

# SPUMS JOURNAL

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South Pacific Underwater Medicine Society Incorporated

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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. 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 sickness (DCS) can thereafter be referred to as DCS.

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Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references which should be limited to 5 per letter). Accuracy of the references is the responsibility of authors.

### 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.<sup>1-2</sup> 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: 17-23

### Computer compatibility

The SPUMS Journal is composed on a Macintosh using Microsoft Word and PageMaker. Contributions on Macintosh discs, 400 or 800 k, preferably in Microsoft Word 3, or in any programme which can be read as "text" by Microsoft Word 3, save typing time. They must be accompanied by hard copy set out as in **Minimum Requirements for Manuscripts** above.

### Consent

Any report of experimental investigation on human subjects must contain evidence of informed consent by the subjects and of approval by the relevant institutional ethical committee.

### Editing

All manuscripts will be subject to peer review, with feedback to the authors. Accepted contributions will be subject to editing.

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### Address for all Contributions

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Telephone enquiries should be made to Dr John Knight (03) 417 3200, or Dr John Williamson (08) 2245116.

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Information may be sent (in confidence) to:

Dr D. Walker

P.O. Box 120, Narrabeen, N.S.W. 2101.

## EDITORIAL

### *The Editor's Offering*

In this issue we again have an editorial from the President of SPUMS. This has been prompted by letters to the Editor and other matters. Turn the page to find out what he says about SPUMS and the diving industry.

Our first original paper is an excellent review of carbon monoxide poisoning and the use of hyperbaric oxygen (HBO) by Paul Mark from Fremantle, written for the Diploma of Diving and Hyperbaric Medicine and therefore published here. Further information is to be found among the abstracts from the Undersea and Hyperbaric Medical Society's 1991 meeting. These are now grouped under various headings.

Carl Edmonds and Roy Damron demonstrate that Hawaiian scuba divers die from much the same causes as do other Americans and Australians and New Zealanders. Again far too large a percentage of the deaths were in people who had completed fewer than 6 dives with almost half of those dying during their basic open water course. Bob Halstead again contributes food for thought with his survey of 265 of his clients. There is no doubt that they are experienced divers with an average of 885 dives each. His divers tend to dive alone, which is what most divers who buddy with a photographer actually do, but these divers admit their "crime". A third had to make an emergency ascent because they had no air. A third of those who dived with a buddy had to make an emergency ascent because the buddy had run out of air. The favourite method of emergency ascent, used by half those who had made an emergency ascent, was a swimming ascent.

Jeff Wilks and Lindsay Christie offer more information about diving incidents. It would appear that far too many people who go diving on the Great Barrier Reef are quite inadequately trained and would be in trouble but for the kindly care of observant divemasters.

Alison Drewry and Des Gorman describe a protocol designed to test if oxy-helium at less than 2 bar is as effective a treatment for decompression illnesses as oxygen at 2.8 bar. This has been prompted by the much less than satisfactory results of the conventional (oxygen at 2.8 bar) treatment. It is only long term follow up, by which we mean after discharge from the initial treatment, that this sad failure rate has been discovered. Many of the symptoms are attributable to central nervous system dysfunction, giving rise to personality changes and motor and sensory losses. Here is an opportunity for co-operation between hyperbaric units.

Carl Edmonds, whose idea SPUMS was, was the first President of SPUMS. His letter to the Editor draws attention

to the difficulties the Journal, and the Society, faces in offering an opportunity for all to have a say. There is no desire to implement Editorial censorship, so as long as letters are not libellous they normally get published. Unfortunately some people seem to be unable to modify their views in the face of evidence of their being wrong, and this leads to friction or worse.

Bill Hamilton's reply to Des Gorman's last editorial is a very clear exposition of the way that experts get involved in diving problems and beyond. Bill is a very experienced and successful calculator of decompression tables and a respected authority. His emphasis is much the same as Des Gorman's, that divers must have the appropriate training and know the risks they undertake before diving. This applies to air as much as to any other gas.

John Parker's two letters are of interest. Painless grade 3 barotrauma is an unexpected finding. Coming across affordable equipment to do a saline provocation test is well worth bringing to everyone's attention.

Glen Egstrom's papers from the 1991 Annual Scientific Meeting are edited transcripts and suffer from not being illustrated by his dazzling slide display. We hope that we have caught all the messages. The rates of ascent that a diver in an inflated large size buoyancy jacket can attain in just under 3 m are astounding and frightening. That the experimental divers came out of the water up to the waist is an impressive achievement of deliberately mismanaged buoyancy. All divers need to learn to manage their buoyancy with skill is the message of this paper and there is plenty of advice about the difficulties of buoyancy control and how to overcome them. The safety stop has a rate controlling effect on a diver's ascent rate as well as providing a hedge against decompression illness.

The paper on investigating diving deaths presents an on-the-job view of the process that leads to papers like that on the Hawaiian diving deaths. The Californian system is, as would be expected of anything Glen Egstrom had a hand in setting up, sensible and thorough, and could well be adopted by all law enforcement bodies dealing with diving deaths. The stupidity of many, and lack of forethought about their equipment of others figure largely in causing the Californian deaths. Everyone is guilty of lack of forethought at some time. How many of his audience had never given thought to how to disconnect the scuba feed from a rapidly inflating compensator before Glen Egstrom mentioned it in the Maldives. Perhaps Australians maintain their equipment better than others or have just been plain lucky that very few defective BCs have reached these shores.

## *Lambada Dancing on a Tightrope*

The relationship between SPUMS and the recreational diving industry is under considerable threat and strain. It will take both good luck and good management to avoid a return to the hostilities of 10 years ago. Indeed, some of SPUMS' most prestigious members believe that the SPUMS Executive Committee has already capitulated to the instructor organisations, and in particular to PADI.

This view of a capitulation may seem extreme, but is not difficult to see how and why it has arisen. Firstly, SPUMS has given the recreational divers access to our Journal and some of their published views have not been complimentary about diving physicians. Secondly, one of "them", Drew Richardson (PADI Vice-President in charge of training), has been admitted to full SPUMS membership. This has even upset some of the other training agencies. However, it must be pointed out that only PADI has ever bothered to "turn-up" at our annual scientific meetings. Thirdly, all of the recreational diving groups involved in the creation of the Australian Standard for recreational diving voted against any form of requisite medical examination of candidates for scuba diving instruction! The need for such examinations is seen as essential by SPUMS; and, justifiably so from our published surveys on the inadequacy of fitness assessments performed by physicians who have not been trained in diving medicine and on the poor selectivity of medical history questionnaires as a sole screen prior to diving instruction. The support these groups received for their subsequent vote against the need for trained physicians from the Australian Medical Association was both disappointing in the extreme and a clear demonstration of why so many medical practitioners do not belong to this "out of touch" organisation. Things are no better in New Zealand. Although they do insist on pre-diving "medicals", the New Zealand Underwater Association was not prepared to even encourage scuba diving candidates to be examined in this context by a physician with appropriate training, a remarkable and sad stance from an organisation that had previously shown the way with the funding of both courses for medical practitioners in diving medicine and the New Zealand Divers Emergency Service (DES). The fourth reason that suggests a capitulation has been the failure of SPUMS to encourage and develop widespread funding support from recreational divers for the Australian DES. Since the collapse of the Victorian Division of the National Safety Council of Australia, the funding of the DES has been precarious. The Federal Australian Government has been a part-funder, but has just announced that it is withdrawing all support next as DES does not conform to their definition of a worthwhile group (perhaps a just reward for trying to establish a volunteer self-help system rather than screaming for full Government funding in the first place). While NAUI, PADI and NASDS (formerly FAUI) have provided some funding, this has been interrupted on occasions such as when PADI withdrew funding from DES in the belief that the latter was

marketing DCIEM decompression schedules in competition to the PADI RDP and wheel. As a tangential issue, to illustrate why diving physicians do become cynical about recreational diving instructor bodies, NASDS is now marketing DCIEM schedules in Australia in direct competition to the Royal Adelaide Hospital. The hospital believed (perhaps naively) that it had an exclusive contract for such Australian distribution and donated all of its profits from these sales to diving medicine research. When NASDS and the hospital could not agree on terms, research funds are precious, NASDS simply entered into a separate agreement with the Canadian supplier. Even if this agreement is subsequently found to be legally reasonable, it will inevitably cause conflict between diving physicians and recreational divers. On behalf of all those who rely on such sources of diving medicine research funding, SPUMS would like to thank NASDS for their short-sightedness. To be fair to NAUI, PADI and NASDS however, it must be pointed out that organisations such as SSI and BS-AC did not even bother replying to requests from the DES for funding. The only other consistent funders of the DES have been SPUMS and the Royal Adelaide Hospital in Australia, and the New Zealand Underwater Association and the Royal New Zealand Navy in New Zealand. The recent rise in our annual subscriptions was contributed to, in part, by the assumption that SPUMS could continue to support DES without a subscription rise in 1991.

Given this behaviour, why should SPUMS bother? Why should SPUMS try to encourage dialogue and to reduce the paranoid conviction widely held in the recreational diving industry that SPUMS is trying to "take over"?

There are two primary reasons. Firstly, with the possible exception of club-based organisations such as BS-AC, the orientation of instructor bodies is, by necessity for survival, commercial. In contrast, SPUMS has no such commercial orientation and remains an "unpolluted" guardian of safety. Simply, SPUMS has an important role in maintaining standards of diver safety and health. Secondly, this role can only be exercised through dialogue. This year, PADI alone will certify more than 600,000 new divers world-wide. It follows that Drew Richardson, as Director of Training for PADI, has more direct influence on diver education and hence safety than SPUMS, DES, the Divers Alert Network (DAN), the Undersea and Hyperbaric Medical Society (UHMS) and the European Undersea Biomedical Society (EUBS) all combined.

The Executive Committee has never wavered from the purposes of the Society which are printed on each inside front cover of our Journal. From these arise the absolute requirement to promote safe diving practices. In some instances, safe diving requires a lot of supervised training; this takes time and to some degree conflicts with the eco-

conomic imperatives of running a competitive recreational diving enterprise. It is inevitable then that at times SPUMS will disagree with the practices of these training organisations. This is appropriate and should not consistently lead to sustained conflict.

Contrary to some current claims from both sides of the debate, Drew Richardson is not the first non-medico to become a full member of SPUMS. From its foundation, SPUMS has had non-medical full members. John Pennefather, the foundation Treasurer, Glen Egstrom and Peter Bennett are good examples. The requirement was and is a commitment to diving medical research.

Given the prevalent paranoia, cynicism and hostility (as manifested during recent code of practice discussions in Queensland), SPUMS' attempts at maintaining a balanced

position and of sustaining communications is very much like lambada dancing on a tightrope. The Executive Committee believes that all our members and associates must have access to our Journal, regardless of the polarity of their opinions. However, on some issues, regardless of the debate, there will be no compromise by the Society on such matters as the obligate need for all scuba diving training candidates to have a medical fitness assessment performed by a trained physician. Finally, we will welcome active participation in our annual scientific meeting (1993 - Palau, 1994 - Rabaul) by all of our members and associates. The alternative is an inevitable regression to open warfare, and consequent little benefit to anyone involved.

Des Gorman  
President of SPUMS

## ORIGINAL PAPERS

### CARBON MONOXIDE POISONING: A REVIEW

Paul Mark

#### Introduction

Carbon monoxide (CO) is a colourless, odourless, tasteless and non-irritant gas. It is the commonest agent used in suicide by poisoning in the United States<sup>1</sup>, Britain<sup>2,3</sup> and Australia.<sup>4</sup> In addition eighty percent of immediate deaths in burning buildings are due to CO.<sup>5,6</sup>

Following non-fatal poisoning, 10-40% of victims develop neurological or psychiatric sequelae.<sup>7</sup> The risk of death or major disability is increased in the young, the elderly and those with cardiovascular, cerebrovascular, or pulmonary disease.<sup>8</sup>

Hyperbaric oxygen (HBO) was first used to treat CO poisoning by Smith in 1960.<sup>7</sup> Numerous published series have established its benefit when compared with historical controls.<sup>8-15</sup> CO poisoning is regarded as an "accepted" indication for Hyperbaric Oxygen Therapy by the Undersea and Hyperbaric Medical Society.<sup>16</sup>

A recent review of thirteen published series containing 3,441 CO poisoned patients has shown clearly that administration of hyperbaric oxygen at 2-3 atmospheres absolute (ATA) soon after admission to hospital and repeated daily, or as made necessary by the patient's condition, is the only effective treatment of CO poisoning yet demonstrated.<sup>17</sup>

This paper reviews recent advances in our knowledge of the pathophysiology of CO poisoning and describes its clinical presentation. It outlines the management of the CO poisoned patient in the emergency department and discusses the indications for referral to a hyperbaric facility.

#### Circumstances of poisoning

The clinical features of CO intoxication are non-specific and may outlast the detection of carboxy-haemoglobin (COHb) in the blood. A thorough history often suggests the diagnosis in less obvious cases.

#### ATTEMPTED SUICIDE

Patients attempting suicide usually park their vehicles in isolated places with a hose connecting the exhaust pipe to the interior of the vehicle. Occasionally they park in a closed garage with the vehicle windows open. Even if the motor stops, the exhaust fumes persist for hours. The Australian change to unleaded petrol for new vehicles should reduce the opportunity for suicide as catalytic convertors significantly reduce the output of CO.<sup>18-20</sup>

#### FIRE

Persons trapped in building fires usually collapse from CO poisoning before being burnt. The mortality from CO poisoning is four times higher when it is complicated by smoke induced chemical pneumonitis.<sup>21</sup> The delayed sequelae of smoke inhalation greatly increase the mortality from cutaneous burns.<sup>22</sup> This may be partly due to the concomitant production of cyanide which is difficult to detect specifically.<sup>5,23</sup> A number of other irritant chemicals such as

acrolein, hydrochloric acid, toluene diisocyanate and nitrogen dioxide are produced by fires. These cause bronchospasm and pulmonary oedema.<sup>24,25</sup>

## ACCIDENTAL

It is likely that many cases of accidental exposure go unrecognised.<sup>26</sup> Mechanics occasionally poison themselves when the car engine is run with the garage doors closed, especially in cold weather. It has been shown that a lethal concentration of CO can be reached in a closed garage in 10 minutes.<sup>27</sup>

Faulty exhaust systems in cars or home heaters can result in whole families being affected. Following severe exposure the patients may present in coma or with altered mental states. Confusingly with less severe exposure they may present with symptoms similar to a viral illness.<sup>28</sup>

Most paint strippers contain methylene chloride which may be inhaled or absorbed through the skin. It is metabolised to CO.<sup>30</sup> The COHb level continues to rise after the patient has been removed from the source of poisoning and then falls slowly.<sup>30,31</sup> However the clinical disturbance is not as severe as the COHb level would indicate.<sup>30</sup>

Divers using a compressor for surface supplied air (hookah) are often poisoned when the air inlet is down wind of the exhaust from the pump motor. If scuba divers' cylinders are filled with CO containing air several persons are likely to be affected. CO poisoning can be mistaken for cerebral arterial gas embolism.<sup>32,33</sup>

## Pathophysiology

### HYPOXIA

A small amount of CO is produced by the normal enzymatic breakdown of haem. Unless there is significant haemolysis the normal COHb level is less than two percent. However smokers may have up to 10% of their haemoglobin (Hb) bound to CO.<sup>34</sup>

Research by Haldane demonstrated that Hb binds CO 240 times more avidly than it binds oxygen.<sup>35,36</sup> This creates a functional anaemia which is exacerbated if the patient has a true anaemia.

CO moves the oxy-haemoglobin (oxy-Hb) dissociation curve to the left, further reducing oxygen delivery to the tissues.<sup>37</sup> This will be exacerbated by alkalosis, hypothermia and concomitant poisoning with cyanide.<sup>38</sup> Mild to moderate acidosis is said to improve tissue oxygen delivery.<sup>37</sup>

### HISTOTOXIC EFFECTS

A further consequence of poisoning is the binding of

CO to mitochondrial enzymes, including reduced cytochrome A3 oxidase, reduced cytochromes of the P450 type and tryptophan dioxygenase.<sup>20,36,47,48</sup> This prevents cells from utilising oxygen and is thought to be a major cause of the disordered physiology. In 1975 Goldbaum replaced two thirds of a laboratory dog's haemoglobin with COHb. There was no deleterious effect. However, when dogs were allowed to breathe CO until an equivalent COHb level was reached they died.<sup>49</sup> It is the CO dissolved in the tissues that is important in producing clinical effects.<sup>50</sup>

Short term exposure, especially in an exercising fireman, will lead to high COHb levels but little CO in the mitochondria. In such cases collapse may be due solely to hypoxia.<sup>3</sup> Longer exposure in an overdose victim will give lower COHb levels but more tissue bound CO.<sup>21</sup> This the setting most likely to give rise to neurological damage including delayed neuropsychiatric sequelae.<sup>51</sup> Concomitant intoxication with ethanol may afford some protection, probably because of an ethanol induced decrease in blood flow and CO intake.<sup>52,53</sup>

Cytochromes have been shown, in-vitro, to bind oxygen nine times more readily than they bind CO.<sup>54</sup> Tissue hypoxia, due to cardiopulmonary or cerebrovascular disease and to the effects of COHb, promotes CO binding with cytochromes.<sup>46</sup> With time, the mitochondria may be overwhelmed as the cytochrome-CO bond is probably not entirely competitive.<sup>47</sup>

The tissues with the highest metabolic rate suffer the most damage especially in areas of poorly developed collateral blood supply.<sup>42,44</sup> Children are more at risk than adults.<sup>8</sup>

### THE FOETUS

The foetus is particularly susceptible to the effects of CO.<sup>61-63</sup> Foetal Hb binds CO more avidly than adult Hb resulting in a half life twice as long as that of maternal COHb and COHb levels 10-15% higher. The oxy-Hb dissociation curve is further to the left and the low arterial partial pressure of oxygen facilitates CO binding to cytochrome. The risk period extends into the first year of life, as at three months of age the child still has 25% of foetal Hb.

### OTHER TISSUES

Injury to other tissues is often complex.<sup>34</sup> Trauma to muscle, chemical damage to the lung and sludging in the glomeruli as a result of rhabdomyolysis all complicate the direct cellular damage produced by CO.<sup>44</sup>

### DELAYED SEQUELAE

The events at cellular level are not clear. Early deterioration may be due to death of tissue with previously impaired blood supply or to cerebral oedema. This does not explain why some patients improve for up to three weeks



before relapsing. One theory suggests that enzyme synthesis disruption occurs but the cells continue to function until the existing enzymes are used up. The ability of hyperbaric oxygen therapy to improve delayed function is possibly due to reactivation of these enzyme systems.<sup>6</sup> A variation of this model emphasises the strong binding of carbon monoxide to solid surfaces and its ability to compete for receptor sites slowing the rates of physiological reactions.<sup>17,55</sup>

More recent research has concentrated on the similarity to re-perfusion injury following cardiac arrest.<sup>56,57</sup> Thom has shown that exposure of laboratory rats to carbon monoxide is associated with an increase in brain lipid peroxidation, which commences only after the animals are returned to CO free air.<sup>58,59</sup> Hyperbaric oxygen at 3 ATA, but not oxygen at 1 ATA (sealevel), was shown to antagonize lipid peroxidation possibly by increasing the scavenging of oxygen free radicals by superoxide dismutase.<sup>59,60</sup>

## POST MORTEM

Post mortem findings show widespread petechial haemorrhages consistent with hypoxia, also infarction, particularly in the globus pallidus, substantia nigra and myocardium, and cerebral oedema.<sup>26,34,43,64</sup> In those who die after a delay, the findings are those of cell degeneration and demyelination.<sup>65</sup>

## Clinical features

### MILD EXPOSURE

Low level exposure causes protean symptoms which may easily be mistaken for influenza or gastroenteritis.<sup>6,66</sup> Headache is the most frequent symptom and is often accompanied by nausea and light headedness.<sup>6,34,67</sup> Fatigue, muscle pains, diarrhoea, vomiting and difficulty in concentrating occur as exposure increases.<sup>68</sup> Dilatation of superficial veins occurs early and flame-shaped retinal haemorrhages may occur if exposure has lasted twelve hours or more.<sup>69</sup> A sudden increase in angina or the occurrence of palpitations may result from occult exposure.<sup>21,34</sup>

### MODERATE EXPOSURE

More severe exposure leads to subtle neurological dysfunction reflecting diffuse damage to the higher centres. Many of these patients appear vague and psychometric testing will uncover deficits. Neurologic examination often shows generalized muscle weakness, impaired balance and diplopia.<sup>70</sup>

Studies have shown that psychometric scores correlate better with the eventual outcome than does the COHb level measured on arrival at hospital.<sup>71,72</sup> Psychometric scoring measures the actual neurological deficit. When there has been a delay between the CO exposure and the taking of the blood sample some CO will have been released

from the haemoglobin, but not necessarily from the tissues. The correct first aid treatment of 100% oxygen by mask can reduce the COHb level very rapidly.

A brief psychometric format appropriate to an emergency department includes orientation, short term memory and recall, serial 7's, spelling a word backwards, drawing a three dimensional house, writing a sentence, naming items and following verbal and written instructions. It is useful to have a standard formula available, such as the Mini-Mental State Examination (MMSE).<sup>73</sup>

### SEVERE EXPOSURE

Severe exposure results in gross neurological abnormalities, lethargy, coma, agitation and convulsions.<sup>26</sup> Often these patients are abusive and combative when disturbed. They may appear to be hyperventilating, hysterical or frankly psychotic.<sup>6,42</sup> Muscle spasm, including trismus, has been described.<sup>34</sup> Peripheral neurological involvement is infrequent.<sup>74</sup>

### DELAYED SEQUELAE

Delayed neuropsychiatric sequelae develop between two and twenty one days following exposure, often after apparent recovery from the acute insult.<sup>10,75</sup> Higher functions are impaired, especially short term memory. Psychometric abnormalities may reappear. Korsakoff's syndrome may occur. Personality changes range from apathy to unconcerned incontinence. Ataxia, incontinence and Parkinsonism may develop.<sup>34,44</sup> Almost any neurological syndrome may be produced.

In a series of 206 patients treated by Smith and Brandon in the 1960's, only the more severely affected were given 100% oxygen at atmospheric pressure. There was a 33% mortality rate. Only 2.2% of the 138 survivors had neuropsychiatric sequelae at discharge.<sup>76</sup> When reviewed three years later the situation was considerably different as 10.8% had gross neuropsychiatric deficits, 28.4% had an obvious personality deterioration and 36.5% had some loss of memory.<sup>7</sup>

The clinical state at the scene of poisoning correlates better with the risk of sequelae than does the clinical state on arrival at hospital.<sup>10,14,77</sup> Variable recovery occurs in 75% of survivors at one year.<sup>78</sup> Characteristically the most severe delayed sequelae are seen in the elderly and in children.<sup>8</sup> Delayed sequelae are more common following severe poisoning.

Depression occurs frequently, even in patients who do not attempt suicide. Following an attempted suicide, it is often difficult to diagnose an antecedent psychiatric disorder due to the presence of the neurological and psychiatric sequelae of CO intoxication.<sup>79</sup>

## CARIORESPIRATORY EFFECTS

The cardiovascular effects include ischaemia, arrhythmias and hypotension. Most early deaths from CO poisoning are due to ventricular arrhythmias and are more common in patients with coronary heart disease or cardiomegaly.<sup>34,44</sup>

Non-cardiogenic pulmonary oedema, due to other toxic agents in smoke, is particularly likely if the victim has been trapped by fire in an enclosed space.<sup>46</sup> Aspiration of fluids is a hazard in anyone who is unconscious and is often implicated in the death of patients who die a few days after admission.<sup>80</sup>

## OTHER EFFECTS

In severe cases the effects of CO on other body systems become apparent. Skin blistering, which usually occurs in pressure areas, is uncommon unless the COHb has exceeded 40%.<sup>81</sup> The classically described cherry red discolouration of the skin is rare but venous blood samples may appear arterial in colour.<sup>21</sup>

Rare sequelae include rhabdomyolysis, acute tubular necrosis, pancreatitis, hepatitis, haemolytic anaemia and thrombotic thrombocytopenic purpura.<sup>34</sup> CO exposure during pregnancy may result in premature labour, stillbirth and severe neurological deficits in the infant.<sup>61-63</sup>

## Laboratory investigations

Arterial blood gas analysis shows a normal partial pressure of oxygen if there is no lung pathology. Since the saturation figure given by most blood gas analysis machines is calculated from the measured oxygen partial pressure it will be falsely elevated. The true saturation is reduced by the percentage of Hb bound to CO. Most pulse oximeters are unable to distinguish between oxy-Hb and COHb and also over estimate the true oxygen saturation.<sup>82</sup>

The importance of metabolic acidosis as a measure of severity has recently been questioned. Several authors have demonstrated that both alkalosis and acidosis can accompany clinically severe poisoning.<sup>83-86</sup> The lactate level is probably a more reliable guide.<sup>87</sup>

The ECG commonly shows sinus tachycardia, often with ST depression and flat or inverted T waves.<sup>17</sup> The chest X-ray is often normal on admission but may display the features of pulmonary oedema or of aspiration. Haematological and biochemical analysis usually reveal a neutrophilia and may show hypokalaemia and hyperglycaemia.<sup>21,88</sup>

The COHb percentage can be measured readily by most laboratories and often helps to confirm the diagnosis.

Whereas it was once the sole criterion upon which treatment was based it is now only one of a number of factors to be considered. Attempts to calculate the past likely maximum COHb are unreliable due to variations in its half life.<sup>46</sup>

Only when alternative diagnoses require exclusion should CT scanning from the emergency department be performed. The presence of bilateral low density regions in the globus pallidus 24 hours after poisoning has been found to predict a higher incidence of death or major disability but not all such patients show these lesions.<sup>15,89</sup>

## Management

### IMMEDIATE TREATMENT

Initial priorities are control of the airway and support of respiration and circulation. 100% oxygen should be given as soon as the diagnosis is suspected. In a co-operative patient this can be achieved with a tightly fitting face mask using a non-rebreathing valve and a reservoir bag. The Hudson mask is inadequate and should not be used as it will deliver no more than 50-60% oxygen even at 14 litres per minute oxygen flow. In a comatose or unco-operative patient aspiration must be avoided so endotracheal intubation, and possibly positive pressure ventilation, may be required. Before hyperbaric therapy the cuff of the endotracheal tube should be filled with water or saline. Cardiac monitoring is required and IV fluids should be started. Hypotension usually responds to IV fluids. A urinary catheter is required as in any other severely ill patient. Patients with chronic obstructive airways disease and hypoxia-dependent respiratory drive present problems and require careful assessment.

### COMPLICATING FACTORS

Fire victims are at risk of airway problems from chemical or thermal injury and should be observed closely. Bronchospasm, pulmonary oedema and traumatic injuries may need to be treated. In a rapidly deteriorating patient who does not respond to oxygen, treatment for cyanide intoxication should be considered.<sup>25</sup> Sodium thiosulphate and cobalt EDTA are the preferred treatments.<sup>23,25</sup> Methaemoglobinemia is dangerous in the presence of CO.<sup>24,90</sup>

Suicide victims should be assessed for the effects of ingested drugs, particularly tricyclic anti-depressants. Hypothermia should be corrected.

Intravenous solutions containing dextrose are best avoided as hyperglycaemia may adversely affect neurological outcome.<sup>91</sup> Increased interstitial and intracellular lactate levels produce acidosis which results in neuronal damage. Several retrospective studies both in CO poisoning and in other ischaemic neurological conditions support this theory.<sup>92</sup>

Metabolic acidosis should be treated if it is associated with resistant ventricular arrhythmias or decreased cardiac output not responding to intravenous fluids. The preferred method of correction, if the patient is ventilated, is by increasing the minute volume. Any sudden correction of acidosis will decrease the serum potassium and may precipitate arrhythmias.

## Hyperbaric oxygen

### RATIONALE OF THERAPY

The purpose of hyperbaric oxygen therapy is two fold.

Firstly, it accelerates the removal of CO from Hb, myoglobin and cytochromes. The half life of COHb in air is 320-480 minutes. This is reduced to 60-80 minutes by 100% normobaric oxygen and to 8-23 minutes by HBO at 2.8 ATA.<sup>17,21,47,93</sup> At this pressure the arterial partial pressure of oxygen is around 1,800 mm Hg which should displace any remaining CO from the cytochromes. Intense vasoconstriction due to hyperoxia rapidly reduces cerebral oedema while increasing cerebral oxygen flux. Numerous published case reports attest to the ability of HBO to awaken unconscious patients whose COHb level has been zero for hours and to the additional benefits of repeated treatments in patients with persistent deficits.<sup>6,64,94,95</sup>

Secondly, it prevents delayed neuropsychiatric sequelae. Myers studied two groups of patients.<sup>10</sup> Those with a normal psychometric score, normal ECG and a COHb level of less than 30% were given normobaric 100% oxygen. Delayed sequelae developed in 12%. The more severely affected patients, with gross neurological signs, psychometric abnormalities or a COHb level greater than 30%, received HBO at 2.8 ATA. There were no delayed sequelae in this group. Those patients who developed delayed sequelae following normobaric oxygen were later treated with HBO and all recovered.

While it was not a prospective controlled trial, this study demonstrates two important points. Patients who appear well when they reach the emergency department can develop sequelae, and HBO is effective both for the prevention and treatment of neurological sequelae.

Only one prospective controlled clinical trial of HBO in CO poisoning has been reported,<sup>77</sup> but this trial used oxygen doses which have been shown previously to be ineffective.<sup>10,12,14,17,41,75,96</sup>

In a recent review, Gorman and Runciman collated the results of 13 clinical series in which the long term mortality (at one month) could be determined.<sup>17</sup> A total of 1,847 patients had been treated with varying combinations of normobaric oxygen, a single HBO treatment or HBO

therapy confined to the first 24 hours after admission. These therapies accelerated the natural early recovery from CO poisoning but did not prevent the delayed deteriorations.<sup>55</sup> Another group of 1,594 patients had been treated with HBO on admission and then either daily, or as made necessary by the patient's condition. Mortality in this group varied between 0 and 9.6% and was essentially seen only in those who had a cardiac arrest prior to arrival at hospital. In these patients long term morbidity varied between 0 and 4.4% and none suffered a late deterioration in cerebral function.

### Indications for HBO

HBO should be given as early as possible to the unconscious patient and to the conscious patient with gross neurological or psychometric abnormalities, clinical or electrocardiographic evidence of ischaemia, arrhythmias, significant hypotension, pulmonary oedema or severe metabolic acidosis, even if this requires transfer. Normally transfer should not be attempted until the airway is secure and the cardiac rhythm and blood pressure are stable.

