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South Pacific Underwater Medicine Society Incorporated

CONTENTS

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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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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 5 per letter).

References

The Journal reference style is the "Vancouver" style, printed in the Medical Journal of Australia, February 15, 1988; 148: 189-194. In this references appear in the text as superscript numbers.[1-2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used. Examples of the format for quoting journals and books are given below.

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

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To obtain Diving Incident Report forms write to DIMS, GPO Box 400, Adelaide, South Australia 5000.

THE EDITOR'S OFFERING

A Happy Christmas and a Prosperous New Year to all our readers. We have to apologise for the delay in getting the September issue into the post. Here is the largest issue of the Journal with 24 pages more than usual to fill the gaps in your (Australasian) holiday reading.

In this issue are the papers presented and provided for the Workshop on Asthma and Diving held at the 1995 Annual Scientific Meeting (ASM). There are plenty of divers with asthma who apparently dive successfully. Some divers with asthma come to harm from their diving and it is these unfortunates who provide the hard data either by being admitted to a hyperbaric facility or by being the subject of an inquest. Unfortunately we have no means of knowing how large a proportion of divers are asthmatics. Simpson and Meehan found that 12% of their volunteer experienced scuba diver subjects had a history of asthma or wheezing, which is about the proportion of asthmatics in the general population. If their finding is confirmed by other studies of larger numbers we will be able to make realistic assessments of the risks of diving for asthmatics compared to non-asthmatics in Australia. American statistics from the Divers Alert Network (DAN) suggest that the risk for an asthmatic is about double the risk for a non- asthmatic for both death from cerebral arterial gas embolism (CAGE) and decompression sickness. The thing to remember is that these risks are very low for either group so one is looking at a very uncommon occurrence.

That said the Workshop considered how best to screen asthmatics to prevent those most likely to damage themselves from diving and to discover who could go diving in safety. This is the kernel of the problem. All were agreed that active asthmatics, that is those needing treatment to avoid wheezing in their daily life, were in the high risk category but both the Cairns divers and Peter Chapman-Smith's New Zealanders had people in this category who had done many dives without pulmonary barotrauma or incapacity from breathlessness. So is the received wisdom right ? An Undersea and Hyperbaric Medical Society (UHMS) Workshop recently accepted the concept that asthmatics who are well controlled on inhaled steroids should be allowed to dive if they do not react to exercise or salt water provocation. So things are moving away from asthma = never diving.

Unfortunately there does not seem to be any good clinical indicator that will foretell who will develop bronchial hyperresponsiveness with inhaled 4.5% saline. Developing asthma underwater seems a dangerous situation, but there will be people who would accept this risk for themselves if it was the only way to get underwater. But most diving candidates are put off diving when the result of their saline provocation test is shortness of breath or wheezing. Anderson et al. present a clear overview of the problems of asthma and of provocation testing. Their decision chart, printed on page 244, leads one on a logical path through the pitfalls of deciding what to do about a person with a history of asthma who wants to dive. They do not know whether any of those they advised not to dive disregarded the advice and presented for another medical, suppressing the history of asthma, and passing, going on to learn to dive.

The only way one can find out what actually happens to asthmatics of varying degrees of severity is by long term follow up of individuals and preferably of every individual. Of course this is an almost impossible task involving much time and money.

Douglas Walker started Project Proteus early in 1995 to encourage divers with asthma to communicate with him by filling in a questionnaire. His paper was written as a Workshop contribution, but could not be presented because of time constraints. It is printed with the other Workshop papers. If successful Project Proteus could provide a snapshot of a cross section of the diving public and help establish the incidence of asthma among Australian divers.

The *SPUMS Policy on the Assessment of Risk for a Diving Candidate with a History of Asthma*, printed on page 213, proposes that permanent records should be retained as part of a SPUMS-sponsored study. There is scope for members to provide information and volunteer to assist by writing to the Secretary of SPUMS, Dr Cathy Meehan.

The other result of the workshop was the *Draft SPUMS Policy Statement on the "Certification" of Candidates for Recreational Diving*, printed on pages 214 and 215. As policy on certification was not included in the notices announcing the Workshop the Executive Committee decided that members should be able to put their views forward before the draft is adopted as policy. Readers are invited to put pen to paper, but considering doctors' handwriting finger to keyboard would be preferred, in the form of a letter to the Editor, expressing their views about the draft and the certificate, on page 214, and how both could be improved. There is a lot to be said in favour of the proposed certificate but it will involve a great deal of explaining to diving candidates in order to be certain that the candidate has really understood the implications of going diving with whatever disorder has been found during the examination. However such a policy of risk acceptance would allow asthmatics and diabetics, who often feel that they are discriminated against when they want to dive, to have a chance of learning to dive if their instructor is willing to accept the risk of teaching them. The policy would, one hopes, abolish the need for an intending diver to suppress various parts of their medical history in order to pass the medical.

ORIGINAL ARTICLES

THE ROLE OF LIGNOCAINE IN THE TREATMENT OF DECOMPRESSION ILLNESS A REVIEW OF THE LITERATURE

Simon J Mitchell

Introduction

Decompression illness (DCI) is a complex multisystem disease of diving and aviation, for which definitive treatment comprises compression and administration of hyperbaric oxygen (HBO). Recent studies reveal an undesirably high failure rate for current treatment protocols and indicate the need for alternatives and/or effective adjuvants to HBO. In vivo data suggest a role for lignocaine (lidocaine) in the treatment of DCI, and possible mechanisms of action in this role have been identified. This paper briefly reviews the pathophysiology and treatment of DCI and, in the context of this discussion, examines the evidence suggesting a role for lignocaine as an adjuvant to HBO. Several studies of lignocaine in the treatment of DCI or analogous pathologies are currently under way.

Pathophysiology of decompression illness

Decompression illness arises secondary to the formation of bubbles outside normal gas containing spaces (intracorporeal bubbles) following reduction of ambient pressure.[1] Intravascular[2-4] and extravascular[5-7] bubbles may arise from inert gases dissolved in the blood or tissue respectively. Intravascular bubbles may also arise from pulmonary barotrauma.[8-10]

Intravascular bubbles may: obstruct blood vessels causing tissue ischaemia,[11] disrupt endothelium,[12,13] and activate leucocytes[14-16] platelets,[14,17,18] and biochemical pathways such as the complement[19,20] and kinin[1] systems. Secondary microcirculatory compromise and tissue ischaemia may follow due to clotting,[17,18] intravascular volume loss with red cell sludging,[21,22] increase in interstitial fluid pressure,[1] and leucocyte aggregation.[23] Extravascular bubbles may mechanically disrupt both surrounding tissue[1] and blood flow through adjacent microvessels.[24,25] Secondary activation of leucocytes and inflammatory pathways may also follow extravascular bubble formation.[7] Intra- or extravascular leucocyte activation may cause tissue damage through cytotoxic substance release.[26,27] In summary, intravascular and extravascular bubbles may give rise to tissue damage which may be ischaemic, mechanical, or inflammatory in nature.

Multiple organ systems may be compromised by these processes,[28] however the most dramatic and potentially debilitating consequences of intracorporeal bubble formation are observed when the central nervous system is affected. Aspects of bubble induced ischaemic and inflammatory damage to neurones are particularly relevant to the subsequent discussion of lignocaine.

Cerebral arterial gas embolism (CAGE) may cause neuronal ischaemia in two phases. Occupation of a vessel by a bubble may cause a period of complete ischaemia, which is relieved by redistribution of the bubble into the venous circulation,[29,30] with restoration of flow. Subsequently, there may be a more protracted period of relative ischaemia as perfusion is reduced by secondary inflammatory changes, particularly the accumulation of leucocytes on damaged endothelium.[31,32]

Neuronal ischaemia leads to loss of energy substrate for membrane ion pump function and a consequent disabling of intracellular homeostasis.[33] There is efflux of potassium and influx of sodium leading to loss of excitability (and therefore function), opening of voltage dependent calcium channels, and release of excitatory amino acids. The resultant increase in intracellular calcium, which is enhanced by opening of agonist operated membrane channels and calcium sequestration from intracellular sources, enables a complicated cascade of injurious reactions, involving protein kinase C and calmodulin, and ultimately leads to cell death.

Treatment of decompression illness

First aid management of DCI includes resuscitation, horizontal positioning of the victim, and administration of 100% oxygen (FIO_2=1) and fluids.[34] Recompression and HBO are the major components of definitive DCI treatment.[35] Compression reduces bubble size in accordance with Boyle's Law, thus encouraging the redistribution of intravascular bubbles and relieving the mechanical distortion of tissues by extravascular bubbles. Hyperbaric oxygen administration increases the diffusion gradient for nitrogen between bubble and lungs, thus achieving more rapid bubble resolution and elimination of nitrogen from tissue.[35] Hyperbaric oxygen may also have a role in reducing the accumulation of leucocytes in response to tissue and vessel damage or hypoxia.[36]

Since approximately 3 bar is the greatest oxygen tension that may be breathed in treatment before the incidence of convulsions becomes unacceptably high, modern treatments are most often based on compression to the equivalent of 18 m of sea water (2.8 bar) breathing 100% oxygen,[35] the "minimal recompression oxygen

tables". Animal data do not suggest any advantage for deeper compressions on air or modified oxygen-nitrogen mixtures.[37,38] However the recently described advantages of helium oxygen (heliox) mixtures over oxygen in tissue air bubble resolution,[39,40] have raised the possibility of more effective deeper treatments using heliox, unencumbered by risk of convulsion. Evaluation of heliox tables versus the minimal recompression oxygen tables in DCI treatment is currently underway.[41]

Administration of intravenous fluids to ameliorate the microcirculatory compromise which follows intravascular volume loss, clotting, and leucocyte accumulation, is the only adjunctive therapy to compression which is firmly recommended.[35] Although widespread, the administration of corticosteroids in DCI is controversial and largely unsupported by convincing data.[42] There is some in vivo data to support the use of indomethacin, prostaglandin I_2 and heparin in combination in DCI,[43] but in vivo haemorrhagic complications have been widely reported and this therapy is not recommended.[35]

Recent post-recompression follow-up surveys of several groups of DCI patients consisting mainly of recreational divers, suggest that failure rates for current treatment protocols are unacceptably high,[44-47] with residual cognitive changes being prominent. It follows that therapeutic recompression strategies more effective than the minimal recompression oxygen tables are being tested, and that effective cerebroprotective adjuvants to recompression are sought.[48] One potential adjuvant is lignocaine and the evidence in support of this is discussed below.

The role for lignocaine in treatment of DCI

Lignocaine is a cationic amide compound which blocks membrane sodium channels. It has a high volume of distribution, readily crosses the blood brain barrier,[49] and is rapidly metabolised by the liver with the metabolites undergoing renal excretion.[50] In sufficient concentrations, lignocaine can prevent the propagation of action potentials along excitable membranes. It is used as an injectable or topical local anaesthetic, and as an injectable antiarrhythmic agent (class lB) in the prophylaxis of ventricular tachycardia and fibrillation.[50] Lignocaine has a relatively low therapeutic index,[50] and a therapeutic range for antiarrhythmia treatment of 6-21 μmol/l. Plasma levels are monitored to prevent toxicity which may be manifest as cerebral irritability, bradycardia, atrioventricular (A-V) block, or myocardial depression.[50] Lignocaine should not be administered to patients with a supraventricular arrhythmia or heart block.

In vivo data indicate a protective role for lignocaine in ischaemic cerebral injury and other central nervous system insults. Much of this data relate to experiments in injuries analogous to DCI. A number of experiments have been reported which provide possible mechanistic explanations for lignocaine's protective effect. Key functional protection and mechanistic studies are described below.

Preservation of neural function during ischaemia by lignocaine.

Early in vivo studies of CAGE pathophysiology revealed that such events caused severe cardiac arrhythmias, acute hypertension,[51] severe elevation of intracranial pressure (ICP) and significant increases in plasma catecholamines.[52] It was also observed that lignocaine eliminated or significantly attenuated these changes.[52,53] It was proposed that these beneficial effects might translate into protection of cerebral function, and the first experiment specifically investigating cerebral function preservation by lignocaine in CAGE was reported by Evans et al.[54,55] Anaesthetised cats were pretreated with 5 mg/kg lignocaine in a short infusion five minutes before a single bolus of 0.4 ml of air was injected into the vertebral artery. Mean sciatic/cerebral somatosensory evoked response (SER) in an untreated control group initially fell to 28% of baseline value, recovering to 60% and 73% over one and two hours respectively. The mean SER in the treatment group initially fell to 68% of baseline, recovering to 89% and 95% over one and two hours ($P<0.01$ for all differences). Lignocaine also attenuated the increases in heart rate, blood pressure, and ICP seen in the control group.

The same authors subsequently published another study using a modified model, and administration of lignocaine after the injury.[56,57] Cats received 0.08 ml increments of air into the carotid artery until the SER was reduced to 10% of baseline levels over a period of 5 minutes. Five minutes later, treatment group cats received lignocaine in a 1.5 mg/kg bolus, followed by an infusion at 3 mg/kg over 30 minutes, then 1 mg/kg every 30 minutes thereafter. This regimen was demonstrated to achieve plasma levels of 8-16 μmol/l for the duration of the experiment. Control and treatment group mean SER recovered to 32.6% and 77.3% of baseline respectively over 100 minutes ($P= 0.001$). In an important additional experiment lignocaine alone was found to have no effect on the SER of uninjured cats.

In another CAGE experiment, Dutka et al. produced cerebral dysfunction in anaesthetised dogs using a single bolus of 0.4 ml air to the carotid artery, and a pharmacologically induced post embolic hypertensive spike.[58,59] Animals were not entered into the study unless the embolus reduced SER to ≤10% of baseline. Control and treatment group animals received HBO treatment with a modified USN Table 6A,[60] while treatment animals also received a post-injury lignocaine infusion using the same

dosage regimen used by Evans et al.[57] On completion of the Table 6A, the mean treatment group SER had recovered to 60% baseline versus 32% for the control group (P< 0.01). Average post-injury cerebral blood flow (CBF) was significantly greater in the lignocaine treated group (P=0.019). This study was important in the specific context of DCI therapeutics since a role for lignocaine as an adjuvant to HBO was indicated. Limited anecdotal human data support this contention. Drewry and Gorman reported a case of neurological DCI, refractory to HBO, in which dramatic improvement seemed temporally related to lignocaine therapy.[61]

These CAGE experiments generated interest in lignocaine as a cerebroprotective agent in other forms of ischaemic injury. While the studies performed in this area are not directly related to DCI, the emergent role for lignocaine in brain protection is of relevance.

Gelb et al. describe a feline model of severe focal cerebral ischaemia (middle cerebral artery occlusion for six hours) in which a 5 mg/kg bolus of lignocaine produced a transient protective effect as indicated by preservation of SER compared with controls.[62,63]

Sutherland et al. administered a 5 mg/kg bolus of lignocaine to a treatment group of rats 10 minutes before a 10 minute period of incomplete global ischaemia (achieved by bilateral carotid artery clamping and artificially induced hypotension).[64] A saline control group received a bolus of saline equivalent in volume to the lignocaine dose, and an untreated control group received neither lignocaine nor saline. Rats were allowed to recover for seven days after ischaemia before being sacrificed for cerebral histopathology. In lignocaine treated rats there was significantly less neuronal injury in the CA3 region of the hippocampus (P <0.05 compared with untreated controls). A numerical trend towards less severe grades of injury was recorded in other areas in the lignocaine treated rats, but this was not significant.

Shokunbi et al. administered a bolus and infusion of lignocaine to a treatment group of cats 30 minutes before and then throughout three hours of middle cerebral artery occlusion and three hours of reperfusion with SER monitoring.[65] Their dose regimen achieved plasma levels which peaked at 20.63 μmol/l after the bolus and 30 minutes of the infusion, falling to 12.85 μmol/l after two hours of reperfusion. A control group received a bolus and infusion of saline equivalent in volume to the lignocaine dose. Treatment group mean SER was better preserved at induction of ischaemia, and recovered to higher levels compared with controls (P <0.05). Histopathological analysis post mortem revealed mean infarct size (cross sectional area of a standardised section) to be significantly smaller in the treatment group (P <0.05).

In a complex series of experiments using rabbits, Rasool et al. administered an infusion of lignocaine at 0.2 mg/kg/min for 15 minutes, before, throughout, and for 40 minutes following a 20 minute period of incomplete global ischaemia titrated to produce standard EEG changes.[66] The amplitudes of both the positive and negative peak potentials of the SER decreased significantly less during ischaemia, and recovered more quickly and significantly more completely during reperfusion in the treatment group compared with a control group.

Nagao et al. administered a 3 mg/kg bolus of lignocaine followed by a 2 mg/kg/hr infusion beginning immediately before, and throughout, 12 hours of left cerebral hemisphere exposure to air achieved by craniotomy and dural resection.[67] This model precipitates progressive cerebral oedema and ischaemia. In the lignocaine treated animals there was significant preservation of SER latency duration, preservation of direct cortical response amplitude in the cortex and white matter, preservation of cerebral blood flow, and reduced oedema in the cortex, compared with controls.

Several investigations of lignocaine in cerebral ischaemia have failed to demonstrate any protective effect.[68,69] Shokunbi et al. administered lignocaine to cats in unconventional doses (50 mg followed by 50 mg/kg/hr) to produce and maintain EEG flattening, beginning 30 minutes before, and continuing throughout left middle cerebral artery clamping for four to six hours.[68] Histopathological brain examination at the end of the ischaemic period revealed no difference in the size of the grossly infarcted area between treated and control cats. The extent of the severe neuronal alteration was reduced in the treated group but this was not significant.

Warner et al. administered lignocaine in unconventional bolus doses (mean 23.5 mg/kg), titrated to produce a pre-epileptogenic EEG pattern in rats, immediately before 10 minutes of global ischaemia.[69] There was no significant difference between treatment rats and a control group with regard to post ischaemic EEG recovery, brain water content at 90 minutes post-ischaemia, or histopathological changes at seven days post-ischaemia.

McDermott et al. administered lignocaine to cats concurrent with compression and HBO therapy beginning 15 minutes after air embolism to the carotid artery (0.08 ml increments sufficient to reduce the SER to <10% of baseline for 15 minutes).[70] The dose regimen used by Evans et al.[57] was employed. The lignocaine with HBO group exhibited a significant improvement in SER recovery compared with a group receiving no treatment at all, but was not significantly different from a group receiving HBO alone. No lignocaine only group was tested, so this result may simply reflect salvage of the same population of compromised neurones by HBO and lignocaine, with no additive effect.

Several authors report investigations of lignocaine protection in spinal injuries.[71,72] Kobrine et al. administered lignocaine using the Evans regimen[57] to cats, beginning 15 minutes after a 15 second balloon catheter inflation in the T6 epidural space.[71] There was significant return of the sciatic SER of three of five treated cats, compared with minimal return in only one of five controls. Moreover, there was markedly less haemorrhagic damage on histopathological examination in the cords of treated cats. In a similar experiment using a weight drop method to inflict the injury, no recovery occurred in either group.[72] However, the weight drop spinal injury may be too severe to allow a realistic possibility of neuronal recovery with any treatment.[73]

In summary, lignocaine administered both prophylactically and immediately after injury, in doses designed to achieve plasma levels comparable to conventional antiarrhythmic levels in humans, has been demonstrated to be protective of cerebral function in a number of animal models of air embolism, focal ischaemia, and global ischaemia.[54-59,62-67] It has failed to provide protection when administered in doses achieving plasma levels greater than conventional antiarrhythmic levels, and in relatively severe models of focal and global ischaemia.[68,69] There is conflict regarding its additive effect to HBO in the treatment of air embolism,[58,59,70] and regarding its role in the treatment of spinal injury.[71-73] The key features of the cerebral protection studies are summarised in Table 1.

Mechanisms of protection

The four possible mechanisms commonly proposed to explain the neuroprotective properties of lignocaine are respectively titled: the neuronal membrane stabilisation / ion channel blockade hypothesis; the reduction of the cerebral metabolic rate of oxygen ($CMRO_2$) hypothesis; the haemodynamic modification hypothesis; and the modification of leucocyte and other blood element activity hypothesis. The evidence supporting each of these theories is discussed below.

THE MEMBRANE STABILISATION / ION CHANNEL BLOCKADE HYPOTHESIS

From the earlier discussion of ischaemic neuronal injury mechanisms,[29] it can be reasoned that a delay or deceleration of ischaemic ion shifts might protect neurones. The protective effect of hypothermia in cerebral ischaemia[74] is now universally accepted and it has been demonstrated that in ischaemic cortical neurones hypothermia both decreases the depletion of adenosine triphosphate (ATP)[75] and delays ischaemic ion shifts.[76] It is unclear whether a pharmacologically induced reduction in ischaemic ion shifts would also equate with protection.[77] Nevertheless, Astrup et al. found that lignocaine in extremely high doses (160 mg/kg) significantly delayed cortical potassium efflux during circulatory arrest in dogs at normothermia, and added to the effect of hypothermia.[77] They proposed that by membrane stabilisation, lignocaine might provide clinically useful brain protection during ischaemia by "saving the energy needed for maintaining the membrane potentials by ion pumping".

In another in vivo study using rats, Prenen et al. found that intrastriatal tetrodotoxin (another sodium channel blocker) significantly delayed deflection of the interstitial cortical potential which indicates significant cation shifts early in cerebral circulatory arrest.[78] Moreover, in rats allowed to survive for 24 hours following a standardised cerebral ischaemic insult, tetrodotoxin injected locally into the striatum almost completely prevented the ionic derangements characteristic of significant damage which were seen in other cerebral regions, and in the striatal areas of untreated controls. They suggested that ischaemic sodium influx into neurones may be a pivotal event in neuronal death. Further, they argued that blocking sodium channels and thereby preventing or slowing these changes may enhance neuronal recovery in a reversible injury.

A membrane stabilisation role for lignocaine in functional protection is supported by Fink who found that the C fibre action potential decrement in rabbit vagi incubated in a glucose free medium, was paradoxically delayed by addition of lignocaine to the incubation fluid.[79] Fink's observation by microelectrode studies that lignocaine reduced axonal potassium efflux after membrane pumps were disabled by hypoglycaemia, suggests that preservation of excitability was achieved by membrane stabilisation.

Although Gelb et al. noted that the sodium channel blockers flecainide and mexiletine, administered in high doses, were not effective in reducing infarct size in a feline model of focal ischaemia,[80] the model was particularly severe. The authors' conclusion, that the failure of these agents suggests that lignocaine does not provide protection by ion channel blockade, is unreasonable.

In addition to ion channel blockade, other aspects of membrane stabilisation may be relevant to lignocaine's neuroprotective properties. Lignocaine may participate in hydrophobic and electrostatic interactions with membrane phospholipids[81,82] and these effects may promote physical membrane stability.[83] Certainly, lignocaine reduces erythrocyte fragility[84] and reduces cell to cell fusion.[85] It has also been suggested that membrane stabilisation may reduce the release of free fatty acids and consequent generation of prostaglandins and toxic free radicals.[64] Finally, membrane stabilisation by lignocaine may prevent damaging mobilisation of intracellular calcium stores during ischaemia.[86]

TABLE 1

INVESTIGATIONS OF CEREBRAL FUNCTION PROTECTION BY LIGNOCAINE IN ISCHAEMIC INJURY

Authors	Model	Dose timing	Dose size	Dose regimen	Lesion	Outcome parameters
STUDIES DEMONSTRATING BENEFIT FROM LIGNOCAINE						
Evans et al.[54,55]	Cat	Pre-injury	Conventional	B	CAGE	SER
Evans et al.[57]	Cat	Post-injury	Conventional	B+I	CAGE	SER
Dutka et al.[58,59]	Dog	Post-injury	Conventional	B+I	CAGE	SER
Gelb et al.[62,63]	Cat	Pre-injury	Conventional	B	Focal ischaemia	SER
Shokunbi et al.[65]	Cat	Pre-injury	Conventional	B+I	Focal ischaemia	SER + histopathology
Sutherland et al.[64]	Rat	Pre-injury	Conventional	B	Incomplete global ischaemia	Histopathology
Rasool et al.[66]	Rabbit	Pre-injury	Conventional	B+I	Incomplete global ischaemia	SER
Nagao et al.[67]	Cat	Pre-injury	Conventional	B+I	Cerebral air exposure	SER
STUDIES DEMONSTRATING NO BENEFIT FROM LIGNOCAINE						
Shokunbi et al.[68]	Cat	Pre-injury	Higher than conventional	B+I	Focal ischaemia	Histopathology
Warner et al.[69]	Rat	Pre-injury	Higher than conventional	B	Incomplete global ischaemia	Histopathology

B = bolus dose only. B+I = bolus and continuous infusion.

Although it is difficult to relate the relevance of in vivo tetrodotoxin,[78] or extremely high doses of lignocaine,[77] to clinical lignocaine administration, sodium channel blockade and physical membrane stabilisation are established as potential neuroprotective mechanisms for lignocaine.

THE CEREBRAL METABOLIC RATE ($CMR0_2$) REDUCTION HYPOTHESIS

Early in vitro studies demonstrated that lignocaine in high concentrations reduced the oxygen consumption of rat brain cortex[87] and porcine brain mitochondria.[88] In an important in vivo experiment, Sakabe et al.[89] administered bolus doses of 3 and 15 mg/kg lignocaine to anaesthetised dogs and recorded reductions of $CMR0_2$ to 90% and 73% of baseline respectively. Maximal reduction of $CMR0_2$ coincided with peak lignocaine levels in sagittal sinus blood at 12 and 88 µmol/l for the 3 and 15 mg/kg doses respectively. In a further experiment, the $CMR0_2$ was increased significantly above baseline during seizures induced by a 27 mg/kg dose of lignocaine. Other authors have reported selective activation of hippocampal neurones by large "pre-epileptogenic" lignocaine doses.[90] It follows that the dose response profile of lignocaine in this regard is complex, and that high doses may result in disadvantageous energy consuming seizures.

Astrup et al. investigated the neuroelectric basis for reduction of cerebral metabolic rate by lignocaine.[91] They proposed that, in the healthy brain, the previously demonstrated membrane stabilising property of lignocaine[77] reduced the work of ion pumping and therefore $CMR0_2$. Lignocaine administered to dogs in a dose (160 mg/kg) sufficient to render the EEG isoelectric significantly reduced both the $CMR0_2$ and the cerebral metabolic rate for glucose (CMR_{gluc}). This was attributed to abolition of the metabolic cost of electrical activity, and was proposed to be similar to the effect of barbiturates. Lignocaine also produced a further reduction in $CMR0_2$ and CMR_{gluc} after the EEG had already been flattened with high dose thiopentone. The same was not observed for barbiturate when the drugs were administered in reverse order. Astrup et al. attributed this effect to ion channel blockade, reduced baseline ion leakage, and consequently reduced baseline ion pumping activity. Further investigation of lignocaine as an adjuvant to hypothermia in protection of the ischaemic brain was advocated.

TABLE 2

REPORTED HAEMODYNAMIC EFFECTS OF LIGNOCAINE

Authors	MAP healthy brain	MAP after injury*	ICP healthy brain	ICP after injury*	CBF healthy brain	CBF after injury*
CONVENTIONAL ANTIARRHYTHMIC DOSES OF LIGNOCAINE						
Donegan et al.[92]	Unchanged		Decrease			
Dutka et al.[59]						Increase
Evans et al.[52]		Decrease				
Evans et al.[55]		Decrease		Decrease		
Evans et al.[53]		Decrease		Decrease		
Evans et al.[57]		Decrease				
Johns et al.[93]	Unchanged					
Klein et al.[94]	Increase					
Lescanic et al.[95]					Unchanged	
McDermott et al.[70]		Decrease				
Nagao et al.[67]	Unchanged					Increase
Rasool et al.[66]	Unchanged	Unchanged	Unchanged	Decrease	Unchanged	Increase
Sakabe et al.[89]	Unchanged				Unchanged	
Shokunbi et al.[65]					Unchanged	Increase
Sutherland et al.[64]	Decrease					
Wiklund et al.[96]	Increase					
UNCONVENTIONALLY HIGH DOSES OF LIGNOCAINE						
Astrup et al.[91]	Decrease				Decrease	
Evans et al.[54]		Decrease		Decrease		
Lescanic et al.[95]					Decrease	
Milde and Milde[97]					Unchanged	
Sakabe et al.[89]	Decrease					
Shokunbi et al.[68]	Decrease					
Tommasino et al.[90]					Decrease	

* = compared with control animals not receiving lignocaine

Although the high lignocaine doses used in Astrup's study are not clinically relevant[91] a similar action at lower doses is possible. This may explain the significant reduction in $CMR0_2$ achieved at standard antiarrhythmic plasma lignocaine levels in Sakabe's trial.[89] Reduction of $CMR0_2$ by lignocaine may afford clinically useful cerebral protection in ischaemia.[91]

THE HAEMODYNAMIC BENEFIT HYPOTHESIS

Several authors suggest that haemodynamic alterations by lignocaine may contribute to protection of the ischaemic brain.[53,57,59,65-67]

The haemodynamic properties of lignocaine noted by various experimenters are listed in Table 2.[52,53,55,57,59,64-67,89-97]

Lignocaine in therapeutic doses appears to preserve CBF, reduce ICP, and prevent arterial hypertension after brain injury, while having no clear effect on these parameters in the healthy brain. How lignocaine achieves these haemodynamic alterations is unknown.[53] Reduction in mean arterial pressure (MAP) after brain injury may be due to a decrease in plasma catecholamines.[52] This effect may also explain the observation of an intracranial hypotensive effect for lignocaine during endotracheal suctioning,[92] endotracheal intubation (intravenous lignocaine),[98] and craniotomy.[99] Lignocaine has vasomotor effects but its dose/vasoactive response profile in the healthy circulation is complex. Conventional antiarrhythmic plasma concentrations cause vasoconstriction,[93,100] and unconventionally high concentrations cause vasoconstriction,[93,95] and vaso-dilation,[93,101,102] depending on the lignocaine dose and vascular bed studied.

A vasoconstrictive effect by lignocaine may contribute to reduction of ICP after brain injury by doses of lignocaine. However, the concomitant preservation of post-injury CBF despite attenuation of a rise in MAP suggests that cerebral vasoconstriction is not occurring. Alternatively, post-ischaemic preservation of CBF may be explained by a protective effect on cerebral blood vessels,[53] either by membrane stabilisation of endothelial cells or leucocyte deactivation (see later). Data is lacking on post-ischaemic effects of unconventionally high lignocaine doses/plasma levels on CBF, however it is notable that, in uninjured animals, such doses seem to reduce CBF and cause hypotension (Table 2). It follows that high lignocaine doses seem likely to haemodynamically disadvantage the brain.

It is concluded that, while the bases for the various haemodynamic effects of lignocaine are uncertain, these effects may contribute to its cerebroprotective properties. For example, depression of neural function after air embolism has been correlated against reduction of CBF[30] and lignocaine preserves CBF after this injury.[59] It is unlikely however, that haemodynamic alteration, whatever its basis, is the only cerebro-protective mechanism provided by lignocaine. In several studies,[66,67] a neuroelectrical protective effect was demonstrated before any haemodynamic benefit became significant, which suggested another concurrent protective process.

THE LEUCOCYTE DEACTIVATION HYPOTHESIS

The suggestion that leucocytes have an important role in DCI brain injury has been mentioned and is supported by data demonstrating that chemically induced leucocyte depletion preserves cerebral blood flow and function after air embolism in rabbits[32] and dogs.[23] Activated leucocytes have been observed to block microvessels in animal models of DCI[32] and other ischaemic injuries.[103,104] Of critical importance is the observation that leucocytes are activated and cause further damage, after restoration of perfusion to ischaemic tissue, for example following redistribution or resolution of a bubble in DCI, the so called "reperfusion injury".[105] The possibility of injury maturation by leucocyte activity after bubbles have been successfully resolved by HBO therapy is of considerable concern in DCI therapeutics, and it is fortunate that HBO also seems to have a role in reducing leucocyte activity.[36] It is also interesting that lignocaine seems to reduce a variety of leucocyte activities.[85,106-110]

Lignocaine in concentrations higher than conventional antiarrhythmic plasma levels decreases superoxide release,[108,110] oxygen consumption,[108] lysosomal enzyme release,[107] chemiluminescence[110] and bacterial killing[110] by stimulated leucocytes in vitro, and reduces leucocyte adhesion to venular epithelium in vivo.[106] Of particular interest are the findings of Luostarinen et al. who exposed the microvasculature of an everted hamster cheek pouch to standard laser induced injury and observed the rheological effects of topical saline, lignocaine, and other local anaesthetics.[85] When applied at the time of the injury, lignocaine prevented the irreversible thrombus formation which occurred in all control animals. In particular, injury site leucocyte-endothelium binding was markedly reduced in the lignocaine exposure trials. When applied 15 minutes after injury involving invariable thrombus formation, lignocaine produced restoration of flow in five of six trials. Moreover, during restoration of flow, leucocytes were observed to disadhere from endothelium and each other. The local anaesthetics tocainide and bupivacaine, trialled in the same series of experiments, had no antithrombotic effect.

All of these investigations involved exposure to concentrations of lignocaine higher than conventional safe antiarrhythmic levels. Although the actual leucocyte exposure concentration after diffusion in the Luostarinen experiment is unknown,[85] there is clearly doubt regarding the relevance of these results to DCI therapeutics since such plasma concentrations could not be safely achieved in humans. However, in a complex series of in vitro, in vivo, and human experiments using lignocaine in conventional antiarrhythmic concentrations, McGregor et al. recorded reduced leucocyte adherence, reduced inflammation and reduced migration of leucocytes into inflammatory exudate.[109] In the latter role, lignocaine was found to be more effective than methylprednisolone, a result described as "surprising". These authors proposed a possible protective role for lignocaine in myocardial infarction, arthritis, and "other autoimmune reactions". In another clinically relevant experiment, Peck et al.[110] recorded reduced superoxide anion release from human leucocytes exposed in vivo for at least 12 hours to plasma concentrations of lignocaine between 4-20 μmol/l.

The mechanism by which lignocaine modulates leucocyte activity is not clear, but it may involve alteration of cytoskeletal function[109,111] or inhibition of stimulus-response coupling at the cell membrane.[107] The impairment of neutrophil migration to sites of inflammation[109] is intriguing and, given the importance in this process of the CD18 glycoprotein receptor complex on the leucocyte and the intercellular adhesion molecule (ICAM-1) expressed on vascular endothelium,[112] it would be interesting to investigate the effect of lignocaine on expression of these molecules.

Whatever the molecular basis of its effect on leucocytes, lignocaine may preserve CBF in the injured brain by reducing leucocyte adherence to damaged endothelium. Functional protection and reduction of ischaemic damage may follow preservation of CBF. Lignocaine may also provide protection by reduction of cytotoxic/inflammatory substance elaboration by leucocytes.

THE MULTIPLE MECHANISM HYPOTHESIS

It is possible that lignocaine mediates neuroprotection through a combination of the above mechanisms. Indeed, lignocaine would appear to be an "ideal" approach to the biphasic pattern of CAGE injury, protecting neurones by membrane stabilisation or $CMRO_2$ reduction during initial vessel occlusion, and then ameliorating the secondary inflammatory changes after bubbles redistribute.

Discussion

The key questions to be answered in appraisal of the experimental data presented above are:

a are the models relevant to DCI; and
b do the data suggest a protective role for lignocaine and, if so, what is the ideal administration regimen?

None of the in vivo studies presented above involved treatment of decompression injury per se, and no trials of lignocaine were reported in disease states where autochthonous (tissue) bubbles may be contributory. However, a CAGE model was employed in three studies,[55,57,59] and these studies would seem directly relevant to the predominantly vascular mechanism of injury in cerebral DCI.[1] Other data come from animal models of focal and global cerebral ischaemia. These models share with DCI the common mechanism of ischaemic injury to neurones, but the relevance to DCI is not clear since intracorporeal bubble formation is not involved.[62-70] It is concluded that the models in which the protective action of lignocaine has been investigated may be classified as either substantially analogous to DCI,[55,57,59] or at least partially relevant.[62-70]

The clinical relevance of the somatosensory evoked response as an outcome measure in brain injury is sometimes questioned. Dutka et al.[59] conclude that SER recovery is physiologically significant and suggestive of possible functional benefit. This issue is discussed in depth by other authors.[113,114]

The data suggest a neuroprotective role for lignocaine in several forms of ischaemic injury. However, protection is not afforded in all models, and factors which may influence the protective capacity of lignocaine deserve attention. These include: the nature and severity of the injury, the dose and pattern of lignocaine administration and the timing of lignocaine administration with respect to the injury.

The nature and severity of the injury appear to be important determinants of lignocaine's efficacy. Significantly, lignocaine protected neuronal function in all experiments where a CAGE model was employed. There were mixed results in trials involving local and global cerebral ischaemia (Table 1). The two experiments demonstrating no benefit involved a relatively severe model of ischaemia. It is possible that the pool of compromised rather than dead neurones was too small for any intervention to affect outcome. The relevance of the nature and severity of the model to the neuroprotective efficacy of lignocaine has been emphasised by several authors,[65,68,70] with the general conclusion being that lignocaine seems to be most effective in transient and/or incomplete ischaemia, such as seen in CAGE. This may reflect an interim protective capability, for example, by membrane stabilisation,[78] which may be overwhelmed if ischaemia is either too severe or prolonged. Further, in CAGE, the functionally important post-embolic accumulation of leucocytes in the damaged circulation may be ameliorated by lignocaine, and this may contribute to its particular success in this injury.[59]

Both studies demonstrating no neuroprotective properties for lignocaine utilised doses larger than conventional antiarrhythmic regimens, whereas all studies demonstrating protection utilised conventional antiarrhythmic doses (Table 1). This observation may be coincidental. However, the finding that high doses of lignocaine selectively activate hippocampal neurones, increase metabolic stress, and thereby predispose to ischaemic injury,[70,115,116] may be important. Similarly, the consistent finding of CBF reduction with unconventionally high doses of lignocaine (Table 2) suggests that these doses may be haemodynamically disadvantageous as well as clinically impractical.

The pattern of lignocaine administration (ie. bolus only vs bolus plus infusion), may be an important determinant of efficacy. Several trials of lignocaine in ischaemic brain injuries have been performed where animals have been allowed to survive, injuries allowed to develop over seven days, and where outcome has been determined by histological examination of the brain.[64,69] One study found a marginal protection,[64] and the other found no protective effect[69] In both of these studies the animals were given a bolus dose of lignocaine only, indicating that the experimenters assumed lignocaine would exert no effect on the injury maturation process. Given that maturation of the lesion would involve a reperfusion injury with ongoing inflammatory changes mediated largely by leucocytes,[105] and given that lignocaine reduces the inflammatory activities of leucocytes,[106-110] the failure to administer an ongoing lignocaine infusion in these models seems to be a methodological flaw. Even in shorter term experiments, a possible decrement in protective effect as plasma lignocaine levels fall after the bolus (such as demonstrated with $CMR0_2$ reduction[89]) would suggest that a bolus plus infusion regimen is the most appropriate for assessing protection.

