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CONTENTS

REPORTS OF INTEREST, COURSES AND MEETINGS ARE DETAILED ON THE BACK COVER

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OBJECTS OF THE SOCIETY

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

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All contributions should be typed, double-spaced, using both upper and lower case, on one side of the paper only, on A4 paper with 45 mm left hand margins. Headings should conform in format to those in the Journal. All pages should be numbered. No part of the text should be underlined. These requirements also apply to the abstract, references, and legends to figures. Measurements are to be in SI units (mm Hg are acceptable for blood pressure measurements) and normal ranges should be included. All tables should be typed, double spaced, and on separate sheets of paper. No vertical or horizontal rules are to be used. All figures must be professionally drawn. Freehand lettering is unacceptable. Photographs should be glossy black-and-white or colour slides suitable for converting into black and white illustrations. Colour reproduction is available only when it is essential for clinical purposes and may be at the authors' expense. Legends should be less than 40 words, and indicate magnification. **Two (2) copies of all text, tables and illustrations are required.**

Abbreviations do not mean the same to all readers. To avoid confusion they should only be used after they have appeared in brackets after the complete expression, e.g. decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is 2,500 words or less. Inclusion of more than 5 authors requires justification. Original articles should include a title page, giving the title of the paper and the first names and surnames of the authors, an abstract of no more than 200 words and be subdivided into Introduction, Methods, Results, Discussion and References. After the references the authors should provide their initials and surnames, their qualifications, and the positions held when doing the work being reported. One author should be identified as correspondent for the Editor and for readers of the Journal. The full current postal address of each author, with the telephone and facsimile numbers of the corresponding author, should be supplied with the contribution. No more than 20 references per major article will be accepted. Accuracy of the references is the responsibility of authors. Acknowledgments should be brief.

Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references which should be limited to 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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All manuscripts will be subject to peer review, with feedback to the authors. Accepted contributions will be subject to editing.

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PROJECT STICKYBEAK

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

Information may be sent (in confidence) to:
Dr D. Walker
P.O. Box 120, Narrabeen, N.S.W. 2101.

DIVING INCIDENT MONITORING STUDY (DIMS)

DIMS is an ongoing study of diving incidents. An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report any incident occurring in your dive party, but do not identify anyone. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver damage. Using this information to alter diver behaviour will make diving safer.

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

The Editor's Offering

SPUMS was formed to educate doctors and others about diving and hyperbaric medicine. The main tools that we have are this Journal and the Annual Scientific Meeting (ASM). Unfortunately only a few members, less than 8% of the membership, attend the ASM, so the educational load is carried by the Journal. With this issue comes a questionnaire, with addressed envelope for your reply, about your desires for the ASM and the Journal. We hope that everyone will find the time to fill it in and return it as this will help the Committee serve the membership more effectively.

The theme of the 1994 ASM was the management of diving accidents. Des Gorman's paper gives a practical overview from the stand point of the treating hyperbaric physician. The average diver never expects to be part of a diving accident, which may explain the many incidents reported to the Diving Incident Monitoring Study (DIMS). At present few trainee divers are taught how to rescue an unconscious diver and then give expired air resuscitation (EAR). Most of the training agencies keep this knowledge for their Rescue Diver, or equivalent, qualification. However it is know that not every trainee goes on to do a Rescue Diver course, so the chances are high that the diver who is faced with rescuing an incapacitated buddy will have had no training in what to do. Given this likliehood, one can only applaud Dr Gorman's view that getting the victim out of the water takes priority over EAR in the water unless the tow is a long one. Getting people who cannot help, out of the water is difficult except on a gently sloping beach without surf. Heaving 70 to 90 kg of inert human up even 150 mm (6 inches) onto a rock is hard work. Lifting the person from water over the gunwale into a boat with a freeboard of 300 mm (1 foot) is just about impossible for the normal, non-weight lifter, male. They have to be dragged in by heaving on the arms as the rescuer will severely unbalance the boat and risk hurting his back if he tries to lift the victim by the axillae. So there is a good case for always going diving with at least two buddy pairs in the hope that at least three people are about to help lift the victim (yourself) into the boat ! Besides the man or woman power you will need a flat surface to get under the victim, a short aluminium ladder will do nicely, and at least a pair of ropes long enough to go from the far side of the boat under the ladder and back again. Finally you need somewhere to attach one end of the rope. Just the things for your family, or your concerned buddy, to give you.

Perhaps SPUMS members should set the rest of the diving world an example and practice rescue techniques on every dive, as one never knows when the next diving accident is going to happen. Another safety feature that could do with polishing up by most divers is buddy diving. Most buddies get into the water together but soon after separate to the limit of visiblity. To be of any use to you, your buddy has to be almost within touching distance at all times, and never more than 2 m from you. Only in this way can you get to your buddy easily, and your buddy can reach you rapidly in an emergency. Incident reports and death surveys show that buddies are often absent when trouble strikes.

Rescuing people from the water, especially cold water, is associated with a surprising level of deaths before, during and after retrieval. For years this was attributed to hypothermic after-drop, but deaths during rescue in people who had been conscious at the beginning of the rescue and in those who were actively assisting in their rescue, for instance by climbing the boarding net, led people to question the attribution to hypothermia as the cause of death. Although the German Air-Sea Rescue Service had discovered, in the 1940s, that the death rate in rescued pilots was much reduced if they were lifted into the boat horizontally, no one much took notice of this. It was the horrendous death rate in those rescued, using a strop fitted under the arms which lifted them hanging feet down, by helicopter during the 1979 Fastnet Race, when many of the yachts were disabled or sunk by the storm, which made people seriously question the accepted wisdom. Since then Golden and his colleagues have shown that the cause of death is likely to be the sudden loss of hydrostatic support of the legs, abdomen and chest, as the person is pulled from the water, allowing vasodilatation and pooling of blood in the legs and consequent fainting from reduced, or effectively no, cardiac output. If the person is not put level very rapidly death is likely to follow. The Roman crucifixion killed by keeping the person upright after they had fainted.

Medical knowledge expands faster than most doctors can keep up with all that is known, even in their speciality, and earn a living. Surfactant appears in the medical text books as the agent responsible for opening up the newborn infant's alveoli. But in the past ten years or so surfactant has been found in all sorts of unexpected places in the body. Brian Hills, who came to physiology and diving medicine via industrial chemistry and waterproofing agents, presents an update of the world of surfactant and its uses.

George Jelinek's diploma thesis deals with carbon monoxide poisoning from the use of charcoal barbecues as heaters. Most people use barbecues out of doors because of the smoke and smell of cooking. Many charcoal barbecues are small and easily carried into the house. Few people even know of, much less consider, the risk of carbon monoxide poisoning from a charcoal barbecue where there is not much ventilation. Here is another educational opportunity as many cases of carbon monoxide poisoning go unrecognised in Emergency Departments.

ORIGINAL PAPERS

SURFACTANT UPDATE

Brian Hills

In 1983 it was a great pleasure to be the invited speaker at the Annual Scientific Meeting of SPUMS held in Fiji. One of my lectures was entitled "Surfactant", a topic which I have pursued vigorously since returning to Australia.

In medical textbooks surfactant is mentioned only in connection with the lung and then only as acting at the liquid-air interface of a continuous fluid lining assumed to coat the alveolar surface. Although I have challenged this assumption in the normal adult lung (Fig.1), it is much more likely to hold in the neonate where, at birth, the liquid-air interface needs to expand from about 2 cm^2 to 30,000 cm^2 within a few minutes. Hence the field tends to be dominated by paediatrics in which "surfactant rescue", i.e. the application of exogenous surfactant for the respiratory distress syndrome (RDS), has reduced mortality at birth by the order of 40-60%. The major research thrust over the last decade has been directed at better formulations for this exogenous surfactant. The recent recipes include not only the highly surface-active dipalmitoyl phosphatidylcholine (DPPC) which actually reduces surface tension but also the associated proteins which enable those DPPC molecules to be recruited so rapidly to enable expansion of the interface to occur. Two of these proteins are so hydrophobic as to be "co-extractable" from lung surfactant with DPPC by typical lipid solvents. These are termed "hydrophobic surfactant proteins". Although never referenced in the respiratory literature, these hydrophobic proteins have a remarkable resemblance to proteolipids discovered in the CNS in 1953 and subsequently found known to have a major role in the myelination process, the predominant component of myelin (and the electrical insulator) for the axons being DPPC and similar saturated phospholipids.

Having emphasised the highly desirable role of DPPC and proteolipid in the rapid *expansion* of the liquid-air interface in the newborn, this combination would be highly undesirable in the tissues of a deep-sea diver where the gas phase is the last thing one would wish to create or to have its growth promoted. It was therefore particularly interesting last year to find this same combination of DPPC and proteolipid when I looked at the CNS of sheep and, moreover, to find even more in spinal tissue.[1] Maybe this is the vital entity which renders the spinal cord so vulnerable to decompression injury compared with the brain. Our studies of spinal tissue on the electron microscope with our special "surfactant fixative" have also revealed more lamellar bodies (LBs) in spinal tissue,[2] these being

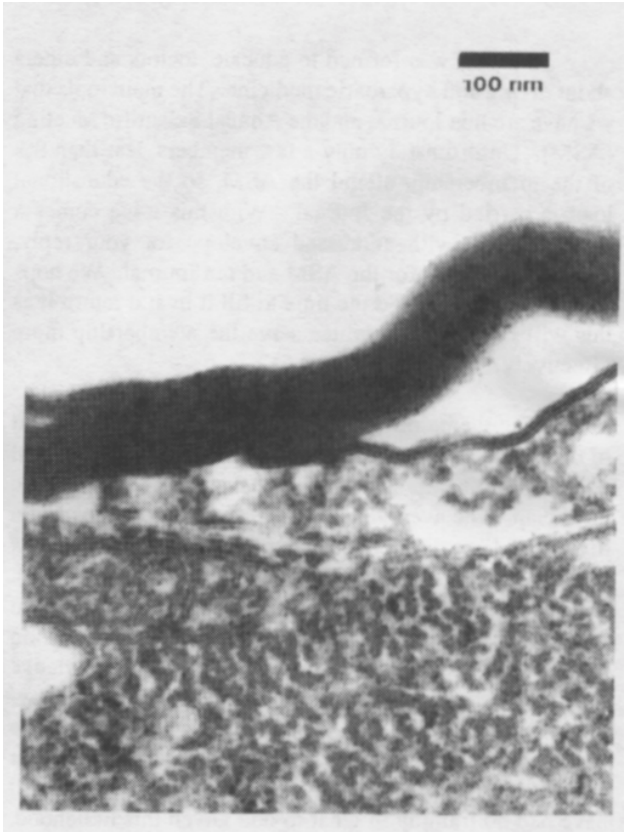

Figure 1. The alveolar lining showing an oligolamellar layer of surfactant both attached to the epithelial wall and detaching from it, although one extra lamellation remains adsorbed to the membrane. Note the traditional "tramlines" of membranes in deeper tissue structures.

the "packages" in which the body stores surfactant (and its proteins) in such a highly surface-active state, especially in the lung. In these "packages" surfactant is instantly available for recruitment to a bubble surface.

Another worrisome aspect of our recent spinal studies is our finding that bubble formation by decompression not only liberates DPPC from spinal tissue[1] but also a proteolipid which is known to be encephalitogenic, i.e. it promotes demyelination and is used to produce experimental animal encephalomyelitis (EAE). EAE is the animal model widely used for studying multiple sclerosis. Hence it is tempting to speculate that this finding might provide the link with multiple sclerosis (MS) in retired divers proposed by James[3] although, apart from one very impressive case known to me personally, published case histories are difficult to find.

To return to more positive aspects of surfactant, it has always amazed me that so little of the vast wealth of knowledge of industrial surfactants has been applied to

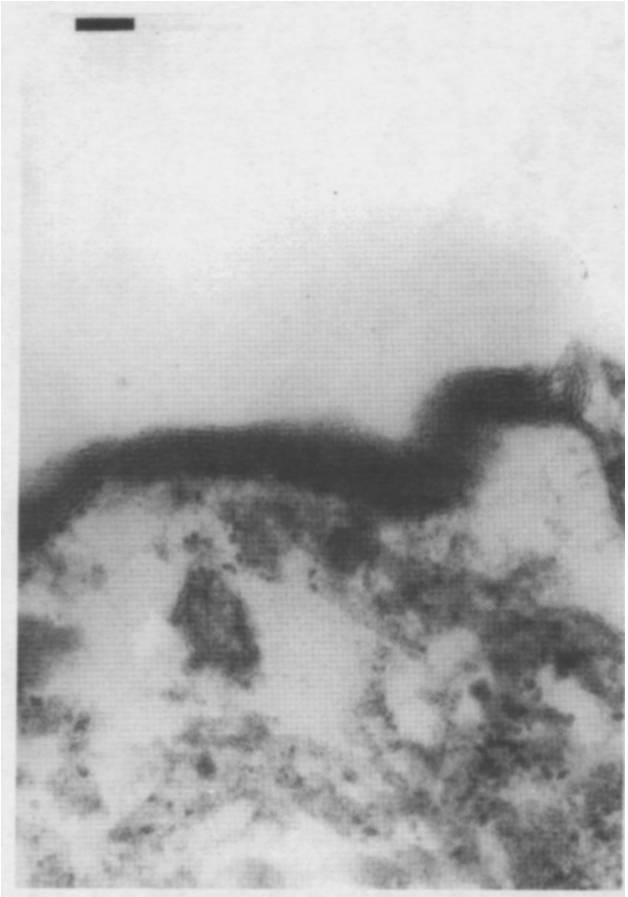

Figure 2. Gastric surfactant found at the interface between the gastric mucosa and gastric contents of the stomach. This layer appears to reform if gastric mucus is rinsed away leaving the stomach wall equally hydrophobic. The bar represents 50 nm.

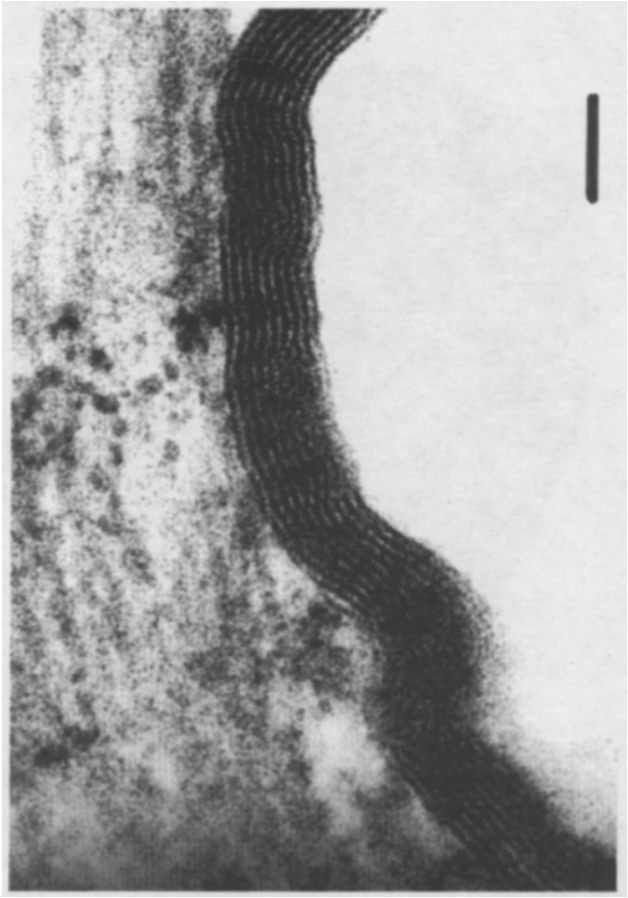

Figure 3. An oligolamellar lining of surfactant coating the epithelial surface of an oxyntic duct through which hydrochloric acid, pepsinogen, etc. at pH = 1 is secreted into the lumen of the stomach. The bar is 50 nm.

Physiology and Medicine and that what has been applied has been limited to the liquid-air interface. The major commercial application of surfactants nowadays is to solid surfaces, to which surfactants can bind reversibly by adsorption and, in so doing, impart many highly desirable properties. One of these is the inhibition of corrosion as typified by the monolayer which is the active ingredient in the underseal applied to motor cars to prevent rusting. Since the early days when we had identified a layer of surface-active phospholipid (SAPL) on the gastric mucosa as a characteristically hydrophobic lining[4] and its elimination by the “barrier breakers”, e.g. bile salts, NSAIDs, etc., there have been several new developments.

Since arriving at the University of New England my major effort has been devoted to the morphology of the adsorbed lining. Three decades ago Davenport[5] had proposed a “gastric mucosal barrier" of unknown composition to the back diffusion of acid, but this term has faded from the literature for lack of ultrastructural evidence. Our approach was to argue that the universal fixative (glutaraldehyde) used in a multitude of previous ultrastructural studies would never reveal any adsorbed SAPL because it is well known to destroy hydrophobic surfaces. Substituting tannic acid we were able to demonstrated a lining of SAPL over the gastric mucosa (Fig. 2). However, it was not a monolayer, as theory might predict, but an oligolamellar layer and also coated the oxyntic ducts, through which acid produced by the parietal cells (at pH = 1) pass into the lumen of the stomach (Fig. 3). Moreover, when we investigated the parietal cells and mucus-neck cells we found lamellar bodies virtually identical to those found in the lung (Fig. 4).

These findings led us (and others) to administer exogenous surfactant as an anti-ulcer remedy with moderate success. My next move was to look for a natural source of lamellar bodies, ideally a vegetable source, and this we found in the ripe banana. This, in turn, has led to a banana-based product which is currently undergoing clinical trials as a very inexpensive anti-ulcer product which avoids suppressing acid and the associated side-effects. To date, the results are most encouraging.

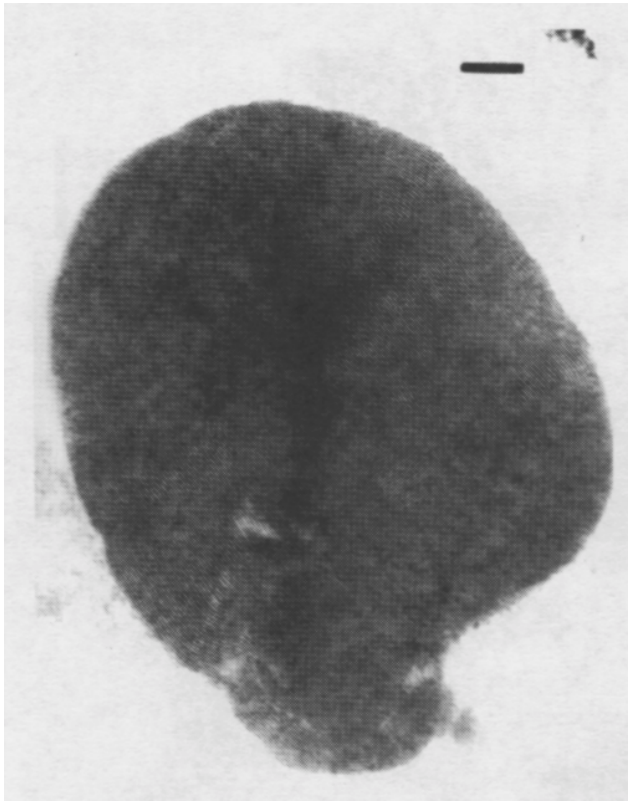

Figure 4. A lamellar body found within an oxyntic duct similar to those found within parietal cells, i.e. the cells which produce hydrochloric acid. The bar represents 100 nm.

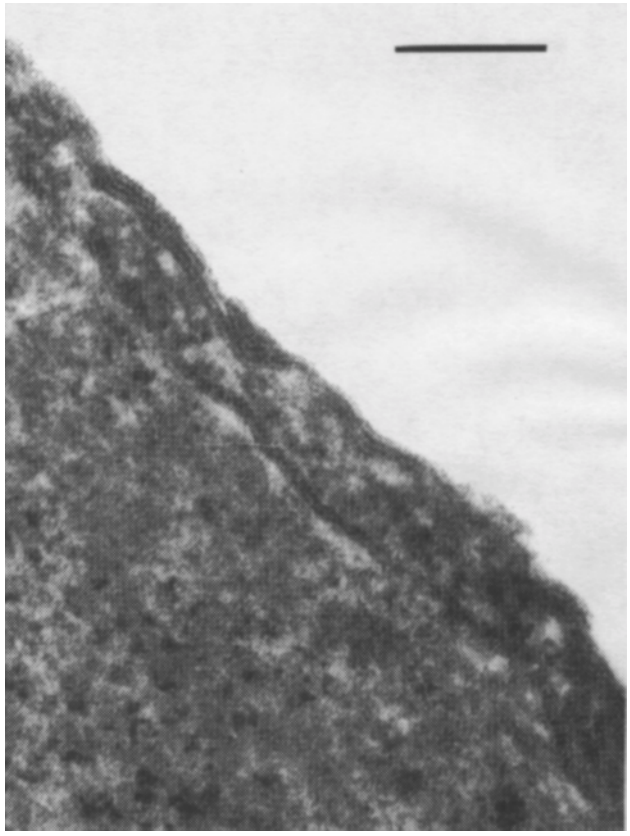

Figure 5. Lamellated surfactant coating the ocular surface and, presumably, adsorbed to corneal epithelium. This lining appears to render the eye hydrophobic causing the tear film to break up, even in saturated air, unless one blinks every 20 seconds or so. The bar represents 100 nm.

In ancillary gastrointestinal studies, I have identified surfactant in lesser quantities in the lower oesophagus, colon and in the duodenum, but only of patients with coeliac disease. Hence we have speculated that coeliac disease could result from unwanted surfactant effectively extending the gastric mucosal barrier into the duodenum where it now impairs absorption.[6] In a recent study we have found morphological evidence of surfactant lining the duodenal epithelium of these patients. Since the discovery in Perth, Western Australia, of *Helicobacter pylori* thriving in the highly corrosive environment of the stomach, and their association with peptic ulcer,[7] these bacteria have become a major, (and controversial) topic in the gastroenterological literature. Hence it was most interesting when our ultrastructural studies displayed how *H. pylori* derive protection against digestion in the stomach by adopting the same defence as the gastric mucosa, i.e. an oligolamellar layer of surfactant. Another surprise was to find essentially the same coating on Barber's pole worms. These parasites thrive in the highly acidic conditions of the abomasum of some ruminants making them the scourge of the Australian sheep farmer.

Another very hydrophobic surface is the articular surface on which we demonstrated adsorbed SAPL and the remarkable lubricating properties of this material.[8] It could reduce the coefficient of kinetic friction (μ) to 0.001 at high load (13 kg/cm^2) and low velocity which is phenomenal when compared with the best man-made boundary lubricants, e.g. Teflon for which $\mu = 0.04$. When applying our novel fixation procedure for electron microscopy, it was fascinating to see the same oligolamellar layers, highly reminiscent of graphite or molybdenum disulphide, which are used widely in many industrial lubricants. We have now found this same graphite-like layer on other sliding surfaces *in vivo*, such as the pleura and cornea (Fig. 5). This has obvious clinical applications to the irrigation of arthritic joints and better artificial tears, both of these applications are being pursued in clinical trials.

Another possible application of lubrication by SAPL concerns premature rupture of the membranes (PROM) which, when it occurs before term, is often termed the enigma of the obstetrician. Amniotic surfactant is an excellent load-bearing lubricant which also deposits a graphite-like layer on the chorioamnionic sac.[9] Hence it was interesting to find a very good correlation between pre-term PROM and lack of lubricity for placentas of the same gestational age. When surfactants impart lubricity to

a surface, they also reduce sticking, acting as release agents, and this could apply to any potential for adhesion of the foetus or maternal viscera to the membrane to cause the local mechanical stress required to initiate rupture.

Returning to diving, there is an interesting application of surfactant in aural barotrauma since we have demonstrated it lining the Eustachian tube where it is a very good release agent.[10] This is also relevant to serous otitis which can occur if the inner ear is not ventilated every 20 minutes or so. Hence the application of exogenous SAPL offers a challenge to the otolaryngologist.

