There are 130 outstanding renewals for this year. There have been nine resignations this year. There are 59 new members since the last AGM.

Once again I would like to thank the SPUMS Administrator, Mr Steve Goble, for all his hard work. I would also like to thank Dr Barb Trytko for her hard work as SPUMS Treasurer, and for agreeing to continue in the position until a replacement is found. I also thank the rest of the Committee for putting the time aside for the arduous tasks of committee meetings and memos.

We are again committing to another year and hope that during this year we will increase the SPUMS membership. Dr Mike Bennett has kindly formulated a membership package along with an information poster. It is hoped this will help spread the word to new doctors and divers. SPUMS will also endeavour to improve the functionality of the web site. We look forward to a healthier membership next year.

Catherine Meehan, Secretary, SPUMS

ANZHMG Report, October 2002

The Australian and New Zealand Hyperbaric Medicine Group Sub-Committee of SPUMS has continued to be extremely active in a number of areas over the last year.

1 Medicare and hyperbaric oxygen

In 1998, a manufacturer of monoplace hyperbaric chambers took exception to the wording of the CMBS relating to Hyperbaric Oxygen Therapy (HBOT). The individual perceived that monoplace treatments had been excluded from the schedule. He then made a submission to the Medical Services Advisory Committee (MSAC) for a separate monoplace hyperbaric treatment item number. As a result of his submission, MSAC undertook to review the whole evidence base for hyperbaric medicine.

Three directors of teaching hospital hyperbaric medicine facilities, Drs Michael Bennett (Prince of Wales Hospital, New South Wales), Bob Wong (Fremantle Hospital, Western Australia) and David Wilkinson (Royal Adelaide Hospital, South Australia) were co-opted to MSAC. They were required by MSAC to sign a confidentiality document until the release of the assessment report. The Federal Health Minister signed off on the document in February 2001, without allowing public comment.

The outcome of the report led to significant restrictions in the number of indications for which Medicare funding would be available for hyperbaric oxygen treatment. It is now limited to:

- Decompression illness;
- Gas gangrene;
- Air or gas embolism;
- Diabetic wounds including diabetic gangrene and diabetic foot ulcers;
- Necrotising soft-tissue infections including necrotising fasciitis and Fournier's gangrene;
- Prevention and treatment of osteoradionecrosis.

The Committee relied solely on evidence from randomised controlled clinical trials to generate this list. It chose to disregard all other evidence, even if the level of evidence in favour of hyperbaric oxygen treatment was equal to or better than available other treatments. There was also considerable selectivity when interpreting evidence.

Executive members of the ANZHMG through intense political lobbying have been successful in obtaining a reprieve from the full impact of the MSAC report. There was no appeals process for the MSAC Committee. However through the Medicare Benefits Consultative Committee, it was pointed out by the Physician Representatives of ANZHMG that the MSAC Report and its restricted list of indications would have serious impact upon the delivery of services at hyperbaric facilities in Australia.

The result was that a temporary hyperbaric item number 13015 was created by the Health Minister – this does not appear in the CMBS Schedule – to allow the conditions of hypoxic non-diabetic problem wounds and soft tissue radiation injury to be treated pending a new submission from the ANZHMG in relation to these conditions. The submission was forwarded to MSAC on 12 December 2001. The MSAC processes have been nothing short of shambolic. In short, there is no appeals process.

MSAC seem to regard themselves as the hanging judge and jury because they have traditionally only assessed new technology. For hyperbaric oxygen treatment, this was the first time they had assessed an existing funded technology and they did not perceive that when funding is withdrawn, affecting particular groups, then there should be an appropriate appeals mechanism. MSAC then proceeded to attempt to forward our appeal submission to the National Institute of Clinical Effectiveness (NICE) in the UK. NICE were unable to assess the submission and as a result MSAC has now convened its own committee again to review the appeal against its own decision. Members of ANZHMG, Drs Mike Bennett, David Smart, David Wilkinson and Bob Wong, have again been invited to join the committee.