Finally, although the effect of significantly delayed administration of lignocaine after ischaemic brain injury is not addressed by any of the studies, it must be assumed that the possible benefit will decline as delay increases. In all of the in vivo experiments described, lignocaine was administered either before or within the 10 minutes following injury. There is no indication of maximum administration delay before protective effect would decline or be absent altogether. It can be hypothesised that protection on the basis of membrane stabilisation and prevention of ischaemic ion fluxes would require either prophylactic or immediate post injury administration. Protection by modulation of injury maturation may be afforded by delayed administration, but no data exist to support or refute this theory.

On the basis of the experimental data reported it is hypothesised that lignocaine, given in a conventional antiarrhythmic bolus plus infusion regime, beginning as soon as possible after the onset of neurological DCI and continuing for a period of at least 48 hours, may provide additional clinical benefit to standard HBO treatment.

Current investigations

A randomised, double blinded, controlled trial of lignocaine in prophylaxis of embolic brain injury in valve replacement cardiac surgery patients has been initiated at Greenlane Hospital, Auckland, New Zealand. This patient population has been chosen because of the significance of the problem in its own right, the pathophysiological similarities to CAGE in divers and the comparative methodological ease with which the population may be studied. Patients 20-70 years old undergoing valve replacement have preoperative neurological examination and comprehensive neuropsychological testing. At surgery a blinded infusion of lignocaine (in standard antiarrhythmic doses) or saline is initiated before cardiopulmonary bypass, and continues for 48 hours. A colour flow Doppler interfaced with an emboli signal counting microprocessor is used to quantify emboli activity in the right carotid artery during surgery. The neuropsychological examination is repeated at 8 days, 8 weeks, and 6 months after surgery. Postoperative decrement and recovery in the two groups will be compared. Twenty three patients had completed the in-patient portion of the protocol, since its inception in December 1994, by May 1995.

A protocol has been generated for a randomised, double blinded, controlled trial of lignocaine as an adjunct to HBO in the treatment of DCI. This awaits completion of the heliox trial at the Royal New Zealand Navy Hyperbaric Unit and other Australasian Hyperbaric Units.

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This paper was accepted as the required thesis for the award of the Diploma of Diving and Hyperbaric Medicine to Dr Mitchell.

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PROVISIONAL REPORT ON AUSTRALIAN DIVING-RELATED DEATHS IN 1992

Douglas Walker

Introduction

Of the 18 deaths in 1992 four were breath-hold (snorkel) divers, nine used scuba, four had a hose supply (one was using a cylinder air supply while the others were compressor (hookah) supplied) and a single diver was using a rebreather set.

Medical conditions were recorded for three of the breath-hold, five of the scuba divers and two of those using hookah. However in several instances the findings were either incidental or possibly so. In only one was a possible missed medical diagnosis apparent and it cannot be known whether the patient gave a full history to her doctor (case SC 92/8). The asthma factor has uncertain significance in case H 92/2 as there were significant other factors (fatigue, cold, rough water, inexperience) present.

Trauma to the head was a factor in two cases, a breath-hold diver and a military diver, and general trauma dramatically ended the life of a hose supplied diver in a dam.

Breath-hold diving deaths

BH 92/1

Because the sea was too calm for them to go surfing or fishing the two friends decided they would go diving. Although the victim was to be snorkelling, something he did infrequently, he was not a spear fisherman. The victim's friend (buddy) was to be using scuba. He was trained but had made no dives in the previous 12 months. They entered the water from rocks and swam out a short distance before the buddy descended leaving the victim at the surface. He was very surprised when, about 5 minutes later, he saw his friend lying on the sea bed. as he did not consider him capable of swimming to that depth (5 m). The victim was without his mask, snorkel and fins and it was apparent that he was unconscious. The buddy described trying, unsuccessfully, to give him air from his regulator and then pulling him up to the surface after partly inflating his buoyancy vest.

The buddy managed to bring the victim back onto the rocks and was then assisted in giving CPR by other people. This was unsuccessful. There was a suggestion that they had been attempting to share the buddy's regulator underwater but the autopsy revealed no evidence of pulmonary barotrauma.

It is not known why the victim lost all his equipment but retained his weight belt. One witness stated his belief that the victim had been in poor health recently, a result of his life style, but there was no comment by the pathologist bearing on this possible factor. Why he drowned in calm water is an unanswerable question as the event was not witnessed.

AVERAGE ABILITY BREATH-HOLD DIVER. FOUND ON SEA BED BY SCUBA BUDDY. POSSIBLE DRUG RELATED ILL HEALTH. NO EVIDENCE THAT HE SHARED AIR WITH BUDDY. NO INQUEST.

BH 92/2

According to his wife this man liked to dive solo in rough water and to surface near rocks, but "always" had someone diving with him. This dive was an exception. His wife raised the alarm when he did not return at the expected time and she found his car still parked where he had left it when he went diving. The sea was described as being quite rough and choppy. A search was made using the surf club inflatable. After some time they located the victim's weight belt and catch bag, but an attempt to retrieve the belt failed due to the rough sea conditions. His body was found floating next morning.

At the autopsy a fracture of the right mid temple area of the skull was noted, so it was concluded he had hit his head on a rock and drowned while dazed or unconscious. There is no information in relation to his breath-hold diving skill.

EXPERIENCED BREATH-HOLD DIVER. SOLO. LIKED ROUGH WATER NEAR ROCKS. NO BUOYANCY VEST. WEIGHT BELT OFF WHEN FOUND. HEAD TRAUMA. NO INQUEST.

BH 92/3

No information is at present available other than the cause of death was coronary insufficiency, a 60% obstruction of the (dominant) left coronary artery. This was diagnosed as having caused vagal inhibition, followed by drowning.

SNORKELLING. CORONARY ARTERY ATHEROSCLEROSIS PROBABLE FACTOR IN DROWNING. NO INQUEST.

BH 92/4

The skipper of a charter boat was a trained scuba diver but much preferred to go breath-hold spearfishing. He had taken a group of divers out to the Barrier Reef. When the scuba divers returned he asked one of them whether he had seen any fish, and was told there was a nice sized one near to the boat. His wife, who assisted with the running of the boat, was aware of his keen desire to do some spear fishing and encouraged him to go for a dive with this man, also a breath-hold spear fisherman.

The skipper entered the water wearing a short wet suit, fins, mask, weight belt. He had a spear gun but not the catch bag he usually carried. No reason for this omission was suggested. The buddy saw him spear the fish he had been told about, then stuff it down the front of his wet suit. The buddy had to help zip him up again. He was later seen to spear another fish before the buddy lost sight of him. The buddy suffered cramp and allowed himself to drift back to the boat. He was apparently a little anxious at being unable to see the victim at the surface, so asked the man in a dinghy, who was watching over the snorkellers, to make an unobtrusive search of the surface around the boat. This was unsuccessful but no great alarm was felt as the victim's wife stated that it was his custom to get involved in his diving and stay away till he had had enough. However his continued absence ultimately became too long and it was realised this was no ordinary absence. A radio call was made and a formal search was initiated.

His weight belt and discharged spear gun were found next day, about 2 m from an area of damaged coral, but no trace of the missing man was ever found. Although he had a long history of right temporal lobe epilepsy this explained neither the ditching of his weight belt nor the failure of the body to float. No sharks were seen in the area at the time of the dive or in the following week so this disappearance must remain a mystery.

EXPERIENCED BREATH-HOLD SPEAR FISHERMAN. CALM SEA. SEPARATION AS BUDDY HAD CRAMP AND LEFT THE WATER. NO CATCH BAG SO FISH PLACED INSIDE WETSUIT. NO BUOYANCY VEST. BODY NEVER FOUND. HISTORY OF TEMPORAL LOBE EPILEPSY.

Scuba diver deaths

SC 92/1

The instructor was taking 4 pupils, including the victim, on their first open water dive. On the sea bed, at 6 m, each was required to demonstrate mask clearing after the instructor lifted the mask to partially flood it. When the victim had partly cleared his mask he suddenly began to make a panic ascent, a little delayed by having the instructor holding on to one of the shoulder straps of his buoyancy compensating device (BCD). As soon as he

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and experience		Dive	Dive	Depth m (ft)		Weights	
		Victim	Buddy	group	purpose	Dive	Incident	On	kg (lb)
BH 92/1	23	No training Some experience	Trained Some experience	Buddy separation before incident	Recreation	5 (15)	Not stated	On	2.5 (5)
BH 92/2	59	No training Experienced	Not applicable	Solo	Spear fishing	Not stated	Surface	Off	Not stated
BH 92/3	61	Not known	Not known	Not known	Recreation	Not known	Not known	Not known	Not known
BH 92/4	45	Trained Experienced	Training not stated Experienced	Buddy separation before incident	Spear fishing	Not stated	Not stated	Off	4 (9)
SC 92/1	27	Some training No experience	Trained Experienced	Group separation during incident	Class	6 (20)	6 (20)	Buddy ditched	10 (22)
SC 92/2	27	Trained Experienced	Trained Experienced	Group separation before incident	Recreation	46 (153)	46 (153)	On	Not stated
SC 92/3	15	Trained Some experience	Trained Experienced	Buddy separation before incident	Cray fishing	9 (30)	9 (30)	On	10 (22)
SC 92/4	18	Some training No experience	Trained No experience	Buddy separation before incident	Army test	3.6 (12)	3.6 (12)	On	10 (22)
SC 92/5	54	Not known	Not known	Not known	Recreation	Not known	Not known	Not known	Not known
SC 92/6	29	Trained Experienced	Trained Experienced	Buddy separation before incident	Recreation	56 (183)	Not stated	On	5 (11)
SC 92/7	54	No training No experience	Trained Experienced	Group no separation	Resort	5 (17)	5 (17)	Not stated	Not stated
SC 92/8	41	Trained Some experience	Trained No experience	Group separation before incident	Recreation	3 (10)	Not stated	On	Not stated
SC 92/9	63	No training No experience	Not known	Not known	Recreation	Not known	Not known	Not known	Not known
H 92/1	28	Trained Experienced	No training No experience	Buddy separation before incident	Recreation	10 (33)	10 (33)	On	4 (9)

DIVING-RELATED DEATHS IN 1992

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	
None	Not applicable	Not applicable	Not applicable	Own	Possible ill health. Buddy had scuba. Unproven buddy breath and CAGE.
None	Not applicable	Not applicable	Not applicable	Own	Rough, choppy water near rocks. Possibly hit head.
Not known	Not applicable	Not applicable	Not known	Not known	Coronary insufficiency.
None	Not applicable	Not applicable	Not applicable	Own	Ditched weight belt and spear gun. Temporal lobe epilepsy. Body never found.
No infomration	Yes	Yes	No fault	Dive shop	Panic ascent. Drowned at surface. Tuberose sclerosis. Instructor's action criticised.
No information	Yes	Yes	No fault	Own	Wreck dive. Recent advanced course. Trio group. Separation at start of ascent. Nitrogen narcosis. Pneumothorax and CAGE.
No information	Yes	Yes	No fault	Own	Night dive. Lost inside groyne.
No information	Yes	Yes	No fault	Army	Night dive. Buddy line came off. Sudden ascent. Cold. Ditched backpack but not weights. CAGE.
Not known	Not known	Not known	Not known	Not known	Heart attack. No inquest. No further information available.
No information	Yes	Equipment lost	No test	Hired	Drift dive. Vertical currents. Recent advanced diver course. Body never found.
No information	Yes	Yes	Not known	Hired	Past history of liver disease. Said to be fit. Cerebral haemorrhage.
No information	Yes	Yes	No fault	Borrowed	Unrecognised heart symptoms before dive. Probable cardiac death.
Not known	Not known	Yes	No fault	Dive shop	Apparently fit. Dissecting aneurysm after dive.
Not known	Not applicable	Not applicable	Faulty	Own	Off work with back pain. Intake hose fault. Carbon monoxide poisoning.

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and experience		Dive	Dive	Depth m (ft)		Weights	
		Victim	Buddy	group	purpose	Dive	Incident	On	kg (lb)
H 92/2	36	Trained Scuba experience	Trained Some experience	Buddy separation during incident	Cray fishing	10 (33)	10 (33)	Not stated	12 (26)
H 92/3	49	No training Some experience	No training Experienced	Buddy separation during incident	Cray fishing	10 (30)	10 (33)	On	Not stated
H 92/4	20	Trained Some experience	No training Experienced	Buddy separation during incident	Work	3 (10)	3 (10)	On	11.5 (25)
RB 92/1	33	Trained Experienced	Trained Experienced	Group no separation	Army exercise	Not stated	0.5 (2)	Not known	Not known

reached the surface he tore off his mask and spat out his regulator. When the instructor tried to restrain him he broke free and sank. The instructor reached him at one metre depth. He refused the offered regulator and struggled free again, managing to regain the surface. Here he failed to respond to the instructor's calls to inflate his BCD. Instead he grabbed the instructor, then clung onto the marker buoy carrying the diving flag. This lacked sufficient buoyancy to support him and his head went underwater.

Although the instructor claimed to have managed to ditch the victim's weight belt and BCD the victim lost consciousness. These events were observed by another instructor from the shore who recognised the need for intervention. He swam out and began giving in-water EAR while towing the victim back to the beach. He later made highly adverse comments on the manner in which the victim's (inexperienced) instructor had shouted advice to his panicking pupil from a distance and failed to inflate the latter's buoyancy vest or ditch his weight belt.

SCUBA COURSE. SECOND SEA DIVE. POOR SWIMMER. ANXIOUS UNDERWATER. SUDDEN PANIC ASCENT DURING MASK CLEARING EXERCISE. CRITICISM OF MANAGEMENT BY INSTRUCTOR. REGURGITATION OF FLUID MADE EAR DIFFICULT. INCIDENTAL FINDING OF TUBEROUS SCLEROSIS.

SC 92/2

As there were seven divers on a charter boat they decided to form two buddy pairs and a trio group. The victim was a member of the threesome. All were reputedly experienced divers but their actual experience of deep dives is not known. They were to be diving on a wreck, maximum depth 46.7 m (155 ft). Conditions were good and the interest in underwater photography of several of the divers was naturally associated with a relaxed interpretation of dive discipline. At the end of their planned bottom time the trio group returned to the anchor line and started their ascent. At the decompression bar the leader of the trio checked and recognised the diver nearest him and saw there was a third diver further away. He incorrectly assumed this diver was the other member of the trio

The last pair to ascend looked down while still close to the bottom and saw a solo diver lying motionless on the sea bed. They descended to investigate and found he was unconscious, had blood in his mask, and was not breathing, so started to bring him up. They were observed by the ship's skipper, who was making a bounce dive to release the anchor. This was part of the agreed dive plan. He assisted them and took the victim the last part of the ascent while the others made their decompression stop. There was no response to resuscitation efforts, which were made difficult by the blood and vomit coming from the victim's mouth.

The autopsy showed that he had suffered a right sided pneumothorax and a cerebral air embolism, findings indicative of a severe barotrauma The equipment was examined and appeared to be in good condition. His tank still had 80 bar of air. Although close to others he was not closely observed at the critical time so the reason why he suffered this fatal barotrauma cannot be known.

EXPERIENCED SCUBA DIVER. UNKNOWN DEEP DIVE EXPERIENCE. GROUP OF THREE

DIVING-RELATED DEATHS IN 1992

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	
No	Not applicable	Not applicable	Minor faults	Borrowed	Unfit. History of asthma. Rough seas. Inadequate hookah air supply. Subarachnoid haemorrhage. Drowned on surface.
No	Not applicable	Not applicable	No fault	Borrowed	Unexplained sudden ascent. Myocarditis or sarcoidosis. Symptoms suggest of CAGE.
No	Not applicable	Not applicable	Minor faults	Work	Attempting to find and close a hole in a dam wall. Sucked into the hole.
No information	Not applicable	Not applicable	No test	Army	Hit by propellor of following boat's outboard motor during exercise.

DIVERS. DEEP WRECK DIVE. GOOD VISIBILITY. NITROGEN NARCOSIS. UNRECOGNISED SEPARATION AT BEGINNING OF ASCENT. BUOYANCY VEST NOT INFLATED. ADEQUATE REMAINING AIR. PNEUMOTHORAX AND CAGE AT DEPTH.

SC 92/3

Danger is always present while diving and safety requires a constant awareness of potential dangers. These two youths failed to take into account the dangers of making a night dive in the interior of a groyne constructed from rocks of various sizes. Over time the filling had washed away and left passages between the larger stones, fine places for crayfish. Although they had hunted here previously this was apparently their first night time search. The victim had been trained for 10 months, his friend for 3 years.

Each had a torch when they entered the water near the end of the groyne. Naturally they soon became separated as they searched for crayfish. After a time the buddy's torch began to fail and he left the water, returned to the car and waited for his friend to return. He gradually grew increasingly anxious at the non-appearance of his friend and after about three hours spoke to a security guard patrolling the area. The police were notified and the police divers arrived. He offered to assist the search and re-entered the underwater passages after putting new batteries in his torch.

The buddy found his friend at the end of a blind passage, minus his mask and obviously dead. The victim's tank was empty. The buddy managed to pull the body a short distance then the police completed retrieval of the body. The passages were deemed too dangerous for there to be any prolonged search for the missing mask, torch, and cray hook. The victim's depth gauge showed a maximum depth of 8.5 m and it is assumed this was during his last dive. It is unknown if either had made any previous night dives.

TRAINED. LIMITED EXPERIENCE. NO TRAINING IN CAVE OR WRECK DIVING. NIGHT DIVE. CRAY FISHING. SEPARATION TO ENTER PASSAGES IN GROYNE. LOST WAY IN PASSAGES. RAN OUT OF AIR.

SC 92/4

As part of their training the army apprentices were given experience of several stressful situations, these included abseiling and scuba diving. Although they were given a little more instruction than for a Resort Dive Experience, they were to be more stressed. They received a lecture and a pool session and then were ordered on a night dive in still water above a weir on the river. Those who attempted to opt out were told they could do so but would have to perform the dive at a later occasion. Though the officer in charge had the assistance of an experienced diving instructor, the latter was not in a position to alter the plan of the dive despite suggesting a daytime alternative. This was not the first group to undertake this dive protocol and nothing untoward appears to have occurred during those dives.

There were not enough wetsuits so only the first few divers had dry ones to put on. It was a cold night but bonfires were lit to keep the waiting and post-dive

apprentices from getting too cold. The medical orderly was instructed to watch for signs of hypothermia in any of those present.

The plan was for trained divers to guide the novices, attached by a buddy line, round an underwater course marked by a sunken rope. The free outer end of this buddy line was attached to a float, carrying a light so that those ashore could monitor their progress. None of these guides had any diving experience other than their initial scuba course, except for some who had acted as guides on a previous dive similar to this. The dive appeared to be proceeding normally until they was more than half way round the course. Then the buddy became aware that the recruit was no longer attached to him. There had been no indication that separation was about to occur, no behaviour suggestive of stress, no jerking of the line to indicate the victim was trying to remove it from his wrist. Visibility was poor, this being night time, so the buddy surfaced after a quick look around underwater.

He found the victim at the surface, obviously panic stricken. After inflating his own buoyancy vest he tried to calm the victim but found himself at risk of being drowned as the victim climbed onto him. He was unable to inflate the victim's buoyancy vest and the victim was not capable of following instructions to ditch his weight belt, inflate his vest, or replace the regulator in his mouth. The buddy retrieved him at least once after he submerged but then lost contact and when he surfaced again the victim was no longer there. He swam to the bank to give the alarm, then swam back and dived, happening to come into contact with the victim, who he managed with difficulty to bring up and get to the bank. His weight belt was in place but his mask and back pack were missing. There was no surface safety boat as this was considered a possible risk to the divers.

Resuscitation efforts were vigorous but the victim died some hours later, in hospital. The autopsy showed aspiration of vomit, a pneumothorax, surgical emphysema, and air in the superior vena cava. It is probable that pulmonary barotrauma occurred during the victim's ascent, followed by inhalation of water, but that the damage was greatly increased by the resuscitation procedures. In addition to the factors of gross inexperience, natural anxiety, the cold water and night conditions, the victim was almost certainly overweighted. The equipment was seemingly provided on a de facto one-size-suits-all basis. The behaviour of the buddy was highly creditable and beyond what could reasonably have been expected.

RESORT DIVE TYPE SITUATION. FIRST OPEN WATER DIVE AFTER ONE POOL DIVE. NO-CHOICE DIVE. ANXIETY. COLD. NIGHT. INEXPERIENCED DIVER AS GUIDE. UN-NOTICED SLIPPED BUDDY LINE. SURFACE PANIC. VALIANT BUDDY ATTEMPT TO RESCUE. FAILED TO DROP WEIGHTS. IN ERROR DITCHED BACK PACK UNIT. EXCESS WEIGHT. PNEUMOTHORAX AND CAGE. VIGOROUS RESUSCITATION EFFORTS MAY HAVE INCREASED AIR ENTRY INTO TISSUES.

SC 92/5

No details are available as no inquest was held. The autopsy found a small myocardial infarct involving the right ventricle and extending into the right auricle, and the left coronary artery was found to be almost completely obstructed near its origin.

ACUTE MYOCARDIAL INFARCTION.

SC 92/6

After completing an Advanced course with a total experience of eleven (11) dives, she joined a diving trip on the Barrier Reef. During this she made several successful dives, including one drift dive. On the fatal day, with 23 dives to her credit, she was the least experienced diver on the trip, though regarded as competent. It was to be a drift dive in a channel which was up to 56 m (185 ft) deep, with a 5 knot current and known to have vertical currents of water from run off from the adjacent reef. Although, for safety reasons, she had previously maintained hand contact with her buddy she decided this would not be necessary for this dive.

The plan was for a group dive to go no deeper than 20-30 m (65-100 ft) and to surface after 20 minutes or when the current petered out, whichever was sooner. Water entry was to be in rapid succession, and separation inevitably occurred. The divers formed two groups and as each assumed the victim was with the other group her absence was not noticed until they were picked up at the end of the dive. The down draft of water affected at least one of the divers and it is assumed that the victim was similarly taken deep and events became more than she could control. Her body was never found. Afterwards the diver in charge admitted that during this trip several of the group had deliberately dived deeper than the dive plan permitted. This was not a factor in this tragedy but indicated the problem of enforcing a dive plan. However a wise instructor will not take those not known to him to a location deeper than their permitted maximum depth.

RECENTLY TRAINED. SECOND DRIFT DIVE. ADVISED TO DIVE AT LESS THAN WATER DEPTH. DECIDED NO NEED FOR DIRECT CONTACT WITH BUDDY. GROUP DIVE. SEQUENTIAL WATER ENTRY. SEPARATION. DOWN DRAFT CURRENTS. BODY NEVER RECOVERED

SC 82/7

The victim signed on for a Resort Dive Experience on the Barrier Reef. He told the instructor in charge of

proceedings that he had already made three similar dives. All intending divers were given a basic talk on scuba diving before being kitted up, checked, and allowed to enter the water. The instructor took three for a scuba dive and descended to about 5 m (16 ft) without incident. Then he noticed that the victim "looked unusual", his mouth was open, the regulator hanging free and he was moving away from the group, one side of his body apparently not functioning. The instructor immediately brought him to the surface and began attempting to resuscitate him.

The victim was evacuated to a shore hospital but died there 2 days later. The autopsy confirmed that he had suffered a large right sided cerebral haemorrhage, though his death was due to a basal pneumonia. Only a limited health history was obtained, that he had been taking unnamed tablets for swollen legs and had previously suffered from some kind of liver disease.

RESORT DIVE. MASSIVE CEREBRAL HAEMORRHAGE. RAPID INSTRUCTOR RESPONSE.

SC 92/8

At the time there was no reason for anyone to anticipate that this would be other than an normal dive. The victim had passed a Dive Medical examination 13 months before, later taking a basic scuba course. This had been completed three months before this dive, and she had made several dives after her course. In the interval between her medical and commencing her course she had apparently suffered some symptoms of breathlessness and chest pain. These symptoms had been present at the time of her course and since. She had apparently reported these symptoms to her doctor but seemingly made them seem more minor than was very probably the case. None of her friends questioned her fitness.

Three divers snorkelled out from the shore together. Only now did the victim notice that she had forgotten her weight belt, so the other two divers waited until she had retrieved it from the shore. They then made an uneventful 30 minute dive and surfaced. After a discussion they opted to dive again to use up their remaining air. The water was shallow but the sea was choppy and visibility had fallen to only 4-5 m (13-16 ft). The victim became separated from her companions during this dive and was not in sight when they surfaced. After a short search she was found on the sea bed nearby. They started resuscitation as they brought her back to shore and it was continued during transport to hospital where she was formally pronounced dead.

Autopsy showed fairly severe atherosclerosis but there was no clear evidence of any recent myocardial infarction. The cause of death was given as cardiac disease, a diagnosis in accord with the incident history.

TRAINED. EXPERIENCE NOT STATED. ONSET OF UNDIAGNOSED CARDIAC SYMPTOMS AFTER DIVE MEDICAL BUT BEFORE TRAINING COURSE. TRIO GROUP. SEPARATION. SOON FOUND. WEIGHTS ON. BUOYANCY VEST NOT INFLATED. VALIANT RESPONSE BY NEWLY TRAINED BUDDIES. X-RAY FAILED TO SHOW APICAL EMPHYSEMA CHANGE. PROBABLE RECENT MYOCARDIAL INFARCTION. NO INQUEST.

SC 92/9

During a tourist trip to Australia this man included a trip to the Barrier Reef to dive. His dive was without adverse incident but later, on the boat, he complained of a severe chest pain and he was taken to a shore hospital. He died there about 8 hours from the onset of his pain. A ruptured aortic aneurism was found at the autopsy. No history of previous ill health was obtained.

RESORT DIVE. POST DIVE DISSECTING ANEURISM. HEALTH HISTORY UNKNOWN.

Hose supplied diver deaths

H 92/1

The victim was owner of the boat and the compressor system (hookah). He was reported to have obtained diving instruction some time in the past. He had years of diving experience and for this reason had been asked by other residents in the caravan park to take them diving. One of those with him for this dive had been shown how to use hookah in the caravan park pool, the other being content to watch because he had made some scuba dives in the past (though never used a hookah) so considered he had no need of this "training".

Three of the four rubber engine mounts for the compressor were broken so it tended to move around when it was working. The air intake hose was fixed to the middle of the windscreen but it was not only worn and had small holes but was liable to come free from the compressor. The boat was anchored so rode with its bow to the wind, but there were exhaust fumes in the stern area. It is probable that the air supply smelled "dirty" but the victim was used to the conditions and the other two accepted this as they knew no different. There were two hoses and the three were to take turns in using them.

The one whose instruction had been a single period in the pool, became panicky on his first dive, but later swam about with a spear gun using the hookah supply. After one dive the victim surfaced and asked for his power head as he had seen a wobbegong shark and wanted to kill it. Many would deprecate such an action. A short time later he was seen from the surface lying on the sea floor, motionless. It required the combined efforts of the two

remaining divers to pull him to the surface using his air hose.

Tests showed that the mask was ill fitting, letting in water so requiring frequent clearing, and that the air the compressor provided was heavily contaminated with carbon monoxide. This was present even with the intake pipe attached but was naturally worse if it was detached. The regulator was defective, giving a fine spray of water with each inhalation. Resuscitation attempts were unsuccessful.

TRAINED DIVER. POORLY MAINTAINED HOOKAH. BUDDIES BOTH UNTRAINED. WATER LEAKS MASK AND REGULATOR. CARBON MONOXIDE POISONING.

H 92/2

This was a dive which never should have happened. The diver who owned the boat and the hookah had taken a scuba course 8 years before, overseas, and admitted that he had never understood diving tables. His experience using hookah is unknown. The equipment was 18 months old and had not been serviced for 12 months. He is called "the buddy". The victim had also trained overseas, in 1983, and had made over 200 dives subsequently. He had a history of asthma, once requiring hospitalisation, and had been suffering a sinusitis problem during the 2 weeks preceding this dive. It is not known whether he told the doctor who performed his diving medical about his asthma history. The third member of the group had made a single scuba dive with a friend some 9 years previously and this was his first use of hookah apparatus. He is called "the novice".

The buddy had heard in a pub that there was a good supply of abalone and crayfish in a rocky area some distance away. That it was a marine reserve did not trouble him. The trip out there was cold and rough and their success in the hunt is uncertain, as later search found nothing and the survivors naturally claimed they had caught nothing. Their compressor had a capacity to supply two at a maximum depth of 18 m (60 ft) if two hoses were used, but they had a single hose ending in a T-junction to which were connected hoses to the divers regulators. These hoses were secured by rope to the weight belts. The novice seems to have managed well on his first dive, correctly accepting the signal to ascend given by his buddy when the latter began to find his air supply inadequate. During this ascent the novice realised he was short of air also, and felt he was lucky to reach the surface. It is probable they had been deeper than 18 m (60 ft), but neither depth nor time was measured at any time.

They took turns in diving, one remaining in the boat while the other two dived. On his third dive the novice found his weight belt had come loose without his knowledge, this becoming apparent when the regulator was pulled from his mouth unexpectedly. He was fortunately a very level headed person and remembered hearing, some 9 years before, about the need to exhale during ascent. He made a successful but worrying ascent thanks to this information and his breath-hold diving experience. The belt had come loose, but not fallen off, once previously. He decided this was his last dive !

The fatal dive proceeded without incident, the victim wearing the weight belt which the novice had just used plus a second belt, as his new wet suit was thicker that those worn by the others. After about 15 minutes they noticed the underwater current had now become far stronger, so decided to ascend. They found the surface conditions had also deteriorated. The buddy managed, with the help of the novice, to pull himself back to the boat along the air hose. He looked back once and saw that the victim was making no effort to return to the boat, merely holding onto the air hose and keeping the regulator in his mouth. He looked tired. As they were sharing a common hose he was being pulled back towards the boat along with the buddy. After the buddy had been pulled into the boat he looked again and saw that the victim was no longer at the surface. He at first thought the victim had chosen to submerge but soon after he saw his body floating face up and drifting away.

The buddy immediately jumped into the water, but then had to be assisted back into the boat as he had removed his regulator but was still wearing his weight belt. The novice now donned one of the boat's life jackets and effected the recovery. They found it very difficult to pull the victim into the boat. The novice knew little about resuscitation, the buddy even less, and their efforts were not successful. They were unable to summon assistance as they had no radio and it took several hours to return, in the rough conditions, to their starting point, load the boat and start their return drive. They later explained their passing many places from which they could have phoned the police as due to their panic, though this was not unreservedly accepted by the fisheries officers.

Examination of the equipment showed that it could not supply adequate air to two divers at 18 m (60 ft). and it was noted that there was twice the permitted carbon monoxide in the air which it supplied, though neither of the survivors reported suffering any ill effects. The boat was found to fail almost every requirement for safety; out of date flares, no lifebelt, no radio, no light for night time use, no diving flag, they had no charts of the area and it was unregistered.

The pathologist reported that he found some small bubbles in the epicardial vessels and over the surface of the brain. These were almost certainly of no significance. The heart was healthy and the lung findings were consistent with drowning. There was some hypertrophy of the smooth muscle in the bronchioles and some mucus plugs were seen

in peripheral bronchi, but he found no evidence of pulmonary barotrauma. A diffuse subarachnoid haemorrhage was noted, chiefly over the left cerebral hemisphere. Analysis showed evidence of the use of both Ventolin and cannabis but their significance was not discussed. At the inquest the pathologist first stated that the cause of death was primarily barotrauma, though his report noted that he found no evidence of this, secondary to asthma. He later stated the subarachnoid haemorrhage was due to bubbles in the blood but in later correspondence denied making this statement. Regrettably, medical evidence is not infrequently the weakest element in the investigation of the deaths of divers.

EXPERIENCED SCUBA. INEXPERIENCED HOOKAH. COLD. ROUGH WATER. FATIGUE. NO UNDERSTANDING OF DIVE TABLES. HOOKAH PROVIDED INADEQUATE AIR FOR TWO DIVERS AT WORK DEPTH. SURFACE DEATH. ASTHMA HISTORY AND SUBARACHNOID HAEMORRHAGE OF UNCERTAIN SIGNIFICANCE. POST INCIDENT IRRATIONAL BEHAVIOUR BY SURVIVORS.

H 92/3

Three friends were using a hookah to dive for cray fish. It had a single hose ending in a T-piece to which were attached two 6 m hoses, each with a regulator. They took turns to dive in pairs, keeping close together. Suddenly and without warning the victim was seen by his buddy to start to ascend, regulator out of his mouth. He had been behaving normally during the previous 30-45 minutes of the dive. The buddy ascended to join him at the surface to discover the reason for his ascent and found he seemed stressed. He checked to make sure his regulator was working and gave it back to him. The victim began to breath from it in an apparently normal manner and the buddy thought that all was well. But as he was about to descend again he saw the victim was “kicking around” in a purposeless manner and the regulator had fallen from his mouth. It was not retained when the buddy replaced it. The buddy tried to keep him at the surface and pull them both back to the boat by the air hose but lost contact and the victim sank out of sight.

Attempts to pull him back to the surface failed when the hose snagged on the reef. When the buddy descended to free it he found the victim was no longer attached. After about 15 minutes his body was found floating by searchers in another boat. There was no response to resuscitation efforts. He was reported to have done a lot of scuba diving 15 years ago and recently to have renewed his interest. His recent diving history is not recorded and it is not known how much, if any, hookah diving he had done nor whether he had any formal training.

The autopsy appeared to show that he had been a very fit man and no evidence of air embolism or pulmonary barotrauma was found, though the history is diagnostic of CAGE. Histology changes suggested he had myocarditis or sarcoidosis but the story does not support a suggestion he experienced cardiac symptoms. No equipment fault can be blamed as the buddy experienced no problems and later testing found it worked correctly. The 15 years break from diving was probably the decisive factor.

UNTRAINED. NO DIVING FOR 15 YEARS. AMOUNT/TYPE OF RECENT DIVING UNKNOWN. CRAY FISHING. UNEXPLAINED SUDDEN ASCENT. SYMPTOMS OF CAGE BUT NO SIGNS. MYOCARDITIS OF UNCERTAIN SIGNIFICANCE. NO INQUEST.

H 92/4

Once again the dangers of diving anywhere near any underwater outflow from a dam or similar head of water have been dramatically illustrated. A recently completed earth dam was seen to have water gushing out from a hole in its base near a pipe which passed through its base. This pipe had been included in the design at the request of the local authority and construction specifications had been calculated by professional engineers. During construction it had been noticed that this pipe had become deformed by the weight of the dam above it but no significance was apparently attached to this observation.

The first diver to attend found the water was cold and the visibility poor. He was careful to have a line attached to a fixed point underwater, the inner end of the pipe, before beginning sweeps to locate any hole through which water might be escaping. He found a current of water entering holes in the dam floor over the pipe and informed the dam’s owner and the dam’s construction company owner of his findings. At their request he took down an old mattress and had this sucked from his hands into the hole he had found. He approached it carefully, feet first, holding onto his rope and then reported to those ashore that the problem would not he solved by mattresses, that the dam should be allowed to empty and that it was dangerous to any diver. He then left, having used all the air in his tank, complaining of being cold.

Soon afterwards two other divers reached the scene. Through a failure of communications they were not informed that the first diver regarded the situation as dangerous to divers, but they were obviously aware of the need for caution when near where water was escaping through a restricted area. They also took a mattress down and had it enter the hole. A suggestion that hay bales be used met the problem of their refusal to sink. They wore ropes held by men on the inner wall of the dam and carried lines to enable communication. They descended a short distance apart and unexpectedly one received a line call to return. He was informed that his colleague’s line had gone

slack, then too tight for them to move it. He descended carefully, feet first, and when he reached the hole, which had now formed in the base of the dam, he was nearly sucked into it. With the assistance of those holding his line he was dragged free. It was hoped that the missing man might survive. as they were using a hose supply system from a cylinder on the wall of the dam. Hopeful listening made them think he was still breathing, though unfortunately this was not true.

The police were called and more rope attached to the rope attached to the victim, this enabling the direction of pull to be in line with the hole and not down the dam wall and angled back into the hole. A police diver who checked the situation was nearly sucked into the hole although fully aware of the problem and proceeding with great caution.

Although the divers were aware of the danger posed by this escape of water they underestimated its true power. It was later suggested that had the victim carried a knife he could have cut himself free from his line and passed through the passage which had developed under the dam. However, events occurred too rapidly for anyone to react sufficiently rapidly, and passage through the dam would undoubtedly have been too traumatic for him to survive. His death was undoubtedly caused either by the water flow pressing him onto the wall so firmly that he was unable to make respiratory movements of his chest, or he died from the sudden trauma of the water flow.

Poor construction of the dam and inadequate awareness of the dangers were the critical factors. None of the divers who were involved in the attempt to close the leak were registered as commercial divers and only one had any training, a basic scuba course. Though they had a variety of experience in commercial diving jobs, none had undertaken any job like this before. It was noted that the two divers believed they would be likely to lose their jobs if they refused tasks they considered to be unduly risky.

SOME EXPERIENCE BUT UNTRAINED AND UNREGISTERED AS COMMERCIAL DIVERS. DIVE TO PLUG WATER ESCAPE FROM DAM. COLD. POOR VISIBILITY. DIVER AWARE OF DANGER FROM FLOW OF ESCAPING WATER. BUT UNDERESTIMATED THIS POWER. CYLINDER AIR SUPPLY. NEARLY MULTIPLE TRAGEDY. DAM CONSTRUCTION FAULTY.

Rebreather unit diver death

RB 92/1

The operation of Murphy's Law is irresistible and no amount of training, experience and planning can guarantee it will not suddenly influence events disastrously. In this incident a well practiced and strictly controlled procedure was changed into one which resulted in one fatality and two lesser injuries among the group of divers.

This army exercise involved the "recapture" of an oil rig by assault divers. Four boats carrying the divers were to approach in a diagonal array, each succeeding boat astern and to starboard of the boat ahead. In the event one boat became unmanageable for a short period and changed course, over running not only its own divers but those in the boat ahead of it. One diver suffered fatal head injuries from its propeller, one was hit on the foot but was fortunately wearing a hard fin which protected his foot and another was fortunate to hit his head on the relatively soft bottom of the boat as it passed over him.

