# *The Editor's Offering*

In this issue of the Journal we have a study of diving habits and divers' behaviour from the UK. It is an unusual study for the Journal to publish as it has no close connections with underwater or hyperbaric medicine and very little to do with diving safety, more a catalogue of disasters. There is no control group and it is purely anecdotal. But it made the Editor laugh so much when he read it in the December 1997 issue of DIVER that he thought that all our readers should be able to share the fun. Turn to page 120 for the first instalment of Rico's *The Sea People's Guide to Divers*. Unfortunately we cannot run to colour for these illustrations so if readers want to see the original colours they can contact DIVER (the address is on page 120) to see if they can get a back copy.

Publishing informative cartoons in the Journal has a long history. From the first issue to Volume 18 (3) 41 issues had a cartoon dealing with issues of diving safety or underwater medicine on the front cover. Many of the 18 issues with cartoonless covers had cartoons among the text.

Another new item we wish to bring to our readers' attention is on page 78 where Best Publishing Company is advertising Bennett and Elliott in hard cover at about half the price it was when we reviewed in the Journal in 1993. Exactly the same book at a price lower than most medical textbooks, a definite "must" for every diving doctor's library.

With this Journal comes the brochure for the 1999 Annual Scientific Meeting (ASM) to be held in Layang Layang, a Malaysian island reached from Kota Kinabulu. SPUMS has booked the whole resort, but there are only 78 rooms available so when they are full that is it. There is nowhere else to sleep for many hours of boat travel. The meeting is "An International Workshop on the Treatment of Decompression Illness". Besides an interesting scientific program there is lovely diving only minutes from the resort.

Which leads us to the paper on page 84 where we see in print the system for scoring severity and measuring recovery in decompression sickness presented at the 1997 ASM in Waitangi by Simon Mitchell. It appears complicated at first sight, but the complications are a more appearance than reality as every step is simple. The great thing about the system is that can give an index of severity (a number) at presentation and an index of recovery, obtained by subtracting the index of severity at review from that at presentation. The difference is the recovery , or improvement, index.

A group of papers, again from Waitangi, focuses on First Aid training for divers. In the Editor's opinion leaving First Aid teaching to a course taken after open water training will miss a large proportion of new divers as the proportion of divers going on to do other courses is low, as admitted by Bill Day from New Zealand. Oxygen administration and fluids by mouth will help the vast majority of sufferers from diving accidents, most of whom do not report to hospital. In Australia the Resuscitation Council (ARC) thinks that a knowledge of general first aid is a prerequisite for training in oxygen administration.

With no good statistics of how divers injure themselves we are forced to rely on anecdotal evidence from divers. And of course the afflictions of divers vary with the weather, water temperature and competence of the divers themselves. Running out of air underwater is not as common as it was (In 1977 5 SPUMS members attending the ASM in Truk had to be rescued and brought to the surface by the safety divers, brought from Australia, hovering above them for just such occasions, on the first day's diving) but it still happens. The whole of the first aid for air embolism can be summed up as position and oxygen. A breathing air embolism victim has a chance, but an apnoeic and pulseless one is probably best left as dead. Water inhalation is a very common problem for divers so it seems sensible to relax the first-aid-first rule in the case of divers, who are seldom apnoeic when they would benefit from oxygen administration and usually have intact bodies, except for bubbles. Divers are liable to unusual ailments which do not afflict those who confine themselves to land. Oxygen administration is the most useful first aid routine for diving associated injuries. There is a good case to be made for training all divers in oxygen administration to a breathing patient without having to learn normal first aid first.

It is interesting that the BS-AC, which used to teach oxygen administration as an add-on course, has changed to including it in the basic teaching. This has been associated with a demand by divers for diving boats to carry oxygen which has been responded to by boat owners. If most divers know how to use oxygen apparatus the chances of having a competent administrator in each dive boat will be vastly increased. However the likelihood of this happening in Australia is low due to both the practices of the training agencies and the requirements of the ARC for a first aid training before learning to administer oxygen.

However our members with an interest in hyperbaric medicine should not feel that they have been ignored. There are two papers on ventilators under pressure in a chamber. It is clear that most of the ventilators used in Australian hospitals are a long way from the ideal hyperbaric ventilator for they cannot provide an adequate minute volume at depth. However, some ventilators, designed for hyperbaric use,which maintain their output unchanged as the patient dives to 18 m are available.

# ORIGINAL ARTICLES

## VENTILATOR FUNCTION UNDER HYPERBARIC CONDITIONS

Marcus Skinner

**Key Words**

Equipment, hyperbaric research, ventilators.

**Abstract**

Assisted ventilation in the hyperbaric chamber presents challenges and risks due to machine and patient related problems. These problems and how to minimise them are discussed. The requirements of the ideal ventilator for use under pressure are detailed and so far few ventilators have satisfied these requirements.

### Introduction

In addition to diving related injury, hyperbaric oxygen (HBO) therapy is used in the treatment of some critically ill patients for whom ventilatory support is often required (Table 1). The indications for endotracheal intubation and mechanical ventilation may include acute respiratory failure, the need for airway patency and protection from aspiration due to loss of airway reflexes and for the manipulation of blood pH. Currently accepted indications for hyperbaric oxygen therapy have been reviewed by Jain[1] and are beyond the scope of this paper.

The use of hyperbaric oxygen in the treatment of critically ill patients requires prolonged respiratory support in the chamber. Little information exists, in the respiratory and hyperbaric literature, both on the use of ventilators under hyperbaric conditions[1-6] and the evaluation of individual ventilators under hyperbaric conditions.

**TABLE 1**

**DISORDERS WHERE VENTILATORY SUPPORT UNDER HYPERBARIC CONDITIONS HAS BEEN INDICATED**

| |
|---|
| Cerebral air embolism |
| Decompression illness |
| Carbon monoxide poisoning |
| Smoke inhalation |
| Closed head injury cerebral oedema |
| Cyanide poisoning |
| Near drowning |
| Severe sepsis |
| Multiple trauma |

The majority of patients receiving ventilatory support require augmentation of alveolar ventilation to decrease the work of breathing in hyperbaric chambers. No outcome data exists to guide the choice of ventilatory support in hyperbaria.

Assisted ventilation in the hyperbaric chamber presents challenges and risks due to machine or patient related problems. The characteristics of particular ventilators need to be understood to appreciate the likely degradation of performance under hyperbaric conditions.[7] The majority of ventilators used in chambers are pneumatically controlled and time cycled. The characteristics and physiological effects of these ventilators are reviewed below.

To use a ventilator safely the hyperbaric specialist must be able to predict any changes that may affect ventilator function under hyperbaric conditions in order to choose the most appropriate ventilator for the patient's needs. This can only be done with a thorough understanding of ventilators and how they function.

### Ventilators used in the hyperbaric environment

Intermittent positive pressure ventilation (IPPV) did not receive widespread use until the 1950s when positive pressure ventilation was used effectively on patients during the polio epidemics in Denmark and Sweden. During this period ventilators were also shown to be reliable for use during anaesthesia and for post-operative ventilatory support.

The first published reports of ventilator function under hyperbaric conditions appeared in the mid 1970s.[8] In 1982 Saywood et al. recommended the use of the Penlon Oxford ventilator as it maintained a set tidal volume up to 6 bar of air and up to 31 bar ATA in oxy-helium.[9]

### Ventilator classification

A ventilator is a device used to move gas into the lungs. The classification of ventilators has been confused by various authors producing different classifications.[10-14]

Two aspects of performance are particularly important when classifying a mechanical ventilator.

1 Functional characteristics, which include factors controlling the pressure and the flow rate of the gas delivered to the patient.

**TABLE 2**

**VENTILATOR CLASSIFICATION BASED ON PHASES OF THE MECHANICAL CYCLE**

| | |
|---|---|
| **1** | **Inspiratory Phase** |
| | Flow generation |
| | Pressure generation |
| **2** | **Inspiration to Expiration trigger (Cycle)** |
| | Pressure cycled |
| | Volume cycled |
| | Time cycled |
| **3** | **Expiratory Phase** |
| | Positive end-expiratory pressure (PEEP) |
| | Negative end-expiratory pressure (NEEP) |
| **4** | **Expired to Inspired trigger** |
| | Intermittent mandatory ventilation (IMV) |

2 Operational features, which include its power source (electrical, pneumatic, spring tension or weighted bellows), patient circuit, alarm capability, controls and the provision of special modes of ventilation, e.g. positive end expiratory pressure (PEEP) and continuous positive airway pressure (CPAP).

It is now generally accepted that the most useful classification of ventilators for clinical application is according to the phases of the cycle.[15] Table 2 summarises this classification.

**Inspiratory Phase (Gas Flow Production)**

Inspiratory gas flow occurs when the proximal airway pressure is higher than alveolar pressure. In the non-breathing person tidal volume may be produced by flow or pressure generation.

FLOW GENERATORS

Constant flow generators produce flow at a constant rate despite changing pulmonary compliance throughout the inspiratory phase and therefore need to be powered by a high pressure source. The larger the pressure gradient between the gas source and the alveolar pressure the less effect the airways and pulmonary impedance will have on the flow. For these the airway pressure will vary accoding to the changes in pulmonary compliance. An example of such a constant gas flow generator is the IMV-Bird. This ventilator has relatively low driving pressures and output and would be expected to deteriorate significantly under hyperbaric conditions. The Bear 1, Bear 2 and Bennett 7200 ventilators have higher working pressures (up to 50 times the physiological alveolar pressure) and are less prone to deteroriate at pressure. In most Australian intensive care units ventilators are of the constant flow generator type.

Non-constant flow generators, such as the rotary driven piston Engström 150 ventilator, permit variable gas flow throughout the inspiratory phase of ventilation.

PRESSURE GENERATORS

A constant pressure generator ventilator maintains a constant pressure regardless of changes in pulmonary impedance. Ventilator pressure can be adjusted to provide the inspiratory pressure needed to deliver the required tidal volume.

A non-constant pressure generator will permit variable pressure during inspiration.

**Cycling from inspiration to expiration**

Cycling refers to the change from inspiratory to expiratory phases. Ventilators may be pressure, volume or time cycled. The limits of these various options may be preset. Pressure cycled ventilators are most commonly used for IPPV and short term ventilator support whereas volume cycling is more common during anaesthesia.

Pressure cycled ventilators, which terminate flow when a preset pressure is reached, may have controls which alter the cycling pressure. Tidal volume may vary with pulmonary compliance. Large leaks may cause the machine to fail to achieve the desired cycling pressure. Most pressure cycled ventilators use compressed air or oxygen at 50 psi (340 kPa or 3.4 bar) to power a venturi flow generating device. The volume of entrained gas is dependant on the ambient/venturi pressure gradient. Under raised pressure they may lack sufficient flow and pressure capabilities to ventilate the patient adequately.

Volume cycled ventilators deliver a preset volume of gas to the circuit. In its simplest form this is done by compressing a bellows. The pressure will build up to overcome any obstruction until the preset volume has been delivered. This may be a disadvantage in the hyperbaric environment due to volume expansion with ascent. A safety blow-off at 30-50 cm water is usually built in to the patient circuit to prevent pulmonary overpressure.

Time cycled ventilators have a timing mechanism which is not influenced by the condition of the patients lungs. With all ventilators inspiratory time needs to be limited to prevent cardiac output being reduced by too long a period of raised intrathoracic pressure. Inspiratory flow rate becomes a limiting factor under hyperbaric conditions. In the chamber an increase in ventilator rate is usually seen, because, as inspiratory flow becomes maximal, the timing circuit pressurises faster and cycles sooner.

**Fluidic Ventilators**

Barila, and other anaesthetists from the Walter Reed Army Institute of Research, evolved the first fluidic ventilators in 1964. Fluidic systems such as the Campbell Mark 2 use moving streams of gas to perform sensing and control functions without mechanical moving parts. When a high speed stream of gas emerges from a nozzle it entrains surrounding gas causing a local fall in pressure and drawing further gas into the stream. When a fixed wall is placed close to the jet it is less easy for further gas to enter so the pressure quickly falls on that side pulling a pivoted jet over against the wall, the "Coanda effect". This system has the advantages of no wear from moving parts and no electronics, so it is exceptionally safe in flammable environments and is not affected by moderate temperature changes or vibration. The disadvantages are that it is sensitive to dust contamination, has a relatively high consumption of driving gas and is noisy.

**Clinical considerations for ventilator use under hyperbaric conditions**

There is no evidence in the literature of normobaric IPPV that any particular wave form gives a better clinical outcome. No such work in hyperbaria has been reported.

When using a ventilator in the chamber the most important consideration is the ability of the ventilator to deliver clinically acceptable minute volumes to the patient. This will be determined by the behaviour of the ventilator under pressure and whether ventilator output is significantly affected by changes in the patient's lung and chest wall compliance. Inspiratory and expiratory cycling characteristics change under hyperbaric conditions and tidal ventilatory requirements may increase.

To understand the relationship between ventilator performance under pressure and the effect individual patient lung compliance has on ventilator gas flow requires further information on the physiological effects of IPPV.

**Effects of IPPV on patients**

During intermittent positive pressure ventilation (IPPV) under normobaric conditions there is an increase in the ratio of physiological dead space to tidal volume. In spontaneously breathing patients physiological dead space is usually less than 30%. It may reach values of 40-70% in ventilated anaesthetised patients. This may become the limiting factor in the critically ill patient at pressure. Satisfactory tidal volumes will not be achieved, with resultant hypercapnia, unless tidal volumes can be increased.

Pulmonary compliance normally varies with age, body position and a number of pathological conditions. Even under normobaric conditions, there is a fall of about 50% in compliance with the commencement of IPPV. I have been unable to find any reports about the effect of IPPV on compliance under hyperbaric conditions.

Positive pressure ventilation changes the normal physiological responses to spontaneous ventilation. Changes in the haemodynamics of venous return, cardiac output, pulmonary circulation and its distribution (ventilation/ perfusion relationship) during normobaric IPPV are well reviewed in respiratory texts[14,16-18] and are beyond the scope of this review. I have not been able to find any controlled studies on the effect of IPPV on ventilation/ perfusion relationships under pressure.

Breathing is more difficult at higher pressures because gas density (the number of molecules packed into a given volume) is increased in direct proportion to absolute pressure and this movement of denser air requires more effort (work) to move.

Increased respiratory resistance at depth leads to increased energy cost of breathing increasing oxygen requirement, carbon dioxide retention, dyspnoea, sometimes adverse cardiovascular changes.

Figures 1 and 2 illustrate the relationships between increased gas density and the development of $CO_2$ retention and increased oxygen requirement. The cardiovascular changes under hyperbaric conditions are beyond the scope of this review.

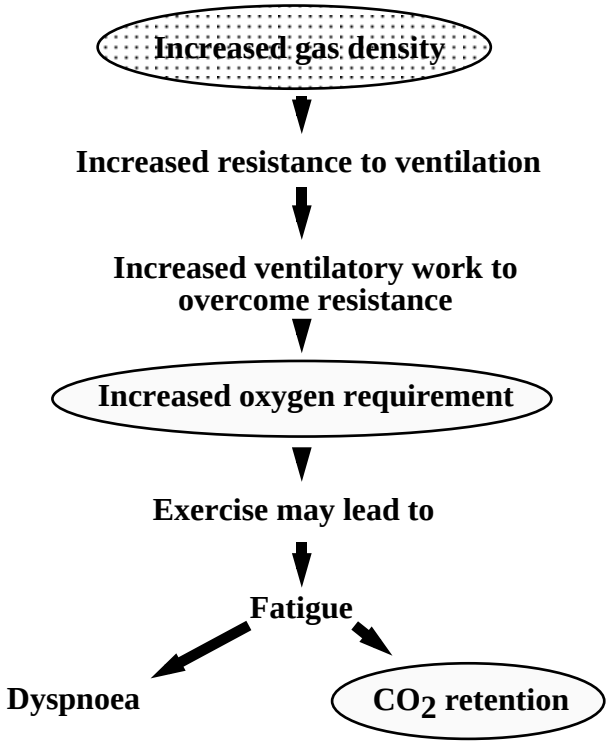


**Figure 1.** Model for normal subject at depth

**Work of breathing in the hyperbaric environment**

During spontaneous ventilation, because the inter-pleural space (between lung and chest walls) only contains a thin fluid layer, the lungs follow the outward movement of chest and diaphragm generating a sub-ambient intrathoracic pressure. Inspiration occurs as air flows into the chest at atmospheric pressure. Diaphragmatic ventilation provides approximately 60% of tidal ventilation and chest wall expansion the remaining 40%.

The study of human work performance under hyperbaric conditions has been well documented in the awake normal subject. The increase in spontaneous minute volume under hyperbaric conditions corresponds to the increased oxygen requirement and the primary reason for this is the increase in respiratory resistance due to the alteration in gas density.[19]

Critically ill patients already have increased metabolic demand, with substantially higher oxygen requirements and invariably have respiratory muscle fatigue. The work of breathing in these patients is also increased by reduced lung compliance. The relative hypoventilation that occurs in this situation can place an excessive demand on spontaneously breathing patients. IPPV in these patients will relieve the excess work of ventilation. The energy expended by the ventilator is best described as the work required to overcome the increased breathing resistance.

**PEEP under hyperbaric conditions**

Many critically ill patients also require positive end-expiratory pressure (PEEP) to maximise oxygenation. Positive pressure on the airway at the end of expiration reduces hypoxia, limits alveolar collapse and decreases the amount of intrapulmonary shunting. However PEEP increases dead space, impedes venous return, increases the risk of barotrauma and can reduce cardiac output. The therapeutic range of PEEP is 5-40 cm $H_2O$. The Monaghan ventilator PEEP function operated well under hyperbaric conditions when tested by Moon in 1984.[20]

Youn et al. reported on PEEP valves in 1991. All the tested valves increased the level of PEEP by 2-4 cm $H_2O$ as pressure increased. This was due to the increased gas density not to increased flow. They found that the valves had to be adjusted when pressure changes occurred to maintain the original level of PEEP. They recommended the Emerson water column PEEP valve which had demonstrated the least rise in pressure with increasing chamber pressure and this did not change with as pressure was raised further. This valve is cheap and easy to adjust. PEEP valves that cannot be adjusted should not be used in hyperbaric chambers. PEEP may subject the patient to an increased risk of barotrauma unless airway pressure is constantly monitored and the valve adjusted to maintain a steady level of PEEP.[21]

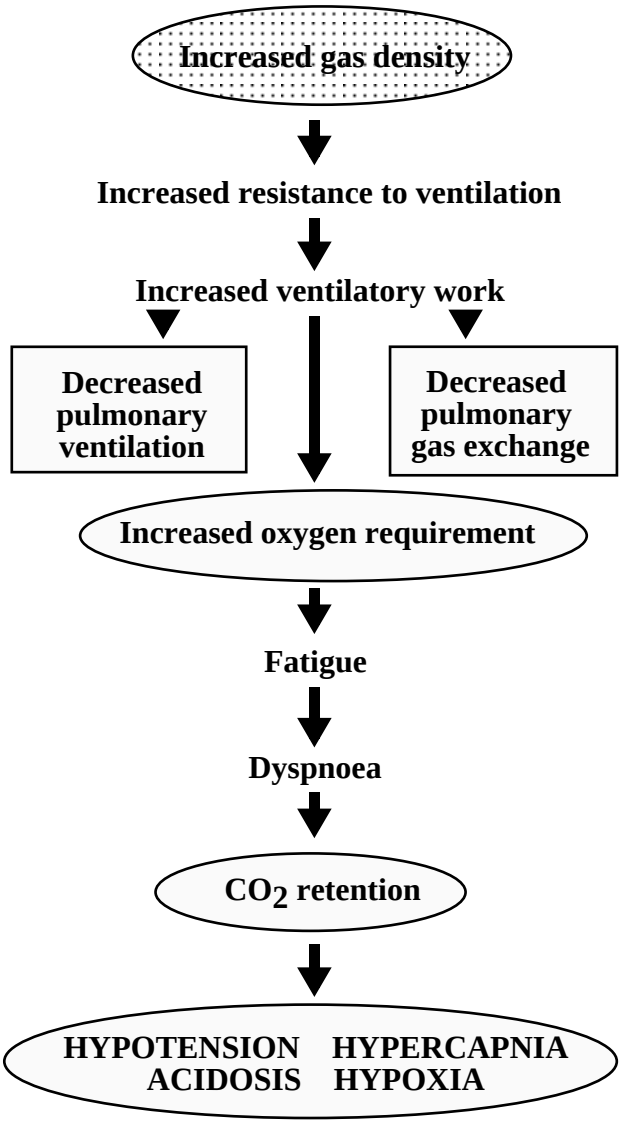


**Figure 2.** Model for respiratory compromised subject at depth

**Intermittent Mandatory Ventilation**

Intermittent mandatory ventilation (IMV) is used for spontaneously breathing patients still attached to, but not triggering, a ventilator. If the minute volume falls below a predetermined figure the ventilator cuts in to make up the preset volume. This mode must be used with caution in the hyperbaric environment as "stacking" of breaths may occur and could produce barotrauma from hyperinflation.

**Respiratory heat loss during ventilation in the hyperbaric environment**

At surface pressure (1 bar), breathing dry gases, respiratory heat loss accounts for about 10% of metabolic heat production. This is primarily through evaporative heat loss in the upper airway while saturating the dry inspired gas to a water vapour pressure of 47 mm Hg (6 kPa). At

depth the inspired gas is denser and higher in heat capacity. Helium mixtures increase this heat loss as helium has 6 times the thermal capacity of nitrogen and 5 times its thermal conductance. The increased respiratory heat loss which has been shown, in normal individuals breathing very cold air at sea level as well as under extreme hyperbaric conditions (25 bar) breathing heliox, is induced by the high thermal capacity of helium.[22]

Physiological studies of divers revealed that significant heat loss occurs via respiratory tract when breathing helium containing mixtures[23,24] this heat loss is a major threat to diver safety and performance.

I have been unable to find clinical data, or outcome studies, on the effect of respiratory heat loss during mechanical ventilation under hyperbaric conditions at "standard" HBO treatment depths (i.e. at pressures < 6 bar).

**$ETCO_2$ monitoring under hyperbaric conditions**

The reduction in tidal volume provided by ventilators under pressure makes it necessary to monitor the adequacy of minute ventilation. Volume measurements give volume delivered or exhaled but not the adequacy of ventilation. End tidal $CO_2$ ($ETCO_2$) is the most informative monitoring of the physiological adequacy of an individual's ventilation. Moon, in 1988, recommended that, because of the limitations of ventilators used under hyperbaric conditions, direct arterial $CO_2$ monitoring should be used during treatment.[25] However, with the development of end tidal $C0_2$ ($ETCO_2$) monitoring arterial puncture is no longer necessary. In 1992 Handell et al. used the Siemens infra red $CO_2$ analyser 930 to provide direct measurement of whether the preset minute volume ventilation was matched to the patient's ventilatory needs. They concluded that end tidal $CO_2$ monitoring is a valuable tool in the hyperbaric treatment.[26]

**Characteristics of the ideal hyperbaric ventilator**

The provision of ventilatory support in the hyperbaric environment has created unique difficulties and the ideal hyperbaric ventilator does not yet exist. However, attempts have been made, e.g. Dräger Hyperlog. Few recommendations have been made in the literature about standards for the "ideal" hyperbaric ventilator. Moon recommended that hyperbaric ventilators should deliver a preset tidal volume at a constant rate and over an ambient range from 1-6 bar.[25]

The ideal ventilator should provide an unchanged pattern of ventilation or, at a minimum, provide a combination of tidal volume, ventilatory frequency and I/E ratio within clinically acceptable limits, for both adults and children, to depths where treatment will occur.

Ventilators used in the hyperbaric environment should have no external or internal electrical requirements that might provide a fire hazard. They should have an ability to deliver a wide range of tidal volumes at clinically relevant rates. Standard operating procedures in multiplace hyperbaric chambers require the fractional inspired oxygen ($FIO_2$) in the chamber to remain below 0.25, therefore exhaled gases from patients and ventilators must be vented outside the chamber.

Other desirable features include:
Robust construction.
Economical to purchase and run.
Simple to operate and maintain.
Clearly marked and simple to use controls.
Gas driven.
Provision for manual ventilation in an emergency.
Ventilation range 1-20 l/min.
Tidal volumes between 50 and 1,500 ml at frequencies between 5-50/min.
An alarm system to notify failure to achieve adequate ventilation.
Positive pressure during expiration when desired.
Humidification of inspired gas.
Easy to clean and sterilise.
Suitable for paediatric use.

**Ventilator function under hyperbaric conditions**

At constant absolute temperature, the volume of a given mass of a gas varies inversely with the absolute pressure (Boyle's Law). This basic gas law is of considerable importance in understanding the use of ventilators under hyperbaric conditions. In monoplace chambers any ventilator placed outside the chamber will be limited by this law. As the pressure within the chamber increases the tidal volume delivered diminishes.

With ventilators in hyperbaric chambers gas flow rates are proportional to the gas density so as the ambient pressure increases the flow rate will fall. When the ambient pressure is increased to 2 bar (10 m gauge) density is double that at the surface.

Jaffrin and Kesic reviewed the fluid mechanics of pulmonary gas flow and found that when the pressure was doubled (to 2 bar) the actual flow was 71% of indicated flow.[27] With a constant orifice the pressure gradient required for a given flow will be greater under hyperbaric conditions, i.e. pressures will read high but flow delivery will be less than set.

The major component of mechanical resistance in the ventilator system is the resistance to flow which, in turn, is depenent upon the gas density. Increases in airway resistance affect IPPV by increasing the inspiratory time constant. Under hyperbaric conditions the power of the

ventilator, i.e. its maximum capacity to deliver a set volume, may be insufficient to overcome the resistance to flow causing inadequate ventilation. It may be impossible, in some ventilators, to achieve adequate ventilation even using a prolonged inspiratory time setting. With no change in the expiratory time, or with an inadequate inspiratory/expiratory time (I/E) ratio, air trapping may result, with an increased risk of barotrauma and impaired gas exchange.