In the interim AMA has forwarded a letter to the Health Minister protesting about the inadequate processes generally of the MSAC and seeking a meeting with the Health Minister as a matter of urgency. MSAC has recently dealt a poor outcome for the profession for trans-oesophageal echocardiography in cardiac surgery. The profession lacks confidence that the MSAC is able to deliver fair and reasonable outcomes.

At this point, we do not have a definite date for the MSAC Committee Meeting however all members have again signed confidentiality agreements and the members will need to hold themselves separate from political processes taking place outside of the MSAC's proceedings.
(**Editor's note:** At the time of publication this committee had met, but no report has yet been published.)

2 Status of submissions to MSAC
Detailed submissions were made to MSAC covering soft-tissue radiation injury and hyperbaric oxygen, hypoxic non-diabetic problem wounds and transcutaneous oximetry. These were in sufficient detail to be reworked into publishable documents and this is occurring progressively in between other commitments for the ANZHMG members.

3 ANZHMG list of approved indications
The current list of September 2000 has not been altered and despite the changes to Medicare, ANZHMG believes that its list of indications is reasonable, based on available evidence and in particular when comparing HBO_2 evidence against the evidence for other available treatments.
(**Editor's note:** The list was published in the last issue of the journal. *SPUMS J* 2003; 33: 111-112)

4 Introductory course in hyperbaric medicine 2002
This course run by Dr Ian Miller was successful and preparations are underway for the course in 2003 (see page 179, this issue). The course complements other available diving medicine courses held around the nation in that the emphasis is 80% hyperbaric and 20% diving medicine.

5 Minimum data set hyperbaric medicine
There are moves in the Australian hyperbaric units to collect a minimum data set. The ANZHMG executive is working on this and a discussion paper will be circulated in 2003.

6 HTNA ASM, Hobart 2003
This year's meeting is being held in Hobart in the last week of August at the Hotel Grand Chancellor Hobart. Invited speakers are David Elliott and Valerie Flook.

7 UHMS Sydney 2004
Mike Bennett and his team are to be congratulated on their success in being awarded the UHMS Conference in Sydney at the end of May 2004.

David Smart, Honorary Chairman, ANZHMG

Letters to the Editor

Children and diving

Dear Editor,

I was interested to read the June 2003 edition of the SPUMS Journal[1] with various articles concerning children in diving. All four of my children (now adults) were keen young snorkellers. Three of them went on to Open Water Scuba certification in their early teens.

I support the current SPUMS recommendation of 14 years for Open Water certification.[2] Younger children can clearly be safely introduced to scuba activities in a swimming pool. However, fundamental skills to be developed early are competent swimming, snorkelling and confidence in the open water environment. Once these skills are well established the addition of scuba equipment is a natural progression.

Thus one must ensure that all trainee divers (both adults and children) are not blindly reliant on a tank of compressed air and other scuba gear to survive in the ocean.[3]

Parents of children undertaking a scuba course preferably should be divers themselves. Unless you are a diver yourself, it is difficult to appreciate the challenges and risks as well as the joys of this sport.

Bill Douglas
201 Wickham Terrace, Brisbane, Qld 4000
E-mail: *<b_douglas@optusnet.com.au>*

References

1 *SPUMS J* 2003; 33 (2)
2 Walker RM. Assessing children's fitness for scuba diving. *MJA* 2002; 176: 450
3 Davis M. The editor's offering. *SPUMS J* 2003; 33: 61

Diving in Antarctica ñ dangerous marine animals

Dear Editor,

I refer to David McD Taylor's article on scuba diving in Antarctica.[1] He refers to the leopard seal (*Hyruga leptonyx*) and states that there have been no reported incidents of these animals injuring divers, but that harassment, which necessitated the abandonment of the dive, has occurred.

Writing in the News section of *The Daily Telegraph* on 24 July 2003, David Derbyshire, the Science Correspondent, reported that Kirsty Brown, 28, a British marine biologist was dragged to her death the day before by a leopard seal as she snorkelled in the bay close to the Rothera research station of the British Antarctic Survey (BAS).

