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SPUMS

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OBJECTS OF THE SOCIETY

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

OFFICE HOLDERS

MEMBERSHIP

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

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

The 2002 subscriptions are Full Members $A121.00 and Associate Members $A 60.50, includes GST in Australia. All those outside Australia will be charged the same amounts as the GST component to partly cover the cost of having the Journal delivered to them by Air Mail.

Those interested in joining SPUMS should write to

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THE EDITOR'S OFFERING

In 1979 I took over the process of preparing the SPUMS Journal for the printer. This issue is the last that I will prepare for the printer. The next issue will be the responsibility of my successor as Editor, Dr Mike Davis, the Director of the Hyperbaric Unit at Christchurch in New Zealand. Those who intend to write a letter to the Editor or to submit a paper can use the addresses that they have been using as the Journal has arranged for the <spumsj@labyrinth.net.au> e-mail address to arrive on Dr Davis' computer rather than mine and all mail to the Editor will continue to go to the Australian and New Zealand College of Anaesthetists.

It is a pleasure to be able to print two SPUMS Diploma of Diving and Hyperbaric Medicine theses in this issue. One, about the effects of cerebral arterial gas embolism in sheep is reprinted as it first published in another journal. The other, dealing with the under pressure performance of a ventilator opens the batting on page 62. Number two in the batting order is a paper from New Zealand dealing with 20 years of diving accidents leading to death in snorkellers and scuba divers. Mike Davis and his colleagues have produced a summary of their findings which suggests seven areas where actions should be taken to provide better training and elimination of a number of current situations which fail to provide for divers' and snorkellers' safety.

Diving safety and activities to enhance diving safety now and in the future appear in the paper by John Lippmann about the activities of the DAN SEAP. For the details turn to page 80.

The paper on the medical care of those working in tunnel bring the railway to Sydney Airport introduces readers to a hyperbaric world which divers know nothing about. Things have changed over the years.

No longer are tunnels dug by pick and shovel while the men labour in a pressurised environment. Nowadays tunnels which have be driven through wet ground are more often excavated using a tunnel boring machine which has a pressurised cutting head and produces a fully lined waterproof tunnel as it eats it way through the ground. The large gangs of workers exposed to pressure are largely a thing of the past. Now the only people exposed to pressure are those who enter the pressurised head to do maintenance. Because the tunnel behind the pressurised head is at atmospheric pressure the tunnels can be driven deeper with less risk of decompression sickness (DCS) because although the intervention pressures are higher the times in pressure are shorter. Using oxygen decompression tables, a first for Australia, the UK and the US, allows much shorter decompression times, and therefore longer working times in a shift, than air decompression tables.

James Francis' paper on the use of lignocaine in the treatment of neurological DCS exposes the problems of organising a double-blinded trial of a treatment for a rare disease. Even worse is getting ethical approval for doing a such a trial on a group of people who are by definition suffering from a disease which interferes with the normal function of the brain so rendering the sufferer incapable of giving informed consent to the treatment. Evidence based medicine has advantages but sometimes gathering the evidence is frustrated by the rules.

Last year's Annual Scientific Meeting theme was drowning and near-drowning. Drew Richardson's paper outlines the PADI teaching about how to rescue an unresponsive, probably unconscious, diver.

Chris Acott presented a summary of the Near Drowning UHMS Workshop, touching on the papers that he thought of most interest. One of the most interesting was Carl Edmonds' paper about the similarities and differences between a group of dead divers and a group who had survived near drowning. One of the few differences between the groups was that the survivors mostly had some air left when rescued while most of those who died did not. The Journal has been able to arrange a special reduced price for the published papers of the workshop with the Undersea and Hyperbaric Medical Society. These books will be available from the SPUMS Administrator who can be contacted C/o Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004, Australia or at <stevegoble@bigpond.com>. This information is printed on the back cover. It is a very worth while book which was reviewed in the Journal in 2000; 30 (2): 89.

Guy Williams provides an overview of the Reverse Dive Profiles Workshop organised by the Smithsonian Institution to discover whether reverse dive profiles were inherently more dangerous than the usual deep dive first type of profiles recommended by the training agencies. The upshot was that there was not enough evidence to settle the matter. This workshop was reviewed in the Journal 2000; 30 (3); 115-117, 144-145 and 145-146. The fact that this is the only time that a book has been reviewed by three different people for the Journal shows how important and interesting the proceeding were.

Our final paper is reprinted from DIVER. It is an irreverent, informative comparison between the attitudes of the average recreational diver to safety procedures and those of working (occupational), police and military divers.

I say farewell to our readers, with best wishes for the future of SPUMS and the Journal.

John Knight

ORIGINAL PAPERS

EVALUATION OF THE CAMPBELL EV 500 VENTILATOR UNDER HYPERBARIC CONDITIONS

Radha McKay and Michael Bennett

Key Words
Hyperbaric research, ventilators.

Abstract

The Campbell EV 500, an Australian anaesthesia ventilator, was tested in a hyperbaric chamber using a lung analogue as patient simulation. Three modes of ventilation were assessed: time-cycled, pressure-cycled and time-cycled/volume preset. The ventilator was adjusted to deliver a set tidal volume at 100 kPa and tidal volume (TV), inspiratory time, rate and inspiratory pressures were measured as chamber pressure increased. Time-cycled/ volume preset was the only mode that delivered consistent minute ventilation over a wide range of compliance and resistance settings. With one exception, mean tidal and minute volume remained within 10% of the original value, inspiratory time and rate were unaffected by the increase in ambient pressure and inspiratory pressures were within clinically acceptable limits (7 to 25 cm H_2O). The exception occurred at a pressure (400 kPa) greater than any treatment pressure that we currently use in our facility with the use of a high set tidal volume (1,000 ml) and a low lung compliance (12.5 ml/cm H_2O). Accurate control of inspiratory time, rate and tidal volume contributed to the ability to provide consistent minute ventilation. The ventilator compared favourably with others previously reported in the literature.

Introduction

For the past two to three decades there have been sporadic descriptions in the literature of mechanical ventilation in the hyperbaric environment.[1-11] In recent years, with increased indications for use of hyperbaric oxygen therapy, the number of critically ill patients requiring mechanical ventilation in the chamber has grown. During this time intensive care ventilators have become more sophisticated but there has been a relative lack of progress made in the development of ventilators for hyperbaric use.

There are a number of reasons for this. The hyperbaric chamber provides particular challenges for mechanical ventilation.

1 Gas density increases proportionately as ambient pressure increases.[12] As gas density increases, turbulent flow through fixed orifices within the ventilator decreases and increased pressure is required to overcome airway resistance and inflate the patient's lungs.

2 Any ventilator needs to comply with the strict fire safety regulations for safety in a hyperbaric chamber. These specify that the electrical power supply must be no greater than a 12 volt DC battery, no flammable lubricants that could act as a fuel source should be used within the ventilator and the percentage of oxygen within the chamber be kept at less than 23%.

3 Any enclosed gas pockets within the ventilator mechanism (for example, hermetically sealed transistors) may be damaged during compression and decompression.

4 Hyperbaric chambers tend to be small, with limited space for any equipment.

5 Because the patient is often cared for by a lone attendant, it is desirable that the ventilator requires as little adjustment as possible when the pressure within the chamber alters, leaving the attendant free to react quickly to a patient's changing clinical condition.

In view of these problems, and despite the fact that there have been some ventilators specifically designed for hyperbaric use, to date there is no "ideal" ventilator available for use in hyperbaric chambers. There is a suggestion, however, that volume-preset ventilators are, as a class, superior to others. Blanch et al., in the largest comparative ventilator study to date, found that volume-preset ventilators outperformed other classes of ventilator in maintaining tidal volume, rate and inspiratory times over a wide range of ambient pressures with the Oxford Penlon being the best of these.[3,9]

This paper describes the evaluation of the Campbell EV 500 ventilator under hyperbaric conditions up to an ambient pressure of 400 kPa (4 Atmospheres absolute). Although this ventilator is not specifically designed for hyperbaric use, it has a number of features that make it a desirable candidate for assessment. These include compatibility with fire safety regulations, compact size, pneumatic power by either air or oxygen and the ability to operate in a volume-preset mode. Assessment involved measurement of tidal volume, inspiratory time, rate and inspiratory pressure in the three different ventilatory modes available and evaluation of the ability of the ventilator to function under the varied lung compliance and resistance settings likely to be encountered in clinical practice. Our hypothesis was that with this ventilator, tidal volume would remain in clinically acceptable ranges under hyperbaric conditions.

Materials and Methods

The Campbell EV 500 Ventilator (ULCO Medical, Marrickville, New South Wales) is a time-cycled, gas driven machine which is primarily used as an anaesthetic ventilator (Fig 1). It is divided into two main systems, electronic and pneumatic. Inspiratory and expiratory times are controlled electronically using a microcontroller that generates timing signals using a quartz crystal oscillator. This, in turn, controls pneumatic valves that allow gas flow. The electronic system also powers the battery charger, the alarm system and the pressure transducer used during pressure-cycled mode.

Due to strict fire safety requirements when operating within the hyperbaric chamber, the ventilator is powered by a 12 volt DC battery (life 10 hours). There is a 6 volt DC back-up battery (life 3 hours) if power is low. The batteries are automatically recharged when the ventilator is connected to a 240 volt AC mains power supply. The pneumatic system is designed to be powered by a driving gas of medical air or oxygen at 350 – 450 kPa, the standard operating theatre supply line pressure. The ventilator trials were undertaken using 100% oxygen both as a driving gas and in the patient circuit.

Two minor modifications were made to the gas delivery system to improve its utility under hyperbaric conditions. Initially, a regulator designed by the manufacturer to keep driving pressure at 800 kPa (allowing 400 kPa driving pressure at chamber pressure of 400 kPa) lost pressure unexpectedly. This problem was solved by connecting the ventilator directly to the chamber oxygen line so bypassing the faulty regulator. The driving pressure was then maintained by altering the line pressure to 400 kPa above chamber pressure by a direct link with the chamber control systems. Secondly, the flowmeter used in the patient circuit was not accurate at low flows (below 2 l/min) and a flowmeter calibrated for low flows has subsequently been acquired for use with the ventilator.

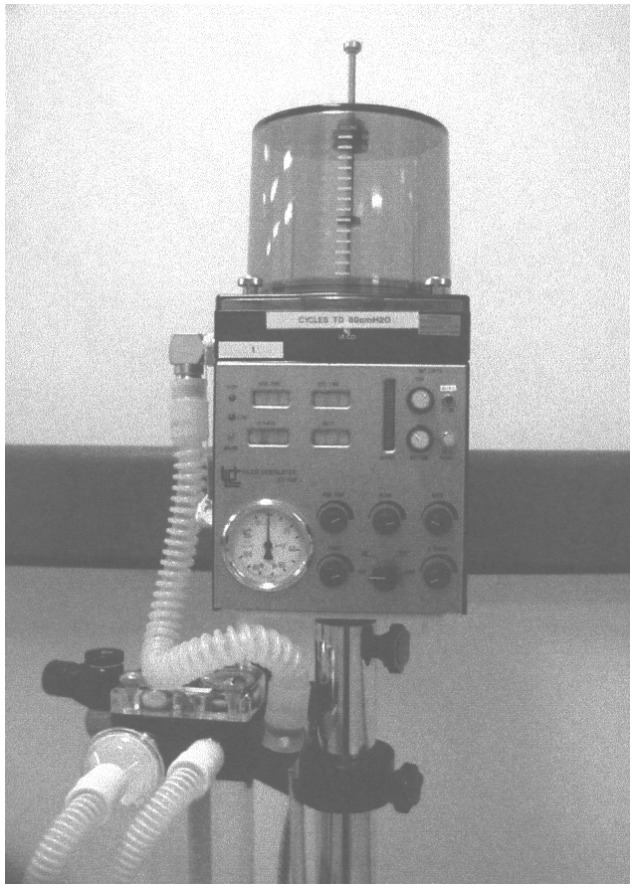

Figure 1. Campbell EV 500 ventilator with bellows attachment.

The ventilator was set up to operate in 3 different modes; time-cycled, pressure-cycled and time-cycled/volume-preset (Table 1). In the first two modes the driving gas also provides gas for ventilation and during passive exhalation gas is vented into the chamber via an expiratory valve. In the time-cycled mode, the inspiratory/expiratory

TABLE 1

SUMMARY OF VENTILATOR MODES

Mode	Circuit	Inspiratory Time/Rate	Tidal Volume	Pressure Limit
Time-cycled (TC)	Driving gas powers ventilator and ventilates patient	Set electronically	Set at surface using inspiratory time and pressure.	Upper limit 60 cm H_2O
Pressure-cycled (PC)	As above	Dependent on time to reach pressure limit	Set at surface using pressure limit	25 cm H_2O
Time-cycled/ Volume-limited (TC/VL)	Driving gas compresses bellows. Separate patient circuit	Set electronically	Set by limiting excursion of the bellows	Upper limit 60 cm H_2O

times and rate are set electronically allowing tidal volume (TV) and inspiratory pressure to vary as ambient pressure changes. In pressure-cycled mode, inspiratory time and pressure are set to maximum and the upper limit LED is set to the required switching pressure. When the cut-off pressure is reached the ventilator cycles into expiration. Rate is determined by the time taken to reach the pressure limit in the inspiratory phase in combination with the expiratory time and not the rate that is displayed on the ventilator (the rate set for time-cycled mode).

In the time-cycled/volume-limited mode a rising bellows is added to the ventilator and this can be adjusted to deliver any tidal volume between 25 and 1,300 ml (Fig 1). The driving gas is used to compress the bellows during the inspiratory phase and a separate patient circuit provides one way flow around the circle system for ventilation and CO_2 absorption. Oxygen is supplied to the patient circuit by a ball flowmeter and need only be set at low flow to replace oxygen consumption once the patient has been denitrogenated. In practice we usually set flows at 2 l/min to provide oxygen for metabolic and therapeutic needs and to overcome any minor leaks in the circle. Inspiratory time and rate are set electronically with inspiratory time long enough to allow the bellows to empty completely.

Other features of the ventilator include a maximum pressure limit of 60 cm H_2O, the ability to provide PEEP (positive end expiratory pressure) to 20 cm H_2O (not tested under hyperbaric conditions in this study) and a flow control. This has the ability to change the inspiratory characteristics of the ventilator from a flow generator to a pressure generator or any selected position in between these two extremes. This control was left in the mid position and not altered during the trials.

The ventilator performance was assessed by an observer within a multiplace hyperbaric chamber in the Department of Diving and Hyperbaric Medicine at the Prince of Wales Hospital, Randwick, NSW, at pressures of 100, 130, 240, 280 and 400 kPa. Observation time was limited to 15 minutes, the maximum time allowed for a no-decompression dive to 400 kPa using Canadian DCIEM dive tables. The maximum pressure of 400 kPa was chosen as it was considered to exceed the greatest possible pressure at which 100% oxygen would be used to ventilate a patient.

Patient simulation was provided by a lung analogue (Michigan Instruments, Grand Rapids, Michigan) in which both compliance and resistance were adjustable. We evaluated ventilator function at lung compliances of 50, 25 and 12.5 ml/cm H_2O/lung and airway resistances of 2.48 and 5.44 cm H_2O/litre/second. These values were chosen to reflect both the range of values for compliance and resistance previously studied in the literature and found in the patients we encounter in clinical practice.

Inspiratory time was set using the inspiratory time control knob in time-cycled and time-cycled/volume-limited settings and the inspiration to expiration (I/E) ratio remained constant. In pressure-cycled mode, inspiratory time depended on the time taken to reach the pressure limit. All times were measured using a digital stopwatch (average of five readings) at each pressure.

A Wright respirometer, calibrated before the start of the study, was used to measure tidal volumes at all observation pressures. Five tidal volume readings were taken at each pressure and then averaged.

Temperature within the chamber was recorded using a Spacelabs thermometer and was established to have no measurable effect on tidal volume readings. Tidal volume measurements quoted in the results were measured at ambient chamber temperature and pressure.

Before each test run the ventilator was set to deliver a specific tidal volume at 100 kPa, with other parameters such as inspiratory time, rate and pressure limits controlled as described in Table 1. Once the settings on the ventilator were fixed, they were not altered in the time-cycled and pressure-cycled modes. Due to volume lost in the patient circuit as a result of Boyle's Law, flow through the patient circuit was transiently increased in time-cycled/volume preset mode to refill the bellows at 400 kPa. We recorded tidal volume, airway, ventilator and driving pressure, inspiratory time and rate, and chamber temperature at each ambient pressure.

One-way analyses of variance (ANOVA) were used to evaluate the variation in tidal volume and minute ventilation at different pressures. Two tail t-tests were used to assess differences in tidal volume and minute ventilation at 100 and 400 kPa with 95% confidence intervals of the mean difference being quoted where appropriate. A P value of <0.05 was accepted as statistically significant.

Results

CALIBRATION OF THE WRIGHT RESPIROMETER

Using a 2 litre Rudolph gas calibration syringe and a similar protocol to Gibson et al.[6] showed that the respirometer consistently over-read the tidal volume by approximately 4.5% at low tidal volumes (less than 500 ml) at 240 and 280 kPa. At these pressures the over-reading decreased to 3% at higher tidal volumes. The error increased slightly (6% going down to 4%) at 400 kPa. The recordings have been adjusted for this error.

TIDAL VOLUME MEASUREMENTS

The variation of tidal volume measurements was low with a less than 1% difference from the mean measurement for all measurement conditions.

TIME-CYCLED MODE

Tidal volume decreases as ambient pressure increases (Figure 2 and Table 2), dropping to approximately one half of the original volume at 4 kPa in time-cycled mode. For example, a tidal volume set at 500 ml at 1 kPa (TV 500 ml) was reduced by -265 ml at 4 kPa, P value = <0.01, 95% confidence interval (CI) in means = -254 to –276 ml. Inspiratory time (1.5 seconds), rate (10) and I/E ratio (1:3) remained unchanged and peak airway pressure (PAP) decreased from 14 cm H_20 at sea level (1 kPa) to 8 cm H_20 at 4 kPa (Table 3).

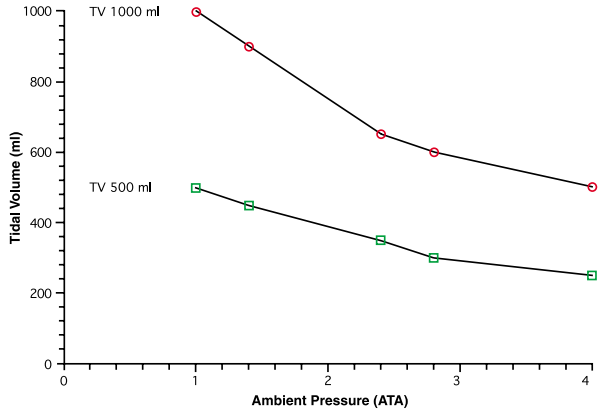

Figure 2. Mean tidal volumes with ventilator in time-cycled mode. Compliance 25 ml/cm H_2O/lung and resistance at 2.48 cm H_2O/l/sec. (1 ATA =100 kPa approx.)

PRESSURE-CYCLED MODE

This mode delivered a consistent tidal volume to 400 kPa (Figures 3 and 4 and Table 2). As chamber pressure increased, however, the time to cycle to the selected cut-off pressure also increased such that inspiratory time increased three-fold at 4 kPa. For example, for a 500 ml TV the mean increase in inspiratory time at 400 kPa was 1.54 seconds, P = <0.01, 95% CI 1.38 to 1.69 seconds. With expiratory time unchanged, minute ventilation dropped. The mean decrease in minute volume was 2,462 ml, P= <0.01, 95% CI 2,075 to 2,848 ml.

TIME-CYCLED/VOLUME-PRESET MODE

As with the pressure-cycled mode, there was little variation in delivered tidal volume with the time-cycled/ volume preset mode over the range of pressures tested (Figure 5 and Table 2). However, the electronically set inspiratory time and rate also remained constant thus providing consistent minute ventilation. Peak airway pressures (PAP) were in the clinically acceptable range, starting at 12 cm H_20 at 100 kPa, and remaining within 2 – 3 cm H_20 of the original value throughout the observation period (see Table 3). We observed, but did not quantify, that with increasing ambient pressure the bellows tended to empty more slowly, resulting in a shorter inspiratory pause phase of ventilation.

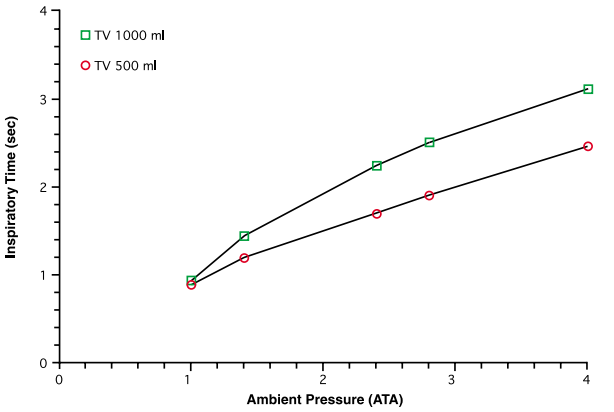

Figure 3. Mean inspiratory time with ventilator in pressure-cycled mode. Compliance 25 ml/cm H_2O/lung and resistance at 2.48 cm H_2O/l/sec. (1 ATA =100 kPa approx.)

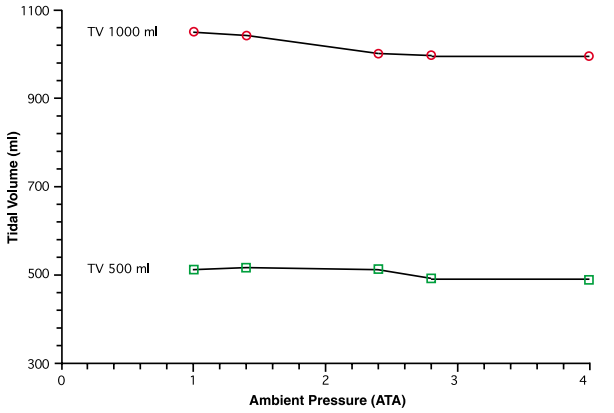

Figure 4. Mean tidal volume with ventilator in pressure-cycled mode. Compliance 25 ml/cm H_2O/lung and resistance at 2.48 cm H_2O/l/sec. (1 ATA =100 kPa approx.)

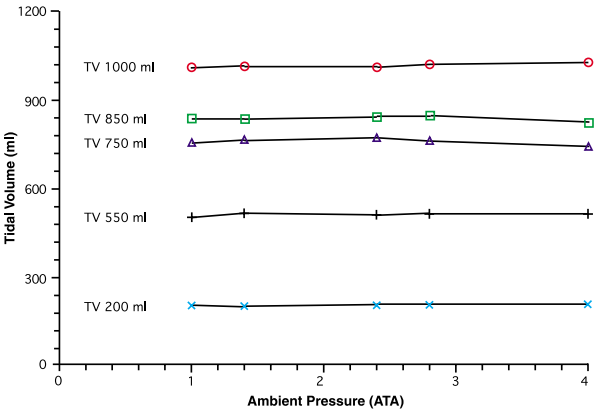

Figure 5. Mean tidal volume with ventilator in time-cycled/ volume-limited mode. Compliance 25 ml/cm H_2O/lung and resistance at 2.48 cm H_2O/l/sec. (1 ATA =100 kPa approx.)

TABLE 2
TIDAL AND MINUTE VOLUMES IN ml AT 1 AND 4 ATA (100 kPa and 400 kPa)

95% Confidence Intervals and P values given only for differences of <10% of the initial value. TC = Time-cycled, PC = Pressure-cycled, TC/VP = Time-cycled/ volume-preset.

Mode	Compliance ml/cm H_2O	Resistance cm H_2O/l/s	Tidal Volume 1 ATA	Tidal Volume 4 ATA	Mean Difference	95% Confidence Interval	P Value	Minute Volume 1 ATA	Minute Volume 4 ATA	Mean Difference	95% Confidence Interval	P Value
TC	25	2.48	1,000	488	-512	-500 to-523	<0.0001	10,000	4,880	-5,120	-5,000 to -5,230	<0.0001
TC	25	2.48	508	243	-265	-265 to -276	<0.0001	5,080	2,430	-2,650	-2,540 to -2,760	<0.0001
PC	25	2.48	1,022	980	-42			15,656	9,666	-5,990	-5,475 to -6,506	<0.0001
PC	25	2.48	514	496	-18			7,963	5,501	-2,462		
TC/VP	25	2.48	1,016	1,050	+34			10,160	10,500	+340		
TC/VP	25	2.48	850	826	-24			8,500	8,260	-240		
TC/VP	25	2.48	758	744	-14			7,580	7,440	-140		
TC/VP	25	2.48	514	516	+2			5,140	5,160	+20		
TC/VP	25	2.48	212	219	+7			2,120	2,190	+70		
TC/VP	50	2.48	1,046	994	-52			10,460	9,940	-520		
TC/VP	50	2.48	754	732	-22			7,540	7,320	-220		
TC/VP	50	2.48	500	456	-44			5,000	4,560	-440		
TC/VP	50	2.48	244	223	-21			2,440	2,230	-210		
TC/VP	12.5	2.48	1,008	828	-180	-168 to -192	<0.0001	10,080	8,280	-1,800		
TC/VP	12.5	2.48	754	754	0			7,540	7,540	0		
TC/VP	12.5	2.48	510	538	+28			5,100	5,380	+280		
TC/VP	12.5	2.48	256	246	-10			2,560	2,460	-100		
TC/VP	25	5.44	945	940	-5			9,450	9,400	-50		
TC/VP	25	5.44	752	710	-42			7,520	7,100	-420		
TC/VP	25	5.44	530	540	+10			5,300	5,400	+100		
TC/VP	25	5.44	249	237	-12			2,490	2,370	-120		

TABLE 3

SUMMARY OF PEAK AIRWAY PRESSURES

Mode	Compliance ml/cm H_2O	Resistance cm H_2O/l/s	Tidal Volume (ml)	Peak Airway Pressure cm H_2O 100 kPa	400 kPa
TC	25	2.48	1,000	20	15
TC	25	2.48	500	14	8
PC	25	2.48	1,000	25	25
PC	25	2.48	500	25	25
TC/VL	25	2.48	1,000	22	25
TC/VL	25	2.48	500	12	12
TC/VL	25	2.48	250	7	7
TC/VL	50	2.48	1,000	16	18
TC/VL	50	2.48	500	12	9
TC/VL	50	2.48	250	10	8
TC/VL	12.5	2.48	1,000	44	38
TC/VL	12.5	2.48	500	25	25
TC/VL	12.5	2.48	250	12	11
TC/VL	25	5.44	950	26	26
TC/VL	25	5.44	500	15	13
TC/VL	25	5.44	250	15	9

Having established that consistent minute ventilation could be achieved in this mode without the need for readjustment, the ventilator was assessed over a range of tidal volume, airways resistance and lung compliance. The ventilator displayed consistent performance in this mode over the range of tidal volumes tested (200 to 1,000 ml). Inspiratory time, respiratory rate and I/E ratio all remain close to initial surface values. The limit of the ventilator in dealing with high tidal volumes in the setting of very low lung compliance is shown in Table 2 where the set tidal volume of 1,000 ml was reduced to a mean of 828 ml. This was a difference of 172 ml, $P < 0.01$, 95% CI 168 to 192 ml. At these extreme parameters the PAP was also high (44 cm H_20 at 100 kPa), but decreased at higher ambient pressure (see Table 3).