The COHb level upon arrival in the emergency department has repeatedly been shown to an unreliable basis for determining treatment.<sup>8,10,11,14</sup> Rather arbitrarily, an isolated level greater than 40% has been recommended as indication for urgent HBO.<sup>21,34</sup> Some authors recommend a level of 25% in the emergency department on the basis that the maximum level at the scene would have been higher.<sup>97</sup>

Patients who were unconscious or who had an abnormal mental state at the scene but who are clinically normal on arrival in the emergency department are at risk of delayed sequelae.<sup>10,14,17</sup> Adults with an isolated carboxyhaemoglobin level greater than 25% and children with a level greater than 15% are also at risk.<sup>98</sup> Transfer to a hyperbaric facility should occur within 24-28 hours of presentation.

In the absence of indications for early HBO therapy 100% oxygen should be administered for six hours.

The question of timing is important since most patients will have to be transferred for hyperbaric therapy. Goulon demonstrated improved results in comatose patients when HBO was given within six hours.<sup>96</sup> Norkool reviewed 115 cases over six years and concluded that it was possible to achieve good results as late as 20 hours after exposure.<sup>11</sup>

### REMOTE LOCATIONS

Patients in remote locations with minor psychometric abnormality should be treated with 100% normobaric oxygen but if they do not improve within six hours, transfer should be arranged. If they improve they must be reviewed closely over the ensuing weeks for the development of delayed neuropsychiatric sequelae. If these occur they should be treated by HBO as soon as possible.<sup>10</sup>

## SAFETY IN PREGNANCY

There has been concern about the safety of HBO in pregnant patients. A careful review of the literature by van Hoesen has revealed that fears regarding teratogenicity, foetal blindness and alterations in foetal circulation are unfounded.<sup>61</sup> It has been found that the maternal condition at the site of exposure predicts the risk of foetal sequelae.<sup>62</sup> Normobaric oxygen may decrease the maternal, but not the foetal, CO load.<sup>99</sup> HBO should be given for any maternal intoxication, foetal distress or a maternal COHb level exceeding 15%.<sup>61</sup> In all cases it should be given as early as possible.

## Treatment schedule

In 1992 HBO is a very safe treatment. Multi-place chambers allow medical staff to remain with any seriously ill patient. All patients are accompanied by a specially trained registered nurse. Intensive care, mechanical ventilation, therapeutic infusions and a range of other procedures can be performed in the chamber. Continuous cardiac monitoring and invasive blood pressure monitoring can be employed. Regular "air breaks" are provided which almost eliminate the risk of oxygen toxicity. The pressure changes are gradual and the difficulty in clearing the ears is little worse than descending in an aeroplane. Claustrophobia is overcome by distracting the patient, constant reassurance and the judicious use of benzodiazepines.

At Fremantle Hospital the initial treatment is conducted at 2.8 ATA. Following compression (often referred to as descent) there are two 25 minute oxygen sessions during which the patient breathes 100% oxygen, each followed by five minutes breathing air (air break). The patient also breathes oxygen during the 35 minute decompression (ascent) including a five minute stop at 1.9 ATA.

## Follow up

Psychiatric review is important after attempted suicide. Precautions to prevent recurrence are essential after accidental cases. Reviewing patients seven days after exposure provides an opportunity to detect delayed sequelae. Relatives should be told to bring the patient back to hospital earlier if there is any change in their behaviour or personality.

## Summary

Patients with carbon monoxide poisoning present many challenges to emergency physicians and hyperbaric unit staff. The role of hyperbaric oxygen has been well researched and indications for its use are now clear. It is essential treatment to prevent post-poisoning neuropsychological sequelae.

In view of this, emergency physicians should know how to arrange treatment at the nearest hyperbaric facility.

## References

- 1 *Accident Facts*. Chicago: National Safety Council, 1982: 80-84.
- 2 Office of Population Censuses and Surveys. *Mortality statistics 1985 - cause, series DH2, table 2, ICD code 986*. London: HMSO, 1987.
- 3 Broome JR, Skrine H and Pearson RR. Carbon monoxide poisoning: Forgotten not gone. *Br J Hosp Med* 1988; 39(4): 298-300, 302, 304-305.
- 4 *Suicides Australia 1961-1981 (including historical series 1881-1981) Table 8 (Cat. no. 3309.0)*. Canberra: Australian Bureau of Statistics 1983: 16. .
- 5 Lundquist P, Rammer L and Sorbo B. The role of hydrogen cyanide and carbon monoxide in fire casualties: a prospective study. *Forensic Soc Int* 1989; 43(1): 9-14.
- 6 Chance JF. Carbon monoxide poisoning. *Audio digest Emerg Medicine* 1987; 4(6).
- 7 Smith JS and Brandon S. Morbidity from acute carbon monoxide poisoning as 3 year follow up. *Br Med J* 1973; 1: 318-321.
- 8 Thom SR and Keim LW. Carbon monoxide poisoning: a review of epidemiology, pathophysiology, clinical findings and treatment options including hyperbaric oxygen therapy. *J Toxicol Clin Toxicol* 1989; 27(3): 141-156.
- 9 Smith GI and Sharp GR. Treatment of carbon monoxide poisoning with oxygen under pressure. *Lancet* 1960; 1: 905-906.
- 10 Myers RAM, Snyder SK and Emhoff TA. Subacute sequelae of carbon monoxide poisoning. *Ann Emerg Med* 1985; 14: 1163-1167.
- 11 Norkool DM and Kirkpatrick JN. Treatment of acute carbon monoxide poisoning with hyperbaric oxygen: a review of 115 cases. *Ann Emerg Med* 1985; 14: 1168-1171.
- 12 Krantz T, Thisted B, Strom J and Strensen MB. Acute carbon monoxide poisoning. *Acta Anaesthesiol Scand* 1988; 32(4): 278-282.
- 13 Kindwall EP. Carbon monoxide poisoning treated with hyperbaric oxygen. *Respirat Ther* 1975; 5: 29-33.
- 14 Mathieu D, Nolf PM, Durocher A et al. Acute carbon monoxide poisoning. Risk of late sequelae and treatment with hyperbaric oxygen. *J Toxicol Clin Toxicol* 1985; 23(4-6): 315-324.
- 15 Sawada Y, Takahashi M, Ohashi N et al. Computerised tomography as an indication of long term outcome after carbon monoxide poisoning. *Lancet* 1980; 1: 783-784.
- 16 Myers RAM, ed. *Hyperbaric oxygen therapy: A committee report*. Bethesda Maryland: Undersea and Hyperbaric Medical Society Inc. No 30. 1986; 33-36.

- 17 Gorman DF and Runciman WB. Carbon monoxide poisoning. *Anaesth Intensive Care* 1991; 19(4): 506-518.
- 18 Landers D. Unsuccessful suicide by carbon monoxide: a secondary benefit of emission control. *West J Med* 1981; 135: 360-363.
- 19 *Motor vehicle emission test results. Vol. 5* (ISSB 0817-3044) Sydney: NSW State Pollution Control Commission. 1987; 10-57..
- 20 Lester D and Clarke RV. Effects of the reduced toxicity of car exhausts on accidental deaths: a comparison of the United States and Great Britain. *Accid Anal Prev* 1989; 21(2): 191-193.
- 21 Kindwall EP and Goldmann RW. *Hyperbaric medicine procedures*. St. Luke's Hospital, Milwaukee, Wisconsin 1984; 90-98.
- 22 Clark RJ and Beeley JM. Smoke inhalation. *Br J Hosp Med* 1989; 41(3): 252-255, 258-259.
- 23 Baud FJ, Barriot P, Toffis V et al. Elevated blood cyanide concentrations in victims of smoke inhalation. *N Engl J Med* 1991; 325: 1761-1766.
- 24 Heimbach DM and Waecherle JP. Inhalation injuries. *Ann Emerg Med* 1988; 17(12): 1316-1320.
- 25 Kulig K. Cyanide antidotes and fire toxicology. *N Engl J Med* 1991; 325: 1801-1802.
- 26 Meredith T and Vale A. Carbon monoxide poisoning. *Br Med J* 1988; 296: 72-79.
- 27 Stewart RD. The effect of carbon monoxide on humans. *Annual review of pharmacology* 1975; 15: 409-422.
- 28 Ilano AL and Raffin TA. Management of carbon monoxide poisoning. *Chest* 1990; 97(1): 165-169.
- 29 Rudge FW. Treatment of methylene chloride induced carbon monoxide poisoning with hyperbaric oxygenation. *Milit Med* 1990; 40(9): 154-155.
- 30 Fagin J, Bradley J and Williams D. Carbon monoxide poisoning after accidentally inhaling paint remover. *Br Med J* 1980; 281: 1461.
- 31 Roix JP and Myers RAM. Hyperbaric oxygen for methylene chloride poisoning: Report on two cases. *Ann Emerg Med* 1989; 18: 691-695.
- 32 McKee J. Cerebral gas embolism or carbon monoxide poisoning: a case report. *SPUMS J* 1987; 17(4): 143-144.
- 33 Hackman C. Air embolism and carbon monoxide poisoning. *SPUMS J* 1983; 2: 44-47.
- 34 Ellenhorn MJ and Barceloux DG, eds. *Medical toxicology - diagnosis and treatment of human poisoning*. New York: Elsevier 1988; 820-829.
- 35 Haldane J. The relation of the action of carbonic oxide to oxygen tension. *J Physiol* 1895; 18: 201-217.
- 36 Haldane J. Carbon monoxide as a tissue poison. *Biochem J* 1927; 21: 1068-1075.
- 37 West JB. *Respiratory physiology - the essentials. 3rd ed*. Baltimore: Williams & Williams 1985.
- 38 Satariya BB, Penny DG and Nallamotheu BG. Hypothermia following acute carbon monoxide poisoning increases mortality. *Toxicol Lett* 1990; 52(2): 201-208.
- 39 Coburn RF. The carbon monoxide body stores. *Ann NY Acad Sci* 1970; 174: 11-22.
- 40 Olson KR. Carbon monoxide poisoning, mechanisms, presentation and controversies in management. *J Emerg Med* 1984; 1(3): 233-243.
- 41 Ginsburg MD. Carbon monoxide intoxication: clinical features, neuropathology and mechanisms of injury. *J Toxicol Clin Toxicol* 1985; 23(4-6): 281-288.
- 42 Dolan MC. Carbon monoxide poisoning. *Can Med Assoc J* 1985; 133(5): 392-399.
- 43 Myer-Witting M, Helps SC and Gorman DF. The effect of an acute carbon monoxide exposure on cerebral blood flow in rabbits. *Undersea Biomed Res* 1990; 17(Suppl): 64.
- 44 Thom SR. Smoke inhalation. *Emerg Med Clin North Am* 1989; 7(2): 371-386.
- 45 Anderson GK. Treatment of carbon monoxide poisoning with hyperbaric oxygen. *Milit Med* 1978; 143: 538-541.
- 46 Williams SJ, Scott AA and Norman PH. Carbon monoxide off-gassing during hyperbaric and normobaric oxygen therapy of CO poisoned patients: a prospective clinical series. *Undersea Biomed Res* 1985; 12(1) Suppl: 55.
- 47 Piantadosi CA. Carbon monoxide, oxygen transport and oxygen metabolism. *J Hyperbaric Med* 1987; 2(1): 27-41.
- 48 Loumanmaki K and Coburn R. Effects of metabolism and distribution of carbon monoxide on blood and body stores. *Am J Physiol* 1969; 217: 354-363.
- 49 Goldbaum LR, Ramirez RG and Absalom KB. What is the mechanism of carbon monoxide toxicity? *Aviat Space Environ Med* 1975; 46: 1289-1291.
- 50 Orellano T. Studies on the mechanism of carbon monoxide toxicity. *J Surg Res* 1976; 20(5): 485-487.
- 51 Rhodes RH, Skolnick JL and Roy TM. Hyperbaric oxygen treatment for carbon monoxide poisoning: Observations based on 8 years experience. *J Ky Med Assoc* 1991; 89(2): 61-64.
- 52 Tomita M, Okuyama T, Skimosato K, Kondo Y and Ijiri I. Effect of ethanol on fatal carbon monoxide poisoning in awake mice: *Toxicol Lett* 1990; 50(2-3): 151-157.
- 53 Sharma P and Penny DG. Effects of ethanol in acute carbon monoxide poisoning. *Toxicology* 1990; 62(2): 213-226.
- 54 Ball EG, Strittmatter CF and Cooper O. The reaction of cytochrome oxidase with CO. *J Biol Chem* 1951; 193: 635-697.
- 55 Gorman DF. Problems and pitfalls in the use of hyperbaric oxygen for the treatment of poisoned patients. *Med Toxicol Adverse Drug Exp* 1989; 4(6): 393-399.
- 56 Marklund SL. Oxygen toxicity and protective systems. *Clin Toxicol* 1985; 23: 289-298.
- 57 James PB. Hyperbaric oxygen in the treatment of carbon monoxide poisoning and smoke inhalation injury. *Intensive Care World* 1989; 6(3): 135-138.

- 58 Thom SR. Carbon monoxide mediated brain lipid peroxidation in the rat. *J Appl Physiol* 1990; 68(3): 997-1003.
- 59 Thom SR. A delayed carbon monoxide induced change in rat brain and its antagonism by hyperbaric oxygen. *Undersea Biomed Res* 1987; 14(Suppl 2): 40.
- 60 Thom SR. Antagonism of lipid peroxidation by elevated partial pressures of oxygen. *Undersea Biomed Res* 1988; 15(Suppl): 22.
- 61 Van Hoesun KB, Camporesi EM, Moon RE, Hage ML and Piantadosi CA. Should hyperbaric oxygen be used to treat the pregnant patient for carbon monoxide poisoning: A case report and literature review. *JAMA* 1989; 261(7): 1039-1043.
- 62 Caravati EM, Adams CJ, Joyce SM and Schafer NC. Foetal toxicity associated with maternal carbon monoxide poisoning. *Ann Emerg Med* 1988; 17(7): 714-717.
- 63 Farrow JR, Davis GJ, Roy TM, McCloud LC and Nichols GR. Fetal death due to nonlethal maternal carbon monoxide poisoning. *J Forensic Sci* 1990; 35(6): 1448-1452.
- 64 Anderson RF, Allensworth DC and De Groot WJ. Myocardial toxicity from carbon monoxide poisoning. *Ann Intern Med* 1967; 67: 1172-1182.
- 65 Lourey C. Treatment of carbon monoxide poisoning in childhood. *SPUMS J* 1990; 20(1): 29-32.
- 66 Gemelli F and Cattini R. Carbon monoxide poisoning in childhood. *Br Med J* 1985; 291: 1197.
- 67 Mills J, Ho MT, Salber PR and Trunkey DD. *Current emergency diagnosis and treatment*. 2nd ed. Los Altos: Lange Medical Publications, 1985: 460-461.
- 68 Burney RE, Wu SC and Nemiroff MJ. Carbon poisoning: clinical effects and results of treatment in 184 victims. *Ann Emerg Med* 1982; 11: 394-399.
- 69 Kelley JS and Sophocleus GJ. Retinal haemorrhages in subacute carbon monoxide poisoning. Exposure in homes with blocked furnace fires. *JAMA* 1978; 239: 1519-1717.
- 70 Rosen P, Baker FJ, Braen GR, Dailey RH and Levy RC. *Emergency medicine - concepts and clinical practice*. St. Louis, Toronto: CV Mosby Co, 1983: 242-243, 438-439.
- 71 Myers RAM, Messier LD, Jones DW and Crowley RA. New directions in the research and treatment of carbon monoxide exposure. *Am J Em Med* 1983; 2: 226-230.
- 72 Brown JR and Pearson RR. Carbon monoxide poisoning: Forgotten not gone. *Br J Hosp Med* 1988; 39(4): 298-305.
- 73 Folstein MF, Folstein SE and McHugh PR. "Minimal state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975; 12(3): 189-198.
- 74 Joiner TA, Sumner JR and Catchings TT. Unilateral diaphragmatic paralysis secondary to carbon monoxide poisoning. *Chest* 1990; 97(2): 498-499.
- 75 Choi IS. Delayed neurologic sequelae in carbon monoxide intoxication. *Ann Neurol* 1983; 40: 433-435.
- 76 Smith JS and Brandon S. Acute carbon monoxide poisoning - 3 years experience in a defined population. *Postgrad Med J* 1980; 46: 65-70.
- 77 Raphael JC, Elkharrat P, Jars-Guincestre MC et al. Trial of normobaric and hyperbaric oxygen for acute carbon monoxide intoxication. *Lancet* 1989; 2(8660): 414-419.
- 78 Lee H. Clinical studies on delayed sequelae of carbon monoxide intoxication. *J Kor Neuropsychiatra Assc* 1978; 15: 374-385.
- 79 Jaekle RS and Nasrallah HA. Major depression and carbon monoxide induced Parkinsonism: diagnosis, computerised axial tomography and response to L-dopa. *J Nerv Ment Dis* 1985; 173(8): 503-508.
- 80 Mark P. Carbon monoxide poisoning. *Emergency Doctor* 1990; 2(3): 10-15.
- 81 Myers RAM, Snyder SK Majerus TC. Cutaneous blisters and carbon monoxide poisoning. *Ann Emerg Med* 1985; 14(6): 603-606.
- 82 Vegfors and Lennmarken C. Carboxyhaemoglobinaemia and pulse oximetry. *Br J Anaesth* 1991; 66(5): 625-626.
- 83 Strohl KP, Feldman NT, Saunders NA and O'Connor N. Carbon monoxide poisoning in fire victims: a reappraisal of prognosis. *J Trauma* 1980; 20(1): 78-80.
- 84 Myers RAM and Britten JS. Are arterial blood gases of value in treatment decision for carbon monoxide poisoning? *Crit Care Med* 1989; 17(2): 139-142.
- 85 Leiby TI, Zalenski R, Hryhorczuk DO and Leikin JB. The usefulness of the arterial blood gas in pure carbon monoxide poisoning. *Vet Hum Toxicol* 1989; 31(2): 138-140.
- 86 Myers RAM. Do arterial blood gases have value in prognosis and treatment decisions in carbon monoxide poisoning? *Undersea Biomed Res* 1987; 14(2): Suppl: 1.
- 87 Sokal JA and Kralkowska E. The relationship between exposure duration, carboxyhaemoglobin, blood glucose, pyruvate and lactate and the severity of intoxication in 39 cases of acute carbon monoxide poisoning in man. *Arch Toxicol* 1985; 57: 196-199.
- 88 Leikin JB, Goldenberg RM, Edwards D and Zell-Kantor M. Metabolic predictors of monoxide poisoning. *Vet Hum Toxicol* 1988; 30(1): 40-42.
- 89 Vieregge P, Klostermann W, Blsumm RG and Boris KJ. Carbon monoxide poisoning: clinical, neurophysiological and brain imaging observations in acute disease and follow up. *J Neurol* 1989; 236(8): 478-481.
- 90 Moore SJ, Norris JC, Walsh DA and Humer AS. Antidotal use of methemoglobin forming cyanide antagonists in concurrent carbon monoxide/cyanide intoxication. *J Pharmacol Exp Ther* 1987; 242: 70-73.
- 91 Penney DG. Hyperglycaemia exacerbates brain damage in acute severe carbon monoxide poisoning. *Med Hypothesis* 1988; 27(3): 241-244.

- 92 Siesjo BK and Wieloch T. Cerebral metabolism, in ischaemia: neurochemical basis for therapy. *Br J Anaesth* 1985; 57: 47-62.
- 93 Fang GC, Xu GH, Wang FM and Hua B. Clinical significance of monitoring blood carboxyhaemoglobin. *J Hyper Med* 1986; 1(4): 233-238.
- 94 Myers RAM, Snyder SK and Linbert S et al. Value of hyperbaric oxygen in suspected carbon monoxide poisoning. *JAMA* 1981; 246: 2478-2480.
- 95 Neubauer RA. Carbon monoxide and hyperbaric oxygen. *Arch Intern Med* 1979; 139: 829.
- 96 Goulon M, Berois A, Rapin M, Nouilhat F, Grobuis S and Labrosse J. Carbon monoxide poisoning and acute anoxia due to breathing coal gas and hydrocarbons. *Ann Med Interne (Paris)* 1969; 120(5): 335-349.
- 97 Hyperbaric Centre Advisory Committee, Emergency Medical Service. A registry for carbon monoxide poisoning in New York City. City of New York. *J Toxicol Clin Toxicol* 1988; 1 26(7): 419-441.
- 98 Croker PJ and Walker JS. Paediatric carbon monoxide toxicity. *J Emerg Med* 1985; 3(6): 443-448.
- 99 Margulies JL. Acute carbon monoxide poisoning during pregnancy. *Ann J Emerg Med* 1986; 4(6): 516-519.

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## HAWAIIAN SCUBA DEATHS

Carl Edmonds and Roy Damron

### Background

During the 1980's a number of surveys were carried out on the causes of recreational scuba diving deaths. Also, the death rate in recreational divers was revised upwards.<sup>1</sup> Previously and also during that time the National Underwater Accident Data Center (NUADC), under the control of John McAniff carried, out annual surveys on the causes of diving deaths.<sup>2,3</sup> NUADC have recorded almost 3,000 fatalities, but the documentation relies heavily on second and third hand information. Nevertheless the great numbers ensure that the information is of value. More recently the Divers Alert Network (DAN) has also become involved in

the compilation and analysis of diving deaths in Northern America.

In the Australia and New Zealand survey (ANZ series), by Edmonds and Walker<sup>4,6</sup>, the deaths were less numerous, but more data was available and it was far more comprehensively catalogued. It included, as a routine, comprehensive police reports, autopsy details, equipment analyses and re-enactment trials. The information so obtained was used as a basis for a series of reports showing the factors contributing to death, and not merely the "final" cause.

This paper covers a number of scuba deaths in Hawaii over the same period. It is reminiscent of the NUADC reports, relying more on newspaper and unofficial reports than did the ANZ series.

It is hard to quantify the relative amount of material available in the three series. The ANZ series had far more detail than either of the American series. The Hawaiian series may well be a little more comprehensive than the NUADC reports because of the simpler logistics of obtaining information from within a single State of the USA, as opposed to trying to obtain information from all States and overseas.

### Methods

These case files were compiled by one of the diving experts in Hawaii (RD), initially obtained from newspaper reports but supplemented with follow up investigation, both on an official and personal level, to ascertain more details. The analysis was then made by an independent expert in this field (CE).

### Diving deaths data

For the 80 deaths, the data was often not complete and therefore the percentages recorded are those of the number on which that specific data was available, usually not of the total 80 cases. The data available from this survey does give some appreciation of the population being studied.

Approximately half the deaths were in divers aged below 30. Another third were aged between 31 and 40. None were over 60 (Table 1). The great majority of the deaths were in males. Only eight out of the eighty (10%) were females.

Diving qualifications were not always recorded. This information was available for 53 (66%) of the 80 deaths (Table 2). Three died on their first dive; one alone and two with companions whose diving expertise was unstated. Although 15% died while under instruction during their basic "open water" diving certificate training, this figure

TABLE 1

## AGE DISTRIBUTION OF DEATHS

11-20	5	(6%)
21-30	34	(43%)
31-40	22	(28%)
41-50	10	(13%)
51-60	8	(10%)
<b>Total</b>	<b>80</b>	<b>100%</b>

TABLE 5

## BUDDY SYSTEM

Diving alone at all times	12	(16%)
Separated voluntarily before the accident	23	(32%)
Separated voluntarily after the accident	10	(14%)
Separated by the elements or the accident	14	(19%)
Stayed together	14	(19%)
<b>Total</b>	<b>73</b>	

TABLE 2

## QUALIFICATIONS

Initial dive	3	(6%)
Under basic training	8	(15%)
Not certified	10	(19%)
Certified	30	(57%)
Professional	2	(4%)
<b>Total</b>	<b>53</b>	

TABLE 6

## DEPTH OF THE ACCIDENT

0-3 m	30	(48%)
3-9 m	14	(23%)
9-30 m	10	(16%)
>30 m	8	(13%)
<b>Total</b>	<b>62</b>	

TABLE 3

## EXPERIENCE LEVELS

Inexperienced (0-6 dives)	18	(33%)
Moderate experience(7-30 dives)	13	(24%)
Experienced (>30 dives)	24	(44%)
<b>Total</b>	<b>55</b>	

TABLE 7

## MAXIMUM DEPTH OF DIVE

0-3 m	3	(4%)
3-9 m	37	(50%)
9-30 m	19	(26%)
>30 m	15	(20%)
<b>Total</b>	<b>74</b>	

TABLE 4

## DIVING ACTIVITY

Recreational diving	25	(39%)
Under training (basic and advanced)	10	(16%)
Spear fishing	10	(16%)
Diving for black coral	8	(12%)
Crustacean collecting	3	(5%)
Photography	2	(3%)
Tropical fish collecting	2	(3%)
Scientific diving	2	(3%)
Fish feeding	2	(3%)
<b>Total</b>	<b>64</b>	

TABLE 8

## DURATION OF THE DIVE

Within the first 5 minutes	8	(14%)
At the end of the dive, after a low-on-air or out-of-air, (compromised air supply) situation	32	(54%)
During the intermediate part of the dive	19	(32%)
<b>Total</b>	<b>59</b>	



might have dropped to 10% if information was available on all cases.

In the 55 cases where the number of dives that the diver had done were recorded it was possible to group the degrees of experience (Table 3). A separate assessment was made to determine whether the diver was experienced enough to undertake the dive during which he ultimately died. It was decided that 30 (58%) were not experienced enough to undertake the fatal dive, whereas 22 (42%) had sufficient experience.

Many different activities were carried out during the fatal dive by the 64 divers for whom this information was available (Table 4). The buddy system was more honoured in the breach than in the observance (Table 5).

When the depth of the accident was recorded nearly half occurred in the surface to 3 m zone (Table 6). In 10 (16%) the accident developed during ascent. When the depth of the dive was recorded the majority of the divers were shallower than 9 m (Table 7).

In only 59 cases was there information about when the accident happened. In over half of these the accident happened at the end of the dive, after a low-on-air or out-of-air, (compromised air supply) situation (Table 8).

### Causes of Death

The causes of death were assessed for all 80 fatalities. At least 49 (61%) appeared to have died from drowning. At least 12 (15%) suffered pulmonary barotrauma, with or without cerebral arterial gas embolism. At least 7 (9%) died from the Sudden Death (cardiac) Syndrome.<sup>7,8</sup> Four (5%) died from decompression sickness following the dive. Trauma (injuries by boats) was responsible for 3 (4%), as was coincidental medical illnesses (cerebral haemorrhage in one instance, epilepsy in two.) In 2 of the cases (3%) there was a previous history of loss of consciousness at depth. This was also thought to be a contributory factor to death in both those cases.

### Factors contributing to death

The percentages in this section are of the 80 deaths.

#### HUMAN FACTORS (44%)

In 35 deaths (44%) significant human factors (medical disorders, physiological or psychological problems) contributed to the death, or prevented successful rescue and resuscitation.

Panic was noted in 16 cases (20%). Salt water aspiration was present in 9 (11%). Fatigue was noted in 5

(6%). Vomiting was present in 4 (5%). The following conditions were also noted, epilepsy in 2, nitrogen narcosis in 2, cramps in 1, cerebro-vascular accident in 1, cardiac disease in 1, chest infection in 1. Two divers were physically impaired.

Although there was very little medical information available on the majority of the divers, it was evident that at least 8 (10%) of them should have been classified as permanently medically unfit for diving because of their significant illnesses. As well as these, in 7 (9%) there was evidence of significant drug taking, and 5 (6%) had evidence of significant alcohol ingestion.

At least 14 (18%) were in the US armed forces at the time of the accident, although none were on active duty at the time of death.

#### EQUIPMENT FACTORS (40%)

In 32 (40%) cases, equipment either contributed to the death or prevented adequate rescue or resuscitation. In 11 (14%) there was an actual fault in equipment. Most of these failures (6) occurred with the buoyancy compensator. As well in 2 cases the regulator failed, in 1 the hose burst, in 1 the pressure gauge gave a totally misleading reading and in 1 the harness failed.

In 9 (11%) cases there was evident misuse of equipment. This was commonly associated with weight belt, harness or tanks. In 8 (10%) cases there was entanglement with lines, ropes, weight belt or harness.

In 20 (25%) cases, there was a failure to carry equipment which would have probably prevented the accident or allowed the diver to be rescued. In 13 cases (16%) there was no buoyancy compensator. Four divers did not wear a wet suit (5%), while 4 did not have a snorkel (5%). Not having a direction line in a cave led to the death of one diver, while the lack of a reserve lever for a J valve contributed to another. Some divers were without more than one life saving piece of equipment.

#### ENVIRONMENTAL FACTORS (56%)

In forty five deaths (56%) environmental factors contributed to death or prevented rescue. By far the commonest problem was related to water movement, either in the form of white water (reduced visibility, reduced buoyancy, increased water speed, trauma, etc.) or from waves, surf and tidal currents. In 32 of the 80 cases (40%) water movement contributed to the death.

The next most common environmental contributor was depth. Depths in excess of 30 m were required before a death was included in this category, but even then they were only so classified if it was thought that the depth itself was a definite contributor. This was so in 7 (9%) out of the

80 cases. Nitrogen narcosis or a loss of air supply contributed to most of these deaths. Depth was not specifically incriminated as a contributing factor in the deaths from decompression sickness, even though it obviously was a factor.

In 4 of the 80 (5%) cases, impaired vision associated with night diving, was a contributing factor. In 3 of the 80 deaths (4%) the diver was run over by a boat, and in 2 of these it was his own "safety boat"!

There was evidence of shark attack in two cases, but it was not clear whether the attack caused death or occurred after the death.

### Techniques contributing to deaths

#### COMPROMISED AIR SUPPLY (36%)

Incorrect technique contributed to many of the deaths. In 29 of the 80 cases (36%) the fact that the diver had a compromised air supply lead either directly or indirectly to death. In some of these the diver had reached reserve levels, and therefore had to take the action which caused the death, whereas in others the diver seemed to have inadequate air at depth.

#### BUOYANCY (27.5%)

With 22 of the 80 cases (27.5%) there were significant buoyancy problems which contributed to the death. Of these 19 (24%) were due to negative buoyancy and 3 (4%) had catastrophic positive buoyancy problems.

#### BUDDY DIVING

Buddy diving techniques were not carried out in most cases. In 42 deaths (52.5%) the failure to comply with the buddy system was a significant factor in preventing rescue and first aid. In 3 of these cases the buddy was in an invidious situation, being the dive leader of a "follow-me" team, who could not possibly have been aware of the victim's state until it was too late.

In 2 deaths there was attempted buddy breathing during ascent.

#### DITCH AND RECOVERY

In one case a "ditch and recovery" training technique was the direct cause of death.

### Conclusions

The data available on this population suggests that the Hawaiians were a very similar group of divers to those in

both the ANZ and NUADC series, and also to that reported in the general diving medical literature.<sup>7</sup> Specifically the high incidence of males compared to females, the surprising number of divers who died either during their initial dive or while under training, the observation that approximately half were experienced enough to undertake the dive and the neglect of the buddy system, was consistent in all three series.

About half had an accident either on or near the surface, but there was a greater number in the Hawaiian series who had dived to depths in excess of 30 m. All three series showed that accidents tended to happen at the start of the dive or following a compromised air supply situation.