A qualified and experienced diver, Miss Brown was studying the impact of icebergs on marine life on the sea floor. She had been making dives regularly, checking and laying hundreds of concrete and clay markers on the sea floor. The soft upper surfaces of the markers record the scrapes of the bottom of icebergs as they float overhead.

She and her buddy, who were both wearing wetsuits, were swimming on the surface when she was pulled underwater by the seal. She was recovered, but attempts to resuscitate her, including by the station doctor, were unsuccessful.

BAS confirmed that leopard seals, which can grow up to 12 feet long, are fast and powerful hunters. While they are often inquisitive, the BAS had no records of unprovoked attacks on Antarctic researchers. One theory is that the seal mistook the divers for penguins, their main source of food. After pulling Miss Brown under the water, the seal may have realised its mistake and let go.

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

Reference

1 Taylor McD D. Scuba diving in remote locations: Antartica. *SPUMS J* 2003; 33: 6-10

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

All enquiries to:
Diving Historical Society Australia, SE Asia,
PO Box 2064,
Normansville, SA 5204, Australia
Phone: +61-(0)8-558-2970
Fax: +61-(0)8-558-3490
E-mail: <bramsay@iaccess.com.au>

The Ballad of Blue Corner

(reflections on the SPUMS ASM 2003)

Dear Editor,

Despite all the wind, the clouds and the rain,
Expectations were high as we walked from the plane.
Acquaintance renewed, many new faces met,
I unpacked my dive gear and prepared to get wet.

Des Gorman, guest speaker, was noted among
International and local colleagues, old and young
(From howls of ageism I wish to reject,
My reference to 'old' was a term of respect).

Paradise maybe, Palau's weather annoyed.
While Blue Corner beckoned, I still enjoyed
Countless coral and ferns, fish, turtles and rays
Within sheltered waters in those first few days.

Then late in the week, our guide with surprise
Said "Let's go Blue Corner, pool's open guys!"
Once hooked to the rocks I was blown away
By the myriad of life on display there that day.

Not all of us dived at this memorable site,
But as diving physicians I hope that we might
Recall the dive creed – we all dive together.
In unity is strength with all forms of weather.

Cathy Meehan, Mike Bennett with countless aside
Have hosted a meeting in which to take pride,
And whatever fate has in store for me,
I'll 'Allways' remember SPUMS 2003.

David Wilkinson
Royal Adelaide Hospital,
Adelaide, SA 5000, Australia
E-mail: *<Dwilkins@mail.rah.sa.gov.au>*

FREE.....

SPUMS WEB SITE DEVELOPMENT

SPUMS is looking for a volunteer from the membership to take an interest in redeveloping the Society's web site with the aid of a web site professional and working closely with the Society's Administrator, Steve Goble.

This is your chance to have input to the direction your Society takes in regard to its appearance in the growing world of electronic media. Computer literacy is essential; some web site development experience would be desirable. Once the site has been revamped it is hoped that you would take an ongoing interest in the running of the site.
Any expressions of interest should be addressed to the SPUMS Journal e-mail address: <spumsj@cdhb.govt.nz>

Book reviews

Unexpected odyssey

Eric P Kindwall

440 pages, hardback
ISBN 1-930536-09-7
Flagstaff, Arizona: Best Publishing Company, 2002
Available from Best Publishing Company, P O Box 30100, Flagstaff, Arizona 86003-0100, USA.
Ph +1-928-527-1055; Fax: +1-928-526-0370
E-mail: <divebooks@bestpub.com>
Copies can be ordered online at <www.bestpub.com>
Price US$19.95, postage and packing extra

Writing about oneself must be a difficult task, and I have never been a great fan of autobiography, with a few notable exceptions. However, I looked forward to learning at both a personal and professional level about a man who has been one of the father figures of hyperbaric medicine. I purchased a copy of *St Luke's hyperbaric procedures* back in the early 1980s. This and Kindwall's subsequent textbook have been important resources for our hyperbaric service ever since.