**Ventilator gas flow under hyperbaric conditions**

The capacity of a ventilator to maintain performance under hyperbaric conditions is dependant on its power reserve. The increase in gas density (D) is proportional to the increase in environmental pressure ($P_{ata}$) by the formula:

$D = D_o \times P_{ata}$

where $D_o$ is the specific density of the gas.[6,7]

At a given volume V and at a pressure P, the compressibility of the gas ($G_c$), is defined by the modification of the volume per unit of pressure according to the equation:

$G_c = V/P$

Using Boyle's Law one can derive:

$G_c = V/P_{ata}$

i.e. if the volume remains constant then as the pressure increases the more the compressibility of a gas decreases. The increased gas density produces a decrease in flow from the orifice, the size of which is controlled by a needle valve.

Flow of a gas in a smooth tube is normally laminar until a "critical flow rate" is reached when it becomes turbulent. For a given flow rate, the resistance and pressure gradients required are greater for turbulent flow than for laminar flow. Narrowing in a tube forces the gas to accelerate which produces eddies and turbulent flow.

The Hagen-Poiseuille equation correlates the factors that determine laminar flow:

$\text{Flow } (Q) = \pi P d^4/128hl$

Where

P = pressure difference across the tube,
d = diameter of the tube,
l = length of the tube
h = viscosity of the gas. In the therapeutic range of pressures viscosity of fases rmains constant.

The theory of turbulent flow is complex. However the property of a gas which most influences turbulent flow in a hyperbaric environment is its density (r) and the pressure needed to produce a given flow will increase as the density increases.

The probability of turbulence can be predicted by an index known as the Reynolds number which is calculated according to the formula:

Reynolds number = nrd/h

where
n = velocity,
r = density,
d = diameter of the tube,
h = viscosity

As the density increases the Reynolds number also increases and turbulent flow becomes more likely and eventually predominates. Studies have shown that in 9 mm diameter endotracheal tubes flow will become turbulent when flow rates exceed 9 l/min. In 15 mm tubes (equal to the tracheal diameter) flow will become turbulent at 15 l/min and in 22 mm tubes (delivery tubing) it will become turbulent at 22 l/min.[28] Turbulent flow requires higher airway pressures to achieve adequate ventilation and this will be worse under hyperbaric conditions when ventilating with air and less so for helium.

**Studies of ventilator function under hyperbaric conditions**

Reports of assessment of ventilator function under raised pressure first appeared in 1977.[30] Table 3 summarises the studies I have found.[6,8,9,20,25,29-37]

1977 Ross and Manson assessed the behaviour of three portable ventilators/resuscitators under hyperbaric conditions.[29] These were the "Pneupac" Ventilator/Resuscitator, the "Motivus" Resuscitator (Type PV) and the "Stephenson Minuteman" Resuscitator. None provided adequate ventilation at pressure. The first two are time cycled, volume limited flow generators and neither was able to provide an adequate tidal volume at 2.0 bar. The Stephenson Minuteman ventilator is a pressure cycled flow generator. Tidal volume was adjusted using the unit pressure regulator. It delivered 100% oxygen at a constant tidal volume but at a reducing ventilatory frequency as the pressure increased. It was clinically unacceptable because the minute volume fell. The unit was incapable of producing a ventilatory rate greater than 6 a minute at 3 bar (20 m).

Gallagher et al. evaluated the IMV Bird and Mark 2 Bird up to 2.8 bar and showed that neither ventilator could maintain a tidal volume of 1,000 ml. They also evaluated modified IMV Bird and modified Mark 2 Bird ventilators using a test lung. At 4 bar (30 m) these units failed to provide a tidal volume of 1,000 ml.[8]

Moon tested 3 models of the Bird Ventilator and all failed at 3-4 bar.[27] These ventilators tended to be unstable during periods of changing ambient pressure. Moon also found the pneumatic Emerson ventilator delivered constant tidal volumes up to 6 bar (50 m). This ventilator used a leather bellows with a hydrocarbon lubricant which is well recognised as hazardous in hyperbaric conditions.

**TABLE 3**

**STUDIES OF VENTILATOR FUNCTION UNDER HYPERBARIC CONDITIONS**

**Note. Failed = unable to deliver clinically acceptable tidal volume**

| Year Author | Ventilator | Notes | Hyperbaric conditions |
|---|---|---|---|
| 1976 Campell[37] | Campbell Mark 1 | Stated to work satisfactorily | No data reported. |
| 1977 Ross and Manson[29] | Pneupac Ventilator-Resuscitator | Time cycled and volume limited flow generator | Failed at 2.0 bar |
| | Motivus Resuscitator | Time cycled and volume limited flow generator | Failed at 2.0 bar |
| | Stephenson' Minuteman | Pressure cycled flow generator 6 breaths/minute at 3 bar | Failed at 2.0 bar |
| 1978 Gallagher, Smith and Bell[8] | IMV Bird | Unstable with changing pressure | Failed at 2.8 bar |
| | Mark2 Bird | Unstable with changing pressure | Failed at 2.8 bar |
| 1979 Moon[25] | Bird Various models | | Failed at 3-4 bar |
| | Emerson Ventilator | Bellows with hydrocarbon lubricant | Constant tidal volume (TV) up to 6 bar |
| 1982 Saywood et al.[9] | Penlon Oxford | Volume cycled | Constant TV to 6 bar for air and to 31 bar for heliox. |
| 1987 Lewis et al.[31] | Penlon Nuffield 200 | Pneumatic time cycled flow generator. Modification needed | Adequate TV at 1.8 bar |
| 1989 Hipp et al.[32] | Siemens Servo 900B | Internal electrical components potential electrical hazard | Adequate to 3 bar |
| | Siemens Servo 500C | | |
| 1986 Moon et al.[20] | Monaghan 225 | PEEP function tested | Maintained preset TV to 6 bar |
| 1989 Blanch, Desautells and Gallagher[33] | 18 Various ventilators | Pneumatic time cycled, pneumatic pressure cycled and volume cycled | Necessary to have minute volume monitoring |
| 1991 Spittal, Hunter and Jones[34] | Pneupac HB | Time cycled, constant flow Set tidal volume | 16 l/min at 2.5 bar |
| 1992 Gibson, Davis and Wilkinson[35] | Pneupac HC | Pneumatic controlled time cycled Adjustable | Functioned to 6 bar |
| 1990 Dragerwerk[36] | Drager Hyperlog | Time cycled pneumatic specifically designed for hyperbaric use | Limits not stated No reports of function |
| 1992 Oriani, Marroni and Wattel[6] | Iper 60VF | Electropneumatic time cycled | Assured ventilation in assist/control mode |

The Penlon Oxford, a volume cycled ventilator, was tested by Saywood to 6 bar (50 m) in compressed air and to 31 bar (300 m, 1,000 ft) in a helium/oxygen environment.[9] The rate and delivered tidal volume was stable over the entire pressure range. At the time it was, reportedly, the only commercially available ventilator to maintain rate, tidal volume and inspiratory time under hyperbaric conditions. Youn in 1989 combined the Penlon 200 Ventilator with the Ohmeda volume monitor to provide rate, volume, apnoea and minute ventilation data.[30]

Lewis et al. assessed the Penlon Nuffield 200 Ventilator, a pneumatic time cycled flow generator, for HBO treatment, in a monoplace chamber, of carbon monoxide (CO) poisoned patients.[31] Modification was necessary to achieve adequate tidal volumes.

Hipp et al. reviewed the Siemens Servo 900B ventilator at pressures up to 3 bar.[32] They found that the delivered minute volume decreased continuously with increasing chamber pressure and that the display of minute volume showed falsely high values. Despite these limitations it has been used by these authors for hundreds of hours without malfunction. The Siemens Servo 500C has also been used; however the internal electrical components make this ventilator a fire hazard which has reduced its use.

The Monaghan 225 ventilator was used extensively at Duke University Medical Center and tested by Moon et al. in 1986 for use in a hyperbaric chamber.[22] It was found to maintain preset tidal volume to 6 bar and deliver clinically acceptable maximum minute ventilation. It delivered 38 l/min at 1 bar reducing to 18 l/min at 6 bar. PEEP functions were tested to 6 bar and operated well. However the preset respiratory rate decreased as the pressure was raised. This was the first “off the shelf” ventilator, with these features, shown to function well under pressure. It is driven by compressed oxygen.

In 1989 Blanch et al. published studies 18 different mechanical ventilators, grouped as pneumatic time cycled, pneumatic pressure cycled or volume cycled (piston or bellows) ventilators.[33] They recommended that clinicians measure the delivered tidal volume after any significant alteration in chamber pressure.

The Pneupac range of ventilators has been accepted widely for resuscitation and anaesthesia. These ventilators are time cycled, constant flow generators capable of delivering a set tidal volume irrespective of airway resistance and lung compliance. When initially tested under hyperbaric conditions the tidal volume decreased exponentially with ambient pressure and the frequency increased linearly until performance was inadequate.

The Pneupac HB was designed specifically for monoplace hyperbaric chambers and its performance was reviewed by Spittal et al. in 1991.[34] The ventilator delivered minute volumes of 11-23 l at 1 bar and 7.6-16 l at 2.5 bar.

The Pneupac HC Hyperbaric Ventilator was specifically designed to ventilate patients over a range of pressures up to 10 bar. The ventilator is a pneumatically controlled, time cycled ventilator providing independent control of inspiratory time, expiratory time and flow rate. It is also autocompensated for pressure by a pneumatic system and can be fed with air or oxygen. Gibson et al. carried out a calibration of this ventilator to derive a series of tables for its clinical use.[35] They found it to be a robust ventilator with consistent performance that could provide clinically appropriate tidal volumes for the vast majority of adult patients up to 6 bar. It is cheap and simple to operate and is capable of providing clinically significant tidal volumes to common treatment depths.

The Dräger Oxylog has been well proven as a robust and capable ventilator. It is generally limited to 2 bar but its advantage is that it does not need modification of any kind. The Dräger Hyperlog, designed for use under hyperbaric conditions, is simple to operate and no adjustments are required with changes in pressure.[36] This is important for critical care nurses who may use these machines infrequently. It works well with air, oxygen or heliox, however there is no IMV mode and it has no alarms.

The Iper VF60 is an electropneumatic, time cycled ventilator which has a variable I/E ratio. The pneumatics are supplied at 3 bar with variable $FIO_2$ capability. The electrical circuit is low voltage and low resistance. An environmental pressure sensor automatically compensates the gas feed pressure and respiratory control.[6]

Campbell stated that the Mark 1 ventilator had been used under hyperbaric conditions with success,[37] but I am unable to find such a report. It requires continual adjustment during pressure changes. It is not easy to use as there are no set marks for identification of inspiratory times, expiratory times or flow, only graduations.

The Sechrist 500A ventilator is a commonly used in chambers in the United States. It is a compact but delivers less than the preset tidal volume whenever ventilator compliance load falls and is severely limited if chamber pressure is greater than 2 bar. It requires a driving pressure of at least 450 kPa to operate normally. This exceeds the usual hospital wall outlet oxygen pressure of 350-400 kPa.

The Bird Mark 7 has also been used in Australia but modifications have been required. No literature reference for its use under hyperbaric conditions has been identified. This machine is oversensitive and difficult to operate but provides a cheap alternative to other more expensive models.

**Summary**

For volume cycled ventilators the change from inspiratory to expiratory phase occurs when a certain tidal volume has been supplied. Under hyperbaric conditions the increase in resistance reduces flow. Increased inspiratory time is therefore needed to supply a given tidal volume. Therefore a decrease in ventilatory frequency (breaths/min) occurs with a reduction in minute volume.

For pressure cycled ventilators the change from inspiration to expiration occurs after a given pressure level is reached. Due to the increase in resistance at increasing hyperbaric conditions, tidal volume is reduced as the cycling pressure is more rapidly achieved.

Time cycled ventilators represent the majority of current ventilators. In these the timing chamber of the pneumatic circuit senses the effect of reduced compressibility of the gas and will cycle more quickly, reducing inspiratory and expiratory time. As the pressure rises reduction in tidal volume and increase in ventilator rate is seen.

The selection of an optimal I/E ratio on the ventilator is important to avoid elevated end expiratory pressures. Inspiratory pressure will vary and understanding of the altered lung mechanics in ventilated patients and the altered ventilator mechanics is essential to operate a ventilator safely under pressure.

**Future research**

In the last 15 years developments in mechanical ventilatory support have allowed patient initiation of ventilator function via various modes.[15] The original modes of mechanical ventilation (control and assist/control) provide full ventilatory support[38] and are the norm under hyperbaric conditions. The introduction of IMV and proportional assist ventilation allow partial support and newer modes include pressure support and airway pressure release.

No clinical data presently exists for the use of such modes in the hyperbaric environment and this area provides a challenge for further research.[39]

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## ASSESSMENT OF THE CAMPBELL "D-MODE" VENTILATOR UNDER HYPERBARIC CONDITIONS

Marcus Skinner

**Key Words**
Equipment, hyperbaric research, ventilators.

### Introduction

The Royal Hobart Hospital commissioned a new Hyperbaric Chamber in February 1993. Since that time a review of in-chamber equipment has been undertaken. A Penlon 200 is used in this facility and a backup ventilator was needed. An assessment of currently available ventilators for use in the chamber was carried out.[1] A Campbell D Mode ventilator was made available for assessment by ULCO Engineering. No performance reports on the function of Campbell ventilators under hyperbaric conditions have been identified in the literature.

### Methods

The Campbell D-Mode Portable ventilator is an Australian made pneumatic, time cycled, volume and rate preset, constant flow generation ventilator developed by ULCO Engineering. Its driving gas supply is medical air or oxygen supplied at 60 psi (415 kPa) gauge of which it consumes 1 l/min in addition to the minute volume. The ventilator was assessed under normal and hyperbaric conditions. Its ability to deliver a preset volume or rate was assessed at three pressures.

At 1 bar it delivers tidal volumes from 50 ml to 2,000 ml with inspiratory times of 0.5-2.0 seconds and expiratory times ranging from 1.0-6.0 seconds. Timing is controlled by graduated scales. Inspiratory time for the ventilator, on each of its graduations, was measured using a digital stopwatch (average of five readings) at each pressure.

The ventilator was attached to a Siemens test lung (compliance rated at 50 ml/cm $H_2O$/l) to simulate a patient's lung. The manufacturer states that the specific compliance of normal lung is in the region of 60-70 ml/cm $H_2O$/l.

A Wright's respirometer, calibrated using a 2 litre Rudolph gas calibration syringe, was used to measure tidal volumes at 1 bar (surface), 2.4 bar (14 m) and 2.8 bar (18 m). Calibration at 1 bar and 2.8 bar showed that the Wright's Spirometer over-read the tidal volume by an average of 6% at low volumes and by 3% at high volumes. The recordings have been adjusted to reflect this error.

The "D" Mode ventilator flow control was set at 1.0, 0.75 and 0.5 and the tidal volume measured for each inspiratory time setting (0.5, 0.75, 1.0, 1.25, 1.5 and 2.0 seconds). Expiratory time was left at the 2.0 seconds setting for convenience and to allow for a greater than 1:2 inspiratory to expiratory (I/E) ratio. The setting of the expiratory time does not affect the inspiratory time setting. The tidal volume at each inspiratory time setting was measured three times and averaged. These measurements were repeated at each pressure.

From the data obtained a series of graphs indicating the tidal volume for given ventilator settings and tidal volume at maximal flow for three depths was constructed. A further graph depicting the minute volume at maximal flow was constructed for each depth.

## Results

The results of the study are shown in Figures 1-5. The narrow variation within each group of readings reflects the consistent performance of the ventilator. Figures 1-3 present the tidal volumes achieved for different flow settings over the ventilator's range of inspiratory times at 1 bar, 2.4 bar and 2.8 bar. Figure 4 presents the tidal volumes achieved at these three depths at maximal flow. Figure 5 presents the minute volumes achieved at these three depths for maximal flow.

It is clear that the performance of the Campbell D Mode ventilator decreases with pressure as at 1 bar it delivers more than twice the volumes available at 2.4 bar and approximately three times the volumes available at 2.8 bar. To achieve a minute volume of 10 l at 2.4 bar, flow must be set to maximum and inspiratory time to at least 1.25 seconds with an expiratory time of two seconds. At 2.8 bar a minute volume of 10 l cannot be achieved.

## Discussion

As would be expected for a pneumatic, time cycled, constant flow ventilator the performance of the Campbell D Mode ventilator decreased significantly as the chamber

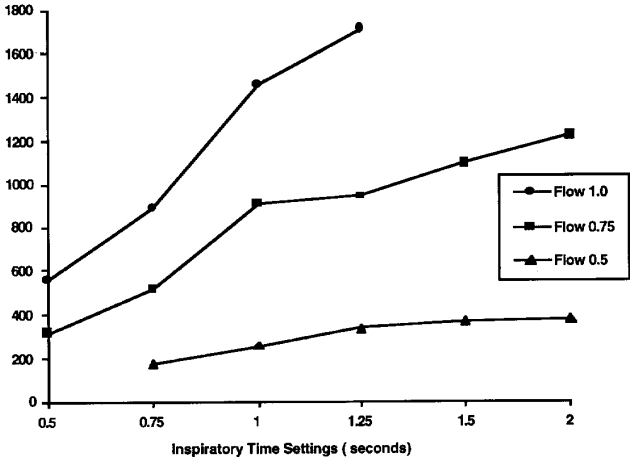


**Figure 1.** Tidal volumes at 1 bar for each inspiratory time setting (0.5-2 seconds), with the expiratory time constant at 2 seconds, for three flow settings (0.5, 0.75 and 1).

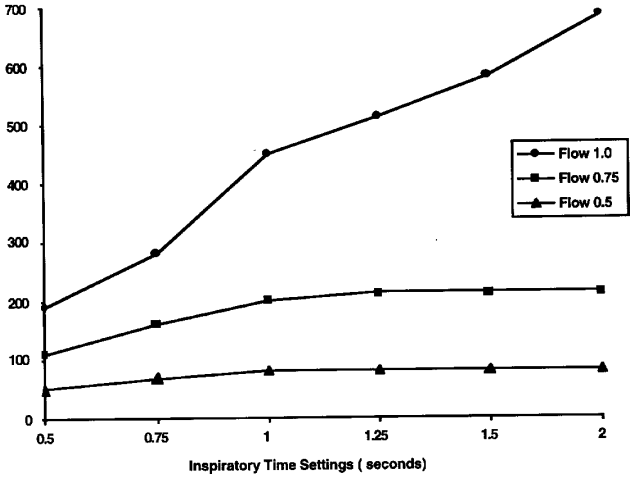


**Figure 2.** Tidal volumes at 2.4 bar for each inspiratory time setting (0.5-2 seconds), with the expiratory time constant at 2 seconds, for three flow settings (0.5, 0.75 and 1).

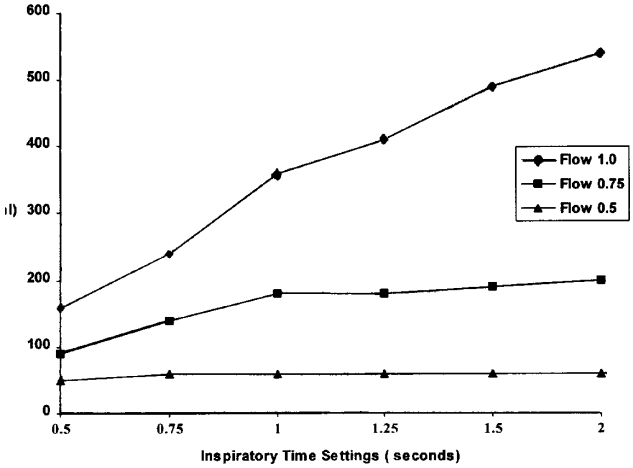


**Figure 3.** Tidal volumes at 2.8 bar for each inspiratory time setting (0.5-2 seconds), with the expiratory time constant at 2 seconds, for three flow settings (0.5, 0.75 and 1).

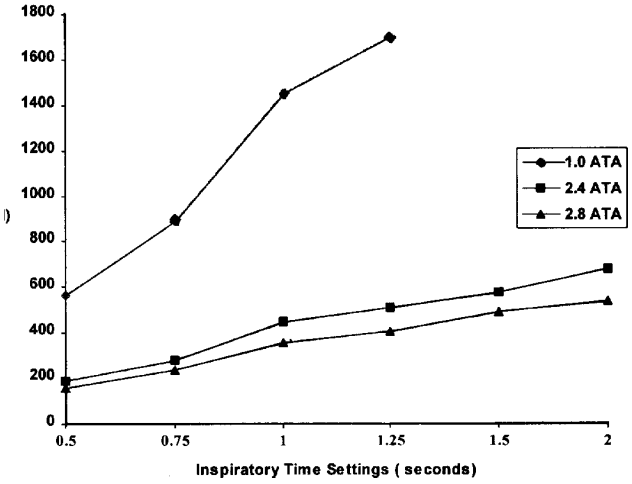


**Figure 4.** Tidal volumes for each inspiratory time setting (0.5-2 seconds), with the expiratory time constant at 2 seconds, for the maximum flow setting (1) at 1. 2.4 and 2.8 bar.

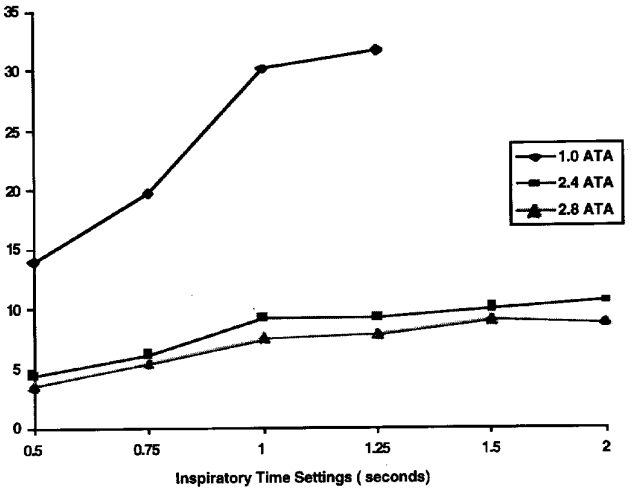


**Figure 5.** Minute volumes for each inspiratory time setting (0.5-2 seconds), with the expiratory time constant at 2 seconds, for the maximum flow setting (1) at 1. 2.4 and 2.8 bar.

pressure increased. Such changes have also been documented for the Oxford Penlon 200 ventilator.[2] The Campbell D Mode does not approach the extended range or the reported performance of the Pneupac HC hyperbaric ventilator.[3]

It is apparent that the Campbell D Mode ventilator is incapable of providing clinically acceptable tidal volumes to the average adult patient at pressures beyond 2.8 bar (18 m). At the Royal Hobart Hospital facility patients are routinely treated at 14 m (2.4 bar) and 18 m (2.8 bar). At the maximal flow setting (1.0) and with the inspiratory time setting of 1.0 second and an expiratory setting of 2 seconds (I/E Ratio 1:2) the tidal volume was 460 ml at 2.4 bar and 380 ml at 2.8 bar. With the inspiratory time increased to 1.5 seconds these values increased to 590 ml and 500 ml respectively. In order to maintain clinically acceptable minute volumes in patients the tidal volumes must be monitored and the rate adjusted accordingly. This requirement has been mentioned in other studies in which the minute ventilation was measured by a suitably calibrated spirometer.[3-6]

In clinical practice the delivered tidal volume may alter with changing lung compliance, whereas in this study the test lung compliance remained unaltered. It is accepted that patients' lung compliance may alter, particularly in the critically ill patient. In previous studies the clinical significance of changes in patients lung compliance at depth and its effects on positive pressure ventilatory tidal volumes was not investigated.

Use of the Campbell D Mode ventilator on patients in our chamber suggests that the changes in patient lung compliance at pressures of 2.8 bar are of minimal significance. This ventilator was not designed specifically for hyperbaric use. When considering the aspects required of the ideal hyperbaric ventilator[1] the Campbell D Mode is robust, simple to operate and easy to maintain. The controls are clear and simple to use with well defined graduations. It has visual and auditory disconnect alarms and may operate on air or oxygen. The ventilator driving gas (oxygen) pressure remains constant at depth and has no significant influence on the delivery of an adequate tidal volume.

The inspiratory and expiratory times of pneumatically time cycled ventilators, including the Campbell D Mode, shorten with increasing ambient pressure.

The Campbell D Mode ventilator has been found to be an acceptable alternative to the Penlon 200 that has been in use in our chamber. It has the same disadvantages as the Penlon but has the major advantage of being capable of being preset by dialling up set graduations on the machine. This has been found helpful for Intensive Care ventilator trained staff who use the ventilator only occasionally. Spirometry is used in the clinical setting and adaptation for end tidal $CO_2$ monitoring is being undertaken.

## Conclusions

The desirable features of the ideal hyperbaric ventilator have been proposed elsewhere.[1] The Campbell 'D' Mode ventilator meets some of these requirements. It is robust and simple to operate and maintain. The driving gas can be air or oxygen. The ventilator's controls allow for known values to be set. A warning system for disconnection or reduced inspiratory pressure is part of the ventilator.

The Campbell D Mode ventilator provides an alternative to the Penlon 200 for the average adult patient. However it does not achieve the desired maximal inspiratory flow rate of 80 l/min at depth (2.8 bar) where its maximum flow is only 35 l/min. At 2.8 bar tidal volumes above 600 ml cannot be achieved. It would be unable to provide clinically acceptable tidal volumes in some clinical circumstances (e.g. the morbidly obese) and further studies are needed to identify its clinical limits.

The PEEP function has not been evaluated. Controlled ventilation in the hyperbaric chamber presents a variety of challenges and risks that require further evaluation.