Table 2 summarises the differences in tidal volume and minute ventilation at extremes of ambient pressure on each test run, with 95% confidence intervals and P values for those runs where the difference exceeded 10% of the values at 100 kPa. We considered smaller differences were not clinically important. The largest differences were obtained from the data collected with the ventilator in time-cycled and pressure-cycled modes. In these modes minute ventilation falls to between one half and one third the value set at 100 kPa. In the time-cycled/volume-preset mode on the other hand, the maximum mean difference is less than 10%.

A summary of PAPs obtained through out the trial is contained in Table 3. Change in chamber pressure had minimal effect on peak inspiratory pressure, which remained within the clinically acceptable range. As expected, in the setting of high tidal volumes and very low lung compliance, peak airway pressures were high at sea level and remained so at 400 kPa.

Discussion

The decision to study the Campbell EV 500 ventilator under hyperbaric conditions came about partly as a result of dissatisfaction with the existing ventilators available in our department and partly due to the non-availability of an "ideal" alternative. We considered it advantageous to trial a ventilator which was manufactured and maintained in Australia and which the manufacturers were willing to modify to meet our requirements.

Use of the Wright respirometer as the device to measure tidal volume was based on its ready availability and acceptability by Whittle et al.[13] Although in their

comparison of the Dräger Volumeter and the Wright respirometer the Dräger was considered slightly more accurate, the authors acknowledged that both instruments were suitable for hyperbaric use. The accuracy of our Wright respirometer compared favourably with that of Gibson et al. which over-read by 8% with low tidal volumes at 280 kPa which became 5% at higher tidal volumes. The device was assessed for systematic error as described and the observations were adjusted for this.

On review of the literature it was evident that the vast majority of ventilators studied were pneumatic time-cycled (PTC) devices.[1,2,9,11,12] There are good reasons for choosing this type of ventilator because they have many desirable features, including compact size, ease of operation, robustness, use during patient transport and lack of electrical components. In practice, however, all these ventilators are significantly affected by changes in ambient pressure. In the largest study to date, Blanch et al. noted that as pressure increased, tidal volume and both inspiratory and expiratory time decreased while the rate increased dramatically.[9] The most likely explanation for these changes is that the operation of these ventilators is affected by the increase in gas density that occurs as pressure within the chamber rises. As gas density increases, turbulent flow through an orifice or needle valve decreases as predicted by the equation for bulk gas flow, flow α 1/gas density.[9] Therefore, at increased ambient pressure, flow through fixed orifices within the ventilator decreases, so reducing the delivered tidal volume over any given inspiratory time. Additionally, the timing devices in the PTC ventilators were affected by change in gas density, altering rate, inspiratory and expiratory times, a factor discussed at length in the paper by Blanch et al.[9]

The results recorded with the Campbell EV 500 in time-cycled mode reflect the findings in other studies with the exception that the EV 500 was able to maintain rate, inspiratory and expiratory times. The reduction in tidal volume is consistent with reduced flow through fixed orifices that occurs with other time-cycled ventilators but the major advantage with the EV 500 is that it has an electronic timing device that is unaffected by pressure or gas density changes. It was possible to return the tidal volume to its original value by altering the inspiratory time and inspiratory pressure with each change in ambient pressure but this is at the expense of expiratory time, reducing I/E ratio. At the completion of the treatment period these changes need to be reversed in order to avoid the risk of significant hyperventilation and barotrauma during decompression.

In pressure-cycled mode, the EV 500 performed similarly to those studied by Blanch et al.[9] While tidal volume remained relatively constant, inspiratory time increased as the time taken to reach the cycling pressure lengthened. In the present study expiratory time was held constant electronically so that as inspiratory time rose, rate fell, ultimately decreasing minute volume at 400 kPa to one third of that originally set at sea level. Again, it was possible to return minute ventilation closer to its original value but this could only be achieved by decreasing expiratory time, which risked incomplete exhalation before the commencement of the next breath (breath stacking). Additionally, as Blanch et al. point out, pneumatic pressure cycled ventilators are a poor choice for hyperbaric therapy as tidal volume depends directly on lung compliance and airway resistance and a sudden change in either or both of these parameters can result in a potentially dangerous change in tidal volume.[9]

The only mode in this study that achieved clinically acceptable consistent minute ventilation at all ambient pressures tested was time-cycled/volume-preset mode. In the study by Blanch et al. the group of ventilators classified as “volume-cycled” generally out-performed those in other categories but there was considerable variation among the three ventilators tested.[9] If, as implied by the term volume-cycled, the change from inspiratory to expiratory phase occurred once a certain tidal volume has been supplied, then one would expect that as ambient pressure increased, inspiratory time would lengthen and a decrease in ventilatory rate would result as predicted by Skinner.[12] The ability of the ventilator to maintain rate in this mode was therefore dependent on the timing mechanism. On closer study of the timing mechanisms of volume-cycled ventilators from previous studies,[2-4] it would appear that the Oxford Penlon was the only ventilator that had a timing device that was unaffected by change in gas density and was therefore able to maintain minute ventilation to 3100 kPa.

The Campbell EV 500 has a timing device that is similarly unaffected by change in gas density and is therefore able to maintain rate and I/E ratio. The combination of the ability to set tidal volume by limiting the excursion of the bellows and the electronic timing device that confers accuracy in inspiratory time and rate are the means by which consistent minute ventilation can be achieved. Our results suggest this can be achieved over a large range of tidal volumes from paediatric to large adults and over a wide range of lung compliance and resistance settings. Mean tidal and minute volume remain within 10% of the original value, a figure well within the acceptable clinical limits quoted in at least one other study.[10] The one exception occurred at a pressure greater than any treatment pressure we currently use within our facility. The observation of a decreased inspiratory pause phase at higher ambient pressures was not considered to be clinically significant.

To the authors’ knowledge this is the first ventilator with an electronic timing device that has been studied in a hyperbaric chamber. Previous studies have suggested it may not be ideal for a ventilator used in a hyperbaric environment to have any electrical power source because of the risk of fire.[4,9,12] On the other hand, a device that has no electrical power frequently has no pressure or disconnect alarms, a factor noted to be a disadvantage in a number of the

ventilators studied to date.[12] Youn and Myers have published a study recommending that the battery powered Ohmeda volume monitor be used as an adjunct to the Oxford Penlon ventilator for this reason.[14] Because of its battery power, the EV 500 ventilator has in-built loss of power (gas or electric), disconnect, overpressure and non-cycling alarms.[15] The means of power and electrical safety are important issues in the hyperbaric chamber. Sealed batteries are considered the safest form of electrical power and the battery used in the EV 500 is similar to those in common use in the hyperbaric environment.

Another criticism that has been levelled at "volume-cycled" ventilators is that they tend to be bulkier than other varieties. Certainly, with the addition of the bellows and the requirement for the separate patient circuit, the EV 500 takes up more room in volume-limited mode than it does while operating in either of the other two modes. In our practice, the consistent performance in volume-limited mode more than outweighs the inconvenience of accommodating the bulkier circuit.

A further potential advantage of having a separate patient circuit is that the ventilator could be powered on air rather than oxygen, thus reducing the amount of oxygen needing to be vented from the chamber to maintain the O_2 concentration below 23%. This question was not addressed in the current study, but Gibson et al. found that the slightly different densities of these two gases did not alter their delivered tidal volumes to a clinically important degree.[6]

The PEEP function on this ventilator has not been formally evaluated. However, given that many of the goals of PEEP, such as improvement in oxygen delivery and reduction in FIO_2 (fraction of inspired oxygen) to reduce the risk of oxygen toxicity, are of less concern during hyperbaric oxygen therapy, there may be a limited role for PEEP in the hyperbaric chamber.

In conclusion, the results obtained using the Campbell EV 500 ventilator in time-cycled/volume-limited mode can be compared favourably with other ventilators studied to date, including those ventilators specifically designed for hyperbaric use. It provides consistent minute ventilation without the need for adjustment from 100 to 400 kPa throughout a wide range of tidal volumes and lung compliance and resistance settings. However, it does not provide the capability for intermittent mandatory ventilation (IMV), synchronised IMV (SIMV) or assist/control ventilation as the Monaghan 225 does, but the Campbell outperforms it significantly in other regards. Australian manufacture confers the advantage of ease of maintenance and relatively low purchasing cost to Australasian Hyperbaric Medicine Units. In our institution it also has the advantage of being a ventilator that anaesthetists are familiar with as it is used in other areas of the hospital. Clinical trials are underway to ensure that this ventilator performs as well as would be predicted by this study.

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The original research described in this paper was undertaken by Dr McKay as the research component of the Diploma of Diving and Hyperbaric Medicine.

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SNORKELLING AND SCUBA DIVING DEATHS IN NEW ZEALAND, 1980-2000

Michael Davis, Margaret Warner and Brendon Ward

Key Words
Deaths, drowning.

Abstract

Drowning is the third leading cause of unintentional injury death in New Zealand (NZ). Between 1980 and 2000 there were 184 unintentional diving related deaths in NZ, 61 snorkellers and 123 scuba divers. The mean age was 34.3 years, and 24% were Maori. Diving related deaths were 5.4% of the total 3449 unintentional drownings recorded over this period. The death rate in NZ scuba divers is estimated to be at least 5.8 per 100,000 divers per year. Many deaths were associated with inadequate training, poor water skills/fitness and poor equipment. Over 75% of scuba divers were either diving alone or became separated from their buddy. The bodies of 173 people were recovered. Blood alcohol was measured in 72 victims and this was positive in 21% of snorkellers and 8% of scuba divers tested. Pre-existing medical conditions may have contributed to death in 24% of snorkellers and 20% of scuba divers, many of whom were uncertified. If the coroner's autopsy was performed to recommended guidelines, arterial gas embolism was identified as a contributing factor in 55%. The reporting of diving accidents needs to be better standardised. The number of untrained divers and those with pre-existing medical conditions suggest tighter standards for medical assessment and some means of restricting scuba equipment use by untrained people may be required. Diving activities contribute importantly to water recreation fatalities in New Zealand although no increase in the annual diving death rate is evident over this 21-year period.

Introduction

Drowning is the third leading cause of unintentional injury death in New Zealand (NZ).[1] The Injury Prevention Research Unit (IPRU) recently analysed drowning related incidents in NZ for the period 1980-1994, using linked data files.[2] A brief section on diving fatalities in this report prompted one of the authors (MD) to review these data in greater detail and to update these with Water Safety New Zealand (WSNZ) records to year 2000. There were three purposes in mind. Firstly, to provide a comprehensive review of the epidemiology of diving related deaths over a 21-year period in NZ. Secondly, to report on diving practices and other factors that might contribute to these deaths and thirdly to document the way diving accidents were reported.

Methods

The study was performed in several stages. A database of scuba and snorkelling related deaths was built from several sources. Information in the database was verified and supplemented using WSNZ files and, where necessary, missing coronial files were obtained. First, cases for the period 1980-1994 were captured with the IPRU database developed by linking information from the NZ Health Information Service (NZHIS)[3,4] and from the WSNZ database called DrownBase™. For the period 1995-2000, DrownBase™ alone was used to capture cases. Since WSNZ and NZHIS now collaborate, this should be comparable to the IPRU database. The IPRU and WSNZ databases matched for all but five deaths, whilst five divers were incorrectly coded - two scuba divers and three snorkellers.

Next, the individual case files held by WSNZ for the whole period, 1980-2000, were examined by one author (MD). Finally, where the information in the case records was deficient, the coronial records were obtained from the Tribunals Division, Department of Courts. Data for the year 2000 were incomplete as several coroner's hearings had not been held at the time of analysis. The WSNZ and coronial

case records were searched for demographic details, specific aspects of diving practice, environmental factors, medical history and the autopsy reports. A total of 30 fields were included in the final MS Access® database (Table 1).

Autopsy reports were examined to identify pre-existing disease, especially respiratory or cardiovascular pathology, which might have been contributory. In scuba divers recovered early after their deaths particular attention was paid to whether the autopsy was carried out in compliance with the guidelines of the Royal College of Pathologists of Australasia (RCPA).[5]

TABLE 1

VARIABLES RECORDED FROM EXAMINATION OF THE IPRU AND WSNZ DATABASES AND CORONER'S REPORTS.

ID code	Solo diver	Buddy's action
Snorkel/Scuba	Separation	Body recovery
Age	BC inflation	Basic Life Support
Sex	Weights dropped	Asthma
Ethnicity	Out of Air	CVS disease
Qualification	Panic	Epilepsy
Experience	Sea state	Other medical
Region	Cave/wrecks	Autopsy (PM)
Depth	Entanglement	Gas embolism
Dive Time	Trauma	Other PM findings
	Free text	

Results

There were 186 diving deaths recorded. These cases were all corroborated by inspection of the individual files. Two deaths were excluded - one scuba death subject to a murder investigation and a boat occupant not snorkelling or diving - leaving 184 divers, 61 snorkellers and 123 scuba divers. The annual rate varied from a low of four (1999) to a high of 16 (1985) (average 8.8 per year, range 4 – 16), but there was no obvious trend over these two decades (Figure 1). Diving related deaths were 5.4% of the 3449 unintentional drownings recorded in NZ over this period (Table 2).

WSNZ divides drownings into various categories related to the activity, type of water and region of the country. One of these is "Sporting and Recreational", which includes angling, board-riding, diving (as in jumping), duck shooting, net & shell fishing, rafting & tubing, river crossing, snorkelling, scuba diving, swimming and windsurfing. During 1980-94 there were 425 deaths in this category, 275 of them in the sea of which 129 (47%) were divers. Diving drownings occurred throughout NZ but with 40% in the

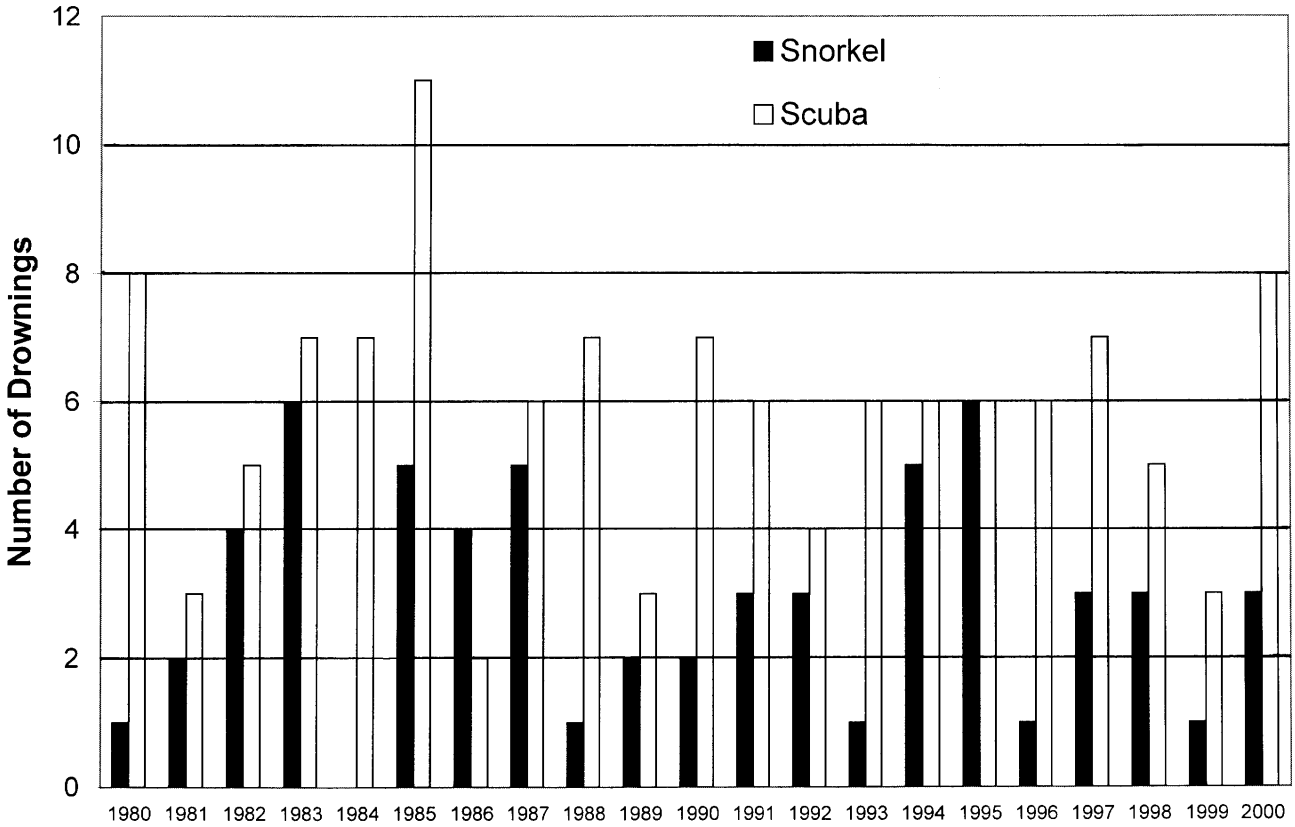

Figure 1. Annual snorkeling and scuba diving drownings in New Zealand, 1980-2000.

TABLE 2

NEW ZEALAND DROWNINGS, 1980-2000. DIVING DROWNINGS ARE 5.4% OF ALL DROWNINGS.

	All drownings	Snorkeling	Scuba
1980-1994	2606	44	88
1995-2000	843	17	35
Total	**3449**	**61**	**123**

two northern regions (Table 3) and were most common in the summer months as shown in Figure 2.

DEMOGRAPHICS

The sex, ethnicity and age of drowned divers are shown in Table 4. All but one snorkeller was male, whereas 14.6% of scuba deaths were in women. Of the 44 Maori drowned, only two were women. Thirty four percent of snorkelling deaths and 19% of scuba deaths occurred in Maori (overall, 24%). The youngest scuba drowning was aged 13, and snorkeller sixteen. Age distribution is slightly skewed (Figure 2), with 10% being over 50 years old.

TABLE 3

184 SNORKEL AND SCUBA DIVING DROWNINGS IN DIFFERENT REGIONS OF NEW ZEALAND

	Number	%
Northland	36	19.6
Auckland, Hauraki Gulf and Coromandel	38	20.6
Bay of Plenty	16	8.7
Hawkes Bay and Gisborne	14	7.6
Wellington	25	13.6
Marlborough and Tasman	19	10.3
Canterbury, Otago and Southland	21	11.4
Other and unknown	9	4.9
All sea drownings	**178**	**96.7**
Lakes and Rivers	**6**	**3.3**

DIVING EXPERIENCE

Diving experience was documented in 88 scuba divers, but their diving qualifications in only sixty-seven. Experience was defined loosely for this study as inexperienced – less than two years diving, moderate – two to four years or a qualification above open water/basic and experienced – more than four years or at least Dive Master.

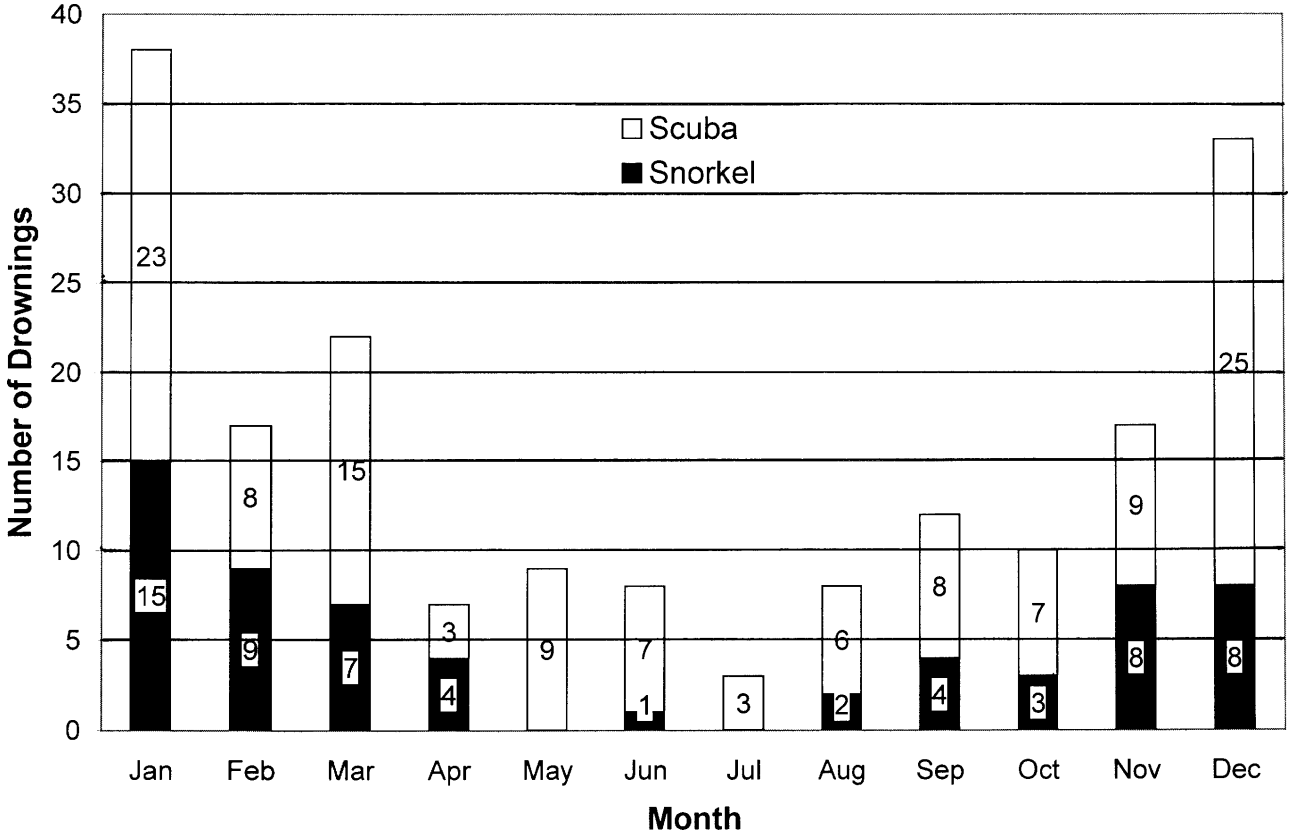

Figure 2. Snorkeling and scuba diving drownings in New Zealand, 1980-2000, by month.

TABLE 4

SEX, AGE AND ETHNICITY OF DROWNED SNORKELERS AND SCUBA DIVERS, 1980-2000.

		Snorkel (N=61)	Scuba (N=123)
Male		60	105
Female		1	18
Age:	Mean	34.3	34.3
	Median	31	33
	Mode	23	30
	Range:	16 to 66	13 to 73
Asian		3	4
European		31	89
Maori		21	23
Pacific Islander		3	1
Unknown		3	6

The majority of deaths occurred in inexperienced or unqualified divers (Table 5). Six divers drowned on their first or second dive, three on basic diving courses. Three divers died in a triple fatality on a Dive Master course. The level of experience of only 19 snorkellers was recorded, of whom nine were described as experienced. Whether any of the snorkellers had a scuba qualification was rarely recorded.

DIVING CONDITIONS

Information on the depth was recorded for 93 scuba divers. This might be the depth where a diver disappeared, or from which the body was recovered or the diver is known to have got into difficulties and was therefore of little value. The surface, tidal or underwater conditions were recorded in 109 scuba accidents, and were believed to be contributory to 29 (27%) of these deaths. Examples included seven divers who suffered head injuries from being swept off or against rocky shores in breaking or rough seas. In total, 10 divers died on the surface. Surges and rips, both surface and underwater, contributed to at least 11 scuba deaths, the most notable of which was a triple fatality in a party of seven divers who were swept to a depth of over 90 m by strong currents during a dive. Two inexperienced divers became

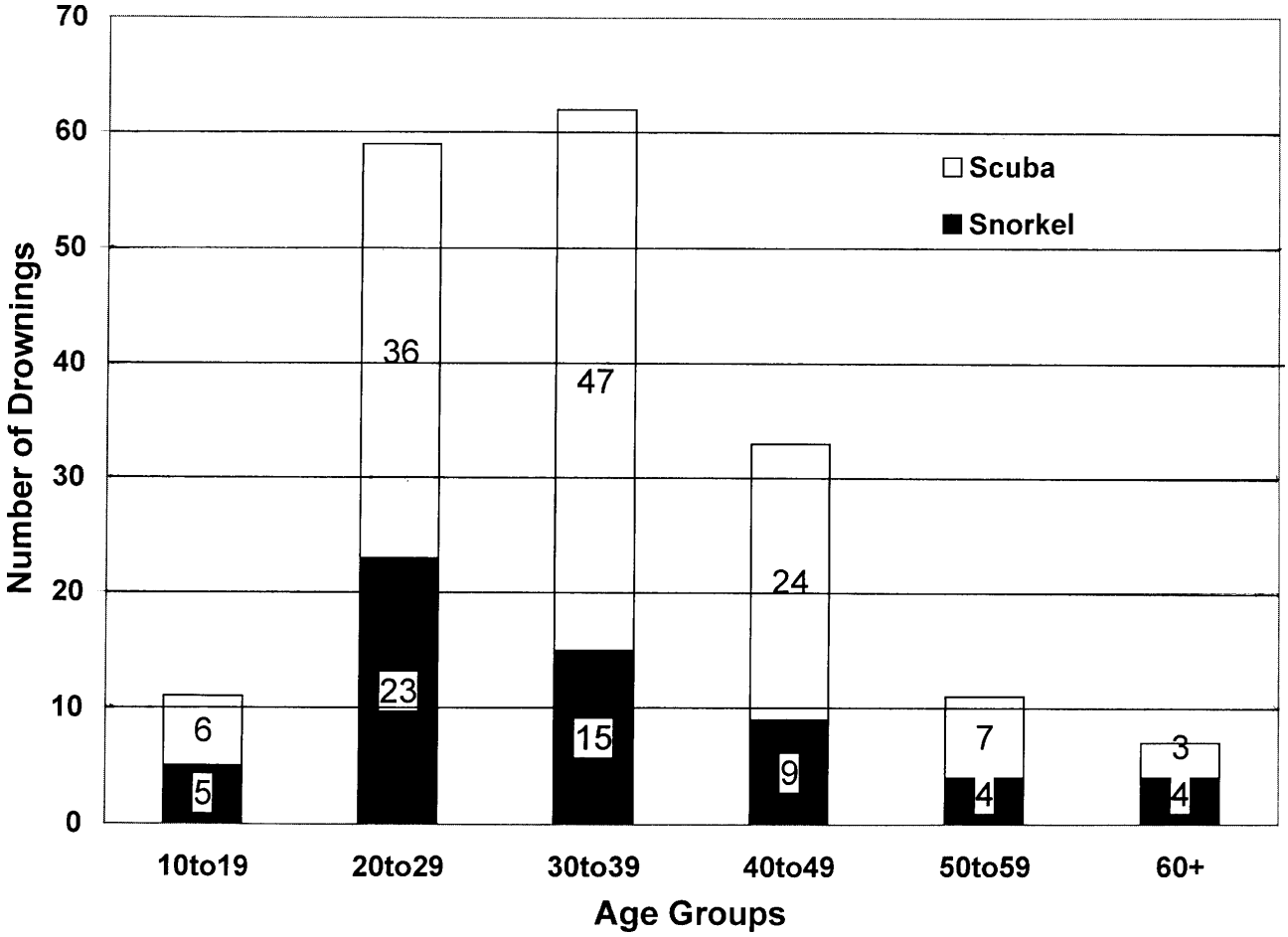

Figure 3. Snorkeling and scuba diving drownings in New Zealand, 1980-2000, by 10-year age groups.