The critical elements in this tragedy were in themselves harmless. The unit had just been supplied with new, more powerful outboard engines to replace weaker, old and unreliable ones and was the first time they had been fitted. The second factor was that the assault divers had to remove but retain their fins in order to climb (and later leave) the oil rig. For this reason it was the custom to have a stiff loop of cord through each fin so it could be easily carried on an arm. The factor which critically directed the course of events was one never considered by either the designers of the engine nor those who approved its use. It had a T-shaped engine gear lever. As the last diver seated on the starboard side of this boat made his backward roll the loop of one of his fins caught this T lever and twisted the engine from the control of the driver for a critical moment. The change of course proved fatal to one of the divers who had just entered the water from the boat ahead.

TRAINED EXPERIENCED DIVERS. HEAD TRAUMA FROM OUTBOARD PROPELLER. NEW MORE POWERFUL ENGINES HAD T-PIECE HANDLE TO GEAR CHANGE. DIVER'S FIN CAUGHT THIS DURING BACK ROLL WATER ENTRY. CAUSED DRIVER TO LOSE CONTROL.

Discussion

A total of eighteen (18) divers were identified as dying in Australian waters during 1992. Details are still not available for two of these fatalities (one breath-hold, the other a scuba diver), which will be reported at a later date. The critical factors were different in each case but there was an unexpectedly frequent incidence of medical conditions, although these were not, in some cases, necessarily significant in either the initiation or progression of the incident.

In the three breath-hold fatalities where there are details available there was one common factor, the absence of any witness of the actual incident, though in case BH 92/1 there is an element of doubt concerning the buddy's

evidence. This victim's health is believed to have been compromised by drugs. Rough water close to rocks is the assumed cause of death in one person, though there was no evidence the conditions were any worse than he usually liked to encounter. Similarly there is no evidence as to whether, in case BH 92/4, the temporal lobe epilepsy played any part in his death and neither is the suggestion of a shark attack more than a possible explanation for the absence of the victim's body. Though coronary artery disease was found present in case BH 92/3 and was given as the critical factor, details concerning this death are not yet available.

In the eight scuba diver deaths discussed, cardiovascular factors were noted in two, cerebral haemorrhage in one, and tuberous sclerosis was a surprise finding in another. Two night-dive deaths occurred, both with avoidable factors. Not for the last time it was shown that the management of drift dives requires careful thought and that control of a group of divers may be difficult or impossible. Case SC 92/5 was apparently also a coronary artery disease death, however no details of this incident are available at present.

There were four fatalities among those who used hose supply for their diving, the critical factors being different in each. In the first the state of the equipment was such that even after the air intake hose was attached there was too high a carbon monoxide level in the air supplied and when this hose came loose the level was further increased. Only the gross ignorance of the divers can explain their lack of awareness of this fact. In this as in other instances there seems to be an assumption that using a compressor to supply air obviates the need for training (and the application of common sense) on the part of the users. While it is reasonable to assume a dive shop will provide clean air, a similar assumption is unwise concerning compressor supplied air.

The second hookah death was the result of an even worse example of irresponsible behaviour. Not only was the condition of the sea adverse, the chosen dive location a nature reserve and the boat inadequately equipped but one participant, not the victim, was almost totally ignorant of diving. While it is possible that there was some reduction in the victim's survival chances due to his asthma reducing his respiratory efficiency, the effects of cold and fatigue were a significant element in his drowning. It should be noted that when two divers are supplied by a common hose their air supply is very significantly compromised.

No reason can be advanced for the sudden ascent made by the diver in case H 92/3 but he was probably lacking in confidence, having only recently resumed diving after a 15 year break. Though he may not have panicked, his symptoms at the surface are those of classical cerebral arterial gas embolism even though there was no autopsy proof that this occurred.

The force exerted by even a modest height of water was once again tragically demonstrated in case H 92/4. It should be plain that caution is not enough, a diver should maintain a significant distance from any place where water is escaping forcefully and in no instance assume that a rope held by others offers a guarantee of safety.

The rebreather fatality resulted from trauma to the head as an out of control outboard passed over him. The cascade effect of even an apparently minor change in diving procedures or equipment is here strikingly demonstrated. Every change has more than the predicted consequences in any living environment.

Acknowledgment

This report could not have been prepared without the generous help and forbearance of those charged with the management of the documentation concerning such fatalities. This is true of every State and includes the Police service in some States in reference to cases where no Inquest was considered necessary.

Others who have supplied identification of cases or supplied information are also thanked. It is hoped that one day there will be a wider involvement in this project by members of the diving community.

Information and correspondence to
Dr Douglas Walker
PO Box 120
Narrabeen
New South Wales 2101

Continued from page 211

Plans for a Fiordland trip before the conference have fallen through, so the meeting will be preceded by a diving charter in Marlborough Sounds. Space is strictly limited on this diving tour which will be an excellent opportunity for Australian and North Island members to see this lovely part of New Zealand. Bookings will be on a first come basis. Divers wishing to confirm their booking should write immediately to

The Hyperbaric Medicine Unit
Department of Anaesthesia
Christchurch Hospital
Private Bag 4710
Christchurch
New Zealand

enclosing a cheque or bank draft for NZ$200 made payable to "SPUMS NZ 1996".

Other enquiries should also be addressed to the Hyperbaric Medicine Unit.

THE WORLD AS IT IS

AUSTRALIA AND NEW ZEALAND HYPERBARIC MEDICINE GROUP

STATEMENT ON THE USE OF HYPERBARIC OXYGEN THERAPY AT SITES OTHER THAN PUBLIC HOSPITALS

1 Preamble

Periodically, and usually for indications not generally accepted by hyperbaric medical practitioners, enthusiasm is generated in the community for the use of Hyperbaric Oxygen Therapy (HBOT) in locations other than mainstream hospital or Naval facilities. The compression of patients for therapeutic purposes in such out of hospital locations exploits the current situation by which the administration of oxygen is not governed by the therapeutic goods administration acts currently in force in Australia and New Zealand. It is the opinion of the Australia and New Zealand Hyperbaric Medicine Group (ANZHMG) that the practice of HBOT requires regulation to maintain the current standards of safety and appropriate use in the best interests of the community.

This statement outlines the position of the ANZHMG with regard to these matters for the consideration of the various Health Administrations in Australia and New Zealand.

2 Definitions

ANZHMG

The professional body of the trained practitioners of Hyperbaric Medicine in Australia and New Zealand and is at present a sub-committee of the South Pacific Underwater Medicine Society (SPUMS). All presently operating hospital-based and military facilities for the practice of HBOT are represented by this group.

Hyperbaric Oxygen Therapy

The administration of oxygen for therapeutic purposes at pressures greater than one atmosphere. This requires the application of pressure to the body and simultaneous administration of oxygen for breathing. This is carried out in a vessel designed for the purpose called variously a compression, recompression or decompression chamber. Such chambers may be designed for single occupancy or multiple occupancy and have an atmosphere of either air or 100% oxygen. When the atmosphere is air, the patient is required to breathe oxygen (or sometimes other gas mixtures) through a mask or via a hood. Many chambers are designed to operate at a range of pressures as required for the treatment of a variety of conditions.

HBOT is at present carried out in a number of facilities around Australia and New Zealand. Most are officially called *Hyperbaric Medicine Unit*, *Hyperbaric Therapy Unit* or similar and they provide a 24-hour service, commonly in association with the intensive care or emergency medicine departments of major hospitals.

3 Current Situation

Currently there are eight facilities operating in tertiary hospitals around Australia and New Zealand and three operated by the Navies of the two countries. One civilian facility is located in each State of Australia and one in the Northern Territory, while the NZ Navy operates a facility in Auckland and a civilian facility is located in Christchurch. While there are some geographical gaps in coverage, for the most part each State has elected to concentrate resources in these single facilities. Smaller hospitals have chosen not to enter the field both because of the extensive specialist back-up required and the probable under-utilisation of an expensive resource. However technical advances are beginning to lower the capital cost of at least the smaller, monoplace chambers.

4 Position Statement

Physician Requirements

It is the opinion of the ANZHMG that HBOT must be prescribed by a physician with appropriate training in Hyperbaric Medicine. There are two appropriate courses operating in Australia at present, being those at the Royal Adelaide Hospital and at the Submarine and Underwater Medicine Unit at HMAS PENGUIN in Sydney, which satisfy a minimum level of theoretical instruction. At present practical experience is obtained by an informal process through the various facilities. There are many equivalent theoretical courses and training fellowships internationally.

At present the local qualification in the field is the Diploma of Diving and Hyperbaric Medicine (DipDHM) which is administered by SPUMS. The minimum requirements are successful completion of one of the courses noted above, six months supervised training in a registered hyperbaric facility and presentation of a written thesis (accepted by appointed referees) for publication in the South Pacific Underwater Medicine Society Journal.

It may be that for management of specific recognised indications in facilities expressly built for that purpose, a modified curriculum would be appropriate theoretical training. This area is controversial and there are no current plans for the definition of such criteria.

Physicians prescribing this treatment are medically accountable for the safety of the patient and staff involved in the treatment. This requires both a knowledge of the indications, contraindications, side-effects and complications of therapy and the provision of an environment where there is immediate availability of emergency medical skills and equipment sufficient to treat any problems that may reasonably be anticipated. In the field of HBOT, this most definitely includes advanced life-support facilities.

It is important to bear in mind that the staff in such facilities are subject to risk directly as a consequence of compression themselves when acting as medical attendants in multi-place chambers and indirectly by the proximity and operation of equipment requiring the use of high pressure gas supplies.

Chamber Requirements

All chambers operated for the purpose of HBOT must comply with appropriate technical and Worksafe standards. These are currently under extensive review to improve their relevance to hospital practice and the new Australian standard entitled "Guidelines for Clinical Multiplace Hyperbaric Facilities" is, now in its second draft. The current standard is AS2299-1992 Occupational Diving.

The ANZHMG feels that hyperbaric facilities should adhere to the guidelines in this document and make extensive reference to international standards and guidelines until the revised local document is published. The most relevant international standards are Z2751-93 Hyperbaric Facilities (Canadian) and two reports from the safety committee of the Undersea and Hyperbaric Medical Society (UHMS) Monoplace Hyperbaric Chamber Safety Guidelines and Guidelines for Clinical Multiplace Hyperbaric Facilities. The UHMS is the largest international body representing the practice of hyperbaric medicine to which the great majority of local practitioners belong.

Chamber Operator Requirements

Any person charged with the responsibility of operating a vessel for the purpose of HBOT must have had appropriate recognised training in the field. The minimum requirements for such operators in Australia and New Zealand are currently under review by the Hyperbaric Technicians and Nurses Association (HTNA) but may be chamber-specific and less comprehensive than those currently derived from the commercial diving industry. These standards are however currently required for all operators in the facilities previously mentioned in this document.

Chamber Attendant Requirements

In any operation which requires a medical attendant present with the patient(s) in the chamber, such attendants must have appropriate training in the field and be medically fit for compression. The HTNA is about to publish a national curriculum of minimum requirements for such training. Courses are currently offered in a number of the hospital-based facilities around Australia, primarily for the provision of sufficient attendants for those facilities. At present all such attendants are either registered nurses, medical practitioners or Navy trained medics who have satisfied such requirements.

Indications

The ANZHMG believes that treatment should be limited to accepted indications for HBOT and for the proper investigation of potential new indications, ideally through the initiation of appropriate randomised controlled trials after sufficient anecdotal and case-descriptive evidence has been documented to justify such studies. Prior approval by an appropriate ethics committee is mandatory.

Exceptions

The only currently acceptable exceptions to the above principles, in the view of the ANZHMG, are the on-site commercial chambers required for the safe execution of diving and tunnelling operations. Such chambers are operated by technicians with extended training and for specific purposes. They are viewed not primarily as therapeutic vessels but as integral to safe diving operations and for the purposes of on-surface safe decompression schedules. They are regulated by a comprehensive set of standards and legal requirements which are also under review at the present time. In practice, such chambers often maintain a close liaison with their local HBOT units.

5 Conclusions

The ANZHMG accepts that many currently proposed out of hospital facilities will not easily be able to comply with all the above principles. We feel, however, these represent the minimum requirements for the safe and rational use of HBOT. Facilities not meeting the above principles cannot be endorsed by the ANZHMG as being appropriate for the administration of this potentially harmful therapy.

The ANZHMG would be glad to assist in the development of further hyperbaric facilities in the region where there is a desire to establish such safe and appropriate use of hyperbaric oxygen.

6 Addendum - The Treatment of Sports Injuries with HBOT

The ANZHMG supports the investigation of this potential indication for HBOT. It should be stressed that, at this time, treatment of such conditions with this therapy remains unproven. People presenting for HBOT with

sports-related injuries should be made aware of this, be under the care of appropriately trained medical staff and ideally be willing to participate in controlled trials to assess the efficacy of such treatment. At present, the only facilities in a position to do this are the hospital-based facilities in co-operation with those trained in Sports Medicine or related medical practice.

Dr Michael Bennett
Secretary, ANZHMG
October 1995

MEDICAL SUPPORT FOR DIVING OPERATIONS: TRAINING THE ON-CALL DOCTOR

David Elliott

All diving activities need medical support, not only in case there is a medical emergency but also for routine cover, in particular for the assessment of fitness to return to diving after some illness or injury. Arrangements for the provision of medical cover vary around the world but usually there is a local medical service which responds to the local need and this is supported by a distant and more experienced medical service providing consultant advice when it is required. There are many different ways in which these medical services are provided but, whatever their organisation, accountability, resources and regulatory constraints, the supreme need is for the competency of the diving doctor on call.

Even the phrase "diving doctor" can be inappropriate because many diving doctors do not dive. A few may be recreational scuba divers and even fewer may have had commercial or naval mixed gas training but the majority are occupational or family doctors who have no need to dive. Attendance at a one-week introductory course is more than is required in most countries. In contrast, for those who are on-call and responsible for providing medical care in a diving emergency, there are no national requirements and few courses, but specific and appropriate training is essential. For those doctors who not only treat diving illnesses but also advise diving companies or government agencies on aspects of diving health and safety, the opportunities to learn the important subtleties of diving physiology are few.

The Diving Medical Specialist must have received some practical training in order to understand the underwater working environment and its medical emergencies. At an intermediate level, while gaining experience under a consultant, the essential medical skills are those of managing the various medical emergencies in diving. At the consultant level, additional skills include applied physiology, understanding decompression theory and reviewing aspects of operational diving, for example assessing modifications to underwater breathing apparatus. Competencies need to include experience of treating difficult diving incidents and the ability to use applied physiology in the assessment of the divers' working environment and all associated equipment. A postgraduate qualification in occupational medicine is an appropriate foundation for this training but, in practice, accreditation in a major clinical speciality has also proved suitable.

A two-week course is a minimum introduction to this subject and must be supplemented by appropriate and sufficient experience. To complete their training, such doctors should be fit to dive and they must maintain this fitness for compression chamber work for as long as they continue to treat diving casualties.

The first course to focus upon the medical needs of commercial diving was organised in Italy by Shell in 1975. Since then there have been a number of advanced courses in Europe of two weeks or longer to give doctors practical training appropriate for providing emergency medical cover for air and mixed gas diving.

Another such international course is planned and will take place in Fremantle in October 1996. Open to doctors from all corners of the world this course will be the first to be located geographically convenient for those in Asia, Australia and around the Pacific rim. The theme of the first week (30 Sep to 4 Oct) is working dives to 50 metres and that of the second week (7 to 11 Oct, 1996) is working dives deeper than 50 metres. The courses will have practical sessions which, with the support of local and international diving companies and the Fremantle Hyperbaric Unit, will include in-water training and simulated emergencies.

Details are available from Professor David Elliott or from Biomedical Seminars, 7 Lyncroft Gardens, Ewell, Surrey KT17 1UR, England. Fax (44) 181 786 7036. Also see the advertisement on page 264

YET ANOTHER FUSS ABOUT (PROBABLY) NOTHING.

Des Gorman

As much of the Australasian media-attentive population are aware, there has been yet another media release of a study showing that diving, even in the absence of decompression illness (DCI) is injurious to your health. The specific study alluded to here was from a German group and published in the British journal called The

Lancet (Ruel J et al. Lancet 1995; 345: 1403-05). The Lancet is famous for early publication of work, not all of which has scientific merit. Given the attention that this paper has received, it is certainly worthy of commentary .

The basis of the German study was the use of a sensitive imaging technique (magnetic resonance imaging or MRI) to examine the spines and spinal cords of 52 recreational divers and a control population of 50 sports club members. The central finding reported was that the divers had a greater prevalence of both spinal cord changes and deterioration of intervertebral discs. Notwithstanding the observation that the extent of diving exposure (decompression stress) is not well measured in this study (it is actually very difficult to obtain a simple estimate of this phenomenon), three more significant comments are relevant here.

First, the frequency of spinal cord changes demonstrated in the divers is essentially the same as that shown previously in other surveys using MRI in both divers and non-divers. The question here then may be better written as: Why do the control subjects in the German study have such a low prevalence of spinal cord changes ? In their media release in response to the Lancet paper, the Divers Alert Network (DAN) propose reasons for the findings in the German control group. These are conjectural, but do make the point well.

Second, as is true for all of these current "imaging surveys", which include brain imaging and electrophysiological studies, and analyses of retinal blood vessels, there are no data to show that recreational diving, in the absence of an episode of DCI, has any effect on well being or function (professional, domestic, social, biological etc). This is very well described by Professor David Elliott in his textbook (Bennett PB and Elliott DH. Saunders: London, 1993).

Indeed, Professor Elliott spoke to this subject at the 1994 SPUMS Annual Scientific Meeting (ASM)[a full transcript has been published in the SPUMS Journal] and there is nothing in the German paper to change his fundamental observation that the changes shown by sophisticated imaging techniques have not been shown to correlate with actual disability.

Third, the finding of intervertebral disc changes in recreational divers and the claim by the German authors that these may be due to gas microbubbles is implausible. Certainly, these discs are not a recognised target in DCI (even in severe untreated disease). While cervical spine degeneration is seen in fisherman divers (due to the long periods they spend swimming underwater with their neck bent right back or hyper-extended) and often cause them to leave the industry, the time that recreational divers spend underwater is relatively brief. Indeed, the disc changes reported in the German divers, casts doubt on both the selection of the divers and the sensitivity of the MRI technique in this context.

It follows that, on the basis of this German study, that there is no reason for concern about uncomplicated diving. However, to state the obvious, the Society does strongly support the Safe Diving programs of the recreational diving organisations.

A version of this paper has appeared in DiveLog New Zealand in response to many enquiries from divers.

SPUMS NOTICES

SPUMS ANNUAL SCIENTIFIC MEETING 1996

PARADISE ISLAND, THE MALDIVES
20th to 28th APRIL 1996
Theme Technical Diving

The guest speakers will be Professor David Elliott (UK) and Dr Bill Hamilton PhD (USA). Professor Elliott's background is in naval and commercial diving and diving safety as well as co-authoring *The physiology and medicine of diving*. Dr Hamilton is a diving physiologist with special interest in decompression schedules. His advice has been sought, and taken, by many of the growing company of "technical divers" in the USA.

The conveners will be Drs Chris Acott and Dr Guy Williams. Intending speakers should contact Dr Williams at 8 Toorak Street, Tootgarook, Victoria 3941, Australia. Phone (059) 85 7161. Fax (059) 81 2213.

As the hotel can provide no more than 75 rooms, members and associates are advised to book early as late bookings may not be able to get accommodation.

The official travel agents are
Allways Dive Expeditions
168 High Street, Ashburton, Victoria 3147, Australia.
Phone (61) (03) 9885 8863
Fax (61) (03) 9885 1164

S0UTH 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 financial member of the Society.

2 The candidate must supply documentary evidence of satisfactory completion of examined courses in both Basic and Advanced Hyperbaric and Diving Medicine at an institution approved by the Board of Censors of the Society.

3 The candidate must have completed at least six months full time, or equivalent part time, training in an approved Hyperbaric Medicine Unit.

4 All candidates will be required to advise the Board of Censors of their intended candidacy and to discuss the proposed subject matter of their thesis.

5 Having received prior approval of the subject matter by the Board of Censors, the candidate must submit a thesis, treatise or paper, in a form suitable for publication, for consideration by the Board of Censors.

Candidates are advised that preference will be given to papers reporting original basic or clinical research work. All clinical research material must be accompanied by documentary evidence of approval by an appropriate Ethics Committee.

Case reports may be acceptable provided they are thoroughly documented, the subject is extensively researched and is then discussed in depth. Reports of a single case will be deemed insufficient.

Review articles may be acceptable only if the review is of the world literature, it is thoroughly analysed and discussed and the subject matter has not received a similar review in recent times.

6 All successful thesis material becomes the property of the Society to be published as it deems fit.

7 The Board of Censors reserves the right to modify any of these requirements from time to time.

NEW ZEALAND CHAPTER 1996 ANNUAL SCIENTIFIC MEETING

The format for the 1996 NZ Chapter ASM will be different from previous years with no diving component to the weekend, though there are opportunities for diving during the week before. Since this meeting will be hosted by he first formally established civilian Hyperbaric Medicine Unit in New Zealand we hope that SPUMS members will give their strong support to this meeting.

The themes of the meeting will be " Hyperbaric Medicine in New Zealand: Past, Present and Future" and Tek Diving, a new diving illness?

The principal guest speaker will be Associate Professor Des Gorman and it is hoped that the meeting will be combined with the official opening of the Hyperbaric Medicine Unit in Christchurch on Friday April 12th 1996.

Continued on page 205

SPUMS ANNUAL SCIENTIFIC MEETING 1997

The 1997 Annual Scientific Meeting is to be held at the Waitangi Resort, Paihia in Northland, New Zealand from April 13th to 20th.

The theme of the meeting will be "The Pathophysiology and Treatment of Decompression Illness" and the Workshop will be devoted to the "First Aid Management of Diving Accidents".

Confirmed speakers are Dr James Francis, until recently at the Naval Medical Institute, Alverstoke, England and Dr Richard Moon of Duke University Medical Centre, Durham, North Carolina, U.S.A. Both are excellent speakers and acknowledged experts in the field of decompression illness.

The venue is a first class resort hotel immediately adjacent to the historic Waitangi Treaty House and situated right on the foreshore of the beautiful Bay of Islands. There is outstanding temperate water diving in the region, especially at the Poor Knights Islands with water temperatures at that time of the year around 20-22°C. The region is renowned for its sailing and game fishing and there is a huge range of land based activities for registrants and their families.

Conference conveners are Dr Michael Davis, Medical Director, Hyperbaric Medical Unit, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand and Associate Professor Des Gorman, Department of Occupational Health, University of Auckland School of Medicine. Enquiries should be addressed to Mike Davis.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING

held on 24 May 1995
at the FIJI ASM on Castaway Island

Opened 1330 Fiji Time

Present

Drs D Gorman (President), A Slark (Past President), C Meehan (Secretary), S Paton (Treasurer), J Knight (Editor), D Davies (Education Officer), M Davis (Chairperson of the NZ Chapter), C Acott and G Williams.

Apologies

Dr J Williamson

1 Minutes of the previous meeting

Accepted as a true record. Proposed Dr Acott. Seconded Dr Williams.

2 Business arising from the minutes

2.1 More suitable booking form and protocol needed to alleviate the problems encountered with the registration fee and booking fee both being needed for confirmation of travel arrangements but being paid to different agents. Dr Williams and Dr Paton have suggestions to look at and comment upon.

2.2 Budget for the coming year was presented by the treasurer and it was discussed in regard to setting the subscription fees for 1996. It was decided to recommend that full membership fees rise to $90, and that associate membership fee rises slightly to $45 (ie. half the full membership fee).

2.3 Letter in Dive Log and its origin. Dr Gorman will address this and have the article reprinted in its full form.

2.4 Upgrade/replacement of the Treasurer's computer. As the Treasurer's computer was to be upgraded it was decided to change to IBM format. It was also agreed that SPUMS would purchase a computer for the Secretary who at present uses her own computer for SPUMS business.

3 Treasurer's report

Accepted. Proposed Dr Davies. Seconded Dr Knight.

4 1996 ASM Maldives

4.1 Dr Gorman to present from Allways clarification of the following three points in writing:

4.4.1 Extra booking fee to PADI, for Americans. This no longer appears to be an issue.

4.4.2 Renegotiation of excessive afternoon tea costs.

4.4.3 Costing of travel arrangements for the guest speaker on Singapore Airlines versus Air Lanka.

4..2 Timing of the 1996 ASM for April confirmed.

5 1997 ASM:

5.1 Destination for the 1997 ASM discussed and New Zealand suggested. Dr M. Davis to present a report on this to the committee in July. A revisit to Palau was a suggestion for the 1998 meeting.

5.2 That a two staged tender process be looked at. The first stage to suggest suitable venues at the desired destination with skeleton costing only and the second stage to then look at full costing on the agreed upon venue. This will be discussed before the next tender goes out.

5.3 Protocol for advertising for and obtaining of tenders for our ASM travel arrangements discussed.

6 Correspondence:

6.1 Issue re Dr Tim Marks. The Board of Censors to look at the curriculum and then to assess further. Dr Meehan to write to obtain the curriculum.

6.2 Asian Diver: this issue has been dealt with.

7 Other Business

7.1 Letter from Lori Barr and Steve Dent offering resignation as Chairperson and Secretary/ Treasurer of the North American Chapter for personal reasons. Dr Meehan to send them a letter of thanks and to offer suggestions as to possible nominees for the vacated positions.

7.2 Upgrade of monitor and printer for the Editor was approved and printer to be replaced when it next breaks down.

7.3 Payment of magazine subscriptions for the Editor was agreed upon, however it was suggested to try to do journal exchange where possible.

7.4 ANZHMG are writing standards of protocol and training at present.

7.5 Secretarial Service at this stage was not followed up any further.

7.6 S.O.S paper. A donation of $250 was decided on.

7.7 Donation to Fiji Recompression Chamber was agreed on from funds received from ASM registrations.

The meeting closed at 1500.

MINUTES OF THE EXECUTIVE COMMITTEE TELECONFERENCE
on the 16 July 1995

Opened 1000 Eastern Standard Time

Present

Drs D Gorman (President), S Paton (Treasurer), C Meehan (Secretary), D Davies (Education Officer), M Davis (NZ Chapter), G Williams (Public Officer), C Acott, R Walker and J Williamson.

Apologies

Drs J Knight and A Slark.

1 Update on the feasibility of the 1997 ASM to be held in New Zealand

1.1 Drs Mike Davis and Rees Jones visited the Bay of Islands to research the feasibility of the 1997 conference being held there. The Waitangi Resort fulfilled all the requirements needed for venue. Fullers Northland is the main organiser in this area. The diving side could be organised with some co-ordination of the dive operators. There would be some full day diving commitments with educational components on the boat and some full day educational commitments.

1..2 Speakers suggested are Richard Moon and James Francis. The proposed theme of the conference is *Treatment of DCI* and the workshop will be "to produce a coherent SPUMS policy on first aid".

1.3 Modes of transfers to the region were discussed. Possibility of transfer by sea was discussed. Other possibilities included charter flight.

1.4 Water temperature postulated to be around 20°C at the proposed time of the conference. The whole enterprise seemed eminently feasible.

1.5 The committee unanimously voted to go ahead with this option. The timing should be as early in April as possible.

1.6 Dr Davis to be reimbursed for airfare to the site inspection. He suggested opening a special conference account in NZ to facilitate payment of the different components of the NZ 1997 meeting.

1.7 Definite date to be confirmed and an article to be written by Dr Davis for inclusion in the December Journal.

2 Discussion of the registration breakdown of the 1995 ASM Fiji:

2.1 Monies outstanding to Allways from registration fee discussed and the convenor Dr Davies to finalise this.

2.2 Re currency fluctuation shortfall
Final decision of the committee was to pay 50% of the amount in question.

3 Update on 1996 ASM Maldives

3.1 The perceived conflict with the EUBS meeting is avoided by 1 week. Guest speaker airfares are not as yet worked out. Last week Paradise Island told Allways that there will an extra cost because of changing the dates of the meeting to April. However this would not be a great deal for those taking full board and is still to be negotiated. The extra cost for full board would be approximately $10 per day. The site inspection is planned for August 23rd. Bill Hamilton and David Elliott are the guest speakers. It was suggested to redo how we pay for the expenses that come out of the registration fee. An account could be opened especially to hold the registration fees and some of the costs could be paid as they arise. During the week of conference we could pay the resort directly for the expenses covered by the registration fee i.e. afternoon teas and Conference Dinner. This would avoid some of the conflict that has arisen this year. Dr Williams needs to come back to Dr Gorman and Dr Paton with the final costing.

3.2 Dr Acott reported a very positive response from EUBS concerning the workshop on technical diving. The meeting format proposed is three half evenings of free papers and the rest of the time workshop.

3.3 The booking form for the meeting was discussed. The treasurer reported that there was no requirement to be a financial member of SPUMS in order to attend the ASM but that it was strongly encouraged. It was decided that non-members would pay an extra levy and that this would be in the range of $90. There would be no extra levy for members who belong to other recognised societies in a similar field. There should be a place on the registration form which reads: I am a member of another society called..... There would be a mail out of the conference details to the EUBS members.

3.4 Dr Williams was approached by Dive Australia to give a donation to the organisation to help with advertising costs. It was decided that a free advertisement in the SPUMS journal would be more appropriate.

4 Correspondence

4.1 Dr Gorman re UHMS attendance at the proposed workshop in Maldives.

4.2 Letter from Secretary dated 29 June 1995.

SPUMS POLICY ON ASTHMA AND FITNESS FOR DIVING

Des Gorman and Andy Veale

Introduction

The Society's workshop on asthma and diving was held as part of the 1995 Annual Scientific Meeting at Castaway Island, Fiji. The open forum component of the workshop was prefaced by a series of presentations (a single written submission was received from Dr Douglas Walker) which are published on pages 222 to 263 of this issue of the Journal.

Considerable consensus was apparent from these presentations. In particular, it was clear that there are few controlled data on which to base risk assessments for asthmatics who dive (let alone a consistent definition of asthma) and that some data sets (e.g. BSAC survey) were of little or no value (at the least being rich in selection bias).

Drs Fred Bove and Andy Veale agreed that what data did exist suggested that the relative risk for those asthmatics who dived was about twice that of the non-asthmatic population (not withstanding the observation that these data did not reach statistical significance) and that it was important to remember that this was in effect a doubling of a very low rate of injury and illness.

Dr Sandra Anderson described various provocation tests and in particular, exercise and hypertonic saline challenges. The most interesting aspect of her presentation was the report that regular budesonide (Pulmicort) inhalation eliminated the hyperresponsiveness in many asthmatic patients. The fitness of such a patient to dive is obviously difficult to determine. It is noteworthy in this context that the Undersea and Hyperbaric Medical Society (UHMS) has just conducted a similar workshop and it was agreed at this forum that asthmatics who are well-controlled (i.e. not responsive to exercise or salt water) on inhaled steroids are "fit for recreational diving".

The utility of provocation testing was also discussed in their presentations by Drs Cathy Meehan, Graham Simpson, Peter Chapman-Smith and Robyn Walker. Despite the differences in approach, it was clear that there was a significant false negative rate in asthmatics (and especially those on regular steroid medication). Again, it is worth mentioning the UHMS Workshop, at which there was considerable support for exercise as the primary form of provocation testing (remembering that inhalation of hypertonic saline is a "model" of exercise-induced asthma).

SPUMS POLICY STATEMENT ON THE PREVALENCE OF ASTHMA IN AUSTRALASIAN DIVING CANDIDATES

1 A history of asthma is common in diving candidates.
2 The assessment of risk for a diving candidate with a history of asthma should be conducted by a medical practitioner who has had training in diving medicine (i.e. suitable for admission to the SPUMS list).

SPUMS POLICY ON THE IMPORTANCE OF ASTHMA IN DIVING

1 Asthma is a potential cause of morbidity and mortality in divers. The level of risk in this context needs to be measured.
2 Diving may precipitate (an) asthma (attack).
3 Asthmatics may have limited exercise capacity and are at risk of shortness of breath, panic and drowning on the water surface.
4 Asthmatics who dive may be a self-selected (i.e. survivor) population and hence their experience may not be representative of the risks of diving for the general asthmatic population.
5 Current information (from descriptive databases) suggests that the relative risk for asthmatics who dive (compared with non-asthmatics) for a decompression illness is about 2.

SPUMS POLICY ON THE ASSESSMENT OF RISK FOR A DIVING CANDIDATE WITH A HISTORY OF ASTHMA.

1 The determination of risk for diving in someone with a history of asthma requires a gradation of the severity and currency of their asthma.
2 Risk stratification for someone with a history of asthma who wishes to dive will require a thorough history and examination and often lung function testing, which may include provocation testing (and especially with exercise and/or hypertonic saline). This may need to be repeated if the person elects to dive.
3 Provocation testing with exercise and/or hypertonic saline (rather than with histamine and methacholine) may be more specific for asthma that is of concern in diving. The significance of a positive result is more easily understood by the diver.
4 As the risk for diving in someone with a history of asthma is uncertain, permanent records should be retained as part of a SPUMS-sponsored study.

Key words

Asthma, diving fitness, provocation testing, hypertonic saline.

DRAFT SPUMS POLICY ON MEDICAL PRACTITIONER CERTIFICATION OF FITNESS FOR DIVING

Andy Veale, Drew Richardson and Des Gorman

Introduction

The Society's 1995 Annual Scientific Meeting (ASM) at Castaway Island, Fiji was dedicated to the theme of fitness for diving. It became apparent during the meeting that the traditional prescription-based approach to diving fitness assessments in recreational diving candidates is no longer appropriate. Indeed, there was consensus that a more discretionary approach was warranted. This would involve the medical practitioner in a risk assessment rather than a regulatory role, but would require a greater level of practitioner insight and training.

Dr Andy Veale proposed a system of tiered review, with a cascade of assessments, performed to AS4005.1 standards, from a general practitioner without training in diving medicine to a trained practitioner to a specialist in diving medicine. This was said to be the *status quo* in New Zealand and Dr Mike Davis read a letter from the New Zealand Underwater Association arguing the efficacy of this approach. A consensus was not reached on this issue, but it is considered to be of sufficient importance to warrant a review at next year's ASM.

A consensus was however quickly reached on the current practice of certifying a person "fit to dive". This is clearly nonsensical for an air breathing mammal. Dr Veale proposed a risk related liability-acceptance style of "certification" that met with the approval of the members at the meeting and was considered by the diving instructor agencies to be compatible with their practice. This is proposed as Society policy (see below).

STATEMENT OF HEALTH FOR RECREATIONAL DIVING

This Section to be completed by the Medical Practitioner.

This is to certify that I have today interviewed and examined:
Name.....
Address.....
.....
Date of birth...../...../.....

Initial those that do and delete those statements that do not apply:

..... I have assessed the candidate in accordance with AS 4005.1.
..... I can find no conditions which are incompatible with compressed gas, scuba and surface supplied breathing apparatus (SSBA) and or breath-hold diving.
..... I have explained the potential health risks of diving to the candidate and we have discussed how these risks may be reduced. The candidate appears to have a good understanding of these risks.
..... Based upon my assessment, the candidate should not dive with compressed gases (Scuba and SSBA).
..... Based upon my assessment, the candidate should not breath-hold dive.

.....	.....	...../...../.....
(Signature of Medical Practitioner)	(Name of Medical Practitioner)	(Date)

This Section to be completed by the Candidate.

Initial those that do and delete those statements that do not apply:

..... I understand the health risks that I may encounter in diving and how these risks may be reduced. I also understand that the Medical Practitioner's recommendation herewith is based, in part, upon the disclosure of my medical history. I agree to accept any responsibility and liability for health risks associated with my participation in underwater diving, including those that are due to or are influenced by a change in my health and or my failure to disclose any existing or past health condition to the Medical Practitioner.

.....	.....	...../...../.....
(Signature of Candidate)	(Name of Candidate)	(Date)

Workshops are now used as the vehicle to produce Society policy. This is certainly a better system than the previous approach of "someone" on the Society's Executive Committee being responsible for such policy development. These Workshops are advertised and anyone wishing to be involved, but being unable to attend, is invited to submit their views in writing. Consequently, there is no need for a review of the outcome of these Workshops through the Journal. This is clearly not the case here, as the membership was not informed of any intent by the Society to produce a policy on diving fitness certification. It follows that this policy is submitted as a draft and members and associates are invited to comment on the draft in writing through the Editor of the Journal. These comments will be considered by the Society Executive before the draft policy is accepted and forwarded to Standards Australia for inclusion in Standard AS 4005.1.

DRAFT SPUMS POLICY STATEMENT ON THE "CERTIFICATION" OF CANDIDATES FOR RECREATIONAL DIVING

A medical practitioner's statement of the compatibility of a candidate's health and recreational diving must include both an acknowledgment of "health risk" and an acceptance of liability by the candidate. The format on page 214 should be used.

Key words

Health surveillance, diving fitness

Details of the authors of these position statements

Dr Andy Veale, FRACP, is a consultant to the Royal New Zealand Navy and Auckland HealthCare. His address is 42 Omahu Road, Remeura, Auckland, New Zealand.

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Dr Des Gorman, FAFOM, PhD is a consultant to the Royal New Zealand Navy and Head, Occupational Medicine, School of Medicine, The University of Auckland. His address is Private Bag 92 019, Auckland, New Zealand.

LETTERS TO THE EDITOR

DIAGNOSIS OF A DIZZY DIVER

ENT Department
Bergen University Hospital
N-5021 Bergen
Norway
13/7/95

Dear Editor

I read with interest Carl Edmonds' article "Diagnosis of a dizzy diver" in SPUMS J 1995; 25(1): 29-31. I agree entirely on his diagnosis and advice to cease scuba diving. However, I have a few minor comments.

Having been the holder of a private aircraft licence for many years I clearly envisage Edmonds' concern at being the passenger in an aircraft piloted by a potentially dizzy airman. However, after central compensation of a peripheral vestibular lesion, the system has become "recalibrated" and is probably not more prone to alternobaric vertigo than in persons with symmetrical peripheral vestibular function. At least I can not recall to have seen any documentation that they are, and nor in my experience in divers continuing to dive after such lesions. A test in a hypobaric chamber could decide that.

Eventually, the National Civilian Medical Aviation Board would have to decide on his flying ability when he applied for his medical recertification. The North American Federal Aviation Administration (FAA) Aero-medical Certification Division's (AMCD) current policy is: "An airman may receive a medical certificate if the condition is in remission and the airman can meet the medical standards for the class of certificate applied for. .....the condition has stabilized and the airman is asymptomatic".

Edmonds also advocates the use of a nasal decongestant (locally, I suppose) before sky diving. I do not think that will harm, but unless he has a blocked nose I am not convinced it will be of any help. I know it is being used by divers, but I have seen no documentation of its effect. In my own experience as a military sky diver with jumps from 13,000 feet and in excess of 1 minute free fall I have never had to perform equalisation manoeuvres, although I have been meticulous about that when diving in the sea. Neither have I heard of anyone else needing to equalise.