The causes of death were very similar in the three series, with drowning the dominating diagnosis as a final cause of death. The only way in which this series differed was in the higher incidence of death from decompression sickness (5%), compared to the 0-1% in the other series. This tended to correlate with extremely deep diving and black coral collecting.

The factors contributing to death were consistent with the NUADC reports but were not of the same magnitude as the ANZ series, as would be expected because less data was collected.

Human factors contributing to death were seen in 44% of cases compared to 55.7% (NUADC) and 74% (ANZ).

In 40% of cases, equipment problems contributed to the death. In 14% the equipment was faulty; in 11% it was misused, while entanglement in equipment was present in 10%. In 25% adequate equipment was not available. This compares with equipment faults in 35% and misuse in 35% (with considerable overlap) in the ANZ series.

Environmental factors contributed to the death in 56% compared to 34.8% (NUADC) and 62% (ANZ). These incidences probably reflected the amount of data available.

The contribution of various diving techniques in the Hawaiian series was similar to the ANZ series. Unfortunately these categories are not easy to compare with the NUADC series. In 36% a compromised air supply contributed to the death. Buoyancy problems contributed in 27.5% and in 52.5% the failure to comply with the buddy system either contributed to the death or resulted in rescue being excessively delayed.

There are other specific references to Hawaiian diving accidents in the diving literature.<sup>9-11</sup>

### References

- 1 Monaghan R. The risks of sport diving. *SPUMS J.*

- 1988;18 (2):53-60
- 2 McAniff JJ. *United States underwater diving fatality statistics 1970-79*. Washington DC: US Department of Commerce, NOAA, Undersea Research Program, 1981
  - 3 McAniff JJ. *United States underwater diving fatality statistics 1986-87*. Report number URI-SSR-89-20, University of Rhode Island, National Underwater Accident Data Center. 1988
  - 4 Edmonds C and Walker D, Scuba diving fatalities in Australia and New Zealand. 1. The human factor. *SPUMS J* 1989; 19 (3): 94-104
  - 5 Edmonds C and Walker D, Scuba diving fatalities in Australia and New Zealand. 2. The environmental factor. *SPUMS J* 1990; 20 (1): 2-4
  - 6 Edmonds C and Walker D, Scuba diving fatalities in Australia and New Zealand. 3. The equipment factor. *SPUMS J* 1991; 21 (1): 2-4
  - 7 Edmonds C, Lowry C and Pennefather J. *Diving and Subaquatic Medicine 3rd Edition*. Oxford: Butterworth/ Heinemann, 1992: 354-361
  - 8 Lourey CJ. The cardiac reflexes revisited. *SPUMS J* 1981; 11 (Supp): 11-16
  - 9 Kizer KW. Dysbarism in paradise. *Hawaii Med J* 1980; 39 (5):109-116
  - 10 Farm FP, Hayashi EM and Beckman EL. *Diving and decompression sickness treatment practices among Hawaii's diving fishermen*. Sea Grant Technical Paper 86-01, University of Hawaii. 1986
  - 11 Erde A and Edmonds C. Decompression sickness - a clinical series. *J Occup Med* 1975; 17; 324-328

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**A PRELIMINARY REPORT ON A PROSPECTIVE RANDOMIZED, DOUBLE-BLIND, CONTROLLED STUDY OF OXYGEN AND OXYGEN-HELIUM IN THE TREATMENT OF AIR-DIVING DECOMPRESSION ILLNESS**

Alison Drewry and Des Gorman

**Abstract**

The treatment of Australasian recreational divers with decompression illness using the United States Navy

recompression algorithms has a high failure rate. Oxygen-helium gas mixtures may have some advantages over oxygen alone in such therapy, and consequently, a prospective randomized double-blind controlled study of oxygen and oxygen-helium in the treatment of air-based decompression illness has been initiated at the Royal New Zealand Naval Hospital in Auckland. Thirty patients have been studied in the first 4 months of 1992.

**Introduction**

The treatment of recreational air-divers with decompression illness (DCI) in Australasia is largely based on the "minimal recompression oxygen" tables promulgated by the United States Navy (USN) in 1965.<sup>1</sup> Although the USN, both initially and still, reports high resolution rates with the use of these treatments in its own naval divers,<sup>2,3</sup> this is not the current experience in injured recreational divers in Australasia. Failure rates (incomplete resolution of symptoms and signs) vary between 37% in Melbourne (1991; 100 divers),<sup>4</sup> 32% (neuropsychiatric sequelae) and 48% (abnormal EEG recordings) in Sydney (1987; 87 divers),<sup>5</sup> 54% in Auckland (1990; 125 divers)<sup>6</sup> and 54% in Adelaide (1988; 64 divers).<sup>7</sup> These failure rates do not vary significantly between facilities and the total number of patients treated and surveyed is large. It is also noteworthy that these failure rates exceed those reported in 1964 for both the 30 and 50 msw oxygen-nitrogen (air) recompression treatment tables.<sup>8</sup> Although these injured USN divers and their follow-up are not directly comparable with the nature and assessment of contemporary injured Australasian recreational divers,<sup>4,5</sup> the "high" failure rates reported in 1964<sup>8</sup> were used to justify the development of the 1965 alternatives (those in current use)<sup>1</sup> The same arguments then, used to introduce these "minimal recompression oxygen" tables can now be used to justify the development and testing of alternative therapies, at least for the treatment of recreational divers in Australasia.

The "minimum recompression oxygen" tables are a compromise between ambient pressure and oxygen toxicity, however the use of 2.8 bar inspired oxygen tension is nevertheless toxic to the injured brain.<sup>9</sup> An alternative is to use oxygen-helium mixtures at the same or greater ambient pressures, but such that the inspired oxygen tension is kept between 1 and 2 bar. The ideal inspired oxygen tension for treatment of DCI in vivo is 2 bar<sup>10</sup>, but the optimal dose of oxygen to inhibit bubble-induced polymorphonuclear leucocyte (PMNL) accumulation (see below) has not yet been determined.<sup>11</sup> Although some studies of cardiopulmonary decompression illness in dogs and guinea pigs have failed to demonstrate any advantage,<sup>12,13</sup> oxygen-helium breathing has resulted in faster shrinkage of air bubbles in rat adipose tissue<sup>14</sup> and spinal cord white matter<sup>15</sup> than when either air or oxygen are breathed. This is explained by net gas flux being determined by both gas solubility and diffusion. Importantly, there is no evidence that oxygen-helium breathing causes air bubbles to grow in aqueous tissues such as

skeletal muscle and tendon; instead, such bubbles have been shown to shrink.<sup>16</sup> There is also anecdotal clinical support for oxygen-helium treatment of DCI which develops following air diving.<sup>17</sup>

It is now apparent that significant pathology in DCI is due to the "biochemical" effects that bubbles have and not solely to the long-espoused "mechanical", "compressive" or "occlusive" effects. Both DCI evolution and sensitivity in rabbits is dependent upon complement protein activity.<sup>18-20</sup> The brain dysfunction that follow air embolism of the brain in rabbits<sup>21</sup> and dogs<sup>22,23</sup> is largely due to an accumulation of PMNLS and a consequent fall in brain blood flow. Not surprisingly, measures adjuvant to recompression that may ameliorate these "biochemical" effects are being sought; at present, only lignocaine infusion is ready for human trials. Lignocaine may act either by stabilising membranes or by inhibiting PMNL accumulation and its toxicity or both. It is effective in cats and dogs with DCI both prophylactically<sup>24</sup> and therapeutically.<sup>25,26</sup> The benefit of lignocaine may actually be additive to that of hyperbaric oxygen.<sup>26,27</sup> Lignocaine has already been used in humans with DCI.<sup>28</sup>

Consequently, it was decided to start a prospective, randomised, double-blind, controlled study of oxygen-helium in the primary treatment of air-diving DCI, and a secondary study of lignocaine versus a placebo in DCI refractory to recompression (This will be the subject of a further report).

## Methods

The trial has been approved by the Royal New Zealand Navy Human Ethics Committee and has begun at the Naval Hospital at Auckland. This facility treats about 40 to 50 recreational divers with DCI annually.<sup>6</sup> When database management and other protocol-specific issues are settled, it is intended to recruit both the Royal Australian Navy School of Underwater Medicine, at HMAS PENGUIN in Sydney, (about 25 to 30 divers a year)<sup>5</sup> and the Royal Adelaide Hospital Hyperbaric Medicine Unit (about 25 to 30 divers annually)<sup>7</sup> into the study, as both of these facilities are capable of oxygen-helium recompression.

All recreational divers with DCI after air diving who present for treatment, regardless of the duration of their signs and symptoms, are compressed to 2.8 bar absolute. All receive intravenous fluids (1 litre of normal saline over 1 hour and then 1 litre 4 hourly), but no chemotherapy. These patients are randomly allocated (Alpha and Bravo cards) to Group Alpha (50/50 oxygen-helium) or Bravo (100% oxygen). The randomisation is stratified into those presenting within 48 hours and those 48 hours or more after the onset of their symptoms. Neither the attendant medical officer nor the patient is made aware of the allocated group. The allocated gases are breathed on arrival at 18 m and for a further 45 minutes. If this produces an 80% or greater

improvement (see scoring system below), then a USN Table 6<sup>29</sup> pressure profile is completed, with extensions if the response is less than complete. Group Alpha breathes oxygen-helium instead of 100% oxygen and has no air breaks. Group Bravo has a standard USN Table 6.

However, if the initial period produces a less than 80% improvement, then the patient is compressed to 30 msw and given the pressure profile of USN Table 1A.<sup>29</sup> Group Alpha patients breathe 50/50 oxygen-helium and Group Bravo breathe 50/50 oxygen-nitrogen.

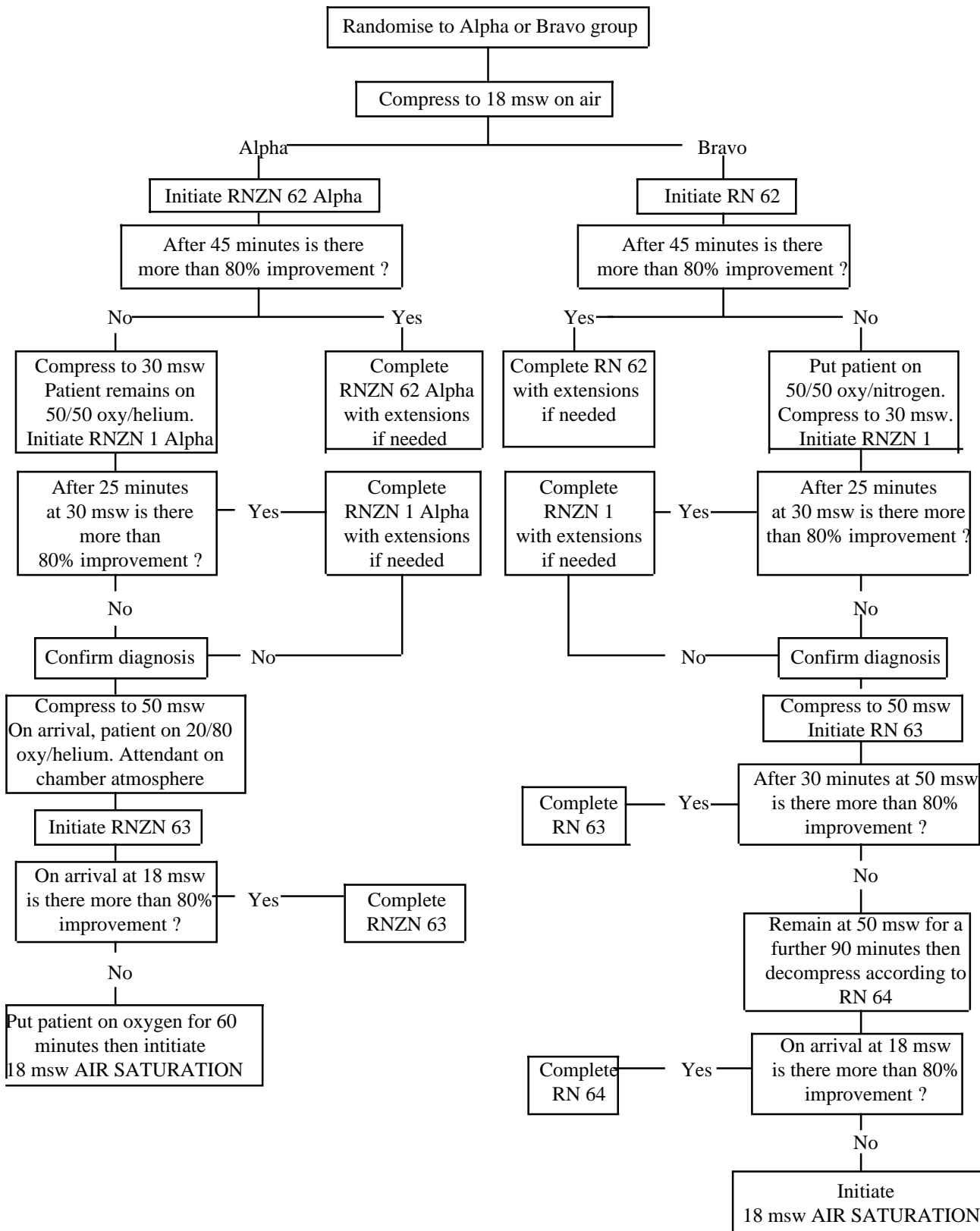
If the response to this is less than 80% improvement, then the patient is compressed to 50 msw and given the pressure profile of USN Table 6A<sup>29</sup> or RN Table 64.<sup>30</sup> Group Bravo have a standard Table USN 6A or RN Table 64, breathing air and oxygen. Group Alpha patients are limited to USN Table 6A and breathe 20/80 oxygen-helium and 50/50 oxygen-helium instead of air and oxygen. Repeat treatments, made necessary by relapsing or persistent problems, are conducted according to the Royal Adelaide Hospital protocol of 18 m for 60 minutes on oxygen with a 30 minute decompression (RAH 18/60/30). Group Alpha have 50/50 oxygen-helium, Group Bravo breathe 100% oxygen. Treatments are given daily until the problems resolve or two successive treatments do not produce any sustained improvement. A treatment algorithm for the study is shown in Figure 1.

Individual informed consent is obtained from each patient. In the event of any patient being unconscious or confused, consent for participation in the trial is sought initially from a first-degree relative. Women who are known or suspected to be pregnant are excluded from the trial.

Response is based on history and examination findings; and in particular, visual analogue scales for pain and sensation,<sup>31</sup> serial-7 performance (time and errors), memory of a Babcock sentence (number of trials), muscle power percentages and sharpened Romberg testing (percentage time stable). Outcome is assessed clinically as this is still the most sensitive measure available.<sup>32</sup> Assessments are made during the first treatment (frequency of compression to 30 and 50 msw and cost), after the first treatment (signs and symptoms), at the time of discharge from hospital (number of treatments, adjuvant care, signs and symptoms), at one and six months and one year later (signs and symptoms).

Data are stored in a dBase IV database and are subject to sequential analysis by an independent blinded observer. The study will be stopped either when the difference in outcome at one month reaches a significance level of 0.05 or group sizes of 50 are exceeded for both groups (and providing the groups are directly comparable). Relative group composition and outcome are tested by t-test (parametric data) and by calculation of Fisher's exact p value. When multiple simultaneous comparisons are made, the Bonferroni correction is applied.<sup>33</sup>

**RNZN HYPERBARIC UNIT  
TREATMENT FLOW CHARTS**



**NOTES**

All follow up treatments are to be on Table 18:60:30.

- Follow up treatments are to be on the gas they were treated on.

Follow up treatments are to continue until there is no sustained response to two successive treatments.

## Interim results

Thirty recreational divers have been treated for DCI at Royal New Zealand Naval Hospital in the first 4 months of 1992 and have participated in the study. No significant differences are manifest to date and the small groups are not yet directly comparable.

## Discussion

The need to test alternative treatment regimens for recreational divers in Australasia has been well established by an unacceptably high local treatment failure rate.<sup>4,7</sup> A potential role for oxygen-helium in lieu of 100% oxygen in recompression is shown in vivo,<sup>14-16</sup> and clinically, although anecdotally.<sup>17</sup> The trial described here should demonstrate any utility for oxygen-helium treatment in injured Australasian air-breathing divers.

The reasons for the difference in outcome between Australasian divers<sup>4,7</sup> and USN divers<sup>2,3</sup> when treated for DCI on USN algorithms<sup>29</sup> are uncertain (perhaps the delay prior to treatment is critical<sup>32</sup>), but are not the subject of and will not be answered by this study.

## References

- 1 Goodman MW and Workman RD. *Minimal recompression oxygen breathing approach to treatment of decompression sickness in divers and aviators*. USN Experimental Diving Unit Research Report 5-65, Washington, DC, 1965.
- 2 Workman RD. Treatment of bends with oxygen at high pressure. *Aerospace Med* 1968; 39: 1076-1083.
- 3 Green JSW, Tichenor J and Curley MD. Treatment of type I decompression sickness using the US Navy treatment algorithm. *Undersea Biomed Res* 1989; 16: 465-470.
- 4 Weinmann M, Tuxen D, Scheinkestel C and Millar I. Decompression illnesses. 18 months experience at the Alfred Hospital Hyperbaric Unit. *SPUMS J* 1991; 21(3): 135-143.
- 5 Gorman DF, Edmonds CW and Parson DW, et al. Neurologic sequelae of decompression sickness: a clinical report. In: Bove AA, Bachrach AJ, Greenbaum LJ Jr, Eds. *Underwater and hyperbaric physiology IX*. Undersea and Hyperbaric Medical Society, Bethesda, 1987, pp 993-998.
- 6 Brew SK, Kenny CT, Webb RK and Gorman DF. A factorial analysis of 125 diving accidents treated at HMNZS PHILOMEL. *SPUMS J* 1990; 20(4): 226-230.
- 7 Gorman DF, Pearce A and Webb RK. Dysbaric illness in South Australia, 1987. *SPUMS J* 1988; 18(3): 95-101.
- 8 Rivera JC. Decompression sickness among divers: An analysis of 935 cases. *Milit Med* 1964; 129: 31-334.
- 9 Holbach KH and Caroli A. Oxygen tolerance and the oxygenation state of the injured human brain. In: Trapp WG, Bannister EW, Davidson AAJ and Trapp PA, Eds. *Proceedings of the 5th International Hyperbaric Congress*. Burnaby: Simon Fraser University, 1974, pp 350-361.
- 10 Leitch DR and Hallenbeck JM. Oxygen in the treatment of spinal cord decompression sickness. *Undersea Biomed Res* 1985; 12: 269-289.
- 11 Zamboni WA, Roth AC, Russell RC, Suchy H and Kucan J. The effect of hyperbaric oxygen treatment on the microcirculation of ischemic skeletal muscle. *Undersea Biomed Res* 1990; 17(Suppl): 26.
- 12 Catron PW, Thomas LB, Flynn ET, McDermott JJ and Holt MA. Effects of He-O<sub>2</sub> breathing during experimental decompression sickness following air dives. *Undersea Biomed Res* 1987; 14: 101-111.
- 13 Lillo RS, MacCallum ME and Pitkin RB. Air vs He-O<sub>2</sub> recompression treatment of decompression sickness in guinea pigs. *Undersea Biomed Res* 1988; 15: 283-300.
- 14 Hyldegaard O and Madsen J. Influence of heliox, oxygen and N<sub>2</sub>O-O<sub>2</sub> breathing on N<sub>2</sub> bubbles in adipose tissue. *Undersea Biomed Res* 1989; 16(3): 185-194.
- 15 Hyldegaard O, Moller M and Madsen J. Effect of He-O<sub>2</sub>, O<sub>2</sub> and N<sub>2</sub>O-O<sub>2</sub> breathing on injected bubbles in spinal white matter tissue. *Undersea Biomed Res* 1991; 18: 361-371.
- 16 Hyldegaard O and Madsen J. Effect of heliox breathing on air bubbles in aqueous tissues after decompression. *Proceedings of the XVII annual meeting on diving and hyperbaric medicine*. European Undersea Biomedical Society, 1991: 75-80.
- 17 Douglas JDM and Robinson C. Heliox treatment for spinal decompression sickness following air dives. *Undersea Biomed Res* 1987; 15: 283-300.
- 18 Ward CA, Koheil A, McCulloch D, Johnson WR and Fraser WD. Activation of complement at the plasma-air or serum-air interface of rabbits. *J Appl Physiol* 1986; 60: 1651-1658.
- 19 Ward CA, McCulloch D and Fraser WD. Relation between complement activation and susceptibility to decompression sickness. *J Appl Physiol* 1987; 62: 1160-1166.
- 20 Ward CA, McCulloch D, Yee D, Stanga D and Fraser WD. Complement activation involvement in decompression sickness of rabbits. *Undersea Biomed Res* 1990; 18: 51-66.
- 21 Helps SC and Gorman DF. Air embolism of the brain in rabbits pretreated with mechlorethamine. *Stroke* 1991; 22: 351-354.
- 22 Hallenbeck JM, Dutka AJ and Tanishima T, et al. Polymorphonuclear leucocyte accumulation in brain regions with low blood flow during the early postischemic period. *Stroke* 1986; 17: 246-253.
- 23 Dutka AJ, Kochanek PM and Hallenbeck JM. Influ-

- ence of granulocytopenia on canine cerebral ischemia induced by air embolism. *Stroke* 1989; 20: 390-395.
- 24 Evans DE, Koblitz AI and LeGrys DC, et al. Protective effect of lidocaine in acute cerebral ischaemia induced by air embolism. *J Neurosurg* 1984; 60: 257-263.
  - 25 Evans DE, Catron PW and McDermott JJ, et al. Therapeutic effect of lidocaine in experimental cerebral ischaemia induced by air embolism. *J Neurosurg* 1989; 70: 97-102.
  - 26 Dutka AJ. Therapy for dysbaric central nervous system ischaemia; adjuncts to recompression. In: Bennett PB and Moon RE, Eds. *Diving accident management*. Bethesda MD, UHMS, 1990, pp 222-234.
  - 27 McDermott JJ, Dutka AJ, Evans DE and Flynn ET. Treatment of experimental cerebral air embolism with lidocaine and hyperbaric oxygen. *Undersea Biomed Res* 1990; 17: 525-534.
  - 28 Drewry A and Gorman DF. Lidocaine as an adjunct to hyperbaric therapy in decompression illness: A case report. *Undersea Biomed Res* 1992; 19: in press.
  - 29 *USN Diving Manual*. NAVSEA 0994-LP-001-9010.
  - 30 BR 2806. *Diving Manual D/DNW 102/4/31*.
  - 31 Scott J and Huskisson EC. Graphic representation of pain. *Pain* 1976; 2: 175-184.
  - 32 Moon RE and Gorman DF. Treatment of the decompression disorders. In: Bennett PB and Elliott DH. *The physiology and medicine of diving. 4th edition*. Balliere-Tindall, London, 1992, in press.
  - 33 Wallenstein S, Zucker CL and Fleiss JL. Some statistical methods useful in circulation research. *Circ Res* 1980; 47: 1-9.

**KEY WORDS**      Decompression illness  
 Oxygen  
 Oxygen-helium  
 Recompression

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## NATURAL OBSERVATIONS OF DIVING INCIDENTS

Jeffrey Wilks and Lindsay Christie

### Introduction

Safety has always been a concern for the recreational diving industry. While the major accident rate is very low compared to other sporting groups,<sup>1,2</sup> the consequences of an underwater accident can often be more serious than injuries on land. At a time when there is an increased willingness for the public to sue sports coaches and administrators for any alleged breach in their "duties of care",<sup>3,4</sup> the diving industry needs to look carefully at cost effective methods of improving safety. While formal legislation<sup>5</sup> or industry Codes of Practice<sup>6</sup> may provide frameworks for safety, other informal methods of preventing accidents should also be examined for their potential contribution.

The term "diving incident" has been used by Acott and his colleagues<sup>7</sup> to describe "An error by a diver, or a failure of his or her equipment to function properly. The error or failure could have led to more serious consequences, had it not been detected or corrected in time". An incident has the makings of an accident, where things actually do go wrong, but does not necessarily lead to an accident.

While the term "diving incident" is relatively new, scuba instructors have appreciated the importance of early recognition in accident prevention for some time.<sup>8,9</sup> Reviews of diving accidents consistently highlight some common factors contributing to the accident scenarios. These include medical and psychological factors, dangerous environmental conditions and equipment difficulties.<sup>10-14</sup>

Some of these problems may be difficult for a dive supervisor to overcome (e.g. undetected faulty personal equipment), whereas other problems might be prevented with detailed dive briefings and greater awareness of areas where accidents are likely to occur. For example, after reviewing 264 Japanese diver fatalities Mano and Shibayama<sup>15</sup> concluded that poor diving technique and reckless diving were the main causes of fatalities. They also noted that many accidents they investigated could be predicted on the basis of their non-existent or inadequate dive planning, and only a few accidents occurred that could not have been prevented.

The Diving Incidents approach to accident prevention suggested by Acott and his associates<sup>7</sup> focuses attention on those areas of activity where problems are likely to arise. In their pilot study they asked divers to record any incidents they had observed during a dive on a questionnaire report form. Of the total of 69 incidents reported, 36 occurred during the dive itself. A further 15 incidents occurred during preparation for the dive, and another five during entry.

The written report methodology presents several problems if this type of study is to be extended beyond a small group of interested divers. First, written reports require commitment and a large degree of organisation. Popularising reporting requires education of the reporters. Obtaining reports about serious accidents is a difficult process, only made possible through legal requirements and insistence by training agencies and government. Harmless incidents are likely to be overlooked entirely.

The second problem with written reports is the embarrassment factor.<sup>16</sup> Some divers feel embarrassed about asking others to spend time on a buddy check, especially if the buddy pair are strangers before the dive. In this situation, minor problems with setting up equipment and preparing to enter the water will either be not detected, or not commented upon by the buddies.

Lastly, new divers are most likely to experience difficulties, especially if they are using unfamiliar rented or borrowed equipment.<sup>11</sup> In Japan Mano and Shibayama<sup>15</sup> found that 30.8% of their 130 fatal cases involved novice divers on their first open-sea dive. While new divers are concentrating on not making mistakes themselves they would not be in any position to observe and report on the incidents occurring around them.

The limitations of a written record format for investigating diving incidents in no way detracts from the extremely valuable pilot work of Acott and his colleagues.<sup>7</sup> Recording patterns of incidents provides an excellent mechanism for identifying the early warning signs of diving accidents. An alternative method of gathering such data is to record observations of divers on a commercial dive boat, which was done in the present study.

## Methods

### PILOT STUDY

To identify focal areas of diver safety for the main study, a pilot project was conducted on the 35 metre M.V. Capricorn Reefseeker. The Reefseeker is a commercial vessel that runs between Great Keppel Island and North West Island on the outer Barrier Reef three times a week. Between October and December 1990, observations about 65 certified divers were recorded.

Data were collected unobtrusively at the time these divers completed their paperwork on the day of the dive. From their certification cards and log books the second author noted age, sex, date certified, certification level, total number of dives since certification, number of dives in the past 12 months, and city or country of origin. Where this information was not readily available from written records, friendly questions were asked in general conversation to elicit the missing data.

The pilot study included 38 men and 27 women; 58% of the sample were Australian and 42% were overseas visitors. Most divers held an open water certification (85%), though all levels of training and all major training agencies were represented. Just over half of the sample (54%) had been certified for 12 months or less, while 22% had been certified for more than five years. Diving frequency in the previous 12 months ranged from none to over 100 dives (a travelling instructor); 15% reported no dives in the past year, 54% less than 10 dives, 11% with 10-20 dives, and 20% with more than 20 dives. Overall, the pilot group seemed to represent a wide cross-section of recreational divers.

Comments on diving incidents were made next to each diver's identification on monitoring sheets disguised under briefing notes on the Divemaster's slate. The comments were unstructured during the pilot phase and included all observations that might later be useful in the design of the major study. On board the Reefseeker divers kit up on a 10 x 5 m rear deck, then descend six stairs to a 10 m platform just above the water. A Giant Stride entry is made from the platform. The Reefseeker moors stern in to the reef face, with the water depth under the rear platform about nine metres. Diving at North West Island is very easy, with no necessity to dive deep or to swim very far away from the vessel. Under these conditions it is not difficult for the Divemaster to observe accurately and record most behaviour on the surface.

### THE MAIN STUDY

Drawing on the observations of the pilot project, and also previously identified problems associated with open water dives,<sup>7,17</sup> the Queensland Diving Incident Checklist was developed. This was pilot tested by two independent instructors and further refined to the final version presented in Figure 1.

The unobtrusive observational design of this study, and the use of a "dry" divemaster, restricted reporting to those events witnessed on the surface. The Incident Checklist therefore concentrates on diving incidents as they occur during pre-dive equipment assembly and dive preparation. Minor breaches of diving safety on the surface of the water and during the post-dive period were also recorded. Since several recent studies in Queensland had revealed a very similar demographic profile for certified recreational divers,<sup>18-20</sup> a decision was made not to alert subjects in the main sample to the research by collecting information about them or their diving history.

### Subjects and procedures

A total of 192 certified scuba divers visiting North West Island on the M.V. Capricorn Reefseeker between December 1990 and May 1991 were included in the study.



**FIGURE 1**

**QUEENSLAND DIVING INCIDENT CHECKLIST**

D.M. Date	No. in Group	TOTALS
<b>Equipment and dive preparation</b>		
1	Faulty assembly	
2	Forgotten/lost gear	
3	Gear breakage	
4	Not using octopus (O) watch (W)	
5	Unsafe turning air on	
6	Free-flowing regulator	
7	Don tank over head	
8	Mask on forehead	
9	Snorkel wrong side (WS) /missing (X)	
10	Carrying too much gear	
11	No buddy assistance	
<b>Incomplete pre-dive check</b>		
12	Buoyancy	
13	Weights (LH release = LH, missing = X)	
14	Releases	
15	Air (not turned on)	
16	No pre-dive check	
<b>Other</b>		
17	Overheating	
18	Clearly nervous	
<b>During and After Dive</b>		
19	Lost buddy	
20	Equipment problems	
21	Needed assistance (A) /rescue (R)	
22	Exceed tables (T) /briefing (B)	
23	Return with less than 50 bar	
24	Not acknowledging boat from water	

**OTHER COMMENTS (WEATHER ETC)**

The second author again recorded diving incidents in an unobtrusive manner, this time on the structured Incidents Checklist. Records were kept for 36 separate reef visits. Groups ranged in size from only one diver (buddied with a divemaster) to a total of 14 on one occasion. On only two of the 36 days were there no incidents observed.

**Results**

Table 1 presents the main diving incidents observed in this study.