TABLE 5

DIVING EXPERIENCE AND QUALIFICATIONS IN DROWNED SCUBA DIVERS.

Experience (N= 88)	Number
Inexperienced	44
Moderately experienced	21
Experienced	23
Qualifications (N=67):	
None	22
Basic	40
Advanced	5

TABLE 6

SOLO AND BUDDY DIVING PRACTICES IN DIVING DROWNING INCIDENTS.

	Solo	Buddy diving	
		Separated	Not separated
Snorkeling (N=59)	26	10	23
Scuba (N=120)	36	58	26

TABLE 7

ACTIONS OF DIVING BUDDIES FOLLOWING AN IMMERSION INCIDENT IN SNORKELLERS AND SCUBA DIVERS

	Scuba (N=55)	Snorkelling (N=32)
Buddy breathe	5	Not applicable
Deep water rescue	2	Not applicable
Surface rescue	26	23
Attempted rescue		2
Search	10	4
Alert nearby boats/people	10	3
Self-care	2	

separated underwater from an instructor-led group in poor visibility and never surfaced.

The average depth recorded for 27 snorkellers was 5 m, range – surface to 24 m. The surface or tidal conditions were recorded in 43 snorkel accidents, with eight snorkellers getting into difficulty in currents or choppy and rough seas.

SCUBA DIVING PRACTICES

All the scuba fatalities occurred whilst diving on air. The presence or otherwise of other scuba divers was recorded in 120 scuba deaths. Solo diving and buddy separation were common, occurring in 78% of scuba drownings (Table 6). Whether panic was a factor was either unknown (and therefore only surmisable) or only recorded in a minority of cases. Panic was thought to have contributed in 30 of 44 scuba deaths (68%) in which the diver's state was noted.

In four divers the weights could not have been released because of incorrect assembly of the equipment, whilst four divers were considerably over-weighted. The buoyancy compensator was not connected to an air supply in three divers whilst the inlet/exhaust hose was tangled in a catch bag in one case. Two divers were thought to have removed their breathing equipment to get into narrow cracks or holes for crayfish. Two divers suffered convulsive-like loss of consciousness (possibly due to acute CNS oxygen toxicity) on deep air dives at 55 and 80 m in separate incidents.

Five scuba divers died in wrecks, including a double fatality. One diver surfaced in a shipping lane and was hit by a powerboatsuicide soon after related to the accident. Two surface tethered divers became entangled in their lifelines in strong current conditions and could not be retrieved. One of these drownings highlights the often multi-factorial nature of diving accidents, in that the diver was inexperienced in the working environment, was reported to be cold, was diving in a strong current, became entangled and almost certainly panicked. At autopsy he was also found to have previously unrecognised severe coronary artery disease.

The actions of the buddy were recorded in 88 accidents and are summarised in Table 7. Excluding the two double fatality dives where no details are known, no buddies died attempting to rescue or search for the victim, though it is known that five suffered decompression illness requiring recompression, one was near-drowned and one committed suicide soon afterwards. A non-diving rescuer drowned when his dinghy capsized whilst he was trying to assist a snorkeller.

There is a complex, often unknown inter-relationship between the air supply, ditching of weights and use of buoyancy devices (BC) in any scuba diving incident. Because of this complexity all recorded combinations of these three factors as known from the coroner's reports are listed in Table 8. The weight belt was ditched by only seven divers and only ten inflated their BC. Seven divers were recorded as not wearing a BC. The contents of 94 cylinders were checked. Forty-six divers still had a useable air supply in their cylinder whilst 48 were probably out of air.

TABLE 8

THE RELATIONSHIP BETWEEN INFLATION OF A BUOYANCY DEVICE, DITCHING OF WEIGHTS AND LOSS OF AIR SUPPLY IN 106 SCUBA DIVING DROWNINGS

BC inflated	Weight belt dropped	Out of Air	Number
Unknown	Unknown	No	5
Unknown	No	Unknown	2
Unknown	Yes	No	1
Unknown	No	No	4
Unknown	Yes	Yes	1
Unknown	Unknown	Yes	9
No	Unknown	Unknown	1
No	Yes	Unknown	1
No	No	Unknown	5
No	Unknown	No	2
No	No	No	23
No	No	Yes	32
No	Yes	No	1
No	Yes	Yes	1
Yes	No	Yes	2
Yes	Yes	Yes	1
Yes	No	No	3
Yes	No	Unknown	1
Yes	Unknown	No	3
No BC	No	No	3
No BC	No	Yes	1
No BC	No	Unknown	1
No BC	Yes	No	1
No BC	Unknown	Yes	1

Inflated	**10**	**Yes**	**7**	**Yes**	**48**
Not inflated	**66**	**No**	**76**	**No**	**46**
No BC	**7**				

TABLE 9

EQUIPMENT FAULTS IDENTIFIED FROM EXPERT EXAMINATION OF 75 SCUBA DIVERS' EQUIPMENT FOLLOWING DROWNING

No Faults	61
Equipment Faults	
Buoyancy device	8
Regulator	5
Contents gauge	2
Pillar valve	1
Air contamination	1
Mask	1
Unknown	1
Total	**19 faults in 14 divers**

EQUIPMENT

All the diving equipment functioned correctly in 61 of 75 cases for which an equipment report was available. The faults identified in the other 14 divers' equipment are listed in Table 9.

SNORKEL DIVING PRACTICES

Snorkellers' experience or training, aspects of diving practice, the equipment being worn or problems with equipment were often poorly documented. However, solo diving or buddy separation were noted in nearly two-thirds (Table 6). At least 21 were not wearing fins and several were grossly over-weighted. Five snorkels became entangled in nets or ropes, one suffered a severe mask squeeze while diving to clear an anchor and one was hit by a powerboat whilst swimming on the surface. Eight snorkellers were believed to have lost consciousness from breath-hold hypoxia. Five people with epilepsy probably had fits in the water.

RESCUE AND RESUSCITATION

Twenty eight scuba divers surfaced in distress and either disappeared again (18) or lost consciousness and were recovered on the surface (10).

The bodies of 173 divers were recovered. The time to recovery varied from immediate rescue, including two deep-water rescues from over 50 m, to months later. Basic Life Support (BLS) was provided in 78 cases (Table 10).

AUTOPSY FINDINGS

Coroner's autopsies were carried out on 169 bodies. Two snorkellers who were recovered promptly from the surface and received bystander BLS and two scuba divers whose bodies were not recovered for some time after the event did not undergo autopsy.

In scuba divers who were recovered within 24 hours of their deaths, 49 of the 94 autopsies were performed in accordance with or close to the RCPA guidelines. In 27 of these cases (55%) cerebral arterial gas embolism (CAGE) was a contributory cause of death. In the remaining 45 autopsies, whilst possible CAGE was mentioned in a few cases, it was not listed as a contributory cause of death at all (Table 11). Of the 28 divers who surfaced in distress, only nine had a correctly conducted autopsy, of whom six had CAGE, one had severe coronary artery disease and two were out of air with no other cause of drowning.

Blood alcohol levels were measured in 72 divers whose bodies were recovered within 24 hours and were positive in five of 24 snorkellers (21%) and four of 48 scuba divers (8.3%). In three drownings alcohol was a possible

TABLE 10

RECOVERY OF DIVERS' BODIES AND THE PROVISION OF ON-SITE RESUSCITATION.

Recovery (N=184)	Snorkel	Scuba
Surface	24	33
Surface-delayed	12	13
Underwater	6	16
Underwater-delayed	12	47
Unreported	6	4
No body	1	10
Basic Life Support given	36	52

TABLE 11

PERFORMANCE OF AUTOPSIES ACCORDING TO RCPA GUIDELINES AND THE DIAGNOSIS OF CEREBRAL ARTERIAL GAS EMBOLISM(CAGE)

Procedure	Snorkel	Scuba
Correct	52	49
		CAGE 27
Incorrect	Not applicable	45
		CAGE unknown
Late	6	17
No autopsy	2	2
No body	1	10

contributing cause of death. One scuba diver, a known user, tested positive for pethidine (Demerol).

A number of medical conditions were either identified at autopsy or were known pre-existing conditions. Medical conditions were considered to be contributory to death in 14 of 58 (24%) snorkellers and 23 of 113 (20%) scuba divers. The deaths of three divers with a known history of paroxysmal supraventricular tachycardia were attributed to sudden onset arrhythmias. Seven scuba divers with previously known medical conditions contributing to death had diving qualifications and therefore must have undergone a diving medical at some time. Their conditions included rheumatic valvular heart disease, paroxysmal tachyarrhythmia, coronary artery disease, alcoholism (positive for alcohol at time of death), severe schizophrenia and gross obesity. The medical conditions identified are listed in Table 12.

TABLE 12

MEDICAL CONDITIONS KNOWN OR IDENTIFIED AT AUTOPSY IN DROWNED SNORKELLERS AND SCUBA DIVERS. THOSE BELIEVED TO HAVE CONTRIBUTED TO THE DEATH ARE SHOWN IN BRACKETS.

Medical Conditions	Snorkel		Scuba	
Asthma	5	(2)	5	(4)
Other pulmonary (eg. adhesions)	1	(0)	5	(3)
Coronary artery disease	3	(3)	8	(7)
Other cardiac (eg. myocarditis, arrhythmias)	6	(2)	4	(3)
Epilepsy	5	(5)	4	(4)
Gross obesity	3	(1+?2)	4	(1+?1)
Diabetes	0		1	(0)
Severe psychosis	0		2	(?1)
Alcohol	5	(1)	4	(1+?1)

Discussion

Epidemiological studies of sports injury may be of three broad types. Descriptive studies, such as case-series or cross-sectional reports, are concerned with quantifying the occurrence of injury. These do not provide incidence rates, but do give information on the nature and circumstances of injury and may identify risk factors for injury. Both Project Stickybeak[6,7] and the present report are case-series studies.

Analytic studies are used to evaluate the role of potential risk factors in the causation of injury, and may provide some information on incidence. Examples are CAGE in free ascent training and a study on mortality and morbidity at Stoney Cove in England.[8,9] The third type are interventional studies, such as randomised controlled trials, that might evaluate the efficacy of preventative measures in sports injury. We know of no case-controlled or interventional studies in the diving literature.

The methodology used for the IPRU database, linking NZHIS and WSNZ data, has been described in detail elsewhere [2,3]. Classification of death according to a single cause such as drowning has the potential to miss some water related fatalities coded to other causes of death such as blunt trauma and heart disease. As a result, total diving related deaths are likely to be underestimated, since deaths in divers that do not include a reference to drowning in the NZHIS data and not identified by WSNZ as drowning would not have been included in the IPRU database. One of the authors (MD), for instance, is aware of two scuba fatalities in the

1980's documented as due to acute myocardial infarction not in the database. These difficulties have been discussed previously in relation to the Project Stickybeak database.[6]

The overall drowning rate in NZ over 1980-1994 was 4.4 per 100,000 at risk compared to 2.0 per 100,000 (recalculated from the original data) in Australia for 1992-97.[2,10] Australia and the USA have similar rates.[13] Thus there is clear evidence that New Zealand compares poorly internationally in its overall unintentional drowning statistics. Is the same true of diving activities? Unlike overall population statistics, we have only a poor understanding of the size of the population at risk.

All diving accidents requiring hospitalisation from Stoney Cove, the largest inland diving centre in England where all divers must register before diving, are taken to Leicester Royal Infirmary. Over a 5 year period, 1992-1996, there were 25 accidents & seven fatalities in 238,501 divers giving a fatality rate of 2.9 per 100,000 scuba divers per year.[9] The Professional Association of Diving Instructors (PADI) reported that over the five years 1989-1993 the fatality rate in PADI scuba instructors internationally averaged 1.8 per 100,000 scuba divers per year.[1] Other PADI data from Australia and Japan quoted by Monaghan suggested a much higher fatality rate of 20-30 per 100,000 "active divers".[12]

Throughout most of the 1970s and 1980s, the National Underwater Accident Data Center (NUADC), University of Rhode Island, reported data from the USA, which by 1987 amounted to more than 2600 diving fatalities.[13] Based on research on the "active diver" population funded by the Diving Equipment Manufacturers Association (DEMA) and NUADC's own figures, the estimate for the 1987 USA diving fatality rate was 3.22 to 4.14 per 100,000 divers per year. This estimate has been questioned by Monaghan who argued, based on his own analysis of the same DEMA research, that the numbers of divers in the USA used to calculate these rates were overestimated by three to five times.[14]

Turning to NZ, there are no nationwide data on the number of active scuba divers or snorkellers. However, PADI and Scuba Schools International who between them have about 95% of the diver training market in NZ issued 18,387 scuba certificates at all levels (of which there are many) in 1999 and 22,772 in 2000 (Nimb and Scappens, unpublished data). Of these, 6,789 and 8,629 were entry level certifications. DEMA estimated there is a drop-out rate of 80% for new divers in any one year and 10% per year for experienced divers (quoted by Monaghan),[14] while there is an unknown minority of active but unqualified divers. Given these figures, industry estimates of 100,000 active divers per year in NZ may be too high and using Monaghan's model would approximately halve this figure. Assuming, however, 100,000 is a close estimate then it suggests a fatality rate in NZ of at least 5.8 per 100,000 scuba divers per year during 1996-2000.

New Zealand diving fatalities have been reported in the past. Lewis reviewed 28 deaths over the period 1961-1973 using similar methodology to our own.[15] There were 40 deaths between 1974 and 1979 recorded by the New Zealand Underwater Association Accident Reporter (Fraundorfer, unpublished data). All but two of the cases for the period 1981-1984 published by Walker match with the present database,[16,17] and these and other cases to 1987 formed part of the analysis of 100 consecutive scuba diving fatalities in Australia and New Zealand analysed by Edmonds and Walker.[6,8]

The age and sex distributions are similar to other surveys, particularly from the US.[13,19] So far, there has been no shift upwards in the age pattern of NZ divers drownings as has been suggested elsewhere.[6,19] Far fewer women snorkellers die in NZ and the USA[13] compared with Australia where 25% of drowned snorkellers were women.[20] The number and pattern of accidents in Australia is influenced by the large numbers of more elderly tourists snorkelling on the Great Barrier Reef, who account for half the fatalities.[20]

Several factors appear to contribute to the relatively higher overall drowning rates of men over women, including elevated risk for exposure, risk taking and alcohol use.[21] Over the past twenty years the proportion of women entering scuba diving in NZ has risen from less than 10% to 20-25% (various sources, unpublished data). Therefore, deaths in women divers (14.6% of the total) may be in keeping with this participation level.

Ethnicity coding in NZ is complex and inconsistent. Population statistics such as census reports, for instance, may not be based on the same criteria as used in NZHIS and this makes rate calculations problematic. In the 1996 census Maori represented 13 % of the total population aged 16-65. It would therefore seem that Maori are over-represented in diving drowning statistics as they are in the overall drowning statistics. However, adult Maori may be more likely to participate in seafood gathering activities. Coastal Maori communities need to focus, amongst other things, on snorkelling skills in their water safety education programs.

The lack of proper equipment and training in snorkellers was very evident even from the sketchy reports available. A particular feature of snorkelling fatalities in both NZ and Australia is the high proportion of victims not wearing fins.[7,20]

Solo diving, buddy separation, running out of air, lack of proper training and failure to carry out fundamental emergency procedures all figure highly in this report as in all others in the literature.[5,7,13] Reading eye witness reports of drowning accidents leaves one in agreement with Edmonds and Walker, "the real tragedy of this survey was that it shows that the lessons and teachings of yesterday are still not sufficiently appreciated today".[6]

Experienced, trained divers constituted a small proportion of scuba fatalities in NZ. PADI's NZ certification data (Nimb, personal communication) shows that the proportion of divers undertaking training beyond that of the basic course is steadily increasing. Whilst entry-level certifications rose 12% from 1996 to 2000, those for further training rose by 124%. The training agencies have mounted enthusiastic marketing campaigns in recent years to attract divers to undertake further scuba training beyond the basic level. Combined with improved standards of teaching this may help to explain why the annual fatality rates of today are no greater than 20 years ago despite more divers reportedly participating in the sport. Monaghan has questioned these conclusions.[14] Using the industry's own data he claimed there are "fewer divers, fewer dives and greater risk of fatalities" than used by the industry.

The safety of sport diving should not be over-emphasised. This is an adventure sport that requires a strong focus on risk avoidance and emergency procedures training from the very start. It is unclear from international studies of drowning statistics whether training in water safety skills reduces the risk of drowning overall, but it is probably important for diving, both snorkelling and scuba, activities. Diver training agencies may also need to provide more training in snorkelling skills in their entry-level programs.

The large number of untrained divers featuring in all surveys of diving fatalities indicate a possible need for legislation to be considered as has been enacted in Queensland. Somewhat like the drinking age laws, it would seem logical to make it an offence to sell diving equipment to or to fill scuba tanks for individuals without diving certification or on a validated training course. The retail diving industry has always been opposed to such legislation but has never adopted voluntary regulation of this type on a consistent basis.

Blood alcohol levels were recorded in only about a third of cases in a study of 320 NZ drownings in 1992-94 and were positive for 50% of the incidnets.[22] In NZ divers in the present series, 41% had a blood alcohol level measured and this was positive in only 15%, in few of whom was it thought contributory to the accident. Therefore, although some divers still drink whilst diving it does not appear to be as extensive a problem as in other drowning situations. However, blood alcohol levels should always be taken in all drowning victims at autopsy.

The quality of autopsy procedures in NZ divers was as variable as has been reported for other countries.[6,13] Guidelines have been promulgated in many countries but continue not to be followed by many coroner's pathologists. Improved quality assurance in this area is essential for the accurate diagnosis of CAGE.

In addition, the police records were often deficient in essential information. The diving equipment is supposed to be secured and professionally examined in all cases but this did not occur in one third of deaths. Diving accidents are relatively uncommon compared to other causes of death so coroners, police and pathologists may benefit from improved education in the handling of diving deaths. The diving industry and SPUMS could contribute to this by promulgating diving accident report forms to be used in all unintentional diving related deaths. Existing formats (eg. DAN forms) may suffice in a modified form.

Fitness for diving remains a controversial issue. Lack of water fitness and lack of skills cannot easily be assessed by the examining physician, but were clearly a contributing factor to many drownings. That a fifth to a quarter of the divers in this series had a contributing medical condition is of considerable concern. Many were unqualified and therefore had not undergone medical assessment but others must have had examinations, with conditions present that should have excluded the candidate from diving. These issues were discussed at length by Edmonds and Walker.[6] They concluded a decade ago that medical standards for diving were not being appropriately applied, and this study indicates this is still the case.

Asthma is a specific example. There is a current view that it may be safe to relax standards for people with stable asthma despite no good data to support this stance. Six divers with asthma in this series probably died from their disease. These preventable deaths would seem to support the views of Edmonds and others that take a prescriptive attitude to this disease.[23] Clearly a prescriptive approach is also correct for epilepsy, and better protocols are needed for screening in older divers with coronary artery disease. DAN reported that in 1998, the last year for which they have reasonably complete figures, 11% of USA scuba fatalities were insulin dependent diabetics.[19] Thorough medical assessment of entry-level divers by trained physicians applying current knowledge in an informed manner is clearly indicated.

Summary

Scuba diving and snorkelling contributed significantly to water sports drownings in NZ.

1 The scuba diving drowning rate in NZ was at least 5.8 per 100,000 divers per year
2 All but one of the snorkelling deaths were in men
3 Maori are over-represented on a population basis, especially in the snorkelling deaths, for reasons that are not apparent from these data
4 The majority of scuba divers who have drowned lacked experience and/or diving training
5 Poor water skills and poor dive planning (especially running out of air) were common risk factors
6 Emergency drills were frequently omitted
7 Autopsy procedures in many divers were not performed correctly and a blood alcohol level was taken in less than half the cases

8 Where the autopsy was conducted correctly, CAGE was a contributory cause of death in at least half the scuba divers

9 A fifth of scuba divers and a quarter of snorkellers drowning had an underlying contributory medical condition such as asthma, epilepsy or ischaemic heart or other cardiovascular disease.

Summary

The authors believe that this examination of two decades of diving drownings should lead to action on some or all of the following:

1 Promotion of competent medical assessment of all entry level divers

2 Establishment of snorkel training programs for the Maori community

3 Improvement in the teaching of snorkelling techniques and enhance training in emergency procedures in entry-level scuba diving courses

4 Standardisation of the reporting of diving related deaths and autopsy procedures in scuba divers

5 Conduct a study in New Zealand to establish the number of active divers so that incidence rates may be more accurately known

6 Consideration of legislation to restrict the purchase of diving equipment or compressed gases for scuba by unqualified divers

7 Public endorsement by SPUMS of the diving training agencies' current approach to a continuum of training in scuba diving and of water safety programs such as WSNZ/NZ Underwater's "Dive Safe" and "Mini-Dippers".

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Conflicts of interest

None

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THE WORLD AS IT IS

THE DIVERS ALERT NETWORK TODAY

John Lippmann

Key Words
Oxygen, rescue, research, training, transport, treatment.

The Divers Alert Network (DAN) is a not-for-profit organisation which was formed in 1980 in the United States to assist in the treatment of underwater diving accidents by providing a 24-hour emergency hotline. When government funding for this service evaporated, DAN established a membership program. For a small payment, subscribers received a diving accident manual and a regular newsletter, *Alert Diver*, dealing with various issues of diving safety.

As the popularity of diving and dive travel increased, numerous situations arose when divers with symptoms of DCI were stranded in remote locations without the funds required to pay for an expensive evacuation to a recompression facility. In the early 1980s, dive insurance was very difficult to obtain and DAN lobbied hard to enable divers to get access to appropriate insurance coverage. As DAN grew, it was able to provide its members with a worldwide evacuation service and diving injury treatment insurance.

DAN organisations have been established in several regions. These include DAN Europe, DAN Japan, DAN S.E. Asia-Pacific (SEAP) and DAN Southern Africa. DAN SEAP was established in 1994, and provides services to most countries in the Asia-Pacific Region. Together, the DAN organisations form the International Divers Alert Network (IDAN). These autonomous organisations work co-operatively towards a common goal of improving diving safety and treatment services worldwide. At the end of 2001, there were more than 205,000 DAN members worldwide.

The aims of DAN SEAP include improving diver safety through education, providing evacuation and insurance services for injured divers, improving the management of diving and other accidents by the provision of appropriate first aid training, oxygen provider training and equipment, support for regional diving emergency hotlines, diving accident data collection and research.

Improvement of dive safety through education

All DAN members receive a copy of Alert Diver, a regular journal dealing with various aspects of dive safety and health. The various DAN entities also conduct workshops and seminars relating to safety issues, and have large websites with a variety of useful health and safety information on them.

Provision of evacuation and insurance services

All DAN SEAP members automatically become eligible for emergency medical evacuation cover, for up to US$100,000, for diving and non-diving emergencies that occur more than 80 km from home. Members also have access to a variety of economical dive injury treatment insurance plans, and in some cases, personal accident plans.

First Aid Training

DAN SEAP has also introduced some general and workplace first aid programs that teach participants the skills required to provide first aid in the home or the workplace as well as in the field. Topics include bleeding, respiratory and cardiac emergencies, fractures, soft tissue injuries, head injuries, diabetes, epilepsy and convulsions, envenomation, poisoning, and many others.

The courses, which are equivalent to those offered by Red Cross and St John, are suitable for divers and non-divers alike. They have been accredited in Australia under the National Training Recognition Scheme, as well as by certain workplace authorities.

DAN has also introduced short programs on the use of Automated External Defibrillators (AEDs) and on marine envenomation.

Oxygen first aid training and equipment

DAN has been instrumental in the development of certain types of oxygen units which are designed to provide a simple, safe and effective means of providing oxygen to injured divers. Additionally, armed with the knowledge of the importance of oxygen in the first aid management for decompression illness (DCI), DAN has created a highly effective and successful oxygen provider program designed specifically for divers.

This program covers the training of oxygen providers, divers able to administer oxygen to casualties, and of instructors who run local courses for oxygen providers.

DAN OXYGEN PROVIDER COURSE

This program is suitable for divers of all levels, from novice to instructor, and is now the most respected oxygen program for divers worldwide with more than 132,000 oxygen providers and 16,500 oxygen instructors trained from its inception to the end of 2001.

It consists of a 4-hour oxygen provider module, supported by an additional resuscitation module, where required. Topics covered include diving accident recognition, the benefits of oxygen provision, precautions and safety guidelines and oxygen provision to both responsive and unresponsive victims.

Participants are certified in the use of the DAN Oxygen Unit, or an equivalent system. In addition, there are several extension modules for divers who require further training in devices such as the MTV100, REMO$_2$ and bag-valve-mask systems.

Regional diving emergency hotlines

IDAN supports a network of diving emergency hotlines throughout the world. These hotlines are available to all divers, whether DAN members or not.

DAN SEAP provides full funding to the DES Australia hotline and to the hotlines in New Zealand, the Philippines and Malaysia.

Diving accident data collection and research

Another important function of the Network is collecting and analysing data on diving accidents and fatalities to improve the understanding of the causes of such accidents and to develop better treatment methods.

DAN entities produce regular reports on dive accident and fatalities. DAN SEAP published a comprehensive report on the Australian diving deaths from 1972-1993.[1]

It is soon to release a compilation of the Australian fatalities from 1994-1998. In addition, DAN SEAP is now actively involved in the collection of dive fatality data throughout the region and aims to produce more comprehensive regional accident reports in the future.