Another surprising finding with implications to underwater medicine is our discovery of the same oligolamellar layer lining cerebral endothelium, rendering it hydrophobic.[11] This has several implications for the diver during decompression. Firstly, it offers a hydrophobic surface which is more conducive to bubble formation as demonstrated so simply and elegantly in the 1950s when Harvey[12] produced profuse bubbling upon plunging a candle into soda water. The second implication is much more speculative in so far as this SAPL lining might provide the elusive blood-brain barrier (BBB).[11] A liquid-air interface in the form of a circulating bubble could be expected to compete much more successfully than endothelium for its SAPL coating in which case, it would open the BBB. This very serious action of circulating bubbles has been known since the work of Broman[13] and, more recently, emphasised by Gorman[14] to explain the neuropathology they cause. It is my contention from early studies of air embolism[15] that small isolated bubbles transverse the cerebral circulation, but clusters of bubbles coalescence to cause the infarction postulated since the work of Paul Bert in the 1840s.[16]

Returning to the lung, and pulmonary barotrauma in particular, it was interesting to find surfactant not as the monolayer located at the surface of the liquid lining but as rafts of solid surfactant either floating at that interface or adjacent to alveolar epithelium. In either case they appeared capable of sealing small pores connecting the air space to deeper structures and even to the vascular space.[17] This could shed some light upon the question asked over a century ago by Ewart and Kobert:[18] "ist die Lunge luftdricht?" for which his answer was "nein". The rafts of surfactant might act as flap valves to raise the overpressure before air could enter the pulmonary vasculature.

Our latest study of the lung has pursued the hypothesis that there is an additional role for surfactant in the lung apart from those traditionally attributed to it. This is attributed to the large hysteresis in surface tension (γ) versus surface area (A) during the respiratory cycle and, hence, a large hysteresis in the collapsing pressure acting upon stretch receptors in the lung as its volume changes. Hence there is a hysteresis in the afferent neural input to the brainstem vital for normal respiration which would enable the respiratory pattern generator to distinguish between inspiration and expiration at the same lung volume. Hence it was particularly exciting to find that surfactant samples obtain from SIDS victims or from infants experiencing recurrent cyanotic episodes had γ:A loops cycling *anticlockwise* rather than in the normal *clockwise* direction.[18] Thus the brainstem would receive a highly confusing neural feedback from the lungs and might even mistake expiration for inspiration, which would fit the clinical findings. Even if this hypothesis proves incorrect the reversal of γ:A hysteresis offers a simple test of those infants at risk of SIDS, enabling them to be monitored carefully until they have outgrown the problem. This test would be particularly useful for detecting the risk *at birth* by using nasopharyngeal aspirates routinely obtained by the standard clinical practice of suctioning neonates during delivery. These aspirates are currently flushed down the drain but they actually provide a very good sample of lung surfactant and one routinely available just at the age when it is needed. I have joined the staff of the Mater Children's Hospital in Brisbane to pursue this very exciting avenue in more detail.

In conclusion, the very wide range of diseases discussed above emphasises the ubiquity of surfactant, while there are other locations in the body in which we have identified it such as the kidney and the cochlea. The area of immediate clinical application, however, is the ubiquitous barrier it offers to potentially corrosive and abrasive agents.

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UNINTENTIONAL CARBON MONOXIDE POISONING FROM CHARCOAL BARBECUES

George Jelinek

Summary

In two weeks in the winter of 1993 eight members oftwo families suffered paoisoning with carbon monoxide produced by charcoal barbecues used indoors to provide heating. Following these incidents a review of the literature was carried out. This paper presents clinical details and reviews the literature. Recommendations are made for public health measures to combat this unintentional form of CO poisoning.

Introduction

Carbon monoxide (CO) is an agent frequently used for deliberate self-harm. Poisoning with CO is the commonest cause of death by completed suicide in industrialised nations.[1-3] Unintentional CO poisoning is also common although the incidence is more difficult to determine. Many cases are probably unrecognised.[4-6] Dolan suggests that it is the protean nature of symptoms of CO poisoning which causes its true incidence to be grossly underestimated.[7] It is likely that many cases are also missed at autopsy because the pathological lesions are non-specific.[8] Common mechanisms include leaky motor vehicle exhausts, faulty home heaters, and inadequate ventilation around appliances producing CO.[1,9-11]

This paper describes two non-English speaking families who presented to the Emergency Department of Fremantle Hospital approximately two weeks apart in the middle of winter 1993. Eight people, six in one family and two in the other, suffered CO poisoning following indoor use of a charcoal barbecue for heating. There have been occasional reports of CO poisoning in similar circumstances in the medical literature. In January 1994, a large series of 79 patients was reported from Washington.[12] CO poisoning from charcoal barbecues has not previously been described in Australia.

The cases reported here illustrate the ease with which accidental CO poisoning can occur. This leads to a review of the epidemiology and effects of unintentional CO poisoning with particular emphasis on chronic exposure which has thus far not been discussed in detail in the literature.

Case reports

FAMILY A

This family of six (mother 24 years old, father 37 years old, two daughters 11 and 9 years old, and two sons, 10 and 6 years old) presented to the Emergency Department at Fremantle Hospital at 0400 hours on 30 June 1993. They had taken a charcoal barbecue indoors to use as a heater overnight after cooking the evening meal. In the early hours of the morning, the mother lost consciousness on the way to the toilet. On regaining consciousness after an estimated period of four minutes, she alerted the rest of the household. Her 11 year old daughter was roused but lost consciousness twice for a total of 15 minutes and vomited once while being taken outside the house. The six

year old boy lost consciousness once for approximately five minutes and the nine year old girl once for one minute. The father and the other boy left the house uneventfully.

On arrival in the Emergency Department, only the 10 year old boy was asymptomatic. The mother felt very tired and the father complained of a severe headache and felt drowsy. The 11 year old girl felt drowsy, and the nine year old girl complained of a headache and drowsiness. The six year old boy had a slight headache.

There was no significant past medical history apart from a history from the six year old boy of mild asthma treated with salbutamol inhaler. None of the family smoked cigarettes. The family was Vietnamese and had been in Australia six months. The children spoke sufficient English to allow communication.

TABLE 1

	Age	MMSE	COHb	LOC
Father	37	30	4 %	No
Mother	24	26	2 %	Yes
Girl	11	28	4%	Yes
Boy	10	30	*	No
Girl	9	30	4%	Yes
Boy	6	*	3 %	Yes

* not done

Age (years), mini-mental state examination results (out of 30), carboxyhaemoglobin levels (%), and whether there was loss of consciousness (LOC) for members of Family A.

Physical examination of all six patients was normal. Mini-mental state examinations (MMSEs) were performed with the assistance of the children and results are listed in Table 1. These are scored out of a possible 30 according to the standard format of Folstein et al.[13] Carboxyhaemoglobin (COHb) levels were done on five of the patients and are also listed in Table 1. No blood was taken from the 10 year old boy who was terrified of needles. Electrocardiographs performed on both adults were normal.

Treatment was commenced with 100% oxygen by tight-fitting mask, non-rebreathing valve and reservoir bag. All six patients were treated that morning with hyperbaric oxygen on a standard 18 m table, breathing 100% oxygen at an absolute pressure of 2.8 atmospheres. The indication for treatment was loss of consciousness for the three females and the six year old boy. The other two patients were treated at the same time as they were felt to have received a similar dose of CO, and the chamber could accommodate them. They received a second 18 m treatment as outpatients next day. All were discharged symptom-free.

FAMILY B

A 42 year old woman presented to the Emergency Department at Fremantle Hospital at midnight on 13 June 1993. She had cooked indoors with a charcoal barbecue and left it burning inside as heating. Her 13 year old son had also been in the house.

TABLE 2

	Age	MMSE	COHb	LOC
Mother	42	28	12.0%	Yes
Boy	13	23	4.4%	No

Age (years), mini-mental state examination results (out of 30), carboxyhaemoglobin levels (%), and whether there was loss of consciousness (LOC) for members of Family B.

After the barbecue had been inside about three and a half hours, she began to feel unwell with headache, dys-pnoea and pounding in her ears, and she vomited four times. She telephoned her sister and lost consciousness briefly on her arrival. There was a past history of bronchiectasis and asthma, treated with salbutamol by inhalation. She was a non-smoke born in El Salvador.

Physical examination was normal. MMSE result with interpreter assistance was 28/30 and COHb level was 12%. White cell count was 17.1×10^9/L.

She was treated with 100% oxygen by tight-fitting mask, non-rebreathing valve, and reservoir bag. While awaiting hyperbaric oxygen therapy, her 13 year old son was contacted by telephone. He complained of headache, palpitations, difficulty with coordination and generalised weakness. He was advised to present to the Emergency Department.

On arrival in the Emergency Department, there were no abnormal physical findings. MMSE result was 23/30 and COHb level was 4.4%.

For similar indications to Family A, both patients were treated with hyperbaric oxygen on an 18 m table, followed by a second 18 m treatment as outpatients next day. Both were discharged symptom-free.

Background and literature review

The literature on CO poisoning due to charcoal barbecuing is reviewed. To put this problem into perspective, the epidemiology of other causes of unintentional CO poisoning is examined. Accidental exposure to CO as described in this report can clearly occur regularly in

TABLE 3

REPORTS OF UNINTENTIONAL CO POISONING DUE TO CHARCOAL COMBUSTION

Authors	Year	Number of patients	COHb levels	Ethnic background
		Charcoal barbecues		
Anonymous[14]	1966	1 fatal	Not stated	Not stated
Wilson et al.[15]	1972	5 fatal	65%	Not stated
		2 non-fatal	71%	
			82%	
Fain & McCormich[16]	1988	2 fatal	30%	Not stated
			41%	
Baron et al.[11]	1989	1 fatal	Not stated	Not stated
Geehr et al.[17]	1989	Not stated (<22)	Not stated	Not stated
Gasman et al.[18]	1990	12 non-fatal	6.9%	Yes
			15.1%	
			17.1%	
			17.4%	
Sternbach & Varon[19]	1990	5 non-fatal	18.5%	Yes
			21.1%	
			25.5%	
			31.9%	
			38.3%	
Hampson et al.[12]	1994	79	Average 21.6%	73%
		Charcoal fires in enclosed spaces		
Finck[8]	1966	2 fatal	Not stated	Not stated
Wilson et al.[15]	1972	2 fatal on review	Not stated	Not stated

certain situations. This leads to a consideration of the circumstances in which such exposure to CO may occur, particularly for certain occupational groups, and its possible effects.

CO poisoning due to charcoal barbecuing

There have been several reports in the literature of CO poisoning due to charcoal briquets burned indoors. A report in 1966[14] described several fatalities from CO poisoning in confined spaces. One of these was the case of a man who was using a charcoal barbecue grill outside when a rain shower caused him to take the grill into his tool shed. He was found dead by his wife some time later.

In 1972, five fatal and two non-fatal cases of CO poisoning were reported following the use of charcoal barbecues in confined spaces.[15] The deaths occurred in a camper van, a station wagon, a trailer and in a cellar. The three victims on whom autopsies were performed had COHb levels of 65%, 71% and 82%. Review of the medical literature by the authors also highlighted five other deaths in similar circumstances, three of which are further described below.[8,14] Because the hot coals after cooking on a charcoal barbecue do not emit irritating fumes, the authors felt that they were a particularly attractive option for indoor heating for people not acquainted with the dangers of CO poisoning. They suggested that warnings be printed on such barbecues, as they are on packets of charcoal heat beads.

In 1988, two deaths were reported from CO poisoning due to charcoal barbecues being used in enclosed camping facilities.[16] The two patients, a 53 year old man and a 12 year old boy, had COHb levels of 41% and 30% respectively. In 1989 a large series of fatal CO poisoning from non-vehicular sources was described.[11] One of the deaths was listed as due to charcoal use in a confined enclosure although further details of that case were not provided.

In 1989, the emergency health impact of a severe storm in New York was detailed.[17] Five hospital emergency departments in the region, accounting for 190,000 annual patient visits, were surveyed. Most attendances were for injuries such as fractures, abrasions, and lacerations. Surprisingly, the most common non-surgical reason for attendance was CO poisoning which caused 22 attendances. The precise number of cases of CO poisoning

due to charcoal barbecues was not stated, however it was noted that common to all such cases was indoor use of barbecues in poorly ventilated areas and that two incidents involved multiple family members.

In 1990, 12 members of a non-English speaking family were reported to have developed CO poisoning from indoor use of a charcoal barbecue.[18] They had initially presented over several hours in groups of four with non-specific symptoms suggestive of food poisoning. COHb levels on the four patients present in the emergency department were 6.9%, 15.1%, 17.1% and 17.4%.

This prompted a report of two 31 year old, non-English speaking women who presented to the Emergency Department at Stanford Medical Centre, and three other family members at home, all poisoned with CO from indoor cooking on a charcoal barbecue.[19] COHb levels on these patients were 38.3%, 31.9%, 25.5%, 21.1% and 18.5%.

A large series of patients with CO poisoning due to charcoal barbecues was reported in 1994.[12] Seventy-nine patients were treated over 14 years to October 1993, referred from 10 counties in the state of Washington. The criteria used for treatment with hyperbaric oxygen (HBO) were a COHb level of 25% or more, angina or ischaemic changes on ECG, or neurological impairment including transient loss of consciousness.

These 79 patients represented 16% of all cases of unintentional CO poisonings treated during the study period. As expected, most occurred in winter, and ethnic minorities were over-represented (73%). Most incidents (69%) involved more than one individual. COHb levels averaged 21.6% (SD 9.6%), and a third of patients lost consciousness. Headache was the most common (67%) symptom. Two other deaths from CO poisoning due to a charcoal fire in a foxhole have also been reported.[8]

Interestingly, levels of COHb above 5% have been found in over half of studied non-smoking workers in charcoal grilling occupations and in over 80% of smoking workers.[20] It is possible that charcoal burning in confined spaces may be particularly liable to cause CO poisoning due to the high levels of CO released in charcoal combustion. Charcoal barbecues have been shown to produce an air stream containing CO at a level of 20 to 2,000 ppm, with 75% producing levels of 200 ppm or more. This is the level defined as the maximum safe level by the US Department of Labor's Occupational Safety and Health Administration.[21]

CO poisoning from appliances used for heating and cooking

There have been many case reports of individuals or groups poisoned with CO due to heating with other appliances in enclosed spaces. In 1978, three incidents of CO poisoning from blockage of flues for home heaters were reported, with three deaths and nine other casualties.[22] Seventeen deaths were reported from Switzerland in the early 1980s due to CO poisoning from gas water heaters without proper ventilation.[23] In 1983, two reports from Denmark detailed several deaths and near-fatalities from hot water systems.[24,25] Caplan et al. reported 11 incidents of CO poisoning from home heating systems in 1986 in which 16 patients died.[26] A study in Brussels in 1987/88 confirmed the danger of faulty water heaters.[27] It showed that about two thirds of all incidents of unintentional CO poisoning in the homes surveyed were due to hot water systems.

In 1988, two mountain climbers were reported to have succumbed to CO poisoning after cooking inside a tent at 14,200 feet.[28] It should be noted that there is some experimental animal work suggesting that high altitude results in higher COHb levels for a given exposure[29] and that it exacerbates some cardiovascular effects of CO poisoning.[30] The same year, several deaths from gas water heaters were reported from Copenhagen.[31] A 1989 report described a family of three poisoned from a central heating system running on butane gas.[32] The three family members had COHb levels of 4.8%, 6% and 8.5%. All three developed ECG abnormalities which improved after hyperbaric oxygen therapy. In the same year, Wharton et al. detailed five people at a hotel poisoned by CO drawn into the air conditioning from gas heaters for the indoor swimming pool.[33] One died. A report in 1991 described six civilians poisoned with CO during the Gulf War after being advised to stay inside in sealed rooms due to the risk of chemical weapon attack.[34]

Several instances of mass CO poisoning from home heating during a severe storm in Washington were reported in 1993.[35] Because of low temperatures and power failures, many residents of the area developed CO poisoning from improvised indoor heating. Recently, Rudge provided details of 300 patients treated at a military hyperbaric facility.[36] Infants were shown to be particularly likely to have been affected by faulty home heaters.

Epidemiology of unintentional CO poisoning

These reports highlight the dangers of using certain appliances indoors for heating or cooking. There are however other, more common causes of unintentional CO poisoning. Motor vehicle exhaust is the most common, followed by exposure to CO during fires or burns. There are many other causes also reported in the literature, with home heating and cooking appliances contributing significantly to overall incidence.

A 1991 study reviewed 10 years of death certificate reports in the United States.[9] The number of cases where

unintentional CO poisoning had contributed to death was determined. Of 56,133 deaths in which CO was a contributing factor, 25,889 (46%) were suicides, 15,523 (28%) were associated with fires or burns, and 11,547 (21%) were unintentional. They found that the number of unintentional deaths was gradually decreasing. Most of these unintentional cases (57%) were due to motor vehicle exhaust, usually while stationary, and most were elderly males in winter in colder climates. It was felt that improvements in motor vehicle emission systems and home heating appliances had contributed to the fall in incidence. A further study analysing the contribution of weather conditions to the incidence of unintentional CO poisoning confirmed that winter was the peak season.[37] The authors suggested that during certain high-risk periods such as particularly cold weather, health authorities could play a preventative role by alerting physicians and the community to these risks.

A three year study in France[10] found that 735 cases (17.5 per 100,000) had been reported to emergency organisations or laboratories. There were 291 incidents responsible, with 12% due to fires and 4% to car exhausts. Of the remaining 196 domestic causes, most were due to water heaters (57%), followed by boilers (20.5%), coal stoves (9%), braziers (4%), cookers (2%) and heating devices (1.5%). The types of cooking and heating devices were not specified. There were a few other miscellaneous causes.

A study from West Virginia showed that, from 1978-1984, there were 62 unintended fatalities not involving a motor vehicle.[11] This represented nearly half (42%) of all unintentional CO fatalities in that time. The deaths were predominantly due to faulty or poorly vented or ventilated cooking or heating appliances.

Other studies have shown a similarly high rate of CO poisoning from motor vehicles. Fatalities have been recorded not only from faulty motor vehicle exhaust systems but also from cars running in enclosed spaces.

Further West Virginian studies detailed 82 vehicle-related CO-caused fatalities over seven years.[38] Of the 64 incidents responsible for the deaths, 50 were due to defective exhausts and 14 due to poor ventilation in enclosed garages. In Florida, 15 cases over five years were reported of unintentional poisoning from motor vehicle exhaust, mostly due to running the vehicles in enclosed spaces.[39]

It is likely that the incidence of non-fatal CO poisoning from motor vehicle exhaust is higher than reported. For example, seven of 18 drivers in one United States region tested after road traffic crashes had COHb levels of 25% or more, and a salesman driving a car with defective exhaust had four serious crashes in 10 weeks.[14]

After eight schoolchildren developed CO poisoning in a school bus in Seattle, CO levels in such buses were measured.[40] A number had CO levels which were above acceptable limits. After a period of idling in the parking area, over a third had levels which were too high. Interestingly, CO levels in vehicles on busy highways have been shown to be higher with increased traffic volumes and reduced speeds.[41] It is also likely that smoking by drivers in motor vehicles contributes to the higher incidence of crashes in smokers through the effects of CO poisoning.[42]

Occupational CO poisoning

Unintentional CO poisoning has been identified as occurring commonly from car exhaust and fires, and in the home from faulty cooking and heating appliances. However, many occupational groups may also be at risk from exposure to CO. Those studied to date include fire fighters,[43] motor vehicle examiners,[44] charcoal grillers,[20] bus drivers,[40] manufacturing workers,[45] blast furnace workers,[46] and workers using propane-fuelled forklifts in warehouses.[47]

A French study[48] showed that CO poisoning was common in poorly ventilated automobile garages. Pedestrians and workers in city streets may also be at risk, as levels ranging from 10-50 ppm have been found in ordinary city streets in a large urban area. Much higher levels have been recorded in poorly ventilated underpasses and underground carparks.[49]

Discussion

Studies of CO poisoning due to charcoal barbecues

The literature review identified a number of case reports of small numbers of patients with similar poisonings to those reported here. Most involved patients of ethnic background using the barbecue indoors in winter. The paper by Hampson et al.[12] confirmed these observations. It suggested that CO poisoning by indoor use of charcoal barbecues is an important public health problem in industrialised countries with cold climates. Although the cases presented here did not occur during power failures, Hampson's large series contained many such cases, suggesting that public warnings at such times may be appropriate.

In contrast to the reported literature, the COHb levels in the cases reported here were low, and not all of the patients may have been treated under criteria adopted in other studies. The indication for treatment with hyperbaric oxygen for most of the patients reported here was loss of consciousness, although the father and 10 year old boy from family A and the son from family B did not lose consciousness. Nevertheless it was felt that all had suffered a similar exposure to the majority who did lose

consciousness, and warranted treatment at the same time, particularly as this did not entail additional cost. Given that four of the six members of family A lost consciousness despite COHb levels of 4% or less, it may be appropriate to re-examine the COHb level criterion of 25% on which treatment has been based in the absence of other indications.[12] This is important in cases such as these with prolonged exposure to comparatively low levels of CO, in which neurological damage is more likely.[50]

Recognition of unintentional CO poisoning

A mounting body of literature suggests that the problem of CO poisoning from accidental exposure is common and frequently unrecognised. Heckerling assessed 37 patients attending an emergency department in winter with a complaint of headache.[51] Seven (18.9%) had COHb levels greater than 10%. When further information was obtained or gas company officials investigated their homes, six of the seven were found to have suffered toxic CO exposure. It was noted that three had cohabitants at home who also had headache. Most importantly, the diagnosis of CO poisoning had not been suspected by treating doctors in any of the cases. Heckerling and colleagues[52] later derived a model for predicting whether a patient's symptoms were due to CO poisoning and showed that the presence of similarly affected cohabitants was the most reliable method. The model was then validated prospectively.[53]

Dolan and colleagues[54] studied all patients attending an emergency department with flu-like symptoms and found that 23.6% of such patients had COHb levels of 10% or more. Again none of the patients had been diagnosed as having CO poisoning.

The pathological effects of chronic exposure to low-dose CO

The literature review has revealed that both accidental CO poisoning occurs commonly. Clearly this may be an acute, emergent problem, as in this and other case reports, or a chronic problem, particularly in occupational exposures.

The effects of acute exposure to substantial levels of CO are well described in the medical literature and have been comprehensively reviewed by Mark.[1] Clinically the acute effects are principally on the nervous and cardiovascular systems with delayed neurological sequelae in up to 40% of untreated patients.[55]

However, prolonged exposure to low doses of CO may also be hazardous and may present a significant problem in terms of overall incidence as identified in the literature review.

ANIMAL DATA

A considerable body of experimental work suggests that exposure of animals to CO at low doses for long periods may cause pathological changes in many tissues.

A variety of changes in several animals has been noted, on birthweights[56] and behavioural development of animals exposed in the prenatal period, and on the cardiovascular, respiratory, hepatobiliary, nervous, endocrine and haematopoietic systems of young animals chronically exposed to CO. Additionally, higher stillbirth rates in herds of pigs exposed to CO from faulty gas heaters and inadequate ventilation have been reported.[57]

Piglets exposed to 200 to 250 ppm of CO have been reported to have developmental delays and to perform poorly in behaviour tests.[58] Those exposed to CO prenatally have had low haemoglobin levels at birth. Mice exposed to CO in utero have also been shown to perform poorly at tasks involving learning.[59]

Chronic low-dose exposure to CO in laboratory rats has produced myocardial hypertrophy,[60-62] altered coronary vessel growth,[63] altered cardiac adaptation to aortic constriction,[64] accelerated development of hypertension[65] and pulmonary hypertrophy.[62] Aortic intimal injury has occurred in rabbits.[66]

Cardiomegaly, splenomegaly, and elevated haemoglobin and haematocrit have been demonstrated in rats.[65] Increased numbers of erythrocytes cause increased blood volume.[67] Platelet count is altered in rabbits chronically exposed to low-dose CO,[68] and platelet aggregation increased in pigs.[69] CO exposure has been shown to enhance liver cell necrosis in rats with ethanol-induced liver damage.[70]

Guinea pigs exposed to 200 ppm of CO prenatally and as infants exhibited changes in QRS vector loops implying myocardial damage.[71] Prenatal exposure to low doses of CO (75-150 ppm) caused changes in peripheral nervous system activity in rats, some of which were irreversible.[72] Similar central effects have been observed in rats exposed in utero to levels up to 300 ppm, with disruption of neostriatal development.[73] It has also been demonstrated that CO exposure in rats caused negative effects on evoked potentials in a complex discrimination learning experiment.[74] Low-dose chronic exposure to CO produced a mild stress reaction in rats, with elevated catecholamine levels[75] and increased serum steroid and brain serotonin levels.[76]

The effect of combined CO exposure and high altitude has also been investigated. It has been shown that chronic exposure to CO at 35 ppm when combined with high altitude produced no additional cardiovascular effects

in rats over and above the two insults singly.[77] However at 500 ppm, there was a significant interactive effect on mean electrical axis in rats, as well as on haematocrit.[78]

HUMAN DATA

Translating such animal data to humans is difficult, and there is little published research on such effects.