**TABLE 1**

**DIVING INCIDENTS OBSERVED**

Area	Number of Divers	%
<b>Equipment and dive preparation</b>		
No buddy assistance	73	38
Faulty assembly	55	29
Unsafe turning air on	23	12
Not using octopus or watch	18	9
Forgotten/lost gear	16	8
Free-flowing regulator	10	5
Snorkel wrong side or missing	9	5
Mask on forehead	7	4
Donned tank over head	6	3
Clearly nervous	4	2
Gear breakage	2	1
Carrying too much gear	2	1
<b>Incomplete pre-dive check</b>		
No pre-dive check	109	57
Buoyancy	72	38
Releases	19	10
Weights (LH release or missing )	17	9
Air not turned on	13	7
<b>During and after dive</b>		
Not acknowledging boat from water	90	47
Exceeded tables or briefing	28	15
Returned with less than 50 bar	24	13
Lost buddy	23	12
Equipment problems	22	11
Needed assistance or rescue	14	7

Lack of buddy assistance was commonly observed during dive preparation. Divers often struggled into their gear while the buddy stood and talked to them. Just under one third of the sample (29%) had trouble assembling their equipment. This included buoyancy compensation devices (BCDs) facing the wrong way on the tank (or upside down), and attempts to attach the regulator first stage by screwing the yoke into the tank valve opening. Holding the contents gauge in front of one's face as the tank valve is turned on (and risking injury if the gauge explodes) was the next most common equipment incident.

Three divers kitted up without an octopus regulator on their personal gear. Following an explanation about Queensland regulations<sup>5</sup> they were given hire equipment. A total of 17 divers prepared to dive without a watch or timing device. Other common equipment incidents included forgotten or lost gear (8% of the sample), free-flowing regulators (5%), gear breakage (1%) and carrying too much gear (1%). While perhaps not serious, six divers prepared to dive

without a snorkel (another three wore theirs on the wrong side), six divers donned their BCDs (with tank) over their heads, and seven divers unknowingly signaled "distress" by wearing their masks on their foreheads. Two divers were observed to be clearly nervous during the process of dive preparation.

Lack of buddy assistance also emerged in the area of pre-dive equipment checks. In general, 57% of the sample made no attempt to run through a pre-dive check. This neglect was obvious in the area of buoyancy, where many divers did not connect their power inflator to the BCD. Weight belts were often observed to be twisted and the quick-release buckles not done up. Two divers prepared to enter the water without weightbelts, while a further 15 were recorded having the release flap on their weightbelts positioned so as to require the left-hand to be used to open it. Thirteen divers (7% of the sample) were prepared to enter the water without turning their air on!

Upon entering the water initially, or coming to the surface during the dive, 47% of the sample failed to signal the boat that they were OK. Directions given during the dive briefing (usually about time to return to the vessel) were disregarded by 15% of the sample. While the briefing contained a request for divers to return from their dive with at least 50 bar in reserve, 13% of the sample failed to comply. Buddy separation (12%) and equipment problems (11%) were also observed during and after the dive. Finally, one diver was rescued following a panic attack in the water, and 13 others needed assistance during the dive.

## Discussion

Lack of buddy assistance emerged as the main diving incident observed in this study. This included little help by partners in setting up equipment and dive preparation, infrequent pre-dive safety checks, and 23 situations involving buddy separation in the water. The buddy system is a tried and proven method of promoting safety among divers, but it relies on the two partners staying together and being able to communicate effectively during the dive.<sup>16</sup> Supervisors should be aware that many divers may be shy and embarrassed when "buddied up" with a stranger for the first time. They will look to the Divemaster for assistance rather than their buddy, or alternatively struggle through pre-dive preparations by themselves, all the while becoming more nervous or distressed before even entering the water.<sup>8</sup>

Lack of familiarity with diving equipment, especially rental gear, may compound any discomfort a diver may feel about their readiness to dive. The Divemaster cannot always attend to minor trouble-shooting activities (e.g. free-flowing regulators) so the buddy pairs must be encouraged to assist each other.<sup>21</sup> This is particularly important with the pre-dive check. Fead<sup>16</sup> suggests emphasising to divers that the check is for rescue purposes, to save one's

"own" life, and therefore the responsibility is for a person to insist that their partner be totally prepared to help them before entering the water. This includes physically checking that the BCD will inflate and deflate, releases are clear, the weightbelt has a right-hand release, and that the air is turned on prior to preparing to enter the water. These same types of incidents were reported in written form by divers in the Acott et al. study.<sup>7</sup>

Merriman and Conn<sup>4</sup> argue that providing a safe environment is the greatest step that one can take to avoid sport injury litigation. Effective use of the dive briefing will go a long way to ensuring that divers understand what is expected of them prior to entering the water. For example, a review of non-verbal safety signals (including how to signal OK to the boat after initial entry or upon surfacing during the dive) would clarify and standardise signals for buddy pairs who had not dived together before. As the present study shows, some divers will still disobey instructions given during the dive briefing, but safety will nevertheless be enhanced if emphasis is placed on local safety procedures to be followed during the dive.

Supervisors should also be aware that peer pressure and social evaluation are very strong factors that may detract from safety in a recreational diving setting. For example, Griffiths and Heyman<sup>22</sup> found that pre-dive anxiety was not caused by concern over potential danger, but rather by perceived social evaluation. In that study, females were more likely than males to be anxious about performing physical tasks in front of others. While the present study did not distinguish between male and female divers, many of the problems with faulty equipment assembly were clearly related to social nervousness.

Divemasters are trained to correct faults before they become a problem. One of the difficulties in this study, for the second author, was achieving a balance between the working role of a Divemaster and the role of an impartial observer. At times it was very busy on the back deck of the Reefseeker as divers assembled their equipment. Correcting faults as they happen, as well as providing friendly encouragement, definitely boosts the confidence of nervous divers. Unfortunately, there were times when the two roles were at odds. Ideally, in future studies the observer should have no other duties. This would also allow natural observations to be made underwater, where Acott and his associates report that most diving incidents are likely to occur.<sup>7</sup>

## References

- 1 Centre for Health Promotion and Research. *Sport Injuries in Australia: Causes, Costs and Prevention*. A Report to the National Better Health Program. Sydney: Centre for Health Promotion and Research, 1990
- 2 Queensland Dive Tourism Association of Australia.

- Dive Tourism Accident Bulletin. *Underwater Geographic* 1990, 28: 72-73
- 3 Rathie DS. Sporting injuries and the law. *Queensland Law Society J* 1988; 18(2): 101-106
  - 4 Merriman J and Conn JH. Sport law: a social perspective. *J Sport Soc Iss* 1988; 12(2): 97-107
  - 5 Department of Industrial Affairs. *Workplace Health and Safety Regulations*. Brisbane: Queensland Government Printer, 1989
  - 6 Division of Workplace Health and Safety. *Information Paper on the Review of Regulation of the Diving Industry under the Workplace Health and Safety Act, Queensland: 9 September 1991*. Brisbane: Department of Employment, Vocational Education, Training and Industrial Relations, 1991
  - 7 Acott C, Sutherland A and Williamson J. Anonymous reporting of diving incidents: a pilot study. *SPUMS J* 1989; 19(1): 18-22
  - 8 Widmann B. The early warning signs of diving accidents. In: Fead L, ed. *Proceedings of the 7th International Conference on Underwater Education*. Colton, California: National Association of Underwater Instructors, 1975: 448-455
  - 9 Williams W and Williamson J. Recognising the distressed diver. *SPUMS J* 1986; 16(1): 16
  - 10 Hardy J. Diving accidents - why? In: Fead L, ed. *Proceedings of the 9th International Conference on Underwater Education*. Colton, California: National Association of Underwater Instructors, 1977: 97-115
  - 11 Divers Alert Network. *Report on 1988 diving accidents*. Durham, North Carolina: Duke University Medical Center, 1989
  - 12 Edmonds C and Walker D. Scuba diving fatalities in Australia and New Zealand. Part 1. The human factor. *SPUMS J* 1989; 19(3): 94-104
  - 13 Edmonds C and Walker D. Scuba diving fatalities in Australia and New Zealand. Part 2. The environmental factor. *SPUMS J* 1990; 20(1): 2-4
  - 14 Edmonds C and Walker D. Scuba diving fatalities in Australia and New Zealand. Part 3. The equipment factor. *SPUMS J* 1991; 21(1): 2-4
  - 15 Mano Y and Shibayama M. Aspects of recent scuba diving accidents. *Marine Tech Society J* 1989; 20: 38-41.
  - 16 Fead L. Buddies speak out! In: Boone C, ed. *Proceedings of the 11th International Conference on Underwater Education*. Colton, California: National Association of Underwater Instructors, 1979: 37-45
  - 17 PADI. *Instructor Development Course Candidate Workbook*. Santa Ana, California: Professional Association of Diving Instructors, 1988
  - 18 Wilks J. Kitting up: an equipment profile of Queensland divers. *SPUMS J* 1990; 20(4): 200-205
  - 19 Wilks J. Balancing recreation and safety: equipment requirements for Queensland scuba divers. *J Occup Health Safety* 1991; 7(3): 221-227
  - 20 Wilks J. Safety signals and procedures in the recreational diving workplace. *J Occup Health Safety* 1992; 8: in press
  - 21 Graver D. The role and responsibilities of NAUI divemasters. *NAUI Diving Association News* 1989; Jan/Feb: 26-29
  - 22 Griffiths T and Heyman S. Psychological and behavioral factors affecting scuba diving performance. In: Bangasser S, ed. *Proceedings of the 16th International Conference on Underwater Education*. Montclair, California: National Association of Underwater Instructors, 1985, 91-105

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## SPUMS NOTICES

### CHANGE OF ADDRESS

Unfortunately The Australasian College of Occupational Medicine (ACOM) has decided to become a Faculty of the Royal Australasian College of Physicians and will not be able to provide a permanent mailing address for SPUMS beyond December 1992.

The Council of the Australian and New Zealand College of Anaesthetists (ANZCA) has kindly consented to provide SPUMS with a permanent address.

All correspondence, **addressed to office holder concerned**, should be sent to

SPUMS, C/o  
Australian and New Zealand College of Anaesthetists,  
Spring Street, Melbourne  
Victoria 3000, Australia.

### SPUMS ANNUAL SCIENTIFIC MEETING 1993

will be held at  
the Palau Pacific Resort  
**SUNDAY 16TH TO TUESDAY 25TH MAY 1993**

The guest speaker will be Professor David Elliott.  
The theme of the conference will be  
**THE LONG TERM EFFECTS OF DIVING**

There will be a workshop on  
**FREE ASCENT TRAINING**

Anyone wishing to present papers should contact the Convener,

Dr Des Gorman, at the RNZN Hyperbaric Unit,  
Naval Base, Devonport, New Zealand.

Allways Travel have sent members and associates a booking form during September 1992. This should be returned with a deposit of \$Aust 600 per person, before the **14th of December 1992**. Every attempt will be made to provide accommodation for those who decide, after that date, to attend but they may be disappointed.

For further information contact

Allways Travel  
168 High Street, Ashburton,  
Victoria 3147, Australia.

Telephone	Australia	03 885 63
	International	61-3-885 8863
Fax	Australia	03-885 1164
	International	61-3-885 1164

### MINUTES OF THE 1992 ANNUAL GENERAL MEETING OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY held at PORT DOUGLAS on Sunday 5th June 1992

#### 1 Present

All members attending the Annual Scientific Conference.

#### 2 Apologies

Drs G Barry, J McKee, A Veale and A Slark.

#### 3 Minutes of the Minutes of Previous Meeting

These having been published in the Journal a motion that these were an accurate record was moved by Dr J Knight and seconded by Dr D Wallner. Carried.

#### 4 Matters arising from Minutes

No matters arising from the minutes were raised.

#### 5 President's report

Dr D Gorman presented the President's report. See pages 149-151.

#### 6 Secretary's report

Presented by Dr Darrell Wallner. See page 151.

#### 7 Treasurer's report

##### 7.1 ANNUAL FINANCIAL STATEMENT

Presented by Dr J Knight in Dr G Barry's absence. See pages 151-154.

##### 7.2 ANNUAL SUBSCRIPTION

Proposed by Dr J Knight, seconded by Dr D. Wallner, that the annual subscription for Full Members be increased to \$80 and Associate Members to \$40. Corporate Members \$80.

During discussion it was pointed out that a comparable sized society, the Australian and New Zealand Intensive Care Society (ANZICS) ran at a profit, but they did not produce a costly journal. The advisability of maintaining a secretariat to handle routine work was recommended by Dr Tuxen, from his experience with ANZICS. However, ANZICS makes a large profit from the trade exhibition associated with their AGM. This avenue of raising finances is not available to SPUMS and our finances do not allow a costly added service. Our permanent address remains at C/o the Australasian College of Occupational Medicine (ACOM).

It was approved that future financial statements would have a column for anticipated profit and loss.

The cost of mailing the Journal overseas, which often arrived late and in poor condition, was discussed and the practicality of bulk posting to the North American Chapter office and then to be distributed will be investigated.

The motion for the increased annual subscription was carried unanimously.

A second motion, that the SPUMS' subscription year be changed from July to June to January to December to coincide with the Journal year, was passed unanimously.

A third motion, that the subscription due in July 1992 should cover the 18 month period to December 1993, was also passed unanimously.

As a result in July 1992 an eighteen month subscription, to December 1993, will be due.

Members	\$120
Corporate Fee	\$120
Associates	\$60

**8 Editor's report**

Presented by Dr J Knight. See page 154.

**9 ANZHMG annual report**

Presented by Dr J Williamson. See page 154.

**10 Election of office bearers**

As only the following nominations had been received there was no need for an election. Next year's office bearers will be

President	Dr G Gorman
Secretary	Dr D Wallner
Treasurer	Dr S Paton
Education Officer	Dr D Davies
Editor	Dr J Knight
Public Officer	Dr J Knight
Committee Members	Dr C Acott Dr G Williams Dr J Williamson

**11 Appointment of Auditor.**

Mr D Porter, FCA, was re-appointed as auditor.

There being no business of which notice had been given, the meeting was closed at 17.40.

**PRESIDENT'S REPORT**

This is my second report as President of SPUMS and I am happy to say that our progress as a Society continues. Our goal being a mature responsible medical and paramedical group with a declared orientation to recreational diving.

Nevertheless, we clearly are interested in other types of diving and in hyperbaric medicine, as testified by our sponsorship of the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG).

I would like to address some issues briefly. The order is entirely random.

**The Society's Finances**

As John Knight will discuss on behalf of Grahame Barry, our absent and retiring Treasurer, SPUMS has to make some changes to the way in which it conducts its finances, and in particular, sets its annual fees. This arises for the following reasons: firstly, SPUMS has grown very rapidly and now has about 1,200 members and associates; secondly, the radical change in the production of our Journal, has not surprisingly, resulted in increased costs of production and this is compounded by increased postage costs; thirdly, both trials at using secretarial services to perform routine management tasks have been unsuccessful in that they have become clumsy and very expensive; and fourthly, our financial year is out of phase with both our subscription and Journal years, which are similarly out of phase such that there is always a misleading 3 month period of financial excess or shortfall.

If nothing else changed, that is, our membership remained static, fees were held at existing levels and current management practices continued, then in the year ahead, SPUMS will receive about \$50,000 but will incur costs of \$71,000. Our decision at last year's AGM to hold fees was almost certainly an error and in the last year we have consumed our cash reserves. Unless we reduce the number of Journals we publish, for example from 3 monthly to 4 monthly, an increase in fees is inevitable. Our proposal will be outlined later in this meeting. To minimise the effect on our membership, we also intend to reduce our secretarial costs dramatically. By severing our relationship with the Brighton Executive Centre and changing our modus operandi to a casual, and as required, use of local secretarial service we very conservatively, reduce our anticipated operating costs for the forthcoming year from \$25,000 for equipment and secretarial services to \$12,000. This reduces our anticipated overall expenditure for next year to \$58,000, enabling a modest rise in fees.

Not surprisingly, we also intend to bring the financial, subscription and Journal years into line. This will be based on the calendar year and will be achieved by the next subscription fee being levied for 18, rather than 12, months. Finally, we now have a credit card facility for membership fees such that payment should be less trouble for non-Australians.

I hope you will support these initiatives. We should not even consider reducing the frequency of our Journal and

consequently, alternatives such as those I have just briefly outlined, become compelling.

### **The Executive Committee**

The telephone conference system for both ourselves and the ANZHMG is a great success. We have reduced our costs for meetings and travel, despite our involvement with Standards Australia, from \$12,000 in the 1990/1991 year to \$3,000 last year.

I must thank our retiring Treasurer, Dr Grahame Barry, for his efforts and pass on our appreciation to his replacement, Dr Sue Paton. Given my earlier comments about our finances, it should not surprise anyone to learn Sue's job will be demanding and difficult. Nevertheless, I am confident that the new system we have in place will be efficient and cost-effective.

I would also like to thank all members of the Committee and especially our Secretary, Dr Darryl Wallner.

### **SPUMS Journal**

We should all be proud of our Journal. It continues to improve academically without losing its orientation or readership. Our congratulations must be expressed to the Editor, Dr John Knight and to Dr John Williamson, for his assistance in reviewing submissions. Index Medicus recognition is anticipated.

### **Dip DHM**

The Dip DHM continues to be awarded and is now recognised, or about to be recognised, by all of Australia's Medical Boards. Anyone interested in applying should contact our Education Officer, Dr David Davies.

### **ANZHMG**

The ANZHMG is now a reality, operates as a Standing Committee of SPUMS and much credit is owed to its two inaugural office-bearers, Dr John Williamson and Dr David Tuxen. I look forward to their meeting this afternoon and hope that the interests of hyperbaric technicians and nurses from around Australia can be accommodated by the ANZHMG.

### **Membership**

An issue for the forthcoming year in the status of some groups within SPUMS and in particular, full-time employed diving administrators, hyperbaric technicians and

nurses. It might be appropriate to confer full membership on these people, along with voting rights and full membership fees.

### **North American Chapter**

In the last year, more than 100 new members have joined via the North American Chapter of SPUMS. We are delighted with this response and wish to publicly thank the office-bearers, Dr Ray Rogers, who is not able to be here, Dr Lori Barr and Steve Dent.

### **AGM**

The decision to hold an AGM in Australia was taken with some reservation. I am pleased that it has been a success, and again would like to thank the convener, Dr John Williamson. I know that John would insist on acknowledging the assistance he has had from Dr Michel Pichon.

Next year's AGM is at Palau. Our theme is "The Long-Term Problems of Diving" and our guest speaker is Professor David Elliott. The interest already shown by members is extraordinary, such that we are now concerned about being able to accommodate all members at the Palau Pacific Hotel and to supply enough diving boats.

Consequently, we have decided to advance the booking deadline to 14th December, 1992 and to increase the deposit to \$600. Based on the numbers booked at that date, we will reserve rooms and diving boats. Members can apply to attend the meeting after that date, but will have to take "pot luck" with the availability of rooms and diving.

The 1994 AGM is scheduled for PNG and Dr Chris Acott is the convener. Chris will have a program available in December, 1992. The tentative theme is "The Causes of Diving Accidents". Professor Peter Bennett has agreed to participate.

Two final points about our AGMs. Firstly, the registration fee will continue to be received and set by the Committee, and secondly, Allways Travel and four other travel companies have been asked to prepare draft programs and cost estimates for the 3 year period beginning 1995. This does not reflect any dissatisfaction with Allways but is consistent with the policy agreed last year of arranging AGMs in 3 yearly blocks and of reviewing our travel arrangements before each block is accepted.

### **Standards Australia**

Finally, our involvement with Standards Australia in trying to produce a sensible standard for recreational divers, in contrast to the ridiculous occupational standard, has

continued. Our primary belief, that diving fitness medical examination must be performed on all candidates for recreational diving instruction and by an appropriately trained medical practitioner has been widely contested, and surprisingly, to me at least, the latter condition was opposed by the AMA. We have good data showing the need for such training for medical practitioners and the AMA's opposition is consequently difficult to accept. We have no intention of relaxing our stance.

### Conclusion

In conclusion, SPUMS is growing and maturing. Such change is exciting but does have a recognisable cost. We must not lose sight of our primary goals. In particular, SPUMS must remain good fun. I thank you for your support and look forward to seeing you all in Palau.

Des Gorman  
President of SPUMS

as co-conveners. This was well attended and successful with Dr C Edmonds as key-note speaker. We feel that March is the best time for these Meetings. We appear to be succeeding in gaining the support of doctors in these areas who are not normally involved in diving medicine.

We are planning our next Meeting for Nelsons Bay with Dr P Pidcock acting as convener.

### DIVING DOCTORS LIST

I have had many complaints from members who have been left off our list. The reason is that they did not notify me that they wanted to be on it! We are gradually getting this list up to date. In future this list will be updated regularly and automatically by a questionnaire with the the annual subscription notice.

Darrell Wallner  
Secretary of SPUMS

### THE SECRETARY'S REPORT

I was appointed at short notice because of Dr J Robinson's resignation. I have found being Secretary interesting and instructive. I believe that the changed secretarial arrangements just outlined by Des Gorman will be more efficient and cheaper than before, though there will be an increased load upon the Secretary and the Treasurer, who will have the new computer to maintain the membership list and accounts. The computer will make regular reports of our financial state easy.

The Committee has met three times by telephone (teleconference) during the year. These were effective and much more economical than face to face meetings.

Membership has increased to approximately 724 Australian members and associates and 160 Americans.

### NORTH AMERICAN CHAPTER

The formation of the North American Chapter has been a success story due to the energy and enthusiasm of Ray Rogers, Lori Barr and Steve Bent. We welcome the presence of Steve and Lori here. I would recommend that you read the Newsletter on the notice board which indicates how active they have been in promoting the North American Chapter.

### EDUCATION

The second of our weekend Annual Regional Meetings was held at Tathra with Drs G Long and J McKee acting

### TREASURER'S REPORT 1992

Unfortunately I am unable to attend the Annual General Meeting and I have asked Dr John Knight to present the report on my behalf.

Let me start by giving a brief annotation of the accounts (see page 152) before you .

You will notice that our income is slightly down and our expenditure significantly increased. The only bright spot in the "Expenditure" column is that our "Conference and travel" section is down nearly \$8,000 on last year. This is due to the fact that no travel expenses have been incurred this year (whereas the Standards Committee meetings of last year entailed many interstate visits on several occasions), and the fact that in our phone conferences under the iron hand of Des Gorman's nigh legendary chairmanship, all attempts at superfluous verbiage and gratuitous jocularity, except his own, are immediately quashed.

All together, our income-expenditure balance is, for the first time in SPUMS history, in the red by almost \$12,000 and we have had to seek a loan to cover this year's expenditure.

I have explained before that, with our financial year ending on April 30th and our subscription year ending on June 30th, there is always a two month period, after these accounts are prepared, for bills to be paid before the new subscription fees begin to be received.

A further loan will have to be sought to cover this period of time, and I will shortly be proposing a motion to overcome this anomalous state of affairs.

This last year has seen a large and unexpected increase in expenditure. Journal postage is now over \$ 1,000 an issue. Twice this year, when sending out the constitution and the Diving Medical, postage has cost over \$ 2,000. The rise in costs has been dictated largely by changes in the way the Society manages its affairs. Instead of the Secretary doing all his own homework, we have employed a secretarial agency to do the routine office work, such as the sending out letters of welcome and application forms. These routine things are done at the moment, in Melbourne, at the Brighton Executive Centre. All mail goes to the Post Office box, Care of the Australasian College of Occupational Medicine and it is then forwarded onto the secretarial agency. Hiving off the routine work has reduced the workload on the Secretary remarkably. Unfortunately, paid labour costs more than voluntary and we have had to pay out more than was expected. This has largely been due to the extra number of enquiries and the new members we have received this year compared with previous years. As we pay for secretarial services by the hour this means extra costs.

Two years ago we decided to use the Science Centre Foundation (SCF) as a secretariat to relieve the Secretary of routine correspondence. Within months it became obvious that the Society would soon be bankrupted by their charges, which were much larger than suggested at the 1990 AGM, for what proved to be an unsatisfactory service. The Treasurer, Grahame Barry, the Secretary, John Robinson and the Editor, John Knight, formed a sub-committee which recommended that :

- 1 The permanent address problem be solved by using the Australasian College of Occupational Medicine as our address.
- 2 The Secretary should choose a conveniently located secretarial service to handle his work.
- 3 The Treasurer should continue to handle subscriptions (This had not been delegated to SCF) and also payments.
- 4 The Editor should continue to maintain the mailing list (list of members).

The contract with the SCF was terminated in November 1990, but bills continued to arrive until 1992.

The final scheme has worked well, except for complaints from the Editor about the amount of time involved. When Darrell Wallner became Secretary he elected to leave the routine work to be done by John Robinson's chosen secretarial agency in Melbourne and to use a Canberra agency for his own correspondence. Darrell has foreshadowed that 1992-1993 will be his last year as Secretary.

On the Brighton Executive Centre's figures their charges for the extra work for running the mailing list and

sending out bills are expected to be about double what we pay now. I consider it prudent to assume that they have underestimated and that the rise will be twice as much as they think. The Executive Committee has decided that this is too expensive for our weakened finances and that the officers will once again have to do the routine functions, ones that take a great deal of time for the Editor, the Treasurer and the Secretary. They have been told to use casual secretarial assistance as they deem necessary as we can afford this level of expense. SPUMS now has over a 1,000 members.

In this age of cellular telephones, computers, faxes, answering machines and high powered medical technology, an ex-GP who has barely advanced beyond the era of ledger, quill pen and abacus, and who has not practiced medicine in Australia since January 1958, would seem a most unlikely person to lead SPUMS into the heady realms of modern financial management.

In addition, I look forward to becoming even more erratic in my movements and eccentric in my behaviour in the coming years, so a more stable and accessible officer would appear to be indicated.

We therefore propose further changes to make the job of the new Treasurer less onerous, so that one no longer needs to be retired to do it !

We have ordered a Macintosh computer, with appropriate software (M.Y.O.B. and ClarisWorks) and a printer. The mailing list and accounts will be kept by the Treasurer on the new computer. I have to emphasise that those who are more than 5 months behind in their subscription automatically get removed from the Journal mailing list. If they then pay they have to go back on the mailing list and this involves a great deal of extra work as many people send in their cheques any time up to 9 months late. By using a computer it is much easier to send out repeated reminders than it is with the manual system that I have used for many years. We have redesigned the Annual Subscription Reminder form so that those who wish to pay by credit card can do so. This should increase retention of overseas members, especially New Zealanders.

We have, incidentally, taken the somewhat questionable step of regarding our Trans-Tasman colleagues as being no different to anyone else, and they are now billed directly from Australia rather than by a subsidiary agency in New Zealand.

This has proved an unpopular move amongst our Kiwi cousins, but has abruptly terminated six years of accounting whimsicality.

There has been no increase in subscription for 3 years and during that time our reserves have run out. It has been necessary for the Treasurer to carry SPUMS for a short period this year until the subscriptions come in. I am sure



**SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY STATEMENT OF RECEIPTS AND PAYMENTS  
FOR THE YEAR ENDED 30 APRIL 1992**

	1992	1991
<b>Opening Balance</b>		
Standard Chartered Finance Ltd.		1,000
National Mutual Royal Bank		8,305
Savings account	6,023	-
Cheque account	2,963	-
National Australia Bank	-	441
Cash on hand and stamps	221	112
	9,207	9,858
<b>Income</b>		
Subscriptions	48,785	50,318
Interest	804	1,579
	49,589	51,897
Loan advanced	2,500	
	\$ 61,296	\$ 61,755
<b>Expenditure</b>		
Secretarial	13,098	10,217
Stationery	2,930	2,657
Journal	19,111	13,790
Postage	9,180	5,883
Travel and phone conferences	3,136	11,086
Equipment (see note)	8,768	4,583
Miscellaneous	2,087	2,989
Bank charges	330	331
Audit	-	200
Legal expenses	-	812
Donation to Diver Emergency Service (DES)	2,500	-
	61,140	52,548
<b>Closing balances</b>		
National Mutual Royal Bank		6,023
Savings account	136	2,963
Cheque account	20	-
Cash on hand and stamps	-	221
	156	9,207
	\$ 61,296	\$ 61,755

**AUDIT REPORT TO THE MEMBERS OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY  
INCORPORATED**

I have conducted various tests and checks as I believe are necessary considering the size and nature of the the Society and having so examined the books and records of the South Pacific Underwater Medicine Society for the year ended 30 April 1992 and report that the accompanying Statement of Receipts and Payments has been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the year then ended.

29 May 1992  
Newport Beach 2106

David S Porter, FCA  
Chartered Accountant

you all will agree that being dependent on the wealth, or otherwise, of the Treasurer is not the correct way to run a scientific society. My unprofessionalism in the position is borne out by the fact that any Treasurer worth his salt in this day and age would have departed the scene with the boot firmly on the other foot. It essential that the Society's subscription income exceeds expenditure so that a sum of money is built up to cover unexpected expenses, such as losses on meetings and purchasing of new equipment.

It is recommended by the Committee that there be an increase in the annual subscription and that the subscription for Full Members, that is medical practitioners all of whom can claim it as a tax deduction, should be \$80.00, and for Associates the subscription should be \$40.00.

Another alteration we feel necessary, is to change our financial year from the current May to April and our subscription year from July to June so that they both coincide with our Journal year of January to December. You will be voting on this change presently.

Changing the financial year will mean, that for this year only, 1992-1993, members will pay a subscription for 18 months, and then we will go back at the end of 1993, to billing a 12 month subscription for 1994. We have chosen that as less expensive to do than charge a half subscription for the remainder of 1992 and bill again in December. It also will enable the Society to discharge its debts. So for the 18 months from July 1992 to December 1993, the subscription for Full Members would be \$120.00 and for Associates, \$60.00.

I call upon the meeting to support this motion.

Grahame Barry  
Treasurer of SPUMS

### EDITOR'S REPORT

This year has seen a steady supply of original papers for publication. My aim is to have the SPUMS Journal as an interesting and educational quarterly, publishing peer reviewed original work and the proceedings of the Annual Scientific Meetings as well as SPUMS notices, and also to bring to the attention of SPUMS members and associates papers published all over the world by reprinting abstracts and original articles. This latter is at present almost entirely limited to what I find interesting in my reading. It is my hope that others will help by sending photocopies of the articles they find interesting. For an abstract or article to be of use the full reference is needed as reprinting requires obtaining the original publisher's permission.

September 1991 saw the paid circulation of the Journal pass the 1,000 copies mark. Since then NASDS

(which is what our old friend FAUI has metamorphosed into) has placed a bulk order of 310 copies which they distribute to their members both in Australia and North America. Now we print 1,500 copies of each issue.

The continued health of the Journal depends on a steady supply of good quality papers and clinical case reports. Each report contributes its bit to the sum of knowledge. Case reports have shown that trainees do suffer air emboli during swimming pool training. Luckily those reported have been non-fatal, but their occurrence highlights a problem which would otherwise have been unrecognised. So please send in your interesting cases. I am more than willing to help with any rewriting that may be necessary.

I would like to thank John Williamson for his great help with the peer reviewing.