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# THE WORLD AS IT IS

## 1997 ANNUAL MEETING OF THE AUSTRALIAN HYPERBARIC TECHNICIANS AND NURSES ASSOCIATION

Eric P Kindwall

**Key Words**
Hyperbaric facilities, meeting.

The Coogee Beach Hotel was the venue for the 5th Annual Scientific Meeting of the Australian Hyperbaric Technicians and Nurses Association (HTNA) 28-30 August 1997. Coogee Beach is a pleasant seaside suburb of Sydney, which in August was welcoming the beginning of spring "down under". There were well over 100 participants from the nine hyperbaric facilities in Australia, all hospital-based units with multiplace chambers. More than 30 papers were submitted to the meeting; slightly over half of them dealt with clinical hyperbaric medicine and the remainder with diving-related subjects. Laura Josefson, RN, President of the BNA, and I were guests of the HTNA and were given ample time to speak on the program.

The clinical hyperbaric subjects were broad and varied. They included impaired neutrophil adhesion in patients with diabetes, the problem of claustrophobia in the chamber, injury mechanisms in carbon monoxide poisoning, psychiatric profiles of patients with carbon monoxide poisoning, a survey of middle ear barotrauma in unconscious patients, an update on the results of hyperbaric incident monitoring (the HIMS Study) and the use of tympanostomy tubes.

The divers dealt with the treatment of decompression sickness, DCS at very shallow depths, technical diving subjects and the practicality and utility of square hyperbaric chambers.

We were met at the airport by Dr Ian Unsworth, literally the founder of HBO therapy in Australia, who turned us over to the capable hands of John Kershler and Barrie Gibbons of the Prince of Wales Hospital HBO unit, who had made all our travel arrangements.

The highlight of the trip for me and my family was a grand tour of the hyperbaric facilities in Australia, starting in Sydney with the Prince of Wales Hospital and HMAS PENGUIN, the Royal Australian Navy Diving Training Facility. Our travels then took us to the Royal Hobart Hospital in Hobart, Tasmania; the Alfred Hospital in

Melbourne; the Royal Adelaide Hospital in Adelaide; the Fremantle Hospital in Western Australia; to the Townsville General Hospital, which handles diving casualties from the Great Barrier Reef; and finally back to Sydney for the meeting.

One of the most impressive looking chambers was the multiplace facility at the Royal Hobart Hospital founded by Dr Peter McCartney, who remains as consultant. Co-directors Dr David Smart and Dr Margaret Walker are currently running the chamber. Their unit featured 60 cm (23.6 inch) circular view ports in a chamber capable of 6 ATA. These were literally like dramatic picture windows. The Alfred Hospital in Melbourne at the present time has a circular-doored multiplace chamber but has just received approval for a 3-lock rectangular chamber capable of 6 ATA, which will be constructed beginning in 1998, according to Dr Ian Miller, the director.

In Melbourne, Dr Miller treated us to an Australian rules football game, best described as a form of "guerilla warfare" with goal posts. The Adelaide Hospital already had a rectangular chamber with unbelievable space for positioning patients and using critical care equipment. Dr John Williamson and his charming wife were our genial hosts.

During our visit to the Fremantle chamber run by Dr Harry Oxer, we were privileged to visit the ultra-modern Royal Australian Navy Submarine Escape Training Tower located there. The Fremantle chamber facility and the Royal Australian Navy work closely together with regard not only to training but also to management of diving casualties. While there, we had a delightful dinner overlooking Fremantle Harbour with Harry and Sharon Keetley of the Fremantle Hospital HBO Unit. Sharon is president of the HTNA.

The Townsville chamber is very active in the treatment of diving casualties, as well as having a vigorous clinical hyperbaric program. Dr David Griffith is the energetic director of the unit there. If a new hospital facility is built, which is now being actively discussed, they are hoping to get an even more user-friendly clinical chamber. While in Townsville, we had the opportunity to spend a day at the Great Barrier Reef, where we snorkelled amid giant clams and colours that I had never seen in the Caribbean.

The most impressive thing my family and I experienced in Australia was the wonderful hospitality and generosity of everyone when it came to their time and willingness to arrange our travel. We made many new friends and hope that we may reciprocate the warm welcome we received.

We found Australian medicine to be absolutely first-rate. In many ways, the Australians seem to be less bound by tradition and quicker to adopt new methods that work. Their no-nonsense approach to problem-solving is impressive. It appears that their protocols for the treatment of decompression sickness, for example, are somewhat more advanced than those used commonly in the United States, with all the chamber having helium-oxygen available on the manifold.

The Australians face many of the same problems we face here concerning money for chambers and equipment and insurance reimbursement, in many ways it sounded like old home week.

Uniform regulation associated with hyperbaric treatment is in the final planning stage, and a committee is about to establish nationwide minimal standards for training of physicians and chamber personnel.

In summary, it was an enlightening educational experience and a most enjoyable 18-day tour under the Southern Cross.

*Dr Eric Kindwall is the Director of Hyperbaric Medicine at the Medical College of Wisconsin, 9200 W Wisconsin Avenue, Milwaukee, Wisconsin 53226, USA.*

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## ROYAL NEW ZEALAND NAVY DIVING MEDICINE COURSE

A 5 day course in Diving and Hyperbaric Medicine is offered by the Naval Health Services on an annual basis.

The program is aimed at medical practitioners and other health professionals with a special interest in diving medical as well as dive instructors and dive boat operators.

| | |
|---|---|
| Dates | August 3rd to 7th 1998 |
| Venue | RNZN Hospital, 91 Calliope Road, Devonport, Auckland |
| Fees | $NZ 750 (includes GST). |

Cheques to be made payable to NZ Defence-Navy

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

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

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

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

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

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

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

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

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

## 1999 SPUMS ANNUAL SCIENTIFIC MEETING

*An International Workshop on the Treatment of Decompression Illness*

will be held on the island of **Layang Layang**, Malaysia
Friday April 30th to Sunday May 9th 1999

The Guest Speakers will be Dr Richard Moon (USA), who was a guest speaker at the 1997 ASM at Waitangi in New Zealand and Dr Alf Brubakk (Norway), who attended the 1998 ASM in Palau.

The Convener of the Annual Scientific Meeting is Dr Chris Acott.

To present papers contact: Dr Chris Acott
Hyperbaric Medicine Unit, Royal Adelaide Hospital,
North Terrace, Adelaide, South Australia 5000
Telephone +61-8-8222-5116. Fax +61-8-8232-4207.
E-mail guyw@surf.net.au

Speakers at the ASM must provide the printed text and the paper on disc to the Convener before speaking.

The Official Travel Agent for the meeting is:
Allways Dive Expeditions
168 High Street, Ashburton, Victoria, Australia 3147
Telephone + 61-(0)3-9885-8863.
Toll Free with Australia 1-800-338-239
Fax +61+(0)3-9885-1164.

## DIVING MEDICINE COURSE

The School of Public Health and Tropical Medicine, James Cook University, Townsville, with the staff of the Hyperbaric Medicine Unit, Townsville General Hospital, will be conducting a course in

**Diving Medicine**
from Monday 6th to Saturday 10th October 1998.

For further details contact
Dr Peter Leggat
Senior Lecturer,
School of Public Health and Tropical Medicine
James Cook University,
Townsville, Queensland 4811
Australia
Telephone +61-( 0)7-4722-5700

## CONSTITUTIONAL AMENDMENTS

At the Annual General Meeting held in Palau on 15/5/98 the following amendment to the constitution was passed.

That Rule 22 (b), which reads "Each officer of the Association shall hold office until the annual general meeting next after the date of that person's election but is eligible for re-election.', shall be changed by replacing the word *next* by the words *three years*.

The new wording would be: *Each officer of the Association shall hold office until the annual general meeting three years after the date of that person's election but is eligible for re-election*.

The amendments will not come into effect until approved by the general body of members. Any member who objects to the amendment should notify the Secretary of SPUMS, Dr Cathy Meehan, C/o Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004, Australia, in writing before September 1st 1998. If any member objects a postal ballot will be held. If no objection is received it will be assumed that the membership has voted in favour of the amendments.

Cathy Meehan
Secretary of SPUMS.

**Key Words**
Consitutional amendment, notice.

## MINUTES OF THE SPUMS EXECUTIVE COMMITTEE MEETING

held at the Royal Adelaide Hospital Hyperbaric Unit on November 1st 1997

**Opened**
1045 Central Summer Time

**Present**
Drs G Williams (President), D Gorman (Immediate Past President), C Meehan (Secretary), T Wong (Treasurer), J Knight (Editor), D Davies (Education Officer), C Acott and R Walker (Committee members).

**Apologies**
Drs M Kluger (NZ Representative) and V Haller.

**1 Minutes of previous meetings**

1.1 Minutes of previous meeting (14 September 1997) read and accepted as a true record after minor adjustments. Proposed John Knight, seconded Chris Acott.

1.2 Minutes of the Annual General Meeting (April 1997) read and are to be published in the Journal for the information of members.

**2 Matters arising from the minutes**

2.1 North American Chapter. Jeffrey Bertsch will be the SPUMS North American chapter representative. He will be asked to contact the previous representatives Dr Lori Barr and Mr Steve Dent and update us on the present financial situation.

2.2 Indemnity Policy update. Dr Guy Williams is awaiting further advice on this issue.

2.3 The job descriptions of the Secretary and Education Officer have been completed. The Treasurer and Editor are updating their list of duties. The role of the convener document is still pending.

2.4 Oxygen equipment update. Dr Acott will liaise with Dr Mike Davis and co-ordinate transportation of the oxygen equipment to the next ASM in Palau.

2.5 Inventory of SPUMS equipment held by members has been passed to the Treasurer. The Secretary will also hold a copy of this. The audio equipment held by the President needs to be upgraded. Dr Williams has this matter in hand.

2.6 SPUMS on the Internet. It is advantageous for SPUMS to have a recognisable domain name, to facilitate easy asses to the proposed SPUMS web site. The objective will be to have current information available on the Internet as well as the ability to complete and process application forms electronically. The Secretary has had a home site for some time. This has been updated and will be posted onto the Internet as soon as the domain address is formalised.

2.7 Update of SPUMS forms. The Editor will update the introductory form. The Education Officer will update the DDL approved medical course form. The Secretary and Treasurer have updated the application form. The new DDL and SPUMS membership application forms will be reprinted in time for the renewal notices to go out in early December.

2.8 Further discussion was held as to the advantages of longer terms of committee positions. It was decided that it would be beneficial to have three year terms for all the elected committee positions. A motion was presented that the constitution be changed as follows:
"**Officers of Committee** 22.(b) Each officer of the Association shall hold office until the annual general meeting three years after the date of that person's election but is eligible for re-election."

2.9 Project Proteus. There has been some response to the letter of expression of interest.

2.10 Letter Dr M Davis re Diploma. Dr David Davies will write to Dr M Davis with regard to this.

2.11 The Treasurer and Secretary have upgraded to

Microsoft Office 97. The computer used by Steve Goble to hold the DDL database runs on Windows for Workgroups 3.1. It is therefore not possible to upgrade this version of Access to Office 97. In view of this it will not be possible for that computer to hold a current list of SPUMS financial members (which is in Access 97). All correspondence addressed to the DDL will in future be directed initially to the Treasurer who will check the financial status of the applicant. The Treasurer will then forward the details to Steve who will update the DDL database and produce the information for the bi-annual DDL booklet.

2.12 It continues to be beneficial to hold one face to face committee meeting yearly at a time other than the ASM. The possibility of holding this meeting at the time of the annual HTNA meeting, which is usually held at the end of August, was discussed. It was decided that there would not be sufficient time available to hold a satisfactory SPUMS Committee meeting during the HTNA meeting. It was proposed that the yearly meeting be held at the end of October in Adelaide, Melbourne or Sydney, according to convenience at the time.

**3 Annual Scientific Meetings**

3.1 1998 Palau update. There were no outstanding points to discuss.

3.2 1997 New Zealand ASM closure report. This has been finalised and outstanding monies have gone to the ASM account.

3.3 1999 Layang Layang update. Richard Moon has agreed to speak. It is proposed to hold the conference in the first week of May. The proposed theme is "Bubbling".

3.4 2000. A family destination such as Fiji was suggested.

3.5 Kavieng, PNG, was proposed as a possible venue for 2001.

**4 Treasurer's Report**

The Treasurers report was accepted. Proposed Robyn Walker, seconded Des Gorman.

**5 Correspondence**

5.1 Dr Parker, re SPUMS WEB site. Dr Meehan will reply.

5.2 Letter from Tony Turner re Wesley Unit. Dr David Davies will correspond and send the requested information.

5.3 Phone call from Dave King re HTNA meeting. It was agreed that a flier be enclosed in the next Journal at no charge.

**6 Other Business**

6.1 The SPUMS Policy on the Initial Management of Diving Injuries and Illnesses was adopted.

6.2 Relationship between the HTNA, ANZHMG and SPUMS (Drs Williams, Walker). The ANZHMG is a standing committee of SPUMS. This was discussed.

6.3 Secretariat (Dr Knight). Discussion was again reopened on having a secretariat. In the past this has turned into an expensive option. It was thought that it may be possible to find an individual in Melbourne to work part time. Duties would include collecting and sorting the mail, dealing with the general inquires as well as coordinating those that need to go through to Committee members. Such as person is being sought.

6.4 SPUMS Diploma and possible affiliation with a tertiary educational institution. (Dr Walker). During the discussion it was pointed out that this had been tried in the 1970s and no New South Wales University was interested in adopting the SPUMS diploma. Dr Walker to investigate if there has been a change attitude.

**Closed** 1600

# BOOK REVIEWS

**UNITED STATES NAVY DIVERS HANDBOOK**
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, USA.
Price from the publishers $US 27.50. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370.

**Key Words**
Diving operations, tables, treatment, underwater medicine.

The USN Divers Handbook has been modified to reflect the error corrections and changes evident in the latest reprint of its big brother, the USN Diving Manual.

It has been published and issued as a pocket sized ready reference for diving supervisors with quick access to all USN air diving tables, therapeutic recompression tables and procedures, diving medicine, neurological examinations and first aid. It reaches its objectives admirably if the prime purpose of its existence is considered. That is, to provide USN Master Divers, Diving Officers and Diving Medical Officers with readily available information that can be carried in their pockets.

If one further considers the intense pressure and very high expectations placed upon USN Master Divers during their certification evaluation and in upholding the very high standards of the USN diving community during any form of diving duty the existence of this publication is more easily understood.

Having said that, there is some very good information included that is of direct benefit to diving supervisors, chamber operators and medical personnel within the non-military occupational diving sectors. The use of the USN air decompression tables for repetitive dives is forbidden in AS 2299 so the practicality of their use can be basically discounted except where single dives with a little longer duration are required.

The medicine section is ideal for diving supervisors and so is the first aid. Both are probably too simplified for the Diving Physician. The therapy section includes many options of changing to various deeper tables if a patient's condition deteriorates. This information is probably relevant for Diving Medical Physicians but I think it is lost to the average occupational diving supervisor. The continued use of Type l and Type ll decompression sickness terminologies, however, distances the handbook from local directions.

The complexities built into the USN support documentation and their continued policy of allowing on site diving supervisors to make complex decisions with access to 10 therapy tables further distances this handbook from our local occupational diving policy. The handbook appears to fit somewhere between the knowledge required by the Diving Medicine Physician and that required by the Diving Supervisor. It does not quite achieve either objective because it is too basic on the one hand and too complex on the other.

There is some very good information regarding pneumofathometer correction factors, general chamber use and limited emergency procedures, all of which are valuable diving supervisory tools.

The effort to include metric values within the air decompression tables is partially lost with the lack of metric conversion in the therapeutic tables and elsewhere in the text.

Perhaps one day in the future when the Australian diving community finally comes to terms with the implementation of a suitable diving supervisors qualification, which reflects the real responsibilities expected of them, the information contained in this publication will be of more practical value.

When diving supervisors, on site, can conduct a thorough neurological examination, in accordance with the information provided in this handbook, and report accurate findings to qualified medical support the Australian diving community will have made a quantum leap forward.

Larry Digney

*Larry Digney has been a Royal Australian Navy Clearance Diver and a trainer of occupational divers.*

**TACTICAL MANAGEMENT OF DIVING CASUALTIES IN SPECIAL OPERATIONS**
The 46th workshop of the Undersea and Hyperbaric Medical Society
Editors. Frank K Butler and David J Smith.
Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Maryland 2089-2627, USA
Price from UHMS $US 20.00. Postage and packing extra.

**Key Words**
Diving operations, mixed gas, occupational diving, trauma, treatment.

The proceedings of this conference, held in Anchorage, Alaska, on 30/4/96 was published in 1997. As

a window to a secret world it is well worth reading. Very few doctors who dive face the problems of covert approach to a coast, getting ashore in the face of the enemy, completing an attack mission or removing an agent, returning to the beach and rendezvousing with a vessel, usually a submarine out at sea.

The conference discussed what should be the management of those injured during such proceedings. The Editors' Summary of Key Points and Research Issues is daunting reading. Item 1 is penetrating chest trauma. Item 2 is that damage to the underwater breathing apparatus may not be survivable without a back up breathing source. Item 3 points out that the injured diver's survival depends on his buddy who is unlikely to have specialised knowledge. It is considered that a medic with a "medical bag suitable for both diving accidents and combat trauma" would be an asset in the pick up boat for these operations. Sensible comments take the reader up to item 40 and on to the Introduction.

Here Captain Butler (USN Medical Corps) points out, among other things, that Special Operations will provide the team with medical and tactical problems in the case of injury and that doing the right thing medically can lead to the enemy killing both the injured diver and his tender or in the failure of the operation. Another problem is how to train the battlefield workers. The USN uses the Advanced Trauma Life Support (ATLS) system introduced in 1978, and updated continually ever since as the basis of their care. However ATLS is designed for doctors, not medics, and assumes that hospital diagnostic and therapeutic equipment is available. So the US Armed Forces developed their own generic combat trauma management plan, and then modified it to fit Special Operations combat trauma scenarios. This plan is divided into three periods, Care Under Fire, Tactical Field Care (while in the field but not under fire) and Casualty Evacuation (CASEVAC) which starts when they have been picked up by a "helicopter, naval craft or other evacuation asset". This workshop was held to discuss some scenarios of Special Operations and discover what changes would be needed to the generic management strategy to best deal with these scenarios.

There are 6 Combat Swimmer scenarios, and three each for Dry Deck Shelter, SEAL Delivery Vehicle and Submarine Lockout systems. Each scenario is presented by an medical expert in that field. The format is to present the military plan first. Then the management plan, broken into Care Under Fire, Tactical Field Care, CASEVAC Care, Equipment Considerations, Additional Considerations and References. Most scenarios are followed by a short discussion. Unfortunately a number of the discussions were not printed as gremlins got into the recording system.

Although it is not always mentioned, it is clear that the surface swimmer scenarios are for after dark operations. Any operation launched from a coastal patrol craft 12 miles out with a one hour transit in two Zodiacs to put 7 swim pairs, using closed circuit rebreathers, in the water is only possible under cover of darkness. Swimming the last mile, half on the surface and half underwater may well not be the surprise it should be as outboard motor noise carries nicely across calm water. With a high oxygen partial pressure in the breathing mix oxygen toxicity becomes a real risk as maintaining depth is more difficult in the dark.

A Dry Deck Shelter (DDS) is a garage for a SEAL Delivery Vehicle (SDV) which can be fitted to modified ballistic missile and fast attack submarines. They have three compartments; an outer floodable hanger, a transfer trunk, which also communicates with the submarine interior, and a treatment chamber. It sounds like a quick method of releasing a SDV but the workshop was told that "with a good crew the launch process takes about 45 minutes". The DDS scenarios cover hypothermia, with or without loss of consciousness, a crushed leg, and a bullet wound to the chest. These are much easier to handle with the availability of a recompression chamber and crew members other than the diver's buddy.

SDV scenarios include a chest wound and pursuit by the enemy, sudden unconsciousness of the SDV navigator, who will be needed to get home, and loss of SDV buoyancy an hour before it is due to rendezvous with the submarine. In this case the two divers escape from the vessel but one has inflated his buoyancy device and is unconscious at the surface.

The Submarine Lockout scenarios are fascinating with unconsciousness from unknown causes to the recovery and treatment of a diver, who has lost consciousness after a rapid ascent, in the escape trunking.

It becomes quite clear as one reads the scenarios that the logical thing to do with many of the bullet injured divers is to abandon them to prevent the others of the team being discovered and attacked. This is, of course, the opposite of the philosophy of the men, which is never abandon a buddy.

Sorting out what should be done is reasonably straightforward in the comfort of the lecture hall but what to do with an unconscious buddy, who has just been shot as he surfaced to check their position, is a different matter. Towing an unconscious buddy underwater, with both divers in bulky closed circuit equipment, is physically demanding and quite possibly impossible to achieve over half a mile or more. Added to this is the almost certainty that depth control will not be possible for the pair so oxygen convulsions are likely and may claim both lives. Some participants suggested that the injured should be put ashore rather than risk the difficulties of finding the transport a mile or more off shore.

John Knight

# SPUMS ANNUAL SCIENTIFIC MEETING 1997

## EAR DRUM RUPTURE IN SCUBA DIVERS.

Noel Roydhouse.

**Key words**

Barotrauma, ears, recreational diving.

**Abstract**

Barotrauma of the ear is an inevitable part of diving with self-contained breathing apparatus (scuba). The middle ear is an enclosed air space and accordingly is at risk to barotrauma. One of the more severe forms of damage is when the ear drum ruptures. This paper discusses the diagnosis and prognosis of 80 ear drums ruptured as a result of scuba diving. The commonest diagnostic symptom complex is the development of pain in the ear which is suddenly relieved with a sensation or sound of a pop or bang on descent. In 46% of cases a disturbance of the balance apparatus develops at the time of rupture, with a true rotary vertigo only in 25% of cases. There was a spontaneous cure in 85% of cases with only 12 cases requiring operative repair. Predisposing factors were difficulty in clearing the ear in 59% of cases and some atrophy of the ear drum in 55% of cases.

### Introduction

Barotrauma of the ear is an inevitable part of diving with self-contained breathing apparatus (scuba). The middle ear is an enclosed air space with a flexible outer wall, the eardrum. If the pressure builds up, either on the outside of the eardrum, as in descent, or on the inside as on ascent, the eardrum will be stretched and, if the pressure is great enough, the eardrum will split or burst open. Stretching of the eardrum on descent is accompanied by a feeling of pressure. This is a signal for the diver to blow air up the Eustachian tube to equalize the pressure inside the middle ear with the outside or ambient pressure. The greater the pressure differential the greater the symptoms. As the pressure on the eardum increases pain is felt and there may be rupture of blood vessels into the cavity of the middle ear (haemotympanum) or into the substance of the eardrum (bruising). Finally the eardrum can split or burst. This is a different type of perforation from that caused by infection or purulent otitis media. In the latter case, as the pressure of the pus builds up, it pushes the eardrum outwards. The infection plus the pressure results in an area of avascularity, then necrosis with a rupture forming a circular hole. With barotrauma in a normal eardrum the tissue tears in a linear fashion and not infrequently along and just behind the handle of the malleus.

Smith[1] in his article on barotrauma of the ear says that all cases suffer pain before the eardrum bursts and that the in rush of water may cause caloric-induced vertigo. Roydhouse[2] on the other hand in his letter denied the statement that "all cases suffer from vertigo as a result of the inrush of the cold water." Morrison[3] is another one who says that "if cold water enters the middle ear cavity the diver becomes vertiginous, loses his sense of direction and may vomit into his mouthpiece." Since 1970 a record of all scuba divers (1,200) attending St Michael's Clinic has been kept. There were 80 cases of ruptured eardrum. These provide much clinical information which appears to be at variance with the two authors quoted who, in their articles, do not describe the basis of their information.

### Results

It is necessary to define terminology so that, in the future, valid comparisons can be made. Difficulty in equalizing middle ear pressure with ambient pressure has already been defined and it was present in 59% of the 80 cases. In the previously published[4] group of 656 divers, all with problems of the ear nose and sinuses, only 40% had difficulty in equalizing.

"Vertigo" is used otologically to mean a rotary or spinning sensation. It can be caused peripherally in the vestibular apparatus or centrally. In scuba divers it is always considered to be a peripheral disorder, unless there are signs of decompression illness. However it also covers other conditions which are kept in a separate category because of their lack of specificity. This includes terms like dizzy, giddy, loss of balance, loss of direction or a feeling of disorientation. These terms are not often associated with the vestibular apparatus but, in the context of scuba divers and ruptured eardrums, they have been included as minor vestibular symptoms. When the diver denies any sensation of spinning or rotation but describes a definite feeling to which they append these other names, it is taken that these are vestibular symptoms.

For example, Case 1138 said "You know what it is like when you are underwater. You are in an unusual situation and environment. I was holding onto the anchor chain and you know how you swing around anyway. Yes, I felt disoriented, as if I didn't know which way was up. I was slightly dizzy. Now you ask me if there was any movement. There was a little. What do you mean by spinning? Well yes I would say that there was a very slight feeling of going in circles."

An "atrophic eardrum" is an otological term indicating that the eardrum is pathologically thinner than normal, usually due to a decrease or complete loss of the

central fibrous layer of the eardrum. With a pneumatic speculum the eardrum can be seen to move much more freely, it can be seen to be thin, and it is often medially positioned. As such it maybe adherent to the long process of the incus or there may be attic retraction pockets. The cause of the atrophy is either previous multiple middle ear infections or long term otitis media with effusion ("glue ear") in childhood. Atrophic eardrums were present in 55% of the cases. In the ascent cases 41% and in the descent cases 61% were atrophic.

Table 1 gives the distribution of some details of the 80 cases in which 35 perforations (44%) were seen and 45 cases (56%) deduced from the history and symptoms. Tables 2 (major symptoms) and 3 (minor signs and symptoms) divide these into two symptom groups showing the same symptoms to confirm accuracy of the diagnosis of the deduced cases. Of interest is that 6 cases, whose perforations were seen, did not recall any signs or symptoms during the dive.