Mortality rates from atherosclerotic heart disease were shown to be higher by a factor of 1.35 in New York bridge and tunnel workers chronically exposed to CO compared with a standardised comparison group not similarly exposed.[79] Toxic effects of CO on humans have also been demonstrated in those with compromised coronary or peripheral circulation.[80] It has been reported that two workers with coronary artery disease died following occupational exposure to CO, as a result of exacerbation of their pre-existing heart disease.[81] The cardiovascular endurance of a group of Canadian fire-fighters has been shown to be lower than expected, and it was suggested that this might have resulted from chronic exposure to CO.[82]

A historical prospective cohort study of mortality among motor vehicle examiners in the United States was undertaken by the National Institute for Occupational Health and Safety.[44] Such workers were estimated to be exposed chronically to a time-weighted average CO concentration of 10-24 ppm. The only significant finding was that cancer deaths (all types) were increased in these people. The significance of this finding is uncertain.

However, two major reviews of the cardiovascular effects of chronic exposure to CO have not provided conclusive evidence on the case for a direct cardiotoxic effect.[83,84]

There is little else reported in the literature regarding the effects of occupational CO exposure although many papers have described the effects of pollutants attributed to smoking, of which CO is one, both in animals[85] and in humans.[86-90]

Despite the paucity of direct evidence that chronic low-dose CO poisoning produces tissue damage in humans, the animal data strongly suggest that such effects are likely. This accords well with the pathophysiology of CO poisoning, with long term exposure causing more CO to be bound to mitochondrial enzymes[91-94] in the presence of comparatively low COHb levels.[95]

The toxic effects of chronic CO exposure are likely to be greater in smokers, who have been shown to have higher CO levels than non-smokers and to have occupationally-induced elevations of those levels above those currently considered safe.[96,97]

Conclusions

CO poisoning is common. Although the majority of such poisonings result from deliberate self-harm, a significant proportion is unintentional. Most of the unintentional poisonings are due to motor vehicle exhaust, fires, and home appliances used for cooking or heating. Additionally, there is likely to be a number of workers exposed to CO in the course of their employment. While the effects of this long-term exposure are as yet unknown, animal and limited human data suggest a number of pathological changes, particularly to the cardiovascular system. This area requires further research in humans.

The reported literature suggests that unintentional CO poisoning is significantly underdiagnosed. Emergency department medical and nursing staff, particularly the triage nurse,[98] need to be vigilant for CO poisoning which can result in very subtle presentations. Suspicion should be high during cold, inclement weather and in those whose occupations may expose them to significant concentrations of CO.

People in certain occupations, such as mechanics in garages, may be at risk, especially if they smoke cigarettes. Others at risk are those with faulty home heating appliances and those using equipment not designed for heating, such as outdoor barbecues, for that purpose. As identified in the literature review and highlighted by the cases reported here, migrants may be at particular risk in this regard. This was recognised by the California Department of Health Services when it issued a public health warning in 1990 concerning the indoor use of charcoal.[99] Asian immigrants were especially targeted. With the progressive development of a multicultural society in Australia, it may be prudent for public health authorities to consider warning newly arrived migrants of some of the dangers associated with activities which may be acceptable in their countries of origin.

In many developing countries, the same implements are often used for cooking and heating, and are used indoors. This is inappropriate when the materials used, such as barbecue charcoal beads, combust slowly and relatively inefficiently, producing large amounts of CO. Chen et al have warned that several hundred million people in developing countries are at risk of poisoning with CO and other products of combustion from indoor heating and cooking.[100]

It has been noted that public health interventions can drastically reduce the mortality from CO poisoning. The Office of the Medical Examiner of New York, for instance, applied stringent inspection rules for gas refrigerators, reducing the number of accidental deaths from this form of poisoning from 168 in 1951 to two in 1964.[14] Although CO poisoning from charcoal barbecues is uncommon in Australia, there is a strong case for warn-

ings about indoor use to be carried on such barbecues as well as the charcoal beads, preferably in several languages.

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THE WORLD AS IT IS

UNUSUAL DECOMPRESSION ILLNESS
A case report

Peter Cardon

A 37 year old male student diver came to me on a Tuesday, referred by his diving instructor. On the Saturday and Sunday he had been training for an Open Water certificate. He took part in a pool session on Saturday morning and an open water dive to 15 m for 40 minutes in the afternoon. At the completion of the day's diving he was tired and had a mild headache but was otherwise well.

The following morning he took part in a further open water session and after some difficulty with weights he undertook a "tired diver" tow, an emergency ascent from about 6-10 m and an ascent using a secondary air source. The remainder of the dive was given to a successful hunt for crayfish.

The dive plan was for 40 minutes to a maximum depth of 15 m, however his depth gauge showed the maximum depth for the dive was 29 m. He had been working against some surge and was a bit cold toward the end of the dive. He made a comfortable ascent up an anchor warp and felt normal on arrival at the surface.

Within one minute of surfacing and releasing the warp to make his entry onto the dive boat he found his hands would not work for him. He had difficulty hanging onto the boat and managing his gear. Eventually he was helped aboard and fell into the boat where he lay helpless and vague. He described being unable to organise himself, he could not get his gloves off and his hands were clawed. He felt uncoordinated with "spasticity" of his limbs and an unpleasant feeling of entrapment in his wetsuit which verged on panic. He became aware of other divers in the water but his perception was impaired and he was troubled by glare. He felt numbness in his arms and chest. His distress was seen by another diver and the dive instructor was called.

A distinct improvement occurred over a few minutes after removal of the student's hood, however he had difficulty with speaking and complained that his legs felt heavy. It took about ten minutes to take the boat to the beach and by then he had recovered so much that he was able to hold the boat while gear was unloaded. However, ten minutes later he had nausea and vomiting so that the car had to be stopped twice during the half hour drive to town.

When he got home he went to bed. He got up once two hours later to vomit then felt better but had a frontal headache which lasted three hours more. No medication was taken. The following day (Monday) he was aware of slight fogging of his peripheral vision, a mild impairment of mental focus and his ears felt strange.

He had had a full medical for a Pilot's Licence in April 1993 and had been found to be quite fit.

On examination on Tuesday he gave a clear description of the events. He moved normally with no apparent impairment of gait, balance or co-ordination. Basic psychometrics showed some difficulty with simple arithmetic but a good performance with immediate recall, including having no difficulty with the Babcock sentence. A quite detailed neurological examination was within normal clinical limits.

Had I been able to find even a minor neurological abnormality he would have been sent off to Christchurch. However I felt that, as he was now symptom free and normal neurologically, there was a reasonable case to be made for resting and observing him over the next few days, as referral to the nearest Hyperbaric Medicine Unit would have involved many hours of travel and considerable disruption to his life. In the event he produced no sequelae.

At follow-up two months later the patient was well with no residual symptoms and stated that he had taken the personal decision not to dive again.

My interpretation of this history is that this student diver suffered an acute form of decompression illness, possibly cerebral arterial gas embolism, with significant transient cerebral ischaemia and no measurable residual impairment.

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FITNESS TO PARTICIPATE IN ADVENTURE ACTIVITIES: MEDICAL AND LEGAL CONSIDERATIONS ARISING FROM RECREATIONAL SCUBA DIVING

Jeffrey Wilks and Trevor Atherton

Introduction

Adventure tourism activities are becoming increasingly popular with international visitors to Australia, as well as for domestic travellers within the country.[1] The 1992 International Visitors Survey, for example, showed that among the most popular activities for overseas tourists were surfing, scuba diving, snorkelling, bushwalking, sailing, rock climbing, fishing and horse riding.[2]

Some of these activities are traditionally categorised as high risk and are considered by sporting organisations as requiring specific safety guidelines and precautions.[3] Unfortunately, very little information is available to show how safe these activities are, or to clarify where risk management might be improved. Australian studies of sporting injuries tend to concentrate on organised team sports,[4,5] or to report the types of injuries treated at metropolitan hospitals.[6] Since these hospitals are usually not in catchment areas where adventure tourists are likely to be treated, their injury profiles generally do not include adventure activities.

One of the most pressing issues for the adventure tourism industry is that of determining candidates fitness to participate in the various activities offered. Scuba diving is one of the few activities in Australia where a medical questionnaire is used to screen people wishing to try diving for the first time (for introductory or resort diving),[7,8] and a full medical examination is required if a person wishes to be trained as a certified diver.[9,10]

The debate about what constitutes an adequate medical examination for divers, and the appropriate training and qualifications for the medical practitioner conducting the examination, has received considerable attention in this journal.[11-18] The debate has generated a medical information base in recreational diving that surpasses any of the other adventure tourism activities. Even so, accurate figures on the number of active divers are still difficult to obtain,[19,20] and medical conditions continue to be a major contributing factor in morbidity and mortality. For example, from a review of 100 consecutive diving fatalities in Australia and New Zealand during the past decade, Edmonds and Walker report that in 25% of the cases there were pre-existing medical contraindications to scuba diving.[21]

In addition to medical considerations, there are legal issues such as professional negligence and statutory "duties of care" to be considered. On the one hand, there are the legal problems for a medical practitioner who certifies a person fit to dive if later it is found that a medical problem which should have been detected was the cause of an injury.[22] On the other hand, a dive instructor or dive supervisor may be caught up in legal proceedings if a client is injured as a result of a medical condition that would normally have excluded them from participating in diving activities. This is the major concern raised with the use of questionnaires as a medical screening measure, since it is well known that some enthusiastic candidates will conceal conditions (such as asthma) that would preclude them from diving.[14]

Available figures for scuba diving suggest that the relative injury rate is very low.[23] This type of information has given underwriters the confidence to make international travel insurance available for diving under certain conditions, while other adventure activities such as mountaineering and polo are specifically excluded from most travel insurance policies.

Two issues have the potential to jeopardise the availability of current travel insurance policies for diving, and create legal problems for tourism operators. The first is the fact that a diving certification (C-card) does not expire, even if the holder does not participate in the sport for years. Rusty and inexperienced licensed divers may therefore be at increased risk for injuries.[24] Secondly, no regular medical examination is required after obtaining an initial diving certification. This means that many licensed divers, over a period of time, may no longer be fit to dive. In one study of Japanese diving accidents, Mano and Shibayama found that poor physical condition was the cause of 4.9% of the diving fatalities.[25] The following analysis of visitors to one island resort demonstrates the importance of considering these two issues.

Heron Island diver profile

Heron Island on the Great Barrier Reef is well known as an international diving destination.[26] All diving guests visiting the island are routinely asked to complete a diving registration form, which is then used by staff as a source of information for dive planning. Profiles generated from a comprehensive review of these record forms have recently been published.[27]

To gain an insight into visitors fitness to dive, a secondary analysis of 2,577 record forms from 1991 was conducted. Of particular interest were the number of dives in the previous 12 months, and the time since the last medical examination. The forms ask about the last medical examination and not whether this examination was specifically for diving. The information is therefore relevant as an indication of the time between medicals for participants in a range of adventure tourism activities.

TABLE 1

HERON ISLAND DIVER PROFILE

Divers from	Queensland	NSW	Victoria	Other Australians	USA	Europe	Other Countries
Average time since last dive (months)	9.0	8.7	12.4	9.9	13.3	8.8	9.5
Number of dives in previous 12 months (%)							
None	24	23	26	27	33	25	27
1-10	35	40	51	34	42	40	40
11-20	18	16	16	17	15	17	18
> 21	23	21	7	22	11	17	15
Months since last medical examination (%)							
Less than 12	52	53	58	49	77	70	67
13-24	26	23	21	18	15	19	19
25-36	12	8	10	14	4	7	8
37-48	3	7	2	10	2	1	3
49-60	4	2	2	7	1	1	1
> 61 months	3	7	7	2	2	2	3
Average time since last medical examination (months)							
	17.6	19.8	19.9	20.1	10.7	11.1	13.1
Number in each group	348	306	100	88	444	204	186

In order to exclude island visitors who were recently certified and may have had a medical as part of their entry level diver program, only divers who had been certified for more than 12 months were included in the sample (N= 1676). Many countries do not require candidates to undertake a medical examination before commencing an open water scuba program, relying instead on a medical questionnaire as a screening measure.[28] However, since medical examinations are required in Australia,[9] and information on place of initial training was not available, a conservative approach was adopted.

Table 1 reveals that approximately one quarter of the sample had not dived in the previous 12 months, while a further third had participated in 10 dives or less in the previous year. Average time since the last dive ranged from 8 to 13 months, suggesting that most visitors do not dive between their annual holidays.

While at least half the visitors in each group reported having a medical examination in the previous year, a substantial proportion had not had an examination for some considerable time. Average time since the last medical ranged from 10 to 20 months.

Implications of these findings

As noted by Edmonds and Walker, many diving accidents are the direct result of medical complications.[21] Having received their initial open water certification card, there is currently no formal requirement for divers to undertake an annual medical examination to confirm their continuing fitness to dive. Health circumstances may change adversely over the years, and with a licence that does not expire some divers may be at risk without a regular medical examination.

A majority of the visitors to Heron Island are specifically seeking a diving holiday, and have excellent staff and facilities available to them. Nonetheless, the substantial proportion who have not had a medical in the past year is suggestive of the large numbers of general tourists taking adventure tourism activities where fitness to participate could be an issue.

Many tourist operators use Liability Release forms in an attempt to exclude or limit their legal liability should an accident occur. These forms often include some statement that the activity contains some degree of risk, and that the candidate should be free from medical

problems before participating. It appears that this practice has been adopted from the United States. There, properly constructed, clearly worded, and signed by the candidate, these forms will often provide a useful defence against claims under certain circumstances.[29]

However, in Australia the obligations cast upon the operator are more onerous. Here disclaimers will generally fail if there is proof that the tourist operator, their staff or agents have failed to meet their statutory "duties of care" to customers. These duties include informing the client of the very real risks of participating in the particular adventure activity. In terms of Workplace Health and Safety legislation, especially in Queensland,[30] this may include a duty to advise customers of the known risks for diving if they are obese, tired, anxious, have been drinking alcohol, or demonstrate any of the identified predisposing factors associated with decompression illness.[31] Any false, misleading or deceptive conduct on the part of the service provider may also lead to a claim for damages under the Commonwealth Trade Practices Act 1974 (ss 52, 53, 55A),[32] or the equivalent sections of the States' Fair Trading Acts.

While this may seem like an onerous burden for a dive operator, much of the problem stems from the two issues of a licence that does not expire and no formal requirement to remain medically fit to dive.

The importance of providing adequate health information for tourists has recently been highlighted in a European Economic Community Directive that health warnings be included in all tourist brochures.[33] While this directive is not binding in Australian law, it nevertheless an indication of the growing attention given to health issues. For adventure tourism operators the best advice is to screen all potential clients before offering services. This may mean that some people may have to be excluded from participating, but it will also avoid unwanted medical and legal complications for both the operator and the customer.

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SUPPOSE THE THREE WISE MONKEYS FACED A CLASS ACTION

A product-liability scenario

Douglas Walker

There is a general perception that those who control the policy directions of the recreational diving organisations live in hope that, if they say and do nothing to draw attention to the misadventures which inevitably occur to divers, they will escape notice and censure when a major accident occurs. Strangely none of their Insurance companies appear to have drawn their attention to the very real dangers of such a policy. The "Three Wise Monkeys" response is fraught with danger to all who seek to follow it. This stems from the product liability aspect of business which holds that a product should be suitable for its intended use and that every care has been taken to discover and remedy faults. Those who have had to appear before a Coroner after the death of a pupil, or of a diver in a group where they were present, will be painfully aware of the interest taken in examining the training and actions of not only the victim and the dive organiser but also the protocols of the parent organisation. Such are not held to be sacrosanct or safe from severe criticism, and liability suits can feed on such a rich diet. So let us consider the "monkeys" one by one.

The term "evil" will be used throughout because the reporting of problems, even those which have been efficiently managed, has long been regarded as both dangerous to the person making the report and lacking real importance "because everything is already known" about the problems affecting divers. In consequence a report is made only when the reporter thinks that a liability claim may be possible and that the Insurance company will ask whether a report was filed. The attitude of the diving organisations has reinforced this view as they often show no response to the reports they do receive. They appear to neither commend good reports nor request more details where the reports are inadequate. My attempts to obtain their active involvement in research into specific problems have failed because the value of the information has not been recognised and there has been a prejudice against asking for information which may not be to the liking of the organisations. Governments avoid this dilemma by careful choice of the chairman and members of any investigatory committee they set up. The diving organisations can avoid being directly identified with complaints and suggestions for changes in diver training and dive management by supporting surveys by those bound by codes of confidentiality.

The "Hear no evil" monkey is the Pontius Pilate option whereby no responsibility is accepted, an avoidance of any attempt to improve safety by taking notice of problems talked about but not formally recorded, an acceptance of "misadventures" because there has been no serious morbidity, in consequence a failure of any alerting of the generality of divers to observed problems which should be receiving attention before serious consequences occur. If problems continue to be accepted and tolerated (not "heard") they will increase and one day reach a critical level.

To take the next monkey "See no evil". If an organisation has a product which during normal and intended use is associated with injury to the user a tort has been committed. There is an implied warranty that the

product is fit for its purpose. In the context of recreational diving there is the clear implication that the purchase of a course of instruction, or a dive supervised by a person holding appropriate qualifications, will ensure that the purchaser does not suffer from exposure to any of the predictable dangers common to the diving environment. Diving training is given only by those certified as qualified to provide such instruction, and trainees certificated at the conclusion of all courses will assume they have a specified level of skill and an understanding of their limitations. Such is a reasonable even if unstated expectation of those learning to dive.

So it is essential that courses are constantly updated to ensure they maintain the maximum relevance to the conditions and problems which occur, that they incorporate the lessons learnt from an analysis of the results of "quality control", which in the case of diving is represented by monitoring the types and severity of diver morbidity. If problems are not noted, discussed and analysed by those immediately involved, both at instructor and organisational level, they will occur again and again within the diving community until, inevitably, one day there will be a cascade effect and more serious morbidity or even a death will occur. If problems are not "seen" there will be no reason to take any action.

The injunction to "Speak no evil" is a child of the "don't dob in" philosophy, which ensures a persistence of unsafe practices and training inadequate to meet the situations the diver is likely to experience. If problems are not reported by those involved or observing them, either by deliberate refraining from reporting for fear of criticism or from a belief that the problem is too common to bother about (or far too obvious not to be known), the person with the information must share some of the responsibility for failure to update the course. Project Stickybeak, a method for the confidential reporting of diving-related problems, has existed for decades. The Diving Incidents Monitoring Study (DIMS) has been running for a few years. There is no reason for anyone to fail to assist in improving diving safety by reporting what they hear or see even when the exact details of the problem are uncertain.

The law assumes that anyone with special skills should employ them when appropriate, even when they are not immediately contractually involved. Taking notice of, and then reporting, all matters which detract from the safety of divers to whom they owe any duty of care could be considered appropriate. An instructor could be expected to correct dangerous errors observed in a diver or dive group even where the person(s) involved were not employing his or her services. An instructor could be expected to provide assistance in any situation where their training has particularly qualified them.

Failure to seek out problems and dangers can no longer be treated as an acceptable option, or one without potential for expensive and painful proceedings in a Court of Law. If one can demonstrate an active pursuit of information, and its use towards reducing identified problems, a better defence can be made, to claims that a diver was inadequately trained or warned or supervised, if all those involved had followed a course of action based on such an information.

This is a plea for the recognition by diving organisations and individual divers of the need for, and value of, details of all types and degrees of diving-related problems. There is a particular need for reports of effectively managed problems and of occasions where early intervention prevented a potentially adverse situation developing. It is time to recognise the moral (and legal) necessity for divers becoming involved in the collection of data on all types and severities of diving problems, particularly those where the response was effective and the dive was able to continue without further problems. Remember two things, reports are treated as being medically confidential, and the life you save could be your own.

Dr Douglas Walker is the founder of Project Stickybeak, from which the Provisional reports on Australian diving-related deaths, which appeare regularly in the Journal, are compiled. His address is P.O.Box 120, Narrabeen, New South Wales 2101, Australia.

SPUMS NOTICES

REVISION OF THE SPUMS POLICY ON EMERGENCY ASCENT TRAINING

The Society's policy on emergency ascent training (EAT) was published in the Journal in 1993.[1] One of the features of that policy was that buddy breathing was considered to be suspect and to warrant ongoing review. This review is now complete and, especially in the context of the Diving Incident Monitoring Study data that Dr Chris Acott is to present to the 1994 Annual Scientific Meeting of the European Undersea and Biomedical Society, the advocacy or teaching of buddy breathing can no longer be supported, with or without reservations. It follows that the Society's policy

on emergency ascent training should be amended to include the following statement *in-lieu* of the original statement about buddy breathing.

Buddy breathing as a form of emergency ascent appears to cause an unacceptable level of risk to participants in either training or an actual emergency. Consequently, the Society advises divers to discontinue buddy breathing and instead practice less risky out-of-air procedures such as the use of alternative air sources. The Society believes that buddy breathing should neither be taught nor practised.

Des Gorman and Drew Richardson
Co-Chairmen
Workshop on Emergency Ascent Training 1993

Reference

1 SPUMS Policy on emergency ascent training. *SPUMS J* 1993; 23 (4):239

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.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING

held on Saturday 22/4/94 in Sydney

Present

Drs A Slark (Past President), S Paton (Treasurer), C Meehan (Secretary), J Knight (Editor), D Davies (Education Officer), C Acott and G Williams.

Apologies

Drs D Gorman (President) and J Williamson.

1 Minutes of the previous meeting

Accepted as a true record, after minor corrections. Proposed by Dr Davies and seconded by Dr Williams

2 Business arising from the minutes

2.1 Dr N McIver is not able to attend the 1995 ASM, but would be available for 1996.

3 Rabaul ASM

3.1 Purchase of Safety Sausages and DCIEM tables has been finalised.

3.2 Official immunisation recommendations have been provided to Allways.

3.3 The provision of oxygen by CIG will cost $2000, to come out of registration fees. The first aid kits taken to Rabaul will be donated to the local hospitals.

4 Tender for 1995 ASM

4.1 Tenders submitted by Allways Dive Expeditions and Dive Adventures were discussed. Both tenderers were available for questioning. A vote was taken and Dive Adventures tender was accepted. It was decided to announce the decision after the ASM on 1/6/94.

4.2 Timing for calling of and deciding future tenders will be discussed in the future. The tender for the 1996 ASM in Fiji is to be opened shortly.

5 Treasurer's Report

That the Treasurer be congratulated on the management of the assets of the Society. Proposed by Dr Knight and seconded by Dr Meehan.

7 Other Business

7.1 The value of face to face committee meetings was discussed. As most of the committee will be in Cairns in October for the Safe Limits symposium, it was suggested that there should be a committee meeting during the symposium.

7.2 After Dr Knight had left for Melbourne there was discussion about the possibility of an honorarium for Dr Knight if he continues as editor.

7.3 It was pointed out that extra support is needed for the Treasurer and the Secretary because of the work load of routine tasks, which could easily be delegated, involved in these positions.

SPUMS ANNUAL SCIENTIFIC MEETING AND ANNUAL GENERAL MEETING 1995

Castaway Island, Fiji.
Sunday 21/5/95 to Sunday 28/5/95

The Guest Speaker is to be Dr A A (Fred) Bove, Chief of Cardiology at Temple University in Philadelphia. He was the Guest Speaker at Madang in 1982.