The ambient pressure at an altitude of 10,000 feet above sea level is 69.7 kPa. Sky diving to sea level from that altitude will correspond to diving from the surface to 3 msw. I always recommend divers to start pressure equalisation before reaching half that depth, so I do understand Edmonds' concern. However, during ascent in the aircraft prior to the jump the middle ear air will expand, so there should be no need for Edmonds' advice to inflate the middle ears by means of forceful Valsalva manoeuvres before the jump, since the middle ears will already be well inflated. Besides, I advise against the use of forceful Valsalva manoeuvres for middle ear inflation because of the theoretical risk of the resulting increased intracranial pressure being conveyed to the inner ear through the perilymphatic duct. I advocate the use of more gentle techniques, like the Frenzel manoeuvre.

Otto I Molvær

ANTIDEPRESSANTS AND THE DIVING MEDICAL

P.O. Box 635, North Adelaide,
South Australia 5006
10/7/95

Dear Editor

At a recent Diving Medical Examiner Course it was recommended that any person taking antidepressant drugs should be automatically classed as unfit for scuba-diving. Whilst this was a very reasonable disqualification in the past, recent therapeutic developments may merit a review of this general exclusion.

Until recently the only antidepressants that were prescribed in Australia were either members of the Tricyclic (TCA) or of the Monoamine Oxidase Inhibiter (MAOI) classes. The well-recognised side-effects of these drugs upon the cardiovascular system, irrespective of their additional adverse effects upon the autonomic and central nervous systems, are such that it is very reasonable to exclude any person using them from scuba-diving.

However, there are now new groups of antidepressant drugs available that are described as Serotonin Re-uptake Inhibitors (SSRI) or Reversible Inhibitors of MAO-A (RIMA) drugs. Fluoxetine (*Prozac* Eli Lilly), Paroxetine (*Aropax* Smith Kline Beecham) and Sertraline (*Zoloft* Pfizer) are available examples of the former and Moclobemide (*Aurorix* Roche) is the only example of the latter group. Extensive clinical and research experience of these drugs appears to exclude any significant risk of cardiac arrhythmia and they are clearly much safer in this regard than the TCA type of drug. They also do not seem to cause the drowsiness and sedation that is typically associated with TCAs.

Any person who is *currently* suffering from a Major Depressive Illness would almost certainly, irrespective of their medication, be considered unfit for scuba-diving. However, there is increasing recognition amongst psychiatrists of the prophylactic benefits of maintaining sufferers from *Recurrent* Depressive Disorder on antidepressants on an indefinite basis. Given the tolerability of the new classes of medications, this is now not only a valid clinical option but also one that is likely to be accepted by the many people prone to this debilitating disorder, who found the earlier medication difficult to bear.

It is thus increasingly likely that diving physicians will be approached by individuals with no current or recent history of a Depressive Episode, who are well stabilised on long-term antidepressant medication and are seeking clearance to go scuba diving.

Provided that person was taking one of the SSRI or RIMA antidepressants and was otherwise both physically and mentally fit I believe it would be difficult on theoretical grounds to justify excluding them from recreational scuba diving. However, whilst reassured by the literature on the newer antidepressants, I am unable to find any direct clinical references on this topic and wondered if any of my colleagues have any practical experiences to assist us in making such decisions.

John Couper-Smartt

IS THE SNORKEL STILL USEFUL?

201 Wickham Terrace
Brisbane, Queensland 4000
10/8/95

Dear Editor

What would the diving industry do without Bob Halstead? His letter "'I Sink, Therefore I Am" (SPUMS J 1995; 25 (2 June): 106-109) is a classic with his witty and wicked observations as to what defines a diver. His letters should be compulsory reading for all divers.

Although I agree with the bulk of his observations, I do not share his insistence that one should dispense with a snorkel. I find a snorkel a most useful piece of equipment. It is no hindrance to ones diving except perhaps when entering wrecks or caves and there is always the occasion when a snorkel is more valuable and more comfortable than a regulator.

Two examples, first while waiting on the surface of the water to be picked up by a boat after a drift dive or, in the extreme instance where one has to ditch ones tank and weight belt and attempt to swim to safety or to stay in the one position. There have been many instances of divers being left behind by the dive boat (but not by the *Telita*) and being picked up the following day, if they are lucky !

For me, I will stick with my snorkel and reduce surface tension.

Bill Douglas

BOOK REVIEWS

OXYGEN FIRST AID
John Lippmann
(ISBN 0-646-23565-6)
J.L.Publications, PO Box 381, Carnegie, Victoria 3163, Australia.
Price $Aust 20.

When first reviewed in 1992 as *Oxygen First Aid for Divers* it was suggested that this book was a very good reference book for anybody doing a first aid course. My opinion has not changed with the publication of this second edition which has been renamed *Oxygen First Aid*. This book is aimed, not at basic first aiders but at their more experienced colleagues who may be required to administer oxygen as part of the resuscitation.

On first opening the book I thought that the author had added more chapters to cover new material but on further investigation I found that this was an illusion due to the alteration in the sequence of chapters so the book now flows in a more logical manner.

The style of the text has been altered for political correctness so that there are no longer patients or victims, all are now casualties. The elimination of all mention of gender sometimes makes the text a bit fussy and more difficult to read but in these enlightened times it seems to be the thing to do.

I like the way the publisher has introduced better quality paper and has improved the definition of the photographs so that one can actually see what is being demonstrated. The font too is clearer and, being larger, is easier to read in my approaching senectitude.

The first three chapters review the anatomy, physiology and techniques of cardiopulmonary resuscitation in language the laity can understand. Then follow five chapters on the benefits and techniques of oxygen administration, the equipment available, indications for its use and adverse reactions. I was intrigued by the chapter on infection control and the legal ramifications of administering first aid and can fully understand why someone who is a little hesitant about their resuscitation skills might be reluctant to participate.

This is a good book, easy to read and chock full of excellent information. It should be on the bookshelf of every diver in Australia and used by them as a reference book at regular intervals. It is also very relevant for non-divers and should be widely disseminated through state, municipal and school libraries.

David Davies

HYPERBARIC MEDICINE PRACTICE.
Eric P. Kindwall (Ed).
(ISBN 0-941332-29-2) 1994 692 pp.
Best Publishing Company,
P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 110.00. Postage and packing extra. Telephone credit card orders are accepted.

The history of hyperbaric medicine dates from Henshaw, an English clergyman, who 300 years ago used his "Domicilium" to treat a wide variety of disease states with hyper- and hypobaric air. Hyperbaric oxygen therapy (HBO) has a more recent history and was brought to the attention of the medical community by the pioneering work of Boerema in the 1950's. Despite the plethora of scientific articles on HBO, there has been no comprehensive textbook which looked widely at the literature in a critical way. This book, by one of the recent pioneers of HBO therapy, goes a long way to resolving this deficiency.

Forty-three authors, with considerable theoretical and importantly practical experience, contribute to 36 chapters. These authors are mainly from the US and the book does have a North American bias with regard to drugs, doses and treatment tables. There are some contributors from the Netherlands (Bakker), UK. (Elliott), Germany (Lampl and Freyl) and Sweden (Hammerlund). The book is subdivided into 3 sections, General Considerations, Disorders Approved for Treatment with HBO and Investigational Areas and has over 1600 key HBO references.

The layout of the text is clear, however the references are in the same font size as the text and in some chapters this leads to interruption of the text by lines of references. In addition, emphasis is placed on some words by underlining which detracts slightly from the text and does not seem appropriate for a textbook. In general most chapters have a clear style, however in some cases tables and diagrams would greatly benefit the reader, especially when the author is discussing several papers on a single theme.

The basic physiology and pharmacology of HBO are well covered. Complications are dealt with in an organised manner, with slight differences in the classification of middle ear barotrauma between US and Australasian practice. Considerable space is given to monoplace chambers which are much more common in the US than Australasia. This may change with the current interest in the treatment of sports injuries with monoplace chambers. In these days of DRGs and Casemix economic considerations of HBO therapy are discussed with a typically North American attitude to aggressive marketing

and "selling" the specialty. Physicians in this part of the world may not be comfortable with this approach, but these techniques may become necessary when departmental budgets are threatened.

Critical care medicine is now interfacing closely with HBO therapy. To this end the contribution by Weaver on the management of the ICU patient in monoplace chambers is pertinent. This should have been extended to multiplace chambers for completeness. There are 5 appendices to his chapter which are reprints of complete articles in other journals. Interested readers would no doubt look up these references or better still they could have been distilled into a shortened version within his chapter. This seems a little incongruous when other chapters could equally have original article reprints to follow their text.

There is a discussion on the role of hyperbaric nursing. This is limited purely to nursing and there is complete absence of any discussion on technicians and other ancillary support personnel.

Recent thinking on the aetiology and clinical presentation of DCI suggests that arterial gas embolism (AGE) and decompression sickness (DCS) are a spectrum of the same disease process. However this is not universally accepted. In my opinion it would have been better to amalgamate these 2 chapters.

There are some chapters, e.g. AGE and gas gangrene, where statements are made on acute medical and ICU management which would not necessarily be followed in Adelaide. It would be better if authors concentrated on the theory and practical details of the HBO related topics and did not stray into other clinical areas where there expertise may be less than some of their readers. There are many excellent chapters, which are written with both a theoretical and practical viewpoint. This is one of the strong points of the text, but also an area where didactic statements may not be universally accepted.

Despite the above comments, this is a landmark publication in the area of hyperbaric medicine. Dr. Kindwall has done well to restrict the size of the book and produce a consistent style. It will become a standard textbook in all hyperbaric facilities and provide a valuable reference base. It should be read and used by all involved in hyperbaric medicine including doctors, nurses and technicians.

Michal Kluger

MEN AND WOMEN IN DIVING

M St Leger Dowse, P Bryson, A Gunby and W Fife.
(ISBN 0 9525152 0 2)
Diving Diseases Research Centre, Fort Bovisand, Plymouth, Devon PL9 0AB, United Kingdom. 1994.
Price from the publishers £20.00 plus packing and postage. Also available from Dive Log New Zealand, PO Box 55-69 Mission Bay, Auckland, New Zealand and the Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Maryland 20895, USA. US price $US 32.00 postage and packing $US 5.00.

This is a report based on the responses to a questionnaire which is being used by Dr William Fife of the Texas A&M University, USA. A pilot study was conducted in 1990 and this was followed later that year by its wider distribution, 10,620 being distributed over three years in the UK. There were only 2270 replies, a response rate of 21.4%.

The questionnaires were targeted to female divers through the British sports diving associations, and to the wider diving public at diving conferences and exhibitions, to diving medical referees and to dive shops and dive schools. The respondents, who were anonymous, were asked to report their diving history and practices, alcohol, smoking, contraceptive and drug use and the pregnancies during which they had dived. Naturally not all the questions were answered clearly, but this report is an invaluable summary of the information obtained.

An immense amount of work has gone into this analysis. Its value is certainly not negated by the, probably inevitable, fact that it can answer so few of the questions concerning the risks women run in scuba diving. It is a resource which should be used by anyone interested in trying to create a valid information basis for their opinions.

While it must be conceded that all such surveys select out only a special group from the general community, nevertheless this must not be used as an excuse to denigrate its findings. Except where an investigation concerns a rare and easily definable condition it will report on only a fraction of the population which is the subject of the investigation.

What are the conclusions? First, that although there are some doctors who do not understand the medical standards applied to divers, not all the applicants are entirely truthful when presenting their medical history. It is probable that both these factors operate in some cases. Second, it showed that, in those surveyed, the women dived less aggressively than the men and had a lower rate of DCI, possibly because they were not diving so deep. However when divers of similar experience were compared there was no difference in dive depths. Indeed women appeared to dive with less regard for decompression stops but possibly had greater respect for the no-stop times given in dive tables.

Rather surprisingly 36% of respondents, more women than men, reported suffering self assessed DCI. This could be a reporting rather than a factual difference.

This frequency is at odds with the usual DCI incidence and indicates that the respondents may not have been representative of the general recreational diving population. In this survey there was an increase frequency of abortions when there had been more than one dive per day but there was no relationship between depth dived and occurrence of abortions or outcome of pregnancy.

There is no clear picture arising from the findings as they are presented and the authors correctly advise that this demonstrates the need for further studies.

While anonymity is undoubtedly necessary in any such attempt to solicit information of a personal nature, it has its drawbacks. It may be possible in future surveys to provide for the respondents to communicate further, under medical confidentiality, about their health or other problems.

The report ends with a hope, close to the heart of this reviewer, that sufficient discussion and debate will result from the report to result in laying the foundations for a more useful and worthwhile collection of data.

Douglas Walker

FROM SKY TO SEA

Susan van Hoek
Best Publishing Company,
PO Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Telephone (1) 602-527-1055. Fax (1) 602-526-0370.
Price US$22.95 plus packing and postage. Telephone credit card orders are accepted.

There is a common concept that a millionaire is a person who has a million dollars and has no worries and there are many such people around. There is also a much rarer breed, a person who can write a cheque for a million dollars and have no worries. This person is a true millionaire and Edwin Link was one such person.

Edwin Link was obviously a man who was in the right place at the right time and with the right ideas to take full advantage of the situation. Born in Indiana in 1904, Link rapidly developed a mechanical bent and became a tinkerer. He was a non-conformist with a resistance to the regimented process of learning that was required by schools at that time. His father however, refused to accept that a vocational training school was suitable for his son but, at the age of 16, after a nomadic passage through numerous formal schools Link was introduced to and became fascinated by flying.

In 1922 he worked for his father's Link Piano and Organ Company and, with his ingenuity, was able to introduce a number of innovations which improved the quality of the product. His income initially allowed him to take up part time flying and, after being sacked by his father, he became involved in barnstorming and running a flying school.

In the mid 1920s pilot tuition was a slow, hands on, seat of the pants affair, and the death rate in flying trainees was unacceptably high. Being deeply concerned about this, Link devoted his non-flying hours to developing his first "primary flight trainer". This piece of equipment not only increased the speed of pilot instruction as it could be used in all weathers and after dark, but it required no fuel and it did not kill the trainees. Despite early setbacks and inexplicable resistance from governments, later models of this simple flying simulator became accepted throughout the world as the Link Trainer. This device was used in the early education of many allied World War II pilots. Descendants of this machine are now used world wide to train pilots of commercial and military aircraft and all space vehicles.

By 1947 he was exceedingly wealthy and his interests included ocean racing off the Florida coast and, as time went by, the lure of treasure hunting in the Caribbean started to take hold and he was introduced to the new scuba equipment developed in France.

His adventures in marine archaeology are an ecologist's nightmare and extended from the Carribean through the Mediterranean and even into the Sea of Galilee. After being acquitted of illegal underwater archaeological research by the Greek courts in 1961 he initially joined forces with Jacques Cousteau in the development of an underwater habitat in which divers could live and work for an extended time without having to return to the surface. However, before this came to fruition, there were differences of opinion and a parting of the ways, with Cousteau going on to develop his shallow water habitat using air whereas Link developed a model for deeper water that required helium oxygen mixtures as the breathing medium. He went on to do a record breaking dive in this, both for depth and duration and so saturation diving was born.

Once this was successful, he realised that the inhabitants needed some way of getting to and from the habitat without undergoing in-water decompression, so he worked on a lockout submersible vehicle which could be carried on the deck of a tender vessel and then used to carry personnel and equipment to and from the habitat. This was done. Then he found that the ship needed an articulated crane so he designed and built one that was suitable. A later model of this submersible became known as the Johnson-Sea-Link which was used to find and assist in the recovery of the remains of the ill fated Challenger space shuttle, a real coup, as the US Navy submersibles had been unable to do so.

Link was a man who was able to look at a problem and solve it by incorporating ideas and techniques from totally unrelated disciplines. Herein lay his genius. At his death he had over 30 patents credited to his name. He was a man who was able to get and keep a team working together though, I gather, he could well have been a slave driver and tyrant at times. He had unbounding energy and a great capacity for work with little requirement for sleep. Link was at the forefront of developmental diving for three decades and was instrumental in the development of many of the techniques that are now taken for granted.

This book could have been a wonderful history of this man's work. The author has used the diaries written by Link's wife as the source material so that a lot of the really interesting information is glossed over. She spends more time telling us who visited and looked over the ship or what they had for dinner, than she does about what Link was doing with each of the hurdles he was facing, how he was tackling them, and what his thought processes were in overcoming these problems. I was very disappointed with this book. Its prose is dull, its narrative forever interrupted with barely relevant excerpts from the wifely diaries and it glosses over the important features and developments in his life. In its defence however, it did whet my appetite for further research and discovery about this man's life and achievements.

Since reading this book I have gone on to read excerpts of one of his wife's books and it seemed that she is a much more interesting author to read than van Hoek. The problem of this book is just that it was written by the wrong person. Disappointing!

David E. Davies

SPUMS 1995 ANNUAL SCIENTIFIC MEETING

THE PRINCIPLES OF HEALTH SURVEILLANCE

Des Gorman,

Introduction

Health surveillance can either be an exercise in primary health care (e.g. measurement of blood lipids to identify people who should change their diet) or an activity-related exercise (e.g. assessment of fitness for diving). In general, the principles are common. The only possible exception is that primary health care surveillance should be prefaced by the "condition" to be screened being shown to be amenable to modification. This is not a necessary preface in activity-based surveillance. For example, it may be worth screening potential blood donors (an activity based surveillance) for antibodies to HIV, but given the resistance of this virus to current treatment, in most communities this would not be cost effective primary health care surveillance.

This review of the principles of health surveillance is biased to activity oriented assessment and fitness for diving, in the context of asthma, will be used as an illustration.

Principles of health surveillance

It is important to examine critically the potential of a "health condition" (a disease, treatment regimen, state of aerobic fitness etc.) for surveillance, as once introduced most screening procedures are very difficult to withdraw. This is due to the population being screened coming to accept the screen as an important part of their health care (often seen as a warrant of fitness). Individuals (and their medical attendants) will always have trouble accepting population-based rationales for allocation and withdrawal of health care. It follows that any health surveillance program, once introduced, must itself be surveyed for efficacy.

The first step in the process of health surveillance is to decide if the condition is worth screening. To justify screening a condition must be:

a important (to the activity or to long-term health);
b prevalent in the subject community;
c (in the context of a primary health care screen only) modifiable (i.e. treatable);
d able to be detected qualitatively and quantitatively by sensitive and specific tests (such sensitivity and specificity will be influenced by prevalence).

On this basis, few of the health screens introduced in the last 20 years in Australasia could be justified. The Armed Forces in both Australia and New Zealand demonstrate many classic examples of poorly focussed and consequently ineffective health surveillance.

If a condition "survives" this review, it must then be decided if the surveillance is cross-sectional or longitudinal. In the context of an activity-based survey, this equates to either a preplacement or ongoing assessment. Often, screens will be able to be justified both

before an activity and as an ongoing screen, however the latter should be reserved for those conditions that are affected by the activity itself or by coincident events or by age. For example, it is worth screening diving candidates for the size of their lungs (FVC) before they undertake any training (as a screen for risk of pulmonary barotrauma). However, the FVC is neither affected by diving nor by age, such that unless the diver has some other form of lung injury or illness, there is no value in periodic reassessment of lung size.

The next step in health surveillance is to decide if the process is to be prescribed or discretionary. The prescribed format is that usually used by the military, occupational diving regulatory authorities, fire and police services and insurance companies. The assessment is based on a prescription (e.g. to be fit for service the candidate must have uncorrected vision in both eyes of at least 6/9). The strength of such assessments is that the medical practitioner or other health professional conducting the review does not require any special training (i.e. they only need to be able to read the prescription) and the outcome of the assessment is unequivocal (pass or fail). The weaknesses of this type of survey is that many candidates will "fall on the thresh-hold" and for many conditions (e.g. asthma) the diagnostic criteria are controversial, such that some form of central arbitration is needed to ensure consistent application of the prescription. Also, the individual being assessed is excluded from the risk assessment. This often results in people who have "failed" such a prescriptive survey "shopping around" for another medical assessment and if aware, avoiding the history that invoked the original negative response (e.g. not describing a history of recurrent shortness of breath and wheezing). Finally, the prescribed format requires a medical practitioner to engage in "policeman-like" behaviour, a role that is not well suited to most doctors.

The discretionary assessment is based on the practitioner evaluating and explaining to an individual the risks (e.g. in the context of an intended activity) and the individual deciding the merit of undertaking the activity. The strength of this process is that the individual is central to the risk acceptance and the medical practitioner is engaging in "physician-like" behaviour. The weaknesses are that the practitioner requires insight (e.g. knowledge of the physics and physiology of underwater exposures for a diving candidate), other risk acceptors are potentially excluded (e.g. dive instructors and dive buddies) and the outcome of the assessment is often uncertain (e.g. the diving candidate may decide to take the risk of diving in the context of active asthma). Many practitioners who use a discretionary approach do have a prescribed threshold as a base (e.g. a diving candidate with phenytoin (Dilantin) controlled epilepsy would not be given the opportunity for discretion, whereas a candidate with a history of asthma in childhood would be consulted).

Any health surveillance then requires careful examination of purpose and efficacy; and also is dependent on the assessing practitioner being aware of the nature of the process.

Asthma and diving fitness

Asthma and fitness for diving can be used to illustrate these principles of health surveillance, particularly in the context of a pre-placement assessment.

Firstly, does asthma warrant screening in a population of diving candidates? The answer is clearly yes, given that:

a asthma is important in diving (Exercise, breathing a dry cold gas or alternatively a salt water aerosol, anxiety etc. may all precipitate asthma. Active asthma will limit a diver's cardiorespiratory fitness in the water and may cause them to drown. Air trapping in an asthmatic lung may cause pulmonary barotrauma during a decompression. Some bronchodilators will reduce the ability of the lungs to filter venous bubbles during and after a decompression);

b asthma is common in Australasia (perhaps 20 to 30% of the population wheeze at some time of their lives);

c tests of bronchial reactivity do exist and there are claims of reasonable sensitivity and specificity for salt water provocations (the same cannot be said for histamine and methacholine challenges).

The review can be prescribed or discretionary, or a mixture of both. A variety of approaches are used. They vary from considering that any candidate who has asthma is fit to dive providing they do not dive when they have active asthma (this is a common stance in the UK and is based on some of the worst epidemiological data ever published, and ignores the potential for diving to provoke asthma), to considering those who have had no recent (3 to 5 years) history of asthma (or of using asthma medication) as being fit to dive, to only considering those with any history of asthma at any time as being fit to dive if they have demonstrably normal bronchial reactivity (a problem here is that some viral URTIs can cause temporary bronchial hyperreactivity), and finally to the stance that any history of asthma is incompatible with diving. Any of the former approaches do require some ongoing health surveillance.

Summary

Health surveillance is potentially useful, but often expensive, ineffective, time consuming and distracting. This is usually a direct result of a health surveillance program that is inappropriate and or the practitioner involved not being aware of the nature of the process. The principles of

health surveillance should be understood by those who intend to engage in such screening.

Key words
Pre-placement, discretionary assessment, prescribed assessment, asthma.

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OBSERVATIONS ON ASTHMA IN THE RECREATIONAL DIVING POPULATION

A Bove

I will review a little bit about the pathophysiology of asthma. Some of the data that we collected for DAN in an attempt to make a statement about what to do with asthmatics in diving. Asthma is a common disease. Some people estimate about 10% of the US population are asthmatics. I was in the desert for several months during the Gulf War and a large number of the young American marines, who went into the desert, came to our hospital wheezing with significant asthma because of the organic dust that is in the air there. Our surveys in the US indicate that the incidence of asthmatic divers is the same as the incidence of asthma in the general population which means there is no effective screening. Asthmatics are getting into diving, probably by not revealing their past history. So, generally, asthma comes under the list of pulmonary disorders in diving, and I do not want to dwell on the other ones, although they are there. The history of pneumothorax, the history of any other chronic lung disease, pneumoconiosis, all would eliminate somebody from diving. The question about what we do with previous barotrauma is also unanswered.

If one looks up the text book definition of asthma it is usually stated as "generalised airway obstruction due to the contraction of bronchial smooth muscle". It has a series of clinical characteristics. Often it is associated with a cough, dyspnoea (shortness of breath) with mild exertion, wheezing, the over inflation syndrome, that is the lungs are over inflated, and often the auscultatory finding of wheezing and crackles throughout the lungs because of secretions retained in the airways. I do want to make light of the cough because many people who have very mild asthma, develop a cough and do not understand what it is about. People are sent to me with a cough thought to be heart failure, it often turns out they are asthmatics and a bronchodilator gets rid of the cough. The cough was related to airway reactivity, so there are a number of different presentations. The severe obstructive airway disease which causes wheezing and dyspnoea is only one end of the spectrum.

Allergy and infection are the two most common trigger mechanisms. Most asthmatics have a family history, in parents, siblings or children, of other allergies. Infection of the upper airways is often a trigger in the person with hyper-reactive airways. Adult bronchiolitis, when a viral infection of the airways causes wheezing, is a truly transient phenomenon and it is not related to reactive airways. There is a small number of patients who wheeze with bronchitis, but if that is the case one should not classify that patient as an asthmatic. In these cases one needs to wait several months to allow the airways to settle down before doing any testing. Acute anxiety will do this and I think it is probably because of the change in hormone that stimulates the airways. Parasympathetic stimulation will cause reactive airway disease, and of course the catecholamines usually cause relaxation of the airways so we use epinephrine (adrenalin) to relax the airways. Exercise will induce wheezing, and cold will induce wheezing. Cold and exercise are somehow irritants to the airways which can cause bronchospasm under those conditions. So there is a number of trigger mechanisms.

In a chronic asthmatic the process goes beyond just pure smooth muscle activation and bronchial constriction. There ultimately becomes hypertrophy or overgrowth of the bronchial smooth muscle so there is thickening of the bronchial walls. There is mucosal oedema and secretions in addition to bronchial hyperaemia. All these things will cause airway obstruction. In particular the retention of secretions in the small airways is a common complication in asthmatics because with bronchial relaxation, the airways do not always completely clear. Often one must use inhalation therapy with mucolytic agents to clear the bronchial secretions. This is an important part of the chronicity of asthma.

Fishman[1] is a well respected pulmonary physiologist who studied asthma for a long time and classified severity in a range of one (most) to five (least). I think the single most useful measure of an asthmatic is the alteration in airway conductance. The normal person, or the minimal asthmatic, has essentially normal airway conductance. As one goes through the spectrum of severity to what essentially is chronic obstructive lung disease, there is a progressive decline in the airway conductance, that is there is more and more resistance to the motion of air through the airways.

This is manifest by a number of different measurements. The forced expiratory flow, between twenty five percent and seventy five percent (FEV_{25-75}), or any of

the measures that record the rate at which air leaves the lungs, will show abnormalities which are characteristic of asthma. That is the nature of the pathophysiologic limits on an asthmatic. They cannot move air rapidly out of the lung, therefore they cannot ventilate adequately and develop hypoventilation syndromes, relative hypoventilation syndromes, and sometimes even significant CO_2 retention. The ability to move air rapidly through the airways declines as the severity of the asthma increases.

The total lung capacity also increases as an asthmatic becomes worse. The chronic asthmatic who progresses ultimately to chronic obstructive lung disease has an expanded chest, an increase in total lung volume. With the mildest form of asthma the volume pressure curve, the lung compliance, is basically normal. As asthma becomes more severe the lung volume increases and as it increases there are decreases in lung compliance. Increasing lung volume is characteristic of the long standing asthmatic. The residual volume goes up. That is a problem in diving because long standing asthmatics are working at higher lung volumes and have trouble with buoyancy because they cannot get the air out of their lungs appropriately.

There is a gradual increase in total lung capacity as the severity of obstructive lung disease or of asthma increases. This is because of the destruction of the alveolar structure of the lung. In the end stages of obstructive lung disease, the total lung capacity is markedly increased. So two things, the lack of adequate airway conductance, and ultimately an increase in lung volume, cause the asthmatic to breathe at higher and higher lung volumes. These are two characteristics that can get the asthmatic into trouble with any sort of physical activity including diving.

The severe end stage lung disease patient has a very high residual volume. This is obvious when you look at the patient. The diaphragms are flattened, and low, the chest is expanded outward, the clavicles are elevated. The whole lung volume is increased including residual volume and there is a lot of intrinsic lung (alveolar) damage by the time you get to this level of obstructive lung disease. Only the mildest forms of asthma have residual volume unchanged. The more severe have a continuous increase in residual volume. Increasing resting lung volume can cause problems with buoyancy.

An interesting thing is the change in lung compliance as individuals develop hyperventilation. In the normal individual lung compliance is much the same when breathing at ten breaths a minute (resting breathing) and when breathing rapidly. With increasing severity of asthma there is very little change in lung compliance when resting. But with significant obstruction, lung compliance decreases. With rapid breathing rates, because of exercise, the asthmatic's lung compliance goes down. In other words the lung gets stiffer and the work of breathing goes up progressively as the respiratory rate goes up. So the exercising asthmatic has dyspnoea for a couple of reasons. First, they can not ventilate adequately, and two, they are really working the respiratory muscles much harder than a non-asthmatic because of the change in lung compliance. The lung volume is larger. The lung is stiffer, it takes much more energy to move the chest back and forth. So, during the hyperventilation of exercise, the asthmatic is consuming significant amounts of energy in the respiratory muscles and getting more and more severe sensations of dyspnoea because of this and because of the inability to ventilate the alveoli properly. So an asthmatic who develops significant airway obstruction can develop problems with exercise and particularly a problem when diving because of the expanding lung volume and alterations in buoyancy.

During ascent, the expanding gas in the lung can be trapped. Overdistension can occur causing pulmonary barotrauma with mediastinal emphysema, pneumothorax and air embolism. This is of concern because of the difficulty asthmatics have in exhaling properly and getting air out of the alveolar spaces . This is one major concern. The other is that when one looks at diving accidents in asthmatics, some of them are actually failures to be able to exercise appropriately on the surface. They just can not exercise, get severe dyspnoea, panic and drown. This is the key to many deaths.

There are about three million sport divers in the United States doing somewhere between twenty and fifty million dives a year. It is very hard to guess the right number but somebody said if one estimated between eight and ten dives per person per year one would end up with about thirty million dives, so twenty million dives a year is a conservative estimate.

Normally about a hundred diving deaths are recorded every year, sometimes fewer and sometimes more. Of those diving deaths about 30% are due to air embolism. There many reasons for these deaths including such things as acute myocardial infarction and getting run over by boats. The University of Rhode Island kept statistics, which were basically collections of newspaper clippings, for a long time. It is a fairly accurate way to look at reported deaths and they found one death due to asthma in ten years of collecting data. One diving death due to asthma in ten years of collecting data, so it is apparent, from the data bases that are available, that asthma is not showing up as a major player in the causes of death in the diving population.

Carl Edmonds' data would suggest that there is a fairly significant contribution of asthma to diving deaths.[2] He said that with a 1% incidence of asthmatic divers 9% of diving deaths were associated with asthma. The original paper describing the series where 9% of deaths were in asthmatics had no mention of the incidence of asthmatics

in the population studied,[3] I think there are questions about the accuracy of Edmonds' report as it is the only report that suggests that asthmatics are so under represented in the diving community.

The DAN data on diving accidents, collected by voluntary reporting, has established a good reputation in the United States and other countries. The reporting is not compulsory, but reasonably reliable. Up to 1987 they had recorded 95 arterial gas embolism cases and estimated the risk of an arterial gas embolism at about one in two hundred thousand dives. Of the 95, thirty eight cases had enough data to find a history of asthma. Unfortunately there was inadequate data in the other 57. There were five asthmatics in the 38 cases where there was adequate data. So you could argue that the incidence of asthma causing arterial gas embolism was either 13% (5 of 38), or 5% (5 in 95). Remember that the estimated incidence of asthma in the general US population is around 10%. Based on this information one could come up with an estimate that asthma increases risk for arterial gas embolism by about two or three times. If the risk is one in two hundred thousand without asthma, then a factor of two, one in one hundred thousand, is still a very low risk of arterial gas embolism in an asthmatic diver. So, here, unlike the data that Carl Edmonds published,[2] the contribution of asthma as a risk is really quite low in the population of reported injury data from the DAN database.

Corson et al. did some more sophisticated statistical analyses of the DAN database.[4] In the 1991 data there were twelve hundred cases of decompression related illnesses. One hundred and ninety six of them were gas embolism. Sixteen of the 196 (8%)had a history of asthma. There were 755 type two decompression sickness, 54 (7%) with a history of asthma, and 25 of the 54 (3% of the 755) were active wheezing asthmatics when they got their decompression sickness. So, there was some interest in the fact that an asthmatic would not only have an increased risk of arterial gas embolism, but also, for some reason, the risk of serious decompression sickness would also increase.

Using logistic regression analysis, Corson came up with the ratio for an asthmatic versus a non-asthmatic diver of about 1.58 to 1 increase in risk. He came up with a ratio for an asthmatic with clinical symptoms at the time they were diving, versus a non-asthmatic of about two to one. The 1.58 to 1 was not significant and 2 to 1 just barely made significance, so it is hard to say that the asymptomatic historic asthmatic has any higher risk than a non-asthmatic in a large database.

The active asthmatic seems to have roughly 2 to 1 increase in risk for having any kind of a diving accident. That includes arterial gas embolism and serious decompression sickness. But again, 2 to 1 in a population where the risk is 1 in 200,00 leaves the active asthmatic with an average risk of about 1 in 100,000 dives for an accident. That is a very low risk for having a diving accident relating to the active asthma patient. The non-active historic asthma patient essentially has no increase in risk, or if you want you can use this 1.58 to 1 but even that was not statistically significant. That is the data that comes from a large database reported to DAN.

The DAN investigators have reanalysed the data each year subsequently, now that they have got it all modelled in their computer, and it continues to support this idea that the active asthmatic has about a 2 to 1 risk in a pool risk of about 1 in 200,000 to start with.

There was a survey done where people were asked if they dived with asthma and if they had had any accidents.[5] Obviously all the dead asthmatics could not respond to the survey, so it is really hard to tell what the denominator was. I thought it was interesting that there were nine recreational divers in England, who wheezed every day, who had logged about twelve hundred dives over several years and had no adverse effects. All these folk felt that they could dive within one hour of an asthma attack. A couple of them commented that if they had an acute attack of asthma they would take their adrenalin, wait about an hour and then go diving again. Yes, one can dive with asthma. People have done it before. The question is what is their risk. The fact is there are acute serious asthmatics who dive and somehow do it safely. This was not a statistically valid survey because we do not know the denominator. We do not know the number of people with asthma who did not respond.

I think the way we should approach asthmatics is, first of all tell them that if they are an active asthmatic they probably have an increased risk of arterial gas embolism or decompression sickness which is about twice the average pooled risk, which in numbers is about a 1 in 100,000 dives risk of having an accident. This is the Desert Storm approach, young people come with a history of asthma, but no symptoms, enter a new environment, and all of a sudden they have asthma. One concern is that the status of an asthmatic can change while diving and that is an unpredictable process. Generally, we would advise an asthmatic not to dive, particularly the active asthmatic, but not necessarily the asymptomatic historic asthmatic, but the percentage of divers, at least in the United States, who were diving with asthma is the same as the population percentage. So these people are getting into diving and obviously, for the most part, doing it safely because the statistics are not singling them out as a high risk population.

I wanted to finish with the facts that the pathophysiology of asthma is characteristically defined by airway obstruction and by over inflation of the lungs and that there are a lot of asthmatics diving. There are lots of symptomatic asthmatics diving and they do not seem to incur the kind of risk that we hypothesise from the

theoretical aspects. Tom Neumann and I and a few others did a survey of the literature on asthma, published in the Annals of Allergy,[6] from which much of this paper has been taken. The issue for this meeting is to try to define a class of asthmatics who can dive safely and to screen out those who should not dive.

Veale

The pathophysiology of the changes, in lung volumes and in compliance, with worsening asthma, suggest that full spirometry, which would include the measurement of FRC and residual volume and total lung volume, is necessary in the assessment of all asthmatics. Therefore doing simple spirometry in this group is quite inadequate.

Bove

In my institution the pulmonary department is on the same floor and within sight of the cardiology department. So, whenever I get an asthmatic diver referred to me, after taking a history and doing the examination, I walk them down the hall to the asthma team, and have them take care of the patient. If you give a person with asthma to a pulmonologist, you certainly do not get just spirometry. You get a very thorough pulmonary function testing, volumes and all. However I personally think that ordinary spirometry is probably enough to screen out the worst of the asthmatics and the subtleties that one gets by going further may not really give one much more useful information. In other words the asthmatics that show up during spirometry may be the ones that should be screened out and everybody who has normal spirometry probably can dive. I throw that up as an issue because I do not know the answer.

Veale

The other slight problem is that asthma is totally dynamic in that one may have perfectly normal lung function one day and be in a critical care unit two days later.

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A RESPIRATORY PHYSICIAN'S APPROACH TO ASTHMA AND DIVING

A Veale

Prescriptive standards are designed to overcome an area of ignorance. They are designed for people without knowledge to lead them to the right answers. If they do not fulfil that then they are bad standards. Prescriptive standards have been designed for the ignorant and uneducated by those who do not trust us and who refuse to accept responsibility and therefore assume that you and I do not accept responsibility, you and I being the doctor and the patient.

Prescriptive standards by their nature are an easy way out. For example how does the standard handle, a twenty three year old woman who has hypoparathyroidism, who is, I submit, at much greater risk of death than perhaps some of the people with past asthma. But it is not in the standard so one is able, with a clear conscience, to certify this person as "fit to dive". A fourteen year old with a slipped femoral epiphysis may have some risks from diving. I think prescriptive standards are a cop out for those that are not prepared to think.

I must put the medical risks into perspective. Many, many, many, many more people are killed through poor training or absent training or poor practice or absent practice than by medical factors. As we get older medical risks become much more important in the genesis of morbidity and mortality. Training has been long forgotten. Equipment failure from ones buoyancy compensator which has not been serviced for twelve years and lack of practice after an interval becomes a more important. There is a little blip of medical factors in diving deaths that occur at the beginning, but it is pretty small. I think we have to remind ourselves constantly at this sort of meeting that what we are concentrating on here is nothing more than a pimple.

I think every diver should have a medical examination before diving. I think it should be a proper medical examination, not a Mickey Mouse medical.[1] I think it should be done for you in Australia according to AS4005.1. I think that this standard should outline the

elements of history, examination and investigations that should be performed on every diver. I think any diver identified as having a problem within the standard should be seen by a diving medicine trained doctor, and I think we should never use the words fitness to dive, pass or fail, ever again. They are unscientific, unworkable and a load of old rubbish
.