Table 4 gives both the age distribution and the length of the experience of the diver. Thirty four of the cases (42%) were in the age group 20-29 years. The youngest was 14 years and the oldest 53 years. The latter had been diving for 7 years, was ascending fast from 13 m and noted blood from his ear on surfacing. Examination 14 days later showed a raw linear area just posterior to the handle of the malleus. The modal figure for diving experience was 2-10 years (37%). However 15 cases (23%) had ruptured their eardrums during their basic diving course.

Seventeen (22%) of the ear drums ruptured on ascent. They all healed spontaneously even though 6 cases had atrophic changes. The ascent ruptures occurred at depths of 2-30 m after ascents of 2-25 m. The atrophic drums seemed to rupture with lesser pressure changes. The case of the ascent from 2 m was seen to have a perforation and the drum was described as "macerated." He was not seen for follow up so atrophy was not determined. Ascent cases had vertigo or lesser vestibular symptoms in 35% (6 cases) compared with 53% (30 cases) of those who ruptured during descent.

Of the 57 ear drums ruptured on descent 45 (79%) healed spontaneously leaving 12 (21%) requiring operation. All these had atrophic eardrums as did 24 (53%) of those that healed spontaneously.

In this series 7 cases had a single re-rupture, all of which healed spontaneously. Three cases ruptured the same ear 3 times (Table 5). One of whom healed spontaneously each time. He was case 730 and appears in Table 3 as a case with no symptoms but the perforation was deduced. Case 712 healed spontaneously the first time and then needed 2 operations, firstly a minor fat patch and then a full myringoplasty. Case 1090 healed spontaneously twice and then had a myringoplasty.

**TABLE 1**

**DISTRIBUTION OF 80 PERFORATIONS**

| | | | |
|---|---|---|---|
| Male | 64 | Female | 16 |
| Left | 46 | Right | 34 |
| On descent | 57 | On ascent | 17 |
| | | Unknown | 6 |

**TABLE 2**

**MAIN SYMPTOMS OF THE RUPTURE IN EACH GROUP**

| | Perforations | | Total |
|---|---|---|---|
| | Seen | Deduced | |
| **All perforations** | 35 | 45 | 80 |
| **Symptoms** | | | |
| A. Pain suddenly relieved | 9 | 13 | 22 |
| B. A + vertigo | 7 | 13 | 20 |
| C. A + dizziness | 5 | 6 | 11 |
| D. Vertigo alone | 2 | 4 | 6 |
| **Totals with symptoms** | **23** | **36** | **59** |
| | **29%** | **45%** | **74%** |

**TABLE 3**

**MINOR SYMPTOMS AND SIGNS OF RUPTURE**

| Symptoms and Signs | Perforations | | Total |
|---|---|---|---|
| | Seen | Deduced | |
| Pain on ascent | 3 | 1 | 4 |
| Pain on descent | 6 | 2 | 8 |
| Deafness | 13 | 7 | 20 |
| Blood from ear | 6 | 8 | 14 |
| Bubbles from ear | 2 | 7 | 9 |
| Hiss from ear | 2 | 5 | 7 |
| Otorrhoea after dive | 6 | 3 | 9 |
| **No symptoms (Only signs)** | **6** | **1** | **7** |

**TABLE 4**

**AGE AND DURATION OF DIVING EXPERIENCE IN YEARS**

| Age group | Number | Years diving | Number |
|---|---|---|---|
| 14-20 | 6 | Course only | 15 |
| 20-29 | 32 | 0-1 | 15 |
| 30-39 | 26 | 2-10 | 25 |
| 40-49 | 14 | 10-20 | 9 |
| 53 years | 1 | over 20 | 1 |
| **Total** | **79** | | **65** |

Inner ear peripheral vestibular stimulation occurred in 37 (46% of total) cases, being the single major symptom in 6 (10%) of the 57 cases of the ruptures on descending. Four of these had a vertigo and the other 2 had minor vestibular symptoms. Of those descending 21 (37%) suffered vertigo and 10 had minor vestibular symptoms. One case (730) was bottom diving at 13 m and had difficulty clearing his left ear. At 13 m it went bang and he heard a hiss and felt dizzy so he sat on the bottom. When the dizziness went he collected his scallops, ascended and boated them. He then went back underwater. He felt water enter his ear and he vomited. So he finished diving and was seen 2 days later. There was a red line just posterior to the handle of the malleus. This case was the only one of the 80 that vomited. There was another case (936) who had nausea with his vertigo.

**Discussion.**

Ruptured eardrums occur in scuba divers but most (65 cases or 85%) heal spontaneously. Those that do not, all have atrophic changes in the eardrum. One reason for the spontaneous healing is that the perforation seems to be linear in nature. Thus when the pressures equalize on either side of the eardrum, the edges of the perforation come together and healing occurs.

In this series of 80 perforations only 1 case vomited and that was on re-entry to the water after having surfaced from the causative dive. It was said[2] that one of the reasons for this was that New Zealand waters were warmer than English waters. This may be true for surface temperatures but not for temperatures at the depths where ruptures occurred. This lack of vomiting is at variance with other reports.

Of concern is the number of divers rupturing their eardrums during their initial course in scuba diving. However these cases were seen before the introduction of the PADI diving education. Nevertheless instructors should be aware of this and ensure their pupils understand that they must not ignore the sensations of pressure, ache or pain in their ears. Of the 15 divers whose ear drums ruptured doing a course, 10 had atrophic eardrums. Atrophic eardrums could be some bar to such people diving but it would be impractical to police. Anyway there must be many divers with atrophic eardrums who have no problems and such cases do heal spontaneously. Very few re-rupture with normal diving.

There may be some concern about the diagnostic criteria for ruptured eardrums. However the cause and the symptoms are the same in both groups surveyed; that is those cases seen with perforations and the others. Of those seen with perforations, 6 had no symptoms but only signs such as bleeding or discharge from the ear.

**TABLE 5**

**13 RE-PERFORATIONS IN 10 CASES**

| **7 single re-perforation** | **Operation** |
|---|---|
| 1 twice | spontaneous cure |
| 1 twice | first spontaneous cure<br>second operation |
| 1 twice | twice operation |

Conclusion

Ruptured eardrums in scuba divers, when properly handled, do not cause any long term effect. Although possible serious sequelae have been described in the literature, in this series there was no indication that these divers were seriously at risk. Of the cases who required treatment, most went back diving. Of those who had the full myringoplasty operation none ever came back with a re-rupture. It is probable that after operation their eardrums were no longer atrophic.

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*Dr Noel Roydhouse VRD, ChM, FRCS(Eng), FRACS was formerly Head of Department, ENT Department, Middlemore Hospital, Auckland, New Zealand. His address is 110 Remuera Road, Auckland 5, New Zealand.*

**ROYAL AUSTRALIAN NAVY**
**MEDICAL OFFICERS UNDERWATER MEDICINE COURSE**
November 1998

For further details apply directly to
The Officer in Charge, Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Road, Mosman, New South Wales 2088
Telephone (02) 9960 0572
Fax (02) 9960 4435

# A NEW SYSTEM FOR SCORING SEVERITY AND MEASURING RECOVERY IN DECOMPRESSION ILLNESS

Simon Mitchell, Tony Holley and Des Gorman

**Key Words**
Decompression sickness, treatment sequelae.

## Introduction

A prerequisite for the investigation of any therapy for a disease is an objective scoring system for both severity at presentation and response to treatment. Decompression illness (DCI) is particularly difficult in this context as the clinical presentations are protean. The conventional practice of describing recoveries as nil, incomplete or complete can be very misleading, as this results in a relative weighting for a complete recovery of paraesthesiae in a left ring finger over a 95% recovery in a tetraplegic. The overall effect is to introduce a potentially significant bias.

Several classification or scoring systems for DCI severity have been proposed. These systems either classify DCI patients into prognostic groups or score disease severity in individual patients.

For example, Dutka[1] proposed a modification to the currently popular descriptive classification of DCI.[2] The modified system classifies DCI according to the latency of onset; the "tempo" of disease evolution after symptoms appear; and the organ systems affected. This modification separates DCI patients into more clearly defined groups and may have more prognostic value than the original system, although this has not been established. In addition, while this novel system will enable group selection, it will not provide a means of tracking patient progress during and after treatment.

An easily calculated severity score, derived from the sum of a sensory symptom grade and a motor symptom grade, was proposed for neurological DCI by Dick and Massey.[3] Ball subsequently reported this system to have prognostic value and used the percentage change in scores to track patient progress during treatment.[4] However, the system is insensitive to those divers with primarily dorsal column spinal lesions and populations of patients who do not have objective neurological findings; as such it would therefore not be applicable to approximately 50% of patients presenting to Australasian hyperbaric units.[5]

Boussuges et al.[6] have proposed a "gravity score" for DCI which is derived from a summation of sub-scores allocated on the basis of: the presence of repetitive diving; the clinical course of the disease before treatment; and the presence of selected neurological findings. Selection of these criteria and the weighting of the sub-scores allocated to them was based on an analysis of 96 DCI cases. The scoring system itself was then validated on another population of 66 divers. This system has prognostic validity and is promoted as useful for "assessing the gravity of a population with a view to comparing the efficiency of different therapeutic protocols". However, the system is not designed to track a patient's progress during treatment nor to quantify recovery. Also, the system is not applicable to populations of patients who do not have objective neurological findings.

Kelleher and his colleagues[7] have developed a system to predict the probability of incomplete resolution after the first recompression treatment. They reviewed 214 cases of neurological DCI and recorded the type of deficit (for example sensory or motor), the number and anatomical location of sites involved, and outcome after the first treatment. They analysed these data to determine the prognostic significance of: the type of deficit; combinations of deficit types and the number of sites involved; and combinations of deficit types and the anatomical sites involved. While this valuable work allows some assessment of prognosis, it cannot be used to track patient progress or recovery.

The Slark Hyperbaric Unit at the Royal New Zealand Navy Hospital has initiated a randomised, prospective, controlled double blind trial of lignocaine (lidocaine) as a adjuvant to recompression therapy in the treatment of DCI. For the purposes of this trial we required a scoring system for DCI severity which provided:

1. applicability to "all" patients presenting with DCI (not just those with spinal syndromes);
2. a numerical index of severity at presentation;
3. quantitative tracking of patient progress during treatment; and
4. an index of recovery to allow comparison between patient groups.

None of the DCI severity scoring systems currently proposed or in place meets these requirements. Therefore we have designed the system described in this paper.

## Methods

The fundamental premise upon which the RNZN scoring system is based is that each symptom or sign of DCI will be scored. The scores for any symptoms or signs present will then be summed to give an overall DCI severity score. This score can be calculated at any point during a patient's treatment and the degree of any recovery (recovery score) determined by subtracting the current from the initial severity score.

The initial step in the design of this system was to produce a scoring algorithm which accounted for the severity of each symptom or sign of DCI, but with no attempt to define relative significance or importance. A four point scale, from 0 (symptom or sign absent) to 3 (maximum manifestation) was adopted and a descriptive guide to the allocation of these scores was designed for each symptom and sign (see Table 1 pages 86-91). Scores derived at admission are referred to as "admission scores" and scores derived at follow up assessments are referred to as "progress scores".

The second step was to derive a series of conversion factors which would modify the admission or progress scores for each symptom or sign to better indicate its relative prognostic and functional significance. Because of the general lack of objective prognostic data for individual symptoms and signs of DCI, these draft conversion factors were obtained by the independent rating of each symptom and sign by three experienced diving physicians. The scales used in this process were: specificity for DCI; natural history if untreated; and potential for incapacity (Table 2). The natural history scale was numerically emphasised since a symptom or sign which is likely to resolve spontaneously, even if untreated, was considered to be unimportant. A fourth scale, the co-dependence compensation (Table 2), was added to increase the importance of symptoms or signs whose presence would prevent or invalidate both the assessment of certain other manifestations and any contribution by the latter to the DCI severity score. Co-dependent symptom relationships that were recognised are listed in Table 3 (page 91).

For each symptom or sign, the ratings on the four scales were summed to derive an importance index (maximum 20) which was assumed to reflect relative importance (Table 4 page 92). Next, the importance index for each symptom or sign was divided by 3 (the maximum score for any manifestation on the unweighted scales in Table 1) in order to derive a conversion factor. For example, the conversion factor for lower limb weakness is derived from its importance index of 20, divided by 3, to give 6.67.

The third step was to add a second conversion factor to allow for the progression of disease before recompression, as this has been shown to be prognostically important.[6] The four disease progression categories previously used in the descriptive classification for DCI[2] were adopted for use in this scoring system. The conversion factors were arbitrarily allocated as follows: symptom static 1.0; symptom remitting 0.75; symptom progressive 1.25; symptom relapsing 1.25. The "progressive category" would include the group of "abrupt" onset patients considered prognostically important by Dutka.[1] Treatment of lignocaine trial patients is stopped when all symptoms have either resolved or the rate of change over two consecutive treatments approaches zero. It follows that the "static" conversion factor (1.0) is assumed at discharge and subsequent reviews.

**TABLE 2**

**FOUR IMPORTANCE WEIGHTING SCALES FOR A SYMPTOM OF DCI**

1 The **specificity** of the symptom in the context of a diver presenting with possible DCI
- 0 = often not related to DCI
- 1 = attribution to DCI sometimes doubtful
- 2 = almost always related to DCI

2 The **natural history** of the symptom if the diver was untreated.
- 0 = almost certain to resolve spontaneously
- 2 = very likely to resolve spontaneously
- 4 = sometimes resolves spontaneously
- 6 = very likely to persist
- 8 = almost certain to persist

3 The **incapacity potential** of the symptom assuming it persists at a moderate severity
- 0 = almost no potential to incapacitate
- 1 = annoyance potential with no effect on activities of daily living / social / employment
- 2 = potential to cause disruption to work but unlikely to cause loss of job, may affect socially but not activities of daily living
- 3 = potential for profound effect on employability, possible loss of job, but unlikely to effect independence
- 4 = likely to cause loss of job and dependence in activities of daily living

4 The **co-dependence compensation** loading for the symptom
- 0 = no co-dependent symptoms
- 2 = 1 co-dependent symptom
- 4 = 2 co-dependent symptoms
- 6 = 3 or more co-dependent symptoms

The DCI severity score can be calculated by:
1. scoring each symptom or sign using the unweighted scoring system (Table 1);
2. multiplying the unweighted score for each symptom or sign score by its importance and progression conversion factors (CFs); and
3. summing the products of these calculations (excluding co-dependent symptoms).

A DCI recovery score can be calculated by subtracting a current severity score from the initial severity

*Continued on page 91*

**TABLE 1.**

**UNWEIGHTED SCORING SYSTEM FOR INITIAL ASSESSMENT OF EACH SYMPTOM OF DCI**
**(Admission scores are calculated at admission. Progress scores are calculated at later reviews)**

**1 LETHARGY / FATIGUE / MALAISE / FEELING "OFF COLOUR"**
*(Note: this group of non specific "constitutional" symptoms is treated as a single entity. There is a separate scale for mood changes and for cognitive changes)*

Admission score

The patient is asked to grade symptoms as: **0** = nil; **1**= mild; **2** = moderate; **3** = severe. As a guide, score 1 would correlate with comments such as "I feel more tired / demotivated / "off colour" than usual but I'm coping easily"; score 2 would correlate with comments such as "I feel more tired / demotivated / "off colour" than usual and I'm having trouble getting on with normal daily activities"; score 3 would correlate with comments such as "I feel more tired / demotivated / "off colour" than usual and I just want to stay in bed / can't cope with normal daily activities".

At reviews the patient completes a 0-10 visual analogue scale (VAS) which compares the current severity with that *just prior to initiation of treatment.*

Progress score = admission score x (current VAS score ÷ 10)

**2 MOOD CHANGE**
*(Note: be careful not to confuse this with lethargy etc above)*

Admission score

The patient is asked to grade any mood change as: **0** = nil; **1** = minimal change; **2** = marked change; **3** = severe change. As a guide: score 1 might correspond to comments like "I feel a bit down / irritable more often than I used to"; score 2 might correspond to comments like "I feel quite depressed / angry a lot of the time"; and score 3 might be indicated by suicidal ideation or violent behaviour.

At reviews the patient completes a 0-10 visual analogue scale which compares the current severity with that *just prior to initiation of treatment.*

Progress score is given by admission score x (current VAS score ÷ 10)

**3 HEADACHE**

Admission score

The patient is asked to grade headache as: **0** = nil; **1** = mild; **2** = moderate; **3** = severe. As a guide, score 1 would correlate with comments such as "It's there but I only notice it if I think about it"; score 2 would correlate with comments such as "I am aware of it all the time but it doesn't affect my normal activities; score 3 would correlate with comments such as "It's so bad that I can't concentrate on anything else".

At reviews the patient completes a 0-10 visual analogue scale which compares the current severity with that *just prior to initiation of treatment.*

Progress score = admission score x (current VAS score ÷ 10).

**4 NAUSEA**

Admission score

The patient is asked to grade nausea as: **0** = nil; **1** = mild; **2** = moderate; **3** = severe. As a guide, score 1 implies that the patient feels "queasy" but not frankly nauseated or near to vomiting; score 2 implies that the patient is constantly aware of nausea and feels they may vomit; score 3 implies that the patient is incapacitated with nausea or is vomiting.

At reviews the patient completes a 0-10 visual analogue scale which compares the current severity with that *just prior to initiation of treatment*.

Progress score = admission score x (current VAS score ÷ 10).

## 5 TINNITUS

*Note: Tinnitus is only scored greater than 0 if it is suspected as arising secondary to DCI. Barotrauma is not considered.*

Admission score

The patient is asked to grade tinnitus as: **0** = nil; **1** = one ear; **2** = both ears; **3** = tinnitus in either ear can be heard over normal conversation.

At reviews the patient completes a 0-10 visual analogue scale which compares the current severity with that *just prior to initiation of treatment*.

Progress score = admission score x (current VAS score ÷ 10)

## 6 PARAESTHESIAE (TINGLING) AND OTHER SUBJECTIVE SENSORY ALTERATIONS

Two separate sub-scores are used to derive both the admission score and the progress score.

Intensity score

The patient is asked to grade paraesthesiae or another subjective sensory alteration as: **0** = nil; **1** = mild; **2** = moderate; **3** = severe, at the *worst location*. As a guide to paraesthesiae, score 1 implies equivocal and perhaps intermittent tingling; score 2 implies definite constant tingling; and score 3 implies uncomfortable prominent pins and needles. Where other subjective sensory alterations exist, or coexist with paraesthesiae, grade the most prominent alteration. For alterations other than paraesthesiae no guidelines are presented and the patient's subjective grading of mild, moderate, or severe will determine the score.

Distribution score

Consider each limb and girdle, the chest, the back, and the head as a "region": **0** = nil; **1** = one region; **2** = two regions; **3** = more than two regions.

Admission score = (intensity score + distribution score) ÷ 2

At reviews, as at admission, paraesthesiae (and / or other subjective sensory alterations) are evaluated with respect to both intensity and distribution.

Intensity

The patient completes a 0-10 visual analogue scale which compares the current intensity of the worst site and modality with those *just prior to initiation of treatment* (do not worry if the worst site and modality have changed).

Distribution score

Consider each limb and girdle, the chest, the back, and the head as a "region": **0** = nil; **1** = one region; **2** = two regions; **3** = more than two regions.

Progress score = [(admission intensity score x (current VAS score ÷ 10)) + current distribution score] ÷ 2

## 7 MUSCULOSKELETAL PAIN (INCLUDING GIRDLE PAIN)

Two separate sub-scores are used to derive both the admission score and the progress score.

Intensity score

The patient is asked to identify the location of greatest pain and mark a 0 - 10 visual analogue scale according to

how the pain compares to the worst pain they have ever felt. The intensity score is then derived as follows: VAS 0 = score **0**; VAS 1 - 3 = score **1**; VAS 4 - 7 = score **2**; VAS 8 - 10 = score **3**.

Distribution score

Consider each arm / shoulder; leg / hip; the back; the neck; and the chest as a"region": **0** = nil; **1** = one region; **2** = two regions; **3** = more than two regions.

Admission score = (intensity score + distribution score) ÷ 2

At review, as at admission, musculoskeletal pain is evaluated with respect to both intensity and distribution.

Intensity

The patient identifies the *current* worst site and completes a 0 - 10 visual analogue scale which compares the current severity at that site with the pain at the worst site *just prior to initiation of treatment*, **not** against the worst pain ever felt (do not worry if the worst site has changed).

Distribution score

Consider each arm / shoulder, leg / hip, the back, the neck, and the chest as a"region": **0** = nil; **1** = one region; **2** = two regions; **3** = more than two regions.

Progress score = [(admission intensity score x (current VAS score ÷ 10)) + current distribution score] ÷ 2

## 8 WEAKNESS

Two separate sub-scores are used to derive both the admission score and the progress score.

Intensity score

Grade the power in the weakest muscle group using the standard system, viz: **5** = normal; **4** = less than normal but able to resist gravity plus some extra force; **3** = able to resist gravity only; **2** = unable to resist gravity, but movement around a supported joint; **1**= flicker of movement only; **0** = no movement. The intensity score is then derived as follows: Power 0-2 = score **3**; Power 3 = score **2**; Power 4 = score **1**; Power 5 = score **0**.

Distribution score

Score the number of locations at which objective weakness is detected as follows: **0** = nil; **1** = one muscle group; **2** = more than one muscle group on same limb; **3** = weakness in more than one limb.

Admission score = (intensity score + distribution score) ÷ 2

Progress score is derived exactly as for admission score.

## 9 COGNITIVE DISTURBANCE

(*Note: includes problems with memory, attention, concentration)*

Admission score

Perform an MMSE and elicit the degree to which the patient feels they are impaired with respect to functions such as concentration, memory, attention. Score as follows: **0** = no impairment; **1** = mild impairment with no significant difficulty working; **2** = moderate impairment such that work would be difficult; **3** = essentially unable to work because of cognitive difficulty or MMSE score < 25. Note: if there is a clear explanation for a poor MMSE score such as poor educational level, do not consider the MMSE result in allocating a score.

Progress score is calculated exactly as for admission score.

## 10 OBJECTIVE SENSORY ALTERATION

These scores grade objective changes to touch, pain, temperature, vibration, proprioception. *(Note, the pairings of*

*pain and temperature, and vibration and proprioception, are each considered as one modality.)*

Two separate sub-scores are used to derive both the admission score and the progress score.

Intensity score

Score **0** = no abnormality; Score **1** = single modality affected; Score **2** = two modalities affected; Score **3** = three modalities affected.

Distribution score

Score **0** = no abnormality; Score **1** = one limb only affected; Score **2** = greater involvement than one limb but changes unilateral; Score **3** = greater involvement than one limb and changes bilateral (includes saddle area deficits).

Admission score = (intensity score + distribution score) ÷ 2

Progress score is calculated exactly as for admission score.

## 11 VISUAL DISTURBANCE

Admission score

A visual disturbance is scored as follows: **0** = nil; **1** = subjective deficit but no signs; **2** = visual field defect to confrontation or other signs on examination; **3** = blindness (less than 6/60 vision in either or both eyes).

Progress score is calculated exactly as for admission score.

## 12 CO-ORDINATION

Admission score

Assessed by finger-nose-finger, rapid alternating movement, and heel-knee-shin tests. **0** = no deficit; **1** = subtle difficulty, for example, occasional past pointing or tremor; **2** = clear evidence of past pointing, tremor, dysdiadochokinesis, **3** = frank inability to perform any one test.

Progress score is calculated exactly as for admission score.

## 13 GAIT

Admission score

A gait disturbance is scored as follows: **0** = no deficit; **1** = walks unaided at normal pace but gait abnormal; **2** = walks unaided but pace and gait abnormal; **3** = cannot walk without support or cannot walk at all.

Progress score is calculated exactly as for admission score.

## 14 BALANCE

Record the best time (seconds) of four attempts at the Sharpened Romberg test (SRT) (maximum of 60 seconds). If the patient achieves 60 seconds on any attempt, no further attempts are necessary.

Admission score

Scoring is as follows: best SRT time 41 - 60 seconds = score **0**; 26 - 40 seconds = **1**; 11 - 25 seconds = **2**; 0 - 10 seconds = **3**.

Progress score is calculated exactly as for admission score.

### 15 SPEECH

Admission score

A speech disturbance is scored as follows: **0** = no deficit; **1** = subjective abnormality only; **2** = mildly abnormal, for example, slight speech slurring; **3** = definite abnormality, for example, significant dysarthria, dysphasia.

Progress score is calculated exactly as for admission score.

### 16 REFLEXES

Admission score

Examination of the reflexes is scored as follows: **0** = no abnormality; **1** = abnormal reflexes confined to one limb; **2** = abnormal reflexes in more than one limb; **3** = abnormal reflexes plus up-going plantar(s) or clonus.

Progress score is calculated exactly as for admission score.

### 17 GENITO-URINARY FUNCTION

Admission score

GU function is scored as follows: **0** = no problem; **1** = subjective difficulty with any of: initiating stream; power of flow; or stopping stream; **2** = clear objective difficulty with any of: initiating stream; power of flow; or stopping stream, but still able to void; **3** = any of gross incontinence; inability to void; impotence.

Progress score is calculated exactly as for admission score.

### 18 RASH.

Admission score

Rash attributable to DCI is graded as follows: **0** = no rash; **1** = fine macular rash present but difficult to see; **2** = distinct rash; **3** = prominent erythematous rash with raised macules.

Progress score is calculated exactly as for admission score.

### 19 HEARING LOSS

Admission score

If the patient believes that hearing loss has occurred as a result of DCI, perform an audiogram and examine the ears. **If** the audiogram shows a sensorineural loss, there are symptoms (other than audiovestibular) to support the diagnosis of DCI and there is no clear evidence of middle ear barotrauma beyond grade II, then score the patient as follows: score **0** = subjective changes with normal audiogram (no loss greater than 20 dB at any frequency in either ear); score **1** = hearing loss 20-40 dB any frequency either ear; score **2** = hearing loss 40-60 dB any frequency both ears; score **3** = hearing loss greater than 60 dB either or both ears.