John Knight  
Editor, SPUMS Journal

### AUSTRALIAN AND NEW ZEALAND HYPERBARIC MEDICINE GROUP (ANZHMG) (A standing committee of SPUMS) ANNUAL REPORT JUNE 1992

This has been a busy and formative year for our "new" Group. At the outset I should like to pay tribute to the hard work and expertise of our inaugural Honorary Secretary, Dr David Tuxen, Director of Hyperbaric Medicine at the Alfred Hospital in Melbourne. He has kept us well informed and has been instrumental in the Group's successes. We all owe him our thanks.

With nine Australian and two New Zealand Hyperbaric Units in our Group, we now represent Hyperbaric Medicine in a large part of the South Pacific. We are currently attempting liaison with the Indonesian Hyperbaric Medicine Society.

Our initial four meetings have been teleconferences underwritten by SPUMS, for which we are grateful. Our Technicians and Nurses Association (HTNA) is excellent, and our move towards unified treatment and research protocols is beginning.

We have a responsibility to place Hyperbaric Medicine on a rational, objective and sound scientific footing, and we have begun well. I thank all ANZHMG and HTNA members for their continued support.

John Williamson,  
Chairman

**MINUTES OF SPUMS EXECUTIVE MEETING**

held at Port Douglas  
on Sunday 31st, May 1992 at 1800

**Apologies**

Drs G Barry and A Slark

**Present**

Drs D Gorman (President), D Wallner (Secretary), J Knight (Editor), D Davies (Education Officer), C Acott, S Paton, J Williamson and Rees Jones (by invitation representing NZ Chapter).

**1 Minutes of the previous meeting**

The Minutes having been circulated, were taken as a true and accurate copy of the previous meeting after one amendment put forward by Dr Knight, that "The medical examination form published in the Journal is a SPUMS form, not an Australian Standard form". Proposed by Dr J Knight, seconded by Dr C Acott.

**2 Business arising from the Minutes****2.1 PALAU A.G.M.**

Dr Gorman has written to our Guest Speaker, Dr David Elliott, asking if he can vary his available dates to earlier than the first week in June, but his flexibility is limited somewhat by other commitments.

Dr Elliott has already forwarded abstracts of his lectures which will be available pre-conference. It is expected that Palau will be popular and demand may exceed accommodation and boat requirements. Therefore it will be suggested at the AGM, and also in the Journal that a substantial deposit of \$600 be paid by December 14th, 1992 by intending participants to ensure accommodation and diving.

**2.2 PNG A.G.M.**

Some discussion as regards to the conference venue, being Rabaul or Madang. The general feeling was that Madang may be best, with Rabaul as a pre- and post-conference option. Dr C Acott (Convener) will prompt Allways Travel to provide details of organisation for the conference by December 14th.

**2.3 DIVE COMPUTERS**

Draft received from Dr C Acott for comment at next meeting.

**3 North American Chapter**

Deferred to Special Executive Meeting tomorrow night (1.6.92)

**4 Diving doctors list**

The Secretary has received updated addresses from State Registration Boards. Letters of invitation to join will be sent shortly.

**5 Dive shop locations list**

Lists have been obtained from PADI, FAUI and BSAC. These will be correlated these with the Diving Doctors lists. Possibly to be done by medical students at Royal Adelaide Hospital.

**6 Treasurer's report**

The Profit and Loss Statement was not available. Discussion was deferred to the Special Executive Meeting on 1.6.92, but a change of subscription year from financial to calendar year was agreed.

**7 Duties of convener**

Discussion paper presented by Dr J Knight. Deferred to next meeting.

**8 AMA**

Letters to be circulated for next meeting.

**9 Diploma thesis**

Dr P Mark's thesis for the Diploma. It was agreed all papers are the property of SPUMS. Dr Knight will write to Dr Mark asking if the MJA has accepted it for publication. If not we will require him to ask for its withdrawal, and it will be published in the SPUMS Journal.

**10 Annual General Meetings**

10.1 Tenders. Dr S Paton will try to get quotes from five travel agents for a block of 2 AGMs.

10.2 Registration fees for future AGMs. These fees are to be intemised by the travel agent and presented to the Committee for approval before the brochures are sent out.

There being no further business, the meeting was closed at 2030.

**MINUTES OF SPUMS EXECUTIVE MEETING**

held at Port Douglas,  
Monday 1st June 1992 at 1800 EST

**Present**

Drs D Gorman (President), D Wallner (Secretary), J Knight (Editor), C Acott and S Paton, with Dr Lori Barr and Mr Steve Dent representing the North American Chapter.

**1 Finances**

The Profit & Loss Account obtained from Dr Barry was discussed, an estimated \$11,000 in debt appeared likely by the end of June. This lack of cash and Ray Rogers' delay in putting in the required receipts were the explanations for the North American Chapter not being reimbursed. Dr Gorman emphasized we are totally committed to reimbursing the North American Chapter as soon as subscriptions come in.

Economic savings could be made by eliminating the Brighton Executive Centre, transferring their computer to Dr S Paton in Sydney and all mail coming from ACOM to go to the Secretary. Dr Knight presented several budgets for next year with varying increased subscription levels.

It was decided that

- 1.1 Members would pay \$80, Corporations \$80, Associates \$40.

- 1.2 The financial and subscription years would be the calendar year from January to December.
- 1.3 Subscriptions, due in July 1992, would be for eighteen months (to December 1993). Because of the low value of the New Zealand dollar there would be an option for New Zealanders to pay six months and twelve months subscription fees.
- 1.4 These decisions to be ratified at the AGM.

Meeting closed at 1920.

### **SUPPLEMENT TO AS 4005.1-1992 Medical form for prospective scuba divers**

Standards Australia has produced pads of 50 copies of the medical form for prospective scuba divers (appendices B and C of AS 4005.1-1992). They are the same as those in the SPUMS Diving Medical (published March 1992).

The pads cost \$ 18.00 each (36 cents a form). The catalogue number is AS 4005.1 Supp1-1992. Standards Australia have offices in all capital cities.

## **LETTERS TO THE EDITOR**

### **SPUMS JOURNAL - FOR DOCTORS OR DIVERS?**

Diving Medical Centre  
66 Pacific Highway  
St Leonards  
New South Wales 2065  
August 1992

Dear Editor,

In recent journals there has been a disturbing increase in the number of articles by diver training agency representatives.<sup>1,2</sup>

When these people are promoting their own products or attitudes, it would be more fitting for these to be distributed by their agency newsletters or via general diving magazines. Dive instructors and training agencies have far more time to devote to self promotional articles than do hard working physicians. I believe the Journal, for which I have the utmost regard and admiration, would be better served by not assisting these promotions.

In medical journal articles there is usually a presumption of truth. This is often not so with the propaganda from commercial organisations, even those written under the guise of a pseudo-medical discussion.

These articles detract from the value of the Journal and also allow the agency to promote both inaccuracies and falsehoods, often without rebuttal. They are then able to, and do, infer SPUMS endorsement. The commercially orien-

tated authors can quote from the SPUMS Journal, even though they are in fact quoting from their own work. This gives even more authority to their inaccuracies.

I was particularly offended by Mr Cummins' article, which was accepted with minimal critical review, in which he blatantly threatens the DES organisation with reduction of funding if they continue to promote what is internationally accepted as one of the safest decompression tables, that of the DCIEM. He warns us that "agencies do not donate money to competitors", presumably comparing DCIEM to PADI (RDP) tables. In the previous Journal Ray Rogers tried to explain why the RDP was associated with so many decompression sickness cases, but omitting one obvious explanation.

Unfortunately, it is inferred that "he who pays the piper, calls the tune". Both SPUMS and DES have an obligation to divers to promote the tables which they consider the safest, irrespective of the view of any training agency. Also, whether DES combines with the Australian Safety Patient Foundation and/or Dive Safe, should not be determined by a commercial diving organisation.

Some other inconsistencies were evident in the article. He states that "very few, if any, accidents occur in training or under direct supervision of a diving professional". This is certainly promoted in the PADI handbook he referenced. An independent article, written by Dr Robyn Walker<sup>3</sup> in the same SPUMS Journal, described the experience in Townsville in 1990 and demonstrated that "27.5% of

the divers treated for decompression sickness and 50% of the cerebral arterial gas embolism victims were participating in basic certification courses under the direct supervision of a qualified diving instructor". The difference in the two statements is informative. In one case the motivation is advertising and promotion of a diving myth. The other is a presentation of factual data. Unfortunately, as they both occur in the same journal, they are likely to get equal prominence in the memories of the subscribers. No prizes for guessing which one will be quoted to trainees.

Dr RM Walker pointed out that every diver treated for DCS attested that the divers were within the particular table limits. But only in 32.5% were they within the limits of the DCIEM tables. There are also no prizes for guessing which agency tables headed the list for causing DCS.

Mr Cummins' criticisms of medical examinations and the implied value of his agencies questionnaire, as opposed to an examination by a diving medical qualified physician, were unsupported by any data. The facts, supplied by Dr John Parker in his excellent articles on diving medicals<sup>4,5,6</sup> in a previous SPUMS Journal, were not even mentioned, let alone referenced. This was despite the careful analysis of both the value of the medical examination and the relative inadequacy of a PADI "questionnaire".

Agency criticism of the "conditional medical" presumes that the client must only be given an assessment according to what is desired by instructor organisations. I do not doubt that many organisations would prefer medicals performed in a specific manner and in fact the AS 4005.1 is designed for this. Nevertheless the medical is not done as a service to the training agency. It is done as a service to the diver, who is paying for this service. Thus if it is prudent to counsel the diver or potential diver in such a manner as to improve his safety, then this should be done. If it offends the training agency, then so be it. Insurance companies, diving contractors and others who require a medical to be performed as a service to them, pay for it.

Fortunately, Mr Cummins has now reduced his stated cost of the diving medical to \$60, as opposed to the \$170 that was quoted in his Bulletin article. Presumably it was not possible to persevere with that inaccuracy when confronted with a knowledgeable audience.

Another statement that, "I am aware that no agency included (free ascent practice) in their training", is semantic quibbling. Free ascent practice is still being performed while under instruction, although not always in this guise. Sometimes it is part of, or the result of, "ditch and recovery" (doff and don) or "out of air" ascents. The same dangers exist.

During the same week as the publication of Mr Cummins' paper a legal case, specifically related to dive instructors doing free ascent training, was submitted to me

for assessment. Also, that week, PADI took legal action against Dr Douglas Walker, to prevent him from discussing some confidential information on the causes of deaths amongst scuba divers, during his attendance at a Queensland meeting on diving Health and Safety. This was not a good example of the "new age of cooperation", described by Mr Cummins. Nor was his ostracism of Dr Robert Thomas.<sup>7</sup>

I am not in any way criticising individual instructors. My point is that some instructor training agencies are using SPUMS Journal for promoting misleading and sometimes false beliefs. In fact, I think the individual instructors are probably being misled as much as doctors. As Dr R M Walker pointed out, 16% of those treated in Townsville were instructors.

Dr A Sutherland, in the same journal, described two cases of air embolism during scuba training in swimming pools. One of Dr RM Walker's cases was of a similar nature. The instructors are at similarly great risk, as demonstrated by "Project Stickybeak" statistics<sup>8</sup> and those of the deaths amongst recreational divers and instructors, described elsewhere.<sup>9</sup>

It is for this reason that I would encourage individual instructors, not the prodigious instructor agencies, to be associate members of SPUMS. Unfortunately, at this stage, they are likely to read, and get support for, their own agencies propaganda in our Journal!

I am aware that because I have publicly disagreed with the PADI propaganda, I am now likely to be lumped with Dr Bob Thomas as a contemptible "high profile diving medical personality", or receive a threat of legal action or a solicitor's letter, like Dr Douglas Walker. I am proud to be associated with both. I do not represent SPUMS (nor did Dr Thomas), but (like Dr Walker) I do believe that, in the interests of diving safety, I should not be intimidated into silence.

Diving doctors should strive for diver safety, whether it involves dive table recommendations, training contributions to diving accidents, treatment facility availability, medical standards or "conditional" medicals. I think these should be decided on the basis of what is best for the diver, and not what is best for one training agency. Perhaps we also should not "promote the opposition."

Carl Edmonds  
Director  
Diving Medical Centre

## References

- 1 Cummins T. A training agency perspective on DES funding and other topical issues. *SPUMS J* 1992; 22(2): 910-96.
- 2 *SPUMS J* Vols 21, 22.

- 3 Walker R. Fifty divers with dysbaric illness in Townsville General Hospital during 1990. *SPUMS J* 1992; 22(2): 66-70.
- 4 Parker J. The diving medical and reasons for failure. *SPUMS J* 1991; 21(2): 80-82.
- 5 Parker J. The assessment of the PADI resort course questionnaire. *SPUMS J* 1991; 21(2): 82-83.
- 6 Parker J. The relative importance of different parts of the diving medical in identifying fitness to dive and the detection of asthma. *SPUMS J* 1991; 21(3): 145-153.
- 7 Thomas RL. Queensland's new 1989 diving regulations. *Underwater Geographic* 1990; No 31.
- 8 Walker D. Provisional report on diving related fatalities during 1989. *SPUMS J* 1992; 22(1): 3-15.
- 9 Edmonds C and Walker D. Scuba diving fatalities in Australia & New Zealand. *SPUMS J* 1989; 19(3): 94-104.

**DIVERS' EARS**

Whitsunday Diving Medical Centre  
P.O. Box 207, Airlie Beach  
Queensland 4802  
10 July 1992

Dear Editor,

On a recent two day diving trip on the Great Barrier Reef with 20 recreational divers, (19 of whom had medical or nursing qualifications), every diver had their ears examined before diving commenced and after all diving had ceased.

The group consisted of 9 males and 11 females with varied diving experience.

Number of dives	Number of divers	Percentage
1 - 10	9	(45%)
11 - 50	8	(40%)
50 plus	3	(15%)
	20	

Diving the weekend the divers had an average 5.3 dives (range 2-7).

Of the 20 divers 5 (25%) had symptoms of aural barotrauma of descent. Only one had to stop diving prematurely. At the end of all diving 10 (50%) divers were seen, on direct inspection of the tympanic membrane, to have aural barotrauma involving 16 ears.

Grade	Ears affected	Symptomatic ears
1	11	2
2	1	1
3	4	2
<b>Total</b>	<b>16</b>	<b>5</b>

The 10 divers with aural barotrauma came from all the experience groups in approximately the same ratios in the group.

Number of dives	Divers affected	Percentage
1 - 10	5	(50%)
11 - 50	4	(40%)
50 plus	1	(10%)

It was interesting to note that over the weekend 5 divers were taking Sudafed tablets for symptoms of mild upper respiratory tract congestion. Of these 5 divers, 3 suffered aural barotrauma, 2 with symptoms, but no one had to stop diving prematurely.

Also interesting was that 7 divers used transdermal hyoscine (SCOP) patches as prophylaxis for sickness despite a favourable weather forecast.

- 1 Although anecdotal this weekend demonstrated that Aural barotrauma is very common in recreational diving despite diving experience (and medical knowledge).
- 2 Subclinical aural barotrauma is also very common.
- 3 Grade 1 aural barotrauma can be symptomatic and grade 3 can be asymptomatic.
- 4 Decongestants (especially pseudoephedrine preparations) and transdermal hyoscine are commonly used by divers.

John Parker

**HIGH TECH DIVING**

**A response to the editorial in the Jan-Mar 1992 SPUMS Journal.**

Hamilton Research Ltd  
80 Grove Street, Tarrytown  
New York, 10591-4138  
5 August 1992

Dear Editor,

The essay on "high tech" diving by Des Gorman in the 1992 Jan-Mar issue of *SPUMS Journal* stands firmly as the opinion of one of the most knowledgeable and respected members of the international diving community and would not normally require a response. However, the essay mentions my involvement, and lest by default I be assigned the role of the villain in the piece, I feel a response is necessary. That involvement, by the way, has not been very great in Australia, but I seem to have found myself in the middle of several issues in the US related to technical and special mix diving, some of which deserve discussion.

My Australian involvement is simple. Rob Cason of Fun Dive Centre asked if I would provide tables to support his introduction of technical diving into Australia. I agreed to do that, provided of course that he would do things in a responsible way. He also requested decompression support for the use of a rebreather. Another client of mine had had excellent results using rebreathers, so I agreed to work with these as well. Later this was rumoured to be for dives to 200 msw. This, I felt, was so ludicrous as not to merit a response. Such dives could indeed be done with rebreathers, but to do so would require such an enormous support operation that it would not be likely to happen in the "recreational" diving mode. I regret any misunderstandings I may have allowed to develop on this issue.

Since the issue has been brought up, please let me say a bit about the concept of "technical diving" and why I am involved in it. People have been making deep dives on air since the invention of scuba, usually but not always successfully (a good history of deep air diving is in Gilliam et al.<sup>1</sup>). My involvement began when Parker Turner and Bill Gavin asked for help in adding some helium to their breathing mix to reduce narcosis for some extensive 75 m depth cave dives. These "trimix" dives gave good results; we used an enriched air intermediate "decompression mix" and oxygen breathing from 6 m. The use of several mixes is easy in cave diving since many tanks of gas are often needed, and they are relatively easy to find. I had the feeling that in a way I was blackmailed; if I had not agreed to help, they would have gone ahead and done the dives with air.

My role is primarily in the area of decompression procedures, but there are many, many other aspects to these complex operations which make the decompression part look easy.

That set the pace for one aspect of technical diving, and the concept spread, even to the extent of spawning a journal (*AquaCorps*). By no means do I encourage people to make these deep dives, but if they are going to do it anyway then a case can be made that it could be regarded as unethical to deny access to the safer approaches of using a less narcotic mix with an efficient decompression.

But don't let me leave the impression that this is easy or safe. Two recent trimix fatalities in the 75 msw range off the US East Coast attest to the importance of high level training and preparedness. Both of these divers appeared to have run out of gas. Also, Parker Turner died in a cave last November. He was doing things right but got caught in the midst of a geological upheaval that blocked the exit to his cave while he was inside.

This information certainly supports the main thesis of Des Gorman's essay, especially the point that many divers may not fully appreciate the risks involved. There are some other points.

My purpose is not to "refute" but to comment. First, as mentioned, I thoroughly agree with the individuals' right to accept the risk, but the risk should be understood, and the diver should be thoroughly trained to deal with it. This is where we have a great need. At present there is no standard for training or qualification in technical diving. I strongly urge technical divers to form their own member-run association and take control of these needs before the opportunity goes away.

Regarding the several USN heliox divers who died following 200 msw-plus dives and missed decompression, with a modest search I have not been able to locate a report on this series, unless it is the submarine lockout accident that happened in the USS Grayback and which is not particularly relevant to technical diving. The valid message here is that when one moves up to technical diving the nature of the operation has to change. All contingencies have to be planned for in advance. Many diving accidents move from incidents to accidents because the response to a disturbance of some sort is not optimal, for whatever reason.

The issue of decompression is of course a major one, but at this point it appears to be under some control; certainly the technical divers are better off than the USN statistics Dr Gorman cites (20% DCS), but documentation is limited (data collection is a major continuing effort). Divers using the trimix pattern described earlier to depths of 75 msw (not 200 msw) and for times in the 20 minute range have several hundred dives with, so far, a negligible incidence of DCS, but some samples of Doppler bubbles scores have been unacceptably high. Regarding thermal stress, most of these divers use dry-suits, usually with argon as the insulation gas, and this works well for short exposures. Only the long cave dives (12-13 hours in some cases) pose a real thermal problem. The endurance possible with a dry suit is limited for a variety of reasons. The 90 msw "limit" is probably not so binding with argon in the suit, but again, gas limitations keep the thermal limits from getting out of hand. Breathing gas heat will indeed be needed when divers spend more than a few minutes in the 150 msw range, unless the water is unusually warm. This is not likely to be a problem in this community any time soon.

Oxygen toxicity is a major concern in decompression planning. From the beginning we decided to avoid the risk of breathing oxygen at 12 or 9 msw and use it only at 6 and 3 (or at only 6) msw. This puts the diver just at the edge of the toxicity limit cited by NOAA of 1.6 bar<sup>2</sup> and for a diver decompressing at rest this is acceptable.

Regarding treatment, it is not my place to take on a respected authority in this arena, but I think Dr Gorman will agree with me on these points. First, regarding technical trimix diving, since the entire decompression part of the dive is done with enriched air or pure oxygen, standard treatments are entirely appropriate. In any case no diver should ever be refused treatment because of some misguided notions that a

treatment is "not right" because the diver has used a special mix. There is too much more to this issue to tackle here, but we can be sure that a prompt treatment with oxygen at 2.8 bar (USN Table 6) will cure most decompression disorders, regardless of the mix. A good thing to think about at this point is that the tough treatments usually result from operational problems like abrupt surfacing or missing the entire decompression. Good planning, high quality equipment properly maintained, and thoughtful gas management are what it takes to avoid the circumstances that result in such incidents.

Finally, let me address briefly the matter of enriched air, the so called "nitrox" diving. There seems to be more dialogue on this practice (which is not technical diving) than on technical diving, probably because it seems more likely to invade the domain of "recreational" diving and hence is more threatening to the "industry." Actually, not much in the way of extra diving skills are needed to do enriched air diving, but some care needs to be taken in mixing and handling the mixes. The issue of enriched air diving was addressed in a workshop at the time of the big DEMA (Diving Equipment Manufacturers' Association) show in January 1992. Because that Workshop settled some issues and defined others more clearly, most the controversy has died down (at least in the USA). For example, misinformation that enriched air corrodes tanks more than air, or that standard treatments do not work, has been laid to rest, good oxygen-compatible lubricants have been identified and both good and bad practices outlined. These are included in a report on the workshop.<sup>3</sup> Because I have no vested interest in enriched air diving (except perhaps to try to get people to call it by its right name, enriched air, and to save "nitrox" for the mixes lower in oxygen than air), it was my privilege to be engaged to help organize and to chair this meeting. In addition to the report, a working group was organized to deal with several remaining issues.

Let me add one last point to both my essay and Des Gorman's. I, too, want to discourage anyone from technical diving, but especially anyone who is not equipped and inclined to do it right. It involves a considerable investment in planning, equipment, decompression tables, gases, training, practice, organization, team-work and patience, and of course considerable risk. If you must go into this, go into it with your eyes open and be well prepared.

R.W. (Bill) Hamilton

## References

- 1 Gilliam B, Von Mair R, with Crea J and Webb D. *Deep diving. An advanced guide to physiology, procedures and systems*. San Diego: Watersport Publishing Inc., 1992
- 2 *NOAA Diving Manual: Diving for science and technology. Third ed.* Silver Spring, Maryland: NOAA

Office of Undersea Research, U.S Department of Commerce, 1991

- 3 Hamilton RW. *Workshop findings: Evaluating enriched air ("nitrox") diving technology*. Boulder, Colorado: Scuba Diving Resource Group, 1992\*

\* Available from Outdoor Recreation Council of America / Scuba Diving Resource Group (ORCA/SDRG), P.O.Box 3353, Boulder, Colorado (International phone 1-303-444-3353) for \$US 10.00 plus postage (\$US 2.00 in U.S.A., \$US 5.00 outside). Also available by telephone/credit card from DUI (International phone 1-619-236-1203, International fax 1-619-237-0378).

## ASTHMA AND DIVING

Whitsunday Diving Medical Centre  
PO Box 207, Airlie Beach  
Queensland 4802  
25th August, 1992

Dear Editor,

The safety of asthmatics scuba diving has been an continuing controversy. The fact that many asthmatics do dive with little obvious catastrophe has been countered with many anecdotal series of catastrophic cases but none of scientific persuasion.

Most "diving doctors" would agree that "conventional wisdom" would advise "active asthmatics" not to scuba dive. The problem has been how many years without symptoms are needed after a history of asthma before diving can be allowed. Edmonds et al.<sup>1</sup> suggest a history of no asthma for five years is acceptable providing lung function is normal. This is a softening of "conventional wisdom" which used to advise that anyone with a past history of asthma should not dive.

The most useful objective investigation to assess "reactivity" of the airways is a challenge test, usually using inhalation of metacholine, histamine or hypertonic saline. The techniques used are rapid, inexpensive, reproducible and safe.<sup>2-5</sup>

Histamine and metacholine challenge tests require minimal equipment but are fiddly and involve a high patient compliance. Both histamine and metacholine are intermittently hard to get, costly and the solutions need to be constantly refrigerated and changed frequently.

Hypertonic saline challenge tests require an ultrasonic nebuliser (with an output of at least 1.2 ml/minute) and 4.5% saline. They are easy to perform and easily justified to a diver...."If salt water will make you wheeze in the surgery it will also do it while diving underwater". Moreover, when



a diver becomes wheezy breathing the nebulised saline they recognise the potential danger and are more accepting of being told that they are not fit to dive.

Whereas inhaling high concentrations of histamine and metacholine can induce a positive result in normal divers, it has not been possible to induce such a degree of airway narrowing with hypertonic saline.<sup>6</sup>

Furthermore there is a similarity in both the sensitivity and the reactivity of asthmatic subjects to inhalation of hypertonic saline and exercise, presumably due to the water loss induced by hyperventilation causing a hyperosmolar state in the bronchial tree.<sup>7</sup>

In the past the only available ultrasonic nebulisers with an adequate output available in Australia were the MISTOGEN (cost Aus \$2,500) or the Divilbiss ULTRANEB 99 (cost Aus \$1,500). Moreover to deliver the nebulised saline in adequate amounts, a low resistance, non-rebreathing 2 way valve was essential. The Hans Rudolf 2,700 valve is best suited at the outrageous cost of over Aus \$400. Setting up to perform such tests was very costly and not economically viable for the average "diving doctor".

Recently I have been using a new ultrasonic nebuliser, the Omron model NEU06. It has an output of about 1.5 ml/minute. At a cost of Aus \$340 it is most economical. Moreover being hand held with an on/off button at the finger tip the patient can inhale the nebulised spray direct from the nebuliser on demand, negating the need for an expensive non-rebreathing valve. It is available from J.A. Davey Pty. Ltd., P.O.Box 171, Warringah Mall, New South Wales 2100.

Armed with such affordable equipment I now test anyone with a past history of asthma with 5 years or longer free of symptoms or anyone with an uncertain history of

asthma. With experience I feel new indications will become apparent.

For anyone wishing to perform airway reactivity challenge tests I commend the Omron model NEU06 nebuliser (beware other models which might not have adequate output). It makes the selection of asthmatics fit for scuba diving a more rational and logical process.

John Parker

## References

- 1 Edmonds C, Lowry C and Pennefather J. *Diving and Subaquatic Medicine* 3rd ed., Butterworth-Heinemann 1992; 462.
- 2 Cockcroft DW, Killian DN, Mellon JJ and Hargreaves FG. Bronchial reactivity to inhaled histamine: a method and clinical survey. *Clin Allergy* 1977; 235-243.
- 3 Schoeffel RE, Anderson SD and Altounyan REC. Bronchial hyperreactivity in response to inhalation of ultrasonically nebulised solutions of distilled water and saline. *Br Med J* 1981; 2183: 1285-1287.
- 4 Yan K, Salome C and Woolcock AJ. Rapid method for measurement of bronchial responsiveness. *Thorax* 1983; 38: 760-765.
- 5 Hariparsad D, Wilson N, Dixon C and Silverman M. Reproducibility of histamine challenge tests in asthmatic children. *Thorax* 1983; 38: 238-260.
- 6 Smith CM and Anderson DA. Inhalation provocation tests using nonisotonic aerosols. *J Allergy Clin Immunol* 1989; 84: 781-790.
- 7 Hahn A, Anderson SD, Morton AR, Black JL and Fitch KD. A reinterpretation of the effects of temperature and water content of the inspired air in exercised induced asthma. *Am Rev Respir Dis* 1984; 130: 575-579.

## BOOK REVIEWS

### OXYGEN AND THE DIVER.

Kenneth Donald

ISBN 1-85421-176-5

The SPA Ltd., Hanley Swan, Worcestershire.

RRP (UK) £14.95 P&P £2.50

I was surprised and very pleased to receive a letter earlier this year from Professor Donald asking the SPUMS Journal to accept an advertisement for Oxygen and the Diver. I had thought that he must have been long dead as I had assumed that he had been a middle aged, established researcher when he did his work with divers breathing 100% oxygen between 1942-45. It turns out that he was 30 in 1942. This work was driven by the need to combat the Italian

torpedo frogmen's success in sinking ships in Allied harbours and to be able to clear mines in newly captured harbours. I was taught my anaesthetics at the Royal Naval Hospital, Haslar, near Portsmouth, by Bill Davidson who had worked from 1943-45 with Professor Donald, so know about the work, which was largely unavailable to the profession because of wartime secrecy restrictions.

This is an attractive and well produced book. I could only find three typographical errors. In it Professor Donald has provided the full story and results of the 3 years of experimentation. He also has been given access to much unpublished information from the USN and Dr Ed Lanphier. Besides oxygen diving his Admiralty Experimental Diving

Unit, the name came with the need for headed paper, worked on the development of safe oxygen enriched mixtures and schedules which enabled the Allies to clear the ports of Europe of mines, sometimes under fire, without any incidents of oxygen poisoning, anoxia or decompression illness. They also worked on surface decompression and submarine escape.

Chapter 1 discusses reports of the ill effects of oxygen under pressure, from Paul Bert in 1878 up to 1942. At that time the USN allowed oxygen breathing for 2 hours at 30 fsw (15 m) and 30 minutes at 90 ft (27 m). In spite of two RN officers having convulsed, after 13 and 16 minutes, during a dry chamber run at 100 ft (30 m) on oxygen in 1933 these USN limits for oxygen diving were adopted for oxygen breathing divers. As described in Chapter 2, this was soon found to be a mistake!

Chapter 2 is Professor Donald's thesis for the Cambridge MD, presented in 1945, reproduced unaltered. It is a clear exposition of the dangers of high partial pressures of oxygen underwater based on many subjects. At the end of this chapter are found the following words.

"The variations of tolerance between individuals, the variation of tolerance of each individual, the impairment of tolerance with work and underwater, all make diving on pure oxygen below 25 feet of sea water a hazardous gamble."

"The only possible conclusion is that such tensions of oxygen should be scrupulously avoided."

Chapter 3 takes the oxygen story up to the present day. Professor Donald explains the very different allowed oxygen depths of the RN and the USN by reference to the original trials where it is likely that the USN counterlungs had a measurable level of nitrogen, so allowing a deeper exposure because the oxygen partial pressure was less than expected, while the British depths were the pressure equivalents of the air above the water the diver was standing in.

Anyone who wishes to undertake diving using oxygen enriched mixtures should read Chapter 5 before embarking on these dives. The RN experience with these mixtures is an amazing one, brought about by never exceeding a safe equivalent oxygen depth and by using a mass flow (a set volume per minute) system which when the diver works hard reduces the available oxygen partial pressure and so the risk of oxygen toxicity. Other methods of providing the diver with gas, such as the constant ratio breathing apparatus and open circuit always provide the full oxygen partial pressure exposing the diver, when close to the mixture's depth limit, to oxygen toxicity when he works hard. The answer is closed circuit mixed gas with sensors for depth and partial pressure of oxygen controlling the oxygen content of the circuit to safe levels, which rise as the diver ascends in order to assist nitrogen elimination.