The Convener of the ASM is Dr David Davies, Education Officer of SPUMS. The theme of the meeting is Fitness to Dive. The Workshop theme is Asthma.

Those wishing to present papers are asked to contact Dr Davies at Suite 6, Killowen House, St Anne's Hospital, Ellesmere Road, Mt Lawley, Western Australia 6050, as soon as possible.

The same applies to those wishing to contribute to the Workshop on Asthma, but who will be unable to attend the meeting. Dr Davies intends to prepare their written submissions to distribute to those attending the meeting. This means that such contributions will need to be in his hands by the middle of April 1995.

Intending speakers are reminded that it is SPUMS policy that speakers at the ASM must provide the Convener with the text of their paper, ready for publication, before they speak.

The Offical Travel Agent for the meeting is Allways Dive Expeditions, 168 High Street, Ashburton, Victoria 3147, Australia. Telephone (03) 885 8863, Toll Free1-800- 338-239, Fax (03) 885 1164.

SAFE LIMITS

AN INTERNATIONAL DIVING HEALTH AND SAFETY SYMPOSIUM

to be held at the
Radisson Plaza Hotel
Pierpoint Road,
Cairns
Queensland 4870
Australia
from
October 21st to 23rd 1994

The symposium is being organised by
The Queensland Government
Division of Workplace Health and Safety
Department of Employment, Vocational Education,
Training and Industrial Relations
with
Dive Queensland
and
The Queensland Tourist and Travel Corporation.

The symposium is endorsed by
SPUMS
The South Pacific Underwater Medicine Society

Registration fee $Aust 350.00

For further details, program and registration form contact
Total Control Conference Management
P.O. Box 101, Burleigh Heads
Queensland 4200, Australia.
Phone (075) 766-388
Toll Free 1-800-036-105
Fax (075) 200-275

LETTERS TO THE EDITOR

DIVING AFTER ROUND WINDOW RUPTURE AND REPAIR

182 North Kiama Drive
Kiama Downs
NSW 2533

17/7/94

Dear Editor,

I have been a keen diver for the past 25 years. About 18 months ago I ruptured the round window in my ear while snorkel diving. I had an operation which involved a full graft as the whole of the round window was blown out. My hearing was restored almost back to normal, but I was left with tinnitus and of course the doctor said that I should not dive again.

I have heard of divers suffering this injury and being told not to dive again, but they have ignored medical advice and continued diving after a healing period. I now know personally two divers who have had a round window rupture and an operation to repair it. They have stayed out of the water for six months and then returned to diving, without any problems, against their doctors' advice.

I would like to do the same but I do not have the courage to give it a go. I am writing to find out if SPUMS has carried out any research on this subject or is aware of any research that has been done and if you could advise me on how to go about obtaining it.

Anxiously awaiting your reply.

Peter Fitzpatrick

The Editor's reply to this letter appears below.

26/7/94

Thank you for your interesting letter which arrived today. I would like to publish it in the SPUMS Journal as a Letter to the Editor as it an important topic, and one that is seldom discussed.

Unfortunately I do not know of any studies about divers who have gone back to diving after rupturing an inner ear window. Like you, I know of two who did, but one of them ruptured the same window again and was very lucky to get his hearing back the second time. He decided that the risk of permanent loss of hearing was too great for him to accept and gave up diving. The other I saw when he had the second ear's ruptured window repaired. He told the surgeon that the only thing that made his very busy life worth living was diving every weekend. He decided that he would rather be completely deaf rather than not dive.

The reason that divers are advised not to dive after a round window rupture repair is that there is a fairly high risk of repeating the accident unless one changes one's diving technique and makes quite certain that the pressure in the middle ear remains at ambient (surrounding) pressure at all times. Doctors feel that risking total deafness is not sensible behaviour and should not be encouraged.

From your story it is likely that you did not equalise properly on the way down and that as a result your eardrum was pushed in too far, pushing the stapes far into the inner ear and the round window out. The other common cause is trying too hard to equalise and forcing extra fluid into the inner ear, from the inside of the skull, so blowing out the round window.

In either case the repaired window membrane is likely to be weaker than the original so more likely to be damaged if equalisation fails.

The decision to dive is for the diver to make. The doctor's advice is based on the considerable social handicap of complete deafness. The chances of getting one's hearing back the second time in the same ear are very low. Someone who is completely deaf in one ear is able to hear with the good one, but if that goes he is completely cut off from conversation, unless it is in deaf and dumb sign language.

To put the decision in perspective, if I had a round window rupture, and got my hearing back, I would prefer to live out my days with that hearing rather than risk losing it. I would sell my gear. But what you do depends on how much your hearing means to you and only you can decide that.

I would be most interested to hear from your acquaintances who have gone back to diving to learn more about how they did the damage and why they decided to go back to diving. It is only by hearing from people that we can find out what you want to know.

I hope that my explanations have been of some use to you.

John Knight

The Editor is willing to act as a collector of information until some member, perhaps a diving ENT surgeon, comes forward to carry out a study of divers who have gone back to diving after having an inner ear window repair.

BOOK REVIEWS

A CODE OF GOOD WORKING PRACTICE FOR THE OPERATION AND STAFFING OF HYPERBARIC CHAMBERS FOR THERAPEUTIC PURPOSES.
Faculty of Occupational Medicine of the Royal College of Physicians of London
May 1994
Price £10.00. Postage and packing extra.

The Faculty of Occupational Medicine of the Royal College of Physicians of London believe that it is opportune to publish this Report in view of the re-emergence in the United Kingdom of some interest in hyperbaric treatment. During a recent visit, I was fortunate to meet the chairman of the British Association of Hyperbaric Medicine Units, who tells me there are now 28 facilities which can provide emergency treatment in the United Kingdom. Only a relatively small number currently carry out clinical treatment other than of divers, but the number of active chambers is encouraging. There has been little interest in hyperbaric medicine in the United Kingdom for some years.

The report is issued at a time when there is world wide interest in rewriting of standards for hyperbaric care. The Australian Standards Association is revising Australian Standard No. AS CA12-1970 (SAA Compressed Air Code). Documents to be used will include this report, the original standard, and the US Undersea and Hyperbaric Medicine Society report on Clinical Multiplace Hyperbaric Chambers, which is about to be published.

The Faculty of Occupational Medicine Report has an introduction which lays out the reasons for the report. It then considers patient and chamber matching criteria, and considers chambers in 4 categories.

Category 1 are capable of receiving patients in any category and can provide advanced life support.

Category 2 can treat all patients, but not provide advanced life support during Hyperbaric treatment.

Category 3 receive emergency referrals of divers and compressed air tunnel workers.

Category 4 is for monoplace chambers, for treating patients not likely to require any access during hyperbaric treatment.

There are sections on hyperbaric facility buildings, and chamber construction, medical equipment and facilities. Staffing requirements are considered in detail and the responsibilities of the various personnel are outlined. There is a section on training, and the requirement for standard operating and emergency procedures. There is also a note on documentation and relevant legislation.

The report will be of considerable value to those in the United Kingdom who are seeking guidance for new or reactivated facilities. It is of interest to those in Australia who are currently redrawing the Australian Standard, though the level of information is somewhat general.

The Faculty should be commended for producing this little book, and at its published price of ten pounds Sterling it may be a useful document to be available in Hyperbaric Unit libraries.

Harry Oxer
Director Hyperbaric Unit
Fremantle Hospital

CAREERS IN DIVING
Steven M.Barsky, Kristine C.Barsky and Ronnie Lynn Damico.
Best Publishing Company, P.O.Box 30100 , Flagstaff, Arizona 86003-0100, USA.
Price from the publishers $US 19.95. Postage and packaging extra.

I have made diving my career and found this book to be full of good advice. Perhaps I should have read it 25 years ago and followed my mother's advice, and got a proper job.

All of the authors have enjoyed a variety of diving occupations during their careers, this seems to be the norm for professional divers. Their personal experiences are complimented by the many other contributors. Underwater Photography, Marine Biologist, SCUBA Instructor, Commercial Diving, Retail Dive Store Operation, Diving Journalism and Marine Archaeology are some of the 14 careers covered in this book.

The format works well of each career being explained, commented on and followed by several short career portraits from practitioners in that field. These career snapshots are of about one page each and constitute a major part of the text, and were well worth their inclusion. A complete chapter is dedicated to each career and includes a suggested reading list. This approach means a career can be appreciated by the reading of the relevant chapter only. However the introduction must be considered as mandatory reading as it gives the philosophy of the authors on how the book should be used.

It is a sign of the times (in the USA anyway) that the first page carries a WARNING, about how potentially hazardous a career in diving may be. Would a book on truck driving contain a disclaimer that you may die as a result of a road accident? Appropriate training, staffing

levels and equipment for the type of diving you are conducting I feel now reduces risk in professional diving to an acceptable level, so why the paranoia?

Each chapter describes the up and down sides (sorry) of that career, there are no punches pulled and it certainly will destroy the romantic view that many may have on a career in diving. How do I get to be a commercial diver? This question is one of the more frequent that I am asked by sport divers and this book will answer the question. The advice given is particularly relevant to North America, especially California, the institutions and qualifications mentioned are all American so the usefulness of this text is slightly diminished outside America.

While remembering that most professionals find it easy to criticise movies or books on their chosen field of expertise, I found some aspects of the advice given in some chapters at conflict with my experience. However, I found the chapters on careers of which I have only a passing knowledge of, informative. So I suspect a bit of bias there.

In summary the book has a easy readable style and achieves its objective of being; a guide to careers in diving. I recommend this book if you can not get a real job, and consider diving as a career.

Bob Ramsay
Senior Hyperbaric Technician
Royal Adelaide Hospital

THE DIVER'S BIBLE
Phil Henderson
Henderson Diving Services, P.O. Box 153, Dunsborough, Western Australia 6281.
Cost $35.00 including postage from the publisher

Nearly all divers start off with their basic scuba manual from the agency that trained them. After the course its use is minimal and almost never required. As recreational divers progress though to becoming an Instructor their reference library has grown to include many useful books on diving and the sea.

Commercial diving tends not to have the resources placed at its disposal to produce glossy “how to” manuals as found in the recreational industry. Commercial divers tend to gather notes and photocopies of formula and procedures, these compliment the textbooks they also acquire during their career. By the time a diver becomes a dive supervisor this tattered collection of reference material has become his Bible, it is a very personal thing, your trade secrets. Your Bible compliments the Company Procedure Manuals that are usually supplied.

Phil Henderson has produced *The Divers Bible*, it is, I suppose, his tatty collection of dog eared notes gathered throughout his career metamorphosed into a “how to” book for commercial divers. Gas mixing, adjusting analysers and if you want to know at what frequency bell sonar works or need a internal bell check list, then this is the book for you. Calculations and notes on rigging, thread sizes, and a plethora of conversion factors, how to transfer an unconscious diver through a deck decompression chamber hub, it is all in this book.

While there are many large format books on the subject of commercial diving this one is different, it is just the facts with little other text to pretty it up. It comes in normal paperback size and 168 pages packed with information. I will add this quick reference book to my library, it is the most comprehensive book I have ever seen of this type.

Its target reader is the offshore commercial diving population but I suggest that the nitrox and technical divers will gain much from owning a copy. I was intrigued by the phrase “Vol of shattering”, it simply turned out to be a typo for shuttering, as in concrete formwork.

As is often the case with many books on diving, it includes a section on diving medical matters that are much better covered by medicos in other texts. The recommendations for flying after diving and return to diving after DCI (7 days after CNS serious symptoms) are not in keeping with current thinking. I also think that a comprehensive index would be useful. These criticisms do not detract from the real value and purpose of the book.

Bob Ramsay
Senior Hyperbaric Technician
Royal Adelaide Hospital

DIVING ABOVE SEA LEVEL
B R Wienke
Best Publishing Company,
P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 9.95. Postage and packing extra.

There is no doubt that the problems of diving above sea level warrant attention, and in that context, this monograph is welcome. Certainly, the author addresses the important issues for the diver embarking on such dives. However, the monograph has some features that prevent it from fulfilling the role for which it was intended.

First, the literary style is such that the book is often difficult to read. Also, the half-referenced format adopted by the author will frustrate many enthusiastic readers.

Second, the monograph is very rich in theory and poor in data. While this is a reflection of the science of decompression theory, the data presented are essentially limited to a selected series of ultrasonic studies and an even more selective presentation of anecdotal clinical data. The

merit of the latter is, at best, questionable. Finally, the author's review of decompression illness (DCI) proposes a 4 type classification, at a time when most practitioners are abandoning such typing (even 2 type classifications), and the author's pathological concept of DCI is simply outdated.

Overall then, the need for a comprehensive review of diving at altitude remains and is not completely satisfied by this monograph.

Des Gorman
Director of Medical Services
Royal New Zealand Navy

SPUMS ANNUAL SCIENTIFIC MEETING 1994

OPENING ADDRESS SPUMS ANNUAL SCIENTIFIC MEETING, RABAUL

Sir Mekere Morauta

Governor of the Reserve Bank of Papua New Guinea
Chairman of Air Nuiguini

Thank you for inviting me to speak to you tonight. I am sure you are all wondering why I, of all people, was asked to speak. I am not a diver. Nor am I a medico; nor one of those brand of people often asked to speak at such functions, the brand known as (quote) "distinguished politicians". My only claim to fame here tonight is having been, many years ago, a friendly cohabiter of Lincoln College, Adelaide, with Doctor Chris Acott, whom you all do know. Some time last year, after a feast of prawns and crab, accompanied by a couple of bottles of wine from our old stamping ground, the Barossa Valley, I was conscripted by Chris to appear before you tonight.

As I said, I am neither a diver nor an administrator of medicine. I am however very familiar with water, muddy swampy water, of the Lakekamu and Tauri river basins in the Papuan Gulf. Apart from breeding mosquitoes, those waters, although highly unattractive for divers, are very rich in seafood, prawns, crabs, lobster, fish, clams and oysters. Which brings me to the theme of the few words about my country I want to share with you tonight.

Papua New Guinea is very fortunate in having a rich resource base, both on land and in seas and rivers. Other Pacific countries also have rich water-based resources, but are less endowed than we with land-based resources. The challenge South Pacific countries face as nations is to identify, plan and invest our resources. It is through this that we increase the value of our resources. The resources on their own are of value to support traditional life styles. But today's generation has different needs and requirements from those traditional needs, different even from the needs and life styles of our parents. Resources are there to be exploited for the benefit of their owners. What the present generation must not forget is that it has an obligation to ensure that future generations are not denied the use and benefit of the resources.

Papua New Guinea has a well developed policy framework for the exploitation of its mineral resources. Policies relating to the exploitation of forest and marine resources however are poorly developed, and generally end up as being reactionary to exploitative developers. Just this morning I was shown a map of PNG carved out into large forestry permit areas to suit large-scale forest exploitation, but we have no plans for replanting, no plans for alternative land-use. The nation's capacity to safeguard the ecology likely to be affected by such exploitation is non-existent. In another ten years, our topography could look like that of Borneo or Madagascar, denuded and red. The effect of erosion and siltage on land and sea resources may be catastrophic.

Governments alone can not fully safeguard the resources of the people. But Governments can help organisations like yours, interested in the simultaneous use and protection of natural resources, ensure that balanced development occurs.

I know Papua New Guinea, like other islands in the Pacific, is surrounded by beautiful, unspoiled reefs, corals and rare species of sea-life. Like our gold, copper, oil, gas, timber, tuna and prawns, the use of these reefs can contribute positively to the nation's development and people's well-being. Is it beyond the capacity of the Pacific nations to get together to develop a common user plan for these underwater resources?

As a "senior" Papua New Guinean, interested in the sustainable development of my country, I welcome the contribution you are making to better and safer use of our underwater resources. Our task is to assist you and use the knowledge that you have in putting together a plan which will ensure beneficial use and conservation of this divers' paradise.

MANAGEMENT OF DIVING ACCIDENTS

Des Gorman

Summary

A generalised approach to diving accident management is presented, emphasising resuscitation and initial management. It is clear that diving accidents should be anticipated and Diving Supervisors must plan accordingly. Divers themselves must be taught and practice relevant diver recovery and resuscitation techniques, and in particular how to administer 100% oxygen.

Introduction

A generalised approach to diving accidents is possible and is presented here. This will emphasise resuscitation and initial management.

Some problems encountered by divers, such as confusion and impaired consciousness, chest pain and shortness of breath and vertigo, often cause problems in differential diagnosis. Other problems are less dramatic, but may nevertheless contribute to diving accidents. These include headaches, gastro-oesophageal reflux, sensory deprivation, disorientation and claustrophobia. Headaches are frequent and may be due to anxiety, salt water aspiration, cervical strain, cold, barotrauma, ill-fitting masks or helmets, migraines, hypercarbia, the decompression illnesses or may be coincidental.[1] Sensory deprivation and disorientation have caused diving fatalities, especially in caves.[2]

Analyses of fatal[3,4] and non-fatal accidents[5,6] produce limited data, and consequently a multi-centre diving incident monitoring study is underway[7] and strongly supported.

Diver rescue

Many injured divers will need rescue and occasionally rescue from underwater. Both scuba and surface supplied (hookah) divers will invariably lose the regulator mouthpiece if they lose consciousness. Before swimming such an unconscious diver to the surface the mouthpiece should be ventilated free of water and placed in the diver's mouth, if possible, so that any inspiration will be of gas. The diver should be brought to the surface with the neck extended to enhance upper airway patency. Gas should not be blown into the unconscious diver during the ascent as this may cause pulmonary barotrauma.[8]

Divers wearing bandmasks and helmets and those using rebreathing sets must have their breathing circuits cleared of existing gases to reduce the concentration of contaminants such as carbon monoxide and carbon dioxide. If the diver has a separate emergency gas supply this should be used as the main gas supply may be polluted. This also explains the need for a rescue diver (standby diver) to have an independent gas source.

FREE-SWIMMING DIVERS

The injured diver must be made buoyant by removing the weight-belt and inflating any buoyancy compensating equipment, providing this does not cause the diver to float face-down. Direct, or snorkel-based, expired-air resuscitation (EAR) is possible on the surface, but only if the rescuer has been trained and has practised, and even then it is still difficult. The rescuer must maintain the diver's airway and return him, or her, as quickly as possible to a boat or shore. In-water EAR is probably only indicated if immediate exit from the water is impossible. External chest-compression cardiac resuscitation (ECC) is impossible in water and should not be attempted.

STAGES AND BELLS

A diver injured working from a stage or bell must be quickly returned to that platform. Access to stages and open-bells (which consist of a platform with a domed air bubble container above) is simple, but decompression to enable further resuscitation may cause a decompression illness and complicate management of the original injury. The injured diver must be fixed in place by their harness, and in an openbell the head and shoulders must be held above-water so that the mask or helmet can be removed.

A closed-bell overcomes the problems of decompression and thermal balance intrinsic to stages and open-bells; however, access to a closed-bell is limited and resuscitation inside the bell is restricted by lack of space. There are two techniques used to recover injured divers into bells. The most popular is to partially flood the bell and float the injured diver in, subsequently expelling the water with compressed gas. The second is for the rescuing diver or bellman to winch the injured diver into the bell. The point of winch fixation is controversial.[9,10] A posterior fixation enables easier winching but the head flexes on the neck and so the upper airway is compromised. A cervical collar can be used but is time-consuming to fit and delays subsequent resuscitation. An anterior fixation often causes the diver's head and body to foul on the bell man-way (entry trunking). Both EAR and ECC are possible in closed-bells, but specific techniques related to the configuration of the bell are necessary.[11]

SATURATION SYSTEMS

Diving accidents, coincident injuries or illnesses such as vestibular decompression illness,[12] water-jet injuries[13] and pseudomonas otitis externa[1] often require medical intervention and a diving physician or medical

technician may have to be compressed to the storage depth of saturated divers. Such interventions are limited by inert gas narcosis (and perhaps the high pressure neurological syndrome) in the medical attendants, difficulties with auscultation, off-gassing in decompressed blood samples, altered drug sensitivities[14] and by decompression illness developing in the attendants during or after their return to the surface. Consequently, medical interventions are usually brief and significant diver injuries or illness may require the dive to be abandoned and decompression to be initiated. The isolation of many saturation diving systems also necessitates adequate local medical supplies.[15] The slow decompressions intrinsic to saturation diving (e.g. 1.5 to 0.5 msw hr^{-1}) causes problems when a diving bell carrying saturated divers is lost or a diving platform has to be abandoned because of flooding, sinking, fire or gas contamination. Successful rescue of these divers will depend upon adequate emergency heating (especially if helium is being breathed), gas supplies, nutrition and fluid supplies for the duration of any subsequent decompression and an alternative decompression venue such as the bell itself or a floating hyperbaric lifeboat.[16]

Divers must learn, and regularly practise, relevant diver recovery and resuscitation techniques. Once the diver is rescued cardiopulmonary resuscitation takes priority, although administration of oxygen, attention to wounds and in particular stopping blood loss, removal of coelenterate tentacles (after deactivation by dousing with vinegar or other appropriate solutions),[17] pressure immobilisation of envenomated limbs and administration of anti-venom,[18] application of hot water to fish stings[18] and ice to traumatic injuries, rewarming, fluid replacement and drug administration can occur concurrently.

Cardiopulmonary resuscitation

Airway management, and if necessary EAR and ECC, must continue until an injured diver arrives at a definitive therapeutic facility and for a hypothermic diver until rewarming has been achieved.[19] Hypothermia is protective to the ischaemic brain and significant recoveries of function can occur despite prolonged apnoea.[20] There is no convincing evidence that ECC should be withheld from a hypothermic diver because of possibly inducing cardiac arrhythmias.[21] Endotracheal intubation and mechanical ventilation may become necessary to correct and avoid hypoxia; ventilation should be adjusted to maintain normoxia and normocarbia as prolonged hypocarbia is not beneficial in brain injuries.[22]

Oxygen administration

Oxygen administration is useful in most diving accidents and, with the exception of oxygen toxicity, is not harmful. Overt pulmonary oxygen toxicity is rarely seen in diving because an inspired oxygen tension of less than 0.6 bar is used in prolonged dives. It is most commonly a complication of hyperbaric oxygen therapy. After an oxygen convulsion, hypoxia is likely in a diver because of airway compromise and/or aspiration of water and vomitus and then oxygen administration may be essential.

Oxygen supports combustion and this is related to its concentration. Consequently, oxygen monitoring is essential in recompression chambers and diving bells and oxygen delivery systems should be free of oil and other combustible lubricants. In general, high pressure oxygen supplies are reduced at their source and piping is limited to stainless steel with a minimum molybdenum content or new alloys such as tungum. First-aid oxygen supplies at a diving site must be adequate to retrieve at least 2 injured divers (20 to 30 litres/min) to a definitive facility. The hyperbaric oxygen therapies commonly used to treat decompression illnesses[23] consume between 5,000 and 10,000 litres STPD of oxygen.

Unless the diver is intubated, administration of 100% oxygen requires the use of either an open-circuit demand system with mouth-piece and nose-clip, or a sealing (anaesthetic-type) mask with high flow rates (at least 10 litres/min) and a reservoir. There is uncertainty about the ideal oxygen circuit to supply to divers and in particular the utility and safety of positive pressure resuscitators (e.g. Robert Shaw) and manual-inflation resuscitation bags. Certainly, such resuscitation equipment make over-inflation and pulmonary barotrauma possible and resuscitation bags require considerable experience to use adequately. It follows that issue of any circuit must be preceded by evaluation[24] and accompanied by appropriate education.

Prolonged administration of oxygen will require interruptions for air breathing (air breaks) to retard pulmonary damage.[25] No breaks are needed for 100% oxygen exposures of less than 4 hours. The breaks increase to 5 minutes after every 25 minutes of oxygen for exposures of between 4 and 12 hours and eventually to alternate hours for exposures of greater than 24 hours. It is important to record both the times of oxygen administration, in order to estimate cumulative oxygen dose and likely respiratory decrement,[26] and the diver's response to oxygen as this may assist in diagnosis.