We have to remember that the administration of inappropriate concepts cost us a lot. Divers do not tell the truth. We, as doctors, lose dive industry respect and they no longer seek our advice on particular issues. I think, too, that we lose the respect of scientific colleagues. So what then should our divers expect of us? I think that they should expect that the medical examination should be performed in a competent manner. The medical should be valid as detecting the potential risks. The doctors should be knowledgeable and articulate in assisting the patient in assessing the risk to themselves, and if they are not able to do that they should know to whom to refer. Doctors should be supportive in the diver's decision and then act to minimise the risk. In other words, this is a matter of risk assessment and informed consent.

There are wider issues in risk assessment than just to the individual, who may die, may become seriously frightened of the water, his wife might have problems. There is also the risk to the dive buddy to be considered. If one has just had somebody disappear from the buddy pair then this can lead to significant long term problems. The buddy may be placed at physical risk in trying to rescue an asthmatic on the surface for example. There are risks to a training organisation and these are legal risks to their reputation and for both of those read income. There is one body where there is limited risk and that is the rescue organisations who exist only to retrieve people and the more people they retrieve, the more likely they are to survive, however included in this are risks to recompression attendants and others that are on the receiving end, and those who are in a single engine helicopter who have to fly off shore to retrieve somebody. There is an issue of cost to insurance companies, government and armed services, and there is a risk to doctors both in terms of medico legal settlements and to our reputations. All of these need to be taken into account in the risk assessment.

Risk assessment involves two people who have a knowledge of the problem. It cannot be done by the doctor alone. Here are two examples where the medical risk is identical. A twenty two year old who has just won a grant of two million dollars over five years to study the sand living population off Castaway Island develops asthma with a bit of a chest infection. If I was this person I would be here next week. However, as a forty year old diver with seven children, a wife who is bigger than I am, a $250,000 mortgage with a big business loan I might elect not to dive. The medical risk has not changed, but my assessment of that risk, as an individual, has changed.

Who should do the medical? I actually believe that the patient's normal GP is the best placed to do the initial examination. They have got past records, and in the case of asthma, which is an intermittent disease that may not be present when the diver sees a remote doctor as a one off examination. I think that there should be some guidance about what should be performed in the examination because these general practitioners may have no knowledge of what is important in diving medicine. I think the term contraindication and relative contraindication should be replaced by indications for referral, and it should have no other implications than that. Anybody identified as having an abnormality, whatever that might be, should be seen by a trained diving medicine doctor, who would interpret the abnormality in the context of thorough training. They may wish to request additional investigations to help them to assign risk more correctly, and if they are not able to do that then they may wish to refer to somebody who may be able to better stratify risk. And because the specialist knows a hell of a lot about the disease and not usually a hell of a lot about diving except for Fred Bove and myself, our addresses will be available afterwards, the diving medicine trained doctor would be able to interpret the specialist opinion in the light of diving medicine knowledge. And I think that this sort of scheme is practicable and workable and that having everybody seen by a diving medicine trained doctor is impractical.

I think that this conceptual change can occur only if the medical form changes. At the moment we put our career on the line by saying that this person is fit or unfit to dive. That implies to a lawyer that if we say that somebody is fit to dive, that they are at no greater risk than somebody without that condition in the environment, and as you know, for every doctor who stands up and says there is no increased risk, there are ten paid doctors who will say the opposite. I think that this is allowing us to assume a medico legal risk where the risks properly belong with the patient and with their training organisation.

In my initial attempt at a revised medical I included a statement that the examination has been performed according to whatever Australian standard is in force. When one has explained the medical risks, identified how those risks may be minimised to the patient, it is important to have some statement about the patient's understanding of that discussion and one should document carefully in the medical record what the discussion was, and then whether you felt it was acceptable. I would put this in the medical certificate because I think many diving medical problems become apparent only after the diver has been diving for a few times.

Particularly ear and sinus related problems. I have not yet had somebody that I could not train to clear their ears adequately. But if you try and teach that before they have an understanding of the pressure dynamics of diving, or if they read it from a book, it is very hard to do. It is also

important to get them early before they have learned bad habits which may later rupture a round window.

Medicals should be done before starting the course. Many intending divers come to me part way through the course after they have paid their money and after our discussions they decide not to proceed. There needs to be some statement to the training organisation that allows the budding diver to get a refund. The annual medical I have no strong feeling about. I think, personally, that it is only worth doing as one gets older.

For screening to be useful there has to be a method of diagnosis. The diagnosis has to usefully change what we do. Changing what we do should change outcome and then we have to decide does the changed outcome justify the costs.

A study done in Auckland on a large number of seven year old school children showed that 27% of people, with no wheeze or who had never wheezed or with only wheezing in the past, had non-specific bronchial hyperresponsiveness. There was a statistical relationship between the degree of hyperresponsiveness and the degree of symptoms.

Malhotra and Wright showed that transthoracic pressures of not a lot could rupture lung. But this effect could be prevented by binding the thorax and binding the abdomen.[2] Now that says that pulmonary barotrauma is not a pressure phenomenon but is due to alveolar over distension, lung over distension. Colebatch looked at a group of divers who had suffered a cerebral arterial gas embolism and showed that they had areas of varying lung compliance, but so did a lot of the control groups.[3] Pearson and a number of others at a range of different times showed that the only abnormality of lung function associated with or that could predict cerebral arterial gas embolism in the submarine escape training tank was small lungs.[4] Asians, Indonesians, and particularly short navy men. The FEV_1/FVC ratio did not, the FEF_{25-75} did not, nor did gas trapping as shown by residual volume/TLC ratio.

Macklin and Macklin did a superb scientific study showing the alveolar over distension in unrestrained calf lungs would result in alveolar rupture.[5] Because it was unrestrained calf lungs it cannot be applied in the restrained situation within the chest. However, they did predict a number of interesting things such as that pulmonary barotrauma would be more likely in those who were hypovolaemic and that was subsequently shown in an intensive care unit, and the reason for that was that alveoli abut pulmonary vessels and if the vessels are contracted and small due to hypovolaemia, then the relative shear forces between the alveolar base and the blood vessels increase. However, I do not believe that is important when talking about pulmonary barotrauma in diving .

This may be more relevant. The bronchovascular bundle moves out through the lung from the central airways right to the peripheral part of the lung and it is surrounded by air containing alveoli. What we tend to forget is that bronchial smooth muscle does not actually run in a circular manner, but spirals down the airway. In the proximal airway there are multiple layers of smooth muscle. But as it near the periphery it becomes first a single layer and then a discontinuous single layer. During an episode of bronchospasm the airway thus has a tendency to shorten. If, at the same time, the lung is over inflating either generally or regionally, the bronchus is being pulled apart so that there is an increased distractional force on the airway. It makes sense that in areas of weakness in the wall the inside mucosal lining may rupture out through the wall and if it ruptures into the peribronchial space the person develops mediastinal emphysema, if it ruptures through into the pulmonary vein then one might get arterial gas embolism. Alveolar rupture is unlikely to explain the sort of radiology that shows the heart, brain, thoracic blood vessels full of air. But this mechanism might because bronchial air will be under some positive pressure and the vessel is large. Have we ever seen this in a diver? The answer is no, but we have seen this in asthmatics. Often, a bronchial duct will rupture and I have a slide, which we keep in a safe because it is the only one I have, which shows an alveolar duct permeating through the wall of the bronchus with peribronchial air. We have not found one breaking into a vessel. So this is as much speculation as the other things one has heard. The reason why pathologists may not ever find this is that this may be one terminal bronchial anywhere amongst millions, and there may be no sign. So that may be one of the contributors. These things lead to the comments that diseases which cause air spaces within the lung, regional gas trapping or areas of regional poor compliance may increase the risk of pulmonary barotrauma.

So we now come to asthma. What might the risks of asthma be on the diver? Well the first, and the one that was most often touted at the beginning, was pulmonary barotrauma. Secondly, and significantly underrated initially, was the fact that an asthmatic might develop asthma during the dive. We have heard already that asthma can be induced by cold air, by dry air, by hypertonic saline and by exercise. Thirdly, they might have a significant limitation of exercise, and close to half of those nine asthmatics died of drowning in the series by Edmonds and Walker.[5] There may be a physical risk to the buddy on the surface and there may be a risk of the drugs increasing the risk of decompression illness. This has been shown, in the case of aminophylline, in laboratory animals where bubbles which would otherwise have been filtered out by the lungs can appear in the pulmonary vein, which is not the case in the absence of aminophylline. The work has not been repeated with a β–agonist but there is no reason why this should not also apply to a β–agonist.

Then like all good doctors, having got a good theory we tried to think about what the mechanism might be, and then how we might prevent it. Unfortunately we forgot the intervening steps. What are the actual risks? Can the risks be reduced by better targeting and is it worth doing?

I think it is possible to put asthmatics into various bands of risk while diving. I have absolutely no idea where the line should be drawn between any of these bands, but one can generate a list of increasing severity. I think there is no doubt from the data that we have available, that having atopic asthma, a past history of asthma or a wheeze with a respiratory infection but normal lung function will place people in the low risk group. There is equally no doubt that some people, who have abnormal lung function between episodes, particularly if there is a high residual volume, marked bronchial hyperresponsiveness, or if they have been in a critical care unit, probably should not dive. Where do we put the bands in between? People with daily symptoms are likely to be at greater risk than if they do not have daily symptoms. People who require continuous medication for control may be at greater risk than if they do not require medication for control. But that is not to say that somebody who uses ventolin twenty times a day in order to stay well and who would be better on a prophylactic medication might be at lower risk. I think we can in every disease state roughly categorise people in some sort of hierarchy of risk.

This is a statistical exercise which I think is quite important to have clear in ones mind. Twenty five percent of New Zealanders have had wheeze at some stage of their life. If one assumes a population prevalence of five to eight percent of adults having current asthma, then if we took an unselected population off the street and turned them into divers we would expect five to eight percent of the divers to have asthma. We would expect about five to eight percent of the dead divers to have asthma if asthma did not contribute to death. We observe, if we take the most, the worst figures available which are those of Edmonds and Walker[6] of nine percent of dead divers having a history of asthma, and if we then take the low range of population prevalence at worst it might be that an asthmatic diver has twice the risk of dying that a non-asthmatic diver has.

If we said that most sensible adults who had asthma and wheezed were a bit frightened of diving because of their efforts on the surface, then perhaps only one percent of divers have asthma. If asthma did not contribute to death then one would expect one percent of the dead divers to have asthma. We observe 9%, therefore the relevant risk is nine times.

If we assume that doctors are very good at screening out asthmatics in the medical examination, maybe only 0.5% of divers have asthma, which might raise the relative risk to eighteen times. If we assume that every intending diver was sent to respiratory function lab and did ten challenge tests to ensure that nobody with hyperresponsiveness became a diver, then maybe the prevalence amongst divers would be 0.1%. I do not think one could ever get it much lower than that because this is an intermittent disorder. That means that the relative risks might be ninety times. Now in New Zealand we train 7,000 new divers each year. They perform ten dives each on average in their first year, so that is 70,000 neophyte dives done in a year. If one includes all the old buggers, then there are lots of dives done, of which ten to twenty die and eighty end in a recompression chamber. For those of you who are not good at mathematics, I must remind you that even ninety times a very small number is still a very small number.

So, in summary, I think the current Australian standard has to go before it gets set in stone. I think there needs to be a new medical consent form. I think we need to be assessors of risk and provide informed consent. I think our divers should have the confidence to come back to us after they have had a bypass, or after they have had abdominal surgery, or after they have had a pin put in a leg, without the fear that we will make them “unfit for diving”, therefore not tell us in a situation where really someone should know, like the thyrotoxic who had hypoparathyroidism. I think that we have to get some science back into the diving medical.

Discussion

Knight

I dislike AS 4005.1 too although I was responsible for getting it through the committee. The problem is your assumption that every GP is interested enough to learn something about diving medicine so that he remembers to look in the ears. We found that this was not so, which is not surprising seeing that diving medicine only gets a mention in one or two medical schools.[1] That was the medical reason for producing a standard so they had a list of things to do. The problem of changing a standard is that the person wanting to change it has got to influence a number of groups, the doctors, which you have done, the instructor organisations, who do not want proper diving medicals, that you may be able to do, and the government regulatory bodies that sit on this committee who really do not know very much about it except that they know that people die when they are diving and that is bad for the tourist industry. I would gladly resign my seat on the committee that looks after AS4005.1 to Andy Veale if he would like to take over and start the process of changing it, because I think he is quite right. It does need changing, but one has got to remember the inbuilt traps in the fact that Australian medicine does not produce any training in underwater medicine in its undergraduate course.

Veale

I quite agree. The comment that I would make is that it should be relatively easy, I hope, to modify the standard. I think GPs without a knowledge of diving medicine do need guidance about what is important. I have no argument with that. Where the problem occurs is in them saying that there should be absolute contraindications, relative contraindications etc. I think they should be indications for referral. The other comment I must make is that I have resigned sixteen of my seventeen committee memberships, the last one just before I came and I am definitely not picking up any others, particularly with government attached to them.

Acott

My incident monitoring study is a demographic picture of what is going on in the recreational diving industry. In the first 1,000 reports I have had about twenty reports involved asthmatics. Some of these people have ended up in the morbidity ranks, but the majority of them involved rescue services when they got breathless on the surface and could not get back to the boat.

Davis

I was asked to pass onto this meeting and this workshop this letter from New Zealand Underwater, which is an encompassing body in New Zealand which does not represent any particular training body these days.

"We ask you to put forward our feelings that within New Zealand the status quo remains as is, to be more precise the potential diver visits their own GP. If there are any doubts about their fitness to dive they are referred to a doctor on the list supplied by SPUMS. This referral is made by either the GP or the instructor. The printed SPUMS list of members trained in underwater medicine is also used as a referral by New Zealand Underwater when receiving enquiries from GPs for further information. Any variations to the above system would be seen as fixing something which is not broken. We are convinced that there is a very low or non-existent number of diving deaths or injuries which could be remedied by altering medical examinations in any way."

Unidentified speaker

Changing the Australian standard obviously needs to be done because it is a load of nonsense at the moment, but one of the things that I have had trouble with in Perth is trying to get respiratory physicians on side. Although a lot of them are dead keen, most of them have read the Thoracic Society guidelines and say “Oh well, we cannot let asthmatics dive. Will there be some mechanism for taking back information from this meeting to the Thoracic Society and say that we need to get into to the 90s rather than staying in the 60s?

Gorman

Do you know the names of the authors of that policy? I know one of them was Anderson. Another one was not Pork, it was

Veale

I have to tell you that the Thoracic Society of Australia and New Zealand (TSANZ) document on diving medicine I found the most difficult negotiation that I have ever taken part in, except coming to this meeting without my wife. The starting position of the TSANZ was that every patient, or rather intending diver, should have full pulmonary function including challenge testing and that is only a slight exaggeration. It really was a very difficult thing to get it as moderate as I thought the statement was, and at that stage the DAN data had only just begun to come available, reporting in an abstract that the relative risk may be as high as two times, but with a wide confidence interval. I think the data is much better now, and the reason that this was not put out as a TSANZ position paper was because of the lack of data. It started off as a TSANZ position paper, which as you know are extremely well researched and authoritative, it became a discussion paper when it became apparent that the data was hardly available

Unidentified speaker

Which asthmatics, if any, would Dr Veale see fit to certify to dive at present?

Veale

I do not think we should certify anybody fit to dive. It is an archaic concept of the ignorant. I think that one can advise asthmatics with current symptoms and certainly those with abnormal lung function that they are probably at a much higher risk than if they do not have those things present. I think one can say to somebody who has features that suggests bronchial hyper-responsiveness, like night cough, cough or wheeze with exercise, that they are almost certainly likely to be at a greater risk of getting into trouble on the surface and placing their buddy at risk. I think that the others are at lesser degrees of risk, and as I indicated, I think that whether the individual accepts that risk or not depends much more on their circumstances than our current level of knowledge.

My current practice is that I sign the bottom of the form that says this person is fit or unfit and if somebody has asthma, I do not certify them fit, and that is because we, if we did so, we would be laying ourselves open to an indefensible medico-legal claim.

Unidentified speaker

I would just like to make a simple statement in support of your views. The only mammals fit to dive are whales, dolphins and seals. Man is a land based, one atmosphere air breathing mammal and by definition is not fit to dive. We should be making a risk assessment.

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This paper is an edited transcript of Dr Veale's lecture, to which he has not objected.

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ASTHMATIC FITNESS TO SCUBA DIVE

Peter Chapman-Smith

New Zealanders enjoy their marine environment. With more trained scuba divers per capita than any other country, and many untrained ones too, diving is a major recreational sport as well as a means of food gathering for many. Sadly between 55 and 76 cases of significant diving illness are treated annually in NZ, with a steady trickle of fatalities annually. These occur more during summer with other deaths from snorkel or free diving.

Asthma occurs in about 10% of the general population and has been said to be associated with approximately 10% of scuba diving deaths in New Zealand and Australia between 1980 and 1987,[1] the actual cause of death often being unclear. But the more recent data certainly has much lower figures of association.

Conventional diving wisdom has declared that asthmatics should not scuba dive at all. The potential risk of barotrauma to the lung from inadequate emptying of the small airways and reduced lung compliance of ascending divers who suffer from asthma may well be unacceptable. The suggested increased risk of pulmonary barotrauma (PBT) on ascent for asthmatic divers is based on consideration of, reduced lung elasticity, greater residual volume, greater resistance to exhalation, variable expiratory time constants of exhalation from alveoli leading to small airway closure with air trapping (closing volumes exceeding functional residual capacity), while exertion, hyperventilation, breathing cold dry air, saline mist inhalation through a faulty regulator, anxiety, increased gas density, increased effort of breathing and wetsuit splinting of the chest (which can be claustrophobic) can all precipitate or worsen asthma. Pulmonary barotrauma is occasionally associated with lung cystic changes. There is a greater risk at shallow depths where the volume changes are larger and rapid ascents are more risky. It is clinical experience that many cases of PBT occur without obvious cause. The potential outcomes include pneumothorax, arterial gas embolism, or mediastinal emphysema. Fatal at worst, with minor to major long term disability at times. Recompression treatment can be difficult and may not be successful. Serious stuff from a leisure sport and quite reasonably diving physicians have taken a conservative view for decades. It is fair to say that there is little hard clinical data to support this advice, and to my knowledge no one has yet demonstrated by section at post mortem the actual pathology of such pulmonary barotrauma.

In the UK a more liberal view has embraced selective risk assessment, with low risk asthmatics allowed to dive if not suffering symptoms for 48 hours before diving.

And how long after wheezing does the label of "asthmatic" linger with one ? All that wheezes is not necessarily asthma. Certainly asthma may also be over diagnosed and over treated in general practice. Bronchial hyperreactivity is well accepted as an entity and of course many asthmatics ignore our advice and continue to dive anyway. Some do so for long periods and are apparently none the worse for it. However, some join the morbidity and mortality figures as well, the non-survivors are not present to put their case.

Prospective dive trainees ideally require a medical clearance from a diving physician. Purists suggest excluding all those with with a history of asthma in the preceeding 5 years, bronchodilator use within 5 years, expiratory rhonchi heard on auscultation, high pitched rhonchi on hyperventilation with the mouth open, high pitched rhonchi 5-10 minutes after exercise and poor respiratory function tests. These are discussed in Sandra Anderson's paper (pages 233-248). An asthma provocation test causing more than a 10% reduction in FEV_1 (a 20%

reduction would verify clinically significant asthma) has also been used to exclude people from diving. The diagnosis of asthma should be supported by a positive reaction to bronchodilator.

Respiratory fitness to allow scuba diving "safely" requires the ability to tolerate severe physical exertion, the ability to tolerate rapid changes in lung volumes and pressures, with equal compliance throughout the lung, no restriction of local airways, no cysts or fibrosis, no current use of bronchodilators (which can be arrhythmogenic and aminophylline is known to reduce the bubble filtration function of the lung) and normal airway resistance

Working in general practice, I have been doing bronchial challenge tests with hypertonic saline using the equipment described by Dr John Parker in the SPUMS Journal.[2] This technique involves the inhalation of 4.5% saline via an Omron NEU06 Model nebuliser with a suitably high output of approximately 1.5 ml per minute.

Subjects

Twenty three subjects were studied, 3 female and 20 male, between the ages of 13 and 51 years. Most were prospective scuba trainees, often referred,to confirm suggested diagnosis of asthma, by colleagues and instructors. Several active asthmatics on regular inhalational treatment were included for comparison.

In each case a medical and diving history was taken. An examination was undertaken to establish the diagnosis. After determining the need for a saline challenge I frequently tell these people that they should not be diving, but they insist on taking the test and some sail through with a negative result! Repeated baseline spirometry was done using a hand held Micro spirometer until reliable data for forced expiratory volume at one second (FEV_1), forced vital capacity (FVC) and peak flow were recorded, then the test was conducted over approximately 30 minutes. Further familiarisation with equipment such as the nebuliser preceded testing. A nose clip was not used.

A twenty percent drop from the base line FEV_1 was accepted as a positive test, or a failure for diving. This figure represented a significant level of reduction where the test would be considered positive and abandoned. Equipment for the treatment of provoked severe asthma was ready but in the twenty three cases the only one person needed treatment and three puffs on a Ventolin inhaler reversed the bronchospasm.

Inhalation testing was with the trigger demand flow provision of nebulised 4.5% saline. Sequential challenges lasting 30, 60, 120, 240 and 480 seconds were given with 3 minute breaks between. During these breaks, spirometry was performed at 30, 60 and 120 seconds. When the FEV_1 fell 20%, a positive result, the test was stopped but spirometry at 60 second intervals was continued until recovery was evident.

Asthma history

Sixteen had been diagnosed with asthma under the age of 10 years. Four since the age of 20. The last episode of asthma was less than 5 years ago for 21, less than 3 years for 16, and for 11 was less than 12 months. 11 had a history of allergic rhinitis. Twelve had exercise induced asthma while 13 had URTI induced asthma.

Asthma medications

Seven had no medications for the treatment of bronchospasm. Six were prescribed prophylactic steroid inhalers. One had been on prednisolone when aged 12, 2 years before being tested (Case 17). Thirteen used bronchodilator inhalers irregularly. Four were using them regularly (Cases15, 16, 21 and 23). One of these had been treated for status asthmaticus 18 years before, had been on prednisolone on about 3 occasions, admitted to wheeze on exertion even in the water, is bilaterally deaf, suffers from intermittent depression, has patchy sensory loss of the lower limbs and perineum, and has been an instructor for years (Case 23). Another was a very heavy smoker (Case 16).

A third was a tourist, whom I advised to not dive, but who intended to proceed anyway despite the risks. She was on regular treatment for asthma (Case 21) and had a negative test.

Dive experience

Thirteen were experienced snorkel divers. Sixteen had not dived on scuba. 5 had scuba dived on less than 4 occasions. 2 had active asthma but had scuba dived often (Case 15 for over 2 years and Case 23 for over 30 years).

Examination findings

Nineteen were entirely normal while four had a wheeze (Cases 10, 16, 20 and 23). Two wheezers had negative provocation tests (Cases 10 and 20). One of these had had a heart transplant, smoked and was functionally normal with unremarkable spirometry (Case 10).

Peak flows at the start were notable in that one was 66%, failing the test (Case 23), 2 were less than 75% with neither failing (Cases 9 and 12) and 18 were better than 80%. Initial FVC recordings were close to that predicted and often better as were initial FEV_1.

Cough and wheeze during testing

Ten were noted to cough, complain of chest tightness or have an audible wheeze during the test. Three of these had positive test outcomes (Cases 2, 16 and 23). It is quite clear that many people with normal lung function devlop a cough during this test.

Outcomes

Four were positive, that is dropping 20% from the baseline FEV_1 (Cases 2, 15, 16 and 23). All those who failed had had symptoms of asthma within the previous month. Case 2 was allergic to cats and horses only and admitted to no other triggers, so presumably truly allergic asthma. The last attack of asthma was three week before the test which was rapidly positive. Case 15, on regular asthma treatment, had dived frequently for 2 years without apparent problems. He had a positive challenge despite being on regular inhaled steroids. Case 16, with a 38 year-pack history of smoking, had chronic obstructive airway disesase (COAD) and responded with wheezing to all the usual asthma triggers. He was very keen to go diving but failed rapidly with a positive challenge. Case 23 had longstanding atopic asthma and his history has been described. In recent years he has been well controlled and has had few problems.

Nineteen tests were negative. Seven of the negative challenges had had symptomatic asthma in the last 12 months. All 7 teenagers in this study had negative challenges. One 14 year old girl had been on oral steroids for asthma 2 years previously and responded with wheeze to all the triggers. Symptom free for 12 months, she was on no regular treatment and surprisingly had a negative challenge (Case 17). A man aged 22 years had had a heart transplant in the UK and been pronounced fit to dive 4 months before at the Great Ormond St Hospital in London. He had a positive family history of asthma, smoked, only wheezed with colds and had a negative challenge result (Case 10).

Twelve subjects admitted to exercise induced wheeze all with negative challenges except for Case 23 (see above) and Cases 15 and 16, who smoked. All three on history and examination alone would normally have been excluded from diving.

Discussion

Exercise induced asthma seems to be caused by osmotic changes in the airways rather than a temperature effect. Water loss per se may be the key stimulus. Swimming hard in a 1 knot current is not unusual and increases the work and volume of breathing. A clinical observation of rhonchi on auscultation (especially after hyperventilation) or a progressive drop in spirometry after exercise would confirm the likely diagnosis of asthma.

Inhalation testing standardisation demands a consideration of nebuliser output, particle size, method of inspiration, airway calibre, drug usage, recent viral infection, exposure to irritants or allergens and individual characteristics. The solution used is critical and either hypo or hypertonic saline solutions are reliable. The equipment must be thoroughly rinsed after use or corrosion will occur. Non-asthmatic subjects do not suffer a 20% or greater drop in FEV_1. Inhaled temperature between 22° and 35° C. seems to not make any difference. Solutions need to be sterile and any bronchoconstriction caused can usually be readily reversed. Lung irritant receptors may be directly stimulated by the altered osmolar solutions inhaled, with subsequent mast cell release of histamine in the bronchial mucosa.

A 20% fall in FEV_1 is considered a positive response, correlating well with the reduction in flow rates in the middle half of the vital capacity. 80% of asthmatics will respond to a cumulative dose of 10 ml or less of either water or 4.5% saline. 40% respond to 2 ml or less. Droplets of hypertonic saline undergo hygroscopic growth in the airways and cross membrane ion fluxes may well be altered. A direct action on bronchial smooth muscle or on afferent vagal nerves is postulated. Although very specific as a test stimulus to detect moderate and severe hyperresponsiveness, hypertonic saline is less sensitive than methacholine and histamine.

Non-isotonic aerosols induce changes in lung function reflected in FEV_1 estimation. The maximum response is usually seen within 60 seconds of a challenge, those patients with an initial FEV_1 of less than 80% of predicted value can be expected to respond to less than 2 ml of inspired saline.

On my figures I cannot agree with the suggestion that we should exclude anybody with a history of asthma in the last three to five years. A saline provocation test is needed. However active ongoing asthma is a clear absolute indication of unsuitability to dive. Saline provocation is cheap, reproducible, safe, quick to do, and if inducing asthma makes an explanation of diving unfitness easier. The similarity to the inhaled mist through a regulator is readily understood. False positive results are virtually unknown, asthmatics with symptoms having a correlation of virtually 1, that is a high reliability and sensitivity. A positive result correlates highly with exercise induced asthma.

To pronounce fitness for diving in prospective dive trainees is an outdated concept. Informed consent is the current approach promoted in Australasia. A wide general awareness now exists amongst general practitioners who perform most dive medical screening in New Zealand that

active asthma is a contraindication. Determining the need for ongoing treatment or prevention of asthma should be reassessed from time to time and the diagnosis of hyperresponsive airways (or bronchial hyperreactivity) needs to be remembered more often.

There are grey areas in the consideration for diving fitness. One active asthmatic has clearly not escaped injury, but continued (and thoroughly enjoyed) diving for years. Is there a case for allowing diving occasionally if one has excellent lung function tests but takes a prophylactic inhaled steroid? (Budesonide especially appears very effective.) If one only wheezes and gets asthma after a specific allergen challenge (such as riding horses) should this exclude the individual from ever scuba diving? Is exercise induced asthma the main diagnosis of exclusion? How many divers have asthma and ignore conventional wisdom? I believe we should be studying that group in much more depth.

A five year asthma free period seems unreasonable for adolescents who often outgrow the disease. Active asthma in the last month appears to be a useful marker. Those with significant asthma still fail the provocation test despite being on regular inhalational treatment. As the actual risks and consequences of pulmonary barotrauma in asthmatics are in fact not well described, perhaps they can be ignored in those who pass a saline challenge. The paucity of clinical data is notable, but ignoring the theoretical risks and consequences of pulmonary barotrauma seems unwise. Guidelines for examining doctors should perhaps urge dividing trivial from more serious asthma. A continuum of risk exists, and perhaps an informed consent approach could be adopted allowing some recreational diving to a wider public. Certainly this would be welcomed by many in the dive industry, but the safety of this advice is ill defined at present.

Questions

Mike Davis, Christchurch

I was not quite clear what your advice was to asthmatics, with a positive history and on medication, who had a negative challenge test with regard to their diving.

Chapman-Smith

The reason it is not clear is I did not mention it. I thought it would be interesting to discuss, rather than say what I had done. In fact I suggested to those who had a negative test that they could do a dive course, after adequate discussion of the risks of barotrauma, which is a dilemma because a number of those people I would never, before doing the test, have suggested they should dive. So I have changed my advice to patients on the basis of this test .

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LUNG FUNCTION AND BRONCHIAL PROVOCATION TESTS FOR INTENDING DIVERS WITH A HISTORY OF ASTHMA

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(presented by Sandra Anderson)

Abstract

With our experience over 7-10 years in assessing intending divers with a past history of asthma we have concluded that full spirometric tests, bronchial provocation and response to bronchodilator should be performed, together with measurements of functional residual capacity and residual volume, if possible. This combination of tests to assess risk has arisen over time and in consultation with our referring medical practitioners. The choice of bronchial provocation test (pharmacological or physical) may present some difficulty. The use of dry air hyperpnea and hypertonic saline have the advantage of being familiar and relevant to the intending diver and having a high specificity for asthma. The use of pharmacological challenges, while well accepted by the medical community, are less acceptable for the intending diver as the stimulus is not relevant to diving. Further, the low specificity for identifying current asthma may lead to the unnecessary exclusion of some persons with otherwise normal lung function. Occasionally a response to a pharmacological agent is negative but the airway response to dry air challenge positive.

Asthma is an inflammatory disease of the airways that can vary widely in severity over a life-time. In assessing 180 adults with a past history of asthma we have found that 50% had no evidence of the disease and had normal lung function and no bronchial hyperresponsiveness. Others who had been symptom free for some years, had abnormal lung function and/or were hyperresponsive. We

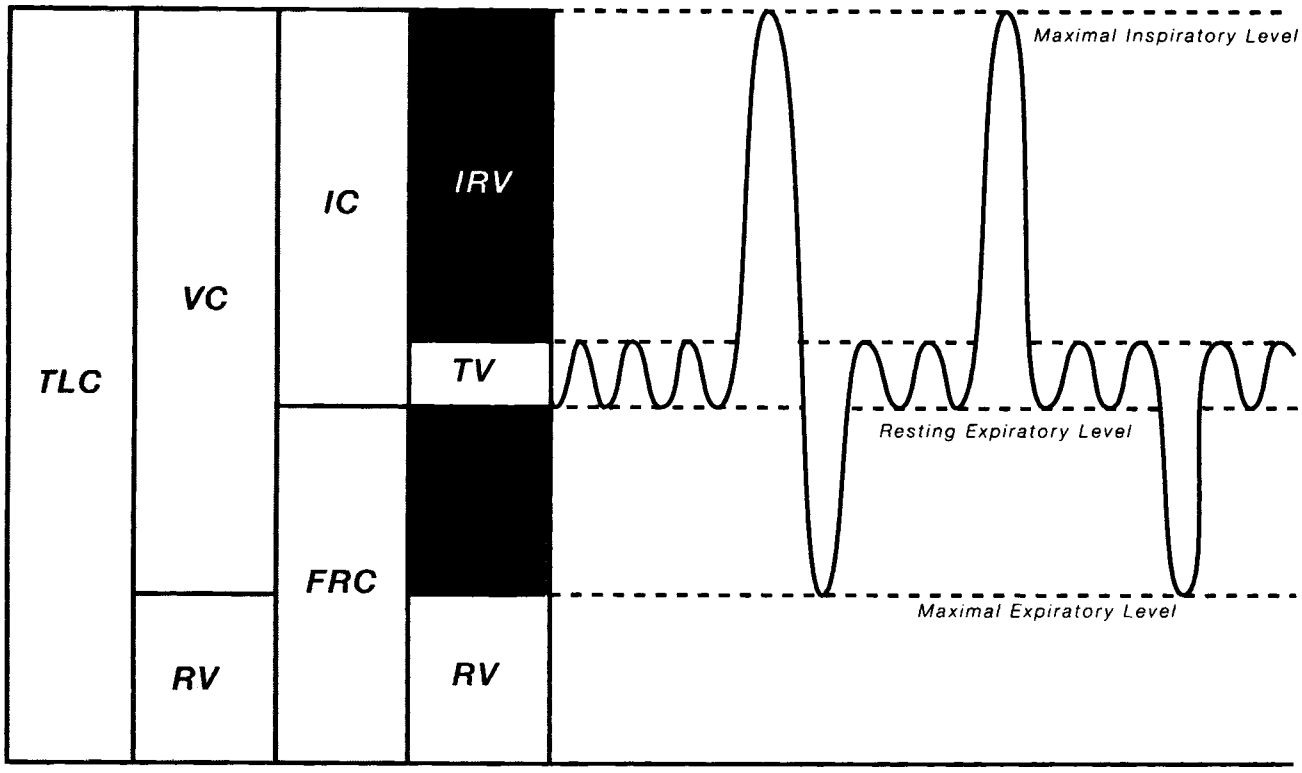

Figure 1. The lung volumes as they appear on a spirogram. After performing a maximum inspiratory capacity (IC) the total lung capacity (TLC) is reached. After a vital capacity manouvre (VC) the residual volume (RV) is left in the lung. When breathing with a normal tidal volume (TV) the functional residual capacity (FRC) is the volume in the lung just before the next breath in. The thoracic gas volume measured during plethysmography is approximately equal to the FRC and TV. The inspiratory reserve volume (IRV) and expiratory reserve volume (ERV) are both utilised to increase tidal volume during exercise but the IRV is used more than the ERV. Taken from Comroe.[5]

believe that persons with abnormal lung function and who are hyperresponsive to the effects of dry air or hypertonic saline are theoretically at an increased risk from pulmonary barotrauma. From our experience of a high rate of abnormality in the referred patients, it would seem cost effective and appropriate to conclude that lung function assessment and bronchial provocation tests serve a useful purpose in identifying people who may be at risk from diving and who may benefit from treatment for asthma.

Introduction

Many candidates presenting for a diving medical examination give a past history of asthma or childhood respiratory illness. This is not surprising given the high prevalence of asthma symptoms reported in the Australian community over the last twenty years.[1,2] For most, the diagnosis of asthma will have been made on history alone with no objective testing of lung function or bronchial hyperresponsiveness, a hallmark of asthma.

It is widely accepted by the diving medical community[3] and by respiratory physicians[4] that persons with a past history of asthma who wish to dive with underwater breathing apparatus, should have tests of lung function and a bronchial provocation test. Such tests usually include the measurement of lung volumes and forced expiratory flow and volume before and after a bronchoconstrictor stimulus or a bronchodilator. These tests have been readily available through most public hospital laboratories for many years. The development of electronic spirometers and easy techniques for bronchial provocation has resulted in some practitioners offering testing at the time of the diving medical examination. This approach has obvious advantages, particularly for the many tourists who wish to dive but have a history of respiratory illness, or, who are heavy smokers.

This paper describes some of the lung function tests and bronchial provocation tests available in major public teaching hospital laboratories in Australia. These tests are discussed for their usefulness in assessing persons with a past history of asthma who wish to dive. It is suggested that specific tests of lung function and bronchial provocation be considered in the assessment of risk of pulmonary complications from diving.

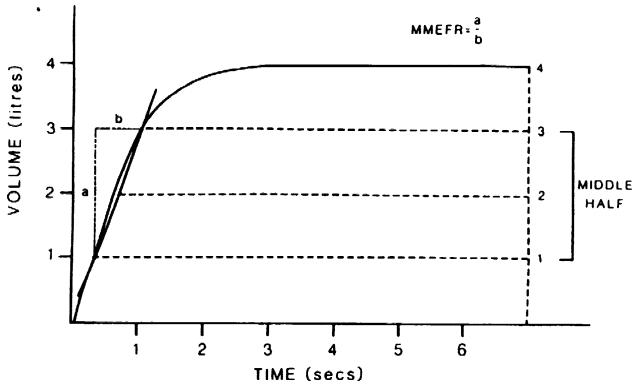

Figure 2. Forced expired vital capacity in relation to time. The maximum mid-expiratory flow rate (MMEFR) also known as the forced expiratory flow rate through the middle half of the vital capacity (FEF_{25-75}). It is calculated from a spirogram by measuring the time (b) taken to expire the middle portion of the vital capacity (a). It is expressed in litres per second. Taken from Alison.[6]

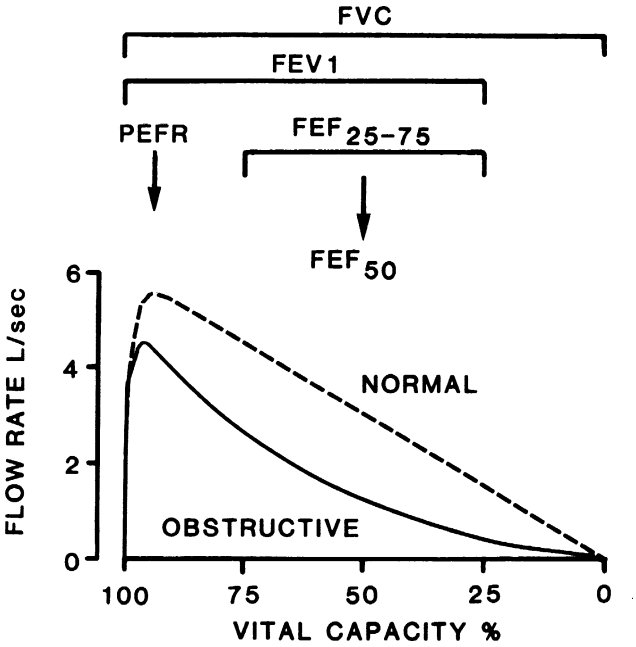

Figure 3. The forced expiratory flow in relation to volume during a forced vital capacity (FVC) manouvre as it would be seen before (normal) and after (obstructive) exercise in a person with exercise-induced asthma or before (obstructive) and after (normal) bronchodilator in a person with acutely reversible air flow limitation. The peak expiratory flow rate (PEFR), forced expiratory volume in one second (FEV_1), and flow rates through the middle portion of the vital capacity (FEF_{25-75}) are shown in relation to volume. In this example the FVC remained the same but the FVC is normally reduced in exercise-induced asthma and is often increased after a bronchodilator. Taken from Anderson.[10]

Spirometry and flow rates

Forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) are the two most common measurements made from a forced expiratory manoeuvre. Traditionally these measurements have been made recording time and volume simultaneously (e.g. bellows type Vitalograph, wet spirometer) and correcting the volume from ambient temperature and water vapour pressure (ATPS) to body temperature and water vapour pressure (BTPS).