Progress score is calculated exactly as for admission score.

### 20 DIZZINESS / VERTIGO

Admission score

If there is vertigo / dizziness in association with non-audiovestibular symptoms typical of DCI, then score the dizziness / vertigo as follows: score **0** = nil; score **1** = subjective “dizziness” without true vertigo; score **2** = true vertigo (accompanied by nystagmus) intermittently or with provocation; score **3** = unremitting true vertigo.

Progress score is calculated exactly as for admission score.

### 21 BOWEL DYSFUNCTION

Admission score

Bowel dysfunction is scored as follows: score **0** = no dysfunction; score **1** = subjective change only; score **2** = decreased anal sphincter tone without fecal incontinence; score **3** = decreased anal sphincter tone with fecal incontinence.

Progress score is calculated exactly as for admission score.

### 22 LYMPHATIC INVOLVEMENT

Admission score

Consider each of the anterior cervical, sub-mental, maxillary, posterior triangle, axillary, supra-clavicular, inguinal areas as a "node region". Where lymphatic involvement arises in relation to other symptoms of DCI, score as follows: score **0** = nil; score **1** = enlarged tender lymph nodes in one node region; score **2** = enlarged tender lymph nodes in more than one node region; score **3** = enlarged tender lymph nodes with associated oedema.

Progress score is calculated exactly as for admission score.

**TABLE 3**

**CO-DEPENDENT SYMPTOM RELATIONSHIPS**

| Primary symptom | Co-dependent symptoms that are not used in calculation of the severity score |
|---|---|
| Lower limb weakness | Gait disturbance |
| | Balance disturbance |
| | Lower limb coordination |
| Upper limb weakness | Upper limb co-ordination |
| Objective sensory change | Paraesthesiae and other subjective sensory change |
| Balance disturbance | Gait disturbance |
| Dizziness / vertigo | Balance disturbance |
| Lower limb co-ordination | Gait disturbance |

*Continued from page 85*

score. Where a full recovery has occurred, the recovery score will be equal to the initial severity score. Where no improvement has occurred, the recovery score will be 0, and if the patient has actually deteriorated the score will be negative.

Clearly, some patients cannot or should not be assessed using this algorithm as this system can only be used for patients who have undergone an assessment which can detect all relevant disease manifestations. Consequently, this would exclude the following patients: those who require emergency recompression; those whose Glasgow coma score (GCS) is less than 15/15; those who the examiner is reluctant to move from the supine position for fear of posturally induced arterial gas embolism; or those who are not fluent in the same language as the examining doctor.

**Case reports**

Two cases are presented below to illustrate the application of the system.

*Case 1*

A 32 year old male had dived to 21 m for 40 minutes. He presented 30 hours after diving.

Admission symptoms

- Intense lethargy
- Mood swings (transient emotional shifts e.g. started crying for no reason)
- Nausea without vomiting
- Bilateral shoulder pain 2/10
- A fine, difficult to see, rash on his chest
- (all symptoms static, except shoulder pain which was remitting)

**TABLE 4**

**DERIVATION OF THE IMPORTANCE INDEX AND CONVERSION FACTOR FOR EACH SYMPTOM**

| Symptom / sign | Specificity | Natural history | Incapacity | Co-dependent | Importance index | Conversion factor |
|---|---|---|---|---|---|---|
| Weakness lower limbs | 2 | 8 | 4 | 6 | 20 | **6.67** |
| Weakness upper limbs | 2 | 8 | 4 | 2 | 16 | **5.33** |
| Genito-urinary disturbance | 2 | 8 | 3 | 0 | 13 | **4.33** |
| Gait disturbance | 2 | 6 | 4 | 0 | 12 | **4.00** |
| Objective sensory change | 2 | 6 | 2 | 2 | 12 | **4.00** |
| Bowel dysfunction | 2 | 6 | 3 | 0 | 11 | **3.67** |
| Coordination deficit lower limb | 2 | 4 | 3 | 2 | 11 | **3.67** |
| Balance disturbance | 1 | 4 | 3 | 2 | 10 | **3.33** |
| Visual disturbance | 2 | 4 | 3 | 0 | 9 | **3.00** |
| Coordination deficit upper limb | 2 | 4 | 3 | 0 | 9 | **3.00** |
| Speech disturbance | 2 | 4 | 3 | 0 | 9 | **3.00** |
| Hearing loss | 0 | 6 | 3 | 0 | 9 | **3.00** |
| Mood disturbance | 1 | 4 | 3 | 0 | 8 | **2.67** |
| Cognitive disturbance | 1 | 4 | 3 | 0 | 8 | **2.67** |
| Dizziness / vertigo | 1 | 0 | 4 | 2 | 8 | **2.67** |
| Tinnitus | 0 | 6 | 1 | 0 | 7 | **2.33** |
| Paraesthesiae or other subjective sensory change | 2 | 2 | 1 | 0 | 5 | **1.67** |
| Musculoskeletal pain | 1 | 2 | 2 | 0 | 5 | **1.67** |
| Abnormal reflexes | 1 | 4 | 0 | 0 | 5 | **1.67** |
| Lymphatic involvement | 1 | 2 | 1 | 0 | 4 | **1.33** |
| Nausea | 0 | 0 | 2 | 0 | 2 | **0.66** |
| Lethargy / fatigue | 0 | 0 | 2 | 0 | 2 | **0.66** |
| Headache | 0 | 0 | 2 | 0 | 2 | **0.66** |
| Rash | 1 | 0 | 1 | 0 | 2 | **0.66** |

Admission diagnosis

Musculoskeletal / constitutional / ? neurological DCI

Severity score at admission (CF = conversion factor)

Admission score lethargy = 3 x CF importance 0.66 xCF progress 1 = 2

Admission score mood = 2 x CF importance 2.67 x CF progress 1 = 5.34

Admission score nausea = 1 x CF importance 0.66 x CF progress 1 = 0.66

Admission score pain = 1.5 x CF importance 1.67 x CF progress 0.75 = 1.9

Admission score rash = 1 x CF importance 0.66 x CF progress 1 = 0.66

Score = 2 + 5.34 + 0.66 + 1.9 + 0.66 = 10.6

Discharge symptoms

None

Severity score at discharge:

No symptoms therefore Score = 0

Recovery score at discharge:

Severity admission 10.6 - discharge 0 = Score 10.6

*Case 2*

A 33 year old male had dived to 55 m for 15 minutes with a rapid ascent. He presented 2 hours after diving.

Admission symptoms

Pain right shoulder 2/10

Paraesthesiae both legs constant and prominent

Weakness all groups both legs, worst 3/5

Unable to pass urine

Unable to walk

Objective deficit to light touch and pain both legs

Balance - unable to stand unsupported

Coordination - Unable to perform heel-knee-shin test (HKS) on left, clumsy on right

Reflexes - clonus at both ankles, bilateral up-going plantars

(All symptoms progressive, except shoulder pain which was static)

Admission diagnosis
Progressive neurological (spinal) DCI

Severity score at admission (CF = conversion factor)
- Admission score pain = 1 x CF importance 1.67 x CF progress 1 = 1.7
- Admission score paraesthesiae = 2 x CF importance 1.67 x CF progress 1.25 = 4.2
- Admission score lower limb weakness = 2.5 x CF importance 6.67 x CF progress 1.25 = 20.8
- Admission score bladder = 3 x CF importance 4.33 x CF progress 1.25 = 16.2
- Admission score gait = 3 x CF importance 4 x CF progress 1.25 = 15
- Admission score objective sensory change= 2.5 x CF importance 4 x CF progress 1.25 = 12.5
- Admission score balance = 3 x CF importance 3.33 x CF progress 1.25 = 12.5
- Admission score lower limb co-ordination = 3 x CF importance 3.67 x CF progress 1.25 = 13.8
- Admission score reflexes = 3 x CF importance 1.67 x CF progress 1.25 = 6.3

*Note. Gait, balance, and coordination are co-dependents of lower limb weakness; paraesthesiae is a co-dependent of objective sensory change. These scores are therefore not included in the severity score calculation.*

Score = 1.7 + 20.8 + 16.2 + 12.5 + 6.3 = 57.5

Discharge symptoms
Reflexes, clonus at left ankle
Gait, limping but normal pace
Objective deficit to light touch and pain in both legs
Balance, sharpened Romberg test score 35 seconds

Severity score at discharge (CF = conversion factor)
- Progress score reflexes = 3 x CF importance 1.67 x CF progress 1 = 5
- Progress score gait = 1 x CF importance 4 x CF progress 1 = 4
- Progress score objective sensory change = 2.5 x CF importance 4 x CF progress 1 = 10
- Progress score balance = 1 x CF importance 3.33 x CF progress 1 = 3.33

*Note. With the weakness resolved, gait and balance are no longer co-dependent and these are included in the calculation of the discharge severity score.*

Score = 5 + 4 + 10 + 3.3 = 22.3

Recovery score at discharge

Severity admission 57.5 - discharge 22.3 = Score 35.2

**Discussion**

Although the development of this scoring system for DCI severity is cumbersome to describe, the system is simple to use. Importantly, it can be applied to divers presenting with a wider spectrum of clinical problems than any of the others proposed. Indeed, this is the first system which allows severity scoring in those divers who present with either trivial or no neurological signs: a presentation which we see commonly in Australasian sport divers and consider to be important.

The prognostic significance of many of the symptoms and signs of DCI is not described by data, and it follows that assessment of their relative importance for a scoring system of the type described here will inevitably involve subjective ratings. We have rationalised this process by the use of rating scales designed to reflect both prognostic significance and incapacity potential. This rating system has resulted in a ranking of relative importance (Table 4 page 92) which seems consistent with the limited data which establishes prognostic significance for some symptoms and signs of DCI.[4,6,7]

This system has the significant advantage of providing a recovery score which should allow comparison of recovery between patient groups. It is notable that the first of the two illustrative cases presented here achieved full recovery, while the second did not. However, the recovery achieved by the second patient was significantly greater from a functional perspective, and this is reflected in the recovery scores. Quite the opposite interpretation would accrue from the traditional consideration of recovery as “complete” or “incomplete”. We have chosen to assess recovery by subtracting the score at review from the initial score, as this method gives the most accurate recovery measurement. Other authors using scoring systems have calculated “percentage recovery”,[4] but unless cases are stratified to account for severity, this system risks creating the same error as the division of recovery into complete or incomplete categories.

We propose that this system is useful in the context of clinical trials in DCI therapeutics. The system is designed to be easily adjusted and patients re-scored either in response to suggestions generated by this discussion paper, or as data describing the prognostic significance of symptoms and signs accumulate. Dr Tony Holley has completed a validation study of 100 cases of DCI treated at the Royal New Zealand Navy Hospital which investigates the prognostic value of the severity score at admission. This will be presented at the Undersea and Hyperbaric Medicine Society 1997 Annual Scientific Meeting and published in a later edition of the SPUMS Journal as his DipDHM project.

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*Dr Simon Mitchell is the Director of the Slark Hyperbaric Unit at the Royal New Zealand Navy Hospital.*

*Dr Tony Holley is a full time Medical Officer at the Royal New Zealand Navy Hospital.*

*Dr Des Gorman is Associate Professor of Medicine at the School of Medicine, University of Auckland, and Visiting Consultant in Diving and Hyperbaric Medicine at the Slark Hyperbaric Unit.*

*Address for correspondence*

*Dr Simon Mitchell, Slark Hyperbaric Unit, Naval Base, Auckland, New Zealand. Phone +64-9-445-5922. Fax +64-9-445-5941.*

## NON-STEROIDAL ANTI-INFLAMMATORY DRUGS IN DECOMPRESSION ILLNESS: A PRELIMINARY REPORT

Mike Bennett

**Key Words**

Decompression illness, drugs, hyperbaric oxygen, treatment.

**Introduction**

This brief presentation is a progress report on the multi-centre, randomised controlled trial currently underway into the efficacy of adjunctive tenoxicam (Tilcotil, Roche Pharmaceuticals), a non-steroidal anti-inflammatory drug (NSAID), in the treatment of decompression illness (DCI). The pharmacology of such an agent and the rationale for administration are subjects for another presentation at this meeting and so will not be dealt with in any detail.

At the Prince Henry and Prince of Wales Hospitals in Sydney it has been the practice of some of our clinicians to administer a NSAID as adjunctive therapy for divers (and others) suffering with DCI. Thus, in addition to standard recompression tables and fluid replacement, divers would typically receive piroxicam (Feldene - Roerig Pharmaceuticals) dispersible 20 mg daily for 7 days in the expectation that such treatment may improve the resolution of symptoms both in the short and medium term. This practice was not based on any objective evidence. This study is to elucidate the efficacy or otherwise of this approach. It is in the early stages and no analysis which involves breaking the randomisation code has yet been made.

**Rationale of the study**

The treatment of DCI has traditionally been limited to recompression, use of 100% oxygen and appropriate decompression schedules. Correction of dehydration and appropriate posturing to prevent any (or further) gas entering the cerebral circulation are accepted as important adjunctive measures.

This regime has proved very effective in treating military and professional divers where recompression facilities are immediately available. However, it has recently become clear that there is a significant rate of incomplete resolution of symptoms and signs in several series of recreational divers with DCI. In Australasia this rate is typically between 20 and 35% of all cases seen.[1-4] This has recently been confirmed in a report from our unit in Sydney.[5] In addition, it is often noted that recreational divers require more treatments to achieve resolution than professionals.

The reasons for these differences are likely to be multifactorial but are assumed to be, at least in part, due to increased times between symptom onset and recompression, although this remains to be clearly demonstrated.[5] A number of adjunctive therapies have been suggested to improve the resolution rate and reduce the number of compressions required. To date no randomised, controlled studies have suggested that any are of practical value. Much of the research that has been done, and is in progress, has focussed on the more dramatic end of the DCI spectrum, including cerebral arterial gas embolism. We do not propose that NSAIDs are likely to significantly alter the course of these profound problems. With regard to the peripheral and less severe forms of the illness, a number of pharmacological modifiers have been suggested, but no formal prospective studies have been reported.

Douglas[6] reported an excellent response to intramuscular diclofenac sodium in a patient with residual pain. The theoretical basis suggested to explain this improvement was the ability of the NSAID to modify the inflammatory response through the inhibition of prostaglandin synthesis. This effect has not been reported in other clinical cases or series.

In a retrospective review, Kizer[7] found that the administration of corticosteroids to a group of patients suffering delays to treatment greater than 12 hours did result in some benefit, but not in the peripheral manifestations of DCI. Aspirin in anti-platelet doses has been reported on by Bove[8] and by Catron and Flynn[9] but no positive benefit elucidated.

While other adjuvant agents are currently under examination, including intravenous lignocaine and high dose steroids, these are aimed at the more severe forms of the disease and we felt it was reasonable to evaluate the role of NSAIDs more effectively at this time.

## Method

Ethics committee approval was granted through the Eastern Sydney Area Health Service for a prospective, randomised, masked and controlled study of the efficacy of tenoxicam for the treatment of DCI. All patients presenting to the department with DCI grades one to five would be eligible, with exclusion only for patient refusal and the presence of known contraindications to NSAID administration (Table 1). The protocol flow diagram is shown in Figure 1 (page 96).

Following fully informed consent, a randomisation schedule is consulted to allocate a trial number and a corresponding course of tablets pre-packaged by the pharmacy department of the hospital. Only the Chief Pharmacist has knowledge of the randomisation code, although this is accessible in an emergency. The patient is

**TABLE 1**

**REASONS FOR EXCLUSION FROM NSAID TRIAL**

| |
|---|
| Refusal to consent. |
| History of sensitivity or complications secondary to NSAID administration. |
| History or symptoms suggestive of peptic ulceration, upper GIT bleeding, NSAID sensitive asthma or renal impairment. |
| Concurrent therapy with analgesic medication, anti-coagulants, frusemide, lithium or methotrexate. |

**TABLE 2**

**THE ADMISSION SEVERITY CODING TABLE**

| Grade | Symptoms and signs | Severity |
|---|---|---|
| ONE | Pain<br>Rash<br>Itching | Peripheral<br>Mild |
| TWO | Muscle/joint pain<br>Numbness/tingling<br>Restlessness<br>Headache | Peripheral and/<br>or Neurological<br>Mild to moderate |
| THREE | Tinnitus<br>Severe pain<br>Fatigue<br>Altered reflexes | Peripheral and/<br>or Neurological<br>Moderate |
| FOUR | Weakness<br>Nausea/Vomiting<br>Hearing loss<br>Personality change<br>Inco-ordination | Peripheral and/<br>or Neurological<br>Moderate to severe |
| FIVE | Visual disturbance<br>Speech disturbance<br>Weakness<br>Paralysis<br>Bladder/bowel dysfunction | Neurological and/<br>or Peripheral<br>Severe |
| SIX | Reduced level of consciousness<br>Paralysis<br>Convulsions<br>Cardiac dysrhythmia | Neurological and/<br>or CAGE<br>Severe<br>early onset |

Taken from Bond et al.[10] with some modification.

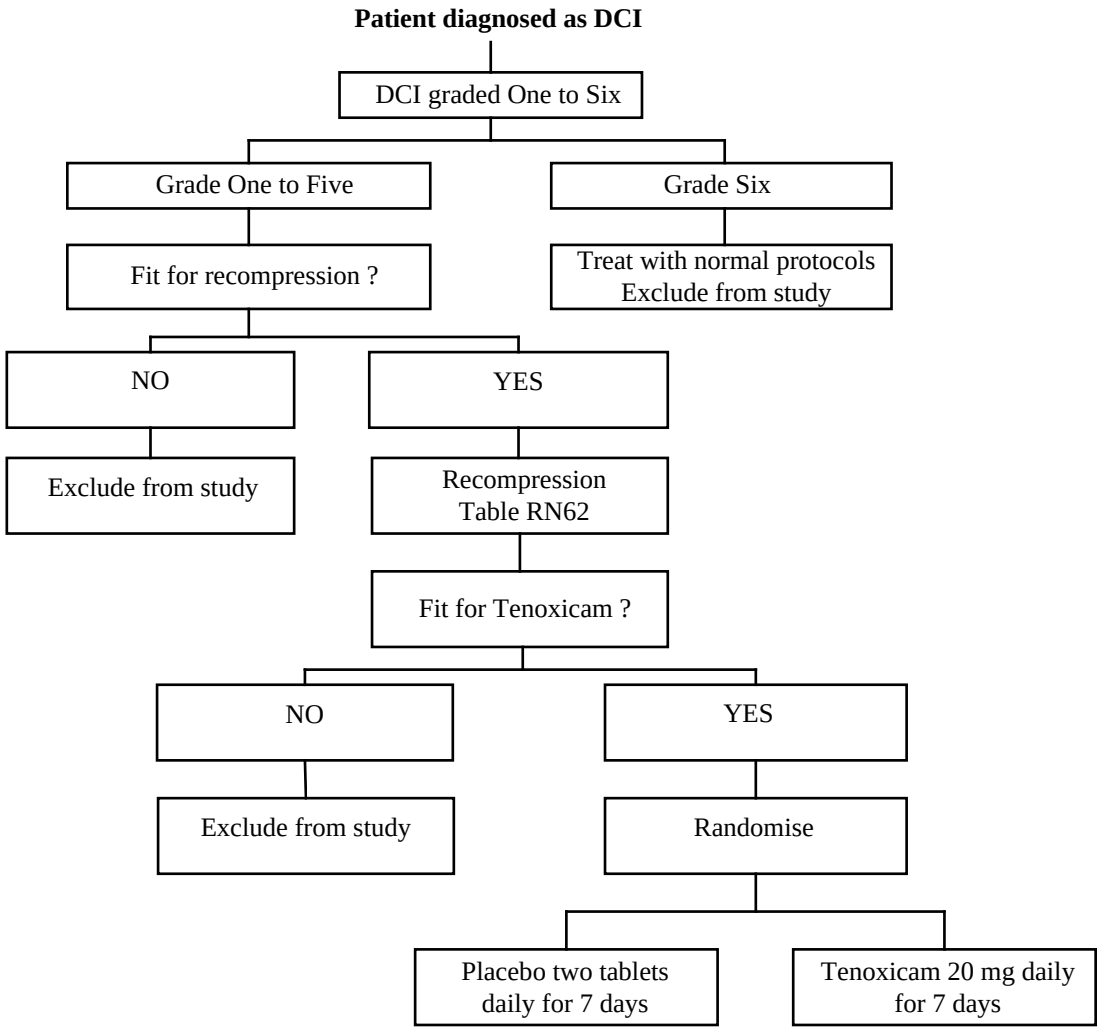


**Figure 1.** Protocol Flow Diagram.

graded for severity of DCI (Table 2). Grades do not imply bubble load or predict outcome but are generated for inter-unit comparative research. The patient is then recompressed using a standard RN 62 treatment table. At the first air break the patient's condition is reassessed and the first dose of Tenoxicam or placebo administered. Recompression is continued as clinically indicated by symptom resolution or attainment of a recovery plateau and the trial drug administered for seven days. The active treatment group receive 20 mg of Tenoxicam daily over this time. Following completion of the recompression phase of treatment, the patient's health status is assessed and recorded. Further assessments are made at 4 to 6 weeks and 6 months following discharge.

Discharge status is graded, on the scale shown in Table 3 , following clinical assessment at the relevant time. The main distinction sought is any significant difference between level one and levels two to five. Routine neuropsychiatric assessment was not included in this status score as this would be difficult to standardise over geographically separate institutions. All effort was made to make these assessments in the Hyperbaric Unit, however data was accepted from telephone consultations if mandated by practical considerations.

It is not considered, given the present state of our knowledge, that there were any ethical dilemmas involved in the withholding of NSAIDs from the placebo group. The

**TABLE 3**

**DISCHARGE STATUS AT SIX WEEK REVIEW**

| | |
|---|---|
| Level one | Well. No symptoms or signs |
| Level two | Minor symptoms or signs |
| Level three | Moderate impairment of function or quality of life. |
| Level four | Major incapacity. |
| Level five | Dead |

risks associated with short courses of Tenoxicam are well known and unlikely to prove a problem during the remainder of this trial. There have been no attributable side-effects to March 1997.

Based on an expected rate of complete resolution of 75% in the placebo group and an assessment that an improvement to 88% would be clinically significant in the active drug group, it is anticipated that about 180 patients will be needed to have a 90% chance of detecting such a difference with 95% confidence. In order to complete such a study within a reasonable time, other centres have been invited to contribute their patients. In March 1997 the Prince of Wales Hospital and HMAS PENGUIN were actively involved. Two other established Australian centres are to join shortly.

**Progress**

By March 1997 we had enrolled 26 individuals in the trial. There have been five other cases who did not enter the trial. Three chose to decline the opportunity while one was not asked and in one there was a contraindication to NSAID administration. The randomisation codes have not been broken for this report.

The majority (18 cases) fell into grade two on the admission scale, while the other four groups have contributed 2 cases each. The average number of treatments before discharge is 2.6 at this early stage.

Three cases have not yet had their 6 week follow-up. At this appointment 18 cases had complete resolution of symptoms while some symptoms persisted in 5. This represents a rate of less than complete resolution (discharge level >1) of 21.7%. Lying between the previously published rate for this unit (27%) and the proposed rate for a demonstration of clinically significant efficacy (12%), this result looks promising.

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*Dr Mike Bennett, MB BS, DA, FFARCSI, is Medical Director, Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, High Street, Randwick (in Sydney), New South Wales 2031, Australia. Phone +61-(0)2-9832-3883. Fax +61-(0)2-9832-3882.*

## LIGNOCAINE AND NON-STEROIDAL ANTI-INFLAMMATORY DRUGS AS ADJUVANT THERAPY IN TREATING DECOMPRESSION ILLNESS.

David Cosh

**Key Words**
Decompression illness, drugs, treatment.

**Lignocaine**

Simon Mitchell has reviewed the evidence supporting the use of lignocaine, an aminoethylamide local anaesthetic with class 1B anti-arrhythmic properties, as adjuvant therapy to the accepted modalities of compression and hyperbaric oxygen (HBO) in the treatment of decompression illness (DCI).[1] It is well known that tissue damage secondary to the presence of intravascular and extravascular bubbles can be caused by ischaemia, mechanical effects and inflammation. Nellgård et al. have demonstrated a potent anti-inflammatory effect of lignocaine in a rat model of bowel obstruction where jejunal fluid loss was converted into net fluid absorption by the administration of intravenous lignocaine in conventional doses (2 mg/kg). Lignocaine applied topically to the serosa proximal to the ligation was also effective.[2,3]

It is unlikely that the anti-inflammatory effect of lignocaine is solely responsible for its role in treating DCI. Three other pharmacodynamic effects of lignocaine, membrane stabilisation, reduction in cerebral oxygen consumption and favourable haemodynamic properties in the ischaemic brain, increased cerebral blood flow and reductions in both intracranial and mean arterial pressure, may also contribute to its efficacy.[1]

In considering the value of any drug commonly used acutely, the pharmacokinetics of the agent are especially relevant. Lignocaine can, and indeed because of a significant first pass effect, needs to be given parenterally. The drug is metabolised with renal excretion of metabolites and dosage adjustment is not normally required, except in cases of major hepatic or renal dysfunction. A short half life (1-2 hours) means that a steady-state can be quickly achieved and, in the event of overdosage, cessation of infusion will be followed by a relatively rapid diminution in blood level and unwanted effects. Lignocaine has a narrow therapeutic index, but the therapeutic range is well established (6-21 μmol/l). Drewry and Gorman, in a single case report of its use as adjuvant therapy in DCI, showed benefit from lignocaine administered in doses sufficient to achieve plasma levels at the lower end of this range.[5] Mitchell, when reviewing the in vivo animal data, showed that in studies where doses higher than those considered to be within the normal anti-arrhythmic range were used benefit was not as great as that obtained from the use of conventional doses.[1]

The adverse effects of lignocaine are predominantly seen in the cardiovascular and central nervous systems and are well known. The properties of the other parenteral aminoethylamide local anaesthetic agents (e.g. bupivacaine, etidocaine, mepivacaine, prilocaine, ropivacaine) do not suggest a role for any of these in preference to lignocaine. Mexiletine, structurally related to lignocaine, and available in both parenteral and oral dosage forms, is the only orally available option but is very poorly tolerated and has no properties that suggest that it should be considered in preference to lignocaine.