Chapter 7 deals with the various papers, using remarkably few subjects, which "established" that some divers have a reduced ventilatory rate in response to raised partial pressure of CO<sub>2</sub>. Only two studies had enough subjects for statistical analysis and one of these concluded that the responses of divers and non-divers did not differ! Many studies showed that the "normals" had a low end tidal PCO<sub>2</sub> at rest, a sure sign of hyperventilation. In many studies the investigators commented on the slow respiratory rate, below 10 bpm, of the divers when they were breathing through the mouthpieces at rest or when working.

This is exactly the pattern of breathing that Glen Egstrom recommends to avoid "beating the regulator". It is a response learnt by divers when using their gear, not their normal respiration. Professor Donald concludes that repeating the experiments using more sensitive measurements and better controlling for the unfamiliarity of the experiment to the "normals" is needed before we accept the theory that some divers retain CO<sub>2</sub> in their normal diving. Certainly some retain CO<sub>2</sub> when working with high resistances to breathing or inadequate CO<sub>2</sub> absorption or inadequate fresh gas flows (Chapter 6).

Appendix 1 is a description of how Professor Donald got into this work and what the Admiralty Experimental Diving Unit did during and after the Second World War. It should have been Chapter 1, it is so interesting and informative. Kenneth Donald cannot be accused of not providing full details of his work. There are 24 tables, even including the names of the divers involved, of the results of oxygen poisoning trials in Appendix 2.

This is a book that should be in every medical library, and it certainly should be in every diving medical library. It should be on the bookshelf by every recompression chamber and in every dive shop that supplies oxygen enriched gas to divers. Any doctor who has any claims to a knowledge of diving medicine should have read it.

John Knight

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## SPUMS ANNUAL SCIENTIFIC MEETING 1991

### BUOYANCY COMPENSATION AND ASCENT RATE

Glen Egstrom

#### Introduction

Archimedes' observation, that a body immersed in water is buoyed up by a force equal to the weight of the volume of water that is displaced, has clearly benefited the diving community. It has also provided frustration, amusement and occasional hazards to health. Proper buoyancy control is being able to achieve neutral buoyancy at every depth and to maintain it throughout a dive. It is a skill that is fundamental for diving comfort and safety. Unfortunately many divers do not seem to understand the benefits of a high degree of skill in buoyancy control.

#### Neutral buoyancy

To teach neutral buoyancy in our training program, we get students to do a one finger handstand on the bottom of a 5 m deep swimming pool. A one finger handstand is not easy. One is not actually standing on the finger, but making negative buoyancy keep the finger in contact with the bottom. With adequate breath control one can keep a finger on the bottom. A big breath will start to float one away. We use this to get people to understand what they can do with breath control. A diver can have about 2-2.5 kgs of differential volume simply by exercising adequate breath control. When one is neutrally buoyant, weighted properly or with the correct sized buoyancy bubble, one can swing to positive or negative buoyancy with breath control alone.

The most important skill that has to be learned with any kind of buoyancy compensation system is to make the specific adaptation to that piece of gear so that, as one ascends, one can hover at any depth. I have to commend those on the boats that I have dived from, they are all able to hover.

#### Gas expansion

Where in the water is the greatest danger of gas expansion? Most people will say "In the first atmosphere of additional pressure below the surface of the water". Actually Boyle's Law produces an exponential curve and the greatest pressure/volume change takes place in the metre just below the air/water interface. Under certain conditions one can develop lung over-pressure accidents with as little as 90 mm Hg or 0.12 bar (less than 2 psi) differential pressure, which is a rise of about 1.2 m. Rising 1.8 m it is

not difficult to achieve an accident, and with a 2.4 m rise it is easy. The statement "I've only used scuba in a swimming pool", tells me that they have only used compressed gas in the most dangerous part of the water column. If they do not know what they are doing, they can easily get into difficulties. Large volume buoyancy compensators (BCs) have shown that very close to the surface of the water is far more dangerous than we had believed. When we only had small bladder BCs, it was more difficult to make a rapid ascent.

#### Development of buoyancy compensators

Until the 1970s few divers used buoyancy compensation devices. Being correctly weighted to be neutral at the depth of the dive was considered adequate. If one needed any additional buoyancy one would blow some air into the wetsuit sleeve. This would form a bubble between the shoulders. To get rid of it, one raised an arm and the bubble ran out. Many people felt no need for a buoyancy compensator.

However as more people took cameras and other heavy objects underwater, it became obvious that it was handy to have some way to displace water so that one could achieve additional buoyancy at depth. At first the bag was in front of the chest. These did little to complicate the rate of ascent because the bladders were generally small, with only 6.8-8.2 kg of lift. Additionally, the divers of the time rarely put air in the bladder unless the need was critical. While this amount of lift was sufficient to increase the rate of ascent, the problem was not significant since the divers were skilled in the use of the devices, which were only intended to be used to maintain neutral states in the water column or float them on the surface. Later back mounted bladders and jacket configurations became popular.

Divers need to be able to displace water close to or just above their centre of mass. On the surface there is a portion of the buoyancy compensator that is only displacing air. To float the diver adequately there must be a significant amount of air underwater. Larger buoyancy compensators displace more air when they are on the surface but displace about the same amount of water as smaller ones, so the diver is not necessarily further out of the water. Underwater they can displace more water, but the bigger bubble causes increased lift, which is a matter of concern.

#### Buoyant ascent rates

A study at the University of California, Los Angeles, (UCLA) Underwater Kinesiology Laboratory in 1980 tested a number of buoyancy compensators, with positive buoyancies of 5 to 24.5 kg,<sup>1,2</sup> in our fresh water pool. The

diver, in full ocean gear, 7 mm wet suit, booties and gloves, tank, regulator, backpack and an adequate amount of weight for neutral buoyancy was placed horizontally at a chest depth of 2.85 m (9 ft 4 inches), holding the sides of a weighted box. The BC was filled until the overpressure relief valve was activated. The diver was then signalled to exhale fully, relax and let go. The ascents were timed and video taped. All the divers changed from horizontal to vertical on the way to the surface. The smallest buoyancy compensator brought the diver up at an average speed of 20.6 m (68 ft) per minute.

Average ascent rates were calculated for the whole 2.85 m (9 feet 4 inches) ascent. We did hundreds of buoyant ascents. Some people came up awfully fast, although they were only coming up two to two and half times faster than they were supposed to when we looked at the average ascent rate.

When we studied the video tapes for the last 1.2 m it was a different story. The smallest vest came up the last 1.2 m at a slower rate than it did overall. The explanation is that the divers started horizontal, became vertical and then, because it was a small bubble high on the chest, they were arched backwards by the vest. This put them at a significant angle and consequent they were slowing through the last 1.2 m. On the other hand the medium and larger vests were accelerating through the last 1.2 m. These took the divers vertically, right up to and straight through, the air-water interface. Sometimes the diver came out of the water to his waist. Some of the larger over the shoulder models reached ascent rates of 76.5 m per minute. Table 1 summarizes the data derived from this study.

### Ascent rates

Since 1951, the USN Diving Manual has told us to come up at 18 m per minute. About two and half years ago I was told how the USN chose 18 m per minute. Doctor Ed Lanphier had the job of recommending an ascent rate. He realized that there were two populations of Navy divers. One was swimmers who wanted to pop out of submarines and go as fast as they could to the surface. They were happy with 54-60 m per minute, and there were the Navy hard hat divers who were very satisfied with, and used to, 7.5 m per minute, which was the ascent rate for the then USN decompression tables. He reasoned that if the hard hat divers came up a little faster and the swimmers came up a little slower, things would be alright. He suggested that 60 ft (18 m) a minute, a foot a second, was a nice round number that sailors could remember. The committee discussed and accepted it. The 18 m per minute ascent rate has been used countless thousands of times, and it has turned out to be a really good guess. That it was nothing more than a compromise and had no significant research behind it was a bit of a shock to me.

Divers are often told to ascend at the rate of their smallest bubble. To find a bubble that goes up at 18 m per minute, one has to be pretty selective. The tiniest bubbles that one can see come up between 13.5 and 18 m a minute. That is the fuzz in the water. Anything bigger than fuzz is coming up faster than 18 m per minute. One of the worst things that a diver can do is to fix on a bubble and follow it up, because as it expands, it goes faster and faster.

TABLE 1

### BUOYANCY COMPENSATOR SIZE, SHAPE, DESIGN AND ASCENT RATES

Inches	Size cm	Shape	Volume litres	Lift		Ascent rates			
				lb	kg	Full distance (9' 4" = 2.85 m)		Last part (4' = 1.22 m)	
						fpm	mpm	fpm	mpm
20x7"	51x18	Single bladder HC	5.1	11.2	5.1	68	20.6	43.3	13.1
23x18"	59x46	Single bladder HC	12.7	27.9	12.7	122	37	147.8	44.8
23x19"	59x49	Bladder in bag HC	15.5	34.1	15.5	132	40	185.8	56.3
24x19"	61x49	Bladder in bag HC	17.2	37.8	17.2	138	41.8	187.8	56.9
24x18"	61x46	Bladder in bag HC	17.5	38.5	17.5	143	43.3	205.7	62.3
25x20"	64x51	Bladder in bag HC	17.3	38.1	17.3	149	45.2	208.7	63.2
19x16"	49x41	Bladder in bag HC	21.6	47.5	21.6	156	47.3	213.3	64.6
		Large Jacket type	21	46.2	21	149	45.2	225.9	68.5
26x10"	67x25	BIB Backmounted	24.9	54.8	24.9	168	50.9	245.3	74.3
		BIB Overshoulder	21.2	46.6	21.2	150	45.5	254.7	77.2

HC donotes horse collar type of buoyancy compensator. BIB indicates bladder in bag. Wet suit expansion was assumed to be constant during the ascent..

In one study of ascent rates there was a wide variation with few ascents being slower than the recommended 18 m per minute. It was commonplace to witness divers ascending at two to three times this rate who, when questioned, would indicate they were travelling at the rate of their smallest bubbles as they did on all of their ascents. Telling people to follow the smallest bubbles as they come up may not be the best advice one can give.

### **Dangers of buoyancy bubbles**

As buoyancy chambers got larger, there were more problems with rapid ascents. The development of larger bladder configurations, which could hold a bubble of gas in a variety of positions depending on whether the bladder was front mounted, back mounted, over the shoulder or around the body, requires careful attention to the size of the bubble since larger bubbles lead to larger changes in buoyancy with changes in pressure.

Large bladders, with buoyancy potentials up to 34.4 kg or more in some extreme cases, offer another potential risk for divers with poor water skills, as the large bladders can compensate for significantly greater amounts of weight. The diver could, by inflating or deflating the BC, move up or down in the water column with considerable speed. No longer was there the need to develop surface diving skills to overcome the slightly positive state which normally existed on the surface at the beginning of a dive where the diver was properly weighted. "Push button diving" permits the diver to constantly adjust buoyancy throughout the dive and the ascent.

Unfortunately the management of a large bubble in a buoyancy compensation system is infinitely more difficult than the management of a small bubble if one is not, unintentionally, to become positively buoyant during ascent. Ninety per cent of the fatalities in our area over the last 5 years were, in our opinion, overweighted. They also had large buoyancy compensators. People are wearing 7 mm wetsuits, with 11.8 to 12.7 kg of lead. Many of them insist that is how much they need to get down. That may be so with some of the newer, thicker foam materials. A thicker suit needs more lead to sink. But as soon as it goes down, the bubbles in the suit compress and the diver becomes considerably negatively buoyant. We no longer believe that one is correctly weighted for a dive when one floats with the water level with one's eyes and if one exhales one sinks. Now we want our divers neutral at 3 m rather than on the surface.

Additional buoyancy does not create a major problem when tackled properly. Anything that one does, putting one's arms out to the sides, spreading one's legs or arching one's back, will slow the ascent rate. All these will cause one to rise at an angle rather than vertically, in which position the ascent rate is faster. But they need to be initiated early in the ascent to be most effective in ensuring control

through the entire ascent. Ascent control is a precautionary skill that must be put into effect early and reinforced often during the rise to the surface.

Jacket type compensators have loose arm holes for easy entry. When inflated underwater the bubble rises to the highest part of the jacket. When ascending the bubble is over the shoulders lifting the jacket up beside the diver's ear. During ascent, the diver may have to reach up well over his head to find the dump valve if it has floated free. There is plenty of room for air to expand in these large compensators. If a diver leaves 27 m with a partially filled compensator when he or she gets to about 6 m the gas will have expanded to practically fill the buoyancy compensator and he is not going to be able to stop.

Many people favour back mounted compensators because they want to have the front open. They need to understand that should they become unconscious, they will float face down. Once I watched a man wearing a newly purchased, back mounted compensator hanging off a line at the end of the dive. He gradually changed position. His feet came up and up. When he got horizontal, I recognised that something was going wrong. He ultimately went feet up and as he did, he let go of the line and made an attempt to pull the dump valve, which was located on the shoulder. Unfortunately, the dump valve was now below the air bubble and he shot to the surface. He embolized but not seriously. He did not have the insight to manage his bubble. As a consequence, he got himself into trouble.

Unfortunately the majority of the divers do not have good buoyancy skills. Most are buying equipment that will give them a large bubble and they do not take the time to learn the skills of how to manage a large bubble. Divers need to be able to vent gas from buoyancy compensators as they swim along. Around the world, we see people who put air in the buoyancy compensator, which is absolutely fine, and then when they start to ascend they forget that they have to start venting early and often. When they get up to somewhere around 4.5 m, even if they start thinking about venting air, in the time it will take to initiate the dump, they are going to continue to ascend and lose control. Accidents occur when a diver loses control. If one is able to regain control, then one avoids injury, but one still had the accident but avoided injury. If one does not regain control, then one can expect there is going to be injury.

The buoyancy compensator is a significant problem but it is not the only problem. Drysuits with large amounts of gas are also difficult to control, and divers have died after becoming inverted and being unable to get head up again at the surface. Drysuits must have a reliable rapid exhaust valve which can be used effectively whether the individual is in the horizontal or the vertical position. It is clear that the dry suit manufacturers, and instructors, who are advocating significant instruction prior to the use of the suit are providing fundamental knowledge and skill to the diver who

wishes to use these devices. The American Academy of Underwater Sciences (AAUS) has made it in as a requirement, for scientific divers, to use a buoyancy compensator with a drysuit, largely because of the problems associated with trapping air in the legs and not being able to get back into a upright position.

### How to stay out of trouble

One should make it a routine to weight oneself and deal with ones buoyancy so that one will be able to come up slower than 18 m per minute. Sixty feet per minute does not seem to create problems for most people. The recommendation however is to slow down and come up at a reduced rate, something of the order of 12 m per minute. This has some advantages.

The safety stop that has been the largest single step for safety that we have taken in the last few years. In my view its purpose is not to provide additional decompression time. It does and that is a benefit. The major benefit is that whatever a person's ascent rate was, a stop close to the surface means they are far more likely to be in control through the last few metres of ascent. They are far less likely to have the BC expanding so rapidly that they will be rushed to the surface and into trouble.

### Safe ascent recommendations

A recent workshop, conducted by the American Academy of Underwater Scientists, published Safe Ascent Recommendations<sup>3</sup>, with the comment that "It has long been the position of the American Academy of Underwater Scientists that the ultimate responsibility for safety rests with the individual diver. The time has come to encourage divers to slow their ascents". The recommendations were:-

- 1 Buoyancy compensation is a significant problem in the control of ascents.
- 2 Training in, and understanding of, proper ascent techniques is fundamental to safe diving practice.
- 3 Before certification, the diver is to demonstrate proper buoyancy, weighting and a controlled ascent, including a "hover" stop.
- 4 Divers shall periodically review proper ascent techniques to maintain proficiency.
- 5 Ascent rates shall not exceed 60 feet of seawater (fsw) or 18 m per minute.
- 6 A stop in the 3-9 m zone for 3-5 minutes is recommended on every dive.
- 7 When using a dive computer or tables, non-emergency ascent are to be at the rate specified for the system being used.
- 8 Each diver shall have instrumentation to monitor ascent rates.
- 9 Divers using dry suits shall have training in their use.

- 10 Dry suits shall have a hands-free exhaust valve.
- 11 Buoyancy compensators (BCs) shall have a reliable rapid exhaust valve which can be operated in a horizontal swimming position.
- 12 A BC is required with dry suit use for ascent control and emergency floatation.
- 13 Breathing 100% oxygen above water is preferred to in-water procedures for omitted decompression.

It was the consensus of the group that produced the recommendations that, because there is no way to stop people from using whatever equipment they want, divers must learn how to to slow down their ascent rate and need to be taught buoyancy control as a skill. They need to be tested on buoyancy control and they need be to able to demonstrate that they can hover in any position before certification. This can be a difficult skill to learn.

### Implications for training

The implications for diving instruction are clear. The training agencies are doing a far better job than they did in the past in getting their instructors to recognise that buoyancy control has to be taught as a skill. Diving instructors have to pay more attention to making sure that their students are skilled in the management of whatever the buoyancy system they are using. It takes time to develop these skills. Divers must understand that the controls for different buoyancy compensators work in different ways. They should take a good look their buddy's buoyancy control system before a dive to ensure that they know how to dump gas rapidly from it if the buddy loses control and starts floating rapidly to the surface. Buoyancy compensation is a tool and not a crutch.

### References

- 1 Egstrom G. A few words on ascent rate, Proceedings: *International Conference on Underwater Education*. NAUI, San Diego 1982.
- 2 Egstrom G. Biomechanics of buoyancy compensation and ascent rate, *Proceedings of the Biomechanics of Safe Ascents Workshop*, Lang M. and Egstrom G. (eds) American Academy of Underwater Sciences, 1990.
- 3 Recommendations from the *Biomechanics of Safe Ascent Workshop* held at Woods Hole, Mass. Sept. 25-27, 1989.

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## INVESTIGATION OF DIVING ACCIDENTS AND EQUIPMENT

Glen Egstrom

### Los Angeles County ESD

Los Angeles County has the distinction of consistently having more diving fatalities than any single community outside the state of Florida. We have anywhere from 6 to 16 fatalities a year. Some of them are pretty bizarre. Years ago I became involved with the ESD (Emergency Services Detail) in Los Angeles County, a highly trained group of individuals who do all the rescue and recovery for the Sheriff's Department. They needed to learn to dive. I had to go through a Sheriff's Law Enforcement Training Academy and become a reserve Sheriff before I could teach them. I have been with them for some time now.

In most communities where search and rescue is combined with recovery there is a tendency to treat a fatality as a rescue. One of the most difficult things has been to get them to understand that one rescues someone if there is possibility, or probability, of bringing the person out alive. If the person has been lying on the sea floor for much beyond an hour, it does not make a sense to tackle it in the same way as a rescue. During rescue operations the ESD people are willing to lay their lives on the line. I found, after having trained with them for a while, that they were as willing to lay their life on the line to go down and recover a dead body, as if it was a rescue.

They felt they had to get the body back because everybody in the family would feel so much better when they saw it lying on the dock. There is a great deal of emotion associated with a person who has died during any kind of activity, but particularly when scuba diving. People think that under the water, the crabs and the sharks are going to do their thing, so one must hurry to retrieve the body. We have been able to get them to recognise that it is not worth risking another human life to recover a dead body. This means that body recovery should be treated like any other salvage operation. There is no need for haste, there is no need for going into conditions that are environmentally marginal and there is no need for anyone to feel they have to make a heroic effort to try and bring back the body.

### Body recovery

In communities where there is enough diving, there is a need to formalise the way in which the chain of evidence is dealt with when trying to get enough information to understand how the accident took place and where correctly to put the blame. One of the difficulties that we have today in the United States, any time there is an accident, is that if anything is found to not be proper that immediately becomes

the proximate cause of the accident. We feel that a thorough chain of evidence is needed to be able to differentiate causes and to make recommendations to the diving community at large about what takes place when there are accidents.

With that in mind, we asked professional law enforcement officers how they go about reviewing a crime scene. What are the kinds of things that they do. They told us about the chain of evidence, the details of the body, the photographs, the identification, where the bodies are, what circumstances might be associated with the environment that the body was found in that might give information about what had happened. This has been taken up by our unit and we have established a group called the Interagency Scuba Committee. This has representatives from the Fire Department, the Sheriff's Department, the Police Department, the Harbours people, life guards and anybody who would be involved in a potential body recovery. As a result everyone knows what to do.

For a scuba diving fatality, or even a serious accident, there is a phone link with the Sheriff's office. This 919 number can be radioed straight into the Emergency Services Detail and they immediately send a trained law enforcement person to the site of the accident or the site of the fatality or to where ever the body is being taken. For example if the person died on a boat, they immediately get the names of witnesses, take charge of the equipment and put into a evidence bag, so that it is isolated, and check that they understand what has happened to the individual and to the equipment during the time since the recovery.

On the other hand, if it is a body that has been down for some time, or we go into a search operation, we use a very different tactic. Instead of finding the body and immediately ditching the weight belt and hauling the body to the surface, as one used to, we now treat it like a crime scene.

When the searchers find a body the first thing is to do a short closer examination to see if there is anything that we will have to spend some time on in relation to documentation. We then do photographs to show things. In one case there was a great deal of swelling in the lips with a relatively small opening and the regulator was out. We could tell that the regulator had been pulled out after the individual had gone into rigor mortis. We check to see if there is anything in the mask or any leaks from the scuba system. We check whether the weight belt been ditched. We take a look at the contents gauge. It is not unusual for the contents gauge to read nearly full. It is really quite rare, less than 1 in 5 cases, for people to be out of air. We have investigated a number of accidents where the tank pressure was 2,800 and 3,000 psi, and at least one of these the diver had simply failed to turn the tank on. We note the maximum indication on the depth gauge and the depth where we find the body, if there are injuries and the state of inflation of the BC. We try to get all the information before we start moving the body.

## Records

There are forms for recording the individual's vital statistics, the names of the witnesses, who they were and the name of the deputy filling in the documentation. We get a ID number from the coroner and a serial number associated with the Los Angeles crime records.

We also have a personal inventory. It includes all the diving equipment. If there is some other personal equipment on a dive boat, we also impound that. We have had a number of interesting surprises with what people have in their personal luggage that they probably used just before their fatal dive. The contents of the luggage can help the medical examiner make decisions about the autopsy protocol.

There is also a Supplementary Diving Report. This is where a lot of the statistical information ends up with a description of where the body was found, how deep it was, what kind of position was the body in, who were involved and places to check off some of the circumstances were. I dislike the term "involuntary separation". It should be "separation" because involuntary is a presumption. Many divers perform what I call "voluntary separations". They have different ideas about the dive and go to different places. They only join up periodically. We have a surprising number of cases where, at the time of the accident, the buddy pair were separated by a distance that made it impossible to participate any kind of a rescue.

The next step is an interview with the personal friends, room mates and members of the immediate family to get some information about the victim's physical condition and their diving physical (medical examination) history. Whenever this information is unavailable, an investigator talks to whoever would be knowledgeable. These interviews provide very interesting and often helpful information. In many cases we have determined, from the autopsy, that victims had used cocaine.

We had a local diving cult called the Jelly Beaners. They derived their name from the fact that a number of drugs came in brightly coloured capsules that looked like jelly beans. They would pop a few and dive and see what kind of fun and mystical things would happen underwater. Many of them survived.

We investigate who trained the victims, their level of experience for that level of training, their diving experience during the past year, whether or not they had any previous kinds of accidents and who owned the equipment. We check whether any adjustments had been made to the equipment by anyone.

## Equipment handling

The directions are that, from the time of recovery, no

one touches the equipment before it has been photographed and examined, unless there is a chance of a free flow. In that case we ask them to make a mark across the handle and the valve and then turn it off recording how far they have to go. We do that because we have had several cases where the divers, instead of turning the valve all the way on and backing it off a quarter of a turn, have turned it all the way off and cracked it on for a quarter of a turn. That does not make a much of difference early on in the dive when there is a sufficient differential pressure. However at the end of the dive, it creates another restrictive orifice which can turn a good breathing regulator into a very difficult breathing regulator, even if the regulator works perfectly.

We have evidence lockers for each of the pieces of equipment so that any time that it is not being tested we maintain its integrity by keeping it in a separated storage area.

## Equipment investigation

Each piece of equipment is examined and tested. We make a record of serial numbers. We make a record of how it is attached to the tank. We check that regulator was attached. It is not as odd as it sounds. Over the past three to five years we have had cases where, when we got the equipment, the regulator was so loose that the tank would not hold air. One of the recent deaths was a commercial diver on hookah, using a tank as back up, while working through kelp. Divers on hookah in kelp often have a quick disconnect on the hose so that they can go onto the pony bottle, get themselves out of the kelp and then re-connect and go about their business. They use the pony bottle partly as a tool and partly as a backup. This man was still attached to his hose, lying dead on the bottom and there was no air in the tank. The assumption was that he must have breathed it dry. On the forms it was recorded that the regulators were attached, the control valve was on and that air was not leaking. However that was because the first stage regulator was functionally separated. What probably happened was that the diver made an attempt to get out of the kelp by plugging into his pony tank, which was upside down. As the air pressure went out of the tank and he started moving up and down water got into the tank, because the first stage was loose, and collected at the lower end, where the valve was. Soon his regulator delivered a mixture of water and air, that had a slight pressure head of air behind it. He then aspirated and drowned, with the regulator in his mouth.

We check all the valves. With anything that can be wound in and out we make a mark on the shoulder, and make a matching mark on the handle and count the number of revolutions available. This also applies to dials that will change the breathing characteristics of a regulator. Scubapro produced a regulator that has been referred to as "dial a death". It in fact is a very good regulator, but by twisting a knob one can detune it and turn it into a very bad regulator.



It has about four and a half turns of adjustment. The designer now agrees with me that it should have been made as a screw driver fitting rather than a knob which gives divers the opportunity to change its characteristics at will. There are people who believe that if they increase the breathing resistance on the regulator, their tank is going to last longer. Unfortunately it only seems to them that it lasts longer.

We check the cylinder size, how it is rated and what kind of condition it is in. We look inside the air cylinder. It is surprising that we find water in about 1 in 4 cylinders. We are not always sure how the water gets into the cylinders. If there is even two teaspoonfuls of water in the cylinder and the diver inverts, the water gets into the valve. Then with the next breath the diver gets the water first, this can be as a bolus or as a spray, and then the air. This is a very stressful process.

We check the kind of floatation, how much lift it had and the amount of weight worn. All these things are recorded in order as we go down the check list. The completed check list gives a record of what took place at that particular point in the chain of evidence.

We see some interesting equipment. Sometimes we suspect that the victims were not used to the diving equipment they were using and had not bothered to take instruction. One dead diver was about 1.6 m (5 ft 4 in) tall and weighed about a 55 kg (120 lb). The wetsuit did not fit but he should have been able to operate with half the 10 kg (22 lb) he was wearing.

Some 1st stages did not have a contents gauges, although there was always a port for one. One person died using a sonic regulator. These make a rattle sound when the tank pressure is low. It was a good operational regulator. But it did not have enough low pressure ports to take care of all the hoses that were needed for a buoyancy compensator, drysuit and octopus. This stubborn individual, decided not to put these hoses on because he did not think he would need them. He had a single first stage, no alternative air source and no auto-inflation system. He did not have the rod that operated the J Valve, however the J Valve had been displaced about 6 mm (a quarter of an inch). That was sufficient to release the spring and allow the reserve air to be used. With a J valve the diver normally takes a breath that is hard to breath, then reaches behind and presses down the loop and gets the reserve pressure. There was no evidence that he had done that.

One diver died wearing an Atpak. These are back mounted buoyancy compensators where the back pack is used as a repository for lead shot as an alternative to wearing a weight belt. The idea is that should one need to get rid of it, you reach around the back, put your finger through a wire loop and pull a 16 cm (7 inch) wire out of something like a piano hinge. Then a trap door will open and all the shot should fall out. Unfortunately all the lead up in between the shoulder blades floats an unconscious diver face down. We

have had a number of these backpacks come through our hands. One had the wire pushed in a little further so that the end could be looped to keep it out of the way. He had not worked out that when one does that one can no longer pull the pin out.

Some of those who died had very heavy backpacks because of their weighting system. An Atpak, with the cylinder full of air, weighed 38 kg (84 lb) which is an awful lot. One innovative individual cut out the centre of his plastic backpack and slid sheet lead down into the hole. The backpack and cylinder normally weighed around 21 kg (46 to 47 lbs), but it weighed 31 (68 lb) with this modification. He fell over in the surf and was never able to get back onto his feet. He drowned in about 50 cm (18 to 20 inches) of water. One very creative diver did not have an accident. He transferred the weights to his backpack with no system of jettisoning, the result could be described as a poor man's version of the Atpak.

We have seen some interesting weight-belt innovations. We think is a bad idea not to be able to ditch a weight-belt. Apparently some divers feel that they do not want to lose the weight-belt because that they put double buckles on. Then the diver has to undo two buckles before the belt starts to fall. Wearing a knife on the outside of the leg means that if one ditches the weight-belt it can come down behind the knife handle and hang there.

If the weight-belt tongue is more than four or five inches long, we note the fact. In this circumstance if one opens the buckle and the belt starts to fall away, the section with the weights is going to move faster than the section without weights. That causes the belt to go crosswise in the buckle and it will usually jam. We have done this experimentally and seen it happen. In one death a belt had closed its buckle around a fellow's ankles, having been hooked up on his knife and lower leg. A belt with 8-9 kg (18 to 20 lbs) of lead, hanging on a knife handle or round one's legs makes survival difficult.

Tucking the long tail of a weight-belt inside, or round and round the belt is not a solution. If one is depending on ditching weights to provide positive buoyancy if one gets into trouble, then a long belt is not in one's best interest. Nor is tucking it inside, because in an emergency, or a panic situation, undoing the buckle will do nothing towards dropping the belt.

We take some of the gear into a swimming pool to test to see if it is operational. In one case the detontating cord on the CO<sub>2</sub> cartridge had one loop around the CO<sub>2</sub> cartridge so that no matter how one pulled the handle, the arm would not come down to puncture the CO<sub>2</sub> cartridge.

On several occasions recently, we have found there was no low pressure hose attached to the BC. This usually indicates some kind of free flow or malfunction and that the

diver separated it deliberately, because we teach them how to do that in our training program. If the power inflator starts to blow, one must immediately disconnect it because that is the only sure way of being able to shut it off. If you have not done it before, because you have not had the problem, you will find it really awkward to do, particularly if you have one of the smaller disconnect mechanisms.

### Equipment testing.

Testing the equipment which is laborious. We have a complete set of catalogues with specifications of all the regulators and equipment that are used in our area. We also have all the appropriate tools as many of the devices take specialized tools. We use our library to see what changes from the manufacturers specifications have taken place.