Spirometric tests are effort dependent and are only accurate if the forced expiratory manoeuvre to residual volume follows maximal inspiration to total lung capacity and no leaks occur during expiration (Figure 1).[5] The flow rates through the middle portion of the forced vital capacity (FEF_{25-75}), also known as the maximum mid-expiratory flow rate (MMEFR), can also be obtained from an accurate volume-time tracing (Figure 2).[6] It is important that the person measuring spirometry is aware of the criteria used to determine acceptability of the results.[7]

For comparison with normal, a set of predicted values is required. Predicted normal values vary according to sex, age, height and ethnic origin. A value greater than 80% or more of predicted normal is usually considered as within the normal range if all other volume values are a similar percentage. The value of 80% usually represents the 95% confidence limit. Table 1 gives normal predicted values and standard deviations, to calculate the confidence intervals, for spirometry for Caucasion persons of European origin.[8] Non-Caucasians usually have smaller lungs[9] and 80-90% of the predicted Caucasian value is considered within normal limits by most laboratories.

Flow volume Curve

The introduction of electronic spirometers with sensitive flow measuring devices has facilitated the recording of expiratory flow in relation to expiratory volume. The electronic spirometers allow the flow signal to be "instantaneously" integrated to volume and a flow-volume curve is obtained (Figure 3).[10] This contrasts with the volume-time curve obtained in classic spirometry (Figure 2). Simultaneous plotting of expiratory flow against volume has permitted a better assessment of flow through the smaller airways and in the less effort dependent part of the flow-volume curve.

In addition to the FEV_1 and VC values, the flow-volume curve gives the forced expiratory flow rates through the middle portion of the vital capacity (FEF_{25-75}). Due to the increase in density of air with increasing depth these flow rates are markedly reduced with submersion. The changes in flow-volume characteristics have been measured in recompression chambers and an example is

TABLE 1

EQUATIONS FOR OBTAINING NORMAL VALUES OF SPIROMETRY RESULTS FOR CAUCASIAN ADULTS AGED 18-70§

The lower 5 or upper 95 percentiles are obtained by subtracting or adding the figure in the last column from the predicted mean.

Variable	Gender	Measurement	Equation to obtain mean	RSD	1.64 RSD
IVC	F	l	4.66H - 0.026A - 3.28	0.42	0.69
FVC	F	l	4.43H - 0.026A - 2.89	0.43	0.71
FEV_1	F	l	3.95H - 0.025A - 2.60	0.38	0.62
FEV_1/FVC	F	%	- 0.19A + 89.10	6.51	10.70
FEF_{25-75}	F	$l.s^{-1}$	1.25H - 0.034A + 2.92	0.85	1.40
PEF	F	$l.s^{-1}$	5.50H - 0.030A - 1.11	0.90	1.48
IVC	M	l	6.1 0H - 0.028A - 4.65	0.56	0.92
FVC	M	l	5.76H - 0.026A - 4.34	0.61	1.00
FEV_1	M	l	4.30H - 0.029A - 2.49	0.51	0.84
FEV_1/FVC	M	%	- 0.18A + 87.21	7.17	11.80
FEF_{25-75}	M	$l.s^{-1}$	1.94H - 0.043A + 2.70	1.04	1.71
PEF	M	$l.s^{-1}$	6.14H - 0.043A + 0.15	1.21	1.99

Abbreviations

IVC = inspiratory vital capacity. FVC = forced vital capacity. FEV_1 = forced expiratory volume in one second. FEV_1/FVC = ratio of FEV_1 to FVC. FEF_{25-75} = forced expiratory flow through the middle portion of the vital capacity. PEF = peak expiratory flow. l = litre. $l\ s^{-1}$ = litres per second. H = standing height in m. A = age in years. RSD = residual standard deviation.

§: between 18 and 25 years, use thevalue of 25 in the equations.
Taken from Quanjer et al. *Eur Respir J* 1993; Suppl 16: 4-40.

illustrated in Figure 4. [11] The importance of these mid-expiratory flow rates may be appreciated better when it is understood that it is this part of the flow-volume curve that determines the maximum flow that can be reached during exercise. At rest the flow generated during a tidal breath is low but with increasing intensity of exercise the flow increases. The capacity of the flow to increase is determined by the maximum flow-volume characteristics of the lung. If the flow rates through the middle portion of the vital capacity are low, then the ability to increase flow is reduced. This problem is exacerbated with increased density of the inspired air at depth.

It is thought that breathlessness on exertion occurs when the flow reaches the limit of the flow volume curve. If this is the case, it may be predicted that persons with a compromised flow-volume curve may experience breathlessness earlier or for the first time whilst diving. This sense of breathlessness could lead to panic at depth with subsequent rapid ascent.

The normal values for FEF_{25-75} are more variable than FEV_1 or FVC. The lower end of the 95% confidence interval is about 65% rather than 80%. However, any reduction in these flow rates is likely to affect maximum exercise performance. An acceptable normal value for divers for this test may need to be more than 65%, if they need to exercise maximally at depth. Simpson and Meehan[12] have measured FEF_{25-75} in 49 current experienced divers and recorded a mean ± SD value of 99.4 ± 26.2 % of predicted normal for FEF_{25-75} (range 50-164 % predicted). Five of the 49 had a FEF_{25-75} below 65% of predicted, the lower limit of normal.

Significance of spirometry and flow-volume curves

Normal spirometry (FEV_1, FVC, and its ratio) and normal flow rates (FEF_{25-75}) excludes airway narrowing at rest. A normal peak flow does not exclude airway narrowing as it is only a measure of the peak of the flow and does not encompass airflow through the small airways (Figure 3). Normal spirometry does not preclude airway narrowing in response to a provoking stimulus. Many persons with a past history of asthma, but normal spirometry and flow rates, can have airway narrowing provoked by breathing dry air at high flow rates or by accidental aspiration of salt or fresh water. Figure 3 illustrates the

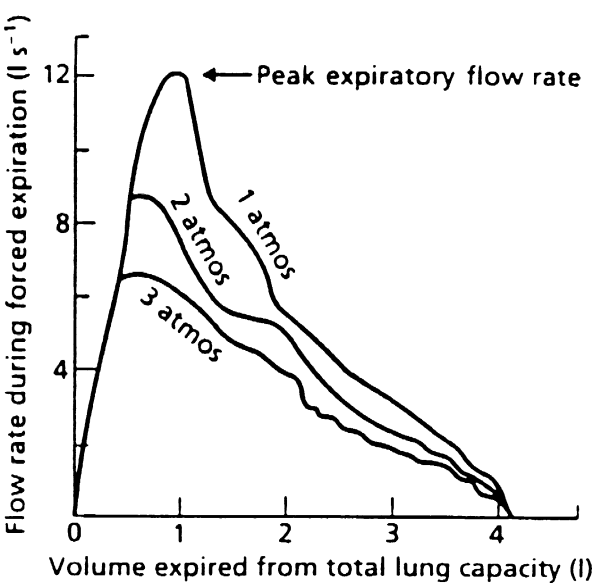

Figure 4. Forced expiratory flow volume curves measured during submersion at 1, 2 and 3 atmospheres (10, 20 and 30 m or 33, 66 and 99 feet of seawater). Note the reduction in peak flow rate and flow rate through the mid-portion of the vital capacity due to the increase in density of the inspired air with depth. It is possible that a person starting with a reduced mid-expiratory flow rate will be limited in their ability to increase expiratory flow rates during exercise at depth. The flow volume characteristics could be further affected by breathing dry air or the inhalation of an aerosol of salt water. In the situation where flow rates are reduced breathlessness and panic could occur. Taken from Cotes. [11]

type of flow-volume curve that would be obtained in a person with hyperresponsive airways. Before a bronchial provocation test the forced expired volumes and flow rates may be normal but after the test the flow-volume curve is concave which represents an obstructive pattern. Such an obstructive pattern may also be recorded in a patient with airflow limitation and this may often be acutely reversed after bronchodilator.

A FEV_1/FVC ratio of 75% or more is usually considered essential for intending divers. This may be unduly stringent as many persons, particularly swimmers and elite athletes, have large vital capacities (e.g.130% predicted) but a normal FEV_1 (e.g.100% predicted) resulting in a low FEV_1/FVC ratio. If the flow-volume curve has a normal shape, it would be inappropriate to suggest that these persons were unfit to dive.

Documentation of abnormal spirometry or flow rates may be all that is needed to decide that a person is medically unfit to dive. However, for the benefit of the person and with a view to treatment, it is advisable to complete the testing to determine if the airflow limitation is acutely reversible. An increase in FEV_1 of 15% or more is considered a significant response to a β_2 adrenoceptor agonist (e.g. terbutaline, salbutamol). It is more difficult to assess responsiveness on the basis of changes in FEF_{25-75}. If the vital capacity remains the same, an increase of 25% in FEF_{25-75} after bronchodilator is considered a significant response. Changes are less easy to interpret if the VC is different after a bronchodilator.

A person with airflow limitation, with or without acute reversibility, should be advised that theoretically, they have an increased risk of problems while diving and they should be alerted to the type of problem. They could also be offered treatment and reassessment to determine whether their airflow limitation is chronic. Chronic airflow limitation is more likely to occur in persons with a past history of asthma who smoke, but untreated asthma, in a non-smoker, can also result in premature and irreversible airflow limitation.

A person with no airflow limitation but values for FEV_1 and VC below normal and a high FEV_1/FVC ratio should be advised to have a more thorough pulmonary assessment to exclude lung disease. Lung volumes were shown to be smaller when a study of victims of pulmonary barotrauma were studied in retrospect[13] so that persons with low volumes may be at an increased risk.

Static lung volumes

In a Pulmonary Function Laboratory it would be usual for a person referred for a diving assessment to have a measurement of all lung volumes (Figure 1). The reason for this is to determine if there is any hyperinflation (abnormally high total lung capacity or functional residual capacity in relation to other volumes) or gas trapping (an abnormally high residual volume in relation to other volumes). The techniques used to make the measurements are most commonly helium dilution, body plethysmography, or nitrogen washout. Values are considered normal in the range of 80-120% of the predicted value. However it would be expected that all the volumes would be a similar percentage. Thus a total lung capacity of 90% and a residual volume of 120% is not normal.

Significance of static lung volumes

The documentation of either increased or decreased volumes, suggests that there would be an increased risk of barotrauma if rapid ascent is required. Demonstrating the presence of hyperinflation or gas trapping with mild airflow limitation would seem sufficient to advise the intending diver of an increased risk. The documentation of normal lung volumes at rest does not preclude the possibility that acute hyperinflation and gas trapping could

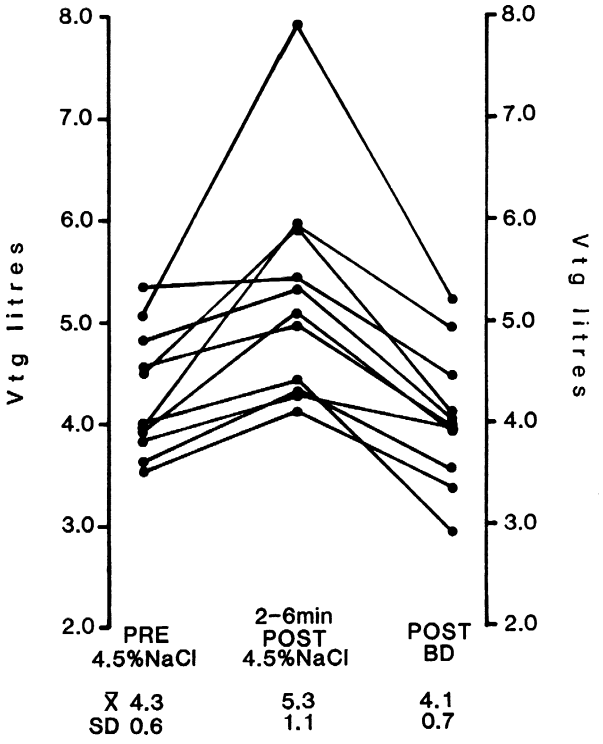

Figure 5. Thoracic gas volume (Vtg) measurements before and 2 to 6 minutes after inhaling an aerosol of 4.5% saline and 10 to 15 minutes after inhaling a bronchodilator in a group of 11 asthmatic subjects. Note the large increases in volume that occurred in some subjects. This hyperinflation occurred at the time the airways were narrowing as a result of inhaling the 4.5% saline. The hyperinflation was quickly reversed by inhaling terbutaline by aerosol (Bricanyl). The thoracic gas volume is equivalent to the functional residual capacity and tidal volume during panting.

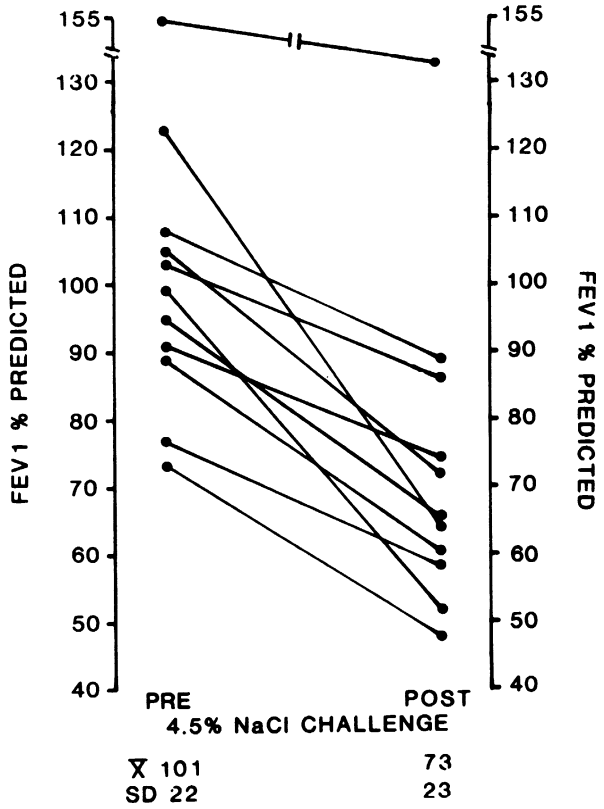

Figure 6. Forced expiratory volume in one second (FEV_1) expressed as a percentage of the predicted normal value in a group of 11 asthmatic subjects before and after inhaling an aerosol of 4.5% saline generated by an ultrasonic nebuliser. For most subjects the FEV_1 was within the normal range (greater than 80% predicted) before the challenge and their lung function at rest did not predict bronchial responsiveness to challenge with 4.5% saline aerosol. Lung hyperinflation occurred in association with airway narrowing to the aerosol and the change in volume

occur in response to bronchoconstricting stimuli such as dry air or hypertonic saline.

Acute airway narrowing associated with acute hyperinflation

The results of 11 asthmatic subjects aged 17-41years (7 M, 4 F) who had measurements of thoracic gas volume (a measurement close to functional residual capacity) in a whole body plethysmograph before and after inhaling an aerosol of 4.5% sodium chloride are illustrated in Figures 5 and 6. Although the reduction in FEV_1 in response to this hypertonic stimulus was abnormal (>15% of baseline) in all subjects (Mean ± SD 28.8% ± 11.7) it was less than 20% in 4 subjects (Mean ± SD, % Fall 17.% ± 1.7). For these 4 subjects there was an increase of 900ml ± 700ml SD in thoracic gas volume when the saline had induced narrowing of the airways. This shows that a relatively small reduction in FEV_1 can be associated with hyperinflation and gas trapping.

Acute airway narrowing provoked by exercise or other stimuli is frequently accompanied by hyperinflation and gas trapping (Figure 7).[14] Indeed hyperinflation acts as a distending force to open narrowed airways. The combination of airway narrowing, hyperinflation and gas trapping would, theoretically, provide a greater risk for pulmonary barotrauma than airway narrowing alone. For this reason, persons with mild narrowing of the airways may not necessarily be at a lesser risk of pulmonary barotrauma than a person who has a greater fall in FEV_1 but who did not become hyperinflated. Thus classification of bronchial responsiveness as mild/moderate/or severe may be misleading when assessing risk for divers.

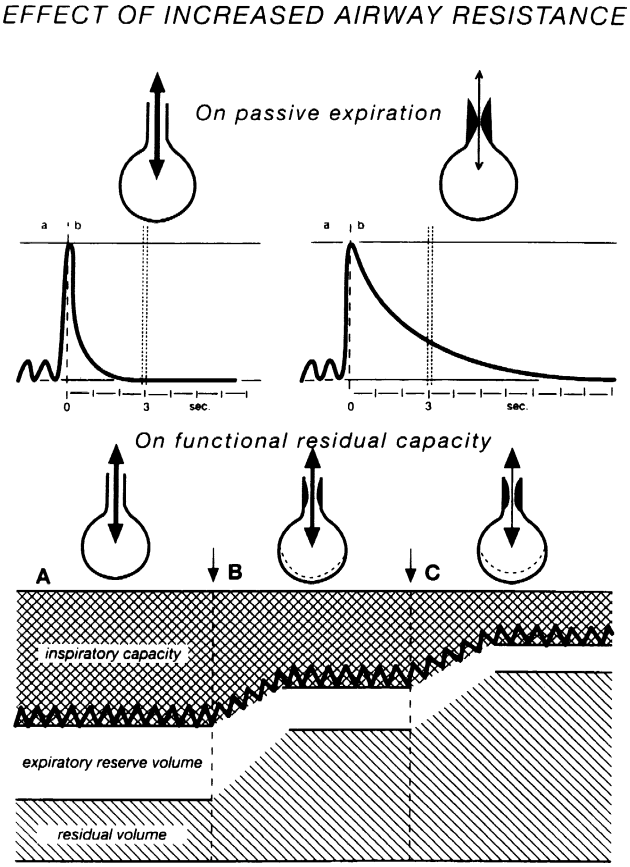

Figure 7. Spirometry and static lung volumes before and during an attack of asthma. Note the reduced inspiratory capacity, expiratory reserve volume, forced vital capacity and forced expiratory volume, and the increased functional residual capacity (hyperinflation) and residual volume (gas trapping). Taken from Comroe.[5]

An indirect method available to most diving doctors to determine the presence of gas trapping is the measurement of vital capacity. As the airways narrow in response to a provoking stimulus, a reduction in vital capacity indicates an increase in volume of trapped gas (increased residual volume). This, in itself, would theoretically put the person at greater risk from pulmonary barotrauma. It is suggested that vital capacity should be measured before and during a bronchial provocation test to determine if gas trapping has occurred or is reversed by a bronchodilator.

Bronchial provocation tests

The role of bronchial provocation tests is to identify persons who would be at risk from acute airway narrowing and hyperinflation when diving. Bronchial hyperresponsiveness (often referred to as bronchial hyperreactivity) can be measured to a wide variety of stimuli. These include pharmacological stimuli or physical stimuli.

Bronchial provocation tests using pharmacological agents such as methacholine and histamine are referred to as direct challenge tests because the administered substance acts directly on bronchial smooth muscle receptors to cause contraction. Thus these tests provide a good measure of bronchial smooth muscle responsiveness to the administered substance.

Bronchial provocation tests using physical stimuli such as airway drying or changes in airway osmolarity are referred to as indirect challenge tests. The reason being that airway drying or changes in airway osmolarity cause the endogenous release of substances that cause the airways to narrow. The presence of these contractile substances (e.g. histamine, leukotrienes, prostaglandins, neuropeptides) and the magnitude of the airway response to them is related to airway inflammation.[15] For this reason the indirect challenges are thought to provide a measure of the cellular and neural contribution to airway narrowing arising from inflammation, the underlying abnormality in asthma.

The range of provocation tests offered by laboratories varies but most offer at least one direct and one indirect challenge test. The diving doctor should ascertain which hospitals provide which challenge tests. It is also useful to know which laboratories use challenge tests with the stimuli relevent for the intending diver (e.g. dry air, hyperosmolar saline). Laboratories such as ours, with an active research interest in asthma, may provide four or more of the challenge tests, e.g. exercise, hyperventilation, hypertonic saline, distilled water, and methacholine. Nowadays, few laboratories offer challenge with histamine.

Advantages and disadvantages of tests

There are advantages and disadvantages in using both the pharmacological and physical challenge tests to identify bronchial hyperresponsiveness in an intending diver. The major arguments to support the use of pharmacological challenges include their high sensitivity to detect airway hyperresponsiveness and the cheapness and availability of equipment to administer the substance, (e.g. jet nebulizer). The major arguments against the use of pharmacological agents are their low specificity to identify asthma, the poor availability of the substances prepared in accordance with British, European or US Pharmacopoeas and the regular need to make up solutions of varying concentration. Perhaps an important argument is the lack of relevance of the stimulus for the intending diver. Further, there is an increasing number of reports of persons, in the random population, being found negative to

histamine or methacholine, but positive to an exercise challenge. This raises the question as to whether the sensitivity of the pharmacological agents to predict bronchial hyperresponsiveness to other stimuli, is as high in the random population as it appears to be in a specialist referred population.

The major argument to support using physical challenge tests is that they represent the stimuli to which the intending diver is exposed. Further, they have a high specificity for identifying persons who have asthma that requires treatment.[16,17] It has been our experience that persons who have a positive response to hypertonic saline are likely to abstain voluntarily from diving and to refrain from seeking another medical opinion.

The major disadvantage associated with the physical challenge tests is that the equipment required to carry out the tests can be expensive and the sensitivity to detect asthma in a general community has been reported to be 50%.[17] This value however is not different to the 53% reported for histamine for a similar population of school children.[18] Persons with a history of asthma, but negative to the physical challenge tests may not have sufficient airway inflammation to have adequate levels of mediators to cause the airways to narrow at the time of testing. It is possible that, for some persons, increasing the strength of the stimulus may render them positive. However it is unlikely that a greater strength of stimulus would be encountered by the intending diver providing that hyperventilation with dry air was performed at maximum ventilation and the concentration of saline used was above that of sea water.

Many medical practitioners would consider a positive response to these challenge tests an increased risk for pulmonary complications from diving. However many persons with well treated asthma in the past, normal lung function and no responsiveness to the dry air hyperpnea or saline will have positive responses to histamine or methacholine. Because of better treatment for asthma it is likely that many persons with this lung function profile will seek to dive in the future.

Medication at the time of test

One would not expect that many intending divers being referred for testing to the laboratory would be taking medications on a regular basis. However, if they are, they should be advised to withhold medications that could affect their airway response to bronchial provocation tests. For short acting antihistamines (diphenhydramine hydrochloride, pheniramine) 48 hours, long-acting ones (terfenadine, astemizole, loratadine) one week; for ordinary preparations of oral bronchodilators, 12 hours and for sustained release oral bronchodilators, 24 hours; for the short-acting β_2 adrenoceptor agonists (salbutamol, terbutaline, fenoterol) and for sodium cromoglycate or nedocromil sodium a period of six hours; the longer-acting β_2 adrenoceptor agonists, such as salmeterol, 24 hours. Inhaled corticosteroids (budesonide, beclomethasone, fluticasone) should be avoided on the day of the study.

Response to bronchodilator

This can be performed using any of a rapidly acting β_2 adrenoceptor agonist such as salbutamol or terbutaline. These are the most commonly used bronchodilators in Australia. Spirometry is done before and after the bronchodilator. The interval between administering the bronchodilator and making the spirometry measurement is important and should be at least 15 minutes, and preferably longer. The response is best measured by FEV_1 and an increase of 15% or more in FEV_1 is regarded as abnormal and consistent with bronchial hyperresponsiveness. In order to ensure that an adequate dose of the drug deposits in the airways we often administer twice the clinically recommended dose by either a pressurised metered dose inhaler or a non-pressurised metered dose inhaler (e.g.Turbuhaler). For persons with a positive bronchial provocation test we usually administer a β_2 adrenocepter agonist in combinations with ipratropium bromide, by a jet nebuliser driven by compressed oxygen.

Methacholine and histamine test protocols

Both methacholine and histamine have been in use in Europe since the 1940s. The most common protocols used in Australia for these pharmacological challenges are those described by Yan et al,[19] and Cockcroft et al.[20] These and other protocols have been recently summarised by Sterk et al.[21] The Yan technique has the advantage of being faster than the others and the nebulizers used are activated by hand rather than by compressed air or electronic devices.

All these techniques require the preparation of a solution of the substance. As doses are low, this normally requires a balance capable of weighing to 0.01 of a gram and sterile containers, pipettes and solutions. The preparation of these solutions has mainly confined their use to public hospitals although some private laboratories do have facilities for preparation. The solutions for the Yan protocol are usually 0.6%, 2.5% and 5.0% and for the Cockcroft protocol 0.05-1.6%. The same strength of solution is prepared for both histamine and methacholine.

Bronchial provocation tests with these substances have been widely used both in the hospital and epidemiological setting to identify persons with bronchial hyperresponsiveness.[1,2,21] Their safety, efficacy and reproducibility have been well established over many years. Their usefulness in identifying asthma specifically

has recently been questioned by the original proponents of the challenge.[22] In population studies between 30% and 60% of persons with positive challenges to histamine have no history of asthma.[1,22] Thus the positive predictive value for identifying asthma is low. It is the high percentage of positive responses to these challenges, without other evidence of current asthma, that makes them less attractive for assessing the intending diver. Further, it is hard to justify the use of pharmacological agents to tourists from countries less accepting of a this approach to measure bronchial responsiveness.

Significance of the results

A positive test result is if the provoking dose of methacholine required to induce a 20% fall in FEV_1 (PD_{20}) is < 3.6 micromols for the Yan protocol although some investigators use a cut-off point of < 8.0 micromols. For the Cockcroft protocol a positive response is recorded if the concentration to induce a 20% fall in FEV_1 (PC_{20}) is less than 16 mg/ml. 8 micromols of methacholine is approximately equivalent to 16 mg/ml. Edmonds[23] suggested that a fall in FEV_1 of 10% or more in response to the inhalation of histamine should exclude a person from diving. This seems to be unnecessarily stringent in that most people, asthmatics or not, will respond to histamine given a high enough dose! Further, as histamine can cause oedema, in addition to contraction of smooth muscle, the airway narrowing in response to challenge may be different in nature from challenges that do not provoke oedema, e.g. methacholine.

There are reports of persons with negative responses to pharmacological agents but positive responses to exercise testing.[24-26] This raises the important question as to whether the documentation of a negative response to a pharmacological agent is an assurance of low risk for airway responsiveness to dry air breathing in a hyperosmolar environment.

Exercise testing

Exercise testing was first used to identify children with asthma and assess asthma severity more than 30 years ago. Studies in the early 1970s identified intensity and duration of the exercise as important determinants of the airway response. By 1978-79 the importance of the water content of the air inspired during exercise was also recognised. This had been convincingly demonstrated by showing that inhaling warm humid air during exercise or hyperpnea completely prevented the airways of asthmatics from narrowing. Since that time it has been appreciated that the major determinants of exercise-induced asthma are the level of ventilation reached and sustained during exercise and the water content of the inspired air.[10] The loss of water, by evaporation from the lower respiratory tract, in bringing large volumes of air to alveolar conditions in a short time is recognised as the stimulus whereby exercise provokes the airways to narrow. The mechanism whereby this water loss acts is thought to relate to both the thermal and dehydrating effects of water loss. Evaporative water loss increases osmolarity of the fluid lining the surface of the airway and probably the submucosa/subepithelium. A hyperosmolar environment enhances release of bronchoconstricting substances from inflammatory cells[27,28] and nerves.[29]

Exercise induced asthma testing protocol

Most laboratories work to a standard protocol[10] that involves the subject exercising sufficiently hard to raise the ventilation rate to approximately 20 times the FEV_1 (50-60% of maximum voluntary ventilation or MVV) and sustaining this for 6 to 8 minutes. Compressed air is inspired via a demand valve and only mouth breathing is used. The FEV_1 is measured before and at regular intervals for 10-20 minutes after exercise.

Significance of the results

If a reduction of 10% or more in FEV_1 is recorded using a standardised laboratory protocol the person is considered to have exercise-induced asthma (EIA), also known as exercise-induced bronchoconstriction (EIB). A value of 15% or more is usually taken as positive for exercise performed in the field. If the test is negative the referring physician should be assured by the laboratory staff that the test was performed with the appropriate ventilation rate achieved and sustained and that the water content of the air was less than 10 mg per litre of air inspired (e.g. temperature less than 23°C and relative humidity less than 50%). Like all bronchial provocation tests, no medication should be taken for the required time before testing.

The temperature of the compressed air inhaled during diving is likely to be less than that inhaled in the laboratory. The cooler air is unlikely to increase the airway response greatly because the inspired air temperature has to be less than 0°C to cause significant enhancement of the airway response. Providing the air is dry there is no significant enhancement in response over the temperature range 9-65°C.[30,31] We use compressed air because it is always dry and it is the same as that inhaled by the intending diver.

Eucapnic or isocapnic hyperventilation testing

This test is a surrogate for exercise and was championed by the US Army to assess recruits.[32-34] The

stimulus and mechanism whereby the airways narrow are thought to be the same for exercise and hyperventilation. Hyperventilation with dry air may be a more potent challenge compared with exercise as the inhibiting factors provided by the increased sympathetic drive are absent.

Hyperventilation testing protocol

The patient voluntarily increases the ventilation rate while breathing dry gas containing 4.9% CO_2, 21% O_2 and balance N_2. The expired CO_2 levels remain the same (isocapnia) and within normal limits (eucapnia) for ventilation rates between 30 and 110 litres per minute. The test can be performed at progressively increasing levels of ventilation (e.g. 30, 60 and 90% of MVV)[35] or at a single level for 6 minutes at a high ventilation rate (30 x FEV_1). We have found hyperventilation a very potent test for provoking airway narrowing in known asthmatics. For this reason we use and recommend a test that comprises progressively increasing levels of ventilation for 3 minutes rather than a single high ventilation rate for 6 minutes for known asthmatics.

Significance of the results

A fall in FEV_1 of 10% or more is taken as abnormal. As diving requires the inhalation of dry gas, sometimes at high ventilation rates, the hyperventilation test would seem an appropriate challenge. Further, asthmatics are known to hyperventilate during exercise and this hyperventilation may be exacerbated by fear or panic making this challenge particularly relevant.

Non-isotonic aerosol testing

In 1980 Allegra and Bianco[36] reported a bronchial provocation test to identify asthma using an aerosol of distilled water generated by an ultrasonic nebulizer. In 1981 Schoeffel and colleagues[37] confirmed the usefulness of this technique. Further they recognised the osmolarity of the solution as important and found hyperosmolarity was also a potent stimulus for provoking the airways of asthmatics to narrow. At the time the mechanism whereby exercise and hyperventilation provoked airway narrowing was thought to be a transient hyperosmolarity of the airway surface liquid. Thus studies were designed to compare responses to hyperpnea with dry air to inhaling aerosols of hyperosmolar saline.[38-40] The studies demonstrated that persons sensitive to the effects of dry air hyperpnea were also sensitive to aerosols of hypertonic saline. This led to a standardized challenge using an aerosol of 4.5% sodium chloride as a cheap surrogate for exercise.[41] As with exercise, there is a period following challenge with hyperosmolar saline, during which the airway response will not be reproduced in response to the same stimulus. This is known as the refractory period and can last for up to two hours.[42]

Non-isotonic aerosol testing protocol

As hyperosmolar challenge combined the natural stimuli of dry air and salt water confronting the diver this challenge has become the popular for assessing intending divers with a past history of asthma. The protocol requires the subject to inhale an aerosol of 4.5% sodium chloride.[43] This concentration of saline is close to sea water. The aerosol is generated by an ultrasonic nebuliser and the rate should exceed 1 ml/minute. If a two-way valve is used the rate should be 1.2 ml/minute, or more, delivered to the inspiratory port of the valve. The FEV_1 is measured before challenge and 60 seconds after exposure to the aerosol. The time of inhalation is doubled for each exposure, 30 seconds, 60 seconds, 2 minutes, 4 minutes, 8 minutes until a 15% reduction in FEV_1 occurs and at least 18.6 ml of 4.5% saline has been delivered to the subject. The dose of aerosol is measured by weighing the canister and tubing before and after challenge or determining the volume loss by subtraction (small nebulizers). If the total time of exposure and the total dose is known, the dose delivered per minute can be calculated and a dose response curve drawn. The dose of aerosol required for a 15% fall in FEV_1 is obtained by plotting the % fall in FEV_1 against the dose (Figure 8). Details of the protocol have been published[43,44] and are available on request. This technique is now widely used in Australia to assess intending divers. It has also been used for epidemiological studies.[18,44] Distilled water can be substituted for the 4.5% saline to study those intending to dive in fresh water.

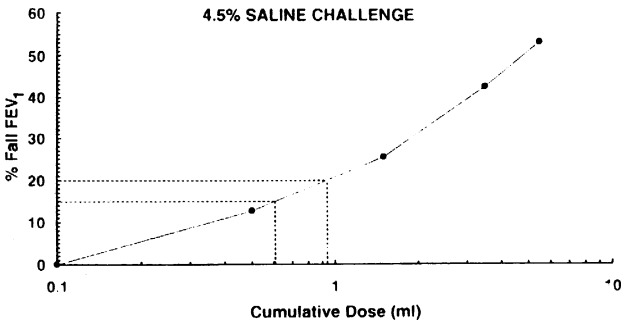

Figure 8. The fall in forced expiratory volume in one second FEV_1 ,expressed as a percentage of the pre-challenge value (% fall FEV_1), in relation to the cumulative dose of 4.5% saline. The broken lines represent the value for the dose of 4.5% saline required to provoke a 15% (PD_{15}) and 20% (PD_{20}) fall in FEV_1. Taken from Anderson et al.[43]

TABLE 2

RESULTS OF 4.5% SALINE CHALLENGE IN 180 PROSPECTIVE DIVERS

The table contains the changes in forced expiratory volume in one second (FEV_1) expressed as a percentage of the pre-challenge FEV_1 after saline challenge. The first column groups the results. The other columns give the mean ± 1 SD of the various groups. The second gives the % fall. The third the % rise in FEV_1 after bronchodilator, expressed as a percentage of the pre-challenge FEV_1. The fourth gives the lability index (% fall after saline + % rise after bronchodilator). The remaining columns give FEV_1 (fifth), FVC (sixth), and FEF_{25-75}(seventh, expressed as a percentage of the predicted normal, and the actual values for the FEV_1/FVC ratio (eighth).

% fall group	% fall 4.5% saline	% rise b'dilator	Lability % change	FEV_1 % predicted	FVC % predicted	FEF_{25-75} % predicted	FEV_1/FVC actual
<15% n=150	4.5 ± 3.7	2.9 ± 4.3 n=121	7.4 ± 6.5 n=121	106.3 ± 14.0	105.5 ± 10.9	81.7 ± 23.5 n = 108	79.8 ± 8.4
>15% n=30	22.3 ± 6.5	3.9 ± 6.5 n=29	26.2 ± 11.8 n=29	100.3 ± 13.7	105.1 ± 12.1	69.6 ± 20.4 n = 26	76.1 ± 8.8
10-14.9% n=21/150	11.5 ± 1.5	2.6 ± 6.3 n=17	14.1 ± 7.2 n=17	103.2 ± 14.4	104.6 ± 11.5	71.7 ± 12.6 n = 12	78.0 ±7.7
<15% FEF_{25-75} normal n=77/108	4.2 ± 3.5	1.9 ± 3.2 n=72	6.1 ± 4.4 n=72	111.8 ± 12.4	105.3 ± 11.5	91.6 ± 20.2 n = 77	84.0 ± 6.1
<15% FEF_{25-75} abnormal n=31/108	4.2 ± 3.7	4.5 ± 3.2 n=29	8.7 ± 4.3 n=29	95.8 ± 7.0	105.3 ± 8.3	57.1 ± 7.2 n = 31	72.1 ± 5.4

Significance of the results

A fall in FEV_1 of 15% or more is considered as abnormal. A person who has a positive test to hypertonic saline would be expected to be positive to challenge with dry air. They would usually, but not always, have a PD_{20} to histamine of less than 2 micromol and methacholine less that 4 micromol[40,43] However a person positive to a methacholine or histamine is not necessarily positive to a 4.5% saline or exercise challenge. Some investigators have found an increase in methacholine responsiveness after hypertonic saline[45] while others have not.[46] This effect may relate to refractoriness to the saline challenge.[47] It would seem advisable that if a pharmacological challenge and a physical challenge are both to be performed that they should be performed on separate days.

Mechanisms provoking airway narrowing.

The mechanism whereby dry air and hyperosmolar saline provoke airway narrowing is thought to involve release of mast cell mediators [28,48,49] and sensory neuropeptides.[29] The evidence for mast cell involvement comes from studies demonstrating the effectiveness of antihistamines given either orally or by aerosol in preventing the airway responses.[50-52] Prevention of the responses by this class of drug however, is impractical and not recommended due to unwanted side-effects, the high dose required with tablets, and inconvenient mode of administration as an aerosol.