IDAN has been involved in extensive research projects that include flying after diving, ascent rates, diabetes and diving, project dive exploration (downloaded dive computer profiles), post-diving Doppler studies, PFO and diving, and many more.

DAN entities are also involved with providing assistance to remote recompression chambers, donating equipment and providing expertise where appropriate and in various other ways to help to further the safety of the magnificent sport we are privileged to participate in.

We remain very grateful to all those who support us in our mission.

The address of the Central Office of DAN S.E. Asia-Pacific is PO Box 384, Ashburton, Victoria 3147, Australia. Phone +61-(0)3-9886-9166. Fax +61-(0)3-9886-9155, E-mail <info@danseap.org>. Web site <www.danseap.org>

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SPUMS NOTICES

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

Requirements for candidates

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

1 The candidate must be a financial member of the Society.

2 The candidate must supply evidence of satisfactory completion of examined courses in both Basic and Advanced Course in Diving and Hyperbaric Medicine at an approved institution.

3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months full time training in an approved Hyperbaric Medicine Unit.

4 The candidate must submit a written research proposal in a standard format for approval by the Education Officer before commencing their research project.

5 The candidate must produce, to the satisfaction of the Education Officer, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

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

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

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

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

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

Key Words
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The dates are September 4th to 8th 2002.

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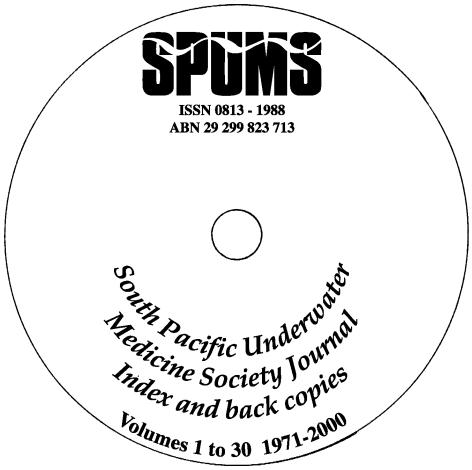
NOW AVAILABLE

The South Pacific Underwater Medicine Society has produced a CD, readable by at least Windows and Macintosh computers, containing every issue of the Society's Newsletter and Journals as Adobe .pdf documents, from the first issue in May 1971 until and including December 2000. All that is needed to read and print these documents is Adobe Acrobat Reader (version 3 or later) which can be downloaded free from the Adobe web site.



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The CD also contains the index for the South Pacific Underwater Medicine Society Journal. This runs from 1971 (Volume 1) to December 2000 (Volume 30 No. 4).

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WORLD CONGRESS OF DROWNING, 2002

To be held in Amsterdam
on 26, 27 and 28 June 2002

Breath-hold, scuba and hose diving

Recreational scuba diving is recognised as a safe sporting activity. There are relatively few accidents compared with other sports although, when an accident does occur in the water, it happens in a very unforgiving environment. What might be an insignificant incident at the surface can start a sequence of events that quickly escalates to become life threatening. The environment in which this happens is also the probable reason why up to some 60 per cent of in-water diving fatalities meet their deaths by drowning. Drowning is the *mode* of their deaths, but not the cause. In examining the *causes* of drowning in divers, one must look at the way in which people dive. To reduce the risk of drowning in divers one must address not only their in-water procedures but also basic issues such as fitness, training and equipment.

For this reason the diving community has been invited to participate in the **World Congress of Drowning** to be held in Amsterdam on 26, 27 and 28 June 2002. This conference was initiated by *The Society to Rescue Persons from Drowning* which was founded in the Netherlands in 1767.

Partners in this venture include the International Federation of Red Cross and Red Crescent, ILS (International Life Saving) and DAN.

The aims of the Congress are

to make recommendations on the prevention, rescue and treatment of drowning victims;
to stimulate and facilitate initiatives to further promote the prevention of drowning;
to reduce the number of drowning victims;
to improve the survival rate and outcome of drowning victims.

"**Breath-hold, scuba and hose diving**" (Chairman: David Elliott, UK) is thus just one of around 10 task forces convened to review particular aspects of this vast topic.

Other task forces and Chairpersons include

Epidemiology	Christine Branche, CDC, Atlanta.
Rescue	Chris Brewster, International Life Saving Federation, USA.
Resuscitation	Paul Pepe, Emergency Medicine, University of Texas.
Hospital treatment	Jean Louis Vincent, Erasmus Hospital, Brussels.
Immersion hypothermia	Beat Walpoth, University Hospital, Insel, Switzerland
Brain	David Warner, Duke University Medical Center, USA.

Each task force has an international group of experts in the appropriate specialities.

The diving task force covers the hazards associated with all types of diving. This includes recreational diving of every variety. It also covers subsistence fishermen-divers in the third world, most of whom have inadequate equipment and no proper training and who have an unknown rate of in-water incidents. The other large group is military and working divers who follow procedures that for them should make the risk of drowning negligible.

A number of drowning fatalities in divers occurs among divers who may have made an avoidable error or who may have been subjected to one. After reviewing such accidents the task force has prepared draft recommendations and reviewed those submitted by others. The following topics are among the questions that they consider deserve discussion at the World Congress.

Should diver certification last a lifetime, or is there a need for re-certification after a few years?

What changes can be recommended in the training of divers and diving instructors that might enhance diving safety?

Should a once-only medical declaration that was made before training potentially last for a lifetime?

Is there a minimum age for diving as one of a buddy-pair?

Should there be a greater emphasis at all levels of recreational diver training on the causation of known in-water fatalities?

Visit the web site (www.drowning.nl) for more details about the Congress, its task forces and the arrangements. Some 60 task force members from 20 nations have prepared formal presentations and reviewed the many recommendations for the Congress. Each task force has a summary of its proposed agenda, each will have a plenary session for all and then a number of sessions on selected diving topics.

Look through the recommendations in the diving section. Because they come from a wide range of sources, some appear worthwhile but others may not be universally acceptable. These will be discussed and, where appropriate, their implementation will be reviewed at the Congress in Amsterdam, 2002.

You can also write for more information to the
World Congress of Diving 2002 Secretariat
c/o Consumer Safety Institute
PO Box 75169, 1070 AD Amsterdam, The Netherlands.
or e-mail <Secretariat@drowning.nl>

Key Words

Breathhold diving, drowning, meeting, occupational diving, recreational diving.

XIV INTERNATIONAL CONGRESS ON HYPERBARIC MEDICINE

October 2nd - 5th 2002
St Francis Hotel
Union Square
San Francisco, California, USA

CALL FOR ABSTRACTS

Abstracts of communication are invited from individuals who are involved in hyperbaric medicine. Abstracts submitted for the 2002 ICHM Meeting should not have been published nor presented before international meetings prior to this meeting. Deadline for abstract submittal is 2002/4/1. Specific instructions for preparing abstracts can be found at the ICHM web page <www.ichm.net>.

E-mail transmission of Abstract Submission Form and abstract should be sent to <ichm@milx.net>. On subject line type: ICHM Abstrac*t*. Persons who do not have e-mail may send a camera-ready hard copy and diskette to:

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Both oral and poster sessions are available for presentations, which will be made in English.

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Accepted abstracts of those who are registered at the meeting will be published in the usual manner in the Program/Abstract book regardless of whether the abstract will be presented in a poster or oral session. Final papers for the Proceedings of the XIV ICHM must be turned in at the Registration Desk at the Congress.

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LETTERS TO THE EDITORS

ARE SOME JELLYFISH TOXINS HEAT LABILE?

Australian Venom Research Unit
Department of Pharmacology, University of Melbourne
Victoria 3010, Australia
2002/2/12

Dear Editor

Thank you for your donation to the Australian Venom Research Unit in memory of our friend, and my mentor, Struan Sutherland. As you know venom research is not well supported by the Commonwealth Government, who are prepared to leave almost all the costs to the Government of Victoria. All donations are very welcome.

I was aware that Struan was a Guest Speaker, with Carl Edmonds, at the 1984 South Pacific Underwater Medicine Society Annual Scientific Meeting held on Bandos Island in the Maldives. I remember being told that he talked about hydroponics as well as toxins.

Thank you for the reprint you sent me, *Are some jellyfish toxins heat labile?*[1] I enclose a copy of a paper published last year in the Hawaii Medical Journal by Craig S Thomas and others which investigated the effects of hot, cold and ambient temperature pack applied to box jellyfish stings.[2] The common box jellyfish in Hawaiian waters is *Carybdes alata*, some 75 to 100 mm in height and about 50 mm wide with tentacles which can be 0.75 m long. These jellyfish appear on the beach around the last quarter of the lunar cycle and sting many, many people, over 800 on 1997/7/29!!

While conditions are different in Australian waters it might be possible for SPUMS members to pursue a similar study. The Hawaiian study showed that hot packs did reduce the pain of the stings but not to a significant degree. However Dr Taylor's personal pain relief with hot water was very effective at relieving the severe pain from Tamoya stings.

If any SPUMS members are interested in such a study I would be happy to offer advice and guidance.

Ken Winkel, Director, AVRU

References

1 Taylor G. Are some jellyfish toxins heat labile?. *SPUMS J* 2000; 30 (2); 74-75

2 Thomas CS, Scott SA, Glanis DJ and Goto RS. Box jellyfish (*Carybdea alata*) in Waikiki: Their influx cycle plus the analgesic effect of hot and cold packs on their stings to swimmers at the beach: A randomized, placebo controlled, clinical trial. *Hawaii Med J* 2001

Key Words

Injuries, marine animals, toxins.

BOOK REVIEWS

MARINE CONSERVATION FOR THE 21ST CENTURY

Oceans of Facts for US Citizens

Hilary Viders
ISBN 0-9411332-46-2 (soft cover). 1995.
Illustrated. pp 350.
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 18.95. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

The cover blurb calls this book "An essential guide", but the five pages of the contents list show it is encyclopaedic in scope. The author explains that she intends it to be a vehicle to teach marine ecology, as well as motivate people. There is certainly a large potential audience, worldwide, for an explanation of marine conservation issues in non-technical language, and obviously to understand these issues one needs to learn about the oceans and marine ecology. This book covers all this without assuming any prior knowledge of the reader. It is liberally sprinkled with good photographs that attract interest, and there are many excellent diagrams (by Ken Ibsen) that aid understanding of key issues. But what information and style is appropriate for such readers?

The six chapters on marine environments and ecological concepts, and the surprising chapter on fresh water ecosystems, present an eclectic flood of facts, from the information that water is made of hydrogen and oxygen to what acoustic tomography is, and the fact that sharks have extraordinary immune systems. Unfortunately numerous collectable facts, such as that there are 325 trillion gallons of water on our planet, appear to have displaced important issues such as the cyclic paths of major ocean currents. The style in some chapters is reminiscent of training manuals: subjects are presented as numbered lists. I find lists of "principles" such as: "Closely related to the life patterns principles is the principle of biotic communities", and "In ecology, the whole is more than the sum of its parts" is a misleading approach to teaching ecology. Technical concepts, such as "spring tides", "genus" and "photosynthesis" are printed in large capitals. Perhaps this assists in memorising and referring back to these terms, but bold type would have been less intrusive. The "environmental professionals" listed as part of the target audience will easily find gaps and errors, for example while the illustration of planetary air currents is correct the text explanation is not, and a picture of a sea anemone is described as a crinoid.

The chapters on natural and human induced stresses are comprehensive, and the 13 chapters on conservation

actions are probably the most valuable part of the book, as they explain how people can make a difference in marine conservation. But it becomes ever more obvious that the target audience is restricted to US citizens, particularly recreational divers and snorkellers.

This book has a myopic focus on what happens in the USA. Marine habitats such as kelp beds are described as if they only occur in the USA. The chapter on "The US Government in Conservation" is twice as long as the next chapter, on "International Conservation Initiatives", and laws in other nations are not mentioned. There are lists of US marine sanctuaries, each with contact details, but the rest of the planet is omitted. Significant marine parks in the world are not too numerous to cover. It is a shock to read, of one of the few non US marine parks mentioned, that: "A large marine park which has met with success is based in Canberra, Australia, on the Great Barrier Reef".

For divers and snorkellers there are useful sections on how to minimise damage by correct buoyancy control, finning, avoiding dangling equipment, etc., how to run a clean up, and other conservation activities. US divers are provided with advice on how to choose an eco-tourist trip, and a long list of organisations that provide diver field trips (all US based). There is a useful glossary, but the reference list is disappointing. It is a great handbook of information for US divers, and it may be useful for divers elsewhere, if you can go past the US focus and "fact-packed" style.

Rob Day
Department of Biology
Melbourne University

Key Words
Biology, book review, ecology, marine animals.

SUBMARINE RESEARCHES
C A Deane
With introduction by John Bevan and edited by Michael Fardell and Nigel Phillips.
ISBN 1 900496 143. 2001.
Historical Diving Society, 25 Gatton Road, Reigate, Surrey, RH2 OBH, UK. Telephone +44-(0)1737-249961.
Price £18.00. Postage and packing about £6.00 for delivery to the Australasian region.

This book is the second in a series of monographs the first of which, *A Demonstration of the Diving Engine* by Jacob Rowe, edited by Michael Fardell and Nigel Phillips, was published in 2000 and reviewed by the Journal in 2001 (31 (2): 84-85). Both books have been published as a limited edition with each copy numbered.

The original title page of *Submarine Researches*, published in 1835 reads "Submarine Researches on the wrecks of His Majesty's late ships Royal George, Boyne and others, by Mr C A Deane in his improved diving apparatus; with an introductory account of the loss of the Royal George by two of her seamen who are yet survivors of that awful catastrophe."

One can understand the words "who are yet survivors" as the Royal George sank on the 29th of August 1782, over 50 years before Deane produced his book to publicise his exhibition of "a number of valuable and curious Relics which he has recently saved from the Wrecks of His Majesty's Ships and East Indiamen; and also 20 oil painting by MEADOWS covering nearly 1400 square feet of Canvass, illustrative of the various ways in which the Apparatus has been so successfully employed".

Unfortunately the paintings have disappeared but the 19 engravings which form the last third of the book are clearly taken from the paintings. They cover many aspects of Deane's work from the Royal George on his first dive on the wreck which lay in 72 feet of water, through shallow salvage, filling a basket with recovered cargo for it to be hoisted to the surface. Anchor recovery has a number of illustrations as does structural work in docks. Another series cover the salvage of a small ship, the sloop Endeavour, from removing her cargo on the bottom, which made the sloop unstable and dangerous to work in. So she was raised by tying empty barrels to her r rigging and hull and using chains from the Endeavour to the diving boat to winch the Endeavour off the bottom. She was then moved six miles to a safer, shallower spot. Not only did Deane's team achieve this, but at the new work site they were able to fill her with empty barrels so that she floated, even though full of water, high enough to allow her to be towed by a "steam boat" from the island of Gigha some sixty miles to Campbelltown harbour where she was "hawled up" and repaired.

This book is a must for anyone interested in the history of the early years of helmet diving. It gives a fascinating insight to the way that Deane dealt with the problems of bending over when wearing an "open" helmet, one which was not attached to the exposure suit but only to a jacket going down to about the waist. If the diver put his head too low air could escape and he would drown. In none of the illustrations does the diver bend, to get low he squats or kneels.

John Bevan's erudite introduction alone is worth the purchase price. *Submarine Researches by C A Deane* can only be described as an essential purchase for a diving history buff and well worth while for the curious diver interested in the past.

John Knight

Key Words
Book review, diving operations, history, general interest.

DANGEROUS AUSTRALIAN ANIMALS
Struan Sutherland and Guy Nolch.
Hyland House Publishers.
ISBN 1 86447 076 3. Paper back. 200 pages
RRP $45.00.

Struan was the most famous of the Australian toxicologists, before his untimely death. His knowledge and humour were legendary. His text on Australian Animal Toxins was second to none in its detail and scope.

This recent publication is written for a more general audience. Nevertheless, it does include basic information on first-aid and simple medical treatments. It is divided into six main sections incorporating; marine creatures, snakes, spiders and other arachnids, insects and other arthropods, other creatures - some of which are aquatic - and first aid.

Because of my interest and expertise, I have concentrated mainly on the marine section and the aquatic animals. I would be loath to venture into the terrestrial sphere.

Firstly, one must acknowledge and respect the conversational style of presentation. It is easy to read, fascinating in its content and informative in its emphasis. The text is interspersed with many anecdotes and case reports, varying from the amusing to the horrifying. It incorporates and presents both an historical and a research perspective.

The illustrations are adequate and sufficient for the purpose of the book. The printing and the lay-out is pleasing to read and professional in appearance. The index, however, is inadequate and sometimes does not even include subjects that are in the Table of Contents.

I have only two major criticisms. The first is that the pages could have been more adequately cross-referenced within the text. For example, under sea snake envenomation, on page 44, antivenom is mentioned but no further reference is given to it although, in fact, it is accurately and fully described later, in pages 190-193, along with various other antivenoms. Similar omissions occurred throughout, when it would have been very simple to have directed the reader to an appropriate area of the text. Otherwise, how is the reader to know that extra information is available?

The second major criticism is that the work is dated. This is seen in two different aspects. Most of the animal injuries and the case reports are taken, almost verbatim, from previous publications of around a decade ago. This may not be of much relevance to those who have not read the original reports, many of which were in the very popular Australian Doctor magazine, but it is a little disappointing to not have the treatment recommendations updated. The explanation for this could well be that Struan had been so ill over the last decade, that he was unable to add his more recent and incisive thoughts to the text.

On a similar line, some of the treatments proposed are no longer considered appropriate, without considerable qualification. Thus the use of the pressure bandage immobilisation techniques for the Chironex (box jelly fish) advised in this text, have been severely questioned, if not discredited. No such doubts are expressed in the text.

But nevertheless, let us put these criticisms in perspective. This is a great little book, a book that one would be proud to have on one's shelf, and it is a delight to read. Buy it.

Carl Edmonds

Key Words
Book review, first aid, land animals, marine animals, toxins

AN ADMIRALTY DIVER'S STUDENT TRAINING NOTES
Leading Seaman W G Harper RN
A4 soft cover
Historical Diving Society, 25 Gatton Road, Reigate, Surrey, RH2 OBH, UK. Telephone +44-(0)1737-249961.
Price £10.00. Postage and packing about £6.00 for delivery to the Australasian region.

Available from the Historical Diving Society, this detailed one hundred page book will be a most interesting read for anybody with an interest in historical diving. Written from a collection of notes taken by Gerry Harper during his diving training in the Royal Navy, the notes are in three parts, basic diver training in 1941, Diver 1 training in 1944 and requalifying for the Korean War in 1951.

The first two pages deal with the subject that all Navy divers had to commit to memory. A complete list of breast rope and airline signals from diver to attendant and attendant to diver. It was interesting to note that they were the same signals I was taught in 1975.

Gerry then moves on to a detailed description of Standard Diving Dress including detailed drawings of inlet and exhaust valves. This section details how the dress is manufactured and assembled. It includes such minute detail as telling us that the corselet lanyard (used to secure the air pipe and breast rope to the corselet) is "of 1 1/4" tarred hemp and fitted with an eye splice and a cut splice, the free ends being 2'6" long".

After describing the dress, Gerry describes the air pump. Once again there is a detailed diagram of the pump plus instructions for its use. This section details the maximum depth for a diver using 1, 2 or 3 pumps plus

instruction on routine maintenance and how to check the pumps for correct volume delivery and pressure tightness.

There are also formulae for working out the efficiency and actual output of a pump if there are leaks in the system. This is essential to determine the number of revolutions per minute the handles must be wound at to provide adequate air delivery to the diver.

After the diving dress and pumps we get treated to the intricate details of Cox's bolt guns and hydrogen oxygen underwater cutting equipment. Once again I was interested to note that the equipment which I was taught to use in 1975 had very few differences from the 1941 equipment. I know that divers don't like change but this was taking things to extremes.

As well as telling us how this equipment works and how to maintain it, Gerry also tells us how to use the equipment to patch holes in ships sides and cut steel plate. There is also detailed instruction in using tubular steel to build bridges.

Of interest to the diving physicians are the sections on the physics and physiology of diving and how to use decompression tables. These sections also describe how to treat carbon dioxide poisoning and decompression sickness.

All in all this is a fascinating book for those with an interest in the subject. Readers who are not familiar with the subject matter may find the text too technical for them, but bear in mind this is a collection of training notes. Everything written down here is what the diver was expected to know to carry out his duties.

Having gone through the Royal Navy's diver training program in the 1970's using different diving equipment but the same working equipment I found it fascinating to note the similarities in equipment and procedures. I would recommend this book to diving historians, owners of Standard Diving Dress and other retired Navy divers. Others may find some of it a bit tedious, but it gives a fascinating insight to the use of Standard Diving Dress in the 1940's.

Steve Goble

Key words

Book review, equipment, history, training,

SPUMS SCIENTIFIC MEETING 2000

MEDICAL SUPPORT FOR THE SYDNEY AIRPORT LINK TUNNEL PROJECT

Michael Bennett, Jan Lehm and Peter Barr

Key Words

Decompression illness, fitness to dive, medical conditions and problems, training, treatment, tunnelling.

Abstract

In 1996 a contract was awarded to construct a tunnel connecting Sydney Airport with the rail network. The project involved driving a tunnel through soft ground over a distance of 5.5 km. Medical input involved planning compression and decompression, assessment of fitness to dive, training of workers and lock operators, health monitoring of workers and treatment of related injuries. Oxygen decompression tables were used for the first time in Australia, the UK or US, although they have been successfully employed elsewhere. Oxygen tables improve efficiency and may reduce the risk of decompression illness (DCI).

22 individuals were declared unfit to work in compressed air. Over the course of the project there were 767 entries into raised pressures (interventions), with a median of 4 workers each time, at pressures ranging from 1.75 to 5.02 bar gauge (175 to 502 kPa absolute) with an average time at the cutter head of 3 hours 2 minutes. Compression related problems included 8 cases of decompression illness. The incidence of DCI was one case every 286 man interventions (0.35%) and this problem affected 5.9% of the workers. This incidence compares favourably with that of projects using air decompression .

The combination of the Tunnel Boring Machine (TBM) and oxygen decompression, in a context of vigilant medical supervision, provided a safe working environment. We recommend this approach in the future.

Introduction

In 1996, the Transfield Bouygues Joint Venture (TBJV), an international joint venture between two companies, one Australian and one French, was awarded a contract to construct a railway tunnel connecting Sydney Airport with the existing suburban rail network. In part, the project involved the construction of a tunnel through soft ground (mainly wet sand) over a distance of 5.5 km. (Figure 1). Soft-ground tunnelling is technically difficult, and the successful bid proposed the use of a self-contained tunnel-boring machine (TBM).

The TBM utilises a rotating cutter head and retractable pressure shield designed so that intervention at the cutting face can be achieved for inspection, maintenance and repair when under pressure. Behind the cutting area and access locks, the rest of the TBM is designed to assemble the shaped, interlocking concrete elements that make up the wall of the tunnel. Thus, the TBM leaves a fully-formed tunnel in its wake, ready for fitting out for road or railway use. The cutter head used in Sydney is shown in Figure 2. The development of this technology has been summarised in course materials from the South Bank University, available on-line.[1]

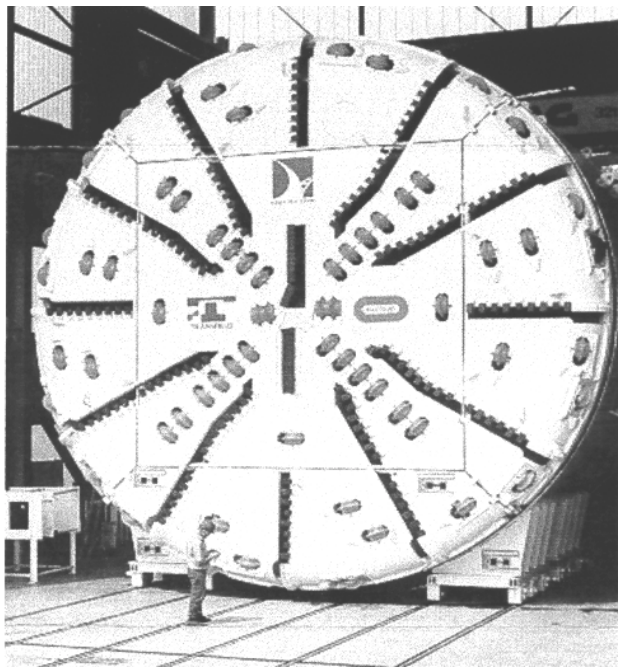

Figure 2. The cutter head and pressure shield after assembly.

While the TBM obviates the need for gangs of workers to spend long periods of time under pressure in order to excavate and build the tunnel, there is nevertheless a requirement for inspection, maintenance and repair at the cutter head under pressure. For this reason, both the local occupational health and safety organisation, Worksafe Authority NSW, and TBJV recognised the need for specialist medical support during tunnelling operations.