The first step is to test the regulator. We use a differential pressure gauge attached to the regulator. Then we put the regulator in someone's mouth and get them to breathe normally. We record the inhalation, exhalation and differential pressures. Then we use forceful inhalation and exhalation and record the same pressures. This enables us to decide whether it is a functional regulator. We do the same for all the breathing equipment, octopus, Air II or alternate air sources.

We always check the intermediate pressure to determine whether there are any leaks, and also to determine whether the appropriate intermediate pressure was operating on the regulator.

After this we test the regulator on a breathing machine inside a small hyperbaric chamber. We can flood the chamber or leave it dry. Our protocol requires that we test the regulator on the surface and at 10, 40, 60 m. It is all automated. We measure at 6, 15 and 30 breaths a minute with a two and half litre tidal volume recording the performance under all these circumstances. We also test the regulator at the depth at which the body was found.

Regulator failures can involved tuning, in-leaks or out-leaks. It is very rare that we have a circumstance where there is not enough air remaining in the cylinder to operate the regulator.

In-leaks sites are going to be at the exhaust valve or the mouthpiece. One has to pull on the mouthpiece to be able to detect these cracks. Under normal circumstances the elasticity of the rubber will hold them together. But in a person's mouth, there is enough distortion to let water leak through the mouthpiece. The other site is the exhaust valve. Debris under the flap valve will prevent it seating and will let water in. Entering the water from a boat with the regulator in the mouth, the water pressure may be sufficiently above the pressure in the regulator to drive part of the exhaust valve back into the regulator. That will provide a wet breathing

regulator throughout the dive. If one blows very hard the flap sometimes pops back out again and then the leak will stop.

Out-leaks are generated by O rings. That is usually because the tank valve does not match the regulator. One has to be very careful lining up the valve and the regulator or there will be a leak. Valve seats and hoses also have to be checked for out-leaks.

### Conclusions

By developing a clear protocol for recording a chain of evidence and co-operation between all authorities concerned the Los Angeles ESD has improved the investigation of diving accidents. The results of careful inspection and testing of the diver's equipment, with evidence gathered at the scene of the accident have allowed us to discover the actual cause of death. This has enabled us to draw attention to dangerous practices in an effort to prevent further deaths.

*The above paper is an edited transcript of a lecture delivered to the 1991 Annual Scientific Meeting of SPUMS.*

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## INTERNATIONAL CONGRESS ABOUT DIVING AND HYPERBARIC MEDICINE

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## ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

### HOW EXPERIENCED DIVERS REALLY DIVE

Bob Halstead

#### Introduction

I love diving, you could say I am fanatical about it. Which is why, 15 years ago, I gave up a good secure job teaching physics to start up a dive school and run dive tours. This has enabled me to make a lot of dives. They are not logged, but if I had to make an estimate, probably over 6,000. Diving is as personal and glorious to me as sex, and would be degraded if I felt I had to write about it after every session. So I do not log my sex life, and I do not log my dive life.

Taking students and tourists diving has also enabled me to see a lot of other people dive and I have observed all degrees of success and enjoyment from euphoria to despair. Estimating again, I have probably supervised over 100,000 dives. All this time I have tried to find ways of reducing the despair and increasing the euphoria for the divers in my charge.

One of the things I have learned is that the way that successful experienced divers really dive often differs from the way that safety nuts say they should dive. I define a "safety nut" as anyone who believes that rules are more important than thinking. Unfortunately, in this life, there are many people who just love to tell you what you should be doing.

Anyone would suppose that the rules for safe diving were inscribed in some Deep Sea Scrolls, true for eternity, instead of being a code of practice constantly evolving in the light of research and experience. It is obvious to me that divers who are at home in, and in harmony with, the sea, and who have contemplated their experience then studied and practised to perfect their skills, will be able to make dives that others less able would find dangerous. At the same time these divers can recognize when the conditions are such that even their abilities are insufficient to make a dive safe, and they will not dive.

Much research is now taking place trying to discover what causes diving accidents. On reading these reports I realized with some shock that they were being analyzed with reference to what the authors imagined was the way the divers should dive which is not the way experienced divers actually are diving. For example, if an analysis showed that 20% of the fatalities in a given year were of solo divers, authors who assume that the norm is "never dive alone" would argue that this demonstrates how dangerous solo diving is, and how important it is to dive with a buddy. Of course if 20% of all dives were in fact solo dives what the

analysis would logically show is that diving solo or with a buddy does not affect diving safety. I could not find any study of how experienced divers actually dived so decided to make a survey to see if I could find out. This article is about the results of that survey.

#### The survey

Over a period of one year I asked our guests aboard Telita to complete, anonymously, a survey form. I also sent survey forms to our past guests and divers on our mailing list. Finally, survey forms were distributed to Australian NAUI instructors. In all 650 forms were distributed and 283 (44 %) were completed and returned. We deliberately market Telita to experienced divers, nevertheless 18 of the responses were by divers who had made less than 100 dives. These 18 responses were not used in any further analysis leaving a sample of 265 (41% of forms sent out). Some divers failed to respond to certain sections which is why the percentages do not always total 100. Percentages have been rounded off to the nearest whole number. The tables below give the actual numbers and the percentages.

#### The results

<b>1 AGE</b>		
30 years or less	23	9%
31 - 40	80	30%
41 - 50	77	29%
51 years and over	85	32%
<b>2 SEX</b>		
Male	181	68%
Female	83	31%
<b>3 NUMBER OF YEARS OF DIVING</b>		
Less than 10	93	34%
10 or more	265	65%
<b>4 CERTIFICATION</b>		
Basic or Openwater	114	43%
Higher	80	30%
Instructor	66	25%
<b>5 NUMBER OF DIVES</b>		
100 - 499	112	42%
500 or more	151	57%
Total dives	234,631	
Average number of dives	885	
<b>6 LOG BOOK</b>		
Log dives	143	54%
Do not log dives	101	38%

<b>7 DEEPEST DEPTH</b>		
39 m or shallower	29	11%
39 - 60 m	170	64%
Deeper than 60 m	61	23%
<b>8 DECOMPRESSION DIVE</b>		
Have made a decompression dive	207	78%
Never made a decompression dive	53	20%
<b>9 DECOMPRESSION SICKNESS</b>		
Have had decompression sickness	18	7%
No decompression sickness	241	91%
<b>10 REGULATOR FAILURE</b>		
Failure with no air underwater	56	21%
Never had regulator failure	204	77%
<b>11 EMERGENCY ASCENT BECAUSE THEY HAD NO AIR</b>		
Had to make emergency ascent	95	36%
Never made emergency ascent	164	62%
<b>12 METHOD USED IN EMERGENCY ASCENT</b>		
Some divers have made several emergency ascents.		
Total number of emergency ascents	173	
Emergency swimming ascent	92	53%
Buoyant ascent	16	9%
Buddy Breathing	31	18%
Octopus ascent	31	18%
Spare air or pony bottle	3	2%
<b>13 EMERGENCY ASCENT BECAUSE BUDDY HAD NO AIR</b>		
Had to make emergency ascent	93	34%
Never made an emergency ascent	176	67%
<b>14 METHOD USED IN BUDDY EMERGENCY ASCENT</b>		
Total number of emergency ascents	190	
Buddy Breathing	89	47%
Octopus ascent	101	53%
<b>15 BUDDY DIVING</b>		
Always dives with a buddy	69	26%
Mostly dives with a buddy	109	41%
Sometimes dives with a buddy	50	19%
Rarely dives with a buddy	29	11%
Never dives with a buddy .	5	2%

**16 BUDDY DIVING HABITS**

The 187 (67%) who said that they always or mostly dived with buddy were asked to make the following choices best describing their usual habits:

<b>i Buddy checking:</b>		
Check buddy continuously	54	29%
Check buddy occasionally	129	69%

<b>ii Buddy contact</b>		
Within touching distance	19	10%
Within vision	161	86%
<b>iii Exits</b>		
Always exit together	67	36%
Sometimes exit separately	116	62%
<b>iv Buddy's air supply</b>		
Check your buddy's air	65	35%
Wait for buddy's signal	108	58%

**17 SKILLS PRACTICE**

In the past year have you practiced:

<b>i Equipment removal and replacement underwater.</b>		
Yes	109	41%
No	154	58%
<b>ii Buddy Breathing</b>		
Yes	106	40%
No	156	59%
<b>iii Octopus breathing</b>		
Yes	122	46%
No	143	54%

**18 USE OF "SPARE AIR" OR PONY BOTTLE**

Usually take on dive	29	11%
Do not use	228	86%

**19 USE OF DIVING COMPUTER**

Use diving computer	215	81%
Do not use diving computer	42	16%

**20 BREATH-HOLD DIVING ABILITY**

Dive to less than 9 m.	113	42%
Dive 9 m or deeper.	148	56%

**21 UNDERWATER PHOTOGRAPHERS**

Take underwater photos or video	191	72%
Are not underwater photographers	72	27%

**22 BUOYANCY COMPENSATOR FAILURE UNDERWATER**

Have experienced sudden failure	74	28%
Never had failure	188	71%

**Comments**

Here is where I show that the statistics prove all of my outrageous ideas about diving. Well, actually no. Statistical arguments seem to follow Newton's Third Law:- "For every argument there is an equal and opposite reply", so my observations will be modest. I claim to be a diver not a

scientist. Whatever minor liberties, if any, I have taken with the analysis, the figures are just as I found them.

### **The experienced diver**

The typical experienced diver surveyed is older, 61% were older than 40 years, has dived for 10 or more years (65%), has made a lot of dives (average 885 each), and is still diving (the survey was of active divers). One third are women (31%). Our experienced divers do not necessarily have good diving qualifications, only 25% of our sample were instructors, and this is only because the survey included Australian NAUI Instructors rather than being confined to Telita clients.

Each diver surveyed was asked the number of dives that they had made. Many were able to give precise figures from their log books (54%), some gave estimates. The survey was anonymous so that there was no motivation for over estimating. Wherever an estimate involved two figures e.g., 6-700, I always used the smaller figure. Several put "hundreds" or "thousands" which I always interpreted as 100 or 1,000. I am confident that the figures are a real indication of the number of dives experienced divers, some of whom are professionals, have made.

### **Deep and decompression diving.**

Only 11% have stayed shallower than the standard recreational limit of 39 m, and 23% have dived deeper than 60 m. 78% have made decompression dives, and 7% have had decompression illness. (Some divers mentioned minor symptoms that resolved without treatment and were not even certain they had been bent). Sixty six per cent of those reporting DCI had never dived deeper than 60 m.

If the 18 divers who had DCI had only been bent once each (I did not ask that question, although some made it clear that only one incident was involved, and no one volunteered that they had been bent more than once), the incident rate works out at less than 0.01%. It was one bend for every 13,035 dives, or 0.0077%.

Our profile of the experienced diver shows that they have typically dived deeper than 39 m and have made decompression dives, yet probably have a bends rate that is less than the average commonly reported for all sport divers. Most (81%) are now using diving computers to monitor their dives.

### **Equipment failures**

Twenty one per cent have had a regulator fail underwater giving no air, and 28% have had a buoyancy compensator (BC) suddenly fail underwater either by leaking or

continuously inflating. These are significant percentages and it is obvious that the possibility of equipment failure cannot be ignored. A common response to this is to say that divers should get their gear properly serviced regularly, and of course I agree. However I am always being asked to fix client's gear that is new, or that has just been, so they thought, properly serviced.

I believe that every diver should be trained to survive a situation which results from regulator or BC failure.

### **Emergency ascents**

Thirty six per cent have had to make a total of 173 emergency ascents because they had no air underwater (regulator failure or ran out of air) and 34% have had to make a total of 190 emergency ascents "because their buddy had no air". Our experienced divers were more likely to have to make an emergency ascent because of their buddies problems than their own. The most popular method by far, for those that found themselves in trouble, was the Emergency Swimming Ascent, 53%, against 18% each for buddy breathing and octopus breathing. A low percentage of divers used "Spare Air" or other pony bottle systems (11%).

Our divers did not use a buddy assisted method often as 64% used an independent method to reach the surface rather than have their buddies help them with an assisted emergency ascent. It is worth noting that less than half had practiced buddy breathing (40%) or octopus breathing (46%) in the past year.

The figures do not surprise me at all. What surprises me is the fact that many divers automatically assume that having a buddy is safer. In fact a buddy may make the dive safer or may add to the risk of a dive. Some have been saved by their buddy. Unfortunately those who have perished because of their buddy are not around to testify. However 34% of our divers were put at risk having to make a total of 190 emergency ascents because of their buddies.

The obvious answer, as far as safety is concerned, is to have a foolproof, independent method of getting to the surface if you have no air, and to make sure that your buddy has one as well.

### **Buddy diving**

Only 26% followed the golden rule of "always dive with a buddy" and, while a further 41% mostly dived with a buddy, some complained that the reason is that they were not allowed by dive boats to dive alone, even if they wanted to.

The next four questions were designed to see if those always or mostly diving with a buddy (the others were not

counted) were conscientious buddy divers. It appears that the majority are not:

- i 69% checked their buddy occasionally, not continuously
- ii 86% stayed within vision but not within touching distance
- iii 62% sometimes exited separately, not always together
- iv 58% waited for their buddy's signal, instead of checking the buddy's air supply during the dive.

### Skills

Of our experienced divers 56% were able to breath-hold to 9 m or deeper. This ability to breath-hold dive may have a significant affect on a diver's ability to survive in an emergency since it develops confidence in surviving zero, or reduced, external air supply for a minute or so. Few recently trained divers have any breath-hold diving ability since breath-hold diving has become neglected in diving certification courses.

Buddy and octopus breathing had been practiced in the past year by only 40% and 46% respectively and these low practice levels support reports that in a real emergency buddy breathing and octopus breathing often fail. This is even more reason to encourage an independent survival technique. For those that actually had to make an emergency ascent, the emergency swimming ascent is three times as popular as any other technique. Experienced divers are mostly able, sometimes to virtuoso levels, to perform the diving skills that they regularly use while diving e.g. buoyancy control, breath control, manoeuvring, navigating etc., but it seems likely that other skills, that are not often used, are forgotten. This does have implications. Diving instruction should emphasise self-rescue skills because divers cannot rely on any one else to rescue them.

### Photography

Three quarters of the divers were underwater photographers. This is a bit depressing since it is going to be even harder than I thought for me to sell my pictures.

### Opinion

Inexperience has been reported to be one of the factors in diving accidents, yet experienced divers (who are safer) do not dive in the same way that novice divers are taught to dive. Possibly some of the problems arise because divers with insufficient skill and knowledge try to emulate very experienced divers.

Instead of limiting experienced divers I would favour far more attention being given to a probationary period for

recently certified divers, until at least 50 dives are logged. After 50 dives in varying conditions a diver may be considered properly certified and after a further 50 logged dives could be classed as experienced. At this stage logging dives would then become voluntary. I would put a time limit of two years for completing the 50 dives. This would mean that the probationary certification lapses if the 50 dives are not completed on time and retraining becomes necessary. This probationary period would emphasise the vital importance of experience for safe scuba diving.

*The above first appeared in the Telita Newsletter and is reprinted with permission.*

*Bob Halstead's address is Telita Cruises, P.O.Box 303, Alotau, Papua New Guinea.*

## REGULATORS V THE MACHINE

In August 1989 DIVER carried the results obtained when 40 regulators, commonly available in Britain, were subjected to performance tests on a breathing machine. As was explained at that time, the type of test that was used formed the basis of an intended European standard.

Response from divers to the results of the survey were very positive and enthusiastic. Response from the regulator manufacturers/distributors was coloured by whether or not their particular regulators had fared well in the tests!

DIVER has now repeated the survey on a comprehensive range of regulators currently on offer, no fewer than 52 models.

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### Key to table on opposite page

A = Adjustable; Adp = Adaptor; A/B = Adjustable Venturi; B = Balanced; B/D = Balanced Diaphragm; B/P = Balanced Piston; C/W = Cold Water design; D = Diaphragm; E/S = Environmentally Sealed; I/C = Integral Cover/Purge; I/P = Integral Purge; L/P Tur = Low Pressure Turret; P = Piston; S = Swivel; S/C = Swivel Connector; S/D = Side Diaphragm; Se = Servo; T/a = Turbo Assist; T/C = Teflon Coatings; VAD = Venturi Assisted Design.

\* As a result of these tests, Oceanic have discovered a quality control problem which has resulted in some Omega regulators failing to reach the figures shown. Oceanic will be making a public announcement shortly.

Make/Model	1st Stage	WP (max)	HP Ports	LP Ports	Features	2nd Stage	Features	Index (m)
Apeks Manta 500	B/P	310 bar	2	4	L/P Tur	Plastic	S/D	33
Apeks Reflex	B/P	310 bar	1	4	L/P Tur	Plastic	A	50+
Apeks Reflex T30	B/P	310 bar	2	4	L/P Tur	Plastic	A	50+
Apeks Reflex T50	B/P	310 bar	2	4	L/P Tur	Plastic	B; A	50+
Beuchat VS111	B/P	280 bar	2	4	L/P Tur	Plastic		27
Cressi-Sub F3	P	300 bar	1	3		Plastic		29
Cressi-Sub FX Pro	B/P	300 bar	2	4		Plastic		36
Dacor 360 XP	B/D	300 bar	1	4	T/A	Plastic	B	50+
Dacor 360 XLS	B/D	300 bar	1	4	T/A	Plastic	B	50+
Dacor 460 XP	B/Pph	300 bar	1	4		Plastic	B	50+
Dacor XLS	B/Pph	300 bar	1	4		Plastic	B	50+
Dacor 960 XLS	B/D	300 bar	2	4	T/A;L/P Tur	Plastic	B	50+
Mares MR10	D	300 bar	1	4		Plastic	I/P	50+
Mares Beta	B/D	300 bar	1	4		Plastic		50+
Mares MR12	B/D	300 bar	1	4		Metal	VAD	50+
Mares Navy	B/D	300 bar	1	4		Plastic	VAD	50+
Oceanic Alpha P	B/P	5500 psi	2	4	S	Plastic	B	50+
Oceanic Alpha D	B/D	5500 psi	2	4		Plastic	B	50
Oceanic Delta P	B/P	5500 psi	2	4	S	Plastic	B	50+
Oceanic Delta D	B/D	5500 psi	2	4		Plastic	B	50+
Oceanic Omega P	B/P	5500 psi	2	4	S	Plastic	Se; S/D	50*
Oceanic Omega D	B/D	5500 psi	2	4		Plastic	Se; S/D	44*
Poseidon Cyklon 300	D	300 bar	1	4	DIN + Adp	Plastic	S/D	50
Poseidon Cyklon 500	B/P	300 bar	1	4	DIN + Adp	Plastic	S/D	50
Poseidon Oceanair	D	300 bar	1	4	DIN + Adp	Plastic	S/D; Se	50
Poseidon Jestream	B/P	300 bar	1	4	DIN + Adp	Plastic	S/D; Se	50+
SCE Navigator Club	P	220 bar	1	3		Plastic		29
SCE Navigator AZ1	B/D	280 bar	1	3		Plastic		32
Scubapro R190/Mk2	P	200 or 300 bar	1	3		Plastic	AN	50+
Scubapro R190/Mk X	B/P	200 or 300 bar	2	5	L/P Tur	Plastic	AN	50+
Scubrpro M5	B/P	200 or 300 bar	2	5	L/P Tur	Plastic	A	50+
Scubapro G250	B/P	200 or 300 bar	2	5	L/P Tur	Plastic	B; A	50+
Scubapro Mk X	B/P	200 or 300 bar	2	5	L/P Tur	Plastic	B; A	50+
Scubapro D350	B/P	200 or 300 bar	2	5	L/P Tur	Plastic	Se	50+
Sherwood Brut	P	300 bar	1	3	A/B	Plastic		41
Sherwood Magnum	B/P	300 bar	1	4	A/B	Plastic	B	43
Sherwood Blizzard	B/P	300 bar	1	5	A/B	Plastic	B; T/C	50+
Sherwood Oasis 2	B/P	300 bar	1	5	A/B	Plastic	B	50+
Sherwood Ultima	B/P	300 bar	1	5	A/B	Plastic	Se; S/D	50+
Sherwood Maximus	B/P	300 bar	1	5	A/B	Plastic	S/C; A	46
Spiro Club	P	3500 psi	1	4		Plastic		26
Spiro Aqua Lung	B/P	3500 psi	2	4	L/P Tur	Plastic		50+
Spiro Supra	B/D	3500 psi	2	4		Plastic		50+
Spiro Supra Arctic	B/D	3500 psi	2	4	E/S	Plastic	C/W	50+
Tekna 660RX	B/P	3500 psi	2	4	L/P Tur	Plastic	A	37
US Divers Conshelf21	B/D	3500 psi	1	4		Plastic		50
US Divers Select	B/D	3500 psi	1	4		Plastic	I/C	50+
US Divers Conshelf SE2	B/D	3500 psi	1	4		Plastic		50
US Divers Conshelf SE2 S	B/D	3500 psi	1	4	E/S	Plastic	T/C	50
US Divers Conshelf XIV	B/D	3500 psi	1	4		Metal		50+
US Divers Conshelf XIV S	B/D	3500 psi	1	4	E/S	Metal	T/C	50+
US Divers Micra	B/D	3500 psi	1	4		Plastic	A	50+

*Not all of the above regulators are available in Australia. The key to this table is opposite*

To remind readers just what the regulator performance test entails, each regulator is attached to a breathing machine, immersed in water in the normal breathing position then "breathed" at a rate corresponding to moderately hard work, 6.5 litres per minute (25 breathes per minute each with a tidal volume of 2.5 litres). Once the breathing cycle has settled down, the water-filled chamber is subjected to pressure, to stimulate depth, and the effort of inhalation, exhalation and the total work of breathing, are accurately measured.

The criteria for an acceptable performance is that the total work of breathing must be less than 3 Joules per litre, and the maximum inhalation or exhalation resistance must be less than 2.5 kiloPascals (25 cm of water). The performance index is the depth, in metres, at which one of the foregoing criteria is exceeded. The limit for any regulator is 50, because tests were not carried out to greater depths than 50 m. However, if a regulator had "plenty of spare" at 50 m, it was given a score of 50+ in the results.

It should be pointed out that most of the regulators that did not make 50 m in the 1989 test actually failed on inhalation resistance not the total work of breathing. The latest results show a similar pattern.

As with the 1989 test, all the breathing performance assessments were carried out at Ansti Test Systems, Portsmouth, so they are truly comparable with those of 1989.

In considering the results, one thing that is immediately noticeable is just how many of the regulator manufacturers have improved the performance of their regulators since the 1989 tests. It is almost as though they needed a bench-mark against which to compare their regulators and towards which they could aim. It is probable that many of them did not realise that regulator performance tests were easily available to the sport diver, since all previous performance testing had been carried out on behalf of organizations such as the Royal Navy and the US Navy. Those manufacturers who did take on board the challenge, and who successfully met that challenge in than three years, deserve great credit.

There have been suggestions that regulators are now being designed to "beat machines" in that it is only by using a breathing machine that high breathing rates can be properly assessed and that a machine is different from a diver. I certainly don't fancy working hard at 50 m to see if a regulator can supply me with enough air! I much prefer to let a machine do the assessment and calculations for me.

I have also been told that 62.5 litres per minute breathing rate is impossibly high. However, breathing rates in excess of 90 litres per minute have been measured on divers working hard at depth! Agreed, they were professional divers being paid for their work, but is commonly believed that a sport diver who gets into difficulties at depth

will pull harder and harder on a regulator and will certainly be breathing at somewhere near the 62.5 litres per minute rate that has been set for the tests.

It should also be remembered that these criteria have already been accepted by a number of agencies, and that they will form part of a European Standard for Underwater Breathing Apparatus. Additionally, the BS-AC does get Incident Reports of divers who have experienced breathing difficulties at depth and of attempts at buddy-breathing that went wrong. In many cases, back at the surface, no fault can be found in the air supply or the regulator. One is forced to wonder whether or not some of these difficulties might have been compounded by the diver demanding more air from the the regulator than it was able to provide!

Another thing that is evident from the latest survey is just how few regulators now have second stages made from once-prevalent metal - chromium -plated brass! It would appear that high-impact mouldable polymers are the norm for today's regulators.

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## GLEANINGS FROM MEDICAL JOURNALS

The following articles have come to the notice of the editorial staff and these notes are printed to bring them to the attention of members of SPUMS. They are listed under various headings of interest to divers and hyperbaric medicine personnel. Any reader who comes across an interesting article is requested to forward the reference to the Journal for inclusion in this column.

### CARBON MONOXIDE POISONING

#### Carbon monoxide poisoning

Gorman DF and Runciman WB. *Anaes Intens Care* 1991; 19:506-511

A literature review article concluding that HBO is the treatment of choice for CO poisoning.

#### Carbon monoxide poisoning

Worthley LIG, Holt AW, Bersten AD and Vedig AE. *Anaes Intens Care* 1992; 20:257-258

A letter drawing attention to papers not in favour of HBO treatment and lack of controlled trials.

#### Carbon monoxide poisoning

Gorman DF and Runciman WB. *Anaes Intens Care* 1992; 20:258-259

Letter in reply explaining why some of the quoted papers should be dismissed and drawing attention to the authors' intention to carry out a prospective randomised trial when they have sufficient new basic knowledge to ethically carry one out. They believe that all patients with early or late neurological sequelae of CO poisoning should have HBO.

#### A longitudinal study of 100 consecutive admissions for carbon monoxide poisoning to the Royal Adelaide Hospital.

Gorman DF, Clayton D, Gilligan JE and Webb RK. *Anaes Intens Care* 1992; 20:311-316

The frequency of neuropsychiatric sequelae in patients who received oxygen at atmospheric pressure was higher (63%) than in those who received one hyperbaric treatment (43%) at discharge and at follow up at one month. Those who had two or more hyperbaric treatments had fewer sequelae, 13% on discharge and 18% on follow up. Delayed hyperbaric treatment was associated with more sequelae.

### BOX JELLYFISH

#### Management of a major of a major box jellyfish (*Chironex fleckeri*) sting

##### Lessons from the first minutes and hours

Beadnell CE<sup>1</sup>, Rider TA<sup>1</sup>, Williamson JA<sup>2</sup> and Fenner PJ<sup>3</sup>. *Med J Aust* 1992; 156: 655-658.

*Objective:* To report the management of a serious box jellyfish (*Chironex fleckeri*) envenomation from the first minutes of bystander first aid and treatment by ambulance personnel to subsequent treatment in hospital.

*Clinical features:* A 14 year old girl sustained a serious *Chironex fleckeri* sting. There was no loss of consciousness, but the patient suffered severe pain, myocardial irritability, acute pulmonary oedema and mild systemic hypotension, due to the direct toxic effects of the venom. Thirst was a dominant symptom.

*Intervention and outcome:* Management involved rapid bystander action and call for ambulance assistance; and early intervention with oxygen/nitrous oxide administration, compression bandaging, antivenom administration and electrocardiographic monitoring at the site by ambulance personnel. Echocardiography in hospital three hours after the sting showed a normal myocardium. In hospital management resulted in recovery. Nocturnal itching of the sting persisted for six weeks.

*Conclusions:* (i) Vinegar dousing may irritate freshly stung skin, but as a nematocyst inhibitor vinegar remains an essential part of the first aid treatment for cubozoan jellyfish stings. (ii) Compression/immobilisation bandaging was not associated with long-term harm to the sting area. (iii) The pain of an intramuscular antivenom injection may not be felt by a chirodroid sting victim, so safe injection protocols must be strictly observed. (iv) Ambulance services in other States where there is a risk of box jellyfish (*Chironex fleckeri* or *Chiropsalmus quadrigatus*) stings should be similarly trained and equipped to deal with serious jellyfish envenomations.

- 1 Cairns Centre, Queensland Ambulance Transport Brigade, Cairns, Queensland, 4870, Australia.
- 2 Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, Australia.
- 3 Medical Advisory Committee, Queensland State Centre Inc., Surf Life Saving Australia Limited, PO Box 36, Newstead, Queensland, 4006, Australia.

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## DIVING SAFETY

### Scuba diving safety

Wilks J *Med J Aust* 1992; 156: 580

Letter about diving related incidents observed by scuba instructors.

### Signals and procedures in the recreational diving workplace.

Wilks J. *J Occup Health Safety -Aust NZ* 1992; 8(4): 323-330

#### Summary

Non-verbal communication is an essential safety element in many workplaces, though the effectiveness of signs and signals to reduce accidents is seldom examined. In the present study, recreational scuba divers were asked to identify 16 non-verbal signals commonly used in commercial dive settings, and also to choose the appropriate safety procedure to follow in the event of separation from their dive partner underwater. Results showed a high level of recognition and understanding of the signals and procedures. Total safety scores were best predicted by the frequency of diving in the previous 12 months. There was an inverse relationship for length of time since certification with newer divers obtaining better safety scores than divers who had been certified for longer periods. Implications of the findings for improving safety in the recreational diving industry are discussed.

## ABSTRACTS FROM THE 1991 ANNUAL SCIENTIFIC MEETING OF THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY

The address of the Undersea and Hyperbaric Medical Society is 9650 Rockville Pike, Bethesda, Maryland 20814, U.S.A.

### CARBON MONOXIDE POISONING

#### Warehouse worker's headache: acute CO poisoning from propane fueled forklifts.

Fawcett TA, Moon RE, Fracica PJ, Mebane GY, Theil DR, Shelton DL and Piantadosi CA. *Undersea Biomed Res* 1991; 17(Supp); 84.

Carbon monoxide (CO) poisoning is a frequent complication of using internal combustion engines in an enclosed space. Although gasoline powered equipment is a common offender, recent reports have shown that engines powered by liquefied natural gas or propane can cause

significant poisoning especially when used in a refrigerated environment such as ice rinks (*Am J Public Health* 1990; May 80(5): 594-8). We reviewed over 220 cases of acute CO poisoning treated at the Duke Medical Center F.F. Hall Hyperbaric Center and report on 17 patients whose poisoning occurred from the use of propane powered forklifts. These patients were treated with hyperbaric oxygen (HBO) based on neurologic symptoms and/or measured carboxyhemoglobin (COHb) level >25%. All patients in this series presented with neurologic symptoms, primarily headache, nausea and lightheadedness. There were two cases of syncope and one case of unconsciousness. The duration of exposure ranged from a 2 to 8 hours with a mean and median of 5.0 hours. Initial COHb level at the time of evaluation ranged from 4.0% to 28% with a mean of 22% and a median of 23%. The delay before treatment, measured from the end of the CO exposure to HBO ranged from 1 to 12 hours with a mean of 4.9 hours and a median of 3 hours. All patients received HBO consisting of 100% oxygen at 2.45 ATA (14.4 m, 48 fsw) for 90 minutes without air breaks. Following HBO, all symptoms resolved without sequelae. These 17 cases occurred in 8 separate episodes where a forklift was operating inside a closed warehouse or garage. Of the 9 episodes, 6(15 of 17 cases) occurred during winter months with the remaining 2 episodes occurring during midsummer in air-conditioned environments. These cases highlight a potentially hazardous complication of the common practice of using propane powered forklifts in enclosed areas. Patients who present with unexplained headache and constitutional symptoms related to working in warehouses warrant routine measurement of COHb level and appropriate treatment with oxygen if indicated.