In addition to the mast cell, we think that the sensory nerves are probably involved in responses to exercise, hyperventilation, and hyperosmolar aerosols. To date the evidence to support this comes only from work in animals.[29,53] However, it is thought that part of the effectiveness of drugs like sodium cromoglycate (Intal) and nedocromil sodium (Tilade) in blocking the responses to these stimuli comes from their action on nerves.[54] The effectiveness of these drugs on many cell types (mast cell, nerves, epithelial cells) and at many different sites in the body (lungs, nose, eye, stomach) probably relates to their ability to block chloride ion channels.[54-56]

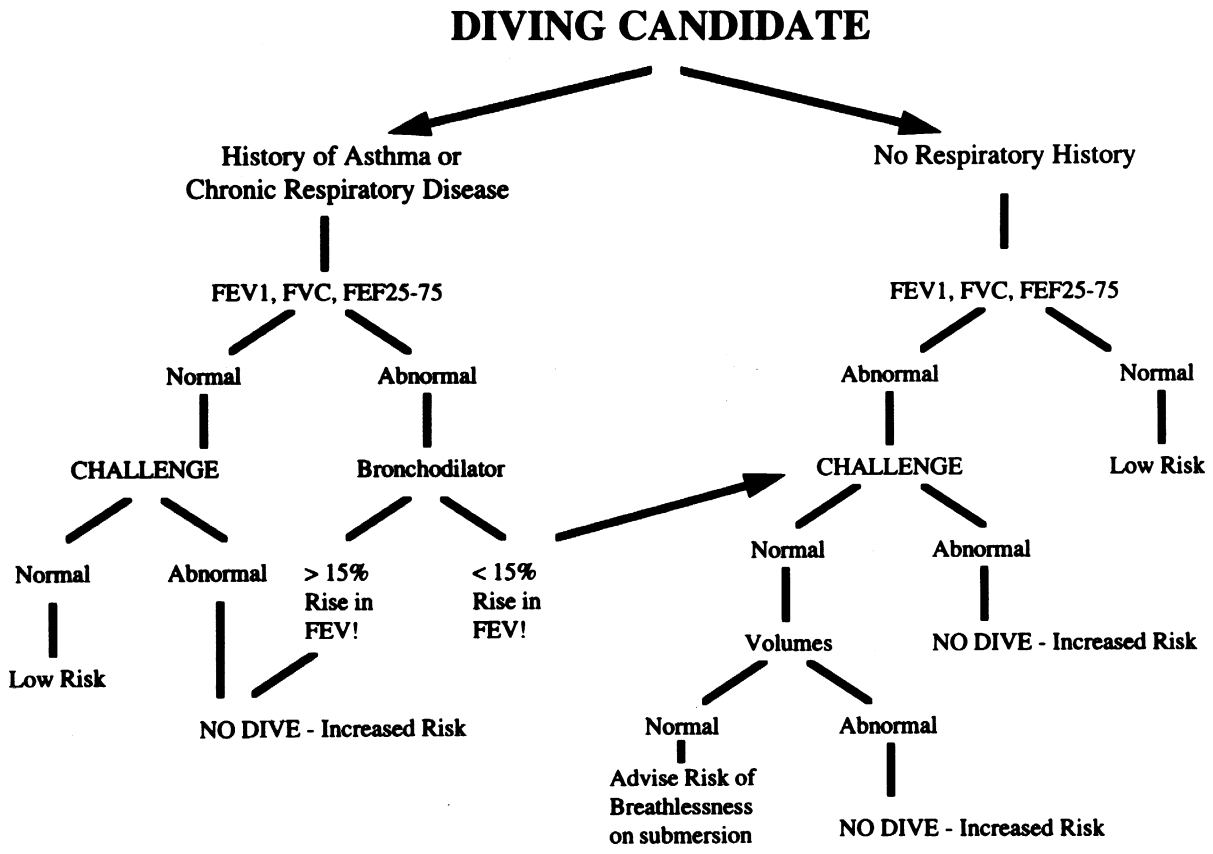

Figure 9. DECISION CHART TO AID IN RESPIRATORY ASSESSMENT OF INTENDING DIVERS

Treatment for those having a positive response

Because false positive responses are uncommon to dry air and hyperosmolar saline it is likely that a person having a positive response to these challenge tests will benefit from treatment for asthma. β_2 adrenoceptor agonists, terbutaline (Bricanyl) or salbutamol (Ventolin, Respolin) sodium cromoglycate (Intal Forte), or nedocromil sodium (Tilade) given immediately before the challenge will inhibit or prevent the airway responses to these stimuli in the majority of asthmatics. Drugs such as sodium cromoglycate and nedocromil sodium are only effective in persons with asthma. Demonstration of prevention of the airway response to hyperpnea or hyperosmolar challenge by these drugs should confirm the diagnosis of asthma. We have found that treatment with budesonide (Pulmicort) (1000 μg/day) for 8 weeks is very effective in inhibiting and even completely preventing airway responses to challenge with 4.5% saline. We found that 50% of our subjects were no longer responsive after this regimen.[56,57] Similar findings have been made in children suffering EIA, taking 400 μg budesonide daily.[58] We have not found beclomethasone as effective as budesonide in this regard.[59] In persons who are still responsive to exercise and to inhalation of 4.5% saline and who are taking aerosol corticosteroids we find the addition of 10 mg nedocromil sodium[60] or 20 mg of sodium cromoglycate[56] to be remarkably effective in blocking airway responses to 4.5% saline.

Laboratory findings

Since 1989 we have been referred 209 intending recreational scuba divers with a past history of asthma or asthma-like symptoms or with a borderline spirometry result suggestive of asthma. Of these, 180 were adults (mean age 27 years, range 18-51) who successfully completed a standardised challenge protocol using 4.5% sodium chloride aerosol.

Measurements of lung function for the groups of subjects separated on the basis of their airway response to 4.5% saline are given in Table 2. An abnormal test is regarded as :

1 Fall in FEV_1, in response to 4.5% NaCl, of 15% or more of baseline
2 Rise in FEV_1, in response to a bronchodilator, of 15% or more of baseline
3 A lability greater than 20% i.e. the % fall in FEV_1

from baseline after saline plus the % rise from baseline in FEV_1 after bronchodilator totals more than 20% (e.g. (9 % fall + 14% rise = 23% lability)

4 FEV_1 % <80% of predicted

5 FVC % <80% of predicted

6 FEF_{25-75} less than approximately 65 % predicted, based on lowest confidence interval.

A decision chart (Figure 9) is given to aid in the respiratory assessment of an intending diver.

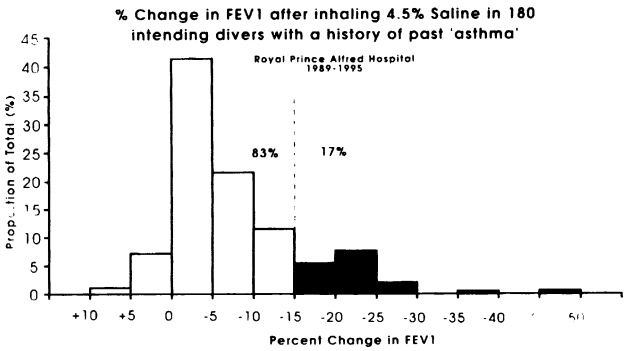

Figure 10. The percent reduction in FEV_1 in response to inhaling hyperosmolar (4.5%) saline in a group of 180 adults with a history of past asthma, or symptoms of asthma, who were referred to the laboratory as a consequence of a medical examination for diving.

Normal lung function

Ninety of the 180 subjects (50%) had normal lung function tests and no bronchial hyperresponsivess (were completely normal) and would be considered as low risk from pulmonary complications from diving with compressed gases.

Bronchial hyperresponsiveness

Thirty (16.6%) of the 180 subjects had a 15% or more fall in FEV_1 after inhaling 4.5% saline for up to 15.5 minutes or more and 28.3 % had a fall in FEV_1 of 10% or more. The mean % fall in FEV_1 ± 1 SD for the entire group of 180 was 7.45% ± 7.9 and the distribution of this fall is given in Figure 10. This distribution is normal and the current asthmatics represent the tail of the normal curve in this population.

The thirty subjects who had a fall in FEV_1 greater than 15% or more to 4.5% saline were also the only subjects to have abnormal responses to a bronchodilator or an abnormal lability index. This finding was a little surprising but it gives confidence that the response to 4.5% saline identified those with bronchial hyperresponsiveness. It should be noted that a positive response to 4.5% saline also identifies persons hyperresponsive to hyperpnea with dry air.[18,38-40,43] As acute narrowing of the airways is associated with gas trapping and acute hyperinflation of the lungs (Figure 5-7) we would consider that persons who are hyperresponsive to 4.5% saline are at an increased risk from pulmonary complications from diving.

Abnormal lung function without bronchial hyperresponsiveness

One hundred and fifty people had a fall in FEV_1 less than 15% after 4.5% saline and no response to bronchodilator, of these 108 had measurements of FEF_{25-75} and 31 (29%) had a value for FEF_{25-75} that was below the lower limit of the predicted normal (Predicted minus 1.64 RSD, see Table 1). Of these all but 2 had an FEV_1/FVC ratio less than 79%. Only one of these subjects had hyperinflation (an abnormally high functional residual capacity) measured before challenge and there was no gas trapping in any subject. These persons with a reduced FEF_{25-75} may be at an increased risk of feeling breathless at depth but may not necessarily be at an increased risk of pulmonary complications from diving. However they could be distressed as a result of heightened perception of breathing. They should be advised of this possibility. It may be more advisable to suggest that they do not dive.

Based on our findings, and those of others, in smaller numbers of persons without a history of asthma, we have previously suggested that an abnormal fall in FEV_1 to be taken as 15% or more. Of our group of 150 persons who had a fall less than 15% in response to saline, the mean fall in FEV_1 was 4.47% ± 3.7. Considering these persons had a past history of asthma, a value of 15%, which represents the mean + 3 SDs of this group representing 97% of the population would seem appropriate. For the 129 people who had a fall in FEV_1 of less than 10%, the mean % fall in FEV_1 was 3.3% ± 2.5. This value was exactly the same for the 70 persons who had normal values for FEF_{25-75} and a fall in FEV_1 to 4.5% saline less than 10%. Using these data the cut off point for mean plus 3 SDs is 10.8%.

Edmonds[23] has suggested that a fall of 10% or more in FEV_1 in response to saline should preclude a subject from diving. Of our 180, 51 had a fall in FEV_1 of 10% or more. A fall of 10-14.9% may be unnecessarily stringent if other parameters of lung function are all normal. These borderline cases, however, need to be assessed in terms of responses to bronchodilator and other indices of lung function to assess risk.

Discussion

We do not know the outcome for those referred to us. We do know that many who were positive to salt water

thought it unwise to dive. Most stated they would not take part or complete their diving course given this finding.

Of those who did go on to complete their courses and to dive we do not know how many had adverse events that could be related to their past history of asthma. Some may have had asthma symptoms again and discontinued their diving. Others may still be diving.

While we consider that we provided a service to evaluate the subject for asthma at the time we would have provided a greater service if we had followed up. By doing so we may have gone further towards establishing what factors contribute to mortality and morbidity in diving persons with a past history of asthma. Perhaps an international effort between interested parties will help to give the additional data required "to define accurately risks of diving in subjects with different forms of asthma".[61]

Audience participation

Unidentified speaker

Why do we not use water?

Anderson

Because water does not necessarily identify persons with exercise induced asthma. We have found that water provokes too much cough. We used it until about 1986. Hyperosmolarity is more likely to be a common stimulus to the airways. However a diver going into fresh water should have a water challenge.

Bove

The test can be graded very nicely in a dose response relationship and I guess the question we have in diving is how does it relate to the outcome results, for example in an asthmatic in general. Is there good data relating to the outcome of the tests to the consequent morbidity, not for diving but for asthma in general.

Anderson

Yes, persons responsive to hyperosmolar saline also have exercised induced asthma. Furthermore treatment with steroids reduces responses to saline and reduces the severity of exercise induced asthma.

Bove

In the States of course we are recommending for athletes that have exercise induced asthma, but not necessarily for divers, that they go on a drug and that they use it on the days of their competition. It seems to work.

Anderson

I am not worried about exercise in air, but I am worried about breathing dry air under water.

Bove

I think that in the States it is agreed that the major problem is the lack of exercise capacity while swimming on the surface and the consequent panic and drowning. This is much more important than the potential for pulmonary barotrauma. Under these circumstances the requirement for athletic performance outweighs concern for pulmonary barotrauma.

Anderson

I agree with this. However we must be wary as we do not wish current asthmatics with exercise induced asthma to dive. I believe that asthma is more severe in Australia and New Zealand compared with the USA. Our vigilance may be the reason that asthmatics are not over represented in our statistics.

Veale

It is probably worth emphasising that the test correlates very well with clinical severity of asthma and allows one to establish a range of mild to severe asthmatics.

Anderson

It also correlates with the pathology but I did not have time to show that.

Acknowledgement

We would like to thank the many medical practitioners who referred the patients. In particular we would like to thank Drs Carl Edmonds and Douglas Walker for their long term interest and enthusiasm that led us to developing this range of tests for the intending diver.

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PREVALENCE OF BRONCHIAL HYPERRESPONSIVENESS IN A GROUP OF EXPERIENCED SCUBA DIVERS

Graham Simpson and Cathy Meehan

Summary

50 unselected experienced scuba divers with total diving experience of over 70,000 dives were investigated to see how many would now be allowed to train as scuba divers under various suggested medical standards in current use in Australia. Subjects had both hypertonic saline and histamine bronchial provocation tests as well as routine spirometry. Using published criteria anything between 10% and 46% of these divers would not now be allowed to train. However there were few reported diving incidents and these were not more prevalent in those with abnormal results. Five (10%) of the subjects had current clinical asthma and bronchial provocation tests failed to identify 3 (60%) of these.

Some current recommendations for medical standards seem excessively severe and should be modified. We suggest that prospective study of novice divers with positive results on bronchial provocation testing would be helpful to quantify any increased risks these subjects may have when scuba diving.

Introduction

Active bronchial asthma is universally regarded as a contraindication to scuba diving. There are good theoretical reasons for this. Obviously an attack of asthma while in the water, either diving or at the surface, could have extremely serious consequences by reducing exercise capacity. In addition airway narrowing falling short of an acute attack of asthma could result in air trapping either globally or regionally within the lung. Such air trapping presents an obvious risk of pulmonary barotrauma on ascent. The scuba diver is in an environment where there are several potential triggers for asthma. These include exercise, inhalation of cold, dry air and possible inhalation of non-physiologically isotonic water, hypotonic fresh water or hypertonic sea water. However, hard evidence that asthmatics are at greater risk of pulmonary barotrauma or death during diving is scarce. Analysis of American diving accident statistics over a long period does not show an over representation of asthmatics.[1] Australian and New Zealand data has been interpreted differently by different authorities. For example Gorman[2] states that "asthmatics are over represented in diving fatalities" and the standard Australian text book on diving medicine states that "only 1% of divers are asthmatic...at least of 9% of deaths were in asthmatics".[3] On the other hand Walker,[4] after analysing the coronial reports on 201 Australian and 120 New Zealand scuba diving related fatalities, could find only four cases in which asthma could have been the probable cause of death.

This divergence of opinion is reflected in the guidelines issued by authorities in different countries regarding fitness to dive. ln the United States there do not seem to be any agreed medical standards, though active asthma is regarded as a contraindication and bronchial provocation testing is felt to be an useful tool.[1] In the United Kingdom asthma is regarded as a contentious issue[5] and British Sub-aqua Club guidelines do not preclude asthmatics from diving unless they have proven exercise induced asthma or have had a very recent attack.[6] In Australia an extremely conservative approach has been adopted in recent years. This is laid down in the Australian Standard AS 4005.1 (1992).[7] This states that current asthma or obstructive airways disease is an automatic disqualification, but that potential divers with abnormal spirometry or possible bronchial hyperreactivity need a specialist opinion and bronchial provocation testing. In its advice to the examining physician the standard suggests for further reading the textbook by Edmonds, Lowry and Pennefather.[3] This textbook takes a more rigid line and suggests disqualification if there is a history of asthma or of use of bronchodilators over the previous five years, wheezing on hyperventilation or after exercise, and forced expiratory volume in one second ($FEV_{1)}$ or vital capacity of less than 80% of predicted, mid-expiratory flow rates of less than 70% of predicted or greater than 10% fall in expiratory flow rates after bronchial provocation testing with histamine and with hypertonic saline. The Thoracic Society of Australia and New Zealand has a slightly more liberal approach, but also emphasizes the role of bronchial provocation tests.[8] In practice this testing is usually applied to potential divers who give a history suspicious of previous bronchospasm or who have mildly abnormal spirometry.

Despite the sometimes very specific recommendations outlined above and a widespread use of bronchial provocation tests to disqualify potential student scuba divers there is no information as to whether having an abnormal provocation test in fact puts the diver at increased risk nor of the prevalence of varying degrees of bronchial hyperresponsiveness in the diving population. The present study attempts to address this last point.

Subjects and methods

Volunteers were sought through local dive clubs and organizations. It was emphasized that this was a prevalence study in a normal diving population and no effort was made specifically to recruit known asthmatics. All subjects were experienced scuba divers who had qualified either overseas or before current standards were introduced. Subjects filled in a questionnaire with particular emphasis on the respiratory system and all

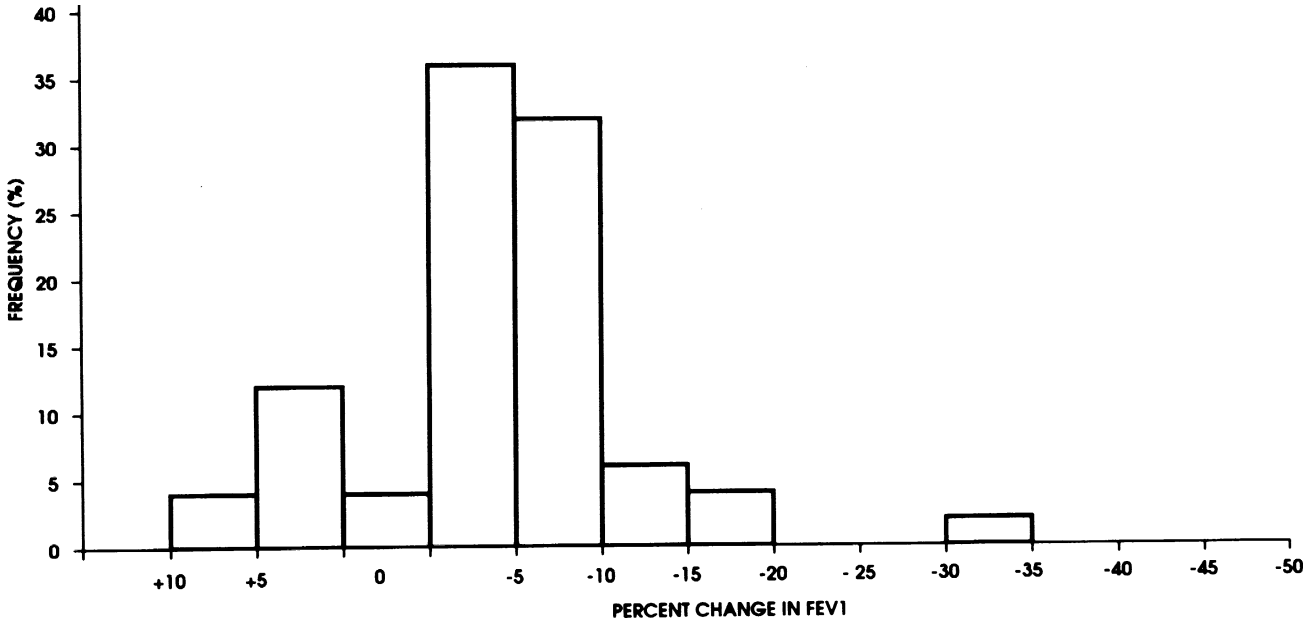

Figure 1. The percentage change in FEV_1 in response to 4.5% saline in 50 current and experienced scuba divers.

underwent routine spirometry using a Welch AlIyn pneumotachograph including expiratory flow volume loops. History included recording of total number of dives and of any problems encountered during diving. All subjects underwent a standard bronchial provocation test with 4.5% hypertonic saline[9] using an Omron NE-U06 ultrasonic nebuliser followed by administration of 5 mg nebulised salbutamol at the end of the test. 47 subjects also had a bronchial provocation test with histamine to a total dose of 7.8 μmoI.[10] Normal predicted values used for spirometric indices were those of Knudsen.[11] All subjects gave informed consent. The protocol included a guarantee of confidentiality of the individual results but subjects showing possibly abnormal results were given full counselling about the theoretical implications of their results. The study was approved by the local ethical committee.

Results

Of the 50 divers studied, 38 were male and 12 female. Mean age was 37 years (range 26-58). Five divers gave a history of current asthma and one of past asthma in childhood. A further 22 had either a family history of asthma or a past or family history of other atopic disease. 24 were recreational divers and the remaining 26 were occupational divers. Overall 23 were smokers (10 (42%) of the recreational divers and 13 (50%) of the occupational divers smoked). Smoking was slightly more prevalent (50% v 45%) in women (6) than in men (17). Smoking was more prevalent in those with abnormal respiratory function tests (RFT).

Spirometry and saline provocation testing

No subjects had either FEV_1 or forced vital capacity (FVC) of less than 80% of predicted normal. Ten subjects had FEV_1/FVC ratio of less than 75%. Five of these also had maximum mid-expiratory flow rates (MMEF) of less than 70% of predicted as did one subject in whom this was an isolated abnormality. Results of bronchial provocation tests are shown in Table 1

Using the most rigid criteria for exclusion from diving,[3] five subjects would be excluded for current asthma, a further ten for abnormal ventilatory function tests and a further eight for a greater than 10% drop in FEV_1 on

TABLE 1

RESPONSES OF 50 EXPERIENCED DIVERS TO BRONCHIAL PROVOCATION TESTING WITH HISTAMINE (47) AND SALINE (50)

Fall in FEV_1	Histamine	Saline	Responding to either
Less than 10%	32	32	27 (54%)
10% to 14%	10	13	15 (30%)
15% to 19%	3	4	6 (12%)
20% or greater	2	1	2 (4%)
Totals	**47**	**50**	**50 (100%)**

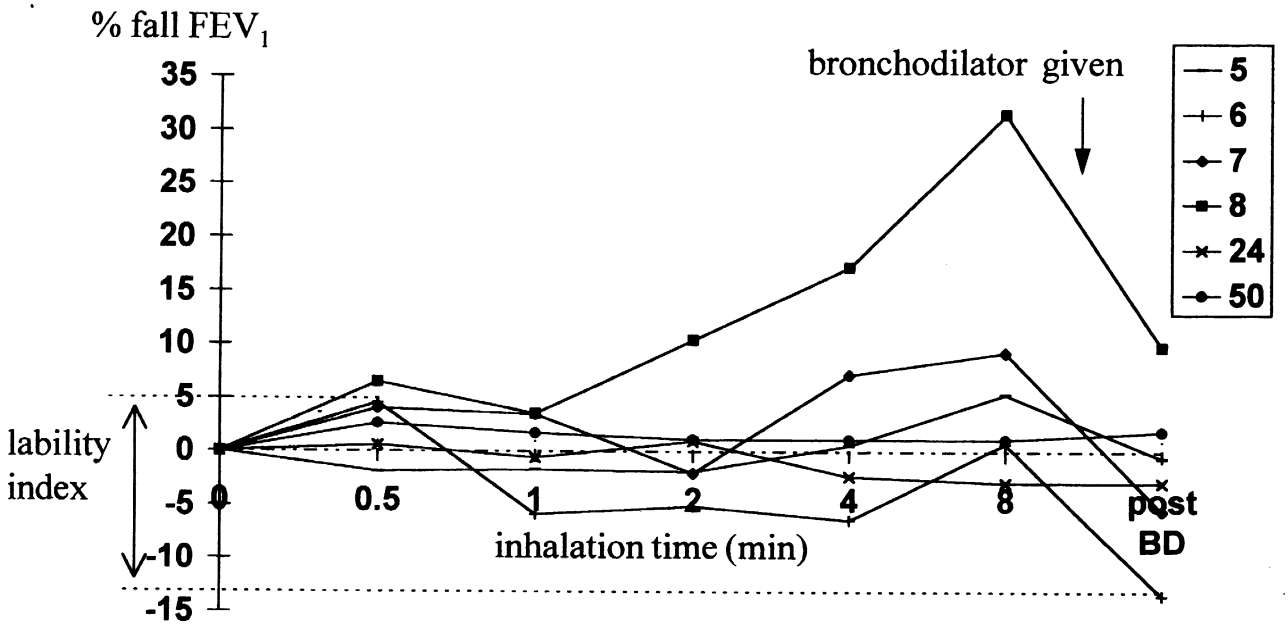

Figure 2. Changes in FEV_1, expressed as % change from baseline, in response to inhaled 4.5% saline followed by bronchodilator in 6 divers with a history of asthma.

bronchial challenge testing using the % fall in FEV_1 from baseline after saline plus the % rise from baseline in FEV_1 after bronchodilator to arrive at the percentage change (e.g. 9 % fall + 14% rise = 23% lability). Figure 1 shows the changes in FEV_1 in the whole sample.

The asthmatic subjects numbered five (10% of the sample). There were three men and two women with the mean age of 35 years (range 26-44). Another male had a past history of childhood asthma. In total they had logged approximately 2,600 dives. One had had an episode of decompression illness not apparently related to pulmonary barotrauma and he also had had an episode of wheezing on the surface. Two of the asthmatics were maintained on inhaled steroids, one on sodium cromoglycate with theophylline and the remaining two took only bronchodilators as necessary. One asthmatic had a low FEV_1/FVC ratio and also failed the bronchial provocation test at the 20% level. Another also failed the provocation tests at the 20% level. The three asthmatics on prophylactic medication had no. abnormalities on spirometry nor on provocation testing. Figure 2 shows the responses of the six divers with a history of asthma.

Results of testing asthmatic divers

Diver 5, a recreational diver with 30 dives, regularly used salbutamol and budesonide. 6 hours after the last dose Diver 5 had a small improvement for the first 4 minutes of saline inhalation, then dropped to 5% of baseline before bronchodilator which returned the FEV_1 to baseline.

Diver 6, a recreational diver with 30 dives, had recent onset asthma, was on salbutamol, beclomethesone and an antihistamine. 1 hour after the last dose Diver 6 had a fall of 5% after 30 seconds, but at 1, 2 and 4 minutes gained 5% above baseline, at 8 minutes this had dropped to baseline and then improved to 14% above baseline with bronchodilator.

Diver 7, a recreational diver with 2,000 dives, took theophylline, salbutamol and sodium cromoglycate regularly. 12 hours after the last dose Diver 7 had an 8% fall with saline and recovered to 5% above baseline with bronchodilator.

Diver 8, a recreational diver with 200 dives, only needed salbutamol intermittently. 2 weeks after the last dose Diver 8 had a 32% fall in FEV_1 after saline and with bronchodilator only recovered to -10% of baseline.

Diver 24, an occupational diver with 350 dives, had used salbutamol in childhood and had had no treatment for 25 years. Diver 24 had a small rise in FEV_1 in response to saline and no change with bronchodilator.

Diver 50, an occupational diver with 400 dives, had intermittent wheezing after viral infections when budesonide and terbutaline were used. One month after the last dose Diver 50 had negligible change with saline and no response to broncholdilator.

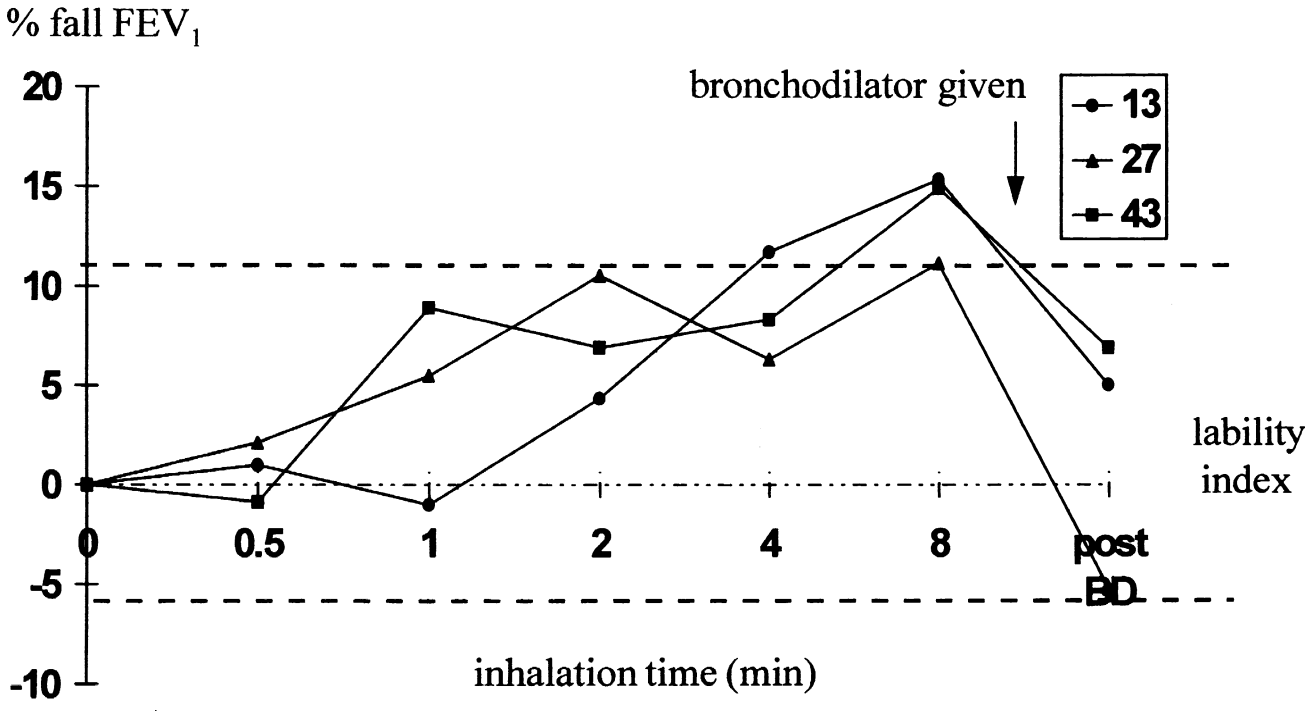

Figure 3. Changes in FEV_1, expressed as % change from baseline, in response to inhaled 4.5% saline followed by bronchodilator in 3 divers without a history of asthma.

Non-asthmatic divers responding to saline

Diver 13 had a low FEV_1/FVC ratio (70% of predicted), low mid expiratory flow rate (60% of predicted and a family history of asthma. Diver 13, a recreational diver who had done 140 dives without problems, developed a 15% fall in FEV_1 from the baseline after 8 minutes of saline and only recovered to -5% with bronchodilator.

Diver 27 had a low FEV_1/FVC ratio (73% of predicted)) and a family history of emphysema. Diver 27, an occupational diver with more than 2,000 problem free dives, developed a 11% fall in FEV_1 from the baseline after 8 minutes of saline and recovered to +6% with bronchodilator.

Diver 43 had a past history of hay fever and a family history of asthma. Diver 43, a recreational diver with 700 dives without problems, developed a 15% fall in FEV_1 from the baseline after 8 minutes of saline and only recovered to -7% with bronchodilator.

These results are shown in Figure 3. The group as a whole reported three episodes of decompression illness in over 70,000 logged dives (one occurring in an asthmatic). There were three episodes of salt water aspiration and one near drowning in addition to the wheezing episode at the surface occurring in one of the asthmatic subjects.

Discussion

Applying standards suggested in the current standard Australian text book on diving medicine, 23 (over 45%) of this group of unselected experienced scuba divers would be prevented from undergoing scuba diving training today. Despite this, this group has reported very few diving problems. lt is possible that this represents selection bias in that those subjects with hyperresponsiveness who were prone to problems may have given up diving, but at the very least such a high prevalence of apparent abnormality in those continuing to dive successfully suggests that the tests are a very poor predictor of problems. The 10% threshold for drop in FEV_1 on provocation testing seems inappropriately severe and this recommendation should be modified. The Thoracic Society recommendations of a 20% fall in FEV_1 seem more realistic,[9] but in this group of subjects, provocation testing seems to add nothing to a clinical history of current asthma.

It is known that there is a very poor correlation between bronchial hyperresponsiveness and respiratory symptoms including symptoms suggestive of asthma. A recent study[12] using histamine found that only about half of those with moderate or severe bronchial hyperresponsiveness had any symptoms suggestive of asthma. In the same study, over 40% of subjects reported one or more chronic respiratory symptoms and over half of these did not show bronchial hyperresponsiveness. lf it is believed that bronchial hyperresponsiveness on

provocation testing is, in itself, a significant risk factor for scuba diving, then this poor correlation between symptoms and provocation test results should logically mean that all potential scuba divers should be tested. Spirometry alone does not seem likely to be helpful here. The commonest abnormality is a reduced forced expiratory ratio but this has been shown not to be predictive of an increased likelihood of bronchial hyper-responsiveness.[13]

In the absence of hard data on absolute risks there are only theoretical reasons for supporting bronchial provocation testing is the assessment of fitness to dive. A 10% threshold for provocation testing is clearly unworkable and most practitioners would probably be unhappy at passing as fit divers with a 20% fall in FEV_1 on bronchial challenge testing. One possible way forward would be to create a national or preferably international register of subjects who have a fall of between 10 and 20% on bronchial provocation testing and to follow their diving progress prospectively should they choose to continue scuba diving after explanation of the increased risks which they may be running. Until this or a similar study is performed no sensible advice to potential scuba divers can be given and it is likely that many people are being excluded from voluntary participation in a recreational activity on purely theoretical grounds.

Only with the results of such a prospective study will we be able to formulate sensible guidelines for the examination of novice scuba divers.

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BRONCHIAL PROVOCATION TESTING IN ROYAL AUSTRALIAN NAVY DIVERS AND SUBMARINERS

Robyn Walker, Jennifer Firman and David Firman

(Presented by Robyn Walker)

Abstract

Royal Australian Navy diving/submarine branch candidates who fail to meet the prescribed respiratory standards are often referred for bronchial provocation testing. Many of these candidates are subsequently passed fit to dive or fit for submarines. A retrospective analysis of 178 Royal Australian Navy members who attended Royal Prince Alfred and Concord Hospitals for bronchial provocation testing was undertaken. Clinical factors which led to the ordering of the test were correlated with the outcome of the testing. The results suggest conventional measurements of lung function parameters and clinical variables are poor predictors of bronchial provocation test outcome in RAN divers.

Introduction

The Royal Australian Navy's (RAN) minimum respiratory standard for entry requires all candidates at their time of application to have displayed no evidence of asthma within the preceding 3 years.[1]

Prescribed medical standards for RAN divers stipulate there must be "No evidence of lung disease and, particular attention must be paid to any condition that might cause retention and trapping of expanding gas in any part of the lungs during decompression. Any past or present evidence of obstructive airways disease (e.g. asthma, chronic bronchitis, emphysema, allergic bronchospasm) are medical grounds for rejection from diving." The RAN also requires all divers and submariners to undergo pulmonary function testing as part of their periodic medical examination. A forced vital capacity (FVC) of less than 3.5 litres or a forced expiratory volume in one second (FEV_1)/ FVC ratio of less than 75% at the initial medical examination are causes for rejection unless further pulmonary function testing reveals no abnormalities.[2]

The Australian Standard for Occupational Diving AS2299[3] has similar guidelines stating "An FVC or FEV1 of more than 20 % below predicted values or an FEV_1/ FVC ratio of less than 75% may indicate increased risk of pulmonary barotrauma. If no other abnormality is present, a finding of fitness may be allowable if additional specialist pulmonary function tests and opinion do not find any fixed or intermittent outflow obstruction that might predispose to pulmonary barotrauma."

Diving/submarine branch candidates who fail to meet these standards are often referred for bronchial provocation testing despite minimal or absent clinical evidence of respiratory disease and many are subsequently passed fit to dive or fit for submarines. Bronchial provocation testing is a recognised technique for identifying bronchial hyperresponsiveness associated with asthma,[4] however, the usefulness of bronchial provocation testing as part of a diving medical work-up in this population has not been substantiated.

Method

A retrospective analysis was undertaken of the Medical Health Documents of all RAN personnel attending Royal Prince Alfred and Concord Hospitals respiratory laboratories from June 1987 to January 1995. 178 subjects were identified from the laboratories' databases. Clinical histories were collected along with the results of lung function and bronchial provocation testing. Age, height, measured and predicted FEV1 and FVC, FEV_1/ FVC, peak expiratory flow rate (PEFR), peak expiratory flow from 25-75% of the vital capacity (PEF_{25-75}), measured and predicted total lung capacity (TLC), vital capacity (VC), inspiratory capacity, functional residual capacity (FRC) and residual volume (RV) were recorded. Clinical details such as the presence or absence of childhood wheeze, recent wheeze, exercise induced wheeze, family history of asthma, smoking history and history of atopy were noted. Details of whether histamine or methacholine or saline bronchial provocation or a combination were performed was recorded with the test result.

Results

Five of the 46 saline challenge tests were positive as were 75 of the 130 histamine and 3 methacholine challenge tests.

Logistic regression analysis was used to model the multivariate association between the various physical measurements, clinical history variables and outcome of histamine or saline testing.

No significant predictors of saline test results were found. There were only 46 saline tests so that tests of association lacked power. However if the results for FEV_1/ FVC in the observed proportions hold true for larger data sets FEV_1/FVC would be a moderately significant predictor of positive or negative result.

There were 130 histamine tests. A number of variables were found to be jointly significantly associated with a positive histamine test. These were height, FEV1/ FVC <75% and a clinical presentation suggestive of asthma,

but the predictive power of this model was relatively poor. Vital capacity was significantly associated with a positive test result but had a high proportion of unmeasured values. Height was associated with VC and probably acts as a proxy for VC in the regression model (but does not have the high missing value rate). This "best" model for histamine test positive result had the optimal sensitivity and specificity values of 83% and 69% respectively.

Variables such as PEFR, TLC and RV were not significantly associated with the test result. Clinical variables such as childhood wheeze, exercise induced wheeze and family history of asthma similarly showed no significant association with test outcome.

Discussion

These results support the hypothesis that physical measurements of lung function and clinical history variables are poor predictors of the results of bronchial provocation testing in the RAN population.

The retrospective nature, absence of complete sets of data on all participants and the small number of saline tests are identified as problems with this study, however important conclusions can be drawn from the data and provide a basis for further study. A FEV_1/FVC ratio of less than 75% at initial examination is used by many practitioners as the sole screening tool of lung function for potential divers. Brooks et al[5] in a prospective study of Royal Navy Submarine Squadron candidates has shown that FEV_1 and FEV_1/ FVC do not predict the likelihood of pulmonary barotrauma with the only good predictor in their study being a low FVC by itself. Our figures show there is an association of FEV1/FVC <75% and a positive histamine test, however the predictive power is low.

Histamine and methacholine challenges have a high sensitivity and negative predictive value but a low specificity and positive predictive value of less than 30%.[6] Up to 30% of people can have a positive result to inhaled histamine but have no symptoms or clinical history of asthma. The absence of any good predictor for test outcome, either measured or clinical, further reduces the value of histamine or methacholine challenge.