Medical support requirements

Operational requirements proposed the employment of approximately 80 manual labourers and engineers capable of working in a compressed air environment at any one time

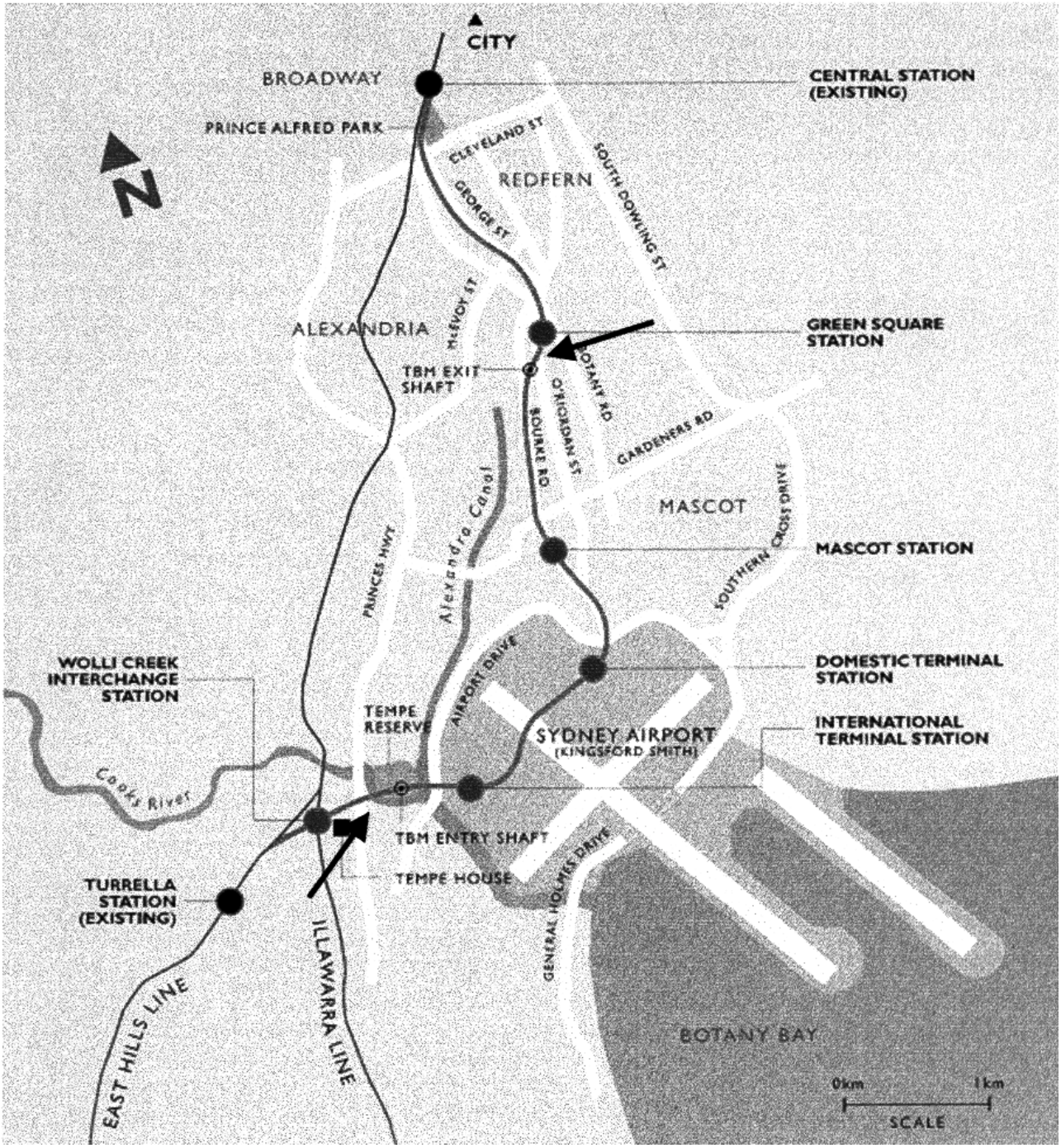

Figure 1. Course of the tunnel. Entry and exit points of TBM are indicated.

over a period of approximately three years. As there was no recent history of similar projects undertaken locally, this involved considerable planning and co-ordination with local ambulance, hospital and medical support. In addition, both workers and their supervising engineers required extensive education and practical training to work appropriately in the compressed air environment. The Prince of Wales Hospital (POWH) Department of Diving and Hyperbaric Medicine (DDHM) was approached early in the planning stages to provide medical support for the project. Wide responsibilities were involved, summarised in Table 1.

PLANNING

DDHM medical staff were required to give specialist medical input to discussions between management and Worksafe NSW. In particular, this discussion concerned the proposed implementation of the French oxygen decompression tables, developed by the French Ministry of Labour in 1992 and discussed by Le Pechon.[2,3] Oxygen had not previously been employed for decompression for compressed air workers in Australia, UK or USA, although the practice is established in Japan, Germany, Denmark and

TABLE 1

SUMMARY OF MEDICAL INVOLVEMENT IN A PROJECT INVOLVING COMPRESSED AIR EXPOSURE OF AN UNTRAINED WORKFORCE

Elements of Medical Support Required

Advise on
- appropriate compression/decompression practices
- appropriate retrieval and hospital facilities required
- specific occupational health risk management

Establish communications for
- effective and timely treatment of workplace injuries

Assess fitness of
- staff required to enter the compressed air environment

Provide
- worker training and education
- lock operator training and education
- medical advice to workers, management and Worksafe NSW
- emergency medical cover during tunnelling operations
- treatment of compression-related problems

Brazil.[3,4] The use of these tables is the subject of review by the Health and Safety Executive (HSE) in the UK. At the time of writing their report was not yet available.[5]

A full discussion of the development of oxygen decompression procedures with regard to compressed air work has been published and Kindwall strongly argues for the introduction of such schedules on the grounds of both safety and efficiency.[6,7]

The use of oxygen breathing periods during decompression facilitates nitrogen elimination and so allows shorter total decompression times. A comparison of air and oxygen decompression schedules demonstrates the significant savings in decompression time for identical workface pressure exposures (Table 2).[2] For example, a period of one hour working at 3.6 bar gauge pressure (460 kPa) will require 150 minutes total decompression time breathing air, compared with a total decompression time of 80 minutes breathing oxygen. Workers undertaking this pressure exposure (intervention) would be required to spend 47% less time in decompression if oxygen was employed.

Work site planning also involved the nature and location of first aid and compression facilities, allowable

TABLE 2

COMPARISON OF WORKING TIMES AND DECOMPRESSION SCHEDULES FOR 3.6 BAR (4.6 ATA) INTERVENTIONS WITH AIR AND OXYGEN DECOMPRESSION.
From Reference 1

AIR DECOMPRESSION SCHEDULE

Working Time	Minutes to first stop	1.5 Bar Air	1.2 Bar Air	0.9 Bar Air	0.6 Bar Air	0.3 Bar Air	Total Decompression	Total Intervention
0 hr 10	11					3	14 min	0 hr 24 min
0 hr 15	11					5	16 min	0 hr 31 min
0 hr 20	10				3	15	28 min	0 hr 48 min
0 hr 25	10				5	20	35 min	1 hr 00 min
0 hr 30	9		3	3	10	25	50 min	1 hr 20 min
0 hr 45	8		10	10	20	40	88 min	2 hr 13 min
l hr 00	7	3	20	20	35	65	140 min	3 hr 30 min

OXYGEN DECOMPRESSION SCHEDULE

Working Time	Mins to first stop	1.5 Bar Air	1.2 Bar Air	0.9 Bar Oxygen	0.6 Bar Oxygen	0.3 Bar Oxygen	Total Decompression	Total Intervention
0 hr 20	10				5	5	20 min	0 hr 40 min
0 hr 25	9			5	5	5	24 min	0 hr 49 min
0 hr 30	9			5	5	10	29 min	0 hr 59 min
0 hr 45	8		3	10	10	15	46 min	1 hr 31 min
1 hr 00	7	3	10	20	20	20	80 min	2 hr 20 min

environmental limits within the access locks and workface, efficient and reliable communication and the minimum time intervals to be mandated before repeat occupational compression.

The planning stage also required liaison with NSW Ambulance and Sydney Aeromedical Retrieval Service with regard to evacuation of serious casualties from compressed air. Particular emphasis was placed on the ability of emergency medical systems within the organisation to safely move an unconscious worker from the tunnel face at pressure, through a period of decompression and to hospital in a timely manner.

ASSESSMENT OF FITNESS TO WORK IN COMPRESSED AIR

Each potential compressed air worker required a detailed examination prior to medical clearance to work in the compressed air environment. This included manual labourers, skilled tradesmen, supervising engineers and safety officers. While detailed standards exist regarding the examination of commercial diving candidates, no specific Australian Standards document exists to protect compressed air workers, although such a document is in an advanced stage of preparation at the time of writing.[8]

We based our examination on existing occupational diving medical standards, modified to allow for the fact these individuals were not planning to be immersed in water during their exposure to high environmental pressure. Routine investigations included an exercise step-test, spirometry, tympanometry, audiometry, chest X-ray and long-bone survey. On satisfactory completion of the medical, the workers were exposed to a short test compression in our main therapeutic chamber in order to learn ear clearing techniques and experience the sensations of compression in a confined space. After successful exposure to pressure, all workers were exposed to a short excursion to 600 kPa at the workplace to further confirm suitability for the planned work. Continued fitness for compressed air work was ensured by regular assessments at six-monthly intervals.

During the course of the project, we examined 162 individuals of whom 142 were passed fit (88%). Of these 136 were actually exposed to compressed air at the workplace. This high pass rate compares to that reported by Lam in Hong Kong of 69% and may reflect an initial questionnaire taken at the workplace to exclude (prior to examination) individuals with active asthma or chronic middle ear conditions.[9] Two hundred and sixty six-monthly reviews were performed. Reasons for failure of the fitness assessment are summarised in Table 3. Eight workers were sent for specialist opinion (3 ENT, 2 cardiac, 1 respiratory, 1 haematology, 1 psychologist) at their own request in order to pursue the ability to work in compressed air, however, all were ultimately rejected for this work environment.

TABLE 3

REASONS FOR CANDIDATES BEING DECLARED UNFIT FOR COMPRESSED AIR WORK
Two workers had multiple problems

Reason Declared Unfit	Numbers
Unable to auto-inflate middle ear on increasing pressure	11
Asthmatic or other pulmonary disorder	5
Psychologically unsuited to chamber environment	2
Miscellaneous	4
Total	**22**

WORKER AND LOCK OPERATOR TRAINING AND EDUCATION

All medically fit workers attended a one week course on the practical implications of work in compressed air. This was conducted by an Australian Diver Accreditation Scheme approved operator (Descend Training Centre, Albury, NSW) and included familiarisation with the compressed air environment, the conduct of safe practice in that environment and the medical and health aspects of compressed air work. As part of this course, DDHM medical staff conducted sessions in common pressure-related illness (in particular the symptoms and signs of middle ear barotrauma (MEBT) and DCI, basic first aid, and the emergency management of DCI.

Thirty five workers, including one female widely thought to be the only female trained as a compressed air tunnel worker in Australia, were trained for a further week as lock operators on the TBM. Medically, they received more detailed instruction on the early diagnosis and management of MEBT and DCI and the mechanisms in place to seek emergency medical help. We relied heavily on instructing these operators to follow a standard diagnostic algorithm and to communicate the findings to the medical officer on duty for such emergencies.

MEDICAL ADVICE TO WORKERS, MANAGEMENT AND WORKSAFE NSW

During the course of the project, DDHM medical staff were available for advice specific to the operation from whatever source. Reports were made on behalf of the workers in relation to the avoidance of dehydration during summer operations, to settle disputes between management and workers with regard to a number of individuals' continued suitability for employment under pressure and to Worksafe NSW to clarify the immediate availability of

medical advice, for example. Considerable interest was expressed by the workers in the long-term effects of breathing compressed air on neurological function and bone necrosis, and the medical staff prepared an extensive review on this subject for all parties. No significant disputes were generated, with all problems being resolved by on-site discussions between the relevant parties.

EMERGENCY MEDICAL COVER DURING TUNNELLING OPERATIONS

Medical staff from the DDHM provided continuous cover during the course of the project. The senior tunnelling engineer at the site contacted the duty medical officer whenever manned intervention at the cutter face was underway in order to ensure rapid response in the event of an emergency. Compression facilities were available on-site at the southern exit of the soft ground tunnel with trained personnel immediately available. The compression vessel there was a twin lock, 600 kPa (5 bar gauge) chamber capable of treating one recumbent or four seated patients with an attendant. Further facilities and staff were available on short notice at the POWH, approximately 10 km away. It was anticipated that most therapeutic compression would take place in the hospital.

The period of heightened medical readiness included a "bends watch" period of low activity following the workers' exit from the decompression lock. This watch varied in length according to the exposure profile, being generally between 2 and 4 hours. It was intended to both ensure the workers' proximity to recompression facilities in the immediate post-intervention period and to reduce the incidence of DCI by ensuring no strenuous exercise was taken at this time.

Several potential hazards of work under pressure were identified, including trauma at the cutter head, environmental disturbance (toxic gases, temperature etc), DCI, barotrauma and confinement anxiety. Specific measures were taken to minimise the risk of each.

Medical incidents related to compression work

There was a total of 767 separate episodes of work at pressure (interventions), with a median intervention crew of 4. In total, workers spent 8,134 man-hours at pressure, of which 4,748 hours (58.4%) were spent working at the cutter head and 3,389 hours (41.6%) in the decompression lock. Working pressures varied considerably during the project (Figure 3) and the mean period of intervention was 3 hours 2 minutes at the cutter head. Twin access locks allowed continuous activity (insertion of second crew) and emergency access during decompression of the first crew. There were no significant mechanical failures experienced with regard to compression/decompression equipment.

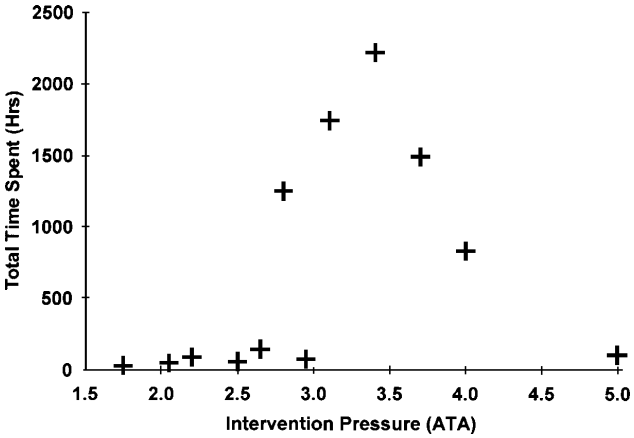

Figure 3. Intervention times and depths (1 ATA = 100 kPa approx.)

TABLE 4

MEDICAL INCIDENTS RELATING TO COMPRESSED AIR WORK DURING THE PROJECT

Medical Incident	Numbers
Middle ear barotraumas requiring medical review	13
Decompression Illness	8
Mechanical injury at cutter head: fractured femur and minor head injury (falling clay), water blast injury, shoulder injury (fall)	4
Significant dehydration requiring active rehydration	5
Incidental finding of significant pathology (lymphoma diagnosed on chest X-Ray)	1
Significant psychological distress	2

A number of medical incidents occurred during the course of the project, including 8 cases of DCI. The details are summarised in Table 4. None of the barotraumatic incidents, mechanical injuries or dehydration episodes resulted in long-term disability and these will not be discussed further. The incidental finding of a lymphoma in one individual resulted in referral to haematology for therapy and he took no further part in the project.

The details of the DCI cases are summarised in Table 5. All involved peripheral joint pain and 4 (50%) also complained of neurological symptoms or exhibited neurological signs. Only Case 3 developed problems while still in the immediate vicinity of the working lock and his symptoms improved markedly on the immediate administration of high flow oxygen as recommended in the standard operating protocols. He was also the only case showing clear cerebral involvement with clouding of consciousness and irrational behaviour. All cases developed

TABLE 5

DECOMPRESSION ILLNESS AS A RESULT OF COMPRESSED AIR WORK EXPOSURE

Case	Intervention to first symptom	Clinical details	Surface oxygen	Therapeutic compressions	Other problems	Outcome
1	14 hrs	Pain L shoulder and knee,	Improved	USN TT6 2.4 x 90 x 1		Full recovery
2	20 hrs	Aching L shoulder, knee, R ankle	Not given	Not treated		Full recovery
3	Immediate	Headache, R arm, knee and lower back pain, confusion	Improved	USN TT6 2.4 x 90 x 1		Full recovery
4	4 hrs	Pain and numbness R hand, lethargy	Improved	USN TT6 2.4 x 90 x 1	Fluid depletion	Full recovery
5	24 hrs	R elbow pain, improves with intervention	Improved	USN TT6 2.4 x 90 x 1	Cervical pain	Full recovery
6	13.5 hrs	Pain R shoulder	Improved	USN TT6 2.4 x 90 x 1		Full recovery
7	6 hrs	Pain L leg	Improved	USN TT6 2.4 x 90 x 1		Full recovery
8	4 hrs	Dizzy, pain, tingling in L arm	Not given	USN TT6		Full recovery

symptoms within the first 24 hours following decompression (mean 10.7 hours) and all those given surface oxygen as first aid therapy improved significantly during that therapy. No cases occurred following compression at less than 250 kPa.

All cases except Case 2 were initially compressed on a standard USN Table 6, with a further oxygen table of 240 kPa for 90 minutes the following day (Case 8 did not return for the second treatment). Case 2 presented to POWH several days after his episode and reported complete resolution of symptoms while resting at home. His history was consistent with DCI and he is included on that basis. All workers returned to light duties 48 hours after the precipitating event and were returned to full active duty, including compressed air fitness, at 2 weeks following injury.

The incidence of DCI during this project was 8/727 (1.1%) of interventions and 8/136 (5.9%) of workers over the three year project. As there were 2,288 individual episodes of compression over this time, the risk of DCI with each individual compression is 0.35%, or an incidence of one case of DCI for every 286 man compressions (number need to compress (NNC) 286). This is similar to the figures for a recent TBM/oxygen decompression project in Kiel, Germany in which 19 cases were reported in approximately 4,000 man compressions (NNC 211) and compares favourably with the previously reported experience with air decompression (Milwaukee, NNC 68; Dartford Tunnel 1957-59, NNC 178; Blackwall Tunnel 1960-64, NNC 94; Tyne Road Tunnel 1960-64, NNC 63, Hong Kong 1975-85, NNC 195).[9-11] [Some figures are as reported by How et al.[12]]

While there is some debate about the possibility of DCI following relatively low pressure exposures, these have been reported.[11,13] In a large project in Singapore using air decompression, for example, the majority of compressions (66%) were at or less than 200 kPa, while the maximum pressure was 335 kPa. One hundred and thirty six of 1,737 workers were treated for DCI (7.8%). The overall reported rate of DCI was 164 cases from 188,538 man-compressions (0.087%, NNC 1,150). Of these, 10 (6%) occurred following an exposure to pressures less than 200 kPa, giving an NNC of 6,406 for these exposures. The authors suggested heavy work, particularly with vibrating tools, repeated exposures and long hours probably contributed to these cases.[12,13]

Reported rates of DCI may be greatly influenced by the consequences to the worker of such a report. Kindwall noted on one project in Milwaukee, for example, that the incidence of DCI was reported as 1.44% based on the numbers requiring therapeutic recompression (the figure quoted above in a different form), while his anonymous reporting system suggested up to 26% of a shift had suggestive symptoms. While previous authors have highlighted a low apparent rate of DCI secondary to poor reporting, our medical team, with the active participation of the management, attempted to generate an environment that encouraged such reporting. On this project, we specifically

made it clear that presentation with DCI would not result in loss of income or removal of fitness to continue compressed air work in the absence of dramatic "undeserved" DCI.

No workers exhibited changes suggestive of dysbaric osteonecrosis on skeletal X-ray survey at entry or exit medical. No reports have reached us of the later development of such changes, however this may be due to by the transient nature of the workforce and the absence of further compressed air projects in Australia.

Conclusion

The safe conduct of this tunnel project resulted in an acceptably low incidence of medical problems related to compressed air work. All cases of DCI resolved completely following treatment. The use of TBM technology and oxygen decompression tables are likely to have contributed to this outcome, although the relative contribution of each is not clear.

While there is no substitute for vigilant attention to workplace safety and appropriate medical oversight of compressed air tunnelling, we believe this project to have provided further evidence for the safety and utility of these techniques. Oxygen decompression remains controversial and regulatory authorities in many countries continue to avoid it. Our experience leads us to recommend the use of oxygen decompression tables for suitable future tunnelling projects on the basis of improved worker comfort through shorter recompression times and possibly a reduction in the incidence of decompression illness.

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SPUMS ANNUAL SCIENTIFIC MEETING 2001

A RANDOMISED PROSPECTIVE TRIAL OF LIGNOCAINE IN THE MANAGEMENT OF ACUTE NEUROLOGICAL DECOMPRESSION ILLNESS – AN UPDATE

James Francis

Key Words

Drugs, decompression illness, treatment.

Abstract

There are good reasons to believe, from both animal experimental data and human studies, that lignocaine, given alone or in combination with hyperbaric oxygen, should improve the outcome of acute neurological decompression illness (DCI). The evidence supports the hypothesis that the cases in which there is brain involvement will benefit particularly. Unfortunately, it is only in a small minority of cases of DCI that there is overt evidence of brain involvement. Since DCI is, in any case, a rare disease, studying the potential benefit of lignocaine in a human population will require a multi-centre approach and a large number of centres will be needed if a result is to be reached in a reasonable time. The logistics of reaching an agreed protocol and coordinating the trial will be considerable. However, such constraints do not render clinical studies of this kind either impossible or impractical. My colleagues and I believe that it is possible to conduct a randomised, double-blinded trial of lignocaine in acute neurological DCI.

What will be more difficult will be to find a willing funding agency. The original proposal for this study was to be funded by the US Navy. Unknown to us at the outset, it is a requirement of US law that all studies funded by the US Department of Defense that involve human volunteers must provide for informed consent before the volunteer's entry into the trial. Since the study population is required to show evidence of brain disease, which renders the individual incapable of giving informed consent, this condition cannot be met. As divers do not always dive with their next of kin or legal representative, and because the treatment of DCI should not be delayed so that such a person can be contacted and asked to consent, it is impractical to attempt this study using US Navy funding. With any luck, an alternative source of funding will be found. In the meantime, the study is on hold.

Introduction

A workshop sponsored by the United States Special Operations Command (USSOCOM) and conducted by the Undersea and Hyperbaric Medical Society (UHMS) at its meeting in Anchorage, Alaska on 30 April 1996 examined the management of diving casualties in the tactical Special Operations environment.[1] Several issues with respect to the treatment of decompression illness in these operations emerged during the workshop. Among these was the issue of adjuncts to recompression therapy.

Most commercial and military diving operations are conducted so that any victim of DCI can be recompressed rapidly. When treated without delay, the success rate for treating these casualties has been reported by several authors to be over 95% for a single treatment.[2-4] However, Special Operations are often conducted in remote areas where there may be considerable delay in access to recompression facilities, with an accordingly higher probability of severe or refractory disease as a result.

It would be of great benefit to have a medication which would relieve some of the negative impact of delays inherent in remote military operations. Equally, in civilian life, a treatment that could slow the progression of disease during transit to a compression chamber would be of value, particularly when the transit time is great. Lignocaine is a widely used drug with the potential to have this effect. However there is, as yet, no evidence from human clinical studies of its efficacy in DCI. This is part of a larger problem in the management of DCI. Despite more than 150 years of observation and study, the diagnosis of DCI is based entirely on the patient's history and examination, and treatment is limited to the provision of adequate hydration and largely empirical hyperbaric oxygen protocols.[5,6] To date, no randomised, blinded, prospective trial of any therapeutic intervention for DCI has been reported in the literature.

There are many reasons why this is the case. DCI is a rare condition. World-wide there are probably no more than 5,000 cases a year. Treatment is commonly provided by hyperbaric units that receive fewer than 100 a year. Of those cases, only a small minority have serious neurological manifestations. Consequently, for any prospective clinical trial of the management of serious neurological DCI to be completed in a reasonable time, more than one hyperbaric unit needs to be involved. With units being separated by distance and, not infrequently, language it is hardly surprising that the logistic difficulties of organising a multi-centre trial have proved to be considerable.

In 1998, USSOCOM agreed to fund a proposal to determine if it is feasible to conduct a randomised, prospective, double-blinded trial of lignocaine in the management of acute neurological DCI. I undertook this study with Dr Ed Thalmann of Duke University and this report is a summary of our deliberations and conclusions.

The potential benefit of lignocaine

There is good evidence that the class 1b anti-arrhythmic agent and local anaesthetic lignocaine may ameliorate the effects of bubble embolism to the brain. Evans et al. using a feline animal model were the first to show that lignocaine given prophylactically in clinically relevant doses preserves neuro-electrical function after arterial gas embolism (AGE).[7] Subsequent in vivo studies, employing a variety of gas embolism protocols, have demonstrated that lignocaine preserves neuro-electrical function and blood flow;[8-10] reduces the extent of cerebral oedema and lowers intracranial pressure.[11-13] There have been similar findings in models of focal and global cerebral ischaemia.[14-17]

Possible mechanisms whereby lignocaine causes these observed effects include inhibiting the transmembrane ion shifts that occur early in neuronal ischaemia;[18-22] reducing the cerebral metabolic rate;[23,24] modulation of leucocyte activity and reducing the release of ischaemic excitotoxins.[25-34]

Because of the considerable body of evidence for believing that lignocaine may be beneficial in the management of DCI, it has been used at a number of centres although in a somewhat haphazard manner. The results of two such interventions have been published as case studies and, although anecdotal, these provide some evidence that lignocaine at least has no detrimental effects.[35,36] Showing efficacy, however, will require a formal study. Of the many potential candidate drugs for consideration in the management of acute neurological DCI we feel that lignocaine offers the best chance of success at the present time.

Study population

In the First World, the standard of care for the management of acute DCI is rehydration, recompression and the provision of oxygen. There are a considerable variety of protocols for the delivery of these agents.[6] Since it would be impossible from an ethical perspective to deny First World victims of DCI a conventional recompression protocol, any investigation of the efficacy of lignocaine on this population must be as an adjunct to recompression and oxygen. Clearly, for this approach to be successful, every effort will have to be made to ensure that the hyperbaric limb of the trial is as consistent as possible between participating units.

An alternative approach would be to investigate the efficacy of lignocaine in a population of divers who do not normally have access to recompression facilities. Such an approach would evaluate the drug in a situation that closely resembles the likely scenario to which USSOCOM forces may be exposed. There are substantial populations of diving fishermen on the Mosquito coast of Central America and in the Far East who do not routinely have access to recompression facilities. However, the political, logistic and ethical difficulties in conducting such a study render this option impractical at present.

Participating centres

A major part of the feasibility study was to identify centres that would be willing to take part in the study. The criteria that such centres should satisfy are:

1 Willing to comply with a standard therapeutic protocol (see below).
2 Appropriate ethical supervision of clinical protocols.
3 Willing to complete and submit the documentation in a timely fashion.
4 Availability and cost of monitored beds (if necessary).
5 Willing to follow up patients.

We decided at the outset that, in order to make communication as easy as possible, only English-speaking countries would be considered. We approached a number of centres in the USA, UK and the Antipodes and Table 1 shows the caseload of those centres that fulfilled the above criteria. As will be discussed later, another important consideration is that the more centres that are involved, the more difficult the coordination of the trial is likely to be. Ideally a small number of centres, each with a large caseload, would be involved.

TABLE 1

ANNUAL ACUTE NEUROLOGICAL DCI CASES WITH EVIDENCE OF CEREBRAL INVOLVEMENT AT POTENTIAL PARTICIPATING CENTRES

Unit	Yearly cases	Suitable for trial
Townsville	90-110	20-30
Melbourne	~80	~20
West Palm Beach	75-90	10-20
INM, Alverstoke	50-70	10-15
Plymouth	35-50	10-15
Poole	50-60	10-12
Aberdeen	15-30	5-9
Miami	50-70	5-7
Sydney	50-60	5-6
Honolulu	60-80	4-5
Fremantle	~30	4-5
Adelaide	20-25	4-5
Christchurch	30-40	3-5
Taverner	20-40	2-4
Auckland	50-60	2-3
Brisbane	~20	2
TOTALS	**725-915**	**106-163**

Criteria for entry into the trial

It is important that only cases of DCI are entered into the trial. Consequently no patient with a latent interval of more than 24 hours from completion of the last dive and the onset of manifestations of DCI should be admitted because the diagnosis is likely to have an element of doubt associated with it. Equally, there is no point in admitting cases in which there has been an apparently complete recovery, since it would be impossible to score any therapeutic benefit. This could be taken one step further, with a minimum score being set as an entry criterion.