**Non-steroidal anti-inflammatory agents (NSAIDs)**

If the anti-inflammatory effect of lignocaine is considered beneficial in DCI then it is not unreasonable to consider the use of other pharmacological agents that interrupt inflammatory pathways. Hallenbeck et al., using a dog model, have shown that the NSAID indomethacin, in combination with heparin and prostaglandin $I_2$, speeds cerebral neuronal recovery from a standardised ischaemic insult. The effect of any of the three agents used alone was not statistically significant.[4]

NSAIDs exert their anti-inflammatory effect by inhibition of cyclooxygenase which subsequently leads to inhibition of the synthesis of prostaglandins.[6] Cyclooxygenase inhibition causes reversible inhibition of platelet aggregation. The individual contributions of the anti-inflammatory and anti-platelet effects of NSAIDs to the overall effect of the agents in DCI have not yet been defined.

Inhibition of prostaglandins in the kidney and gastrointestinal mucosa is responsible for the well known adverse effects, in both organs, seen with NSAIDs.[7,8] Which agent to use is a reasonable question to ask, given that there are fifteen different NSAIDs in a wide variety of dosage forms available in Australia. The desire of pharmaceutical companies to have at least one of these widely prescribed agents in their inventory, and the fact that no one drug stands out as being significantly clinically superior, are two reasons for the multiplicity of agents on offer. In practice, patient preference often remains the final arbiter.

While some of the agents are locally irritant to the gastric mucosa, toxicity is principally mediated systemically. The dramatic decline in renal function from normal to acute renal failure in otherwise well relatively young patients presenting for elective surgery and receiving the parenteral NSAID ketorolac (Toradol®) either peri- or post-operatively, as an alternative to traditional narcotic analgesic agents highlights the dangers associated with the administration of NSAIDs at a time when renal circulation is stressed.[9] Applying the same analogy to otherwise fit divers presenting with symptoms of DCI, resuscitation and fluid repletion should be completed and baseline urea and creatinine measured before using a NSAID. Ongoing monitoring of renal function during treatment with a NSAID in this setting would be prudent.

Case control studies in UK populations have shown that there is a difference in the propensity for different NSAIDs to cause damage to the gastric mucosa. Longer acting NSAIDs, while popular because of the need to take the drug only once daily, appear more toxic than some of the older shorter acting agents.[10-12] Piroxicam (Feldene®) consistently compares unfavourably with ibuprofen (Brufen®) while agents such as naproxen (Naprosyn®) and diclofenac (Voltaren®) fit somewhere in the middle. Accepting that some of the difference in toxicity may be explained by not using equivalent anti-inflammatory doses, a case can still be made for starting patients on ibuprofen before moving on to longer acting agents in the event of lack of response.[13] While known risk factors for gastrointestinal bleeding associated with NSAID use, such as old age, smoking, history of peptic ulcer disease and the presence of cardiovascular disease, are unlikely to be present in the majority of those presenting with DCI, providing anti-inflammatory efficacy is not compromised, it is reasonable to start with a drug with less gastrointestinal toxicity.

New developments in NSAID research include the combination of a nitric oxide releasing moiety with

conventional NSAIDs. In animal studies these agents have demonstrated anti-inflammatory efficacy comparable to the NSAID alone with less gastrointestinal toxicity.[14] Cyclooxygenase (Cox) exists in vivo as two molecules (Cox-1 and Cox-2). Whereas Cox-1 is ubiquitous occurring in most tissues, Cox-2 is far more localised and is inducible at sites of inflammation. Conventional NSAIDs inhibit both isoenzymes to varying extents. Naproxen and diclofenac are relatively Cox-2 specific when compared with piroxicam, which is not, and this ranking is consistent with the greater gastrointestinal toxicity seen with the latter agent. Highly specific Cox-2 inhibitors have the potential to be safer agents and one such drug, meloxicam is currently undergoing clinical trials in Australia.[15]

If studies currently in progress provide further evidence for a role for lignocaine and NSAIDs as useful agents in the adjuvant treatment of DCI, then combination therapy using lignocaine and a NSAID may prove worthy of investigation. While in the acute setting of any illness, including DCI, the use of NSAIDs should be tempered with an appreciation of their potential to cause damage to an already stressed gastric mucosa and under-perfused kidney, lignocaine has an established role in the emergency treatment of ventricular arrhythmias. While lignocaine may be the preferred initial agent, if on-going anti-inflammatory medication is indicated oral NSAIDs may have a role to play. If this proves to be the case, which NSAID to use, out of a confusing array of agents which will only get larger, remains to be determined.

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*David Cosh is a Clinical Pharmacist at the Repatriation General Hospital, Daw Park, South Australia 5041 and Senior Lecturer, School of Pharmacy and Medical Sciences, The University of South Australia.*

*Address for correspondence*

*David Cosh, Pharmacy Department, Repatriation General Hospital, Daw Park, South Australia 5041. Phone +61-(08)-8275-1799. Fax +61-(08)-8374-0225. E-mail scoshdg@rgh.sa.gov.au* .

# THE PADI APPROACH TO DIVER RESCUE TRAINING

Drew Richardson and Karl Shreeves

**Key Words**
Recreational diving, rescue, training

**Abstract**

Designing instruction for rescue skill training in recreational divers must address the question of what to teach (content), and when to teach it (sequence amid the development of prerequisite cognitive and motor abilities). The "old school" of diver training attempted to teach all aspects of rescue to the beginner diver. This has given way to training rescue skills by building basic skills in steps to assure prerequisite learning and psychological preparedness.

Teaching rescue skills content is a question of sequence. Examining rescue skills and student characteristics against the doctrines of instructional system design theory (ISDT), learning theory and cognitive psychology shows that premature rescue skill training has several potential pitfalls. Among these are potential failure to perform, failure to assimilate, failure to retain, and failure to transfer skills to rescue situations after training. Students may not recognise these failures in themselves and therefore have unrealistic estimations of their rescue abilities. The potential for these problems, despite otherwise appropriate teaching methodologies, arises, among other reasons, because of limitations in human attentional resources, and the absence of experienced-based mental frameworks and related domain-specific prerequisite intellectual skills. The preferred timing of practice sessions may also be an issue.

Sequence is also content related. There is a potential for increasing the complexity of a rescue when rescuers learn techniques as isolated skills. Both patient and rescuer benefit when the content integrates rescue and first aid in a priority hierarchy that parallels emergency medical care, though at the lay level. Adapting to local emergency medical protocols enhances this approach to content yet further.

The PADI system of diver education presents diver rescue training in a content based on established medical and rescue protocols and sequence based on ISDT, cognitive psychology and motor learning theory. This approach minimises or eliminates the problems associated with "old school" recreational rescue diver skill training to assure the assimilation, retention and application of appropriate rescue techniques and philosophies.

## Introduction

This paper covers the application of instructional theory and cognitive psychology to diver rescue skills assimilation, retention and transfer. It is a summary of the PADI system's educational approach.

Since the birth of recreational scuba diving, training rescue techniques has been associated with training divers. Scuba training texts from the 1950s into the 1970s reflect an approach that wrapped rescue among many other topics in a single course that was intended to teach all aspects of diving, including the broad range of rescue skills, first aid and rescue breathing. The approach of the "old school" was often to combine rescue procedures with discussions on human physiology; specific first aid for specific pathophysiologies was provided, but the broader topics and techniques of preparation, management, and the rescue itself were largely ignored.[1-4]

Diver training matured with adoption of educational principles derived from Instructional System Design Theory (ISDT), cognitive and educational psychology, and other theories of human learning. Training moved away from the invalid one-course-teaches-all concept and evolved into training in stages, mirroring mainstream education. This for allowed more attention to specific topics, including rescue training, which then became a large part of advanced diver programs in the late 1970s and early 1980s.[5] By 1985, in the PADI system of diver education, the training of rescue techniques had evolved into a distinct and separate program, with the beginner diver taught fundamental self-rescue and assisting skills which were built upon during subsequent training.[6] Though PADI courses have been revised since then to keep pace with change, the instructional hierarchy remains essentially the same, as reflected in current PADI materials.[7] Today, before rescue training, the student completes the PADI Open Water Diver course, the PADI Advanced Open Water course and then does the PADI Rescue Diver course, with CPR and first aid training a required component.[8]

A brief look at ISDT, cognitive psychology and other human learning principles as they relate to rescue training reveals why PADI's approach to rescue training has endured and been successful, while the "old school" approach of presenting novices with virtually all rescue techniques has declined. We can follow this with a brief look at the sources of training content, particularly emergency oxygen and CPR protocols.

## Instructional system design theory

ISDT is concerned with what the student must be able to do after being taught and in reaching that goal, analysing what to teach when. The design of instruction calls for a task analysis of the educational goal that looks at

both sequence and the information processing characteristics of learners.[9,10] Improper sequencing of material can hinder education because a student may lack prerequisite skills or knowledge for the task being learnt. However, instruction must also be sequenced and timed considering the student's psychological and physical preparedness to learn at a given time as well as the sequence called for by the information.[10,11] It is the question of psychological preparedness that leads to the inadequacy of teaching the full spectrum of rescue techniques to beginner divers.

**Attentional resources**

The human mind has a limited capacity to pay attention to stimuli. There are various models that address attentional resources, but most relevant to training divers in rescue skills are the effects of controlled processes compared with automatic processes.

Controlled processes are those that require conscious thought. Controlled processes require mental effort, consume attentional resources and are performed step-by-step. The individual must focus on the task to accomplish it. However, with repetition over time, a controlled process may become an automatic process. Automatic processes require little or no mental effort, consume few attentional resources and are performed in parallel with other tasks.[12] The automatic process level is called automacity, particularly where motor skills are involved.[11,13]

To teach complex motor skills (which includes diver rescue skills) instruction must be sequenced so that automacity is first attained in sub-skills. This frees the mind to concentrate on the aspects of the new motor skill, or new aspects of the same skill. Otherwise the skill may be too complex for the student's attentional resources. Beginner instruction must first develop those sub-skills that must be performed automatically.[13] (Magill 1993, 169-171)

The novice diver who has just learned basic diving skills, which are prerequisite to rescue skills, operates primarily at the controlled process level. The diver may have attained mastery and performs the skill competently, but must pay attention to the steps. The "old school" of diver training disregarded this, expecting novices to learn complex rescue skills while still in the controlled process stage with prerequisite skills.

It is not psychologically valid to expect the novice to learn complex rescue skills such as in-water artificial respiration when the student must still consciously think about needed skills such as buoyancy control or air way control. On the other hand, simple skills such as a tired diver tow or removing a cramp, are not particularly complex and are reasonable to teach at the novice level.

Because the novice operates on the controlled process level, the novice is self-focused. Therefore, at the novice level, the PADI system's philosophy is to emphasise simple rescue skills and problem management, such as cramp removals, tired diver tows, establishing buoyancy and other self rescue skills. The novice masters prevention, which is the most important rescue skill, and develops automacity with basic diving skills. Also, though novices are not ready to develop complex motor skills, they are ready to learn some of the concepts behind them. Therefore, novice divers in the PADI system are introduced to the basic concepts behind rescue procedures, including complex ones.[14,15]

The PADI Rescue Diver course follows both the novice course and the PADI Advanced Open Water program, which emphasises diving in different environments and while engaged in specialised activities. This sequence is consistent with the need for automacity because it ensures that the student has a minimum of nine dives, compared with no dives in the old school methodology when complex rescue training began in the pool. After nine actual dives, it can be expected that a diver's basic skills will be completely, or nearly completely, under automatic processes.

**Domain specific experience and schemata**

As mentioned, novice divers are initially self-oriented. As their controlled processes become automatic processes, the demand on their attentional resources drops. This allows them to focus beyond their basic diving skills and become more task oriented. In keeping with this psychological change the Advanced Open Water program trains the diver to accomplish task specific learning objectives ranging from deep-diving and navigation knowledge and skills, to underwater photography and studying nature.[15,16]

From an ISDT perspective, the Advanced Open Water has a function in training divers to handle rescues. The program, along with the initial open water training, creates a structured environment in which students develop experience (increased automatic processing) and schemata. A schema is cognitive framework the mind uses to organise interrelated concepts in a way that permits access to those concepts and their relationships. The mind uses these for semantic memory, problem solving, and both inductive and deductive reasoning. Specialised schema organise and activate motor skills.[11,12,13]

The "old school" of training divers in rescue neglected the development of experience and schemata, whereas the PADI system is particularly concerned with these. The minimum of nine dives, plus task-focused training, assures that students entering the rescue diver program have several well-established schemata. It is reasonable to expect these divers to have experience and

schemata relating to basic problem solving and self-rescue skills (which the Rescue Diver course builds upon), nuances of equipment configurations, environmental conditions, diver personalities and other topics that all affect handling a rescue. Among the many ways schemata and experiences relate to rescue training, two that stand out in particular are problem solving and the development of mental scripts and procedures for rescue.

**Problem solving**

Besides basic techniques for specific emergencies, such as mouth-to-pocket mask rescue breathing for an unresponsive, non-breathing victim, diver rescue is a problem-solving skill because there is no way to know in advance the specifics of the situation. Problem solving is a higher-level cognitive skill; good problem solvers in a given field have, among other attributes, well developed schemata that they access in solving a problem. This has been demonstrated by many studies.[10-12]

These show that better problem solvers do not necessarily have better problem solving abilities per se, but have better organised knowledge (schemata) about the problem subject. Those with better schemata and experience more readily identify problem components. They have previously encountered more sub-steps in the problem and may therefore deal with these sub-steps through automatic processes, freeing attentional resources to work on the novel aspects of the problem. Well developed schemata and experience are associated with greater problem solving efficiency and these problem solvers have greater accuracy in assessing their own progress as they attack the problem. In short, experience and schemata allow the solver to apply more mental resources to finding the solution.[11,12] The PADI system approach to rescue training accommodates the development of basic schemata and experience that student will apply at the Rescue Diver level.

**Mental scripts and procedures.**

Scripts are components of a schema that the mind uses to sequence action. These range from what to expect, such as what happens when visiting the dentist, to procedures to follow in a particular situation, such as, first you check in with the receptionist at the dentist's office, then you wait, etc. Scripts do not handle problems (i.e. situations never previously encountered), but help the problem solving process by letting the mind assign probable roles to elements in a given situation.[11,12]

Procedures are rules for doing something; complex procedures have variables that call for judgment and require combining simpler rules into the proper sequence.[27,28] Much of rescue training falls into these categories.

The "old school" of rescue training neglected the roles of scripts and higher-order rule development. The tendency was to teach rescue as a set of isolated procedures that were not well integrated. First aid and rescue breathing were treated separately.[17,18]

The problem with this approach is that the student may not be able to integrate these skills when faced, for example, with a non-breathing victim in the water who is bleeding profusely and who is negatively buoyant. What does the rescuer do first? Without training that integrates the skills, determining the basic steps is a problem solving situation that demands significant attentional resources.

The PADI system accomplishes rescue training through an approach that not only provides for the development of schemata, but that also integrates rescue skills with each other and Basic Life Support (BLS). The BLS approach itself integrates CPR and first aid according to the protocols followed by emergency medical personnel, but at the lay level.[7,19] This is one reason why CPR/first aid certification is required prior to certification as a PADI Rescue Diver.

It should be noted that the preferred CPR/first aid BLS course for the PADI Rescue Diver course is the Medic First Aid (MFA) course, which was developed by Emergency Medical Planning, Inc. MFA was the first lay person level course to integrate CPR and first aid along emergency medical technician (EMT) protocols.[20,21] With a field record of more than 15 years, the MFA system has proved itself effective not only in initial training, but in the willingness of students to render aid when needed. Surveys show that more than 85% of MFA trainees have offered to use their skills when faced with an actual emergency, which is a far greater proportion than trainees from conventional, non integrated CPR/first aid courses.[20] The philosophy of integrated training in the MFA program parallels, and integrates with, that in the PADI Rescue Diver course.

In the example of the negatively buoyant, non-breathing, bleeding victim, through the integrated approach, the rescuer knows that the priorities are to make the victim buoyant and begin artificial ventilation, handling the bleeding as soon as possible. This aspect of the rescue follows a developed script and higher order rules (procedures) that enact the component rescue skills largely under automatic control, leaving the rescuer's attentional resources free to address the unique aspects of the situation, such as distance to the boat, etc.

In an actual rescue situation, integration is important because it reduces the number of stimulus-response choices by automating decisions where appropriate. Hick's Law states that reaction time increases logarithmically with the number of stimulus-response choices. Therefore, reducing choices reduces reaction time.[13,22]

**Mass practice versus distributed practice**

In the development of motor skills, one area of conflict is whether learning benefits more from massed practice, with relatively short intervals between practicing, or from distributed practice, with longer intervals. What constitutes "short" or "long" depends on the skill in question, which has contributed to some of the debate as to which is better.[13]

Research in the 1930s, 1940s and 1950s suggested that distributed practice made for more efficient learning, but with more recent research that view is no longer valid. Based on current research, the prevailing view is that distributed practice is better for continuous motor skills, repetitious skills with arbitrary beginning and end points, but only marginally. Mass practice is better for discrete motor skills, those with clearly defined beginning and end points, especially with respect to short term retention. Students show about the same long term retention of discrete motor skills after mass practice or distributed practice; therefore, mass practice is preferred for the short term advantage.[13]

Dive and rescue skills include both continuous and discrete motor skills. Continuous motor skills include swimming with fins, scuba breathing and buoyancy control. Discrete motor skills include dropping a weight belt, removing equipment from the rescuer and victim, and approaching a panicked diver. Discrete motor skills that are performed in series are called serial motor skills.[13] These would include buddy breathing, alternate air source breathing, exiting the water, and in water artificial respiration (which has a continuous sub-skill, swimming with fins).

The PADI system accommodates the differences in preferred practice for continuous and discrete motor skills. Most of the continuous motor skills are basic diving skills. The novice learns these during the Open Water Diver course, which has at least five sessions and fits the description of distributed practice.[23] By the time the student reaches the Rescue Diver course, the continuous basic skills that are sub-skills to rescue skills have been practiced over at least nine dives, which is also distributed practice. Further, the continuous components are usually at the automatic process level by the time the diver reaches the Rescue Diver stage, so that learning them is no longer an issue because they have already been learnt.

At the Rescue Diver level in particular, students begin to develop most of the discrete motor skills related to rescue. The common format is a two day program with closely spaced, increasing-complexity, skill training and practice. This creates practice conditions that most conform with the preferred, mass practice conditions.

**Learning transfer**

ISDT is not just concerned with learning, but learning transfer, the student's use of the learned skill or knowledge in the actual situation for which it was intended. In rescue training, the question is whether the student, having performed rescue skills in class, will use the skills when faced with an actual rescue situation.

Transfer is encouraged by embedding the new skill(s) in a schema, and more importantly, by having a variety of practice.[11] Assuring that students have rescue-related schemata is well addressed by the PADI system. In addition, the PADI Rescue Diver course consists of multiple scenarios where students repeatedly apply their skills in varied situations with increasing complexity.[23] This contrasts with the "old school" approach, which not only presented skills in an isolated, unintegrated context, but which did not typically include variety of practice in the training.

Students who are practicing a complex procedure, or problem solving skill, without the prerequisite establishment of automated processes and schema will be thinking hard. This may be exacerbated by having to listen to and process instruction being provided. The result may be that the students solve the problem (complete the rescue), but have not actually learned much.[11] We are concerned that these students may believe they have mastered the rescue skills. Perhaps such a belief would dissuade students from continuing their diving training to include rescue, or they might put themselves in situations they would otherwise avoid due to their skill limitations.

**Emergency protocols**

Apart from applying ISDT methodologies, an issue is deciding what emergency medical protocols to include in a course. PADI's expertise in this area lies in instructional design, not in medicine. Therefore, the PADI system relies on those with relevant expertise for the specific protocols. Rescue-related training, such as BLS protocols, providing emergency oxygen, whether or not to incline a patient with decompression illness (DCI) head down, etc. draws upon the current protocol of the American Heart Association,[24] from the Divers Alert Network, the hyperbaric medical community including the South Pacific Undersea Medicine Society, Undersea and Hyperbaric Medical Society, European Underwater and Baromedical Society, and other expert sources internationally.

PADI Instructors follow these protocols, but adapt them to their local area to mesh with local emergency medical services (EMS) and local protocols.[7] The intent is two fold. First, to assure that students are trained in rescue protocols that include the steps needed to get the victim/patient into local emergency care, and second, to follow any

local laws that may apply. Therefore, rescue techniques taught under the PADI system follow established and accepted protocols, but with adaptations suited to the area.

Some rescue issues have no clear cut protocols. One example is how to handle a diver convulsing due to oxygen toxicity. In seeking advice, PADI was given two possible protocols. After assessing the protocols, the more widely recommended one, get the diver to the surface, was presented first. However, the second protocol, hold a breathing diver at depth until after the convulsion, was also presented.[25] When a single, clear protocol for this is presented by the hyperbaric medical community, then PADI materials will reflect that single protocol. Such a protocol appears to be evolving.

**The PADI approach to rescue training**

The PADI system applies ISDT, cognitive and educational psychology principles and other tenets of learning theory to train divers in rescue. Due to the need to develop automatic process in cognitive skills and motor skills, the need for experience and schemata, the benefits of appropriate practice intervals and the importance of assuring learning transfer, such training cannot be a single step. Rather, it is an ongoing process that begins at the novice PADI Open Water Diver level and continues through the PADI Rescue Diver level. Each training level has an important role.

PADI Open Water Diver. The student is self-oriented and most skills are consciously controlled. Students learn primary self-rescue skills and practice low complexity assists. Students begin to develop automacity in their motor skills and general diving schemata. Course materials establish basic concepts for a rescue-related schema, including problem recognition, basic assisting procedures, first aid for decompression illness and the basic steps for rescuing an unresponsive diver. Motor skills learned in this course directly related to rescue include:

entries and exits
swimming with fins
cramp removal
weight system removal at the surface and underwater
tired diver tow
buoyancy control at the surface and underwater
alternate air source use
free flow regulator breathing
controlled emergency swimming ascent
no-mask swimming
buddy breathing (optional)
scuba unit removal at the surface
basic compass use and navigation

PADI Advanced Open Water program. The student is less self-oriented; skills are increasingly under automatic control. Students gain experience in a variety of diving situations, broadening the diving schemata that they will use in rescue problem solving. By the end of the course, primary diving skills are generally automatically controlled, which frees attentional resources for new skill acquisition and problem solving at the next stage. Any additional specialty training, and/or non-training dives, contribute to the experience and schemata as well. Besides the crucial development schemata, the core deep dive includes more detail and review of first aid and treatment for DCI. Motor skills directly related to rescue include:

compass use, patterns and navigation
night navigation

PADI Rescue Diver course. The student is more externally-oriented and primary dive skills are under automatic control; the student has a basic rescue schema, as well as general diving experience and schemata to draw on as resources. Students learn the concepts and principles behind diving related first aid, stress and stress management, emergency management, emergency oxygen and how equipment can contribute to emergency situations. Other topics include the pathophysiology of lung expansion injuries and decompression sickness. Students practice rescue skills in a sequence that emphasises skill and concept integration, rising complexity, problem solving and variety of practice. These assure rescue skill mastery at the higher-order rule level, as well as learning transfer after the course. Motor skills learned include:

evaluating a victim's mental state (panicked or not)
approach, rescue and assistance of tired diver
tired diver tows
approach, rescue and assistance of panicked diver
underwater and surface contacts
non-swimming assists
exits with a responsive victim
post rescue attendance
search patterns and search for a missing diver
surfacing the unresponsive diver
in water rescue breathing
equipment removal of rescuer and victim while rescue breathing in water
exits with an unresponsive victim
first aid for DCI
responding to an accident from a boat or shore

After initial mastery in multiple practice sessions, these are practiced again in two fully integrated accident scenarios.

The PADI Divemaster course. The PADI Divemaster course is the first leadership level program and was not mentioned in previous discussions because it is beyond the

scope of this paper's topic. However, it is appropriate to note that as students enter this program, they enter a leadership orientation. Rescue training continues at this level, first to refresh skills and foster retention and second, to develop skills to the demonstration quality level. The skills acquired in the previous three levels continue to be refined with the goal of providing role model demonstrations for lower level students in training. Divemaster students also learn more about managing other divers (rescue and non-rescue scenarios), and the physics and physiology of diving, include rescue-related pathophysiology.[26]

Throughout these programs, the PADI system applies protocols derived from the emergency medical care and hyperbaric medical communities. The course adapts these protocols to the specifics of local protocols and emergency medical care.

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*The references have been rearranged to consolidate multiple references to one work so that the page numbers, separated by commas, are in the order that the references refer to them. This has reduced the number of references from 44 to 26.*


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*Drew Richardson is Vice-President, Training, Education and Memberships of PADI Worldwide and President, Diving Science and Technology, Inc. His address is PADI International, 1251 East Dyer Road #100, Santa Ana, California 92705-5605, USA. Phone + 1-714-540-7234. Fax + 1-714-540-2609. E-mail 748-3543@mcimail.com .*

*Karl Shreeves is Vice-President, Technical Development, Diving Science and Technology, Inc. His address is PADI International, 1251 East Dyer Road #100, Santa Ana, California 92705-5605, USA. Phone + 1-714-540-7234. Fax + 1-714-540-2609. E-mail 205-2396@mcimail.com*

# FIRST AID TEACHING FOR SPORTS DIVERS: WHAT AND WHY FROM SSI

Bill Day

**Key Words**
Accidents, first aid, oxygen, rescue, training.

## Introduction

SSI and PADI compete in exactly the same markets, we have the same customers, we just have different points of entry into that market. Basically I agree with what Drew Richardson, of PADI, has said. There is not a lot that I can add.