Posture

The ideal posture for an injured diver is controversial, although some consensus is possible.[5] Van Allen et al. showed that arterial bubbles did not embolise the brain of head-down dogs.[27] These results have been confirmed in both cats[28] and rabbits.[29,30] Consequently, it was recommended that divers with arterial gas embolism be placed in a (30°) head-down posture.

This advice is challenged by the following: it is often difficult to distinguish cerebral arterial gas embolism from cerebral decompression sickness;[31] significant arterial gas embolism probably occurs in decompression sickness by arterialisation of venous bubbles[32,33] and a head-down posture will enhance this by increasing venous return; in small vessels bubbles travel with blood flow;[34] a prolonged head-down posture increases cephalic venous volumes and makes subsequent middle-ear inflation (e.g. during therapeutic compression) difficult; a head down posture limits resuscitation; and, in air embolised dogs recovery of brain function was slower in those nursed head-down (45°) than in those nursed horizontally.[35] These observations must be balanced against the lethal deterioration seen in some divers suspected of arterial gas embolism when they have been sat up ;[36] although most will tolerate this procedure.

Consequently, while there may be benefit in a slight head-down posture for those with arterial bubbles, the balance of opinion and data favour a horizontal posture for injured divers.[5] If the diver is unconscious, vomiting, or if airway patency is suspect the diver should be nursed on the side. There are no convincing data favouring either side. Divers who are or have experienced neurological symptoms (the natural history of air embolism of the brain is for spontaneous recovery and then relapse[37]) must not be allowed to sit up until inside a recompression chamber. The only exception is in a diver with isolated inner ear barotrauma where sitting up will reduce perilymph losses.[38,39] If in doubt, the horizontal posture should be adopted.

Thermal balance

HYPOTHERMIA

Hypothermia contributes significantly to morbidity and mortality in divers and especially if helium is breathed. Helium's high thermal conductivity and capacity underlie the requirement for diving-bells to have an independent heat source.

Hypothermia can be arbitrarily divided into mild (35-32°C), moderate (32-28°C) and severe (less than 28°C) core temperature categories. A low-reading thermometer is essential to make this distinction. The diver should be both removed from the water and nursed horizontally.[40] Handling of the diver must be minimised to reduce the risk of cardiac arrhythmias. Manoeuvres that can precipitate hypotension (e.g. standing, hot showers) should be avoided as removal from the water may by itself reduce intrathoracic blood volume and hence cardiac output.[41] Neither EAR nor ECC should be withheld,[21] standard resuscitation routines should not be altered and resuscitation must continue until the diver is rewarmed.[19]

Wet clothing must be removed, including woollen and synthetic garments which lose insulation when wet.[19] The diver should be covered in the thickest driest coverings possible and protected from the environment. Re-warming is based on metabolic heat production and anything that inhibits shivering should be avoided (e.g. alcohol, vigorous rubbing). As shivering increases oxygen consumption, concurrent oxygen administration is beneficial. Hot bath rewarming in the field is poorly tolerated, although bath temperatures can be rapidly increased once the diver is immersed;[19] given the consequent loss of patient-access this procedure should probably not be used. Placing hot-water bottles or chemical heat packs around critical areas such as the head, neck, axillae and groins may be beneficial, although they have caused severe burns when applied to hypoperfused areas in hypothermic children.[42] Warming the inhaled air is advocated,[43] but has not been shown to have any benefit over dry insulation alone in adults.[19]

Intravenous rehydration should involve warmed fluids, but should not include dextrose solutions if brain injuries are suspected[44,45] or lactate-containing solutions which a hypothermic liver may not be able to metabolise. If the patient is conscious and capable of protected swallowing, food and warm drinks (as they will not contribute to any after-drop in core temperature) should be offered to the hypothermic diver.

Hospital management of hypothermia has included endotracheal intubation and warm air ventilation,[43,46] infusion of warmed intravenous fluids[47], peritoneal lavage or dialysis,[48] extracorporeal blood warming[49] and thoracotomy and bathing the heart with warm fluids.[50] Arrhythmia prophylaxis (e.g. Bretylium or lignocaine) remains controversial.

HYPERTHERMIA

Hyperthermia is likely to be encountered in warm climates and in recompression chambers or habitats. For example, heat exhaustion and cramps have been experienced in small transportable recompression chambers in tropical regions of Australia.[51] Given the limited inside space, the solution has been to protect the vessel from the sun and or to cool the vessel from the outside by using either water evaporation or ice packs. The latter have also been used successfully by the Royal Navy on small chambers in the Arabian Gulf.[52]

Deep oxygen-helium saturation exposures are also liable to hyperthermic stress[53] as the diver's thermal comfort zone is increased and narrowed, sweating is impaired and helium has 5 to 6 times nitrogen's thermal conductivity and capacity. Failure of habitat conditioning units can rapidly lead to hyperthermia. While the units are being repaired or replaced, immediate solutions include

protection from the sun and external cooling of the habitat, transfer of the divers into the water at their storage depth, applying ice packs to the divers themselves, venting (flushing) the habitat and the divers breathing cooled gases via a built-in-breathing system (BIBS).

Fluid administration

With the possible exception of a diver with either pulmonary oedema from near-drowning or brain oedema from air embolism, liberal intravenous fluids will not disadvantage injured divers and will usually be helpful. There are no data to support colloid over crystalloid solutions and bicarbonate solutions are probably only ever indicated if cardiac arrest is prolonged.[54,55] Oral fluid administration is not supported by data and should not be given to divers who are unable to protect their upper airway or who are vomiting. Similarly, diuretics such as alcohol should never be given to injured divers.

DECOMPRESSION ILLNESS

Intracorporeal bubbles compromise the microcirculation either directly by trapping in systemic[56] or pulmonary[57] arterioles or indirectly by damaging endothelial cells,[58] activating and causing focal accumulations of platelets[59] and polymorphonuclear leucocytes,[60,61] causing extravasation and haemoconcentration[62] and activating a variety of plasma proteins including those of the coagulation system, complement proteins, kinins and lipoproteins.[63-65] There is both *in vivo* [66-68] and anecdotal[69, 70] data to show that rehydration significantly improves outcome. While the dextrans have obvious theoretical advantages (oncotic and anticoagulant),[66] a practical advantage has not been shown. Glucose-containing solutions should be avoided as they may lead to a worsening of neurological lesions.[44, 45]

Intravenous infusion of fluorocarbon emulsions into animals with decompression illness has been shown to be beneficial both prophylactically[71] and therapeutically.[72,73] This is not surprising as these emulsions are rich in surfactants, have a high solubility for both oxygen (increasing oxygen transport) and nitrogen (increasing nitrogen removal), increase plasma volume and decrease blood viscosity and may also be anticoagulant. A trial in humans awaits demonstration of their stability and safety. Ethanol has similar properties to the fluorocarbon emulsions, but any defrothing activity or increase in nitrogen solubility is unlikely *in vivo* because of the low molar concentration of ethanol in blood. Despite a reported improvement in four Chinese divers with decompression illness after drinking a combination of ethanol and glucose and an increased survival of rabbits with decompression illness given an intravenous infusion of ethanol,[74] ethanol cannot be advocated because of its diuretic properties. Also, in a controlled prospective study of divers oral ethanol and glucose did not influence either the frequency or extent of bubbles detected ultrasonically after a bubble-provoking decompression.[75]

Fluid regimens in the decompression illnesses can either be arbitrary (e.g. 1 litre of crystalloid as fast as possible and then 1 litre 2 to 4 hourly) or titrated against haematocrit (to be reduced to less than 50%) or urine output (to be maintained above 0.5 ml/kg/hour). Accurate fluid balance recording is essential as oliguria or anuria despite fluid administration may indicate persistent haemoconcentration or bladder dysfunction; either warranting bladder catheterisation and an increase in fluid load.

PULMONARY OEDEMA AFTER NEAR-DROWNING

While some authors recommend use of plasma expanders,[76] others are sufficiently concerned about aggravating any pulmonary and brain oedema that they will limit fluid loads, even in the presence of systemic hypotension, to near-drowned patients until they can measure intracranial and pulmonary artery wedge pressures and cardiac output.[77,78] These measures are not available at a dive site and an unconscious diver may have brain air embolism or drowning, or both and distinction is often difficult. Pulmonary oedema can itself contribute to systemic hypovolaemia. Thus, oxygen and a fluid load should be given early, as preserving brain function is the primary aim and this depends upon adequate arterial oxygen content and cerebral perfusion.[79-82] Hypoxia from drowning needs 100% oxygen administration and, if necessary, application of positive end-expiratory pressure (PEEP).[55,83] If decompression illness is not suspected in near-drowning, then oxygen and intravenous fluids should be given to maintain normoxia and normotension.[20]

BRAIN OEDEMA

It has also been argued that brain oedema is a major determinant of outcome after air embolism and consequently that therapy should include fluid restriction, corticosteroids (discussed later) and diuretics.[84] Bubbles do increase the permeability of the blood-brain-barrier,[62,85,86] but this is transient (several hours) and in air embolised dogs brain function and water content did not correlate well.[87] Similarly, in brain injuries in other animal species and humans brain water content does not correlate with function unless the brain is so swollen that it is distorted or displaced.[88-93] In contrast, there is usually a good correlation between brain function and blood flow after air embolism[79-82] and consequently the fluid load should be adequate to maintain cerebral perfusion. Given that autoregulation of brain blood flow is lost after air embolism,[30,82,94,95] hypotension must be avoided and corrected quickly and there is probably no role for either fluid restriction or diuretics.

Drug administration

Although nasal decongestants, anti-emetics, antibiotics, analgesics, local anaesthetics, corticosteroids and anti-inflammatories (especially aspirin) are widely needed and used in divers, there are no drugs of proven benefit in the definitive treatment of the decompression illnesses.

CORTICOSTEROIDS

Some anecdotal data have been presented to support corticosteroid use in the decompression illnesses,[84,96,97] but these have either been individual case reports or retrospective studies in which numerous variables were changed simultaneously. Unless given prophylactically, corticosteroids have not been shown to be of benefit in animal decompression illnesses,[35,98] single factor analyses of corticosteroid use in air-embolised humans similarly cannot establish therapeutic benefit[99] and corticosteroid administration will increase the probability of overt oxygen toxicity.[100] Large doses of methylprednisolone have recently been shown to ameliorate spinal cord injuries,[101] but these doses have not been trialled in divers. It follows that while there are still no supportable indications for corticosteroid administration in diving injuries, the issue is uncertain.

ANTI-INFLAMMATORIES AND ANALGESICS

There is an obvious role for anti-inflammatories and analgesics in traumatic injuries in divers, but indomethacin is the only one of these agents which has a supportable role in the decompression illnesses.[102,103] However, support is exclusively from *in vivo* data and then only if prostaglandin $Pg1_2$(a vasodilator and anticoagulant) and heparin are also given. Concern about heparin has prevented human trials.

ANTICOAGULANTS

Bubbles induce platelet accumulation, adherence and thrombus formation.[59] Surprisingly, a variety of antiplatelet agents, and especially aspirin, have been extensively tried prophylactically and therapeutically without success.[69,70] These agents will also contribute to any evolving gastric stress ulceration.

Heparin and the other anticoagulants have some theoretical,[104] *in vivo* [105-108] and anecdotal[70,109] support for use in decompression illness treatment. However, heparin given either prophylactically or therapeutically to dogs with a decompression illness was not beneficial[110] and other animal-models of the decompression illnesses have either shown no advantage for heparin[111] or have shown haemorrhage into either the brain or spinal cord,[30,112-114] and into the inner ear.[107,115] Consequently, any anticoagulant is thought to be contraindicated.

LIGNOCAINE

Infusion of lignocaine either prophylactically[116] or therapeutically[35,117] has been shown to accelerate recovery of brain function in air-embolised cats and dogs, and this effect may be additive to that of hyperbaric oxygen in severe brain injuries.[35,118] Consequently, a multi-centre trial of lignocaine is underway in air-embolised humans.

DIAZEPAM

Diazepam has been widely used in divers.[14] It is effective in either preventing or treating oxygen toxic convulsions and in controlling vestibular symptoms. It makes titration of treatment of divers with vestibular symptoms impossible and is therefore not advocated.[119]

Recovery to a definitive treatment facility

Injured divers must first be resuscitated, stabilised and then retrieved under controlled conditions.

If any of the decompression disorders are present the diver must be immediately taken to a facility with a recompression chamber, avoiding decompression (i.e. to more than 300 m above sea level).

The decision then between air and ground transport should be based on minimising delay, although road contour, surface and altitude may make even a short ambulance retrieval unacceptable.

A successful series of 19 diver retrievals in a single-person monoplace recompression chamber was reported in Israel[120] and of more than 70 such retrievals in a 2-person monoplace chamber in Australia.[51] In the latter series, 4 of the patients had to be paralysed and mechanically ventilated en route. To be suitable for such retrievals, a transportable recompression chamber must have a working-pressure of at least 2.8 bar, provide access to the injured diver for resuscitation, have adequate lighting, have facilities for oxygen administration, mechanical ventilation and suction of the airway, have a carbon dioxide scrubber and adequate gas supplies to conduct a USN 6/ RN 62 therapeutic table, have a medical lock and a communications system and have a transfer-under-pressure (TUP) capability to a definitive recompression chamber. A TUP is a logistically demanding exercise involving risk and significant time-savings must be achievable to justify the use of transportable recompression chambers.

The two pre-requisites of air transport are that cabin altitudes do not exceed 300 m above sea-level and that access to the injured diver is adequate. With few exceptions, helicopters are unsuitable because of noise and vibration, limited space and unpressurised cabins. They should only be used as a last resort.

Similar transport requirements exist for divers with pulmonary barotrauma and if venous, arterial or chest cannulation is needed it should be performed beforehand. Also, divers who are near-drowned should be intubated and mechanically ventilated if spontaneous oxygen breathing alone does not significantly improve hypoxia and sustain cardiopulmonary function and mental state. It is essential that adequate communication with the definitive facility precede retrieval of the injured diver. Other measures such as resuscitation, posture, oxygen administration, thermal balance and fluid administration must be maintained throughout any retrieval.

IN-WATER OXYGEN THERAPY

In-water oxygen therapy is controversial and is contraindicated if the injured diver is unconscious, vomiting or incapable of protecting the airway. An in-water oxygen treatment of decompression illness is probably supportable only if the above contraindications are absent and a delay of greater than 24 to 36 hours will occur before retrieval to a recompression chamber. The Edmonds' regimen should be used (a 9 msw depth treatment),[1] and there must be adequate supplies of oxygen; some form of thermal protection for the injured diver and the attendant; a fullface mask and dedicated oxygen delivery system; a cradle or chair that can be lowered to and kept at 9 m depth; and, access to safe (i.e. free from surge, swell, current and animals) and preferably warm water.

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REFLECTIONS ON DIVER FATALITY AND SAFETY STATISTICS

Drew Richardson

Every death of a scuba diver is a human tragedy that diminishes the whole of scuba diving. Stories about diving fatalities remind us of the importance of established safety guidelines.

The thought of a person dying while scuba diving should cause us to think about our own mortality, diving practices and dedication to safety. We want to know why the accident happened. We search for the reasons to give us perspective, understanding and control.

When combined, proven diving practices and sound judgment afford a reasonable envelope of safety in which to dive. When rules are broken, limits stretched or ignored, we increase our risk. Occasionally, factors beyond our control compound the difficulty of a dive, leading to stress and in the extreme, death. Fatality statistics are an important barometer on the safety of our sport and the adequacy of our controls.

By carefully studying the causative factors leading to diving deaths, we help educate ourselves to avoid repeating tragedy. An excellent source of this information is produced annually by Divers Alert Network (DAN).

The National Underwater Accident Data Center (NUADC) at the University of Rhode Island, USA, has been reporting diving fatalities since 1970. Beginning in 1989, DAN has collaborated with NUADC in reporting diving fatalities. The data used in this paper are drawn from the 1991 and 1992 DAN Reports.[1,2]

In the past 23 years a total of 2,404 U.S. recreational scuba fatalities have been recorded.[2] In the first 10 years of fatality reporting, the average diver fatality per year was 123.[2] Approximately half of all recorded recreational scuba fatalities occurred from 1970 through 1979, when both the dive industry and number of divers were smaller.[2] Today, the average number of fatalities is approximately 104 deaths per year,[2] as the number of divers has grown into the millions. In February 1994, PADI alone certified its five millionth diver. These facts reflect the trend that diving safety has improved greatly over the years. The estimated fatality rate, per 100,000 active dives per year, has dropped from about 8.6 in 1976 to about 2.7 in 1991.[1] Figure 1 shows the total numbers of U.S. recreational diving fatalities by year, as reported by NUADC and DAN.[1,2] The incidence of scuba deaths has generally shown a decreasing trend since 1970. Improved training standards, equipment and diver awareness are in part responsible.

Throughout the world, the popularity of scuba is increasing. Fortunately, diving deaths are rare; while this is good news, we must persevere with the fundamental educational issues of diver health and safety. Fewer deaths each year are only possible when we all continue to emphasise the importance of diver training and awareness.

It is important to analyse and review the factors involved in diving fatalities. To help us understand and avoid diving deaths, we need to know more about the divers and what they were doing during the dive. In a general review of the top three causes of diving deaths between 1970 and 1992, drowning/asphyxia was the leading cause, followed by embolism and heart attack/cardiac problems.

Table 1 reflects the primary diving activity at the time of death as reported by DAN.[1,2] Note that 10.4% of the deaths occurred in divers who were under instruction. This is an instructor's nightmare. These sobering data remind all involved in diver training of the importance of good judgment, control and emergency procedures in our dive planning for open water diver training.

Twenty-one deaths occurred in technical diving environments in 1991 and 1992.[1,2] At least 10 of these people were not properly trained or equipped for the dive they chose to do. Several of these technical deaths were diving at depths greater than the recreational limit of 40 m (130 ft), at least five were to depths of 60 m (200 ft) or more.[1,2]

Other fatalities were attributed in part to diving conditions. Environmental conditions vary widely throughout the world with the effects such as latitude, temperature, elevation, wind, current and visibility changing from site to site and even on the same site from one moment to the next. Divers should stay within the limits of their training and skills when choosing a dive site. Furthermore the dive conditions should be consistent with one's training, experience and comfort level.

Death usually results from multiple factors. However, there is often one event or condition which precipitates a sequence of events. While single event problem solving may be routine, multiple problems occurring simultaneously or in sequence may overwhelm a diver.

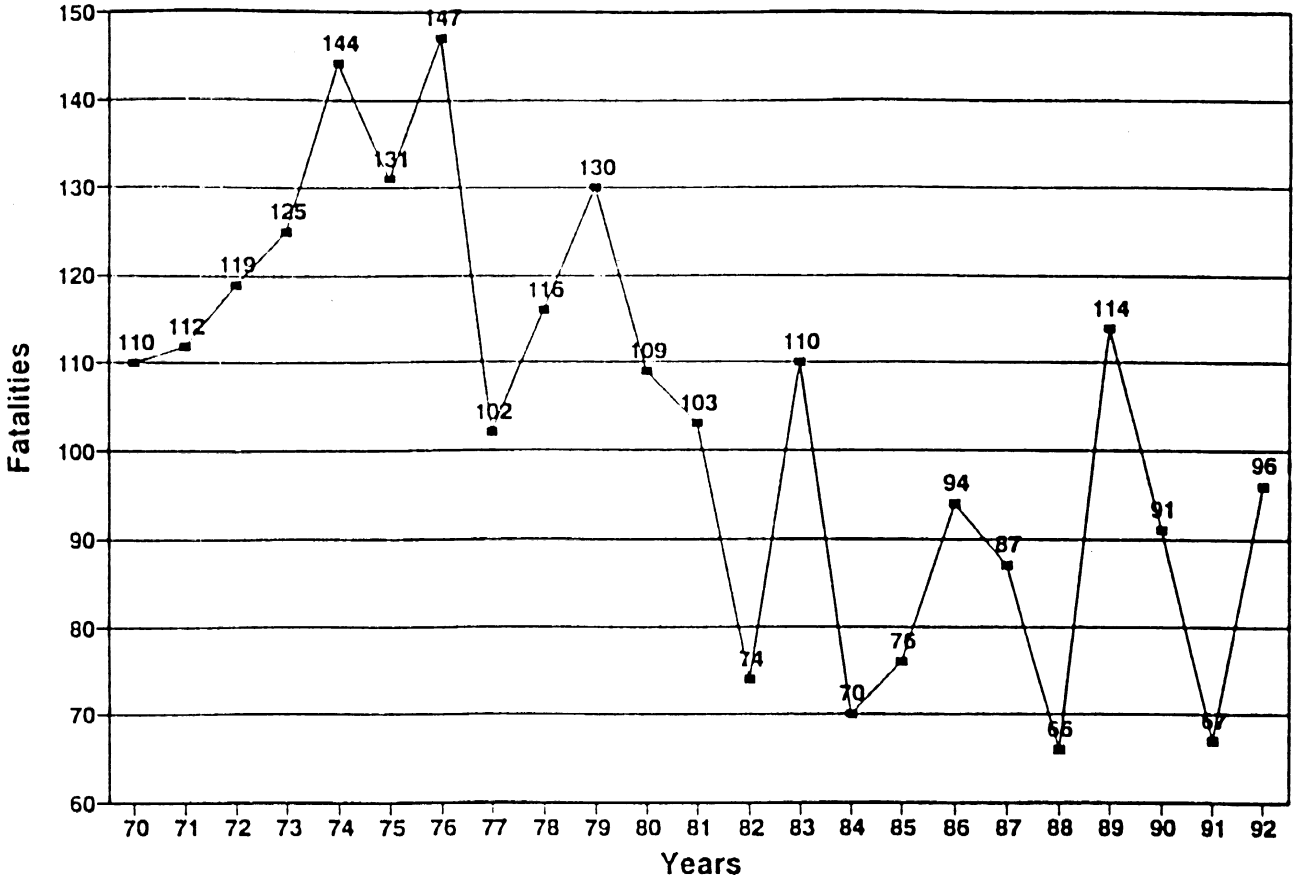

Figure 1. United States recreational diver deaths in the years 1970 to 1972 (Compiled from reference 2).

TABLE 1

DIVE ACTIVITY AT TIME OF DEATH

Dive Activity	Certified to Scuba Dive	Not Certified to Scuba Dive	Certification Unknown	Total	%
Unknown	—	—	5	5	3.0%
Pleasure	63	11	2	76	46.6%
Under instruction	7	10	—	17	10.43%
Spearfishing or hunting	14	1	I	16	9.8%
Wreck diving	15	—	—	15	9.2%
Photography	6	—	—	6	3.6%
Collecting/work/task	4	—	1	5	3.0%
Night	2	—	—	2	1.2%
Non-recreational diving					
Cave	13	—	—	13	79%
Deep more than 39 m (130 feet)	6	—	—	6	3.6%
Under Ice	2	—	—	2	1.2%
TOTAL	**132**	**22**	**9**	**163**	**100.0%**

Table 2 shows when and where the deaths occurred.[1,2] The relatively high number of divers who experienced a problem late in the dive and on the surface after the dive is significant. When one stressful event sets up a chain reaction of events, these events, if unresolved, may lead to death. Examples of events that contributed to deaths are: entrapment, getting lost, running out of air, rapid ascent, panic, narcosis, asthma, cardiovascular

TABLE 2

WHEN AND WHERE THE PROBLEMS OCCURRED IN 155 DIVING DEATHS

	When
Surface Predive	7
Immediately	8
Early Dive	21
Mid-Dive	19
Late Dive	41
Post Dive	33
Unobserved	21
No Information Available	5
	Where
Surface	7
During Descent	15
At Depth	33
During Ascent	39
Surface Post Dive	35
Unobserved	21
No Information Available	5

disease, alcohol and drug use. Table 2 emphasises the need for divers to pay attention to safety during all phases of the dive. Buddy separation was reported in nearly half of the deaths for the period. There were also five divers in 1992 who died while diving alone.

Worthy of special mention is careful air supply monitoring. Air management continues to be of concern. DAN reports that air consumption was the probable starting cause in several deaths. These cases included: running out of air, low on air, insufficient air causing a rapid ascent, running out of air as a secondary event to entrapment or being trapped.