Another potentially confounding factor is the delay to treatment. This varies enormously between centres, with those in the UK generally having the shortest delays and those in the Antipodes the longest. We did not consider that it would be appropriate to exclude cases based on delay to treatment. Not only is there no recognised cut-off at which treatment has been shown to be ineffective, such a policy could potentially exclude a large proportion of cases. Instead, it may be necessary to stratify cases, based on delay to treatment, when the time comes to analyse the data.

The justification for using lignocaine in DCI is based on its effects on the brain. It is therefore considered necessary that there should be at least one manifestation that is referable to involvement of the brain, such as a history of loss of consciousness; sensory or motor loss compatible with a cortical lesion, perturbation of any special sense and any positive finding on the mini mental examination. Since involvement of the brain is relatively uncommon in DCI, this requirement will necessarily limit the number of cases that can be entered into the trial.

The contra-indications for the use of lignocaine will also exclude potential cases from inclusion. These include known sensitivity to the drug, known pregnancy or liver disease and patients taking calcium channel blockers. In addition, patients who have treated themselves with or who have received a non-steroidal anti-inflammatory drug (NSAID) or a steroid preparation (other than using an oral inhaler) should be excluded on the grounds that they have been used therapeutically in DCI and may confound the trial.

In those centres that require patients receiving lignocaine infusions to be in a monitored bed (in ICU, CCU or equivalent), if no such bed is available, no patients can be entered into the trial until a bed is free.

Potential clinical protocol

Although developing a detailed method was not an objective of our study, we did need to consider in outline how such a study might be undertaken. We were greatly helped in this regard by Dr Simon Mitchell who had developed a detailed protocol while working in Auckland. The main steps in the clinical protocol would be:

1 Patient arrives at treating facility and appropriate stabilisation measures are instituted.
2 History and physical examination are completed using standard forms.
3 Determination made as to whether the patient meets the entry criteria.
4 If entry criteria are met and informed consent obtained, the patient is entered into study.
5 IV infusion started and blood samples for specified clinical tests drawn.
6 Drug administration is begun just before recompression at rate specified in the protocol. Subject and all treating personnel are blinded as to whether they are administering lignocaine or placebo.
7 Recompression protocol started.
8 Blood for lignocaine level drawn 8 hours after IV infusion was begun.
9 Recompression protocol completed, lignocaine infusion is continued for 24 hours from the start of infusion, then stopped.
10 Patient is reassessed 12-24 hours after initial treatment. Follow on treatments conducted as appropriate. Lignocaine is not administered during follow up treatments.
11 Patient's condition at discharge is recorded.
12 Patient follow up interviews to be conducted at one week, one month and one year after discharge.

Severity and outcome measures

In preparing his protocol, Dr Mitchell recognised that a formal system was required to score the severity of disease and the extent of recovery from DCI (i.e. treatment outcome). This is particularly important since there is no quantitative or even qualitative clinical test for the condition. The score that results from the application of such a system must be demonstrated to correlate with clinical or other indices of severity. In addition, for such a system to be useful in a multi-centre trial, it must be shown to be applied consistently between observers. Mitchell et al. published such a scoring system in 1998.[37] Although the scoring system is mathematically quite complex, it lends itself to being processed on a spreadsheet program. In terms of the information required from the examining physician it is remarkably simple. Each manifestation is allotted a value of 0-3: 0 = nil; 1 mild; 2 moderate; 3 severe. Another advantage of the system is that it is comprehensive in that it addresses all the potential manifestations of DCI, rather than focusing purely on those involving the nervous system. Finally, the system takes account of important characteristics of each manifestation: how specific it is to DCI; its natural history; its potential to incapacitate the victim and whether other manifestations are co-dependent on it. In this respect, it is considered to be the best system yet developed and appropriate for use in this study.

Since it was published the scoring system has been validated by Holley, who retrospectively scored 100 consecutive admissions with DCI to the Auckland hyperbaric unit.[38] He used the number of hyperbaric treatments the patient received as an index of severity and correlated this against the severity score on admission. He found a linear correlation coefficient, r, of 0.8. Using a score of 25 as a cut-off, he found that 77% of those with a score above 25 (more severely affected) were left with residua or developed sequelae; 89% of those scoring 25 or less had no residua or sequelae. It appears that the scoring system does represent a valid means of quantifying the severity of disease.

There has been no attempt so far to assess the fidelity of the scoring system between observers. While it would be possible to undertake such an assessment using case studies, it would be a less convincing test than using the scoring system live with two observers scoring the same patient independently. The reason being that in a multi-centre trial the scoring system would have to be used live, case study information having already been filtered by the examining physician. We feel that such a trial would have to be undertaken before the scoring system is used in a clinical trial of lignocaine.

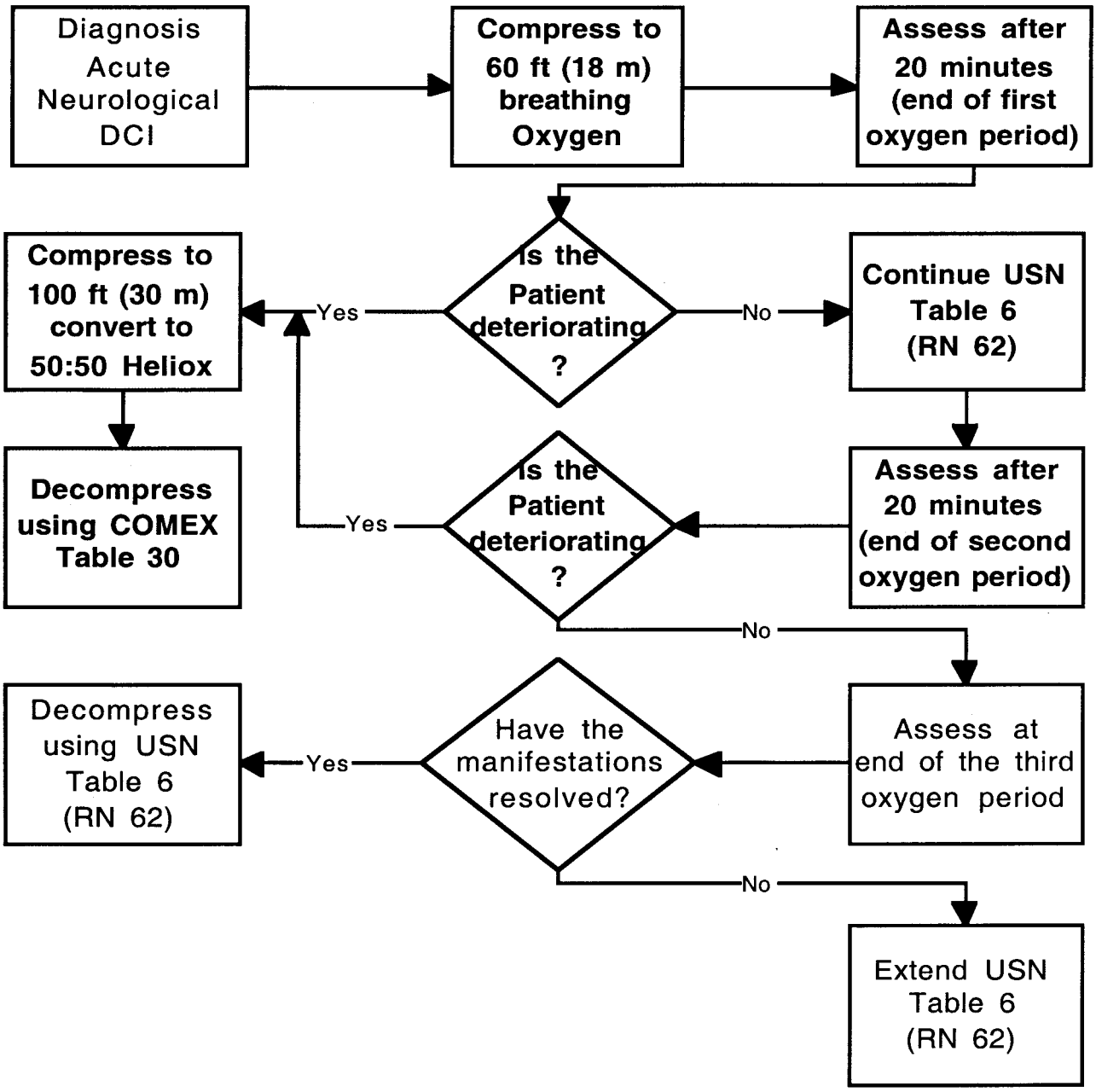

Figure 1. Outline recompression protocol for a multi-centre trial of lignocaine in the management of acute DCI

Subsequent patient evaluation

In the outline protocol it is proposed that patients should be re-evaluated one week, one month and one year after discharge. This is likely to be difficult to conduct in centres in which a large proportion of the caseload is tourists. Potential solutions to this problem are to consider follow up by telephone or for patients to be followed up by their family physician, who consults the original treating centre as necessary.

Proposed hyperbaric protocol

In discussions with the various units we visited it was apparent that one protocol, with small variations, is predominantly used. This is the US Navy Table 6 (RN Table 62), with extensions at 18 and 9 m as determined by the treating physician and based on the response of the patient.[6] Almost universally the patient is assessed at the completion of the first or second oxygen period at 18 m and, only if the patient is deteriorating or has very severe static neurological manifestations, is he or she transferred to a deeper table. Again, almost universally, the deeper table selected is the COMEX 30 table with the patient breathing a 50:50 heliox mixture while deeper than 18 m.[6] It is therefore likely that the hyperbaric protocol, subject to the agreement of the participating units, will be something along the lines of Figure 1.

For repeat treatments, there is a wide disparity in the protocols used. However, most centres expressed a willingness to be flexible. In many centres, if there are residual motor signs, the first re-treatment is a further US Navy Table 6 (RN 62). In the absence of motor signs, or for subsequent treatments, a short, shallow oxygen table is used almost universally and this may be a USN Table 5 (RN 61) or a 60-90 minute soak at various depths (2.0-2.8 bar) on oxygen with a slow (10-30 minute) bleed to the surface. In the interest of consistency it will be desirable to specify a re-treatment protocol and a table such as the Royal Navy Table 66 (Table 2) would be appropriate. When using this table, the attendant should breathe oxygen for the final 20 minute oxygen period and the ten minute ascent to the surface. There was consensus with the view that re-treatments should continue on a once- or twice-daily basis until there is no further sustained incremental improvement in the residual manifestation(s).

Number of cases and controls

In order to justify the study we must determine whether or not there will be sufficient patients to either accept or reject the null hypothesis at a certain level of confidence. The null hypothesis (H0), is that lignocaine has no effect on the outcome of acute neurological DCI. If we reject H0 we want to be 95% sure that the rejection was not due to chance alone, that is there is only a 5% chance that the H0 was rejected when it is actually true. This means we want the Type 1 error to be 5% or less. We must also be sure that we do not accept the null hypothesis when it is in fact false. This Type 2 error is usually set at 20% for most medical studies. If the Type 2 error is 20% the power of the study is 80%. The appropriate formula is:

$$Z_\alpha - Z_\beta = \frac{\overline{x_1} - \overline{x_2}}{sd\sqrt{1/n_1 + 1/n_2}} \qquad (1)$$

where:
Z_α = Z score for 5% Type 1 error (1.96)
Z_β = Z score for 20% Type 2 error (-0.845)
$\overline{x_1}$ = mean score for control group
$\overline{x_2}$ = mean score for lignocaine group
n_1 = n_2 = number of subjects in each group
sd = standard deviation of scores

Equation 1 is easily solved for n, the number in each group:

$$n = \frac{2}{\left(\frac{(\overline{x_1} - \overline{x_2})}{sd\langle (Z_\varepsilon - Z_\beta)}\right)^2} \qquad (2)$$

TABLE 2
ROYAL NAVY TREATMENT TABLE 66

Gauge Depth in metres of seawater (feet)		Stops/Ascent in minutes (breathing mix)		Elapsed Time (hours and minutes)		Rate of Ascent msw/min (fsw/min)
14	**(45 fsw)**	**30**	**(O_2)**	**00:00 –**	**00:30**	
14	(45 fsw)	5	(Air)	00:30 –	00:35	
14	**(45 fsw)**	**30**	**(O_2)**	**00:35 –**	**01:05**	
14	(45 fsw)	5	(Air)	01:05 –	01:10	
14	**(45 fsw)**	**20**	**(O_2)**	**01:10 –**	**01:30**	
14-0	(45 fsw-0)	10	(O_2)	01:30 –	1:40	1.4m (4.5 fsw)
Surface					**01:40**	

Since the standard deviation is not known we use the relative mean difference $\frac{(\overline{x_1} - \overline{x_2})}{sd}$ to estimate the size of the effect. The convention is that if the relative mean difference is 0.1 or less there is no effect. If it is 0.3 there is a slight effect, 0.5 a moderate effect, and if it is 0.9 there is a large effect. Substituting these values for Z we obtain table 3.

TABLE 3

NUMBER OF SUBJECTS REQUIRED

Relative mean difference	Number of subjects in each group
0.1	1,574
0.3	175
0.5	63
0.9	18

Lignocaine levels

In Dr Mitchell's original protocol, he proposed that a plasma lignocaine level be taken at 8 and 24 hours into the infusion and the infusion rate adjusted accordingly. The reasons for this were:

a. To avoid toxicity and
b. To ensure that the blood level was in the therapeutic range.

For those receiving the placebo, a system of sham results should be in place whereby the reporting laboratory would report a value determined by the investigators.

In discussing this with the centres visited, it rapidly became apparent that the measurement of plasma lignocaine levels was not a routine procedure and many laboratories would be unable to process samples in a timely fashion unless an analytical kit were provided. In other instances the samples would have to be sent to a reference laboratory. Either option would be expensive and the latter would also be time consuming to the point that it may not be possible to use the resulting data while the infusion was in progress.

In assessing why measurement of the lignocaine level should be undertaken we concluded that a check is necessary to ensure that patients who were supposed to receive the drug did so and those who were not supposed to did not. It was the experience of a number of centres that the infusion rate proposed, a pump-controlled bolus of 0.5 mg/kg over 20 minutes followed by 120 ml of a 2 mg/ml solution (240 mg) over the next hour, 60 ml (120 mg) over the next hour and 45 ml (90 mg) per hour thereafter, invariably results in a therapeutic dose in the otherwise healthy young patients that are representative of the diving population. Equally, an infusion lasting only 24 hours is unlikely to result in lignocaine toxicity. It is therefore considered appropriate that a single level should be taken just before the infusion is discontinued and that these samples be frozen and batched for processing at a reference laboratory.

Ethical clearance

For this trial to work each centre must conform to a single protocol. Ethical approval for the protocol would have to be granted not only by the US Navy but the human use committees of each participating centre. We do not underestimate the difficulty involved in getting such approval. It will be considerable and will increase with each centre that is added to the protocol. Although the institutional ethics committees at the centres visited offer a processing time of between one and two months, it is inevitable that some rewording, additions or deletions of the first draft will be required to satisfy many of them. Potentially, some of these will be conflicting. The result is likely to be a protracted process of serial revisions, each requiring as long as the slowest review body takes to complete the process, until a final draft is agreed, assuming that this is possible. It is considered that a period of no less than six months should be allowed for this process and longer if more than six centres participate.

Informed consent

In the course of discussing an outline protocol with a number of centres it became apparent that the study contained a potential ethical issue. As the justification for studying the use of lignocaine in the management of DCI is based on its effects on the brain, it is almost certainly necessary to require evidence of involvement of the brain in the subjects. If there is evidence of brain dysfunction, can patients give informed consent to participate in the trial?

Having consulted the director of the US Navy Clinical Investigation Program, the answer to the question is "No." Furthermore, there is a section of the United States Code (No. 10 USC 980) which reads: "Funds appropriated to the Department of Defense (DoD) may not be used for research involving a human being as an experimental subject unless (1) the informed consent of the subject is obtained in advance; or (2) in the case of research intended to be beneficial to the subject, the informed consent of the subject or a legal representative of the subject is obtained in advance." This requirement is imperative, non-negotiable, and can not be waived. If gaining the patient's informed consent is not possible because the patient has brain disease, this would jeopardise the US DoD funding such a trial. Furthermore, 10 USC 980 precludes any DoD study from attempting to invoke legislation known as 21 CFR 50.23, which was written precisely to allow these types of

emergency studies, with minimal time to contact third party consent relatives, to proceed without specific informed consent of the subjects.

We briefly considered the possibility of gaining informed consent from divers prior to their undertaking the dive that causes DCI. It would require a herculean effort to approach, let alone gain the consent of just the estimated 5 million divers in the USA. It would be equally difficult to recruit those who hail from Canada, the UK or the Antipodes. The problem is that there are numerous umbrella organisations for divers and many divers belong to no organisation whatsoever. This idea was therefore quickly rejected. It would be equally difficult to try to gather the consent of a partner, parent, legal guardian or legal counsel who could provide consent on the patient's behalf. Except for married divers who choose to dive with their spouse (a very small minority of the sports diving population) it is most unlikely that anybody who could consent on the patient's behalf will arrive at the hyperbaric unit with the casualty. Since acute neurological DCI represents a medical emergency, delaying treatment while attempting to contact such a person by telephone would be unethical. The provisions of 10 USC 980 have halted this study in its tracks and precludes it being funded by the US DoD. However, in all other important respects, we feel that the study is perfectly feasible although it will be difficult and expensive to undertake.

Conclusions

At the outset of this study we predicted that the most difficult part of this study would be to find suitable participating centres. This turned out not to be the case. There is clearly much enthusiasm for conducting randomised, double-blinded trials in DCI. There are, however, substantial hurdles that will have to be overcome to see such a project through to fruition. Many of these arise because of the need to involve a large number of centres if results are to be available in a reasonable time frame. The logistics of reaching an agreed protocol and coordinating the trial will be considerable, particularly if the entry criteria to the study are restrictive, as they are in this case. However, these constraints do not render clinical studies of this kind either impossible or impractical. We believe that it is possible to conduct a randomised, double-blinded trial of lignocaine in acute neurological DCI.

A difficulty, which I have not covered, is that this trial will be very expensive to conduct and, given the limited number of organisations with deep pockets that are likely to fund such research, this will limit the rate of progress in the future. An important funding organisation in diving medicine is the US Navy. The limitation to getting this study funded by the US Navy turned out to be the constraints placed by the US Congress on how the DoD conducts its clinical research. This constraint is unlikely to be removed in the near term, if ever. Thus, if clinical studies of this kind are to be funded by the US Navy, there must be no difficulty with obtaining proper informed consent. Alternatively, a more liberal source of funding will have to be found.

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AUDIENCE PARTICIPATION

Chris Acott, Adelaide

I have two case histories. The Diver Emergency Service, based at the Royal Adelaide Hospital (RAH) occasionally gets calls from chambers in Thailand where there are a lot of fishermen divers. Three weeks ago I was rung as they had a diver who was paraplegic, with no bladder or anal control. He had presented after a long delay. As they had treated him twice with a US Navy table 6 without improvement they rang me for advice. I suggested that they started him on a lignocaine infusion. He made a full recovery and walked out of hospital. The other was in the RAH. He had six Table 6 treatments and was still hemiparetic. He was starting on a lignocaine infusion just before I left. It will be very interesting to see how he went when I get home.

James Francis

I have also seen this sort of Lazarus effect with lignocaine. I think the potential is wonderful but to get it as an indicated use of the drug we will have to have some trials to prove that it works and, at the moment, I have got no idea how we are going to get this expensive trial funded. We calculated the cost of the trial, for the benefit of the US Navy, to be around $600,000 to $1,000,000 to do.

Mike Bennett, Sydney

In view of the of stories that Chris has mentioned, is it ethical to deliberately not give half of them lignocaine? How long is that trial going to be ethical for us to do, let alone US Navy regulations.

James Francis

I suppose if there is a steady drip of case reports in the literature of this Lazarus type effect, eventually that will provide sufficient weight of evidence for its use in DCI becoming an indicated entity. It really depends upon whether people like Chris and I write up the cases. I have not written up either of the two that I have used lignocaine with. Are you going to write yours up Chris? Lignocaine is used sporadically all over the place now. One of the things I have had to tell people is even if they want to use lignocaine they should wait and hold back and not use it until the trail is completed. Some people use it almost routinely now and as soon as that becomes the case, of course one cannot do the trial.

John Knight, Melbourne

If you are getting Lazarus type results, you are back in the situation of the early antibiotics. Nobody ever did a controlled trial of streptomycin in tuberculosis or penicillin in infected wounds. The results were so different from what had been happening before that it would have been unethical not to treat the patients.

James Francis

That is true but out of date. In the UK we have the National Institute of Clinical Excellence or NICE for short. These people review medications for use in the National Health Service (NHS). Their decisions are evidence based. On the recommendations of this NICE committee people are allowed or not allowed to use drugs in the NHS. There was no NICE committee when antibiotics were being developed. Just as well, but unfortunately we live in a world where one cannot work like that.

David Taylor, Melbourne

While I was at Duke we had a couple of severe spinal hits where they were commenced on lignocaine within half an hour. In one case there was no effect at all. He got worse and we ended up saturating him.

James Francis

This is the point about doing trials. One may get wonderful results reported with a particular drug or regime, but very rarely are the failures reported. If one has done a proper controlled trial that should come out in the wash up.

Mike Davis, Christchurch

I'd like David just to put the other side of the coin. We have used lignocaine in about 10 divers over the last few years in Christchurch. So far I have not seen any enhanced benefit from its use in anyone. So I don't think there is any problem about the ethics of a trial of this nature.

Mike Bennett, Sydney

Our experience at the Prince of Wales Hospital is that one or two have some benefit. But for most there is no apparent change to their course. However in most cases, we are giving lignocaine later than the trial was contemplating.

Barbara Trytko, Prince of Wales Hospital

I have used it in two patients who had cerebral symptoms and were started on it before being retrieved. Both patients did very well.

James Francis

The problem is that cerebral DCI is notoriously good at getting better. The spinal ones often have major residua. Another reason to need a large number of cases to show a difference.

Drew Richardson, PADI

Is it possible to do a pilot study, scale it down a little bit to make it cheaper?

James Francis

The problem is if one did that the trial would not have the power to answer the question. It is not really worth doing a study unless there are enough cases to answer the question. But each case costs money. The patients will stay longer than they normally do. Additional staff will be needed. Monitoring in full is expensive. A study like this is only worth while if it gives clear answers.

Mike Bennett

Unfortunately the words "pilot study" is often code for " I do not have the time, money or inclination to do the proper job".

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NEAR-DROWNING AND THE UNRESPONSIVE DIVER: RESCUE TRAINING FOR RECREATIONAL SCUBA DIVERS

Drew Richardson

Key Words
Near-drowning, rescue, training, unconscious

Introduction

This is an overview of the procedures required for rescuing near-drowned scuba divers and the training recommended. The paper includes definition and causes, four general procedures for in-water rescue, two scenarios for rescues and the training required, one for an unresponsive diver underwater and the other for an unresponsive diver at the surface. Also included are variations of in water rescue breathing technique, seven procedures for assistance out of the water and closing philosophical thoughts

Definition

Near drowning occurs when someone revives a diver (or swimmer) who became unresponsive, unconscious, or unable to respond or act coherently, and stopped breathing while submerged. Although a variety of causes may initiate fatal dive accidents, in most instances drowning ultimately causes death. One can define drowning as death caused by asphyxiation (suffocation) in water. When a diver suffers asphyxiation in water but gets revived, we define the accident as near drowning.

In more than 80% of cases, near drowning causes water to enter the patient's lungs. This interferes with the body's ability to transport oxygen even if the patient is breathing, resulting in hypoxia or insufficient oxygen reaching the tissues.

With near drowning, the most common immediate symptom will be that the patient is not breathing. Therefore, the primary first aid for near drowning is immediate rescue breathing. Be alert for vomiting, especially as the patient resumes breathing on his own. Be prepared to turn the patient and keep the airway clear. Symptoms also include coughing, shortness of breath, blueness of lips (cyanosis), frothy sputum and cardiac arrest. If the patient breathes sporadically, administer oxygen. Treat the patient for shock and contact the Emergency Medical Service.

Although in most instances brain damage occurs in as little as four to six minutes without oxygen, for reasons not completely understood, immersion (especially in cold water) can make revival possible even after fairly long periods. Therefore, begin emergency care immediately, even if the victim has been submerged longer than four to six minutes.

A revived near drowning patient may quickly seem fully recovered. Nonetheless, the patient should always go to a hospital as soon as possible. Physiological complications caused by inhaled water can occur five to six hours after the accident; untreated these complications may be fatal. Proper medical care can prevent this.

Possible causes of near drowning

Near Drowning may be caused by swallowing water, extreme fatigue, entanglement and lung over pressurisation, panic, inefficient breathing, throat blockage, exhaustion, cardiac arrest and unconsciousness.

In-water Rescue

The essentials of in water rescue are:

1 Quickly bring the diver to the surface and check for breathing.
2 Establish ample positive buoyancy for rescuer and victim.
3 Signal for assistance, provide rescue breathing as needed.
4 Remove the diver from the water.

Unresponsive underwater

If the regulator is in the victim's mouth, hold it in place from behind even if the victim is not breathing. This may help keep water out of the victim's lungs, provide air if the victim resumes breathing during ascent and will not interfere with expanding air escaping from the victim during ascent.

If the regulator is out of the victim's mouth, do not waste time putting it back in; expanding air will vent itself and may prevent water from entering their lungs.

For the best control, use your own buoyancy compensating device (BCD) to ascend with the victim. Hold the victim from behind and begin your ascent, venting air from your BCD as necessary to maintain a safe rate. If possible, do not drop the victim's weights or inflate his BCD until you reach the surface. This makes it easier to control the ascent. Keep the victim's head in a normal position. Expanding air will vent by itself.

If releasing air from your BCD does not control the ascent, you may need to release air from the victim's BCD. You can also flare out to reduce ascent speed. If you cannot keep the ascent under control, allow the victim to ascend

separately. Ascend at a safe rate and regain contact with the victim at the surface. When you reach the surface with an unresponsive diver, call for help as you ditch weights, establish buoyancy and turn the victim face up.

Unresponsive at the surface

If you find an unresponsive diver floating, call to the diver to check responsiveness as you approach. As soon as you determine that the diver needs rescue, call for help, if available, and follow these general procedures below.

General procedures

Drop the victim's weights, establish positive buoyancy, turn the victim face up, remove mask and regulator, position the head to open airway, look for chest movement, listen for breathing, feel for a pulse. If the victim is not breathing initiate rescue breathing. Assess towing/ removal considerations based on the situation.

In-water rescue breathing techniques

There are several alternatives available to the rescuer to initiate in-water rescue breathing which include:

Mouth to pocket mask,
Mouth to mouth and
Mouth to snorkel.