However there are a couple of points I want to make. But I will be brief because it is not worth repeating what Drew has done so well.

## What we should teach divers?

There are four areas of first aid for diving that we want our trainees to know, in terms of their final development as a diver.

First, our divers should know general first aid. That is the ABC (Airway, Breathing, Circulation) of first aid, how to stop the blood leaking out of them and how to cope with the things that they will die of immediately. The stuff of a standard first aid course.

Secondly, we would like our divers to be able to rescue other divers.

Third comes oxygen administration and oxygen therapy. Divers need an understanding of the reasons for oxygen administration and the ability (motor skills) to do it.

Fourthly divers need more in depth understanding of the pathophysiology of decompression illness.

There are the four aims. How do we deliver them? The answer is “Not very well at all”.

## What we actually do

We out-source first aid. SSI does not do that training in-house. PADI has moved, over the years, to teaching the medic first aid course in house, which is a really good and positive move. SSI has in fact been throwing away some of their market by farming first aid out. SSI have just started to take a first aid course on board and keep it within their doors, but it is only developing at the moment (April 1997).

We have put rescue abilities, the second skill, in very much the same place as PADI, as a later course. It is what we call our Stress Rescue course. So our customers come in and do the Open Water course where they learn to dive. Typically they do about five dives in that course. Then we have some further training courses for them to gain experience, which they do between learning to dive and the Stress Rescue course.

To date we have been weak on oxygen administration . We aim to pick that up through a DAN course, that is what we are starting on, and in some ways through our Med Dive program. In terms of the oxygen administration part I think the DAN course is far superior to the Med Dive program. We use the Med Dive program to give our instructors or our senior divers, and often just people who have an interest in it, a much greater understanding of decompression illness and how it happens. When divers have that understanding they realise that there are many changes that they can make in the way they dive. Our aim is to give them an understanding of the problems of repetitive diving; an understanding of why it is much better to have a winch on the front of the boat rather than the diver pulling the anchor up by hand at the end of a stressed dive. There are many things they can learn from that course which actually affects them from day to day, and we think that is important.

That is the overview.

## Why not teach rescue at the Open Water level ?

The answer is, “It’s too bloody difficult”.

When people come for Open Water training, it is a big training challenge. Our clients are thoroughly trained to hold their breath when their heads are under wate which has kept them out of trouble all their lives, right up to the moment they come to us. It has been a good skill for them. We have to take that and turn it round to become “Never stop breathing underwater”. This must happen not only when they are sitting quietly and thinking about it, but even when everything has gone wrong and panic threatens. We have to change it from an intellectual skill to something that is integrated at a much deeper level. Of course we have to teach them all the other things as well. It is a difficult task. Dive training is not easy. We know that people learn better when they are relaxed and confident, but we are throwing them into a totally alien environment. It is an inherently hostile environment, for one cannot breathe under water without equipment, and most trainees are scared. At some stage or other, when you all learnt to dive, you were scared. At some point we had fear. Last year I learnt to fly aeroplanes. And suddenly, after 20 something years of diving, I sat back and said to the instructor, “Hey, I’m scared again. This is different.” Learning to dive is a difficult process.

The problem is that not everyone is a good learner. In the medical field it is a bit different. All of medicos are very good learners. They are in the top academic stream and are very good at learning things.

However I must tell you, as someone who has been out in the field teaching diving for a few years, the lowest common denominator is actually pretty low. Many people learn much slower than doctors do, and it is more of a challenge for us to teach them. We cannot actually teach people, within any reasonable time frame, the whole range of skills. That is why they are split up into different courses. As Drew said, we used to try it in the past, and the way we did it was we cheated. We would take them and teach them to dive, but then we would not let them get away! But there was a difficulty. If we wanted to teach them other things, we had to keep them on the course until they were relaxed enough. So what did we do with them? We just dreamed up things. We taught them to take their gear off and throw it onto the bottom of the sea and dive down and pick it up again. We did not care what they did, they just had to do things in the water. Underwater chess and writing the great New Zealand novel, on waterproof paper, would have been fine! The worst part is that we believed that what we taught them in those days was useful! But that is a secret!

What we did learn from the courses that ran over weeks and weeks was that it not work very well, because when we looked at those people later, they were not all that well trained. Even worse we cannot market the damn thing. We are in the real world and we just cannot sell it any more.

**What is the result?**

I do not think the result is very good at all, because there are two gaps. I believe every training agency faces the first and most important gap. Just like PADI, SSI has got a lovely range of courses which divers should go through to pick up their skills. We start off with the Open Water course, then there is some Advanced Training, and then on to the Stress Rescue course. Later on one does the DAN Medical Course.

But the point is, people don't do them! The actual retention into those other courses is extremely low. So the reality is, that despite all this fine rhetoric, what is happening is that the vast majority of trainees do the Open Water course and then go out into the world to dive. They do not have any of the skills taught in these subsequent courses, they do not think about getting them and they are never likely to. Most divers out there do not have first aid skills, they do not have rescue skills, they do not have an in depth understanding of decompression illness and they will never have them.

I think there is a gap, and I do not think it has been well served by any training agency. But I do not know how to fill that gap either. We have tried to bring some of those skills in house, rather than out source them, and as a consequence we are perhaps able to channel people into those other courses. This does two things. One, it is good because they learn at the other courses and two, we are in the business of teaching, so it makes common sense.

The other gap, again from a philosophic standpoint, is that when an instructor is teaching, it is important that he or she has a level of knowledge about the subject that is a degree greater, and in my view a considerable degree greater, than the actual material that they are teaching. That gap is only just being sorted out. For some years now, instructors have learnt how to teach the course by doing it and their level of knowledge is just the same. They are like the Monty Python parrot. They cannot go further.

So I think the biggest challenge that we face is that divers are still going out without any substantial rescue or oxygen administration or first aid skills, despite all the things we have put in place. It is just not happening.

*Bill Day is a director of Seaworks Ltd, a New Zealand based multi-national shipping company. He is an Instructor Evaluator for SSI New Zealand and has been active in diver education for the past 20 years. His address is 162 Owhiro Bay Parade, Owhiro Bay, Wellington 2, New Zealand. Phone +64-(0)4-383-7021. Fax +64-(0)4-383-7621. E-mail billdayatseaworks@xtra.co.nz .*

## DAN S.E. ASIA-PACIFIC OXYGEN PROVIDER PROGRAMS

John Lippmann

**Key Words**
Accidents, equipment, first aid, oxygen, training.

**DAN SEAP structure and aims**

The Divers Alert Network (DAN) is a not-for-profit organisation which was formed in 1981 in the United States to assist in the treatment of scuba diving accidents by providing a 24-hour emergency hotline.

To help fund its hotline, DAN established a membership program, initially providing subscribers with a diving accident manual and a regular newsletter, *Alert Diver,* dealing with various issues of diving safety.

As DAN grew, it expanded its operations to provide a free evacuation service for members and diving injury

treatment insurance as well as a variety of other services, including a highly effective and successful oxygen first aid training program designed specifically for divers but also applicable to general first aid. Another important function of the Network is collecting and analysing data on diving accidents and fatalities to improve the understanding of the causes of accidents, improve preventative measures and to develop better treatment methods.

DAN organisations have now been established in several countries. DAN Europe and DAN Japan have been operational for some years and provide similar services to DAN America. DAN S.E. Asia-Pacific (SEAP) was established in 1994, and provides services to countries in the S.E. Asian and Pacific Region. DAN Southern Africa is to commence operations later in 1997.

DAN SEAP is incorporated as a non-profit diving safety association based in Melbourne, Australia. Its aims include:

1 improving diver safety through education,
2 improving the availability of oxygen at dive sites,
3 providing evacuation and insurance services for injured divers,
4 supporting regional diving emergency hotlines,
5 supporting diving safety research, and
6 accident data collection and presentation.

DAN SEAP provides membership services to divers in Australia, New Zealand, S-E. Asia and some Western Pacific Islands. Membership includes:

1 subscription to *Alert Diver*,
2 a copy of *The DAN SEAP Diving First Aid Manual*,
3 cover for the costs of evacuation to a treatment centre,
4 access to dive injury treatment insurance,
5 access to and discounts on DAN products including oxygen equipment.

As a non-profit company DAN SEAP is subject to an independent annual financial audit. Excess revenue is directed towards improving diving safety within the Region. For example, DAN SEAP provides full funding for the DES Australia hotline and has begun to provide some support to other regional hotlines.

DAN SEAP has recently established a National Office, known as DAN SEAP-Philippines, at the Makati Hospital, Manila. It appears that a New Zealand National Office may be established at the RNZN Hospital in 1997. It is planning to establish National Offices in various other countries within the Region to better serve members in those countries.

Current membership is approximately 3,500 and, after a relatively slow start, DAN SEAP is beginning to enjoy a more rapid growth rate.

**Oxygen equipment for divers**

An oxygen demand valve is usually the simplest system to use with a breathing casualty. A pocket-style mask with oxygen inlet is the easiest method for ventilating a non-breathing casualty. *These techniques require far less initial training and continued practice than other methods and can be done effectively by one trained person.*

Bag-valve-mask devices (BVMs), manually triggered ventilators (MTVs) and closed circuit oxygen resuscitators (CCRs) all require considerably more initial training and continued practice to achieve and maintain proficiency. In addition, it often requires two trained operators to effectively ventilate a non-breathing casualty with these devices in the field.[1,2] So, even though such devices are capable of delivering higher oxygen concentrations for resuscitation (an inspired oxygen percentage of up to 100% with MTVs compared with approximately 50% with a pocket-style mask with supplemental oxygen inflow of 14 l/min), in practice they may often be less effective because of the greater skill and manpower required.

**DAN Oxygen Units**

Although there is a variety of oxygen systems available, many of the units are unsuitable for use by the general diving community. Some units require too much on-going training and practice, others don't provide sufficiently high inspired oxygen concentrations. To remedy this situation, DAN America assembled an oxygen unit, known as the DAN Oxygen Unit, designed specifically to cater to divers' requirements. Various configurations and modifications to this original unit are now available through DAN SEAP.

A DAN Oxygen Unit is not only effective, but also easy to use and so requires minimal training. It consists of a multi-function regulator with at least one high flow outlet; a variable flow outlet providing flow rates up to 25 l/min; a demand valve, hose and tight-sealing mask; a Pocket Mask with oxygen inlet; a non-rebreather mask and a waterproof case.

Adaptors are available to enable a pin-index regulator to be fitted to oxygen cylinders with various types of threaded fittings. These oxygen units are easy to assemble, easy to use, effective and durable.

**DAN oxygen programs**

In the increasingly litigious environment in which we live, there is now more pressure than ever for diving instructors and dive charter operators to ensure that *appropriate* oxygen equipment and properly trained and

qualified oxygen providers are available where diving is conducted. In fact, all divers are strongly advised to undergo training in resuscitation and oxygen provision. The skills developed are extremely valuable, not only in the diving environment, but in all walks of life.

Oxygen provider training is available through several first aid training agencies. However, most of these courses are not diver-oriented and many have not been reviewed and aligned to current experience, recommendations and protocols. Some years ago, DAN America developed a 4-hour Oxygen Provider program to provide divers with appropriate training to use a DAN Oxygen Unit or similar equipment.

The DAN Oxygen Provider Course has become the fastest growing and generally the most highly regarded oxygen program for divers, worldwide. DAN America had itself certified 9,419 Oxygen Providers to 1995.[4] This has lead to an increase in the provision of oxygen as first aid in diving accidents. In 1995, oxygen was provided in 32% (190/354) of the diving accidents reported to DAN America.[4]

**DAN SEAP Oxygen Provider Course**

DAN SEAP has adapted the DAN America program to better conform to the Australian Resuscitation Council (ARC) protocols, to suit the needs of this Region. First aid protocols vary from place to place, as does equipment and standards of training. DAN SEAP has worked hard to address these issues and has made several modifications to the DAN America program.

The DAN SEAP program is suitable for divers of all levels, from novice to instructor. It consists of a 4-hour Oxygen Provider Module, supported by an additional Resuscitation Module, where required, and several Extension Modules for divers who require further training.

Most injured divers are breathing and require supplemental oxygen.[4,5] The Oxygen Provider Module is designed to teach the skills needed to perform this task safely and effectively. Topics covered include:

- diving accident recognition
- the benefits of oxygen provision
- when to provide oxygen
- review of various types of oxygen equipment
- precautions and safety procedures
- the DAN Oxygen Unit
- oxygen provision to both conscious and unconscious casualties
- oxygen supplemented expired air resuscitation
- dealing with a diving accident
- assessment

Participants are certified in the use of the DAN Oxygen Unit, or an equivalent system. Certification is valid for one year in Australia and New Zealand and two years elsewhere.

The Resuscitation Module is also a 4-hour program. Topics include:

- recognition and management of the unconscious casualty
- expired air resuscitation
- cardiopulmonary resuscitation
- modifications for infants and children
- complications
- cross infection
- assessment

Given that the vast majority of diving accident victims are breathing and will benefit from oxygen provision and that only a small percentage require resuscitation (possibly 8 to 9%)[4,5] there is a strong argument for encouraging divers to learn oxygen provision before resuscitation. However, in Australia, the Resuscitation Module (or equivalent resuscitation training) is a pre-requisite for the Oxygen Provider Module to remain consistent with the ARC oxygen training protocols. In certain other countries the Oxygen Provider Module can be taught before resuscitation training.

**DAN Oxygen Instructor Training**

DAN SEAP also provides instructor training to people, who have done the Oxygen Provider course, who wish to teach the DAN Oxygen Provider Program. DAN SEAP Instructors, who are generally qualified scuba diving instructors (or equivalent), have all participated in an extensive training program, usually under the close scrutiny of an experienced diving medical specialist.

DAN America or DAN Europe trained Oxygen Instructors have participated in a one-day Oxygen Instructor Training program in addition to their initial Oxygen Course. However, before June 1996, DAN Oxygen Instructors trained in Australia and New Zealand (under DAN SEAP) had participated in a two-day Instructor Program. One part of the DAN SEAP program consisted of physiology, resuscitation and diving medicine and the other concentrated on oxygen equipment and its administration. These programs have been very successful and participants have generally found them challenging and enlightening. However, it has often been difficult to schedule the programs to suit the requirement to have both an Instructor-Trainer and an approved Medical Specialist available. In addition, it has been difficult to accommodate Instructors who have obtained DAN qualifications in one region and then travel to work in another.

DAN SEAP has now rearranged the components of

the two-day course into two separate one-day modules:

*Instructor Module I*

This includes all of the components necessary to enable the successful participant to teach the 4-hour DAN Oxygen Provider Module. It is compatible with the DAN America and DAN Europe Oxygen Instructor Courses and is presented by a DAN SEAP Instructor-Trainer.

Topics covered include DAN Mission and overview; revision of physiology and anatomy; management of the unconscious casualty; EAR; recognition of a diving injury; benefits of oxygen provision; handling and storage of oxygen; oxygen delivery systems; teaching the use of the DAN Oxygen Unit; cross infection and cleaning; legal issues; and course preparation, standards and procedures.

A person who successfully completes Instructor Module 1 becomes a Level 1 DAN Oxygen Instructor. As previously mentioned, a Level 1 Instructor can conduct the DAN Oxygen Provider Module but cannot teach the DAN Resuscitation Module.

*Instructor Module 2 - Resuscitation and Diving Medicine.*

This module, which is presented by both a DAN SEAP Instructor-Trainer and a Diving Medical Specialist (often with anaesthetics experience), includes advanced physiology, resuscitation theory and practice, injury recognition and management for diving and certain non-diving illnesses, hands-on practice with a variety of oxygen delivery systems and accident management scenarios.

A person who successfully completes both Instructor Modules becomes a Level 2 DAN Oxygen Instructor. A Level 2 Instructor is sanctioned to teach both the Oxygen Provider and Resuscitation Modules to the diving community, and beyond. In other words, they are a DAN Oxygen Instructor as well as a DAN CPR Instructor.

A Level 1 Instructor can upgrade to become a Level 2 Instructor by attending an Instructor Module 2.

DAN SEAP is also conducts DAN Oxygen Instructor-Trainer Programs to prepare people to train DAN Oxygen Instructors.

Although DAN SEAP believes that DAN-style units fulfil the needs of most dive professionals, it has developed Extension Modules to train people in the use of more advanced oxygen equipment for those who have a specific need or desire for such training. Extension Modules include MTV- 100, Bag-Valve-Mask and Closed Circuit Oxygen Resuscitator training.

By attending an appropriate extension program, which vary from 4-6 hours in length (in addition to the initial provider training), Oxygen Providers can, if desired, gain an endorsement to use such devices. The programs concur with recent Australian Resuscitation Council guidelines for such training.[6]

To date, DAN SEAP has certified around 1,700 Oxygen Providers, 320 Oxygen Instructors and 30 Instructor-Trainers (15 trained under DAN SEAP, 15 US trained) within this region.

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**Update**

The above was current at the time of presentation at the SPUMS Annual Scientific Meeting in Waitangi, New Zealand (April 1997).

At the end of April 1998 DAN SEAP membership had increased to approximately 4,200 and DAN SEAP has trained just over 500 oxygen instructors and almost 3,000 oxygen providers. In addition the DAN SEAP New Zealand office has been established at the Royal New Zealand Navy Hospital under the directorship of Dr Simon Mitchell.

*John Lippmann is Executive Director of DAN S.E.Asia-Pacific. For his contributions to diving safety he was elected a member of SPUMS in 1996.*

*The address of DAN S.E.Asia-Pacific is PO Box 134, Carnegie, Victoria 3163, Australia. Phone +61-(0)3-9563-1151. Fax +61-(0)3-9563-1139.*
*E-mail danseap@danseap.com.au .*

# THE INADEQUACY OF CURRENT FIRST AID CARE

James Francis

**Key Words**
Accidents, first aid, oxygen.

## Introduction

From what we have heard about the Australian, New Zealand, UK and USA situations it is clear that the current provision of first aid to divers is inadequate.[1-4] Nowhere do all injured divers get given oxygen first aid. Nowhere does even the majority of injured divers get oxygen. The figures for fluid administration are even worse.

## Why is diving first aid inadequate?

I can only speak about the UK, because I know a bit about what goes on there. I do not know much about what goes on anywhere else.

I think the basic reason is ignorance of what to do in a number of groups, in particular divers themselves and the people who provide diving facilities, such as dive boats. In the UK there has been ignorance of diving first aid in the Coast Guard, which is one of the principal emergency retrieval systems that we have, and in the Royal National Lifeboat Institution, which backs them up. I am not suggesting that these organizations are ignorant about first aid, merely about the specific requirements of divers, notably the provision of oxygen and fluid resuscitation. In 1991,when we first started collecting data in a formalised manner, we were dealing with single figure percentages. One reason could have been that these data were incompletely recorded, however I am quite convinced that they reflect a minimal provision of diving-specific first aid.

Between 1991 and 1995 the situation improved, and the UK struggled into the double figures. Now (April 1997) the provision of oxygen first aid is around 25% or so, but the provision of fluids is less.

Diving medical officers, and particularly those associated with the Royal Navy, have been bleating for years that oxygen treatment has been inadequate. But our bleats were muted and we were bleating in the wrong place. To a certain extent we doctors can blame ourselves.

## How can the situation be improved?

What change that has occurred in the UK has been because the divers themselves have come to realise that more adequate diving first aid treatment is required and they have turned out to be the necessary pressure group to get things to change. In the UK, the British Sub-Aqua Club, which is the principal training authority there, has hoisted this in and they now provide first aid courses very much along the lines of what we have been hearing this afternoon from DAN, PADI and SSI.

Initially these courses, particularly the oxygen delivery course, were provided as an add-on to exiaring training. It was one of the things that one did during a weekend's diving with the BSAC. Now it is going to be introduced into their training qualifications for the sport diver. This is bound to have a snowball effect. If divers are trained in the need for and the provision of oxygen first aid they will expect it to be available at the dive site and we will enter a virtuous circle. So we should soon have the situation where oxygen delivery skills should be quite widespread amongst UK divers.

Another problem, which has inhibited the provision of oxygen, is that the equipment was not always available. Staggeringly, it was not available in the Coast Guard until fairly recently, unless the Coast Guard chopper happened to be a military one, in which case there was almost always not just the equipment available but someone who was trained to deliver it.

Again, until recently, most dive boats did not carry oxygen, and even if they did, there was no gaurantee that there would be someone trained to deliver it. Thankfully this situation has turned around. Divers in the UK have a growing reluctance to go out in dive boats that do not carry oxygen. Quite right too! If they can put pressure on the market to get itself up to speed, good on them and I encourage their efforts.

I am quite convinced that if the lack of provision of oxygen first aid to divers is to be improved, it is divers themselves who need to exert the necessary pressure on their training organisations and on those who provide diving facilities. If organisations like DAN can do that, good for them.

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*Dr T J R Francis, MSc, PhD, Dip DHM, one of the Guest Speakers at the 1997 Annual Scientific Meeting, was Head of Undersea Medicine at the Institute of Naval Medicine, Alverstoke, Gosport, Hampshire PO12 2DL, England. His address is now Naval Submarine Medical Research Laboratory, Naval Submarine Base, New London, Groton, Connecticut 06349-5900, USA. Telephone +1-860-694-4005. Fax +1-860-449-2523. E-mail francis@nsmrl.navy.mil .*

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# HOW DIVER'S DIE

Rees Jones

**Key Words**

Accidents, case reports, cerebral arterial gas embolism, deaths, drowning, injuries

## Introduction

Several years ago the Hillary Commission for Sport and Recreation surveyed the New Zealand population about their sporting and recreation pursuits. They produced meaningful statistics of population involved in activities from archery to shooting and included scuba diving.

The Accident Compensation Insurance Corporation (ACC) provides in New Zealand a no-fault accident insurance for fatal and non-fatal accidents in every sphere of activity. Statistics for 1989-1990 show that claims for compensation scuba diving accidents under New Zealand's Accident Compensation Scheme are relatively low both in total numbers and in comparison to other outdoor sports and leisure activities. It is unlikely that any dive accidents in this period requiring medical treatment or involving death would not have been recorded. Payments for other water sport fatalities including boating, swimming (presumably non-competitive athletic events) and fishing are very high.

Figures from Project Stickybeak and the NZ Underwater Accident recorder show a spread of causes of Australasian diving accidents not dissimilar to other figures such as DAN (Divers Alert Network) USA, except for higher representation of asthma in Australasia.

The Northland environment favours year round diving by local residents, overseas tourists and most recent graduates of Auckland dive schools. The underwater attractions also bring many divers from elsewhere in New Zealand. With the large amount of diving in Northland it also means that this is where many dive accidents occur.

Non-fatal accidents are usually transferred rapidly to the Slark Hyperbaric Unit facilities at the Royal New Zealand Navy (RNZN) Hospital, Devonport by Northland Emergency Helicopter. Fatal accident victims are usually brought to Whangarei for autopsy if the body is recovered.

It is important to establish the cause(s) of death in any fatal accident, and especially in diving accidents, as the death has occurred in a hostile environment. Associated factors include level of training and experience, with medical, psychological and equipment factors. Equipment, if available, is usually examined by the Navy or police divers. Assessments of the other factors, for the coroner, are obtained by police interviews of witnesses (the results are of varying adequacy). The pathologist's role is to establish the mechanism and mode of death. The more information, especially history and equipment examination, one has the more likely is a firm diagnosis.

## Autopsy

Empirical and eyewitness evidence indicates that pulmonary barotrauma leading to arterial gas (air) embolism is a likely cause of loss of consciousness in divers. Recent dive profile examination, if available, may suggest possible causes of death but in recent years protocols for autopsies on divers have suggested that efforts be directed to detecting air in the vascular system.

These range from performing the post mortem under water e.g. in an hydraulic bath, or opening the chest cavity through a water seal maintained by the reflected skin flap and using X-rays, both conventional and computerised axial tomography (CAT). In the absence of X-rays pneumothorax has been demonstrated by direct aspiration of the chest before any skin incision.

Demonstration of gas within the cranial cavity before any skin incision or removal of the cranium, prior to the introduction of CAT scans has been especially difficult. These were first used in Australia by Ansford et al in 1990 for investigation of a naval diving accident and later investigating scallop diving fatalities in Tasmania. Three recent scuba diving fatalities are presented to show the usefulness of computerised axial tomography (CAT) scanning to increase gas embolus detection within the vascular system before the body is opened. Northland Base Hospital (now Whangarei Area Hospital) obtained a CAT scanner in 1992.

Before the availability of CAT scans locally, lateral and AP standard X-ray views of skull and thorax were done. These were occasionally of considerable interest, two examples being one with gas outlining Circle of Willis, another with gas in a coronary artery. Both also showing gas throughout the thoracic great vessels.

The factors leading up to the deaths are considered with the significance of the gas detection in relation to the mode of death. No gas analyses were undertaken. This would be essential in the future to distinguish between nitrogen, air and decompositional gases. In these cases the latter is cause is thought unlikely because of the short time interval between death and refrigeration and post mortem examination.

## Case reports

Three recent fatalities, each illustrating different problems, which have occurred in widely separated areas of Northland are presented as examples of the usefulness

(or otherwise) of CT (computerised tomography) in the diagnosis of cerebral arterial gas embolism (AGE).

This has been available for use in these diving related deaths. In one the body was retrieved from 30 m after immersion for 3-4 hours. The other two patients experienced difficulties at 7 and 10 m, ascended to the surface and subsequently died after unsuccessful resuscitation attempts, in one case by an inexperienced lay person, and the other after attempted resuscitation by a general practitioner and then the Northland Emergency Rescue Team.