Table 3 identifies the certification level of the 1991 and 1992 fatalities.[1,2] Entry-level divers have skills, experience and knowledge that limit them to diving conditions similar to their training environment. PADI and other training organisations recommend additional training to become familiar with different conditions and environments. Quality entry-level training, continuing education, proper attitude and judgment, environmental orientation diving and adherence to safe diving practices all help.

The causes of death are of interest. Of the cases that were autopsied, it was determined that 22 deaths were due to arterial gas embolism (AGE); 11 were immediate and 11 suffered AGE before drowning.[1,2] The factors that led up to this were insufficient air supply problems, panic, lung abnormality, presumed breath holding, rapid ascent, asthma, coronary artery disease, obesity, sudden death (in a presumably healthy individual), diabetes and buoyancy problems at the surface.

TABLE 3

CERTIFICATION LEVEL OF 163 DEATHS IN 1991/1992

Certification	**Number**	**%**
None	12	7.3
Unknown	15	9.2
Student	10	6.1
Basic/Open Water	80	49.0
Advanced	20	12.2
Rescue	2	1.2
Divemaster	9	5.5
Instructor	7	4.2
Commercial	1	0.6
Military	2	1.2
Cave/Cavern	5	3.0
TOTAL	**163**	**100**

Drowning remains the most frequent cause of death in divers (Table4). It follows the occurrence of factors that prevent the diver from either reaching or remaining on the surface. Many people think that “drowning” as cause of death may simply be the response of non-diving coroners who do not autopsy. Buoyancy problems at the surface, where the diver failed to establish buoyancy and sank were reported in several cases. Panic may complicate the diver’s ability to operate equipment, such as a BCD, properly or to drop a weight system. Insufficient air leading to drowning contributed to 32% of the total drownings reported.[2]

These sobering statistics should renew our personal commitment to diving safety and education. If we understand the risks that have led divers to their deaths, we may be more effective in influencing the attitude and judgement of other divers.

Responsibility to Diving Safety

After certification, ultimate responsibility for safety rests with each diver. The responsible individual must make the decision to dive or not for himself. The role of the diving instructor is to train each person to be capable of assessing diving conditions without help after training. For divers to make this choice, thorough training and teaching are important, as are disciplined diving habits within the buddy team in the field.

Over 15 years ago PADI published the PADI Safe Diving Practices in the PADI *Diver Manual* as follows:[3]

1 Maintain good mental and physical fitness for diving. Avoid being under the influence of alcohol or

TABLE 4

FACTORS CONTRIBUTING TO DROWNING

Contributing Factors	Number of Divers
Insufficient air	41
Buoyancy problem*	15
Entrapment	23
Cardiovascular	12
Alcohol/drugs	9
Panic state	9
Nitrogen narcosis	7
Air embolism	10
Hypothermia	1
Obesity	4
Rapid ascent	4

*At the surface, failed to establish buoyancy and sank.

dangerous drugs when diving. Keep proficient in diving skills, striving to increase them through continuing education and reviewing them in controlled conditions after inactivity.

2 Be familiar with your dive sites. If not, obtain a formal diving orientation from a knowledgeable, local source. If diving conditions are worse than those in which you are experienced, postpone diving or select an alternative site with better conditions. Engage only in diving activities which are consistent with your training and experience.

3 Use complete, well maintained, reliable equipment with which you are familiar; and inspect it for correct fit and function prior to each dive. Deny use of your equipment to uncertified divers. Always have a buoyancy control device and submersible pressure gauge when scuba diving. Recognise the desirability of an alternative source of air and a low pressure buoyancy control inflation system.

4 Listen carefully to dive briefings and directions and respect the advice of those supervising your diving activities.

5 Adhere to the buddy system throughout every dive. Plan dives, including communications, procedures for reuniting in case of separation, and emergency procedures, with your buddy.

6 Be proficient in dive table usage. Make all dives no-decompression dives and allow a margin of safety. Have a means to monitor depth and time under water. Limit maximum depth to your level of training and experience. Ascend at a rate of 18 m (60 ft) per minute.

7 Maintain proper buoyancy. Adjust weighting at the surface for neutral buoyancy with no air in the buoyancy control device. Maintain neutral buoyancy while under water. Be buoyant for surface swimming and resting. Have weights clear for easy removal, and establish buoyancy when in distress while diving.

8 Breathe properly for diving. Never breath-hold or skip-breathe when breathing compressed air, and avoid excessive hyperventilation when breath-hold diving. Avoid overexertion while in and under the water and dive within your limitations.

9 Use a boat, float or other surface support station whenever feasible.

10 Know and obey local diving laws and regulations, including fish and game and dive flag laws.

These guidelines evolved from many years' experience. Safe diving guidelines result from accident history and are adopted in the attempt to help others avoid repeating mistakes. Still in use today, it is possible some new divers may not necessarily know why these guidelines were created or the fact that many resulted from accident and fatality analysis.

In 1993 Edouard Lagache, a doctoral candidate at the University of California, Berkeley, produced a paper titled, *An introduction to semi-qualitative methods for the analysis of recreational scuba accident data*. In it he analysed 10 years of diver fatality data from the Ontario, Canada, Underwater Council 1989 report. He compared the events and circumstances surrounding the fatalities in this report to a list of safety rules he derived from the established PADI Safe Diving Practices. Lagache reported his findings in the Undersea Journal in an article entitled, *Are Divers Choosing to Die ?*[4] He suggested that in 87% of the deaths, at least one derived safety rule was broken and in over 80% of these cases, the violation clearly contributed to the accident! His research supports the commonsense notion that violating safety guidelines does indeed have something to do with promoting accidents. The rules that were broken with the most frequency were, dive with good gear and set up for the chosen location, do not use alcohol or drugs, do not lend gear to uncertified people and groups of divers should be broken down into buddy teams.

Certain violations can be classified as those that cannot be violated without choosing to do so. Lagache's research indicates that in 41 % of all fatality cases he analysed, when rules were broken, they may have been broken by the individual diver's choice and not by accident. Additionally, his data supports the premise that series of chain events (multiple events) are most often involved in diver fatality, ultimately overwhelming the diver and prohibiting any chance of recovery.

Diver training and education programs address the problem of ignorance and inexperience in divers. Diver education has indeed reduced fatalities over the years. Education has limits, however, and individual attitude is an important factor. Because people can do whatever they wish to do, responsible choice making and social peer acceptance variables have an important role and influence at the dive site.

The development of proper attitudes about training is the key to maximising the likelihood that the majority of divers will stick to proven safety guidelines. This process begins in the entry level diver course with the instructor. It is reinforced within the various training materials, texts and industry publications available to divers.

To be successful in reducing diving fatalities, responsible diving behaviour must be nurtured at a grass-roots level. Because diving is a social pursuit, divers do learn from other divers, buddies, friends and divemasters. All of these people collectively provide a community of example. In this way, divers go through a type of apprenticeship. Social forces and peer pressure shape diving practice either in a positive or negative way, depending on what the group tolerates. Good diving habits are developed in part by example and safety attitudes. Divers who rush into the water with leaking regulators or faulty equipment without solving the problem, need to stop and think about the fact that they are increasing the risk to themselves and their partners. Responsible group dynamics must accommodate corrective behaviour, before diving, in order to keep risk levels acceptable.

Ideally, safety is freedom from risk. Unfortunately, risk is unavoidable in any sport and diving is no exception. However, risk can be minimised. Good judgment and training will help ensure risks are judged as acceptable and diving will remain "safe." History has shown the benefits of detailed dive planning, performing a pre-dive safety check and following safe diving practices in reducing diving deaths. Diving death is not a random event. While we may never be able to reduce the fatalities to zero, we must never cease in our efforts to do so.

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A PROGRESS REPORT ON DIVING MEDICINE STUDIES IN THE ROYAL NEW ZEALAND NAVY

Des Gorman, Alison Drewry and Simon Mitchell

Abstract

Three prospective, randomised, controlled studies involving oxygen-helium and lignocaine in decompression illness and lignocaine in patients undergoing cardiopulmonary bypass are underway at the Royal New Zealand Navy's Auckland Naval Base. Two other studies are designed to elucidate whether there are objective markers of decompression illness, in either the coagulation cascade or bone imaging; and a survey of the health status of the Royal New Zealand Navy Diving branch has been conducted. No progress has been made in the study of the role of chest and abdominal splinting in the prevention of pulmonary barotrauma.

Introduction

A series of studies relevant to diving medicine have been initiated by researchers at the Auckland Naval Base. These include:

a) prospective, randomised, controlled trials of (i) oxygen-helium versus oxygen, (ii) lignocaine versus placebo in the treatment of decompression illness (DCI) arising from recreational air diving, and (iii) lignocaine versus placebo in the prevention of brain injury due to gas emboli during cardiotomy;

b) a study to determine whether there are any measurable changes in the coagulation system in DCI,

c) a study of bone changes in acute DCI using nuclear magnetic resonance imaging (NMRI);

d) a survey of the current health status of the Royal New Zealand Navy (RNZN) Diving Branch;

e) a study of the role of chest and abdominal splinting in the prevention of pulmonary barotrauma.

These studies are reviewed below:

Prospective randomised controlled trials

OXYGEN-HELIUM STUDY

The rationale for a comparison of oxygen and oxygen-helium as the ideal therapeutic gas mixture to be breathed during the recompression of divers with DCI has been described previously.[1] The study has been in progress for 2 years and 88 subjects have been treated, of whom 12 failed to meet the trial criteria (due to pregnancy, wrong diagnosis or failure to follow the study protocol).

One-year follow-up results are available for 56 subjects. The treatment groups are directly comparable with respect to age, sex and symptoms and a successful outcome was achieved in 80% for each group. However, fewer patients treated with the oxygen-helium required multiple recompressions (9 of 25 compared with 20 of 31 in the oxygen group) and this difference is statistically significant ($p= 0.03$). The cost-effectiveness of the treatment regimens will be compared to assess whether the use of oxygen-helium represents an important advantage. Meanwhile the study continues.

LIGNOCAINE STUDIES.

The need for a controlled trial of lignocaine versus placebo in the treatment of DCI has been demonstrated by a pilot study.[2] A prospective, randomised, controlled trial of lignocaine versus saline in refractory DCI has been initiated at RNZNH and acute cases will be studied after completion of the heliox trial. In the meantime, other centres are being recruited to commence the acute phase of the study, but no results are yet available.

The rationale for employing lignocaine in the treatment of DCI has also been applied to studying the use of lignocaine prophylactically in cardiothoracic surgery. Neuropsychological sequelae are reported with variable but significant frequency in patients following cardiotomy, largely due to the introduction of solid and gaseous emboli into the arterial circulation.[3-8] This has obvious parallels with the pathophysiology in DCI and cardiotomy patients are to be studied in a controlled trial of lignocaine versus placebo, in collaboration with the cardiothoracic surgical team at Green Lane Hospital. Efficacy will be assessed primarily by neuropsychological evaluation before and after operation, and test scores will be correlated with arterial emboli counts.

There is a need for an objective "measure" of DCI, which could be monitored and used to predict outcome and gauge response to treatment. These studies aim to determine whether measurable systemic changes occur in DCI and can be correlated with disease progress.

Coagulation change study.

It has been proposed that the formation of aggregates of blood constituents, and subsequent embolic phenomena, is both a primary and perpetuating event in DCI.[9] Coagulopathies have been demonstrated in animal models of DCI, but reports of abnormal haematological parameters in human subjects are rare, although this may be due to the use of insufficiently sensitive tests. In this study, all recreational divers with DCI presenting for therapy, will have sequential sampling of the coagulation cascade (APTT, INR, bleeding time, factor viii, von Willebrand's factor, fibrinogen and proteins c and s, antithrombin III) throughout the treatment period, and the results compared with clinical outcome.

Bone change study

The pathophysiology of dysbaric osteonecrosis is uncertain, but there is a significant relationship with DCI, although a common cause cannot necessarily be inferred.[10] NMRI is more sensitive in the early detection of avascular necrosis than other imaging modalities, and may detect acute ischaemic changes which subsequently resolve.[11] Patients with acute DCI will undergo initial NMRI within seven days of presentation for treatment and thereafter as the clinical course and imaging results dictate.

Health status of the RNZN Diving Branch

Concern over the long-term health effects of diving and significant changes to the diving practices in the RNZN (the introduction of helium, deep "bounce" diving and saturation diving) prompted a survey of the current health status of the RNZN diving branch. Health status has been evaluated by questionnaire, physical examination, neurocognitive assessment, long bone survey, audiometry and spirometry. Individual diving exposure was determined by recording the number and depth of dives, gas mixes used, decompression schedules followed and any diving incidents experienced. Few exposure-related abnormalities were found. This may reflect the "healthy-worker" effect, but the group now forms a cohort which will be matched with non-diving controls and subjected to sequential evaluation.

Pulmonary barotrauma

A study of the role of chest and abdominal splinting in the prevention of pulmonary barotrauma has not yet proceeded because of technical difficulties.

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

DEVELOPMENT OF SATURATION DIVING : THE HUMAN FACTORS

David Elliott

Abstract

The concept of remaining at depth beyond the time for equilibration with inert gas is attractive commercially because the ratio of working hours to hours spent in decompression is optimal. Saturation diving is a unique work practice with a mere 25 year history and is still developing. It is now a normal feature of offshore diving but, unlike most jobs, it requires the diver to live in his unphysiological workplace. This demands more attention from the occupational health physician than the application of conventional diving physiology and medicine. During compression, attention is usually focussed upon the amelioration of the high pressure nervous syndrome (HPNS), at depth upon the limits of safe excursions and during decompression, on the avoidance of bubbles. Though important, these are not the only issues. The ergonomic design of the chamber system includes details such as control of noise and lighting, and special provisions such as the need to adapt the facilities in response to possible medical emergencies. Bacteriological and environmental control extends to avoiding contamination. Bell design and, in particular, the setting of standards for breathing apparatus have received considerable attention but tool design, communication and thermal balance relatively neglected. For the saturation diver, the management of nutritional status requires careful attention. Work-cycles are carefully defined, but sleep can be disturbed by the noise of others in the chamber and by a sensitivity to small changes of temperature. Finally, on surfacing, reports of fatigue and intolerance are common. What are the medical priorities in the support of saturation diving, and what are the priorities for research?

Introduction

There is no doubt that the development of saturation diving over the last 30 to 40 years has been a simple consequence of man's need to work at depth for longer periods of time than are compatible with the maximum safe durations of surface-orientated bounce (there and back) diving. Indeed, after attaining equilibrium between the tensions of respiratory and dissolved gas any prolongation

of bottom time does not add any time to the obligation for subsequent decompression. From a commercial point of view, the ratio between the diver's effective working time at depth and the time spent in decompression is significantly improved by this procedure.

This paper reviews the origins of this very practical concept, traces its development for both scientific and commercial use and notes the recognition, assessment and control of the various physiological and medical hazards that are encountered.

History

The concept of a saturation exposure was proposed first by Behnke[1] for compressed air workers. "From the point of view of physiologic response and work output, the attempt to decompress men twice daily is not only potentially dangerous but also high uneconomical. It would appear advisable therefore to keep men at work on a job continually under pressure. Following a work shift at maximum pressure, the pressure could be lowered rapidly to between 20 and 30 psi and maintained at this level during the rest and sleep period." (30 psig approximates to 300 kPa or 20 msw or 3 bar).

Kindwall[2] suggests that the first saturation exposure was conducted by End and Nohl in 1938, but it is not clear from that description whether their 27 hours in a chamber at 101 feet (30 m) was planned to demonstrate the decompression benefits of saturation or whether the dive was for some other purpose, the saturation being incidental.

Credit for turning the concept into reality belongs to George Bond. His original proposal in 1957 for the Sealab series of dives[3] states "It is believed that successful completion of this research effort will provide a demonstration of man's capability for free-ranging existence under conditions of high ambient pressure such as might be encountered in life on the ocean floor of a continental shelf." In Workman's "Project Genesis" animal studies preceded human exposures.[4]

Dugan[5] tells of a meeting in his apartment between Captain Bond and Jacques Cousteau in September 1959 when George Bond explained his experiments and his ideas. This story is recounted by Bond who said "in late 1959 Jacques Cousteau and I appeared together on an undersea program in New York. Afterwards the undersea chronicler Jim Dugan invited me to his apartment for further talks with Cousteau. We discussed my concept of saturation diving. In fact, we talked until breakfast!".[6]

The first to take advantage of such developments was Ed Link's *Man-In-Sea* program,[7] largely concerned with underwater archaeology and wrecks, and supported by the Smithsonian Institute. Based upon the work on saturation by the US Navy, he began with a simple submersible decompression chamber (SDC) which would be used to return divers at pressure to the vessel from their seabed habitat. In September 1962 Robert Stenuit made a dive to 74 m from a small saturation chamber at 61 m. Captain Bob Bornmann (whose hard work and enthusiasm later founded the EUBS) was seconded from the US Navy to be the Physiological and Medical Adviser for the dive. Stenuit was very cold in the SDC overnight and, because of a shortage of helium, the bottom time was terminated after 24 hours at depth. Only 4 days later Cousteau began Conshelf I, a 7 day exposure for 2 men in a habitat at 10 m.

It was not until 1964 that the US Navy was able to mount Sealab I in which 4 divers spent 10 days at 58 m.[8] In the interim, Cousteau's Conshelf II had been home to 7 divers at 11 m, 5 of them living there for a month.[9] A year later, in June 1964, Ed Link used his SDC to take 2 divers down to their inflatable house, the SPID, at 132 m for 48 hours.

In the US Navy's Sealab I at 680 kPa (58 msw or 6.8 bar) there was an apparent slowing of all gross ological and motor functions but it was also noted that when oxygen levels were held at 4% (approximately 1 atmosphere) or greater, the aquanauts reported an improved sense of well-being. After some 24 hours of exposure at 58 m, after denitrogenation of the divers in the oxygen and helium atmosphere, exposure to compressed air resulted in an immediate and dangerous level of nitrogen narcosis, equivalent to that experienced breathing air at 108 m. The authors concluded that once the body is essentially denitrogenated, susceptibility to nitrogen narcosis is significantly increased. In the US Navy's Sealab II in September 1965, there were extensive physiological investigations while, for 45 days, 3 teams of 10 men occupied the habitat at 62 m. Following dives into cold water, a marked reduction in temperature of the extremities and the slight rise of the core temperature was recorded. On return to the elevated temperature of Sealab and particularly following a hot shower, rectal temperatures fell sharply. This fall was due to peripheral vasodilatation and partially explained the paradoxical shivering, a subjective feeling of being warm, coupled with an objective pronounced shivering observed by several divers.

There were no neurological alterations resulting from the exposure, although EEG studies revealed slight transient changes during the time at depth. Psychophysiological examinations also revealed no deleterious effects from the prolonged dive. The concurrent Conshelf dives led, in 1965, to Conshelf III at 100 m. The habitat was home for 6 men for 30 days and there were no adverse effects reported.

The year 1962 was certainly a remarkable one in the history of diving for, in that year after a series of

iconoclastic dives with "impossible" decompression times, Hans Keller made a brief dive to 304 m off Santa Catalina Island, California. His companion in the diving bell died during the decompression, and yet that dive not only broke the thousand foot barrier for deep diving but also led to developments which were significant for the future of saturation diving. Keller's dive was a prolonged bounce dive using a series of different gas mixtures calculated by Bühlmann[10] to minimise decompression time. The tragic circumstances of that 304 m open-sea dive led to a conviction in the US oil industry that deep diving should be avoided and, consequently, the one atmosphere seabed habitat was developed for the purposes of well servicing. In contrast, an European view was that, notwithstanding the fatality, Keller's achievement was to show that man could go to and come back from such deep depths. In order to develop such procedures, a commercial diving company with 60% oil company ownership, was set up in Italy. The associated program of deep diving research continued in the chambers of Zurich University. Using compression rates of 1 m per minute to depths as great as 500 m and with prolonged, yet relatively rapid, decompression profiles, much knowledge was gained.

Though now this may be perceived as modest, another breakthrough in 1962 was a working surface-orientated helmet dive made by Dan Wilson to 128 m for the oil industry. From this experience Dan then developed, with Jon Lindbergh, the first submersible compression chamber specifically for commercial purposes.

The first commercial saturation dive was conducted in 1965 by Marine Contractors of Connecticut with technical hardware provided by Westinghouse. The divers spent up to 5 days at 61 m working on the Smith Mountain Dam in Virginia.[11] The following year the same contractors used the Cachalot diving system again in the first saturation dive for the oil industry. This dive to 73 m was to remove parts of a platform destroyed by a hurricane.

In subsequent years research diving continued to progress in depth and duration, whereas dives conducted as commercial diving contracts made much slower progress. Among the significant commercial dives to follow was the recovery of a blow out preventer (BOP) stack from 326 m in 1975. For the installation of the oil platform Cognac, Taylor Diving undertook saturation dives over two seasons which totalled more than 14,000 man hours in saturation at depths greater than 290 m. Though somewhat shallower at 240 m, the salvage of gold by Wharton-Williams (2W) from the wreck of HMS EDINBURGH in 1981 is remarkable for the amount of hard physical work undertaken by the diving team in their lock-outs.

These operational experiences tend to be overshadowed by the deep test dives, in the laboratory and in the sea, that occurred in the United States, the United Kingdom, France and Norway during this time. After a dive in 1992 on hydrogen-helium-oxygen mixtures to 701 m by Comex, one might ask why operational diving has never exceeded about half the depth achieved in such trials. One tempting answer might be that breathing apparatus design, communication systems and engineering requirements to support such deep dives have not yet been fully developed to the standard required for health and safety. A more relevant answer is simply that there is, as yet, no work at such deep depths.

However, in spite of the excellent progress made in the design of remotely operated vehicles (ROVs) to eliminate the need for deep diving, there are still tasks for which the human hand and eye are needed at depth. As long as there is the possibility that just one diver will need to lock out at a great depth, all the supporting services and research must be provided, undiminished.

Saturation diving today

It is not possible to acknowledge all the many pioneering dives done over the last 25 years upon which current procedures have been based. In a review of the high pressure nervous syndrome Bennett and Rostain[12] review more than 70 well documented dives and no doubt there are others. With gratitude to the many divers who have taken part in this development work, one is now in a position to review saturation diving today. The approach will be that of occupational health as seen by the physician responsible for the medical support of such saturation diving operations. While there are some aspects open to further debate, what follows is essentially pragmatic.

The diving systems and equipment

Although a review of compression and decompression procedures might be of greater interest to some, the proper design of the control room from which the dive is to be run deserves its share of attention. Essential aspects include the design of the individual controls and displays in a co-ordinated manner which reflects functional requirements. The interaction with the physical characteristics of the users and the relative importance of anticipated situations such as the critical actions required in an emergency, imply the use of good ergonomic principles.[13] Specifications have been given for adequate illumination of all working areas, for sufficient space to avoid crowding and for work patterns that minimise any reduction of vigilance. The entry of persons not connected with the dive should not be allowed, and the traffic flow in and out of the control room must not interfere with the tasks of the controllers. The ready availability of fire extinguishers, breathing masks and emergency power sources that might be required in an emergency must be tested to ensure compatibility with the continued control of the chamber and diving systems.