Assistance out of the water

Once you have the victim ashore or on board and secure, perform a primary assessment and follow these seven general procedures:

1 Keep the airway open and check for breathing. If necessary, start and continue rescue breathing and/ or cardiopulmonary resuscitation (CPR).
2 Observe the diver constantly, checking breathing and pulse.
3 If the diver does not require CPR or rescue breathing, keep the diver lying level on their side, supporting the head (the recovery position). Do not let this position interfere with transportation or other aid. It should not be used if CPR is required.
4 Administer emergency oxygen if possible.
5 Keep the diver still and attempt to maintain a normal body temperature by protecting the diver from heat or cold.
6 Seek emergency medical assistance.
7 If unable to accompany the diver to medical treatment, write down as much background information as possible and attach it to the diver in a conspicuous place.

Closing philosophical thoughts

As a rescuer, do the best you can with the resources you have under the circumstances. All you can do is give the victim or patient a better chance, not certainty, for a more favourable ending. Realise that even if your efforts, in the end, made no difference to the outcome, they still made a difference in that they improved what chances the victim/ or patient had.

AUDIENCE PARTICIPATION

Chris Acott

I think that perhaps in the next couple of years in South Australia buddy diving may be looked at very carefully. I say this because I am involved in a law suit where a diver is suing her buddy and the people who took her out diving. I suppose what the court will be looking at will be the responsibility or duty of care of a buddy in a diving situation, which may be quite interesting.

Guy Williams

I would be interested in Drew Richardson's opinion of such ideas as mandatory regulator servicing and BCD servicing and actually training divers to use redundant systems.

Drew Richardson

I think that training is key. Some of those recommendations, without training people to cope with the more complicated tasks, are going to give a negative result. Just adding a tank here and there is not going to do anything except probably put people in harms way. The technical community is already going down the pathway of totally redundant systems. Your full face mask recommendation has not been something that they have picked up because they cannot do gas switches. However there is at least one manufacturer who is producing a full face mask.

The mandate for the dive industry is to service divers. I think a lot of operators right now, if a tank comes in empty, are going to open it up and look in it, because first of all there is a workplace health and safety issue. There have been cases where an employee was injured in a tank explosion. So if a tank is empty, quite typically they are going to crack the valve and do a visual inspection. To tell the diver that is going to cost them extra, may be part of the individual store's protocol, or it may just be wrapped into their service. I do no think people are going to be dissuaded from running out of air by that.

Out of air is an on-going, long-standing problem which we try to solve that in the beginner training schemes. I am not sure it is an increasing problem. Even at this conference I have seen people run out of air. It is a Darwinian result as there is a task loading. Some people do not survive well and others do. There are a lot of reasons for running

out of air, but response training does not necessarily have to be more formalised. This conference is an on-going training experience. On day one people were sorting out the cobwebs. By now they are more into an automatic process of getting their kit together and they know the reefs a little bit more, the lay of the current, etc. I think that can happen within a peer community as well, not necessarily in a formalised academic environment although we certainly would encourage that.

We have found for the last 30 years that about 30% of divers actually go forward beyond entry level to undertake higher diving certification. That is pretty consistent even though we promoted quite heavily to look after these interests it does not seem to be budging off of about 30% of the divers and I am not sure what to do about that. On the upside the DAN database, just recently reported, have demonstrated a downward curve in annual fatalities. However, there still are fatalities globally. Unfortunately there always will be. Our goal as educators certainly is to try to eliminate deaths and it is encouraging at least to see that the curve is not going up or spiking. It is clearly a downward trend.

On the buddy diving, solo diving arguments, I think we have to be cautious about throwing the baby out with the bath water. It depends how you interpret buddy diving. There are a lot of pragmatic benefits of course. Just dry suit zippers and things like that, which unless you are a contortionist, there is no way on God's green earth you are going to be able to suit yourself up properly. With the assistance of DAN's research department I have recently looked at the number of fatalities in the last 10 years for divers, either solo diving or diving alone. We found about 538 cases. They may have ended up alone, not intended to go solo diving. Our position is that we will continue to advocate buddy diving in the training scenario for all the long standing benefits. Solo diving has a place, but it should not be a one off thing.

If you are going to solo dive, then I think you have to involve yourself in redundant systems. Make a mental effort, not just disappear off the boat by yourself down to 50 or 60 m and hope that it all works out well because when things go wrong, you have a lot of options taken away from you when you are by yourself. Then there is the intangible option which might not have to do with training or anything to do with equipment and that is another human brain there with you when you are focused on something. That brain might just intervene in an error chain in a very benign point, then no one would ever think the more or the less of it. So you could argue hypothetically if you break error chains early you can have positive outcomes, they might not even be noticed.

Looking at the solo diving argument from a legal point of view, we have had a few cases in the US, which I am sure will stun everybody here, where buddies have sued one another. Shocking for our culture but I think people have realised that more and more in the UK and from what I see from Australia, the lawyers are advertising their services. It might be frivolous but people can bring suit for anything at any given time. I do no think that should run us off inherently safe protocols that have been well established for a number of years.

There have been buddy systems that have broken down and double deaths as well. There is no question of that, but by and large, the safety aspects, at least in the initial training seem to be worth preserving. And I think you asked something on emergency ascent training. A few years ago I presented the various options. They depend on your depth. We do not ask divers to look around endlessly for their buddy to get a bit of air or whatever in 10 m or less. If they are not close enough then divers are trained to do a controlled emergency swimming ascent to the surface. The deeper you get the trickier it becomes whether it has a positive outcome or not. And of course we heavily advocate the use of alternative air sources whether that be an octopus or some of the other things that you described.

Guy Williams

A supplementary question. In my part of the world if you go out on some of the dive charter boats and say "I want to dive the 110 foot sub" they will say "Where is your pony bottle?". If you do not have one they will say "Here we will rent you this, stick it on the back of your tank" and explain how to use it in a very limited way. It might only apply to my part of the world, whether that sort of training should be included in the basic open water course so people have seen it and know what it is before it is thrust upon them.

Drew Richardson

It is included, but there is no motor skill practice. They know these things exist, Spare Air, pony bottles, those are all covered in all the academic preparation so that there is exposure. Whether or not there is actual motor skill practice is left to the instructor.

Reference

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REVERSE DIVE PROFILES

Guy Williams

Key Words

Decompression illness, diving tables, risk, safety.

Introduction

On October 29th and 30th 1999 the Smithsonian Institution held a Reverse Dive Profile Workshop in Washington, DC. The 49 person workshop was called to decide if there was evidence to show that reverse dive profiles were more dangerous than those where the first dive was the deepest and any repetitive dives were shallower. The participants mostly (41) came from the USA, two from Canada and Germany, and one each from Finland, Norway, Switzerland and the UK. Four of the participants have been Guest Speakers at SPUMS Annual Scientific Meetings, Glen Egstrom was our first Guest Speaker in 1978 and again in 1991, Alf Brubakk in 1999, Bill Hamilton in 1996 and Richard Moon in 1997 and 1999. This paper is an attempt to summarise the 295 page book of the Proceedings, edited by MA Lang and CE Lehner, which was published by the Smithsonian Institution in 2000.

Three reviews of the Workshop Proceedings appeared in one issue of the Journal in 2000 [32 (3): 115-117, 144-145 and145-147].

What is a Reverse Dive Profile?

The Workshop adopted the definition that a reverse dive profile was either two dives performed within 12 hours in which the second dive is deeper than the first; or the performance of a single dive in which the latter portion of the dive is deeper than the earlier portion.

For many reasons reverse dive profiles are being performed in recreational, scientific, commercial, and military diving

What is wrong with a reverse dive profile?

Although there appears to be anecdotal and practical support for avoiding reverse dive profiles the exact origination of condemning this profile is unclear. It appeared in recreational dive training in early 70s and became accepted as a standard recommendation by the training agencies by the mid 80s.

The reverse dive profile “problem” is limited to recreational diving as neither the US Navy nor the commercial diving sector have prohibited reverse dive profiles. This may be due to their infrequent usage of repetitive diving or to their more disciplined diving routines.

The prohibition of reverse dive profiles by recreational training organisations cannot be traced to any definite diving experience that indicates an increased risk of DCS.

However, when divers use US Navy tables, reverse profiles always produce less bottom time on the second dive to the no-decompression limits so there are practical reasons to avoid deeper repetitive dives. After all recreational divers want to spend as much time as possible underwater. Intuitively one can understand the theoretical attraction of adding less nitrogen to the body by doing shallower dives after a deep one and benefits that this might bring to avoiding decompression sickness (DCS). Add the legal advantage that teaching deep dive first would bring to the instructor if a diver sued after suffering DCS after a reverse dive and it is small wonder that reverse dive profiles were advised against.

Are reverse profile dives safe?

No convincing evidence was presented that reverse dive profiles within the no-decompression limits lead to a measurable increase in the risk of DCS.

No theoretical or experimental evidence can be found that indicates a repetitive dive must be shallower than the dive that precedes it.

Many divers using PADI recreational dive planner (RDP) or dive computer to do deeper repetitive dives are beyond the tested envelope which is mostly military and experimental dives. The commercial diving industry does not often open its records to outsiders.

Deep repetitive dives that are followed by a direct ascent to the surface have been shown to produce a high incidence of DCS. Repetitive deep decompression dives that do not push the limits do not seem to have the same problems.

However no evidence was produced that showed that reverse profile dives were safe. Only evidence which suggested that they were not dangerous. The General Session discussion which finishes the book makes it quite clear that no one was willing to say that it was safe to use reverse profile dives except in the no-decompression limits. Tom Neuman made it quite clear that, from his experience treating bent divers, doing a deep dive as the last dive was a dangerous practice because many recreational diver paid no attention to keeping some air for emergencies and most of his cases had run out of air and had to make an out-of-air ascent. From a shallow dive this was not likely to be

followed by DCS but DCS was very likely after a rapid ascent from a deep dive.

Safe diving

For divers who use a dive computer and are taking advantage of its multi-level capacity, any rule to avoid reverse profiles would seem irrelevant.

For those still using a dive table, the avoidance of reverse profiles is an important practical rule that results in more bottom time.

Conclusions

The final statement of the meeting was "We find no reason for the diving communities to prohibit reverse dive profiles for no-decompression dives less than 40 msw (130 ft) and depth differentials less than 12 msw (40 ft)".

In the 21 pages of the General Session discussion it appears that the consensus, give and take a few, was that conservatism, staying well within the times at depth, ascending slowly and decompressing longer than required by the tables, appears to be the best way to avoid decompression sickness. This discussion, the Introductory Session and the Medical Session were the easiest for medicos to understand. Two major sessions, Physics/Physiology and Physiology/ Modelling were dominated by mathematics. The Operational Experience Session was very interesting.

Nowadays most divers use dive computers. Computers rarely are victims of DCS, but divers can be! In fact, in some series of Decompression Illness reports, 50% and more of the affected divers were using computers.

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A SUMMARY OF THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY NEAR DROWNING WORKSHOP

Chris Acott

Key Words

Drowning, incidents, near drowning, physiology, treatment.

Introduction

The Undersea and Hyperbaric Medical Society (UHMS) Workshop on near-drowning was held in 1997.

One of the most interesting presentations was by Carl Edmonds on drowning and near-drowning.[1] Table 1, chosen from that paper, shows what I consider to be the interesting parts of his data. This reported 100 Australian divers, who fitted the requirements for being classified as dying from drowning.

TABLE 1

COMPARISON BETWEEN 100 DROWNED DIVERS AND 48 WHO SURVIVED NEAR-DROWNING

Taken from tables in C Edmonds, *Drowning with scuba*.[1]

	Drowned	Survived
Training		
No training	38%	4%
Under training	8%	15%
Experience		
None	37%	31%
Novice	30%	35%
Some	27%	29%
Equipment		
Faults	31%	18%
Misuse	43%	38%
Buoyancy		
Overweighted	25%	27%
BCD not inflated	52%	31%
BCD failed to inflate	5%	8%
BCD inflated before incident	12%	-
Victim inflated BCD	15%	35%
Buddy operated BCD	16%	25%
Overall inflated BCDs	31%	60%
Air		
Out of air	49%	27%
Low on air	11%	8%
1/4- 1/2 cylinder	11%	20%
>1/2 cylinder	29%	45%
Water environment		
Poor visibility	26%	18%
Current	55%	31%
Rough	44%	41%
Weight belt		
Not ditched	66%	48%
Ditched by victim	10%	19%
Ditched by rescuer	20%	25%

They were selected from Douglas Walker's Australian Diving-related death reports. Their stories were compared with those of 48 divers who had survived near-drowning and completed a questionnaire on various diving-related web sites.

In many ways the two groups were very similar. The drowned and the near-drowned had much the same diving experience. However, 38% of the drowned victims had no training while only 4% of the survivors had not been trained. Equipment faults and misuse of the diving equipment featured in both series, but the survivors had just above half the rate of faults of those who died.

Divers were over weighted in 25% of the drowned and in 27% of the near-drowned. Too often people learning to dive are over weighted by the diving instructor. This is to make it easier for them to get down and to keep them on the bottom doing the various things that the instructor want to teach them. Often when people learn to dive carrying 26 kg of weight belt they will always use 26 kg. About 5 years ago, in South Australia, a husband was teaching his wife to dive. They were on the Port Norlunga jetty and he had over weighted her, then told her to jump in with the snorkel in her mouth. He turned his back on her and started to fiddle with his equipment. Apparently he never actually told her how to inflate a buoyancy compensating device (BCD) to stop herself from sinking, so when she jumped in the water with a snorkel in her mouth she sank. Her husband was busy with his equipment and about 2 or 3 minutes later he heard someone say "Hey there's somebody on the bottom down there". She had drowned. She was dead. He had forgotten to tell her about putting the regulator in her mouth before entering the water as well as forgetting to instruct her in inflating her buoyancy jacket. She died because she was overweighted and did not know how to use her equipment.

The BCD was inflated in 31% of the drowned divers whereas in the survivors the BCD was inflated in 60%. In the drowned group, out of air and low air situations featured in 60% of the deaths. Of the near-drowned survivors it was only about 35% who were out of air or low on air. Only 11% of the drowned divers still had between a half and a quarter of their air remaining. Twenty per cent of the survivors had that much remaining air. Only 29% of the drowned divers still had more than half a cylinder remaining, but in the survivors it was 45%. Most of the near-drowned divers (65%) had enough air to get themselves out of their problem.

Rough water exposure was much the same for both groups but currents were reported in nearly double the number of dead divers than in survivors. Poor visibility was a factor in 26% of drowned divers but only in 18% of the survivors. Perhaps they could actually see their way out of the wreck or see their way to the surface. In wrecks and caves it is very easy to stir up silt and lose visibility.

Retained weight belts featured both in the drowned (66%) and near-drowned (48%). It seems that divers want to save money and not their lives when they fail to ditch their weight belts when in trouble. Carl always tells people to take their weight belt off as soon as trouble starts and to hold it well away from your body. Then if it needs to be dropped it will fall away clear of the body, and if you go unconscious you will drop your weight belt as your grip relaxes. I do not understand why people in trouble on the surface, struggling to keep their head out of the water, would not ditch the weight belt unless it was to save money.

From Edmonds' data survival depended on training, education, water skills, good buoyancy control and a rescuing buddy . Failure to ditch the weight belt featured in both drownings and near-drownings and fatalities. In the latter 60% were either out of air or low on air.

Why do divers run out of air?

The Diving Incidents Monitoring Study (DIMS) has data on out of air causes (Table 2). The main cause is failure to check the air supply both before and during the dive. The only safe way to dive is to check whether the cylinder is full and the valve turned on fully before you get in the water. This known failure to check air supplies always worries me when people start talking about redundant air systems . One wonders whether divers with two air supplies will always check the second air supply that is theoretically supposed to be there to keep them out of trouble.

Equipment failure is usually an inaccurate contents gauge. Some people had inaccurate contents gauges and when they found it difficult to breathe and tapped their old analogue contents gauge the needle just suddenly went straight to empty. Debris in the tank obstructing the valve, rupture of an air hose, vomitus in the mouthpiece and sudden failure of the second stage to deliver air also featured. Many people think that rupture of the air hose from the first stage to the second stage will always occur at the start of the dive when the tank pressure high. That does not necessarily happen, sometimes it is well into the dive.

Contributing factors include poor dive planning. Most divers have no idea on how much air they consume during a dive. Sometimes they decide to do a dive to 50 m. But they do not realise that they will need a much greater air supply than for their usual less-than-18 m dive;

a) for getting to 50 metres and
b) to do decompression on the way up.

I was taught to use the rule of thirds, which people do not seem to use these days. A third of the supply to enjoy the dive, a third to get back to the boat, and a third to have on the surface just in case there is some trouble.

TABLE 2

CAUSES OF OUT OF AIR INCIDENTS FROM DIVING INCIDENTS MONITORING STUDY DATA

Failure to check air supply
Before and during dive.

Equipment failure
Inaccurate contents gauge.
Debris in tanks.
Rupture air hose, not necessarily at start of dive.
Vomitus in mouth piece.

Poor dive planning
Air consumption for depth and dive time not calculated before dive.
Failure to apply rule of thirds, one third in, one third out and one third for emergencies.

Stupidity
Inappropriate response such as:
Doing a safety stop when low on air,
Continuing the dive when using octopus regulator,
Inattention.

Poor buoyancy control
Frequent use of air for buoyancy adjustments.

Poor buddy diving
Buddies diving too far apart so that one goes from low on air to out of air while trying to alert buddy to the problem.

Stupidity is the only way to describe some inappropriate responses. One of these is doing a "safety stop", which by definition is not a decompression stop, when both divers are low on air. The result is almost always one, and often two, out of air ascents. Another is continuing to dive when one diver is out of air and is given the buddy's octopus regulator. Amazingly this happens and they continue the dive and the two of them run out of air very quickly. The final inappropriate behaviour is inattention, neglecting the air supply, while being fully occupied by what is going on around you.

Poor buoyancy control occurs usually when the diver is overweighted and air needs to be added to the BCD the frequently in order to maintain neutral buoyancy. This frequent topping up depletes the air supply very rapidly.

Finally poor buddy diving, when the buddies are separated greater than 5 or 6 m. If one buddy becomes low on air, by the time he or she actually gets to the buddy they are often out of air.

The Workshop

Brown and Piantadosi discussed the hospital management of near-drowned people including general measures, management in the intensive care unit (ICU), brain resuscitation and the status of patients and their prognosis.[2] Table 3, compiled from their paper, demonstrates their management plans. One checks for pre-disposing factors, for cervical spine and skull fractures. This will involve X-rays. One also checks for evidence of ear and sinus barotrauma because they may serve later as portals of intracranial infection if they become infected. The management in ICU is standard cardiac support, fluids and monitoring. Inotropes, fluids and the management of various electrolyte abnormalities that may occur are often needed. Salt water, when it is swallowed can act as a very good osmotic diuretic or cause osmotic diarrhoea.

The initial chest X-ray may be normal. Respiratory care using intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP) may be needed. If antibiotics are to be used these must be determined by accurate microbiology sensitivity testing. The treatments for brain resuscitation used in intensive care units in the 1980s, such as Hyper therapy have been reviewed in the 1990s and shown to be of no benefit. Corticosteroids, osmotic diuresis and the use of frusemide is no longer

TABLE 3

HOSPITAL MANAGEMENT OF NEAR DROWNING

From Brown and Piantadosi Near-drowing; hospital management.[3]

General Measures
Check for predisposing factors
Check for spine and skull fractures
Sinus, ear, skin barotrauma may serve as portals for infection

Management in ICU
Inotropes, fluids, monitoring
Electrolyte abnormalities etc.

Respiratory Care
Initial chest X-ray may be 'normal'
IPPV +/- PEEP
Accurate microbiology as required

Brain resuscitation
— HYPER reviewed - no benefit
— Use of corticosteriods, osmotic diuresis, hypothermia not advocated
— ICP monitoring - no benefit

advocated, nor is hypothermia though to be useful. No benefit has been shown from intracranial pressure monitoring which was popular in the 1980s. Brown and Piantadosi used a classification system of near-drowned people. It involved assessment at 1-2 hours after resuscitation. Category A were awake and fully conscious, category B had blunted consciousness but were rousable and category C were comatose. They state that about 80% of child and adult near-drowning victims survive without sequelae and 2-9% survive with brain damage. About 12% of all near-drowning victims die. About 90% of category A and B and about 50% of category C patients survive with full recovery. About 10-23% of category C patients survive with permanent neurological injuries.

Chris Dueker presented a paper on *Myths in near-drowning* in which he discussed laryngeal spasm.[3] He also debunked dry drowning. Breathing against a closed glottis will cause a negative intrathoracic pressure, which may cause pulmonary oedema. Just before death from anoxia the vocal cords relax and may allow fluid to be aspirated. In my early anaesthetic days consultants cheered their juniors by saying "If you just keep trying to oxygenate the patient, the cords will open just before he dies and you can get some oxygen into him then". I never waited for that but used to use suction fairly quickly.

Dueker discussed so-called protective role of hypothermia and discounted this in divers because divers actually do wear suits to protect them from becoming hypothermic. This protection is limited and can be overcome by long exposures to cold water. Dueker considered that in most waters divers will not get any protective effects from hypothermia. He disagreed with the use of the Heimlich manoeuvre (an abdominal thrust), which is the agreed primary treatment of respiratory obstruction by foreign objects, in near drowning as respiratory obstruction is rare in near-drowning and when it occurs is usually aspirated material, which will need to be removed by a finger or repositioning rather than by delaying resuscitation by squashing the belly and perhaps increasing hypoxic injury. The Heimlich manoeuvre should not be used in near-drowned people to try and clear the lungs of fluid because it will not do that.

Other topics discussed were *Open water rescues and field resuscitation* by Dennis Graver.[4] Drew Richardson has a paper to present at this meeting on these topics. Bill Hamilton, who was our guest speaker in the Maldives in 1996, spoke on *Rescues in special circumstances*.[5] His presentation was as good as the presentations he gave in the Maldives, I don't go on any further than that, and Claes Lundgren presented an excellent paper, *Does the cardiovascular diving response have a protective effect in near drowning incidents?* which I decided not to review here.

Carl Edmonds also presented a paper on the mechanisms of the drowning syndromes,[6] and Chris Dueker presented a paper on *Expectations for recovery*.[7]

Two papers about drowning appeared in the South Pacific Underwater Medicine Society Journal in 1997 and 1998 and were forerunners of Carl Edmonds' two papers at the UHMS Workshop in 1977 which was published in 1999.[8,9]

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The following paper, presenting the research carried out by Dr David Williams for the award of the SPUMS Diploma in Diving and Hyperbaric Medicine, is reproduced with permission from DL Williams, DJ Doolette, RN Upton, "Increased cerebral blood flow and cardiac output following cerebral arterial air embolism in sheep", *Clinical and Experimental Pharmacology and Physiology* (2001) Vol. 28: 868-872. The *Clinical and Experimental Pharmacology and Physiology* website can be accessed at <http://www.blackwell-science.com/cep>.

INCREASED CEREBRAL BLOOD FLOW AND CARDIAC OUTPUT FOLLOWING CEREBRAL ARTERIAL AIR EMBOLISM IN SHEEP

David J Williams, David J Doolette and Richard N Upton

Keywords

Air embolism, sheep, cerebrovascular circulation, blood flow velocity, cardiac output, physiopathology, blood pressure, heart rate, capnography.

Summary

1 The effects of cerebral arterial gas embolism (CAGE) on cerebral blood flow and systemic cardiovascular parameters were assessed in anaesthetised sheep.

2 Six sheep received 2.5 ml injection of air simultaneously into each common carotid artery over 5 seconds. Mean arterial blood pressure, heart rate, end-tidal carbon dioxide, and an ultrasonic Doppler index of cerebral blood flow were monitored continuously. Cardiac output was determined by periodic thermodilution.

3 Intracarotid injection of air produced an immediate drop in mean cerebral blood flow. This drop was transient and mean cerebral blood flow subsequently increased to 151% before declining slowly to baseline. Coincident with the increased cerebral blood flow was a sustained increase in mean cardiac output to 161% of baseline. Mean arterial blood pressure, heart rate, and end-tidal carbon dioxide were not significantly altered by intracarotid injection of air.

4 The increased cardiac output is a pathological response to impact of arterial air bubbles on the brain, possibly the brainstem. The increased cerebral blood flow is probably the result of the increased cardiac output and dilation of cerebral resistance vessels caused by the passage of air bubbles.

Introduction

Cerebral arterial gas embolism (CAGE) results from the introduction of gas into the arterial circulation and the passage of gas bubbles to the cerebral circulation. By far the most common cause of CAGE is the introduction of air into arteries during surgical access to cardiovascular system, particularly cardiopulmonary bypass assisted procedures. Massive air embolism resulting in stroke or death is rare but cerebral gas micro-emboli occur in most cardiopulmonary bypass assisted operations and may contribute to the neurobehavioral and cognitive dysfunction found in up to 79% of patients following cardiopulmonary bypass.[1] A much less common cause of CAGE is decompression illness that results from intracorporeal bubble formation during a reduction in ambient pressure (decompression) such as occurs in undersea diving.

Injection of 0.05–1.0 ml/kg air or blood/air foam into the carotid or vertebral arteries of anaesthetised cats, dogs, or rabbits is followed by a changes in brain function such as persistent decrease in amplitude of somatosensory evoked responses[2-4] and profound changes in cerebral blood flow. There is a transient drop in cerebral blood flow during the passage of air through the cerebral circulation.[5] An increased global cerebral blood flow has been demonstrated during the subsequent 1-2 hours.[5,6] There is a persistent dilation of cerebral pial arteries following the passage of bubbles[3,5] and increase in intracranial pressure.[2,6] Other studies have shown localised decreases in cerebral blood flow during the hours following the passage of gas.[2,3]

Cerebral hyperaemia and a rise in intracranial pressure have been proposed as a primary pathogenic mechanisms of CAGE,[6,7] as in other head injury. However, the observations of both high and low cerebral blood flows following CAGE have not been adequately reconciled and the mechanism of increased flow has not been investigated.

We have developed a chronically instrumented sheep preparation that can provide useful insight into cardiovascular and cerebrovascular physiology. Central to this model is the measurement of cerebral blood flow by an ultrasonic Doppler method that allows continuous real-time measurement of global cerebral blood flow. The aims of this study were to assess the effects of CAGE on cerebral blood flow and any parallel systemic cardiovascular changes.

Methods

EXPERIMENTAL PREPARATION

All surgical and experimental procedures received University of Adelaide Animal Ethics Committee approval

and were conducted in accordance with the Australian Code of Practice for the Care and Use of Animals for Scientific Purposes (National Health and Medical Research Council of Australia).