Unexpected odyssey was a little disappointing in that Dr Kindwall allows only limited insight into himself, and the story of his more than quarter century of hyperbaric medical practice does not start till page 350. It is as though this were merely a prolonged epilogue to the most important parts of his life, when as a teenager and young man he spent times in Sweden and as a merchant seaman. Throughout, the picture one has is of a man who rarely reveals his true feelings. There are hints about emotional difficulties, brief references to psychotherapy and "psychic scars", and an admission late in the book that perhaps he was "very rigid" in his demands of his first wife. Personal issues are mere asides to the main, and often extraordinarily precise, record. For instance, there is only half a page on his first wife, but 45 pages on submarine training and his first patrol. There are many extraordinary juxtapositions; when talking about an "on-again, off-again" relationship with a Swedish-born classmate at medical school, Kindwall devotes most of the single paragraph to describing the steam train journey to visit her and the type of car they hired.

Kindwall was the only child of Swedish immigrants. His father trained as a psychiatrist after being "a 15-year-old high-school dropout who punched cattle in Dakota", whilst his mother was a medical technician who had worked in von Euler's Karolinska laboratory before emigration. Mention of his relationship with his parents is brief. He describes his father at one point as a strict disciplinarian and both, particularly his father, appear as shadowy figures interwoven into his story. The most insightful remark about himself is perhaps "In a sense, I never had a childhood because I was expected to act like an adult from very early on." At the age of fourteen he spent a year at school in Sweden, which he describes as one of the defining moments of his life. He describes himself as a "singular" child.

The book is presented as a series of anecdotes separated into dozens of very short chapters, as though one were listening to someone reminiscing in front of the fire on a long winter's evening. Nothing is dealt with in depth unless it is a ship's engine or an old car he and a friend are renovating, and one is not given much insight into most of the major personal and professional events in his life.

A fascinating read because of the tangled style of writing with its frequent jumps from topic to topic but, ultimately, rather shallow. Kindwall the man remains elusive, but you would be happy for him to renovate your vintage 'Model T' Ford, role up his sleeves to put in your recompression chamber gas plant or look after you for a 'bend'.

Michael Davis
Editor, SPUMS Journal

Key words
Book reviews, autobiography, underwater medicine

Submerged

Daniel Lenihan

287 pages, hardback
ISBN 1-55704-505-4
New York, New York: Newmarket Press, 2002
Available from Best Publishing Company, P O Box 30100, Flagstaff, Arizona 86003-0100, USA.
Ph +1-928-527-1055; Fax: +1-928-526-0370
E-mail: <divebooks@bestpub.com>
Copies can be ordered online at <www.bestpub.com>
Price US$29.95, postage and packing extra

This interesting and informative book is subtitled "*Adventures of America's most elite underwater archaeology team*". When the editor first asked me to review this book I knew nothing about marine archaeologists other than that they were usually the most intransigent group at occupational diving standards meetings. I also knew that most of the diving population in South Australia viewed them with suspicion because of their sometimes obscene rush to declare every wreck historic, even those where there was a distinct possibility that the insurer still owned both wreck and cargo.

After reading Daniel Lenihan's wonderful tale of discovery and exploration I have to admit that my views have changed. My views were changed while reading Chapter 2, where the author describes how as an anthropology student he removed an artifact from a local sinkhole, cleaned it up

and took it back to the anthropology lab. The resulting lecture from his professor on the amount of information that had been lost as a result of his actions made him feel that he had acted like an idiot. He swiftly learned that shipwrecks are an even greater treasure trove of information, provided they are archaeologically surveyed and not plundered by wreck hunters.

While studying in Florida, Lenihan became a member of the local cave-diving fraternity, quickly becoming friends with the likes of Sheck Exley and David Desautels. During his time cave diving he learned and refined the techniques and safe practices he would later introduce to marine archaeological surveys. Cave diving also led to Lenihan's employment by the National Parks Service (NPS) as a park ranger/archaeologist.

Lenihan leads us on a chronological tour of his career in the NPS starting off with a project called the National Reservoir Inundation Study and developing into a job as the Director of the Submerged Cultural Resources Unit, or SCRU for short. Through each step one can see the development of techniques for surveying and mapping wreck sites in limited visibility, cold water and extreme depths; techniques now accepted by the growing number of marine archaeologists working for governments and universities around the world.