From the Hyperbaric Center, Duke University Medical Center, PO Box 3823, Durham, North Carolina 27710, U.S.A.

#### Chronic carbon monoxide - a new clinical syndrome.

Youngberg JT, DeFazio A and Myers RAM. *Undersea Biomed Res* 1991; 17(Supp): 78-79.

In the past ten years 8 patients with chronic, untreated carbon monoxide (CO) have presented to and been treated at the MIEMSS. The exposure to CO in women was in the home whereas in the men at the workplace. CO production was from exhaust fumes from fork lift trucks, automobiles in repair shops; invented research hood; clothes dryer and water heater; open cans of paint emitting methylene chloride. The CO exposure was intermittent and over a time frame of from 3 weeks to 3 years.

Common symptoms included weight changes, dizzy spells, trembling hands, memory changes, sleep pattern alterations, headaches, emotional lability and irritability. Neuropsychologist testing (WAIS-R, Aphasia Screening, Trails A and B, finger oscillation) all from the Haistead

Retain battery, logical reasoning and visual reproduction from the Weschler memory scale, and the MMPI one or two.

Base line tests were undertaken followed by daily hyperbaric oxygen therapy at 2 ATA for 90 oxygen minutes for 14 treatments and then a re-assessment of their neuropsychometric responses. If improvement was noted a second and even third series of hyperbaric oxygen treatments were undertaken until no further improvement was noted or the patient's neuro-psychometric profile was normal. Improvement in functional, cognitive and psychiatric capacities were noted.

This clinical syndrome is often overlooked because of its obscure symptomatology, wide range of presentation and physician general lack of awareness to the problem.

From Maryland Institute of Emergency Medical Services Systems, 22 S Greene Street, Baltimore, Maryland 21201, U.S.A.

#### **Assessment of cognitive functioning in carbon monoxide poisoned persons.**

Barger SD, Weaver KL and Hopkins RO. *Undersea Biomed Res* 1991; 18 (Supp): 33

Carbon monoxide poisoning has been shown to cause muscular weakness, confusion, impairment of cognitive abilities, and with severe exposure, unconsciousness and death. Cognitive functioning in carbon monoxide poisoned person was assessed using a battery of psychometric tests adapted from previous carbon monoxide research (Myers and Messier, 1987). The battery consisted of the following tests: Orientation to person, place, time and event; selected subtests from the WAIS-R which included Digit Symbol, Block Design, and Digit Spans forward and backward; Trail A and B; and Story Recall. These tests were used to determine if treatment with hyperbaric oxygen (HBO) was warranted. Treatment was given to individuals who were impaired on these measures. The patient did not have to be abnormal on all tests to be recommended for treatment. The same battery was administered post-treatment to determine if further hyperbaric oxygen treatments were necessary. Twenty-eight CO victims had psychometric screening pre- and post HBO treatment. The most sensitive measures of cognitive dysfunction were Digit Symbol, Block Design and Trails. Digit Symbol ( $t(64)=2.88$ ,  $p<(0.01)$ ), Block Design ( $t(65)=3.16$ ,  $p<(0.01)$ ), and Trails ( $t(65)=5.85$ ,  $p<(0.001)$ ) were all significantly different from a control group (Myers and Messier, 1987). Orientation was not different from the control group ( $t(68)=1.79$ , ns). Simple orientation does not reveal cognitive dysfunction, whereas the other three subtests are more sensitive. We recommended formal psychometric assessment including the Digit Symbol, Block Design and Trails tests for all patients with carbon monoxide poisoning. We further suggest that

patients impaired on these measure be referred for HBO treatment.

From the Hyperbaric Division, Pulmonary Medicine, LDS Hospital, Salt Lake City, Utah and Department of Psychology, The University of Utah, Salt Lake City, Utah.

#### **Immediate and long term neurological sequelae of carbon monoxide intoxication.**

Skeen MB, Massey EW, Moon RE, Shelton DL, Fawcett TA and Piantadosi CA. *Undersea Biomed Res* 1991; 18 (Supp): 36.

A retrospective review of medical records was performed to analyze the frequency and characteristics of neurologic sequelae in patients with acute carbon monoxide intoxication.

187 cases were reviewed ranging in age from 7 months to 99 years with a mean age of 37.2 years. The male:female ratio was 3:1. All patients were treated with hyperbaric oxygen at 2.5 ATA for 90 minutes, 1 to 120 hours after the end of the exposure. Of this group, 103 patients had experienced loss of consciousness prior to arrival at the chamber with 76 of these evidencing no neurological deficits at the time of discharge. Twenty eight patients were determined to have neurologic deficits at the time of discharge. Twenty seven of the patients with neurologic deficits had suffered loss of consciousness prior to arrival at the chamber. One patient was lethargic and confused and became unconscious during treatment when he experienced recurrent generalized seizures while in the chamber. In comparing patients with and without neurologic deficits there was no significant difference in mean COHb levels ( $27.5\pm 17.4$ , and  $27.2\pm 13.7$  respectively). Likewise there was no significant difference in mean duration of exposure reported (8.3 vs. 8.1 hrs.). Of interest those patients who developed deficits had a mean interval from exposure to treatment significantly longer than those without deficits (13.7 vs 6.5 hrs.). Current investigations are underway to evaluate late sequelae or residual symptoms in all patients.

From the Hyperbaric Medicine Laboratory, Duke University Medical Center, Durham North Carolina, U.S.A.

#### **Lactate levels as a measure of severity of carbon monoxide poisoning.**

Britten JS, Baker TL and Myers RAM. *Undersea Biomed Res* 1991; 17(Supp): 79.

A problem exists in quantifying the severity of poisoning by carbon monoxide using laboratory measures. If clinical criteria are used to stratify victims of carbon monoxide poisoning into mild, moderate and severe cases, carbon monoxide blood levels are poorly correlated with the clinical stratification. Blood lactate levels have been sug-

gested as a better indicator of the severity of carbon monoxide poisoning. We have examined retrospectively the data collected from 141 cases of carbon monoxide poisoning admitted to the Institute over the period 1981 to 1986. Linear regression analysis of the blood lactate versus percentage carboxyhemoglobin levels on admission disclosed a weak correlation ( $r=0.45$ ) with wide scatter of data points. Blood lactate was much better correlated to blood pH ( $r=0.69$ ) with an obvious linearity in the data point plot. We have established earlier (J Crit Care Med 1989; (17): 139) that blood pH is poorly correlated with the severity of carbon monoxide poisoning. We suspect that lactate levels would have a similar poor correlation. Direct comparison of lactate levels to clinical symptomatology is under way.

From Maryland Institute for Emergency Medical Services Systems, 22 S. Greene Street, Baltimore, Maryland 21201, U.S.A.

#### **Does late repetitive hyperbaric oxygen improve delayed neurologic sequelae associated with carbon monoxide poisoning?**

Hopkins RO and Weaver LK. *Undersea Biomed Res* 1991; 18 (Supp): 34.

Delayed neurologic and psychiatric sequelae resulting from acute carbon monoxide (CO) poisoning are well known. The medical literature supports the use of late administration of hyperbaric oxygen for delayed neurologic impairments subsequent to carbon monoxide poisoning. After exposure to carbon monoxide there is a period of apparent recovery of approximately 3-40 days, followed by subsequent neurologic and cognitive deterioration. In a three year retrospective study of 86 carbon monoxide victims, five developed late sequelae (5.8%), and two victims had continued neurologic deterioration (2.2%). Carboxyhemoglobin ranged from 22 to 52 with a mean of 35.3. As expected, carboxy-hemoglobin was not a significant risk factor in the development of the neurologic sequelae. The five patients with delayed sequelae were treated with hyperbaric oxygen an average of 5.8 times, had no persistent neurologic or cognitive impairments and then developed delayed neurologic and cognitive deficits. The subjects were followed with psychometric testing, neurologic evaluations, and Magnetic Resonance Imaging (MRI). These individuals showed impairment in short term memory, attention, concentration, and impaired spatial ability. Two subjects experienced cortical blindness. Lesions observed on MRI scans for all seven subjects included one with a left internal capsule and two with bilateral lesions of the globus pallidus, one with bilateral caudate and left putamen lesions, and three scans were normal. These subjects with delayed neurologic sequelae subsequently received an average of 6.0 additional hyperbaric oxygen treatments, with no improvement in the cognitive or neurological deficits. Multiple hyperbaric oxygen treatments do not prevent the development of delayed neurologic and cognitive sequelae re-

sulting from carbon monoxide poisoning. However, the incidence of delayed sequelae may be reduced with hyperbaric oxygen, but pending a formal randomized controlled clinical trial, the role of multiple hyperbaric treatments in the prevention of the delayed sequelae can not be definitively answered. Contrary to previous literature, additional hyperbaric oxygen treatments after the development of delayed sequelae do not appear to improve these deficits.

From the Hyperbaric Division, Pulmonary Medicine, LDS Hospital, Salt Lake City, Utah and Department of Psychology, The University of Utah, Salt Lake City, Utah.

### **DIVING PHYSIOLOGY**

#### **Evidence of altered liver function in a group of amateur scuba divers following a diving holiday.**

Doran GR. *Undersea Biomed Res* 1991; 18 (Supp): 46-47.

In the light of previous findings of altered liver function among professional deep saturation divers a range of biochemical blood tests were performed on 9 experienced amateur scuba divers (8 male; aged 30-60 years) before and immediately after a 12 day diving holiday to Eilat, Israel in March 1989. The average total number of dives undertaken was 18; to depths ranging from 6 to 50 msw, with an overall average depth of 20 msw. With the exception of one male all the divers continued to consume alcoholic drinks while on holiday. Significant post holiday climbs were detected in their plasma activities of isocitrate dehydrogenase ( $P<0.05$ ), alkaline phosphatase ( $P<0.01$ ) and acid phosphatase (0.05). The levels of the plasma glycoproteins thyroxine-binding globulin and fibronectin also rose ( $P<0.01$ ), together with the complement C<sub>3</sub> fraction ( $P<0.02$ ). No significant post-holiday changes were identifiable overall in the activities of the transaminases (AST,ALT),  $\gamma$  glutamyl transferase (GT) or cholinesterase, nor in their plasma bilirubin or  $\alpha_1$  acid glycoprotein levels. While among the male divers the changes were generally moderate, those evident in the only woman in the group were pathologically severe (including raised AST, ALT and  $\gamma$ GT), such as would characterise a mild hepatitis. However, subsequent serology failed to identify any common infective aetiology. The fact that, apart from the case of the woman diver, no elevation of  $\gamma$ GT was evident, coupled with the fact that changes were equally apparent in the male who abstained, argues against these disturbances being attributable to "excessive" alcohol consumption. Overall the results confirm that some significant alterations in divers' liver function tests may be brought about by repetitive shallow diving and that exposure to very high ambient pressures (>6 ATA) is not a prerequisite. The possibility that women may be more severely affected than men requires further careful review.

From the Department of Chemical Pathology, Charing Cross and Westminster Medical School (University of London), St Dunstons Road, London W6 8RP, UK.

#### **Circulatory response to deep breath-hold diving.**

Ferrigno M, Lundgren C, Cerretelli P, Ferretti G, Warkander D and Costa M. *Undersea Biomed Res* 1991; 17(Supp): 87-88.

Three elite breath-hold divers were studied with regard to circulatory response to submersed (25°C) dives to between 40 and 50 m in a hyperbaric chamber. Descents and ascents were at 1 m/s, time at max depth 15 s for a total duration of 95 to 105 s. Heart rate (HR) and stroke volume were recorded by ECG and impedance cardiography, calf perfusion by strain gauge plethysmography and arterial blood pressure was obtained invasively (two subjects). Heart rates during resting conditions were typically 60-70 bpm and immediately pre-dive 110-130 bpm. During the dives mean HR fell by about 30% (from immediate pre-dive level) in diver A, by 50% in B and by about 80% in C. All three subjects showed marked sinus suppression and varying activity by junctional and ventricular pacemakers and variability in HR with rapid beat to beat changes from as low as 8.5 bpm to over 100 bpm. Thus, a very different picture from the classical smooth diving bradycardia was observed. Similarly, there was considerable variability in cardiac output with only a small reduction in subject A, a fall from about 7 L/min to 6 L/min in B and from 6 L/min to 2 L/min in C. At maximum depth all three subjects showed a pronounced peripheral vasoconstriction resulting in cessation of blood flow and even a reduction of calf circumference indicative of a forceful capacitance vessel constriction. In subject A, the blood pressure was 250/145 mm Hg at max depth vs a control value of 150/80 mm Hg and in subject C, the values were 180/100 vs 155/70 respectively. These values at max depth, coinciding with low cardiac outputs, attest to the vasoconstriction which is thought to be the primary factor in the cardiovascular diving response.

From the Department of Anesthesiology, University of Miami, Florida 3301; The Center for Research in Special Environments, Department of Physiology, School of Medicine, State University of New York, Buffalo, New York 14214; Department of Physiology, Centre Med. Universitaire, Geneva, Switzerland; Section of Physiology, CNR, Milan, Italy.

#### **Towards a molecular mechanism for the action of pressure on the central nervous system.**

Daniels S, Price DJ, Shelton CJ and Smith EB. *Undersea Biomed Res* 1991; 17(Supp): 57-58.

The mechanism of action of high pressure in inducing hyper-excitability in the central nervous system has, like that of general anaesthesia (and in a diving context inert gas

narcosis), been attributed to a non-specific physico-chemical interaction frequently associated with a disturbance to the cellular membrane. However, recent evidence has indicated that the action of pressure is more specific; with the site of action located sub-cortically and involving either post-synaptic inhibition, mediated by the neurotransmitter glycine, or enhanced excitation, mediated by the glutamate transmission system. Pharmacological evidence appears unable to distinguish between these possibilities. Accordingly we have begun to utilise the *Xenopus* oocyte as a model system in which mammalian neurotransmitter receptor proteins can be expressed using the appropriate messenger-RNA. The effect of pressure on specific mammalian neurotransmitter receptor proteins can then be investigated using whole cell voltage clamp techniques. We have constructed an apparatus to enable these investigations to be conducted both at atmospheric pressure and under conditions of increased ambient pressure using helium as the pressure transmitting medium. We have found that the glutamate transmitter analogue, Kainate, which is responsible for fast synaptic transmission, is unaffected by pressure (although it is inhibited by general anaesthetics) whereas the glycine receptor, responsible for sub-cortical post-synaptic inhibition, is very sensitive to pressure, showing a threefold increase in  $EC_{50}$  at pressures up to 100 bar. Studies are under way to investigate the role of the pre-synaptic inhibitory transmitter GABA and the excitatory transmitter NMDA. The studies will reveal whether the action of pressure is mediated exclusively at one particular receptor protein or whether a number of different transmitter systems are involved and identify a model system in which the details of the effect of pressure on channel function at a molecular level can be elucidated.

From the Oxford Hyperbaric Group, Physical Chemistry Laboratory, South Parks Road, Oxford OX1, 3QZ, U.K.

### **DECOMPRESSION ILLNESSES**

#### **Use of the neurodermatome chart in the treatment of decompression sickness.**

Smookler M and Cianci P. *Undersea Biomed Res* 1991; 17(Supp): 76.

The use of the standard neurodermatome chart is utilized in many offshore operations. Surprisingly, few medical texts recommend its use in the evaluation of decompression illness. It would seem appropriate to recommend the use of this standardized technique for evaluation of decompression illness in order to facilitate accurate diagnosis and results of therapy. Furthermore, communications would be greatly improved in those instances where a physician is not readily available and in follow-up communications physician to physician. We ask the question, would this not be a suitable area for standardization of evaluation of decompression sickness and air embolization?

A standard overlay and/or a metal-etched version would be part of each chamber operation and perhaps should be further utilized in various diving texts.

Address for correspondence 33036 Regents Boulevard, Union City, California 94587, U.S.A.

#### **Time for saturation in humans as defined by venous gas bubbles.**

Eckenhoff RG and Olstad CS. *Undersea Biomed Res* 1991; 17(Supp): 70-71.

The time required for human tissues to reach equilibrium with respect to inert gas exchange is not clear, and has been suggested to be as long as 48 hours or more. We examined the time required for saturation with nitrogen in humans, using the evolution of venous gas bubbles as a criteria of tissue inert gas load after decompression. Although indirect, we believe that this criteria is relevant to decompression practice, because of the etiologic role of bubbles in decompression sickness syndromes. 128 healthy male subjects were exposed to air at 20.5 fsw; 32 subjects for each of 4 different durations: 3, 6, 12 and 48 hours. After direct decompression (1-2 min.), subjects were monitored with doppler ultrasound over the precordium and subclavian veins at regular intervals for a period of 24 hours, and scored as previously reported (JAP 69: 914). Results indicate that the incidence of detectable bubbles reaches a plateau (~85%) with less than a 6 hour exposure, but that the magnitude of bubbles (both bubble score and duration) continue to increase to exposures of about 12 hours. No significant increase in either score or duration could be detected after the 12 hour exposure. This suggests that saturation, as indicated by venous bubbles, takes less time than previously thought. Further, the data are consistent the notion that a short half-time tissue (~ 120 min.) is responsible for venous bubble generation, and is "fed" by longer half-time tissues.

From the University of Pennsylvania Medical Center, Philadelphia, Pennsylvania 19104 and the Marine Research Development Foundation, Key Largo, Florida, U.S.A.

#### **Doppler analysis of sport diver profiles.**

Dunford RG, Wachholz C, Fabus S, Huggins C, Mitchell P and Bennett PB. *Undersea Biomed Res* 1991; 17(Supp): 62.

Doppler scores for 81 male and 41 female divers were obtained following 640 recreational exposures during 14 DAN sponsored dive trips to warm water locations. Age (X - 39 years), depth (X - 72 fsw) and ratio of dives were similar for both sexes. Doppler measurement was carried out using the 0-4 Spencer scale at the precordial site with an IAPM Doppler 30 minutes post dive. A Doppler score >0 was observed in 46 divers following 89 exposures (145 scores were eliminated as unusable). A Doppler score = 3

occurred following 21 exposures but no score = 4 or case of decompression sickness were observed. Since the number of positive scores was small the data was dichotomized for Doppler score (+/-), age (<40 vs ≥40) and depth (<80 vs ≥80 fsw). A repeated measure logistic regression model estimated relative odds for risk of + score. Males were more likely to produce + scores at fsw ≥80 for both age groups (p<0.001). Females showed an increased risk for positive score only when age ≥40 and fsw ≥80 (p=0.001). Dichotomized age at 35 and 45 years or depth at 70 and 90 fsw showed similar patterns. The first dive day showed less risk of + score when compared to the remaining days (p=0.04). Neither repetitive diving nor the use of a dive computer increased the risk of a + score.

From the Hyperbaric Department, Virginia Mason Medical Center, Seattle, Washington 90101 and Divers Alert Network, Duke University Medical Center, Durham, North Carolina, U.S.A.

#### **Ultrasonic doppler measurement of sport divers at altitude.**

Wachholz CJ, Dunford R and Bennett PB. *Undersea Biomed Res* 1991; 18 (Supp): 23-24.

The Diver's Alert Network (DAN) has since 1985 used ultrasonic Doppler to measure sport divers making unsupervised dives in open water. Most of these measurements have taken place in warm, tropical ocean waters at sea level. In 1990, two groups of sport divers were measured at altitude following sport diving. The first group of 15 divers were measured while diving off the Caribbean Island of Saba, Netherlands Antilles. Measurements of 12 subjects were conducted both at sea level and at an elevation of 1,200 feet, the altitude of the divers' hotel. Excursions to 1200 feet occurred each day following the last dive of the day, between 40-80 minutes after exiting the water from the last dive. Divers made three dives per day on most days. One Doppler measurement was taken between 30-40 minutes following each dive. Divers were measured immediately prior and immediately following the excursion to 1,200 feet. Of the 15 divers 10 (75%) had positive bubble scores, all grade one or two (Kisman Masurel Scale). There was no increase in bubble scores following the excursions to altitude for any of the subjects. Because of Saba's close proximity to St. Marten, tourists regularly fly over to dive for the day. For over ten years, following scuba dives, return flights to St. Marten have occurred at altitudes of 1,500 to 4,000 feet. No cases of DCS as a result of these flights have been reported to Divers Alert Network. This information is important in light of the recently issued minimum 12 hour Flying-After-Diving recommendations (Sheffield, et al., UHMS Publ. No.77, 12/1989). Dive resort operations in the Caribbean that relied on one to three day dive package business have complained that the 12-hour guideline is too conservative. Another group that has taken issue with the necessity of a 12-hour delay to altitude are the freshwater divers of the Rocky

Mountain region. A second group of 12 Doppler subjects from Grand Junction, Colorado, elevation 4,843 feet were measured following single day diving at Lake Powell, Utah, elevation 3,736 feet. In this group, 7 of the divers (58%) had bubbles, all grade one or two, except one individual who had a grade three bubble score. Both the Saba and Utah dives were repetitive, with the deepest dive first in all cases. In both groups, the effect of depth (dive #) produced more bubbles than subsequent repetitive dives or from any altitude effect. As the '89 flying-after-diving workshop guidelines were only concerned with excursions on commercial aircraft assuming an 8,000 foot cabin altitude, alternate recommendations for altitude excursions following sport divers to lower altitudes should be considered.

From Divers Alert Network (DAN), Duke University Medical Center, Durham, North Carolina, USA.

**Perilymph fistula, rapid recompression and middle ear barotrauma: effect on guinea pig auditory function as measured by electrocochleography.**

Stevens DM, Velasquez JL and Dutka AJ. *Undersea Biomed Res* 1991; 18 (Supp): 18-19

Forceful attempts to equalize middle ear and ambient pressures during a dive may result in rupture of the round window membrane and formation of a perilymph fistula (PLF). The symptoms of PLF may be confused with those of cerebral arterial gas embolism (CAGE), resulting from diving related pulmonary barotrauma. Treatment of CAGE requires recompression; however, some investigators have suggested that re-exposure to changes in pressure might increase damage to the inner ear in cases of PLF. We subjected guinea pigs with surgically created PLF to rapid recompression to test the hypothesis that recompression resulting in middle ear barotrauma (MEBT) causes further damage to the inner ear. Electrocochleography (ECoG) was used as a measure of hearing function. We studied 10 male, albino guinea pigs by performing ECoG prior to and after surgery and following a rapid excursion to an depth equivalent of 165 ft of seawater (6 ATA). On the left side surgery consisted of entering the middle ear via a postauricular transbulbar approach. On the right side the same approach was utilized to identify and surgically remove the round window membrane. The bullae were sealed air tight with a paper patch and methylmethacrylate glue prior to the a rapid excursion at a rate of 11 feet per second. MEBT was verified by direct inspection of the middle ear bullae following the excursion. There was no significant difference in the latency of the waveforms (two-way ANOVA with repeated measures,  $p > 0.05$ ) between the control left ears and fistulized right ears. Log transformation of the amplitude of the summing potential (SP), action potential (AP) and the SP/AP ratio were also significantly different between these groups (ANOVA with repeated measures,  $p > 0.05$ ).

We conclude that there is no difference in the effect

of rapid recompression resulting in MEBT when fistulized ears are compared to non-fistulized ears utilizing ECoG. The results suggest that recompression therapy can be carried out safely in divers who surface with symptoms consistent with both AGE and perilymph fistula.

From the Naval Medical Research Institute, Bethesda, Maryland 20889-5055, U.S.A.

**Chest radiographs can assist in the diagnosis of pulmonary barotrauma.**

Koch GH, Weisbrod GL, Lepawsky M and Muller NL. *Undersea Biomed Res* 1991; 17(Supp): 100-101.

Recent cases of pulmonary barotrauma presenting at our facilities showed distinct chest radiographic findings. These findings were originally described by Kidd as pathognomonic for pulmonary barotrauma. Since his description, there has been little mention of these unique findings in the radiology or diving medicine literature. We therefore reviewed several cases of pulmonary barotrauma to determine the frequency with which these findings are present and if there was a pattern. It is of note that these findings present very early, i.e. immediately after the barotrauma, and disappear within 6 to 24 hours in most cases. The findings are usually unilateral, suggesting a unilateral barotrauma. A similar chest X-ray may be seen in 25% of cases of near-drowning but is usually bilateral. The changes are not usual in decompression sickness cases. The presence of these X-ray findings in a scuba diver should result in rapid referral to a hyperbaric facility and not in treatment for near-drowning. These early X-ray changes are probably due to hemorrhage into the lung tissue where the barotrauma has taken place. These findings can assist the emergency physician to differentiate pulmonary barotrauma from near drowning and decompression sickness. Cases will be presented to illustrate the argument.

From The Toronto Hospital, Toronto Canada and Vancouver General Hospital, Vancouver, Canada.

**Is pulmonary barotrauma a reperfusion injury?**

Koch GH and Lepawsky M. *Undersea Biomed Res* 1991; 17(Supp): 101.

Pulmonary barotrauma can lead to pneumothorax, mediastinal emphysema and arterial gas embolism (AGE). The latter is often fatal and because of that, the pathophysiology of AGE is well documented. We have encountered cases of pulmonary barotrauma with AGE in which the patients have recovered from the AGE with recompression treatment but have gone on the progressive respiratory failure similar to adult respiratory distress syndrome (ARDS). ARDS is described as probably being the result of the free radical evolution and inflammation in the lung. It is likely that massive pulmonary barotrauma can initiate a series of

events like that in ARDS with resultant severe lung tissue, hemorrhage, arachidonic acid cascade and tissue factors activated, reperfusion and free radical formation, lipid peroxidation, leukocytosis and continuing inflammatory damage to the lung. Arguments supporting this pathophysiology and illustrative cases will be presented.

From The Toronto Hospital, Toronto, Canada and Vancouver General Hospital, Vancouver, Canada.

## CLINICAL REPORTS

### **Paradoxical pain during the treatment of dysbaric osteonecrosis with hyperbaric oxygen.**

Youngblood DA and Vega RL. *Undersea Biomed Res* 1991; 17(Supp): 103.

Dysbaric osteonecrosis is an environment hazard associated with changes in ambient pressure. We treated a 35 year old diver with no history of recognized decompression sickness during his 15 year career of intensive scuba diving activity involving 10-15 dives per week to depths between 60 and 200 fsw. Symptoms of fatigue and shoulder pain during ascent and after surfacing gradually increased over a three year period to include joint pain exacerbated by flying at normal airline cabin altitudes. Physical examination revealed diffuse neurological abnormalities, and radiographs showed a suspicious humeral medullary lesion. Early dysbaric osteonecrosis in both shoulders was confirmed by Magnetic Resonance Imaging (MRI), and the patient received HBO in a Sechrist monochamber at ATA for 90 minutes once per day over a 5 month period. MRI after 100 treatments showed incomplete resolution of the shoulder lesions despite considerable clinical improvement. During the initial treatments there was a paradoxical increase in shoulder pain during compression which continued to increase while at the treatment pressure. This paradoxical pain became less intense each succeeding day and disappeared by the twentyfifth treatment. Theories which might explain this paradoxical pain will be presented.

From the Baromedicine Center, Straub Clinic and Hospital, 888 S. King Street, Honolulu, Hawaii, U.S.A.

### **Transthoracic pulmonary needle biopsy associated with cerebral air embolism ameliorated by hyperbaric oxygen.**

Lepawsky M and Hashimoto S. *Undersea Biomed Res* 1991; 17(Supp): 102.

Although rare, cerebral air embolism associated with transthoracic needle biopsy is a known clinical entity. It has a potentially severe prognosis and may be fatal. Treatment with hyperbaric oxygen has repeatedly been reported to be helpful. Rapid institution of hyperbaric oxygen is, usually

stressed as being of great importance. Delayed treatment with hyperbaric oxygen has, nonetheless, been reported to be helpful in a number of cases. We report a case of transthoracic pulmonary needle biopsy associated with cerebral air embolism in which delayed hyperbaric oxygen was of significant benefit. CT scan prior to hyperbaric oxygen showed intracerebral gas. CT scan after hyperbaric oxygen showed no evidence of intracerebral gas. CT scans from the time of diagnosis and after hyperbaric oxygen will be shown.

From the Vancouver General Hospital Hyperbaric Unit and Department of Neurology, 855 W 12th Ave, Vancouver, British Columbia, Canada V5Z 1M9.

## INCIDENT REPORTING

### **Incident reporting. Its application in scuba diving safety.**

Acott CJ. *Undersea Biomed Res* 1991; 18 (Supp): 47-48.

Incident reporting is not a new concept. It was first used successfully in aviation by Flanagan in the 1940's. It is the reporting of error - a study of preventable mishaps.

This on-going study was designed to:

- 1 identify the incidence of preventable errors/mishaps (both human and "pure equipment failure");
- 2 to define corrective strategies for the more commonly identified errors;
- 3 to improve safety in scuba diving.

A diving incident is defined as:

"any error that could, or indeed did, reduce the safety margin for a diver on a particular dive. The error may have been by the diver, diver's buddy or somebody else. It may also be due to equipment failure"

A report form/questionnaire was circulated throughout the Australasian diving community. Divers were encouraged to fill out and return these report forms as soon as possible after they either produced, witnessed or corrected an incident during a dive. Data obtained were analysed along the now well established lines developed by pre-existing aviation and anaesthesia incident protocols. Incident reporting represents a valuable technique in improving safety in scuba diving. Its application in diving is discussed.

125 incidents were reported in the 15 months to November 1990. Forty (32%) of all the reported incidents resulted in harm. Overall the main contributing and associated factors identified were: error in judgment (24%), poor dive planning (23%), inexperience (23%), inattention (21%), diving in unfamiliar conditions (18.4%). However, there were a number of common contributing factors in these incidents which when present resulted in a higher incidence of morbidity. These were: drug and alcohol use, lack of medical clearance to dive, failure to understand dive table, insufficient training, inexperience and anxiety.

Majority (38%) of the reported incidents occurred



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during the dive, however few of these resulted in harm. 21% of incidents occurred following exit from the water, most of these were associated with harm. However, when some of these incidents were detected may not have been when the incident occurred, e.g. mis-reading of the decompression tables may not have been evident until the diver developed DCS post exit. Although only 10% of reported incidents occurred during ascent, most were harmful. About half of the post dive cases of DCS were associated with multi-level, multi-day diving. About a quarter of the cases were triggered by flying. The harmful incidents during the dive involved marine stings, panic, faulty contents gauges and salt water aspiration.

Eight (6%) of incidents occurred during training. These incidents highlighted weaknesses during training in: equalization and shared breathing (both octopus and buddy breathing) techniques, safety protocols and supervision.

About a third of all the incidents involved equipment problems, however a few were due to pure equipment failure and did not involve diver error. Equipment failures related to: inaccurate contents gauges and dive computers, rupture of the high pressure hoses and first stage blow out.

From the Hyperbaric Medical Unit, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, Adelaide, South Australia, 5000, Australia.

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