Six adult female Merino sheep with a nominal weight of 50 kg and aged between 2 and 3 years were prepared under anaesthesia (1.5 g thiopentone induction, 1-2% halothane maintenance) as previously described.[9,10] Instruments included a sagittal sinus ultrasonic Doppler flow probe and in one sheep, an extradural Codman type pressure transducer placed via a craniotomy, as well as arterial and central venous catheters and a pulmonary artery flotation catheter, all placed via femoral vessels. Following instrumentation, the sheep recovered from anaesthesia and lived in mobile metabolic crates for at least one week with free access to food and water.

On an experimental day, the sheep were again anaesthetised and mechanically ventilated via an endotracheal tube using a closed circuit anaesthetic system and 40% oxygen (balance nitrogen). Both common carotid arteries were exposed and cannulated with 4 Fr catheters (Cordis Europa NV, Roden, Netherlands) inserted to a depth of 20 cm using the Seldinger technique and flushed with heparinised 0.9% saline (10 i.u./ml) at regular intervals thereafter to prevent clotting. They were sutured to a plastic mounting plate, which was in turn secured to the overlying skin. Hypodermic needles (26 gauge) were placed subcutaneously in both fore legs and the right hind leg as electrocardiogram electrodes.

Ultrasonic Doppler frequency shifts from the sagittal sinus probe were recorded at a sampling rate of 1 Hz using a four-channel pulsed Doppler flowmeter (Bioengineering, University of Iowa, Iowa City, IA, USA) and an analog-to-digital card (DAS 16-G2, Metrabyte Corp., Taunton, MA, USA) in a personal computer. Because sagittal sinus Doppler probe frequency shift is linear with sagittal sinus outflow, this provides a continuous index of global cerebral blood flow . Mean arterial blood pressure (MABP), measured using a transducer on the arterial catheter, and intracranial pressure transducer output were recorded using the same data acquisition system.

Electrocardiogram and heart rate was displayed on a Cardiocap monitor (Datex, Helsinki, Finland). End-tidal carbon dioxide ($ETCO_2$) was continuously displayed by side sampling infrared spectrometry using the Cardiocap monitor and was maintained near 40 mm Hg by altering ventilation. Values of heart rate and $ETCO_2$ were recorded manually.

Cardiac output was measured by the intermittent thermodilution technique, using a dedicated cardiac output computer (Abbott Laboratories, North Chicago, IL, USA) and 10 ml of room temperature 0.9% saline as the indicator injected via the pulmonary artery flotation catheter with integral thermistor (Biosensors, Singapore). Pulmonary artery temperature was otherwise maintained at 38-39.5°C using passive insulation.

EXPERIMENTAL PROTOCOL

The anaesthetised sheep were placed upright in the "sphinx" position, the neck erect and the head facing forwards. A period of one hour was allowed for the induction agent to be cleared from the blood. Once steady state values of all variables were achieved, baseline readings were made of all variables during a period of 5 minutes. Then, at 5 minutes, and accounting for cannula dead space of 0.5 ml, 2.5 ml of air was then simultaneously injected into each carotid artery (5 ml total dose) over a period of 5 seconds. The resulting changes in the above parameters were measured and recorded for a period of 1 hour following injection. At the end of this period, the sheep was sacrificed by lethal injection of 4 g phenobarbitone sodium (Lethabarb, Fort Dodge Animal Health, Sligo, Ireland.

STATISTICAL ANALYSIS

Doppler frequency shift and MABP were smoothed using a 9-point moving average and values at the same time points as the manual cardiac output, heart rate, and $ETCO_2$ measurements extracted for graphical presentation and statistical analysis. Owing to failure of instrumentation, heart rate and MABP were only recorded in five sheep, cardiac output recorded in four sheep, and one experiment was terminated at 25 minutes. For each sheep, the measurements of each variable from 0 to 4.5 minutes were averaged to provide a single baseline value for statistical analysis. As an index of cerebral blood flow, Doppler frequency shift is given as percentage change from this baseline. Each variable was analysed by one-way ANOVA with the single factor time as a repeated measure. The Greenhouse-Geisser adjustment for violation of the assumptions of compound symmetry and sphericity was applied to the *F* test. Where the *F* test was significant, the baseline value was compared to each subsequent time point using Dunnett's post-hoc test (2-tailed $\alpha = 0.05$). Analysis of variance, either excluding data from the shortened experiment or excluding time points beyond 25 minutes gave similar results. $ETCO_2$ and sagittal sinus Doppler signal were tested for potential covariation by product-moment correlation. Statistical calculations were performed using the basic statistics and general linear model modules of Statistica '99 (Statsoft, Tulsa, OK, USA).

Results

CEREBRAL BLOOD FLOW

All of the sheep exhibited a similar characteristic three phase response to injection of intracarotid air that is

evident in the mean data in figure 1a.

Phase 1

Within 30 seconds of injection of the air bolus, there was a rapid, transient drop in individual continuous Doppler signal (data not shown) to near zero and then a rapid return towards the baseline during the subsequent 30 seconds. The minimum mean Doppler signal of only 47% of baseline in figure 1a is an aliasing artefact because the timing of the brief signal loss was not identical between individual sheep. During the return of flow, the characteristic "chirping" sound of bubbles passing the probe could be heard on the audio output.

Phase 2

Accompanied by the frequent bubble sounds, Doppler signal increased above baseline during several minutes following injection of air.

Phase 3

Subsequently, bubble noise became infrequent and eventually stopped and the Doppler signal steadily declined towards and occasionally below the baseline.

In the one sheep where it was measured, intracranial pressure exhibited a similar response to that of the sagittal sinus Doppler signal, a transient drop followed by overshoot of baseline. Product-moment correlation of intracranial pressure and Doppler signal in this sheep produced a Pearson's $r = 0.98$ ($P < 0.001$).

END-TIDAL CO_2

End-tidal CO_2 typically fluctuated around baseline following intracarotid injection of air (figure 2a), these changes were not statistically significant ($P = 0.133$; Greenhouse-Geisser adjusted F test, d.f.=10) and were too small to account for the cerebral blood flow changes. End-tidal CO_2 did not correlate significantly with Doppler signal in any sheep, Pearson's r ranged from - 0.15 to 0.30 ($P > 0.20$). Five of the sheep maintained a relatively steady $ETCO_2$ throughout the experimental period without changing ventilation. However, in sheep no. 5, $ETCO_2$ dropped suddenly to 36 mmHg at three minutes following injection of air and then rose to 48 mmHg at five minutes following injection of air, coincident with a fall and then rise in cardiac output, respectively. These changes required the minute ventilation to be reduced to 50% and subsequently increased to 200% in order to maintain constant $ETCO_2$. By 10 minutes, the ETCO2 had stabilised and returned to normal with baseline minute ventilation. The elevated $ETCO_2$ of 48 mmHg coincided with the maximum cerebral blood flow of 230% of baseline in this sheep, however the level of hypercarbia would only be expected to increase cerebral blood flow to 150% baseline[9]

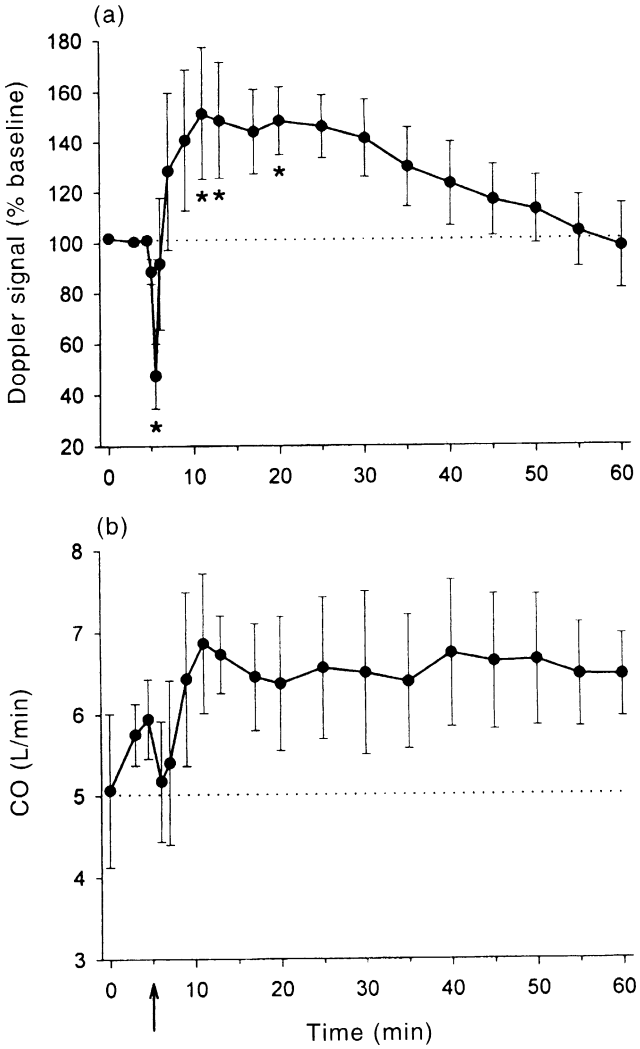

Figure 1. (a) The cerebral blood flow index is shown as the sagittal sinus Doppler frequency shift as percentage of baseline on the y-axis over the duration of the experiment in minutes on the x-axis. (•), selected data points extracted from the continuous signal are the mean of six sheep until 25 minutes and five animals thereafter. *$P < 0.05$ for times where the mean Doppler signal was significantly different from the baseline value (2-tailed Dunnett's test, d.f.=50). (b) Mean (n=4) cardiac output (CO) is shown on the y-axis over the duration of the experiment. On each panel, the horizontal dotted line indicates the mean baseline value and error bars indicate ± 1 SEM. The arrow below the x-axis indicates the time of intra-carotid injection of air.

MEAN ARTERIAL BLOOD PRESSURE, HEART RATE AND ELECTROCARDIOGRAM

Although there was no significant difference in mean MABP ($P = 0.263$, Greenhouse-Geisser adjusted F test, d.f.=5, see figure 2b), there was a brief rise in MABP during the first five minutes following injection of intracarotid air in most sheep.

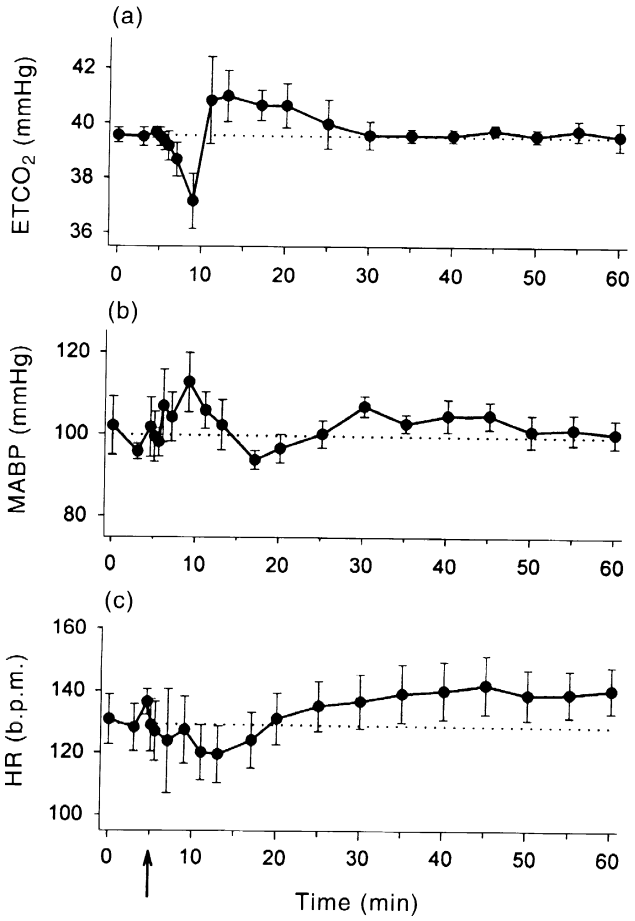

Figure 2. (a) End-Tidal CO_2 ($ETCO_2$) is shown on the y-axis over the duration of the experiment,in minutes, on the x-axis. Data are the mean of six sheep until 25 minutes and five animals thereafter. (b) Selected data points for mean arterial blood pressure (MABP) extracted from the continuous signal are the mean of five sheep until 25 minutes and four animals thereafter. (c) Heart rate data are the mean of five sheep. On each panel, the horizontal dotted line indicates the mean baseline value and error bars indicate ±1 SEM. The arrow below the x-axis indicates the time of intracarotid injection of air. No values were significantly different from the baseline value (see text).

The ANOVA indicated no significant variations in heart rate (P =0.076, Greenhouse-Geisser adjusted F test, d.f.=8, figure 2c). The heart rate response was not homogenous across sheep; following injection of intracarotid air, marked electrocardiogram changes were noted in 3 of the sheep (no.s 1, 4 and 5). Sheep no. 1 developed a sinus bradycardia of 40 beats per minute (bpm) one minute after injection of intracarotid air, which gradually returned to the baseline rate after five minutes. Sheep no. 4 began with a baseline heart rate of 135 bpm. One minute after injection of intracarotid air, ventricular bigemini developed at a rate of 140 bpm, with the addition of many multifocal ventricular ectopics after two minutes. Bi- and trigemini then developed and persisted for four minutes, before alternating with brief periods of sinus rhythm. The periods of sinus rhythm became longer, and those of bigemini shorter until sinus rhythm of 139 bpm was re-established after nine minutes. Sheep no. 5 had a baseline heart rate of 131 bpm. Two minutes after injection of intracarotid air, ventricular bigeminy occurred. This was followed one minute later by an episode of supraventricular tachycardia, which persisted for two minutes, before returning to sinus rhythm at 120 bpm. Upon return to sinus rhythm, T wave inversion was noted, which persisted until the end of the experiment.

CARDIAC OUTPUT

Following injection of intracarotid air, there was a substantial increase in mean cardiac output reaching a maximum of 161% of baseline at six minutes, coincident with the peak mean cerebral blood flow. Cardiac output remained elevated throughout the study (figure 1b). Whereas this increase was significant according to the unadjusted F test and 2-tailed Dunnett's test (P <0.05, d.f.=45), it was not significant according to the Greenhouse-Geisser test (P =0.22, d.f.=5). In one sheep (no. 5), cardiac output decreased briefly during a period of cardiac arrhythmias before increasing markedly above baseline.

Discussion

CEREBRAL BLOOD FLOW

Transient low flow

The initial transient drop in Doppler frequency shift following injection of air into the carotid arteries could coincide with passage of a long column of air beneath the Doppler probe at normal blood velocity. It more likely results from an actual drop in cerebral blood flow due to temporary trapping of air bubbles in cerebral resistance vessels.[3] In support of this interpretation, the persistent bubble sounds for several minutes following return of cerebral blood flow indicate temporary trapping of gas in the cerebral circulation. In addition, continuous measurement of heat conductance using a thermocouple on the frontal cerebral cortex of cats show the same drop in cerebral blood flow index following injection of 0.6 ml air/blood foam into the common carotid arteries.[5] Most other animal studies fail to detect this transient drop owing to use of periodic flow measurements.

Sustained flow increase

The sustained increase in global cerebral blood flow following injection of 5 ml intracarotid air found in the present study is of similar time course to that found by thermocouple or sagittal sinus ^{133}xenon clearance following CAGE in cats.[5] The increases in Doppler and thermocouple indices were of similar magnitude but sagittal sinus ^{133}xenon clearance was greater. External detection of ^{133}xenon clearance during the 5 to 10 minutes immediately following

CAGE finds increased cerebral blood flow in cats and dogs of similar magnitude to the present study.[5,6]

In contrast, cerebral blood flow measured by initial hydrogen clearance shows a progressive and sustained decrease during 3 hours following injection of 0.025–0.4 ml air into the internal carotid in urethane anaesthetised rabbits.[3] Autoradiograms of [^{14}C]-iodoantipyrine clearance show adjacent localised areas of very low and very high cerebral blood flow after 214 minutes of recompression therapy following injection of 0.4 ml air into the internal carotid plus an i.v. bolus of 10 µg/kg noradrenaline in pentobarbital anaesthetised dogs.[2] Areas of low cerebral blood flow were uncommon in dogs following CAGE without concomitant transient systemic hypertension.[2] Whether species and anaesthetic differences can explain these different pathological responses of cerebral blood flow is unknown but, in halothane anaesthetised sheep, cerebral blood flow physiological response to varying $ETCO_2$ is robust[9] and unchanged compared to awake animals (RN Upton et al. unpublished observation, 1999).

The discrepancy in cerebral blood flow findings between studies is, to some extent, explained by the different methods of measurement. The present sagittal sinus Doppler outflow method, the thermocouple method[5] and external detection of ^{133}xenon clearance[5,6] all measure total blood flow of the tissue area under detection, irrespective of any local flow heterogeneity. These methods all find a similar magnitude of increased global cerebral blood flow following CAGE. Sagittal sinus ^{133}xenon clearance indicates flow through perfused tissue only, which shows the largest increase following CAGE . The initial slope of hydrogen clearance curves measures the fastest component of microcirculatory blood flow in a very small volume of the superficial cortical tissue surrounding the implanted electrode tip, and is shown to decrease following CAGE.[5] The photodensity of [^{14}C]-iodoantipyrine autoradiographs are difficult to equate with the present study because only single determinations are possible; these were made following CAGE and therapeutic recompression, there were no pre-embolism measurements and no untreated controls were included for comparison. Nevertheless, these data do suggest local heterogeneity in cerebral blood following CAGE.[2] It seems likely that CAGE causes a marked heterogeneity in cerebral blood flow with an overall increase in global cerebral blood flow but reduced blood flow in some cortical regions.

In addition to the limitations of the autoradiographic method already mentioned, cerebral blood flow measurements following CAGE using ^{133}xenon or hydrogen clearance methods[3,5,6] must be interpreted cautiously. These methods rely on the brain:blood gas partition coefficient; this may change with introduction of an intravascular gas phase changing the effective solubility of the blood/free gas mixture and, therefore, cause an artefactual change in the calculated flow. The ultrasonic Doppler index of blood flow allows continuous real-time measurement of global cerebral blood flow, can catch the rapid changes early after embolism and flow measurement is not influenced by the presence of intravascular micro-bubbles. An additional advantage is that bubbles can be detected due to their high acoustic reflectivity, which produces a characteristic Doppler signal.

SYSTEMIC CARDIOVASCULAR CHANGES

Cardiac output has not previously been measured during experimental CAGE. In the present study, CAGE produced a sustained increase in cardiac output. This is an unusual cardiac response given that heart rate and MABP remain relatively unchanged and is likely a pathological response to CAGE rather than a normal cardiovascular compensatory mechanism. Because there was no significant change in heart rate, the increased cardiac output must result from greater stroke volume. This could be due to increased end-diastolic ventricular volume because of increased venous return or increased ventricular contractility; left ventricular pressure and contractility have been shown to increase transiently following CAGE in cats,[11] but this was not measured in the present study. Increased stroke volume could also result from reduced afterload; although afterload was not measured in the present study, there was no significant change in MABP despite the large sustained increase in cardiac output, suggesting that CAGE must reduce systemic peripheral vascular resistance.

Some studies have similarly shown no change in heart rate or MABP following injection of air into the carotid arteries of dogs and rabbits.[2,3] A transient rise in MABP is found after injection of air into the common carotid artery of dogs[5,7] but this has been attributed to manipulation of the carotid vessel.[7] Cardiovascular changes are more associated with injection of air into the vertebral arteries of cats and dogs, and studies report transient (10 minute) rise in MABP and, variously, a transient increase[11] or decrease[6] in heart rate. In the present study the cardiac arrhythmias observed in the first five minutes after injection of intracarotid air in sheep are consistent with the findings following intra-vertebral air injections in cats and dogs.[5,11]

MECHANISMS

The amount of gas reaching the cerebral arterial circulation as a result of surgical procedures or decompression illness is not well defined, but the 5 ml of air injected in the present study is a relatively small dose (0.1 ml/kg) compared with other experimental studies. Air injected into the common carotid arteries will distribute with buoyancy and flow to the brain. Bubbles may trap temporarily in the cerebral resistance vessels but will mostly be shunted through the cerebral circulation by the perfusion pressure.[3] Bubbles entering the venous circulation will be

filtered by the lungs before reaching the systemic arterial circulation.[12] Thus, both the cerebral blood flow changes and the systemic cardiovascular effects of intracarotid air injection must result from the impact of bubbles on the brain.

Sheep have no internal carotid artery and all blood to the brain passes via the external carotid through the carotid rete arteria and supplies the entire brain including the brainstem.[13] Thus, the air injected into the common carotid should be distributed to all brain structures. The cardiovascular effects of CAGE in other species have been attributed to neurohumoral effects resulting from a greater impact of bubbles on the brainstem via the vertebral-basilar arteries.[11] The cardiovascular effects in the present study may also be due to the effect of bubbles on the brainstem because, in the sheep, the brainstem is supplied by the carotid arteries.[13] Therefore, these cardiovascular effects may be caused by a sustained alteration in autonomic discharge. However, pharmacological and surgical blockade of the autonomic nervous system in cats does not completely abolish the cardiovascular response to CAGE.[11]

The increase in cerebral blood flow following CAGE in the present study mirrors the increase in cardiac output. There was no evidence of increased perfusion pressure since MABP was unchanged but there is evidence of increased intracranial pressure in this and other studies.[2,6,7] It seems likely that there is a generalised cerebral vasodilation. Indeed, cerebral pial arterioles dilate following CAGE.[3,5] In the one sheep where it was measured, intracranial pressure increased in parallel with cerebral blood flow and may reflect such vasodilation. For these reasons and because it may be an important pathogenic mechanism, it is unfortunate that we were unable to measure intracranial pressure in all animals. In the present study, the cerebral vasodilation may result from the same mechanism as the systemic cardiovascular responses, possibly an action of bubbles on the brainstem. However, cerebral vasodilation and rise in intracranial pressure is reported in models where impact of bubbles on the brainstem is not expected[2,3,5,7] and may be a local effect of bubbles on the cerebral vasculature.

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WE ARE JUST A LOAD OF LEMMINGS

Monty Halls

Key words
Equipment, recreational diving, safety, traiuning.

Why do sport divers make life so difficult and unsafe for themselves, using kit which no self respecting professional would countenance? There are three areas crying out for improvement.

We were in the shadowy lee of a wreck in Australia. While I, a mere trainee, satisfied myself with the bizarre sensation of exploring a man-made object in 3D flight, my buddy was after crayfish Spotting a likely nook in the hull, he disappeared into it like a mole, leaving only a pair of huge neoprene buttocks and failing fins in view. His arms must have been working like mechanical grabs, stuffing bewildered crustaceans into a heaving catch bag. Then came the extraordinary sight of my buddy reversing abruptly out of the hole, like a cork from a bottle. He executed a neat handbrake turn and shot towards me, beard trailing attractively in his wake. His eyes bulged like ping-pong balls, and one hand extended towards me in a crooked claw. Remembering my drills, I carefully angled my shiny new octopus towards him. He arrived a nano-second later, 15 stone (95.5 kg) of barrelling Australian. Colliding with me, he grabbed my regulator from my slack jaw, and sucked in whooping lungfuls of air, leaving me to wrestle with him, my octopus and my rising sense of indignation. We moved towards the surface, me glowering reproachfully.

It has dawned on me that we run-of the-mill sport-diver types take part in one of the most dangerous forms of diving. Cave and free-divers might just pip us, but compared to technical, commercial, police, media and scientific divers, we ask for trouble every time we enter the water. Credit for this theory must go to veteran commercial divers Mark Hagger and Martin Versvelt. Through long debate they have brought me round to the view that we sport divers are naive, outdated, resistant to change and bent on ignoring fundamental truths about safety.

The first big issue leaves other sections of the diving community agog at our rampant stupidity. Most of us (BSAC divers with an in-built bottle in their BCs are excused here) dive without an alternative air source. And before you start waving your yellow octopus at me, that isn't an alternative, it's another regulator running off the same first stage. Why do divers in every other sector insist on a totally redundant system to supply them should their main supply cut out? Because they have learned the hard way. Try to get a working dive using only a single cylinder in even just a choppy sea past a Health and Safety risk assessment, and you would be laughed out of the rather oily Portakabin from which these chaps seem to operate.

Try to get a military or police diver to dive under such circumstances and you would be taken round the back and beaten by large men in moustaches. Yet we crawl over wrecks, clamber over line-strewn reefs and drift in limited visibility into God knows what, while breathing wheezily from our single cylinder. If that packs up, there's always your buddy, of course, if you can find him, if he can get his octopus released in time, if you both remember your drills. And you can put that Spare Air away, too, don't even start me on that. But wouldn't it make sense to have some sort of official insistence that all divers carry a totally independent alternative air source?

On to weight belts: why do we insist on holding them on with a simple clip that can be flipped open if you catch it on something, invariably with a flapping length of belt sticking out from the buckle, as we've been carefully taught to leave it loose? Most of us have experienced losing or nearly losing a belt, or know someone who has. The solution? A sequential release (thank you, Martin Versvelt). Not nine different straps and pulleys, but a simple, standardised pin and clip. You pull the pin, you flip the clip. Incorporate it in training, and it would be second nature within weeks. Had we always dived with a Pin-and-clip-release weight belt, and someone appeared with just a clip and a flapping loose end of belt, he would be laughed off the boat.

Dive kit has a large fashion element to it. A few years ago, large knives were out, the smaller the knife, the more PC you were. I sported a veritable toothpick tiny and subtle, sufficiently understated to let people know I had no issues with my masculinity, and didn't require nine inches of gleaming blade to justify the pitiful contents of my PADI posing pouch. Bring big knives back! The ones with a hammer thing in the handle, and a bloody great big serrated edge. And that phone cord thing holding it onto your thigh. Why? Because it's a tool. The chances of having to hammer, hack or saw your way out of a crisis are slim, but if you have to, better to be wielding a Neptune Big Boy than a Gucci Silver.

Next time you dive, pause for thought. You or someone else could be about to enter the water on a 30 m wreck dive with a single cylinder, a tenuously clipped-on weight belt and a tiny daggerette. Would a working diver do the same dive with your gear? No, and I reckon there's a reason for that.

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2 Lippmann J and Bugg S. *The diving emergency handbook*. Melbourne: J.L.Publications, 1985

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Information may be sent (in confidence) to:

Dr D. Walker

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

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

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The aim of this investigation is to establish a data base of divers who dive or have dived with any medical contraindications to diving. At present it is known that some asthmatics dive and that some insulin dependant diabetics dive. What is not known is how many. How many with these conditions die is known. But how many dive safely with these conditions is not. Nor is incidence of diving accidents in these groups known.

This project is under the direction of Dr Douglas Walker and Dr Mike Bennett. The investigation has been approved by the Ethics Committee of the Prince of Wales Hospital, Randwick, approval number 01/047.

If you are in such a group please make contact. All information will be treated as CONFIDENTIAL. No identifying details will appear in any report derived from the data base.

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