Saline provocation is a highly specific challenge for identifying persons with current asthma and is readily recognisable by diving candidates as having relevance to their potential sport. However, our data suggests that in the RAN our criteria for testing have a low predictive value.

Our results reveal that no measured lung function value alone or in combination stands out as having strong properties for predicting a positive test result. The combination of height, FEV1/FVC <75% and a clinical history suggestive of asthma, while being better than tossing a coin in predicting a positive result, is by no means a sensitive or specific model. This suggests we could refer everyone for bronchial provocation without performing any lung function tests or collating medical history and still obtain the same results.

To obtain the absolute predictive value for provocation testing we will need to submit a large number of RAN members who pass the screening tests for diving or submarine selection to bronchial challenge and compare the results with those of the group described here.

Conventional measurements and clinical history in this population are poor predictors of bronchial provocation testing outcome which implies we must continue the search for positive predictors in order to prevent candidates from being exposed to unnecessary physical or financial insults.

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BRONCHIAL PROVOCATION TESTING FOR INTENDING DIVERS WITH A HISTORY OF ASTHMA OR WHEEZING

Cathy Meehan

Abstract

A retrospective analysis of 100 hypertonic saline challenge tests, performed as part of an assessment of medical fitness to dive, was undertaken. The candidates where intending scuba diving students who had passed all other aspects of a diving medical, but gave a history of asthma or wheeze. An analysis of 50 histamine provocation test results was also undertaken, the selection criteria being identical. Twenty one candidates (21%) were deemed unfit to dive as a result of their response to hypertonic saline. These either had a fall in FEV_1 of 20% or greater after hypertonic saline inhalation or developed wheezing or shortness of breath. Of the 50 candidates who had a histamine challenge, 17 (34%) had been deemed unfit as a result of a 20% or greater fall in FEV_1 after inhalation. These results suggest a higher incidence of significant bronchial hyperresponsiveness to a pharmacological challenge with histamine when compared with a non-isotonic challenge with hypertonic saline. The results also suggested an increased incidence of bronchial hyperresponsiveness to hypertonic saline in the group of Japanese candidates compared with other nationalities. Smokers demonstrated a greater response to inhalation than non smokers. A family history of asthma or a personal history of atopy were poor predictors of response.

Introduction

All prospective self contained underwater breathing apparatus (scuba) divers in Australia require a medical examination to assess their fitness to dive before learning to dive. A would be diver who gives a history of asthma or recurrent wheeze may be at risk of developing bronchial airway narrowing while exercising when diving. These diving candidates are therefore referred for bronchial provocation testing to assess the reactivity of their airways. There are several triggers when diving, including breathing dry air, exercise and non-isotonic water (salt water or fresh water) inhalation. The Australian Standard AS 4005.1 for prospective recreational divers states that "any evidence of obstructive airways disease, e.g. current asthma, chronic bronchitis, allergic bronchospasm, shall automatically disqualify. In case of doubt, specialist medical opinion should be sought. Such opinion should include provocative testing if any doubt concerning the possibility of bronchial hyperreactivity exists".[1]

The Thoracic Society of Australia and New Zealand[2] states that "intending divers with a history of current asthma should be advised not to dive.....Intending divers with a past history of asthma and asthma symptoms within the previous five years should be advised not to dive.....Those who have had asthma in the past, but who have normal spirometric tests and no symptoms, and have not taken asthma medication at all in the last five years, should proceed to bronchial provocation testing". Although most physicians consider that if bronchial hyperresponsiveness is present, subjects should not be passed fit to dive, the present recommendations against subjects with bronchial hyperresponsiveness and past asthma are made on theoretical grounds. There are good reasons to suggest that, along with current asthmatics, such divers have an increased risk of pulmonary barotrauma or arterial gas embolism, but there is insufficient data to confirm or refute this.[2] Provocation tests are of practical use in identifying those persons who would seem to be at risk of acute airway narrowing during diving. Those who have demonstrable bronchial hyperresponsiveness should be told that they may be at increased risk of pulmonary barotrauma and details of the possible consequences should be explained. Traditionally tests for measuring bronchial hyperresponsiveness have been challenges with pharmacological agents such histamine and methacholine. These are less acceptable to the intending diver as the stimulus is not seen to be relevant to the diver. The use of non-isotonic stimuli as a physical challenge to the airways is becoming more popular. It has been shown that during exercise, the increased rate of respiratory water loss acts as a hypertonic stimulus to induce asthma.[3] Strenuous exercise is occasionally required from a diver, all air breathed by diver is dry and a fine aerosol of hypertonic saline is often produced through a faulty expiratory valve. Experience has shown that if challenge with salt water causes breathing difficulty, or excessive coughing, the intending diver is immediately aware of the potential for the same thing happening while diving and accepts exclusion more readily.

Method

A retrospective analysis of 100 hypertonic saline challenge tests performed as part of an assessment of medical fitness to dive, was undertaken. These candidates had been referred to my diving medical practice in Cairns, North Queensland, for a diving fitness assessment between May 1994 and April 1995. A further 50 histamine challenge tests were also analysed. These had been performed during 1991 and 1992 for identical reasons. All these subjects had passed a recreational diving medical on all other aspects of their health, but had given a history of asthma or wheeze.

The selection criteria used has been outlined and follows the guidelines of the Australian and New Zealand Thoracic Society.[2] All the candidates had normal lung function as shown on respiratory function testing (RFT)

using Knudson predicted values,[4] and gave no history of significant asthma or wheeze within 5 years. A small group of candidates went onto provocation testing although they had symptoms more recently, but only when the aetiology or severity of the symptoms was questionable.

Hypertonic saline challenge[5] was performed using 4.5% NaCl through an Omron ultrasonic nebuliser (NE-UO6). The nebulising rate was approximately 1.5 ml/min. The protocol used was as outlined by the Lung Function Laboratory in the Department of Thoracic Medicine at the Royal Adelaide Hospital.[6] Baseline measurements of forced expiratory volume in one second (FEV_1), and forced vital capacity (FVC) were taken before inhalation. The FEV_1 was measured before challenge and at 30 and 90 seconds after the inhalation. The time of inhalation was doubled after each exposure, starting with 30 seconds, then 60 seconds, 1 minute, 2 minutes, 4 minutes and 8 minutes or until a reduction in FEV_1 of 20% or greater occurred. A minimum amount of 15 ml of saline was nebulised or the test was extended. Inhalation with a bronchodilator (salbutamol) was given at the end of the test and the percentage rise in FEV_1, from baseline, calculated. A fall in FEV_1, at the completion of the test, of 20% or greater after hypertonic saline or a rise of 15% or greater after salbutamol, was considered to indicate significant bronchial hyperresponsiveness and these candidates were recommended not to dive. The lability index, the sum of the percentage fall in FEV_1 during inhalation and the percentage rise after bronchodilator (from baseline), was also calculated. This calculation was used to identify the candidates who may have had some degree of airway's restriction at commencement of the test. If this result was 20% or greater, then diving was not recommended.

The histamine challenges were performed using the rapid hand operated technique described by Yan et al.[7] using a DeVilbis hand held nebuliser. Responses to these challenges were compared with the history of symptoms, smoking status and nationality, in order to identify any trends. The hypertonic saline challenge test responses were also compared to responses from a group of experienced divers who volunteered to undergo challenge testing as part of another study.[8] Volunteers had various past histories (including some with asthma) and diving experiences. The protocol and equipment used was identical. The volunteers were tested between September and December 1994.

Hypertonic saline challenge results

Of the 100 students presenting for hypertonic saline challenge, 62 (62%) were male and 38 (38%) were female. The mean age of the group was 25 years. Twenty seven (27%) were smokers. Thirty six of the group (36%) were Japanese, 18 (18%) Australian, 12 (12%) were British, 17 (17%) European, 14 (14%) from North America, and there were 3 (3%) others. A greater percentage of the 36 Japanese were currently smokers at 9 (25%) compared with 17% for both Australian (3) and European groups (3). Six of the USA and Canadian group were smokers (40%). The numbers here are too small to be statistically significant. In the group as a whole 9 (24%) of the females smoked compared with 18 (29%) of the males.

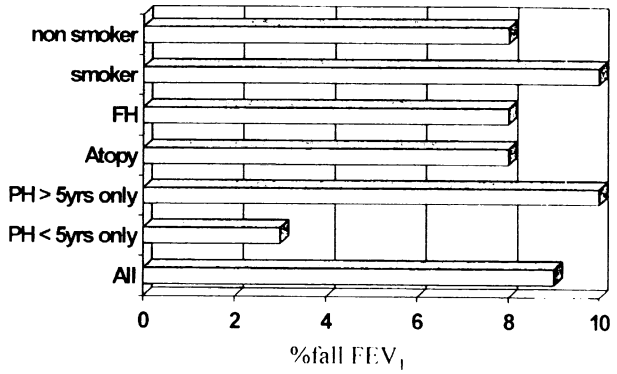

Figure 1. Average falls in FEV_1 after 4.5% saline provocation testing in cadidates with various past histories. FH = family history of asthma or wheezing. PH = past history of asthma or wheezing.

TABLE 1

FALLS IN FEV_1 AND FAILURE RATE

Fall in FEV1 after 4,5% saline	Number of students	%
10% or greater	33	33%
20% or greater	23	23%
30% or greater	18	18%
Total failed		**21%**

The average fall in FEV_1 at completion of the challenge test was 9%. As a group the Japanese fared worst with an average fall of 12%. This was also mirrored in the overall outcome. As a result of the provocation test diving was not recommended in 21% of the group as a whole. Again, the Japanese were over represented in this group with 11 (31% of all Japanese students) completing the test being recommended not to dive. Percentage fall in FEV_1 was also compared in groups with various risk factors and is represented in Figure 1. The results are suggestive but not statistically significant.

Figure 1 shows that smokers had a higher than average fall in FEV_1. Also that candidates who gave a history of symptoms of asthma or wheeze within the last 5 years had the least reaction to saline. Although this may seem contradictory, the students who were selected to do

the provocation test in this group where ones whose symptoms either seemed insignificant or there was question as to the diagnosis. The resultant average fall in this group of 3% shows that the selection criteria imposed on this group was justified. Summary of the percentage falls after inhalation of hypertonic saline is shown in Table 1. Twenty one prospective divers were recommended not to dive because of a 20% or greater fall in FEV1 after inhalation or the development of clinical signs of airway narrowing.

Histamine challenge results

Examination of the 50 histamine challenge tests on diving candidates, using identical selection criteria, showed that 17 (34%) of the candidates were found to have significant bronchial hyperresponsiveness and were advised not to dive. Compared with the 21 (21%) of candidates who showed significant airway reactivity to hypertonic saline, it is clear that a greater proportion of students were unable to dive as a result of the histamine challenge. This reinforces what has been found in a population study showing that a significant proportion (30%) of people can have a positive response to inhaled histamine but have no symptoms or clinical history of asthma.[2]

Table 2 shows the responses in prospective divers to different stimulants and compares them with the group of volunteer divers studied at the same medical practice using identical techniques.[8] These 50 volunteers were challenged with hypertonic saline as part of a study carried out between September and December 1994. The results are published on page 249- 253

Discussion

The results show that the Japanese students demonstrated a higher than average incidence of significant bronchial hyperresponsiveness, with an average percentage fall in FEV_1 after inhalation of 4.5% NaCl of 12% compared with the average of 9%. 31% of the Japanese students discontinued their scuba diving course as a direct result of the challenge compared with 21% of the total. The Great Barrier Reef is a great draw card for travellers to Far North Queensland and to Cairns in particular. Many of these travellers come especially to explore this world wonder and while doing so experience the excitement of scuba diving. A significant proportion of these travellers come from Japan. Analysis of statistics of open water certifications processed from one of the dive schools in Cairns, was kindly provided to me by PADI Australia.[9] The majority of students seen at my surgery were from this particular dive school. Analysis of the statistics showed that during the 12 month period commencing 1 April 1994, 33% of all the students successfully completing their open water certification were

TABLE 2

FALLS IN FEV_1 WITH PROVOCATION TESTING IN 50 EXPERIENCED DIVERS AND 150 DIVING STUDENTS

Group	Divers	Students	Students
Challenge	4.5% Saline	4.5% Saline	Histamine
% fall in FEV_1			
10%	30%	33%	
15%	12%	23%	
20%	4%	18%	34%

The groups above are 50 experienced divers tested with 4.5% saline,[8] 100 diving students tested with 4.5% saline and 50 diving students tested with histamine.

Japanese. This would suggest that more than one third of the open water diving students presenting to this particular dive school were Japanese (remembering that a proportion of those presenting failed to complete the course due to the outcome of the pre-dive medical assessment). Although some dive schools in Cairns may not attract as many Japanese student divers to them, there are other schools that cater solely to the Japanese traveller. I think that we can safely say that in our area a significant proportion of the diving industry dollar comes from Japan. The reason I have emphasised this is that some tour operators feel quite strongly about their loss of commission when a diving candidate does not continue with their diving course. One such operator suggested to me that as Cairns was such a high risk area, for failure to dive on medical grounds, that it was becoming too great a business risk to send their Japanese travellers to Queensland and that perhaps Micronesia would be a better destination as a medical examination was not required there at all. Although obviously to doctors the safety of the individual diver is of paramount importance, the financial side takes precedent in other quarters.

There was also a higher than average response to inhalation of 4.5% NaCl in current smokers. Those that gave a history of symptoms or use of bronchodilator within 5 years showed insignificant responses and this was an indication of the selection methods. Past history of atopy or family history of atopy or asthma was a poor predictor of response.

There was also a greater positive response rate to histamine challenge compared with challenge with 4.5% NaCl. Again this highlights the findings in a population study which showed that a significant proportion of people (30%) had significant response to histamine without any history of asthma.[2]

When comparing the group of experienced volunteer divers with little or no past history of airway

reactivity with the group of prospective divers all having a history of airway's reactivity, we see that both groups had a similar (30% and 33% respectively) response at the 10% level to hypertonic saline. This suggests that the criteria outlined, by Edmonds et al.[10] in *Diving and Subaquatic Medicine*, stating that "asthma provocation producing 10% or greater reduction in FEV_1 after both histamine and hypertonic saline challenge" leads to a FAIL, may be too stringent. In these two studies, fall in FEV_1 of 10% after provocation failed to differentiate between the group of experienced divers and the student divers. Further studies will be required to decide whether a 15% fall in FEV_1 after provocation indicates significant increased risk to diving (as suggested by Anderson et al.[11]) or whether a greater than 20% fall is stringent enough (at present part of our protocol). In order to answer these questions, more data is required. It would be useful to follow up candidates with borderline challenge test results and, if they have chosen to continue to dive, document their progress. This is the only way that guidelines can be set out based on clinical data rather than on purely theoretical grounds.

Acknowledgments

I would like to thank Dr Sandra Anderson for her help and encouragement and John Brannan for his technical assistance.

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DIVERS WITH ASTHMA: AN INVESTIGATION IS REQUIRED

Douglas Walker

"For any complex question there is a solution which is simple, appealing...and wrong."

There is undoubted logic in the medical opinion which states that asthmatics will be exposed to excessive risk if they attempt to scuba dive and should therefore never be granted permission to do so if a medical fitness certificate is requested.[1] Certainly it is the medical dogma in Australia that such people are subject to an unacceptable increased risk of morbidity and death should they be in an environment of changing ambient pressure. Questioning of self-evident truths requires an open-minded attitude which is not always easily reconciled with the advantages of accepting what is the local shibboleth. Unless we continually check the fit of what we believe against new data we are claiming that everything which there is to know is already known and understood.[2,3] In reference to the subject of asthma and diving it is timely to remember that in England a more relaxed opinion is held and there had not been any evidence of increased morbidity among scuba divers as a result.[4]

Nobody researches problems they believe fully understood. It is therefore necessary first to question the obvious, a worthwhile undertaking even if it only confirms the validity of beliefs. The fact that some asthmatics do indeed scuba dive cannot be denied,[5,6] a few coming to

notice in incident reports. Divers are notoriously reluctant to admit to being asthmatic, hard experience making them aware that most doctors (and diving instructors) who hear such an admission will respond with a lecture on the dangers they run.[7,8] If they omitted reference to asthma at their Diving Medical they may fear a loss of their certification should such an omission become known. There is another group of divers who deserve investigation of their diving experiences, those who gave some history of asthma in the past but have been assessed Fit to Dive after testing. The diving experiences of members of both these groups, if known, would greatly increase our understanding of the real natural history of the various degrees of the asthma syndrome in scuba divers. Their experiences are the only valid data base for discussing safety in diving with asthma.

It will be extremely difficult to persuade scuba divers that admitting to asthma will help towards a more flexible attitude by doctors to assessing less arbitrarily on medical fitness to dive, and the first step towards obtaining their involvement is to make them realise the necessity for the data only they can provide, and that there is a 100% guarantee their identities will never become known beyond those involved in this survey. Indeed they must have an assurance that whatever their diving history it will remain as medically confidential information. It is for this very practical reason that the investigation will attempt to follow-up those who have been passed as fit despite a history of asthma when younger, as they are unlikely to harbour such fears.

On page 260 is a draft questionnairefor an investigation into asthma and diving. It is published in the hope that readers can provide the author with feed back to immprove its contents and layout.

There have been a few surveys of asthmatic divers which have achieved a certain degree of success.[9,10] These have relied on use of a proforma in a diving publication and have been criticised for a perceived bias because there would be a tendency for those having suffered problems to have ceased diving, thereby unduly enriching the sampled diver population with the less affected. But this may be considered a counterbalance to relying on morbidity reports which only record the presence of an asthma history where a diver has been involved in some diving incident. There is a tendency to assume a causal relationship is present between an asthma history and any morbidity such a diver suffers. A closer analysis of such incidents will often reveal additional critical factors likely to have been far more significant in the genesis and progress of the incident.

The medical concerns with those who have a history of asthma revolve around two elements of the dive. First there is a risk of pulmonary barotrauma on ascent due to constriction of the airways in response to effort, stress, cold air, or inhalation of ultra fine droplets of salt water. The second is the more general reduction in safety if airways impairment makes the diver unable to perform the required physical effort either underwater or at the surface. Strangely there is little evidence in local or overseas reports of such problems, though they would be a readily identifiable risk factor to report if present. Few things in medicine are “always” or “never” and asthma is no exception. While some claim that once the person has suffered an asthma wheeze the hyper-responsiveness will always remain in their bronchial tubes to some degree,[14,15] others believe this respiratory tract responsiveness to provocation will fall to within “normal” levels in many.[16] Great variations exist in medical attitudes in different countries concerning the safety of asthmatics who dive but no evidence that this is reflected in the morbidity or mortality statistics in the Australia or New Zealand diving incidents reports compared with those of the UK or the USA.

When carrying out a Fitness for Diving examination there are applicants who have to be told that their bronchial system is too reactive for medical acceptance standards, and they complain there are asthmatics who scuba dive. Unfortunately they carefully avoid providing identification of these people, though they certainly do exist. It would be useful if there was a data bank containing the details of such persons, both those who suffer problems from their asthma when diving and those who dive uneventfully, for this would provide a better basis for fitness decisions.

One problem which requires resolution is to define “asthma”, as while some consider any history of a wheeze, even in childhood, as a contraindication to a positive fitness assessment, there will be others who will accept even those who require present use of a bronchodilator as being acceptable. Many more take a middle path and rely on the response to provocation testing.

An examination of Australian scuba diver fatalities reports, between 1955-1993 inclusive (203 cases),[17- 30] shows that in only 9 cases was there mention of asthma in the victim’s medical history. This has to be set against the lack of information concerning both the proportion of divers “at risk” who had a similar medical history. It was assessed as the initiating factor in one incident, possibly a significant factor in two others. In each of these incidents it was the actions of the victim rather than the asthma itself which decided the outcome. No cases have been identified in the reports made to the BS-AC where asthma was identified as being present and it is not treated as a significant factor in the code used to record the USA (University of Rhode Island) cases. This lack of representation of asthma among identified critical factors may indicate that few asthmatics scuba dive, or that few get into serious trouble. There is need to clarify this matter by examination of incident reports where there was a non-fatal conclusion.

ASTHMA AND SCUBA DIVING INVESTIGATION

You need not provided your name and address etc. but it would help the investigator if you do, as then he can contact you for further information if needed. Your personal details will be known only to the investigator (Dr Douglas Walker) **and never revealed to anyone else.**

Name..... Present age

Address..... Phone (...).....
Fax (...).....

Post Code .....

MEDICAL HISTORY OF WHEEZING / ASTHMA / TREATMENTS

Age at onset Age when asthma/wheezes last occurred

Severity Occasional wheezing Wheeze more than once a week Need medication all the time

Trigger events Exercise / Head cold / Pollen/ Weather / Emotion / Other

Treatment regular / occasional / rare / emergency

tablets (name .....)	YES	NO
Ventolin/similar	YES	NO
Atrovent	YES	NO
Bricanyl	YES	NO
Intal	YES	NO
Becotide / Beclofort / Turbuhaler / Pulmicort	YES	NO
Other		

Hospital treatments ?

Present condition wheezing / breathlessness problems YES NO How often.....

Present treatment / management

Other medical or surgical past or present problems

Diving History

Had medical before training ?	YES	NO
Disclosed asthma/wheeze ?	YES	NO
Medical Examiner asked about asthma	YES	NO

Response when told of wheeze/asthma respiratory function tests ?
Special respiratory tests for asthma
Advice given

Instructor
Aware of wheeze/asthma history ?
Response when told of wheeze / asthma history
Training level
Experience

Any diving-related problems (not necessarily due to asthma)	YES	NO
specify		
Any asthma related problems when diving	YES	NO (Describe below)
Use of medication before / after dives		

Any comments on medical / instructor / other advice on asthma and diving.

You are invited to add any additional information or comments, including your views on or experience of "diving medicals" and the attitude of diving instructors to those who admit to "asthma", on the back of this form. Also your views on present medical standards for divers and your observations on those you have seen scuba diving with disabilities either physical (eg paraplegic, amputee) or medical (eg asthmatic, diabetic, or with heart or other problems).

Please return this form direct to Dr D G Walker, PO Box 120, Narrabeen, New South Wales 2101, Australia.

It is hoped to enlist the interest and active involvement of not only divers who have an asthma history and dive (either with, or without, medical agreement) but also those persons, other divers or medical, with an interest in this problem. It is appropriate to seek out those who scuba dive who have been medically assessed as having only a mildly increased responsiveness to a test exposures to saline or methacholine (having admitted a history of wheezing, or use of inhalers such as salbutamol in the past). As assessment of the duration of effective protection against this type of test provocation by use of the most modern inhalations would have real value, as the problem should be examined with regard to treatments of a prophylactic nature which are now available.

The proposed manner of conducting this research requires the involvement of both medical and non-medical persons having either an interest in improving our understanding of asthma as a risk to diving safety or an interest in legitimising asthmatic divers. To this end a request is made for interested persons to communicate with the author. The information may concern personal experiences or observations of others, or statement of a willingness to follow up those who have revealed a history of asthma but been judged as safe to dive after pulmonary testing. There is a guarantee that a Medical Confidentiality management code will apply to all reports and correspondence. The "asthma community" is here presented with an opportunity to perform a useful service to those who suffer or have suffered from this condition.

For far too long the problem of deciding on the influence of some medical condition in the context of giving a formal decision on fitness to dive has been managed on an absolutist basis rather than by examination on a case by case basis. This has the benefit of simplicity and was defensible in court before the intrusion of the concept that medical beliefs must be demonstrably grounded on acceptable data. The basic fault has been our medical assumption, surely ill advised, that diving accidents commonly had a "medical" basis and could be eliminated (or at least significantly reduced) by medically examining every applicant before training commenced. A less proud boast of the profession's prognostic skills might in truth have been more appropriate and engendered an approach which was more open to examining the facts. Undoubtedly there are those whose medical or physical condition makes diving far too risky to be approved and others where the applicant's experience balances out the physical or medical adverse factors. There is need for an acceptance that people cannot be divided into one or other of two absolute groups, the totally fit and the absolutely unfit to dive. There is indeed a need to re-examine Australian standards.

An excellent basis for debating the case for initiating this investigation is given in the 1987 UHMS Workshop report *Fitness to Dive* and the recent paper by Neumann et al. on *Asthma and Diving* in addition to the other references given.[31,32]

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AN INTERESTING CASE OF DECOMPRESSION ILLNESS

Neil Banham

During the final dive of her initial training course a 35 year old female novice diver who developed constitutional and neurological symptoms. As the history of the incident, obtained on admission to the Emergency Department was unclear, the narrative here was compiled from the patient when she presented to hospital, on completion of treatment and at follow up a month later, from the diver's buddy and also from thedive master.

The dive which resulted in the presenting problems was to a maximum depth of 4.4 m for a total dive time was 37 minutes. Of this ten to twelve minutes at least was spent on the surface for instruction and most of the dive was spent around 2 m practising underwater skills such as mask clearing and controlled octopus ascent. Such a profile is well with in all recognised decompression tables and there was no suggestion of a rapid ascent.

Before entering the water she had been quite well. There were some problems with her new mask leaking and slow clearing of her left ear. Some nausea developed during the dive.

Her buddy reported later that the diver complained of feeling absolutely exhausted while she was snorkelling to shore but her was speech normal at this time. It was noted that, when she left the water, she was somewhat unco-ordinated but she was able to carry her gear up to the bus, wash it and to stow it. She complained of feeling unwell with nausea and headache.

At the dive shop, about an hour after the dive, she complained of feeling "weird" and had to sit down. A sensation of numbness developed over the dorsum of her left hand which progressed to paraesthesia extending up her left arm and there was some tingling on the back of her right arm.

The dive master reported that although she was able to answer questions appropriately he was concerned about the progression of symptoms so he took her by car to Fremantle Hospital, some five minutes away.

On examination in the emergency department she was noted to be alert but confused. Her Mini Mental State score of 19/30 indicated a significant psychometric deficit. Peripheral nervous system examination while she was recumbent was normal and while on oxygen in the emergency department her paraesthesia resolved.

On being stood up to assess her co-ordination it was noted that her heel-toe gait was poor, she was unable to perform a sharpened Romberg test and her overall condition deteriorated. She became drowsy, confused and agitated. Her speech was unintelligible. She had been erect for no more than thirty seconds

The differential diagnoses considered were cerebral arterial gas embolism (CAGE) and decompression sickness (DCS). DCS was thought to be unlikely because of the absence of a significant nitrogen load. The possibility of paradoxical embolism of venous bubbles through a patent foramen ovale was later excluded by a normal bubble contrast echocardiography.

Against CAGE were the absence of a history of rapid ascent, no clinical or radiological evidence of barotrauma and the long delay before obvious symptoms. CAGE is usually almost immediately apparent, although delayed cases have been reported many times. Rapid

deterioration on standing has also been reported many times and is presumed to be due to redistribution of bubbles under the influence of gravity.

I would like suggestions about the diagnosis from the audience and then I will tell you what actually happened.

Audience participation

Veale

You did not comment at all about her ventilation nor whether she had a normal $PAC0_2$. I see a lot of people referred with funny neurological symptoms and syndromes who are hyperventilating and I just wonder whether this woman was a nutter or not. I think you can clearly exclude DCS on the basis of her nitrogen load. It is not at all impossible that she could have a cerebral arterial gas embolism from barotrauma as a silent event and in the absence of detectable abnormality in the lung.

Banham

When she presented she was not hyperventilating. She had no evidence of carpopedal spasm. However we did not do a $PAC0_2$.

Bove

It is a strange exercise to propose that this woman had decompression sickness. First of all if it was decompression sickness it was purely cerebral decompression sickness and we are not even sure that exists as an entity, let alone in a situation where somebody comes up from four metres.

To me this is a clear cut classic case of arterial gas embolism following pulmonary barotrauma. The woman was a novice diver using scuba doing things like octopus breathing, ascents and descents. It is very easy to slip for a few seconds and ascend one and a half metres and get an air embolism, even though she would have been totally unaware that it occurred. I think the obvious first diagnosis ought to be CAGE and the patient ought to be taken as quickly as possible, even at the beginning of the behavioural changes, to a chamber for treatment. Again, I think it is a futile exercise to try to propose decompression sickness would be even a possibility in this case.

Banham

Her symptoms are initially quite vague and all of this obviously when it was pieced together became much more apparent. Other things we thought about were did she have a problem with her gas mix but carboxyhaemoglobin was less than one percent. Salt water aspiration crossed our minds but there were no other clinical features to suggest that and a chest x-ray was normal. So, fortunately the diagnosis was made fairly apparent on standing the patient up, despite this being about an hour and a half after the completion of her dive. I was just wondering if anyone else in the audience had actually seen this occur before.

Veale

It is a well documented phenomenon and certainly Des Gorman has a handful of delightful stories he can tell people in the same way. One of the ones I remember vividly is of a female diver at Stony Cove in the UK who lost consciousness at the surface following an abort from a deep rescue dive. She was brought ashore and was fine and regained consciousness and then they stood her up and she promptly died.

Banham

This woman had actually been standing, washing down her gear, sitting in a bus and walked into the dive shop.

Davis

Secondary deterioration is common, and fits very nicely with the modern concept of what is happening to the cerebral vessels as a result of gas embolism. Anybody who has read Des Gorman's papers will appreciate that about this time it is classic for the secondary deterioration in cerebral blood flow. Deterioration does not necessarily have anything whatsoever to do with a residual gas phase in the cerebral circulation, but is a reflection of the pathological injury that occurs as a result of the gas going through in the first place. The shallowest diver I have ever had in the chamber in Christchurch was in two metres from a swimming pool. He was a diving instructor who should never have been diving. He was grossly obese, had a symptomatic hiatus hernia with very clear evidence of recurrent pulmonary aspiration at night who, getting out of the pool during a class, became very dizzy on the pool side and fell over. He walked in to hospital. He began with very vague symptoms, poor on his Mini Mental Score and absolutely hopeless at sharpened Romberg and certainly that assessment is appropriate, in these people it was often the only physical finding. I have seen that in quite a number of divers and I expect Fred Bove has too.

Bove

The Dan reporting system received a case of a woman standing at the shallow end of a pool during a scuba class, water about up to her mid-chest, a mask was put down by her feet in the pool and she was asked, with her scuba tank on, to lie down in the pool, put the mask on, clear it and come up. She embolised and that was about four feet of water. Clear cut classic air embolism. I think that in a pool or shallow water with scuba gear on CAGE is got to be the primary diagnosis any time mental change is seen.

Unidentified speaker

I remember that when I was initially trained as a diver in the Navy in 1958 we had to do an exercise, with a

re-breather set, in a twenty foot tank where we had to take the full face mask off and then swim to the surface. In that short distance I well remember having a gush of cold water on my face, coming half way to the surface, feeling a great distension in my lungs and remembering, at that stage, now is the time to breath out.

Veale

Some of the original studies involved inflating the lungs of cadavers clearly showed lung rupture with a one metre or a 1.3 m ascent so transthoracic pressure changes of 75 to 100 cm of water pressure are enough to rupture lungs.

Banham

The phenomenon of arterial gas embolism from shallow depths is well recognised. What I am really trying to emphasise is the delay in onset of her symptoms which is somewhat atypical of acute arterial gas embolism, and then her sudden dramatic deterioration when she stood up some hour and a half later. Has anyone else actually seen a delayed significant deterioration such as that, rather than acute one?

Veale

Those of you that run, or have run, recompression chambers, hands up if you have seen delayed presentation of CAGE. That is four of us.

Conclusion

Despite the delay in onset the diagnosis was made of cerebral arterial gas embolism (CAGE), and urgent arrangements were made for recompression therapy. She was kept supine and given one hundred percent oxygen by non- rebreathing circuit using a mask with a tight seal. Intravenous fluids were started. By the time she was taken into the chamber she had improved but not back to the state she had been in before she was stood up. She was treated with a modified Table RN62 and made a full recovery.

The modifications to Table RN62 are that there is twice the time (one hour) decompressing from 18 m to 9 m at a rate of 0.15 m/minute, with a five minute air break midway. Instead of having periods of an hour on oxygen they are reduced to twenty minutes by five minute air breaks. It is not routinely extended either at 18 m or at 9 m. The total elapsed time is 5 hours and 20 minutes. Since the introduction of these modifications we have not had to extend at either 18 m or at 9 m.

There was no deterioration after the initial treatment. Because of persistent tiredness two follow up treatments (RN61) were given on subsequent days. After discharge the patient felt tired but was otherwise asymptomatic.

The patient was advised of the possible risks of further diving and chose not to continue diving.

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MEDICAL SUPPORT OF OPERATIONAL DIVING

NOVEMBER 1995

PRACTICAL COURSES IN 1996

Responsibility for providing both routine medical support and emergency medical care for operational diving usually falls on doctors who are geographically situated relatively close to a diving location but who may have had no previous experience of hyperbaric work and of the hazards of manned underwater activities. The purpose of these two weeks is to provide the essential knowledge required by such doctors to meet their obligations. Similar courses have been held periodically in Europe during the past 20 years with particular emphasis on the North Sea but this will be the first at a location convenient for doctors working in the region of the Pacific rim. It will be held at the Hyperbaric Medicine Unit in Fremantle, Australia in co-operation with local and international diving contractors.

The courses are planned for those with little previous knowledge but will also provide experienced doctors in this field with an opportunity to revise and update their knowledge and to discuss case histories and some of the medical problems encountered. The range of study covers applied physiology and both clinical and occupational medicine. Attendees will be doctors working in the oil and gas industry or supporting services, government or military doctors, occupational health specialists, hospital consultants or family practitioners. It is **not** necessary for the attendees to have diving experience or to be fit to enter a compression chamber but either would be an advantage. The courses will have practical sessions which will include simulated emergencies.

The academic directors will be **David Elliott** (Civilian Consultant in Diving Medicine to the Royal Navy and Medical Adviser to IMCA, the International Marine Contractors Association) and **Harry Oxer** (Director of the Hyperbaric Unit at Fremantle Hospital, Western Australia and Medical Consultant to several diving companies).

MEDICAL SUPPORT OF OPERATIONAL DIVING

WORKING DIVES to 50 METRES
30 Sept. to 4 Oct. 1996

The first course will emphasise physiology in the underwater environment and the medical problems that may be encountered. On the basis of applied physics and physiology, the range of equipment used will be reviewed together with the codes of practice that are relevant to safe and effective underwater work. Underwater accidents, emergency procedures and the management of decompression illness will be a major feature. Practical instruction and experience will be given with different types of equipment and the practical work will include simulated emergencies in the chamber and in the wet. Prior experience is an advantage but is not essential. This one-week module is self-contained and is designed to meet the essential needs of doctors who have a medical responsibility for compressed-air and nitrox divers.

Because these medical activities are often offshore, it is intended to include aspects of survival-at-sea including escape from a submerged helicopter.

For reservations made before 30 June 1996, the course fees will be Australian $1500 for one week or $2500 for two weeks. From 1 July 1996, these fees will become Australian $1650 and $2750 respectively and acceptance will be subject to availability because space will be limited.

Further details can be obtained from:
Dr. Harry Oxer,
Hyperbaric Medicine Unit,
Fremantle Hospital, PO Box 480,
Fremantle 6160, W. Australia

MEDICAL SUPPORT OF OPERATIONAL DIVING

WORKING DIVES DEEPER THAN 50 METRES
7 Oct. - 11 Oct. 1996

The second week will focus on mixed gas and saturation diving to meet the increasing needs of commercial diving in South East Asia. The instruction will include applied physiology of compression and the HPNS; equipment; breathing apparatus and environmental control; codes of practice; fitness for work; long-term health effects and, in particular, emergency procedures and medical management for divers in the water, the bell and the chamber complex.

Attendance at the previous week or some equivalent course elsewhere would be a useful foundation.

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT

Basic Course in Diving Medicine

Content Concentrates on the assessment of fitness of candidates for diving. HSE-approved course
Dates February 19th to 23rd 1996
October 28th to November 1st1996
Cost $A 500.00

Advanced Course in Diving and Hyperbaric Medicine

Content Discusses the diving-related, and other emergency indications for hyperbaric therapy.
Dates February 26th to March 1st 1996
November 4th to November 8th 1996
Cost $A 500.00
$A 800.00 for both courses

Diving Medical Technicians Course

Unit 1 St John Ambulance Occupational First Aid Course. Cost approximately $A 500
Unit 2 Diving Medicine Lectures. Cost $A 500
Unit 3 Casualty Paramedical Training. Cost $A 300

Dates

February 1996

Unit 1	5/2/96	to	9/2/96
Unit 2	12/2/96	to	16/2/96
Unit 3	5/2/96	to	23/2/96

July 1996

Unit 1	1/7/96	to	5/7/96
Unit 2	8/7/96	to	12/7/96
Unit 3	1/7/96	to	19/7/96

October/November 1996

Unit 1	14/10/96	to	18/10/96
Unit 2	21/10/96	to	25/10/96
Unit 3	14/10/96	to	1/11/96

Diver Medical Technician Refresher Courses

Dates
12/2/96-16/2/96
8/7/96-12/7/96
21/10/96-25/10/96
Cost $A 350

For further information or to enrol contact
Professor John Williamson, Director, HMU,
Royal Adelaide Hospital, North Terrace
South Australia, 5000.
Telephone Australia (08) 224 5116
Overseas 61 8 224 5116
Fax Australia (08) 232 4207
Overseas 61 8 232 4207

DIVING MEDICAL CENTRE

SCUBA DIVING MEDICAL EXAMINER'S COURSES

Courses for doctors on diving medicine, sufficient to meet the Queensland Government requirements for recreational scuba diver assessment (AS4005.1).will be held by the Diving Medical Centre in 1996 at:

Bond University,
Gold Coast, Queensland.
5th-7th April, 1996 (Easter Holidays)

Royal North Shore Hospital,
Sydney, New South Wales,
8th-10th June 1996 (Queen's Birthday Long Weekend)

Melbourne, Victoria
2nd-4th November 1996
Melbourne Cup Weekend

Previous courses have been endorsed by the RACGP (QA&CE) for 3 Cat A CME Points per hour (total 69)

Phone Brisbane -(07)-3376-1056 for further details

MARINE MEDICINE SEMINAR '96
JUNE 29TH - JULY 7TH 1996

A Seminar on Aquatic and Hyperbaric Medicine, Marine Animal Injuries and Diver's Medical assessments will be held in Bali, Indonesia, at the Bali Padma Hotel, Legion, over 5 half days (morning sessions).

Australian and Indonesian experts in Diving and Hyperbaric Medicine will lecture at the Seminar. RACGP point allocation is pending.

Excellent fare and accommodation packages apply but places are limited due to school holidays in Eastern States.

Phone Brisbane -(07)-3376-1056 for further details

Information and application forms for courses and seminar can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road,
Jindalee, Queensland 4047.
Telephone (07) 3376 1056
Fax (07) 3376 1056