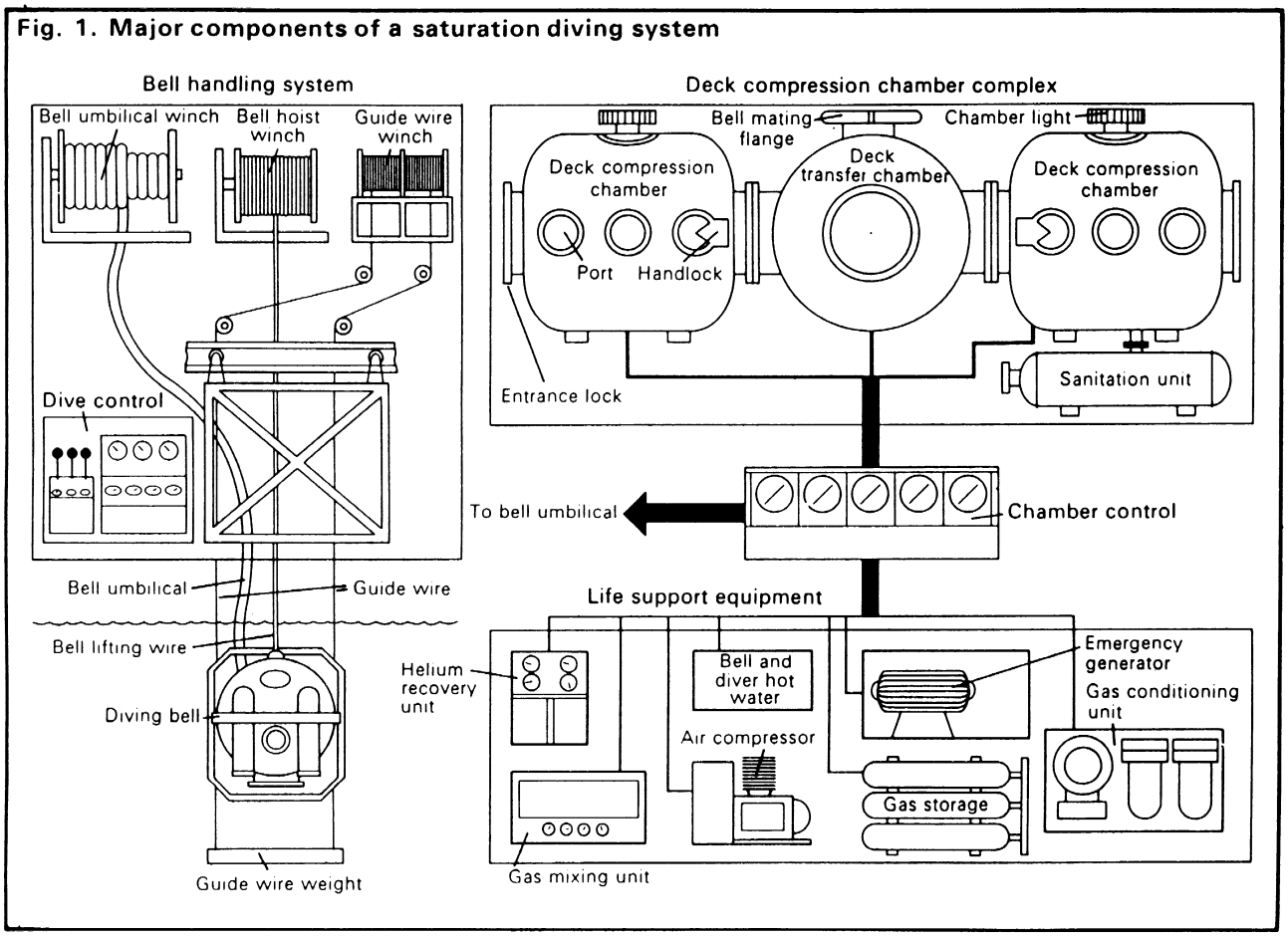

Figure 1. Diagram of a saturation diving complex. Reprinted ,with permission, from Sisman D. Ed. *The Professional Diver's Handbook.* London: Submex, 1982

The chamber complex, diving bell, welding habitat and hyperbaric evacuation unit (Fig.1) need to be designed ergonomically and each must be designed to cope with all foreseeable emergency situations.[13,14]

The design of the breathing apparatus should be based on physiological principles[15] and comply with current national and international standards and, under European Economic Community (EEC) law, also will need to comply with any European Community Norms (CEN) standards. However the community directives address only the need for each individual piece of equipment to meet an approved standard. There is no requirement for the compatibility of different items of approved equipment to be tested by the manufacturer. This is a duty placed upon the user. So the chosen underwater breathing apparatus, mask, helmet, hot-water suit and associated equipment must be assessed together ergonomically as part of the personal diving system. Detailed specifications are needed for the different performance objectives of underwater breathing apparatus, standby-diver breathing apparatus, bail-o u t systems, welders' masks, emergency breathing apparatus for use in a welding habitat and the built in breathing system (BIBS) needed within the chamber complex. For example, the welder's mask should be designed to provide an adequate field of vision for the welder but, if this is not possible then the availability of a nose-clip and mouthpiece should be considered.

The gas hoses used both in the bell umbilical and from the bell to the diver need to be tested to ensure that contaminants do not enter the gas supply. For instance, the degradation of some polymers and plasticisers used in the hose can lead to traces of caprolactam in the breathing gas. In braided bell umbilicals, bacteria can generate H_2S which has a potential to pass through the hose wall.

Operational considerations

There are a number of practical aspects of saturation diving which are not primarily medical or physiological, but about which the occupational physician must be aware. For example, the use of electricity underwater is

particularly hazardous and all use of electricity needs to comply with a code of practice such as that which has been produced by the Association of Diving Contractors.[16] Underwater tools need to be designed in compliance with the ergonomic and safety principles with particular regard to weight, buoyancy, balance and reaction. The majority of such tools will be used by divers who are wearing gloves and, if those tools are to be used effectively and safely, this fact needs to have been assessed in the design. A number of power tools are noisy and all should comply with the appropriate noise level requirements for both impact and continuous noise. Radioactive sources are another obvious hazard but, properly managed, should be associated with a low risk.

The maximum in-water and bell-run times are usually specified in national regulations, as are the maximum durations which the divers may spend in saturation. There has been recent controversy over the minimum interval to be spent at the surface by the diver between saturation exposures and how soon air diving may be resumed after the completion of a saturation dive. There may be good reasons for such restrictions but these various times[17] have little basis in science or medicine and are largely defined by sociopolitical considerations.

Compression

The rate of compression needs to minimise HPNS and, for dives down to about 200 m, such procedures are reasonably well established. Indeed, in Norway the minimum compression times, defined by travel rate, travel time and stop time, are specified in a report, published by the Norwegian Petroleum Directorate (NPD).[18] Deeper than 300 m the compression profile is not so easy to define and it is important that any procedure used is one that is based upon practical experience. Compression to depths beyond 300 m entails a degree of physiological stress and, in order for the divers to be able to cope adequately, they should be well rested prior to compression. Trimix, usually 5% nitrogen in oxy-helium, is not used commercially for a number of reasons. Current oxy-helium profiles tend to follow the principles that have developed over the years[19] and successfully tested in the dive "Aurora '93" at the National Hyperbaric Centre, Aberdeen.

The atmosphere of air in the chamber at the start of the operation need not be flushed out prior to compression. However the further introduction of nitrogen in the chamber, as by use of the food and medical locks, should be kept to a minimum. A full system check is usually conducted at 10 m and if this check discloses problems the chamber may be held at this depth without any need for decompression stops should it be necessary to return to the surface. Compression is then usually made with heliox with PO_2 between 38 and 42 kPa (0.38 and 0.42 bar). A higher oxygen content is available to the BIBS for emergency breathing at all times.

It is also important that during compression the chamber temperature should be maintained at a level at which the divers feel slightly cool. Once at the living depth, a 12-hour stabilisation period is required before the first bell-run.

Storage depth

At the living depth in the chamber the PO_2 should be maintained between 30 and 50 kPa (0.3 and 0.5 bar), a lower oxygen level being considered to reduce the possibility of post-dive pulmonary effects.[20] The storage depth should be within 1 m of the working or welding habitat depth wherever possible so that excursions can be kept to a minimum.

Environmental control

The control of the temperature should be adjusted within the range 22 and 33°C in all the chamber compartments, in accordance with divers' comfort. The relative humidity should be kept as low as 40%, if possible. Noise levels must be kept within the limits required by the regulations for hearing protection but, for those in the living chamber, the noise levels must be sufficiently low that sleep is not disturbed. In addition to the continuous monitoring of pressure, humidity, oxygen, carbon dioxide and temperature there should be daily monitoring for carbon monoxide and other gaseous contaminants. These might range from H_2S to complex hydrocarbons which are off-gassed from the paint on the walls of the chamber. The chamber should also contain video monitoring equipment enabling the supervisor to see what is happening, particularly in an emergency.

Lock-out

While for emergency use a greater excursion may be acceptable, it seems appropriate that routine excursions should be confined to the recommendations of the report by the Norwegian Petroleum Directorate[18] but should never be greater than 10 m.

It is also important that, during the excursion, the depth of each diver and the bell man is recorded continuously and on-line.

The bell should maintain the same oxygen level as in the living compartment but, during his dive, the diver may have a mixture containing 70 kPa (0.7 bar) oxygen with a limit of 170 kPa (1.7 bar) in his bail-out bottle.

The PO_2 in the breathing gas supply needs to be continuously monitored, as does the breathing gas

temperature for dives deeper than 150 m. The hot water temperature and flow to the diver needs to be monitored at the bell and, preferably, also at the diver's suit. For closed or semi-closed breathing apparatus the partial pressure of carbon dioxide also needs to be monitored. In all circumstances the diver's depth needs to be monitored continuously, preferably on a visual display.

Where the diver is to work in a welding habitat there are some additional considerations. The diver should have a welding mask which provides him with a breathing gas supply independent from that of the habitat itself. If a face mask interferes with his field of vision when welding, or if he wears a beard, a nose-clip and mouthpiece may be considered as an alternative. In all cases the provision of an oro-nasal or mouthpiece must be a personal issue.

The welding habitat should be monitored for carbon monoxide, ozone, fumes, oxides of nitrogen and shielding gases, using tubes and other available technology in the habitat. Samples for more rigorous analysis onshore should be taken if only to ensure that the habitat monitoring is effective.

Decompression

Decompression profiles are based upon much theory and many years of trial and error. For depths less than 180 m the NPD has specified the minimum decompression time. As yet there is insufficient evidence to be so dogmatic about decompression from greater depths and the rate should be based upon accumulated experience.[21] The decompression is usually conducted with a PO_2 of 0.5 bar (50 kPa).

Contingency requirements

Perhaps the most difficult task of a physician in support of a diving operation is to be prepared for the unforseen. The majority of accidents are foreseeable, but every time one occurs, it is different in detail. For each it may be enough to follow the agreed emergency procedures but occasionally additional problems require instant decisions and only a broad training and experience in diving medicine can provide the skills needed.

DECOMPRESSION ILLNESS

The treatment of manifestations arising from excursion dives or during saturation decompression is well established.[22]

HYPERBARIC EVACUATION

If all have to share the same hyperbaric evacuation unit, it is necessary for the divers at depth and those at some shallower depth undergoing decompression to meet at an intermediate depth. The decompression of the deeper team is limited by the hazard of decompression illness. An immediate upward excursion followed by an accelerated decompression in accordance with an agreed procedure can be combined with an elevation of PO_2 to 60 kPa (0.6 bar), the use of prophylactic drugs such as aspirin, and the maintenance of adequate hydration, each of which should reduce the risk of decompression illness. However, the greatest distance has to be travelled by the shallower team.

Notwithstanding bad experiences of HPNS by some divers, there is also considerable experience that a compression rate as fast as 1 m per minute is possible for many. In a real emergency, the nausea and vomiting which might occur should be accepted if this were to hasten the launch of the hyperbaric lifeboat. If HPNS led to an individual becoming unconscious he could be carried by others into the hyperbaric evacuation unit. This is a possibility but is thought to be a very low risk. It is therefore appropriate that the final decision upon compression rate should be left to the diving superintendent who is able to take into consideration the many other factors that are relevant to the particular emergency. There is much to be said in favour of the Norwegian rule that, for diving below 180 m there will be two independent pressure compartments for the evacuation of the diving personnel.

COINCIDENTAL ILLNESS AND INJURY

In a saturation diving system at least one chamber should be fitted out so that it is suitable for any emergency medical procedure that might need to be carried out within it. It must be possible to remove, with ease, fittings such as bunks which are not needed during the emergency. An operating table must be available which is capable of being locked into the chamber and fixed to the floor. The table should permit head down tilt, be of adjustable height and facilitate all round access to the patient. The special needs for lighting, heating, ventilators, monitoring, chamber cleaning, hand-scrubbing facilities, and suction have all been considered in a guidance note.[23]

The selection of the medical personnel to enter the chamber implies more than being part of an appropriate organisation and might, for instance, include the preferential selection of individuals who are small in size. The training of persons liable to enter a chamber in a medical emergency must include the use of non-volatile anaesthetics and the interaction between drugs and pressure.

All medications, disinfectants, topical agents, dressings and other materials which might be required for use in the chamber system must be reviewed for their potential contamination of the chamber atmosphere. The items that might be required to meet medical emergencies need to be checked and approved in advance.

IMPAIRED CONSCIOUSNESS, NEAR-DROWNING

The recovery and resuscitation of an unconscious in-water diver by his partner can never be an easy task. The recovery of the diver to the bell, the stowage of his umbilical, and hoisting him by means of his harness into the bell is slow. This is standard procedure but the problem which follows is cardiopulmonary resuscitation. This must be conducted at first in the upright position which is not likely to enhance circulation to the brain.[24]

THE LOST BELL

The diving bell and welding habitat must have storage for sufficient food, water and first aid supplies which are readily retrievable, even in the dark. If a bell or welding habitat becomes isolated underwater from its surface support vessel it will need supplementary atmosphere control, thermal support, food and drinking water. The odds are that, on recovery, there will be a need to re-warm the persons from the isolated bell or habitat and appropriateprocedures must be planned in advance.[25]

Health surveillance

It is generally agreed that periodical medical examinations of diving personnel should be conducted by appropriately trained physicians at set intervals. This is for both the safety of the diver in the water and for monitoring any adverse effects of diving upon health.[26] A basic examination is required at one year intervals or, if so required by the diving medical examiner, more frequently. The diver must also be examined for fitness to return to diving after any illness or injury, not just those due to diving. This is not easy. These examinations must be conducted by someone who is familiar with diving because, although meticulous guidelines are available from the authorities in Guidance Notes, there are so many borderline and other unusual circumstances that clinical judgement often has to be used.[27]

For diving deeper than 180 m, the depth at which, in Norwegian regulations, very deep diving begins, good medical, mental and physical fitness is essential. Conditions relating to in-water safety and the absence of conditions which would be difficult to treat when in saturation, are the primary targets for this examination but it can also be used to establish a baseline so that, at a medical examination one month after surfacing, reassurance can be given that there have been no significant changes. Experience has shown that particular attention must be given to any history of head injury because it seems that such persons are more predisposed to post-dive findings of minor neurological abnormalities. In saturation diving the longer term consequences must not be forgotten and periodical X-ray of the long bones is needed to monitor for the onset of aseptic bone necrosis.[28]

HEALTH MANAGEMENT

To prevent otitis externa and other skin infections there must be meticulous procedures for the divers' personal hygiene and for monitoring that the procedures are followed. These should include the bacteriological sampling of specified locations within the chambers and of the individual's themselves. Cleaning procedures for all the chambers and equipment must be prepared and, with the ear prophylaxis routines, be entered into the daily log.

In many dives a common feature is that divers lose weight. It is necessary to prepare a program for dietary management which ensures adequate nutrition.

Conclusion

Saturation diving has developed as a result of the work of many pioneers. As said once many years ago by an enthusiastic deep diver, "First we do it; then you doctors come along and prove that we did do it." But, in fact, the development of saturation diving owes much to the experience gained in many laboratory dives. Even so, many uncertainties remain and, to review the medical and research priorities in this field, would be to go through a larger and more detailed review than presented here. Within that scope, the greatest priority may not seem obvious but has been for many years a matter of urgency. With the diver as a very small percentage of any national workforce but one who holds the key, in a hazardous environment, to the completion of many important tasks, the urgency is to set up now a longitudinal study on every diver throughout his career and an international diving registry to monitor the potential for long-term effects. This was proposed 20 or more years ago by the European Diving Technology Committee and, if this had happened, we would now know many important answers. The government Health & Safety authorities throughout Europe must understand that, for divers, not just saturation divers but all professional divers, it is still not too late to begin.

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CIRCUM-RESCUE COLLAPSE: COLLAPSE, SOMETIMES FATAL, ASSOCIATED WITH RESCUE OF IMMERSION VICTIMS

F StC Golden, G R Hervey and M J Tipton

Introduction

It has long been known, though it is still not always appreciated, that victims of accidental immersion may collapse and may die during the process of rescue or shortly afterwards. We propose to call this syndrome "circum-rescue collapse". It was at one time thought that it was directly related to after-drop, the continued fall in core temperature that occurs after a subject is removed from cold water, which would imply that circum-rescue collapse is caused by hypothermia. In 1981, however, Golden and Hervey[1] showed that the after-drop can be fully explained by temperature gradients between deeper and superficial layers of the body and the physical laws of heat flow: its association with circumrescue collapse is, therefore, coincidental. They presented arguments that led them to suggest that the key event may be collapse of arterial blood pressure. Since then attempts have been made to test this hypothesis more rigorously, using the cold tank facility at the Institute of Naval Medicine.[2] Regrettably, higher priority tasks, the Falklands Campaign, ethical considerations and career moves have all impeded completion of the project. Meantime the hypothesis has become somewhat widely disseminated and it has prompted changes in rescue procedures. Since there are inescapable difficulties in completing the study to ideal scientific standards but the applications are important now, it seems timely to review the information available and to examine the conclusions for practical rescue procedures.

Historical review and anecdotal evidence

Sudden loss of consciousness or death during or following the rescue of immersion victims came into prominence during the Second World War because of the frequency with which it was encountered, but there are many earlier records of circum-rescue collapse and death.

In the 18th century James Lind[3] was the first to emphasise the danger of collapse in the postimmersion period. James Currie[4] observed the deterioration of his experimental subjects' condition post-immersion. In the 19th century Sir John Richardson appears to have been well aware of the dangers that faced him after rescue[5] and Varigny[6] also recorded death after rescue.

During the enquiry into the sinking of the *Titanic* in 1912, one witness, Fifth Officer H G Lowe, described how the crew of his lifeboat rescued four men from the water, and one subsequently died. Another witness, in a different lifeboat, Mrs E B Reynolds, described how two immersion survivors died in the boat shortly after rescue.

In the First World War the sinking of the *Lusitania* provided several accounts of survivors rescued alive subsequently dying on the rescuing fishing boats.[7] After the first battle of the Falkland Islands it was reported that most of 200 survivors from the German battle cruiser *Gneisenau* died on board one of the rescue ships.[8]

These lessons of the First World War evidently went unheeded and it was not until the Second World War that the problem of collapse and death after rescue from cold water was again highlighted on both sides in the conflict. For the Allies. MacDonald Critchley[9] emphasised the danger for shipwreck survivors, and Wayburn[10] recorded some instances of death after rescue among ditched USAF personnel. In Germany Grosse-Brockoff[11] reported how men rescued alive from Norwegian fjords subsequently died from "sequelae of hypothermia". Also on the German side the problem of "Rettung Kollaps" ("Rescue collapse") came to official notice during the Battle of Britain: the highly efficient German Air Sea Rescue Services recovered a substantial proportion of ditched Luftwaffe and RAF personnel, only to observe many die 20-90 minutes after rescue. The investigation of this problem was offered as justification for the notorious experiments at the Dachau concentration camp.[12]

It is of great interest that in the later stages of the war the German lifeboat service noted, albeit by serendipity, that rescuing immersion victims by manoeuvring a ladder beneath them and lifting them horizontally improved their chances of survival, compared with lifting in a vertical posture or requiring them to assist in their own rescue.[13]

The incidence of post-rescue collapse is difficult to establish as death certificates generally only the terminal event. Although pulmonary changes are usually found post-mortem after drowning, their absence does not decide the diagnosis since it is estimated that 10-20% of drownings are "dry drownings".[14,15]

Some deaths attributed to dry drowning may however, have been caused by other mechanisms. Immersion victims whose history suggests that the airway was clear of the water at all times are sometimes inexplicably classed as victims of drowning! There are no post-mortem findings that can positively identify cold as the primary cause of death. Reliable statistics are therefore an impossibility.

One of the few numerical indications of the incidence of post-rescue problems comes from McCance el al.[16] In their survey of merchant shipping losses during World War II they found that, in a series of 289 shipwreck survivors, of the 160 rescued from water at a temperature

of 10°C or less, 17% died within 24 hours of rescue (the questionnaire did not ask for finer timing). Of the 109 rescued from water above 10°C, none died after rescue. Although the cause of the post-rescue deaths was not known the association with low water temperature was thought to be significant.

MacNalty[17] in his history of the medical services in war, suggests that the problem may be even greater and not limited to very cold waters. He writes:

> "A not uncommon feature, observed in other waters as well as in the Arctic, was that many survivors who had managed to get themselves to the point of being rescued from the sea collapsed when safety was within reach, and required to be handled in the same manner as those who had been helpless while still in the water."

Keatinge's excellent account[18] of the sinking of the *Lakonia* off Madeira in water at approximately 18°C, also shows that post-immersion collapse is not confined to very cold water. He reports that, of the 15 dead victims on the *Montcalm*, the first rescue ship on the scene, most were thought to have been rescued alive.

Post-rescue death is also not limited to subjects who were already unconscious at the time of rescue. Matthes[19] describes how ditched aircrew who had been in the sea for relatively short periods and were fully conscious and able to assist in their own rescue, lost consciousness after rescue and in some instances died. Ortzen[20] describes a survivor from the passenger vessel *Vestris*, sunk in 1928, collapsing and dying shortly after climbing into a lifeboat.

Other cIassic instances of post-immersion collapse include the survivors of the *SS Empire Howard.*[21] The captain, Capt. Downey, reported: "Everyone was conscious when taken out of the water but many lost consciousness when taken into the warmth of the trawler. Nine (out of 12) died shortly after being rescued." Similarly nine survivors from *HMS Kite,* sunk on Arctic convoy in 1943, were rescued by a sister ship, the *Keppel*: all were conscious when rescued but only five survived.[22]

Documented examples of post-immersion collapse and death in swimmers can be obtained from several sources,[23] including the records of the British Long-Distance Swimming Association. Hardwick[24] gives an account of a swimmer collapsing and losing consciousness after "forcible" removal from the water after 11 hours of swimming in the Irish Sea. Lloyd[25] describes the sudden collapse and death of a speleologist who had been rescued in a hypothermic condition from a flooded cave, and had, shortly before, been enjoying a bar of chocolate.

Some recent accounts give a more detailed picture of various forms of circum-rescue collapse. Golden[26] described two cases of teenage boys dying from ventricular fibrillation (VF) in hospital after a boating accident. Although body temperatures were not recorded the circumstantial evidence and the condition of other survivors left little doubt that the victims had been hypothermic. In another accident investigated by one of the authors a 54-year old teacher was canoeing on Lake Coniston with two young female passengers. The canoe capsized and the three were immersed in water at 12°C for about ten minutes, during which time they held on to the upturned canoe. A rowing boat rescued the two girls but the teacher proved too heavy to be helped aboard and was towed ashore. On arrival ashore he complained of cold and numbness but was rational and coherent. His wet clothes were removed and he was massaged. A short while later "he suddenly went rigid and died".

In June 1979 *HMS Jupiter* was involved in the rescue of survivors from the *MV Iris* which sank in the Bay of Biscay in a force 8 gale. The water temperature was 15.5°C, air temperature 12°C and the sea state 6 with a 25 foot swell. Rescue was effected in very difficult circumstances with the Ashanti rig (adapted from a helicopter lifting strop) and with the ship rolling through an angle of 45°. Of the 12 survivors rescued, one "died within minutes of rescue" and one "died halfway up the ship's side".

During the 1979 Fastnet race, in water at 15-16°C, three of the 15 fatalities among the competitors (20%) occurred during rescue: one in the course of lifting by helicopter and two while the subjects were trying to climb up a scrambling net.