During his time as Director of SCRU, Lenihan supervised projects from the Bering Sea to Micronesia, on wrecks from the American Civil War to the fleet sunk at Bikini Atoll during the nuclear testing. They were the first team to dive, survey and map the wreck of the USS Arizona in Pearl Harbour and the first team to dive the wrecks at Bikini Atoll, checking the background radiation and assessing the site's possible future as a tourist dive destination.

The photographs are superb, as one would expect from a marine archaeologist, and the descriptions of various projects at times enthralling. This is a well-written and interesting book, excellent bedtime reading for anyone with an interest in diving, archaeology, history or adventure.

Steve Goble,
Hyperbaric Medicine Unit, Royal Adelaide Hospital

Key words
Book reviews, autobiography, history, cave diving, salvage

FASCINATION APNOEA-DIVING
Physiology, Pathophysiology, Safety and Training
Dates: 8 to 9 November, 2003
Venue: University of Ulm/Donau, Germany
Contact: Dr Claus-Martin Muth
E-mail: <CMMuth@aol.com>

SPUMS 2004
Annual Scientific Meeting
Noumea ñ New Caledonia
Venue - Le Meridien Noumea
May 30th – June 6th 2004
(meeting to run June 1st – 5th inclusive)

SPUMS meeting to follow UHMS Sydney 2004

Themes
Marine Stingers and Marine Envenomation
Modelling Dcompression Tables

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

Convener
Dr Guy Williams
P.O. Box 190, Red Hill South, VIC 3937 AUSTRALIA
***Phone:** +61-(0)3-5981-1555*
***Fax:** +61-(0)3-5981-2213*
***E-mail:** <guyw@surf.net.au>*

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Allways Dive Expeditions
168 High Street, Ashburton, VIC, 3147 AUSTRALIA
***Phone:** +61-(0)3 -9885-8863*
***Fax:** +61-(0)3-9885-1164*
***Toll Free:** 1800338239*
***E-mail:** <Allwaysdive@bigpond.com.au>*

UNDERSEA AND HYPERBARIC MEDICINE SOCIETY
37th Annual Scientific Meeting
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>

FREMANTLE HOSPITAL
DEPT OF DIVING & HYPERBARIC MEDICINE
DIVING MEDICAL EXAMINATION FOR RECREATIONAL DIVERS

A three-day course will be conducted for medical practitioners who wish to perform medical examinations for recreational divers in accordance with Australian Standard AS 4005.1. RACGP CME credit hours applied for.
Dates: 28 to 30 November 2003
Venue: Fremantle Hospital, Alma Street, Fremantle, WA
Contact: Mrs Beth Karlsson, Administrative Assistant
Phone: +61-(0)8-9431-2233
Fax: +61-(0)8-9431-2235

ANZHMG and ANZCA INTRODUCTORY COURSE IN DIVING AND HYPERBARIC MEDICINE

Dates: 1 to 12 March, 2004
Venue: Prince of Wales Hospital, Sydney
Cost: A$1,600.00 plus GST ($160.00)

This course is designed for medical graduates with an interest in the practice of general hyperbaric medicine, including relevant aspects of diving medicine. It is designed both for those wishing to pursue a career in the field and those whose primary area of interest lies in related areas. Prior experience is not required but would be of advantage. Extensive pre-reading material will be supplied. The course is limited to 20 participants.

The course is jointly sponsored by the ANZHMG, ANZCA and the UHMS. It is accredited with SPUMS for the Diploma of Diving and Hyperbaric Medicine and attracts 70 US CME points.