Three further anecdotal accounts of helicopter rescues are worth putting on record:

i) The pilot of a light aircraft flying from Sweden to Denmark in winter, ditched in the sea within sight of the lights of Copenhagen. The pilot scrambled on to the outside of the cockpit to await rescue. He remembers the rescue helicopter flying overhead and the winchman being lowered and putting the strop around him. His next recollection is of awakening in a hospital bed in Copenhagen.

ii) A young woman rescued from the sea off the coast of Cornwall in summer remembers the helicopter winchman putting the strop around her and the lift commencing. She regained consciousness some time later while lying in the helicopter cabin.

iii) A sailor who was washed overboard from an RN frigate off North Cape in 1985 (in sea temperature 5°C, air temperature -2°C and sea state 6) spent 8 minutes in the water before being rescued by helicopter. This was done with a single-man strop. He was successful on the third attempt in putting the strop around his body. His next recollection was of regaining consciousness as he was brought into a horizontal posture to be manoeuvred

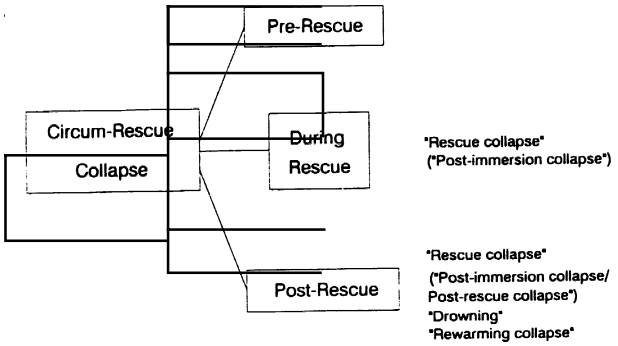

Figure 1. Stages of Circum-rescue collapse and their relationship to previously used terms.

through the helicopter door. He subsequently made an uneventful recovery.

Insights gained

Consideration of the historical and anecdotal evidence suggests that three stages of rescue can be distinguished and each may be associated with particular risks. They are: just prior to rescue (pre-rescue): during the process of rescue (during rescue): and just after rescue (post-rescue). These stages and their relationship to previously used terms are shown in Fig. 1.

Among the possible causes of death, *drowning* as a consequence of inhaling fluid must clearly be considered. It may be the major factor in victims whose airway was intermittently submerged and who gradually lose consciousness. The delayed hypoxic effects of drowning have been well reviewed, for example by Modell.[27] *Hypothermia* will be an important factor in those who have suffered prolonged immersion with an airway clear of the water.

However, the *sudden* deterioration in the victim's condition so often described, with rapid loss of consciousness and sometimes sudden death, suggests a more acute cause than either the progressive cerebral hypoxia of drowning or the loss of cerebral function as body temperature falls. We shall suggest that *collapse of arterial pressure* is much more consistent with the events observed. This suggestion, which would apply at any body temperature, is quite distinct from the well-recognised concept that VF may be the terminal event in those suffering from profound body cooling.

"Rewarming collapse" has been reviewed elsewhere,[28] A suggested mechanism for this is a failure to maintain arterial pressure when extreme vasoconstriction is released during rewarming in subjects whose blood volume has been depleted by prolonged vasoconstriction and cold diuresis. This may explain some postimmersion deaths, particularly those that occur during rewarming procedures. It clearly cannot account for cases in the pre-rescue and during rescue categories. The mechanism of sudden collapse and death at these earlier stages may also be important post-rescue. It merits detailed study.

Pre-rescue collapse

MacNalty"s account[17] of survivors who, at "the point of being helped from the sea collapsed when safety was within reach", implies sudden deterioration in the condition of some victims immediately prior to rescue. Other accounts support this picture but it is inevitably difficult to establish the precise timing and thus the distinction from cases where rescue had begun. Pre-rescue collapse has not previously been distinguished and it is obviously difficult to evaluate hypotheses as to its mechanism. It is, however, worthy of consideration. We would again argue that the suddenness suggests a cardiovascular mechanism.

In hypothermia, prolongation of the isometric phase of myocardial relaxation and increased viscosity of the blood reduce coronary blood flow. McConnell et al.[29] showed that coronary vasodilation capacity (i.e. the maximum coronary flow achievable at any coronary perfusion pressure) is reduced in dogs in profound hypothermia (core temperature 28°C). This makes the heart more liable to ischaemia if perfusion pressure falls. This is compounded by the leftward shift of the oxyhaemoglobin dissociation curve.

McConnell's group examined the effects of reducing coronary (i.e. diastolic aortic) pressure from 100 to 50 mm Hg in 11 dogs at body core temperatures of 37 and 28°C. At normal body temperature the reduction in coronary perfusion pressure was compensated by the vasodilation reserve capacity and endocardial perfusion remained adequate. At 28°C the lowering of perfusion pressure led to a reduction by 44% in subendocardial blood flow and redistribution of blood away from subendocardial tissue; this resulted in ischaemia and changes in the S-T segment of the ECG. If perfusion pressure was immediately restored or if the heart was rewarmed, subendocardial perfusion was restored and the ECG reverted to normal.

Catecholamines, particularly noradrenaline, have been shown to raise the threshold for VF in hypothermic dogs.[30] This protective effect was attributed to a rise in blood pressure rather than to a specific effect on the heart. This was confirmed by Angelakos and Daniels,[31] who showed that infusion of noradrenaline or dopamine reduced the incidence of VF in hypothermic dogs from 60 to 20%, and the LT_{50} from 19.3 to 12.5°C.

There is ample evidence that catecholamine secretion is greatly enhanced in humans immersed in cold water.[32-35] Possibly that in the pre-rescue phase awareness of imminent rescue can reduce sympathetic tone or catecholamine secretion, and thus their protective effect.

Collapse during rescue

HYPOTHESIS 1: THE AFTER-DROP

The experimenters at Dachau reported deaths occurring after victims had been removed from cold water, and attributed these to the after-drop in core temperature which they had also observed.[12] They attributed the after-drop to return of cold blood from periphery to core as the peripheral circulation reopened on rewarming. They observed that the incidence of collapse and death roughly coincided with the nadir of the after-drop. On this explanation if a victim was, for example, within 2°C of a lethal core temperature on removal from the water, the after-drop would bring the core temperature to the lethal level.

This explanation was widely quoted and accepted.[36,37] The Dachau experimenters, however, made no effort to differentiate between early and late deaths. While their explanation might be credible in the context of their experiments, it is not consistent with accounts, anecdotal but not isolated, of individuals collapsing while assisting in the rescue process. To be able to do this they must have been conscious and active, which means that their core temperatures must have been above 30°C and probably above 33°C: the after-drop is typically 1-2°C, and the Dachau records show that death usually occurred at around 25°C.

The after-drop hypothesis for rescue collapse proposed by the Dachau experimenters went unchallenged for many years, until Golden and Hervey[1,38] showed that it was not necessary to invoke any physiological events to explain the after-drop. An after-drop is an inevitable consequence of the physical laws of heat flow in a structure, such as the body, that comprises a heat-producing core surrounded by an insulating shell, if it is immersed in water at a temperature at which heat loss to the water exceeds heat production, and then moved to a warmer environment. Webb[39] subsequently confirmed this.

Although indirect evidence for a possible "cold bolus" effect in some circumstances has been reported,[40] Golden described experiments with pigs in which the recording system was shown to be capable of detecting even a small cold bolus of blood returning to the heart:[41] no evidence of any cold bolus was found when rescue and rewarming were simulated by transferring the anaesthetised animals from cold to warm water.

In any case, it is not clear how a cold bolus of blood could account for the anecdotal reports of a transient loss of consciousness seen in some survivors.

HYPOTHESIS 2: COLLAPSE OF ARTERIAL PRESSURE

An alternative explanation would therefore seem to be required for rescue collapse. It is conceivable that a similar mechanism to that outlined for pre-rescue collapse may also contribute to rescue collapse, particularly in those individuals who have only been immersed for a short period of time. The reported duration of immersion in these victims differs little from that of many subjects in laboratory studies in whom collapse has not been recorded. This leads to the suspicion that the physiological consequences of the greater levels of anxiety, which must be present in the real situation, contribute to the subsequent collapse.

Following prolonged immersion a number of other factors come into play at the time of rescue that, alone or in combination, clearly can cause sudden and dramatic changes in cardiovascular function. We shall argue that their likely effects are consistent with the pattern of circum-rescue collapse as seen in the anecdotal evidence. It is also of interest that the central concept of failure to maintain arterial blood pressure can explain loss of consciousness followed by recovery: this may be due to inadequate cerebral oxygenation, recoverable when the situation improves (in contrast to the irreversibility of VF).

The factors which have been identified on the basis of the anecdotal evidence include:

(i) immersion; (ii) removal from the water; (iii) the restoration of the full influence of gravity; (iv) hypovolaemia; (v) hypothermia; (vi) physical effort at the time of rescue. Although listed separately they are linked and when present in combination are mutually reinforcing.

When a subject is immersed in water, the blood in extrathoracic vessels is surrounded by a medium of approximately the same density and becomes effectively weightless. Hydrostatic compression reduces venous capacitance in the dependent parts of the body. The increase in ambient pressure with depth causes a negative intrathoracic pressure typically around 15 mm Hg during upright, seated, head-out immersion in thermoneutral water:[42,43] this effect is greatest when the body floats vertically and least when it floats horizontally in the water.

These changes have characteristic physiological consequences. The most significant is redistribution of blood to the upper part of the body. This takes place almost immediately on immersion to the neck in the upright position and is the major physiological change in immersion in thermoneutral water. Central blood volume increases by up to 700 ml.[44,46] In cold water vasoconstriction further increases the shift of blood from the peripheral circulation to central vessels.

On immersion, removal of the gravitational effect and its replacement by a positive hydrostatic pressure gradient reduces the demand for work by the heart, particularly in supplying the cerebral circulation, while the shift of blood to central vessels together with the hydrostatic effect facilitates cardiac filling. Cardiovascular changes observed in man during resting, headout, up-

right immersion in thermoneutral water include: increase by 12-18 mm Hg in right atrial pressure;[44,45,47] increase by 250 ml in diastolic filling;[44] and increase in cardiac output by 32-66%.[42,45,47] The increase in cardiac output was described as attributable to increase in stroke volume, there being no change of consequence in heart rate*.

The shift of blood to central vessels during immersion evokes diuresis[48-50] and eventually hypovolaemia. The body appears to sense the initial over-filling of the central vessels and to interpret it as indicating excess total blood volume. Subjects immersed upright and headout in thermoneutral water may secrete 350 ml urine per hour.[51] In cold water, peripheral vasoconstriction exacerbates this effect. If severe hypothermia occurs the direct effect of cold diminishes tubular reabsorption in the kidney; shifts of fluid from intravascular to interstitial and intracellular compartments further deplete circulating volume. This may cause severe hypovolaemia in immersion victims.[52]

When a subject is lifted out of water after prolonged immersion, the hydrostatic assistance to circulatory function is lost and the full effects of gravity are again applied to the body. In the usual vertical posture, more work is required to lift blood to the head; at the same time gravity-induced pooling of venous blood in the lower limbs diminishes venous return. Blood volume may be severely depleted. In normal circumstances, reflex adjustments act to maintain cardiac output and arterial pressure: in the combination of adverse circumstances that occur in rescues, this may be impossible.

There is evidence from experiments with animals that baroceptor reflexes are impaired by moderate hypothermia.[53] This would add an important harmful effect to the factors already described. Even if the reflex controls are working normally, they call for more cardiac work and so for greater oxygen supply to the myocardium. Increased heart rate decreases the time for coronary filling and this may not be tolerated by a cold heart. Increased blood viscosity further increases the demand for cardiac work and reduces coronary perfusion.

***Footnote.** A physiologist might point out that normally events in the heart, other than failure to pump out the volume of blood returned to it, cannot alter cardiac output other than momentarily. Output is adjusted to match venous return, and this, as long as arterial pressure is constant, is determined by peripheral resistance: output in excess of venous return would simply deplete central reservoirs and then cease. In adverse circumstances, however, maintenance of cardiac output and arterial pressure are under threat; the inter-relationships become more complex and the heart's action becomes limiting. Factors that facilitate filling are then valuable in themselves, and increase myocardial contractilily through the Frank-Starling mechanism; output, arterial pressure and venous return are improved together.

Yet another factor that can greatly increase the demands on the circulation at a time when it may already be difficult to meet them, is the physical effort involved in attempts to assist in rescue by swimming, climbing and so on. Although the "muscle pump" should fractionally assist the heart, opening up of the potentially huge vascular bed in muscle calls for correspondingly increased work by the heart and in turn for oxygen supply: if the heart cannot do the extra work arterial pressure inevitably falls. Pre-existing coronary disease clearly adds another factor that can threaten myocardial oxygen supply in the circumstances of rescue.

We consider that the evidence and arguments presented strongly support the hypothesis for the mechanism of circum-rescue collapse first advanced by Golden[1,41] as an alternative to the after-drop and cold bolus mechanisms. This postulates that the sudden deterioration in a survivor's condition is caused by collapse of arterial blood pressure.

In summary the circulatory collapse can come about as a result of a number of factors: primary among these are loss of hydrostatic assistance to venous return and reimposition of the effects of gravity; hypovolaemia; increased blood viscosity; diminished work capacity of the hypothermic heart and reduced time for coronary filling; dulled baroceptor reflexes; unmeetable demands to perfuse skeletal muscle; psychological stress and pre-existing coronary disease.

Death probably occurs as the result of inadequate blood supply to heart or brain. Myocardial ischaemia is likely to cause irreversible VF. The outcome of cerebral ischaemia may be more variable. It may account for cases where the subject recovers after a spell of unconsciousness: it may be significant that such recoveries seem to occur once the subject is horizontal and at rest. Alternatively recovery from cerebral ischaemia may not take place. There is a further possibility that those who lose consciousness while climbing nets or ladders will fall back into the water and drown. The variability in the incidence of rescue collapse may be explained by the variation in the susceptibility of individuals to cooling and/or differences in the state of health of their coronary arteries.

Experimental evidence

There are severe problems in subjecting such a hypothesis to experimental test. Real rescues are diverse and uncontrolled and there is no possibility of making the necessary measurements. Experiments on human subjects are heavily constrained by ethical considerations and the experiment described below may represent about the limit to what can be done. Animal experiments were valuable in investigating the mechanism of the after-drop but the requirements for studying cardiovascular events are more

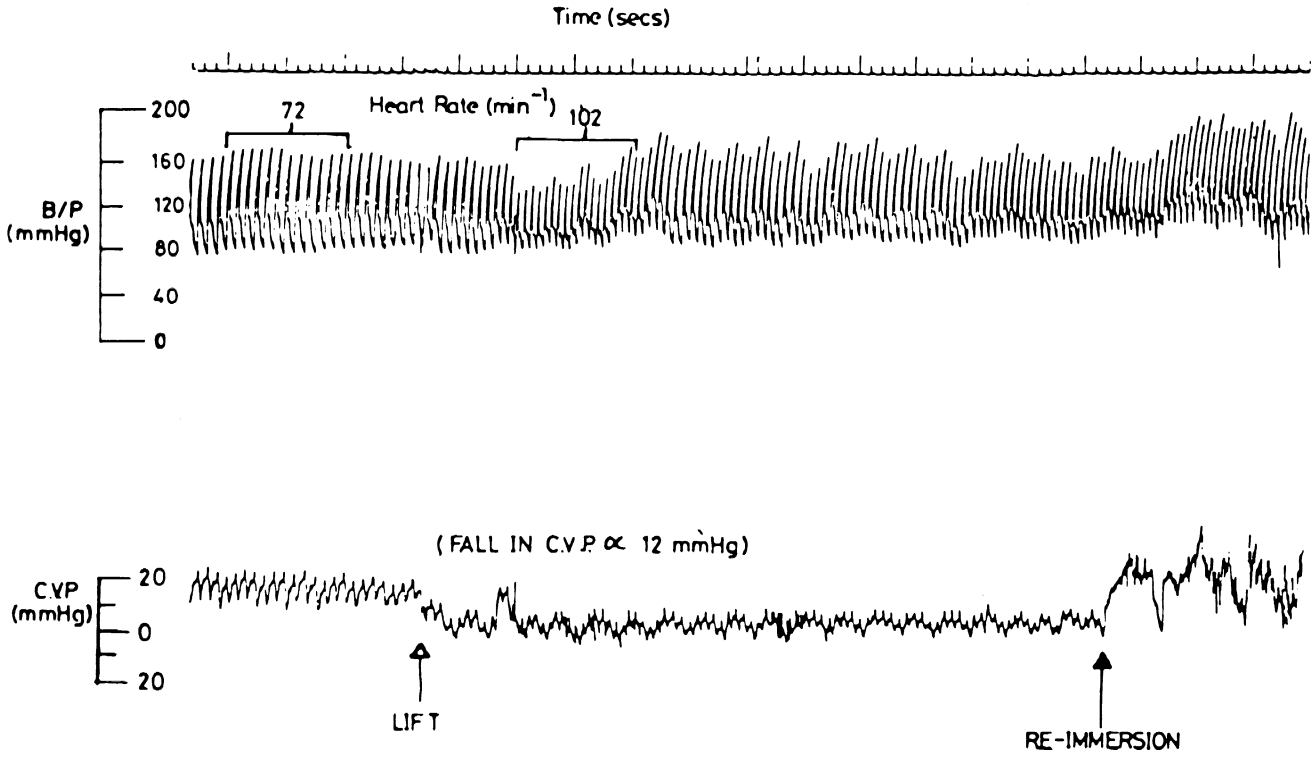

Figure 2. Changes in the aortic (B/P) and central venous pressure (C.V.P.) during vertical lifting by helicopter strop from water at 15°C (n= 1).

critical; a species with similar reflex controls and postural habits to man would be needed, which suggests one of the higher apes, with the practical difficulties that would entail.

Nevertheless it was possible to demonstrate the key cardiovascular effects of removal from water in the unpublished pilot study in man shown in Fig. 2. The subject, dressed in shorts, was lifted vertically from water after immersion for 30 minutes at 15°C. The lift was performed with a single helicopter strop. Aortic and central venous blood pressures were recorded throughout immersion and lifting.

As Fig. 2 shows, during the lift central venous pressure fell by approximately 12 mm Hg. This we interpret as the consequence of removing hydrostatic support and restoring gravity. Aortic pressure fell initially. Heart rate increased by 30 beats per minute (40%). This is part of the normal response to fall in systolic pressure. In this fit, normothermic individual arterial pressure was maintained after the brief fall: if the normal regulatory responses were overwhelmed or impaired, however, the situation would be very different.

In view of the implications for rescue techniques, studies have been made of the effects of varying these in simulated rescues of human subjects. In an unpublished experiment by Golden, 17 subjects wearing swimming trunks were lifted from cold water, vertically by the standard single lifting strop, and more nearly horizontally by a double strop (Fig. 3). The mean changes in heart rate for each method of lifting are shown in Fig. 4. During vertical lifting mean heart rate increased by 16%: this change was highly significant. Horizontal lifting caused only a small, non-significant increase. The change in heart rate is an indication of the regulatory response the heart was called upon to make. In situations where the capacity to respond is diminished, horizontal lifting would clearly be preferable.

Golden's work has been extended by Tipton and Vincent[2] to cover subjects wearing normal and protective clothing. Figure 5 shows the mean changes in heart rate. In all clothing assemblies heart rates were again found to increase significantly more during vertical than during horizontal lifting.

In interpreting these results it must be borne in mind that the clothing assemblies affected body temperature. In water at 5°C, when normal clothing was worn mean body core temperature had fallen by 1.1°C at the time of lifting; in the wet suit by 0.7°C; and in the dry suit by only 0.2°C. The difference between vertical and horizontal lifting was greatest when subjects wore the dry suit and thus had the highest core temperatures. This may possibly been because peripheral venous capacitance was reduced when they were cold, and this decreased the scope for gravita-

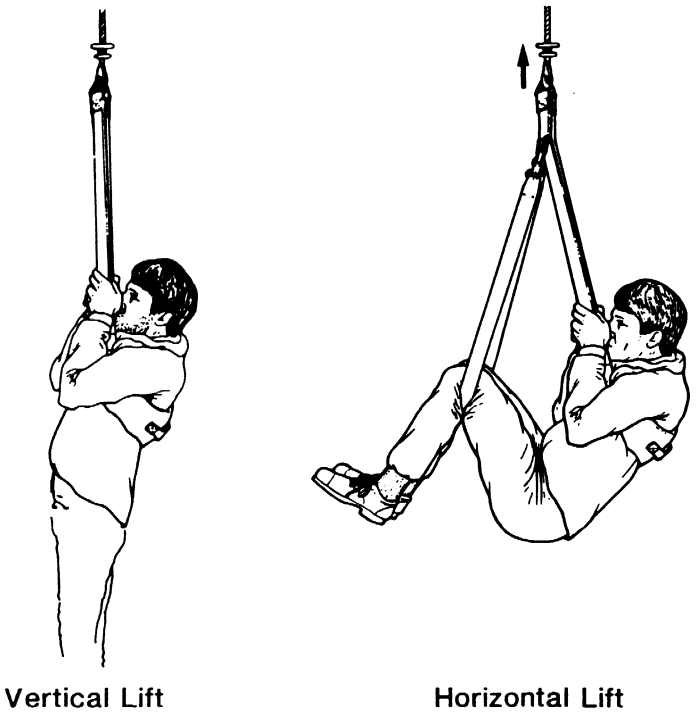

Figure 3. Vertical lift (single strop) and horizontal lift (double strop).

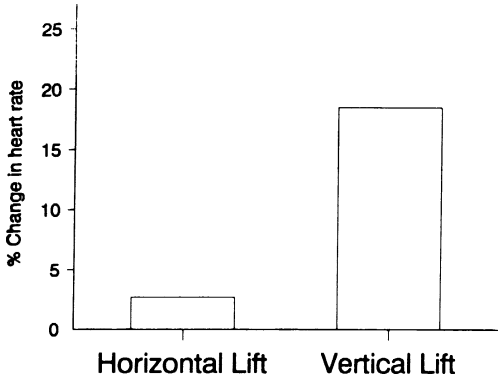

Figure 4. Average percentage changes in heart rate after one minute horizontal and vertical lifts (n= 17). Subjects wearing swimming trunks only. Lifts after 45 minutes immersion in water at 15°C.

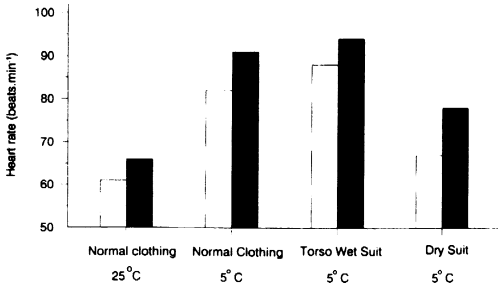

Figure 5. Average heart rates during one minute lifts following immersion in water at different temperatures and with different clothing (n= 18). Normal clothing = cotton underwear, woollen socks and pullover and a cotton boiler suit; Wet suit = normal clothing+a trunk and arms wet suit; Dry suit =normal clothing+a whole body watertight suit leaving only the hands (wet suit gloves worn) and face exposed. White columns = horizontal lift; black columns =

tional redistribution of blood. Only in the dry suit were the subjects horizontal while in the water, due to air in the suit, so there was no hydrostatic element in this situation and it should follow that the change in heart rate reflected only the baroceptor response to the changes in posture.

Practical conclusions

It follows from the suggested mechanism for circum-rescue collapse that the transition from water to air is likely to be least traumatic in subjects who are lifted horizontally after floating horizontally during immersion. The effects of loss of hydrostatic support and exposure to gravity will be most severe in those lifted vertically after floating vertically.

Protective gear tends to encourage horizontal flotation: this applies to aircrew wearing immersion coveralls, submariners in Submarine Escape Immersion Suits and to survivors lying on the floor of a liferaft. It also prolongs the possible duration of survival. During a lengthy flotation, however, a subject in protective clothing may become profoundly hypovolaemic as well as, eventually, hypothermic. Thus in real life, subjects who are found floating horizontally may be at as great or greater risk of circum-rescue collapse than those floating vertically, who probably have less protection from cold and have been floating for a shorter length of time. The latter, however, are the more at risk from loss of hydrostatic support.

The conclusion is absolutely clear that removal from water in the horizontal posture is preferable, in all circumstances, if it can be achieved. It also appears very likely that any demand for physical effort on the part of the victim at the time of rescue carries a risk of precipitating collapse and death. With the inevitable proviso, circumstances permitting, immersion victims should be handled with the utmost gentleness and as the potentially critically ill patients that they are. We fully endorse and approve the efforts now being made in many quarters to modify rescue techniques to meet these principles, and so improve the prospects for survival.

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