Contact: Miss Gabrielle Janik, Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Barker Street, Randwick, NSW 2031, Australia
Phone: +61-(0)2-9382-3880
Fax: +61-(0)2-9382-3882
E-mail: <janikg@sesahs.nsw.gov.au>

ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS UNDERWATER MEDICINE COURSE 2003

Dates: 24 November to 5 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 information and application forms contact: The Officer in Charge, Submarine & Underwater Medicine Unit, HMAS PENGUIN, Middle Head Rd, Mosman, 2088 NSW
Phone: +61-(0)2-9960-0572
Fax: +61-(0)2-9960-4435
E-mail: <Sarah.Sharkey@defence.gov.au>

AUSTRALIAN AND NEW ZEALAND COLLEGE OF ANAESTHETISTS
2004 Annual Scientific Meeting
Preliminary Notice

Diving and Hyperbaric Medicine Special Interest Group
Dates: 1-5 May
Venue: Perth Concert Hall and Duxton Hotel, Perth
Contact: Katie Clarke, Congress West
E-mail: <conwes@congresswest.com.au>

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE COURSES 2003
Medical Officers Course

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
October 2003 3 weeks, 13/10/03 to 31/10/03

DMT Refresher Course
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-(0)8-8222-5116
Fax: +61-(0)8-8232-4207

10TH INTERNATIONAL CONFERENCE ON EMERGENCY MEDICINE (ICEM 2004)

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

CLINICAL MANAGEMENT of DIVING ACCIDENTS
An advanced course for medical practitioners
TRIAGE and MANAGEMENT of DIVING ACCIDENTS
An international workshop
Dates: 10 to 14 November, 2003
Venue: Patong, Thailand
Contact: Professor David Elliott, 40 Poetworth Road, Haslemere, Surrey GU27 2HX
Fax: +44-(0)1428-658678
E-mail: <davidelliott@aol.com>

SECOND INTERNATIONAL MEETING OF EMERGENCY MEDICINE IN THE PACIFIC REGION

Dates: 23 to 25 February, 2004
Venue: Tahiti
All information on this meeting can be found on <www.emergency-tahiti.com> and booking can be made online. Papers accepted until 23 November 2003.
Contact: Dr Yann Turgeon
E-mail: <info@urgences-polynesie.pf>

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 disk (preferred) or by e-mail. The preferred format is Word 6 for Windows. Two printed copies of all text, tables and illustrations should also be mailed. All articles should include a title page, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied. The text should be subdivided into the following sections: an Abstract of no more than 250 words, Introduction, Methods, Results, Discussion, Acknowledgements and References. Acknowledgments should be brief. References should be in the format shown below. Legends for tables and figures should appear at the end of the text file after the references.

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. Underlining should not be used. Measurements are to be in SI units (mm Hg are acceptable for blood pressure measurements) and normal ranges should be included.

The preferred length for original articles is 3,000 words or less. Inclusion of more than five authors requires justification as does more than 30 references per major article. Case reports should not exceed 1,500 words, with a maximum of 10 references. Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references, which should be limited to five per letter). Legends for figures and tables should be less than 40 words in length.

Illustrations, figures and tables should NOT be embedded in the wordprocessor document, only their position indicated. All tables are to be in Word for Windows, tab-separated text rather than using the columns/tables option or other software, and each saved as a separate file. They should be double spaced on separate sheets of paper. No vertical or horizontal rules are to be used. Illustrations and figures should be separate documents in JPG or TIFF format. The firewall has a maximum file size of 5Mbytes. **Photographs** should be glossy, black-and-white or colour. Slides should be converted to photographs before being sent. Colour reproduction is available only when it is essential for clinical purposes and may be at the authors' expense. Indicate magnification for photomicrographs.

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

References
The Journal reference style is the 'Vancouver' style *(Uniform requirements for manuscripts submitted to biomedical journals*, updated October 2001. <http://www.icmje.org/index.html>).
In this system references appear in the text as superscript numbers.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples of the format for quoting journals and books are given below.

1 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 a space after the semi-colon and after the colon, and no full stop after the page numbers. Titles of quoted books and journals should be in italics. For those using referencing software, the format is the same as the *British Journal of Anaesthesia*. Accuracy of the references is the responsibility of authors.

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

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DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA

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

NEW ZEALAND

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

The DES numbers 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>

DISCLAIMER

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

CONTENTS

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