***Case 1***

A dive charter boat at Three Kings received a radio message from the 16 year old deckhand of a cray fishing boat close by saying that his skipper had gone diving an hour earlier, to retrieve a trapped cray pot, and had not returned. The cray fishing skipper (male), aged 28, was an inexperienced diver. The charter boat skipper came alongside and commenced a search, finding and retrieving the body quickly from a reef surface at 30 m. It was estimated it had been submerged for approximately 2 hours.

The weight belt was on but not the mask. When examined about 24 hours after retrieval the body was unremarkable except that the appearance of his eyelids was unusual. He had gross haemorrhage into the eyelids with splitting of the skin, and conjunctival haemorrhage and oedema (Figure 1).

CT scans of the head (Figure 2) and trunk were obtained, which showed the vascular system full of gas. The overall significance of this was uncertain, because of the time underwater and the time since retrieval. It is highly likely that most of this gas was nitrogen which had off gassed from body tissues since the body was brought to the surface. The superficial cerebral blood vessels were full of bubbles (Figure 3 page 115). The lungs show gross oedema consistent with drowning and areas of haemorrhage which suggest probable pulmonary barotrauma leading to AGE.

WHY DID HE DIE?

This diver was totally untrained and this was his first dive! He carried scuba gear on his boat as safety equipment! I was told by the Police that his tank still contained air and his regulator was satisfactory.

My hypothesis is that he descended without equalising the pressure in his mask, producing a gross mask squeeze. This resulted in haemorrhage into his lids and tearing of skin with bleeding into his mask. In pain from the swelling and having suddenly gone blind, he panicked, removed his mask and drowned.

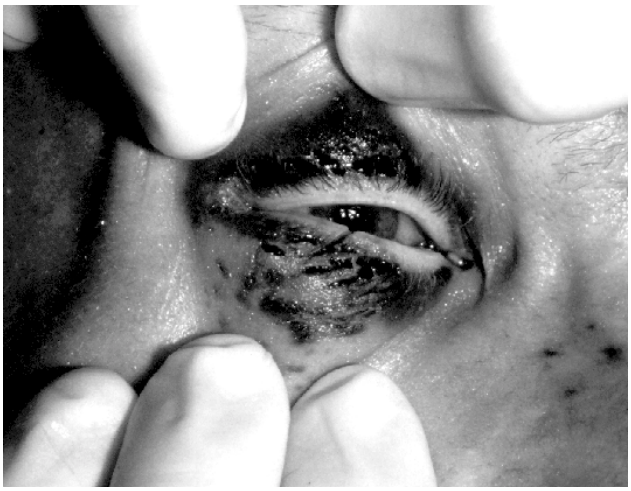

**Figure 1.** Case 1. Haemorrhage into lids with split skin of upper lid.

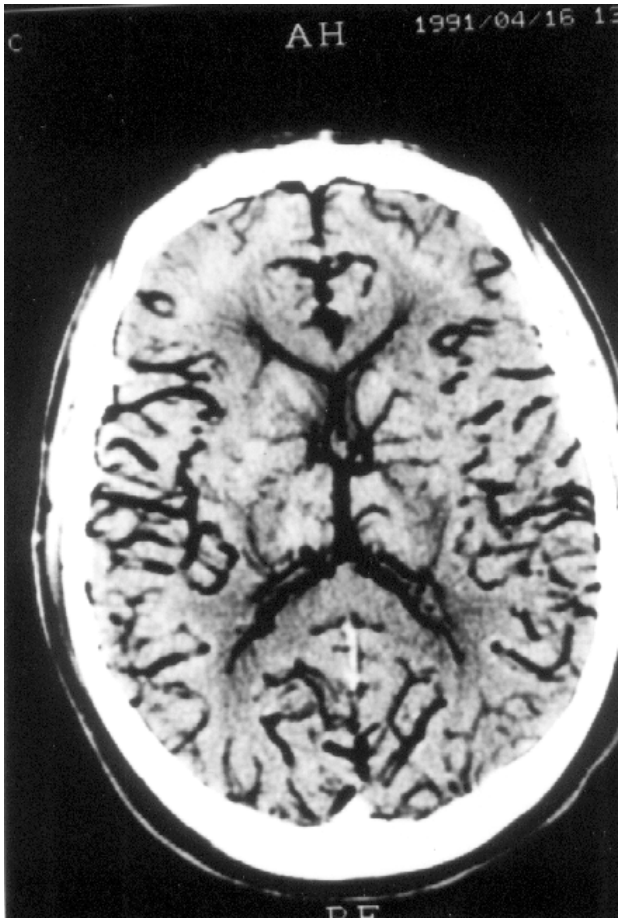


**Figure 2.** Case 1. CAT scan head. Large amounts of gas (black) in all intracranial vessels.

***Case 2***

A couple were diving off Rauol Island in the Kermadec Group 400 miles North East of New Zealand.

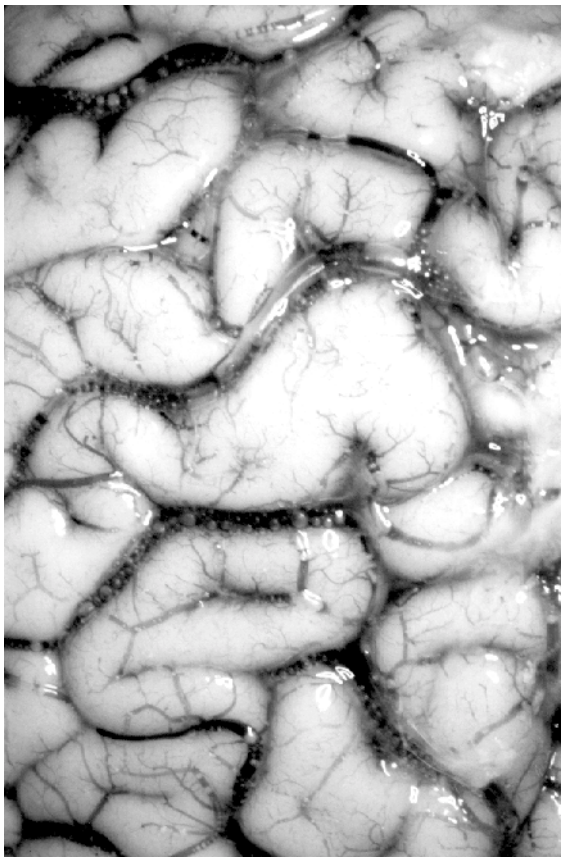

**Figure 3.** Case 1. Cylindrical and spherical bubbles in cerebral vessels.

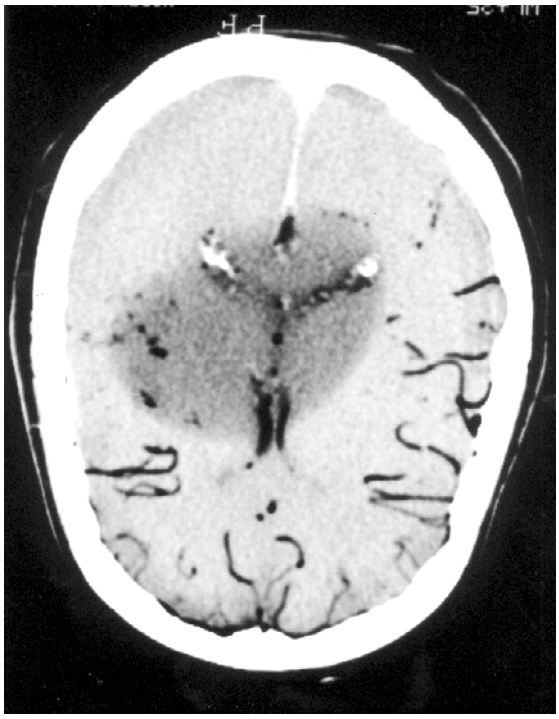

**Figure 4.** Case 2. CAT scan of frozen head. Gas (black) within intracranial vessels. Compare with Case 1.

The wife, aged 49, was an inexperienced, untrained diver. At 10 m she signalled to her husband that she wished to surface. They both ascended. On the surface she told him that she felt uneasy and wished to stop the dive. She also felt too weak to swim against the slight current to the boat. Her husband inflated her buoyancy compensator, then swam to their yacht's dinghy and returned with it to her, by which time she was unconscious. A dive charter boat (with the same skipper as in Case 1) came around the corner of the island at this time. The victim was taken aboard and resuscitation attempts started. These included intracardiac adrenalin (advised by medical personnel using radio). This was unsuccessful. Her body was taken ashore and on Police instructions placed in the walk-in deep freeze at the Department of Conservation Lodge.

As death occurred in "suspicious" circumstances the police arranged for retrieval by Emergency Rescue Helicopter, which refuelled during its return journey from a RNZN fuel dump on Esperance Rock. This involved hovering with only 1 skid on the rock!

No external injuries were evident at post mortem. A CT scan showed much gas in the cerebral circulation and great vessels (Figure 4). There was nothing obvious in coronary arteries, but there was air in ventricles. The lungs showed haemorrhagic areas suggestive of pulmonary barotrauma as a source of AGE. The post mortem was technically difficult due to frozen organs.

WHY DID SHE DIE?

The likely sequence of events was an inexperienced untrained diver in a remote environment ascending, in an emotionally upset state, while breath holding. The resulting pulmonary barotrauma led to embolism with loss of consciousness and respiratory and cardiac arrest on the surface.

***Case 3***

A 34 year old female, with five years diving experience, was on a scallop dive from a launch in Rawhiti Channel, Bay of Islands in 8-9 m. She was diving with her father after lunch on New Year's Eve. He noticed, ten minutes into the dive, that she was actively ascending but continued his dive.

Her non-diving husband was on their boat and saw her surface about 50 m away. She waved, rolled onto her back and swam to the boat. He helped her aboard where she immediately collapsed and lost consciousness. He radioed for help and went ashore at Urapukapuka Island where a tourist helicopter arrived bringing a Paihia general practitioner who continued resuscitation attempts.

The Northland Emergency Rescue helicopter team arrived some time later with an anaesthetist and assisted, but resuscitation attempts were declared unsuccessful after about 2 hours. The body was transferred to Northland Base Hospital for a Coroner's post mortem. CAT was undertaken on skull and thorax. The skull views showed a single segmental bubble obstructing the right internal carotid artery at the siphon (Figure 5). The opposite side was clear. Lung fields showed areas of pulmonary infiltration. There was gas in the aorta, vascular tree and the left ventricle and hepatic artery (Figure 6).

The tracheobronchial tree contained a considerable quantity of poorly digested food similar to that in her stomach down to the peripheral bronchi. The gastric mucosa was reddened, consistent with an acute gastritis.

WHY DID SHE DIE?

The likely sequence of events was that, following a salad lunch, for some reason, probably gastritis, she felt like vomiting soon after submerging. Not being trained in the techniques of vomiting underwater she ascended to the surface while holding her breath and trying not to vomit. She developed pulmonary barotrauma, embolised during the ascent, vomited at the surface and aspirated. This eliminated any chances of survival by preventing adequate ventilation during resuscitation.

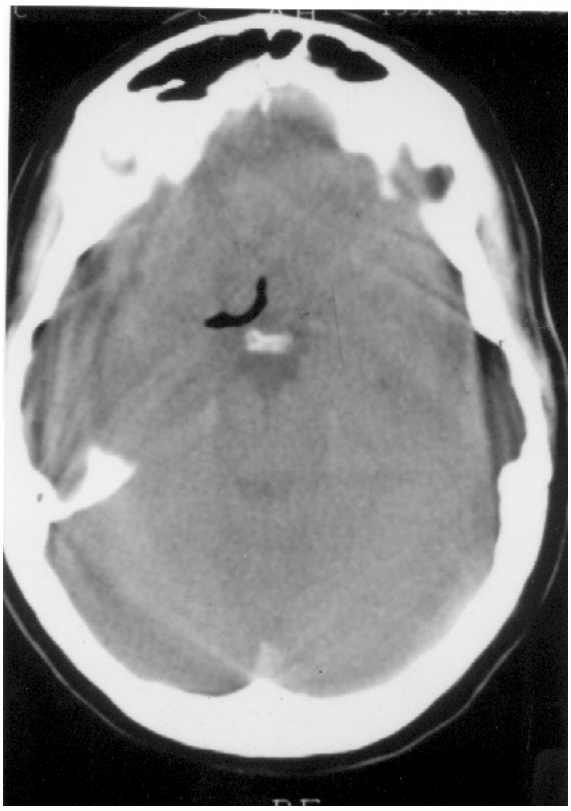

**Figure 5.** Case 3. CAT scan head. Single segmented bubble of gas (black) within right internal carotid artery. Compare with Cases 1 and 2.

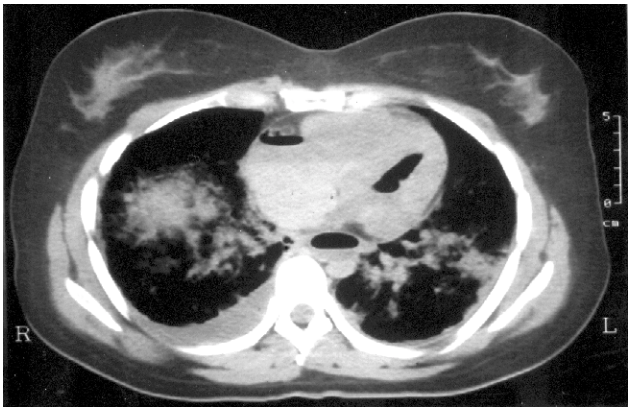


**Figure 6.** Case 3. CAT scan thorax. Gas (black) within aorta, left and right ventricles. Pulmonary infiltrates secondary to aspiration of stomach contents.

**Discussion**

I have endeavoured to show that diving deaths can result in a number of injuries which one would not necessarily expect, that CT scans are a good way of examining the body before starting the post mortem but can show air in unexpected places which makes diagnosis of the original injury less certain as off-gassing has to be considered.

In Case 1 drowning was the probable mechanism of death, contributed to by mask squeeze, panic and perhaps gas embolism. The other two cases undoubtedly died on the surface so drowning can be confidently eliminated. Case 3 involved aspiration of stomach contents which undoubtedly interfered with ventilation. Regrettably no gas analysis of samples from their vascular system bubbles were undertaken in any instance.

These three cases show that intravascular gas can be readily demonstrated and localised by Computerised Axial Tomography.

Does delay in retrieval of a body (Case 1) allow continuing uptake of nitrogen into tissues of lung and blood? If this is so, retrieval from depth with release of pressure allows production of gas from blood in the lung vessels, pushing gas into pulmonary arteries and pulmonary veins, and so into the chambers of heart, and perhaps more peripheral spread.

The sources of intravascular gas in these cases can only be guessed at.[1] Is it as a result of vigorous resuscitation? If intravascular gas is introduced during

resuscitation, what is the mechanism? Should resuscitation procedures be changed to avoid or prevent this? Should resuscitation attempts be advised against because of the risk of initiation or worsening of air embolism?

Analysis of gas samples from great vessels and ventricles should be able to confirm the origin of gas. Air would be from pulmonary barotrauma. Gas with a high nitrogen content would have evolved from dissolved gas in tissues. Various complex mixtures, involving $CO_2$, hydrogen sulphide and short chain fatty acids, develop from putrefactive changes.

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*Dr Rees Jones is a pathologist based in Whangarei. His address is 13 Moody's Lane, Whangarei, New Zealand. Phone + 64-(0)9-437-0608. Fax + 64-(0)9-437-2469. E-mail jonesrhj@igrin.co.nz .*

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# ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

## BUBBLES

**Bubble formation: amount of dissolved gas vs number of micronuclei.**

Raychaudhuri S and Van Liew HD. *Undersea Hyperbaric Med* 1997; 24 (Suppl): 25

**Abstract**

Background

Decompression tables for safe return off human divers to the surface without many bubbles in tissue or blood (decompression sickness, DCS) grew around the idea that supersaturation of tissues was the only issue. As an alternative to that view, this communication focuses on the process of bubble formation.

Methods

We developed a quantitative theory assuming that

1 gaseous micronuclei are necessary to initiate bubbles,
2 some of the micronuclei are crushed by the compression phase,
3 the individual micronuclei vary with regard to the pressure needed to cause crushing and with regard to the magnitude of supersaturation necessary to form a bubble,
4 the number of bubbles formed is equal to the number of micronuclei activated unless there is insufficient excess dissolved gas: if so, there are fewer bubbles than expected.
5 the bubbles generated are influenced by surface tension.

Results

Yount and Strauss counted bubbles in gelatine that had been subjected to various compression/decompression profiles. Application of our theory to the Yount-Strauss data indicates that all of our assumptions are warranted.

Conclusions

In the "critical volume hypothesis" , DCS is thought to be a function of excess dissolved gas alone; the opposing hypothesis is that gaseous micronuclei are crucial. Our theory shows the both ideas may be correct; in some profiles, available gas is limiting. Micronucleus limitation may distort graphs based on the critical volume hypothesis, and limitation by available gas may appear as noise on graphs based on the micronucleus hypothesis. As yet, we have applied the theory only to gelatine data; whether it is applicable to human DCS remains to be seen.

From

Department of Physiology, University at Buffalo, SUNY, Buffalo, New York 14214, USA.

**Key Words**

Bubbles, hyperbaric research.

## TRAINING IN UNDERWATER MEDICINE

**Diving Medical Training in the Canadian Forces. Current issues and preliminary review of 15 years experience.**

Bateman WA and Khan YA. *Undersea Hyperbaric Med* 1997; 24 (Suppl):17

**Abstract**

Background

In late 1982, the Canadian Forces (CF) approach to diving medical training was revised, creating a multi-level strategy that consist of "Basic", "Advanced" and "Consultant" tiers (equivalent to UHMS Levels I-III, respectively). Also given is introductory training, whose aim is to help practitioners recognise complex diving medical problems requiring specialised experience. Diving medical training is given to a wide variety of students.

Methods

A review of the 15 years' training since revision was conducted.

Results

As of end-1996, 476 students had attended 8 courses in diving medicine conducted in 45 serials; 209 students qualified as "Basic" (Level I), 84 as "Advanced" (Level II) and 7 as "Consultant" (Level III); of these, 277 were CF medical officers (MOs), 12 were MOs from other nations and 12 were civilian physicians; a further 182 non-physician personnel (mostly operational divers and paramedics) were trained and more than a further 700 medical, paramedical and other operational personnel received introductory training.

Conclusions

Current issues that were identified include:

1 the need for evidence-based clinical practice and training is increasing (both students and outside authorities are demanding better answers to questions about any intervention's efficacy and cost-effectiveness);
2 the "fitness to dive" issue is becoming increasingly complex (human rights legislation and the blossoming spectrum of diving operations make case-by-case assessments increasingly necessary, thus making clear-cut guidance harder for students to find);
3 the need for update and refresher training is becoming more acute (techniques such as distributed

training are being explored to help those not practising diving medicine full-time to stay abreast with rapidly arising new developments);

4 pressure from civilian agencies to provide training and expertise in diving medicine is mounting because no civilian equivalent of the School of Operational Medicine (SOM) exists and formal training in diving medicine is becoming mandatory for support of commercial operations; and

5 relentless pressure to reduce training costs has cut dedicated SOM manpower to just over two person-years, so extensive use must be made of "multiple hatted" personnel and guest instructors.

From

School of Operational Medicine, Defence and Civil Institute of Environmental Medicine, 1133 Sheppard Avenue W, North York, Ontario M3M 3B9, Canada.

**Key Words**

Training, underwater medicine.

## DEATHS FROM CARDIOVASCULAR DISEASE

**Fatalities related to cardiovascular disease in the recreational diving population.**

Caruso JL, Uguccioni DM, Dovenbarger JA and Bennett PA. *Undersea Hyperbaric Med* 1997; 24 (Suppl): 26

**Abstract**

Background

Cardiovascular disease is the leading cause of death in the United States and other industrialised nations. Older divers with many years of diving experience and individuals participating in dive training in the later years have reached the age at which risk of cardiovascular disease becomes significant. Deaths related to cardiovascular disease represent a significant portion of the total number of fatalities in the Divers Alert Network (DAN) recreational diving fatality database each year.

Methods

DAN collects all available information on recreational diving fatalities involving US citizens and publishes an annual report of diving accidents and fatalities. Sources of information on diving fatalities include DAN accident reports, police and coast guard investigative reports, eyewitness accounts and autopsy findings. Every case in reviewed by the DAN medical and research staff which includes physicians with experience in both diving medicine and forensic pathology. A total of 549 diving fatalities for the years 1990-1995 were reviewed to determine how many fatalities were associated with cardiovascular disease.

Results

A total of 66 recreational diving fatalities had cardiovascular disease as he cause of death or as a major factor contributing to the ultimate cause of death. This represents 12% of all fatalities in the database. Among the divers who were older than 35 years at the time of death, cardiovascular disease was a factor in 26% of the fatalities. Cardiovascular disease was second only to drowning as the leading cause of death in divers over 35 years of age.

Conclusions

Cardiovascular disease is a significant cause of mortality among recreational divers. especially in those over 35 years of age. Because expedient access to advanced cardiac life support (ACLS) is difficult or impossible in most recreational diving situations, pre-existing cardiovascular disease may increase the diver's risk of suffering a diving related fatality. The large number of older experienced divers continuing to participate in the sport and individuals participating in dive training at an older age should make screening for cardiovascular disease an essential part of determining an individual's fitness to dive.

From

Divers Alert Network, Department of Pathology and F G Hall Laboratory, Duke University Medical Center, Durham, North Carolina 27710, USA.

**Key Words**

Accidents, cardiovascular, deaths, recreational diving.

## RECURRENT PULMONARY BAROTRAUMA

**Recurrent pulmonary barotrauma in scuba diving and the risks of future hyperbaric exposures: a case report.**

Carpenter CR. *Undersea Hyperbaric Med* 1997; 24 (3): 209-213

Abstract

A 27-year-old male scuba diving student suffered two episodes of pulmonary barotrauma 6 months apart after 3.6 m (12 ft) training scuba dives. In the interval between these episodes, four uneventful hyperbaric chamber dives occurred. No definite cause or risk factors were identified. A MEDLINE review of diving literature revealed few studies of recurrent pulmonary barotrauma and no studies addressed risks of recurrent pulmonary barotrauma in the hyperbaric oxygen environment following scuba pulmonary barotrauma.

**Key Words**

Hyperbaric oxygen, pulmonary barotrauma, treatment

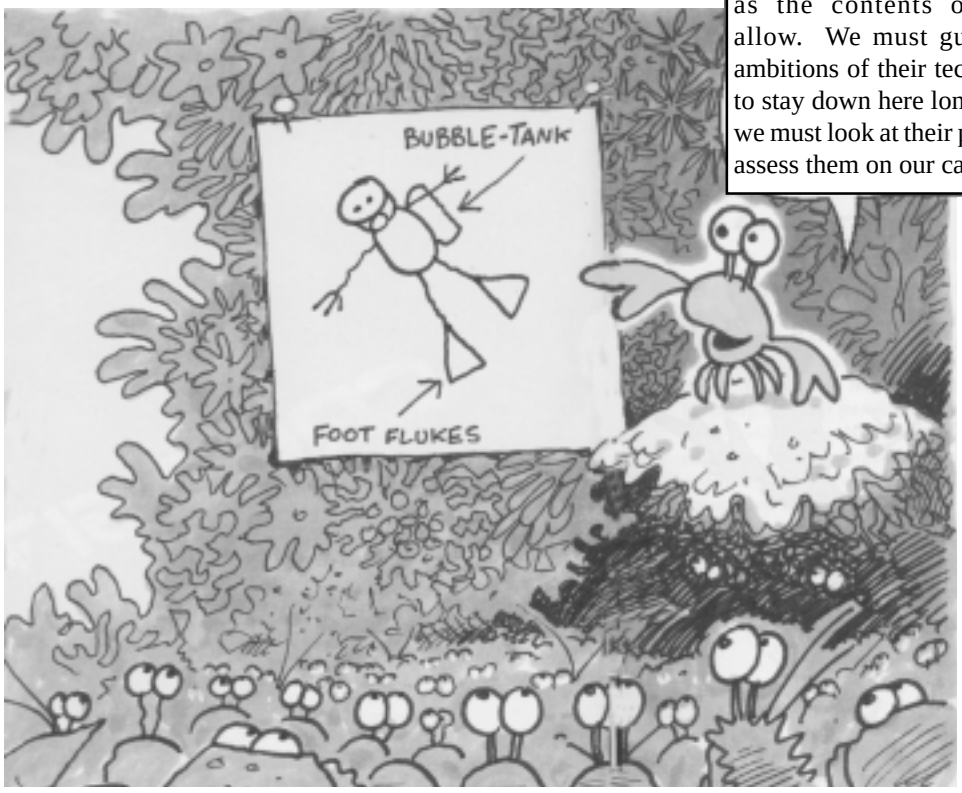
# THE SEA PEOPLE'S GUIDE TO DIVERS

**By RICO**

*Humans say that to see themselves as others see them is a great blessing. Imagine then what a blessing it would be to see themselves as other species see them. If only we could find a way of giving them a Sea People's view of themselves. Well, actually, we can...*

*Thanks to the kindness of Rico, the cartoonist, and of Bernard Eaton, the Editor of DIVER, who have agreed to allow this series of typical divers to be reproduced in the SPUMS Journal. Although the featured diver types originated in the UK, we believe that most of them, at one time or another, have attended a SPUMS Annual Scientific Conference.*

Luckily for us permanent residents, divers come underwater only briefly. They can stay only as long as the contents of their little bubble-cans allow. We must guess, however, that the high ambitions of their technology will soon allow them to stay down here longer and longer. This being so, we must look at their personality types so that we can assess them on our calamity-impact scale **...**




One of the first diver types to catch your attention will probably be the *Sea-Squirt*. Basically he is a harmless klutz whose energies and hapless enthusiasm far outweigh his brain-cell count. Loud noise and calamity are his constant companions. He is the guy who treads on the pillar valves of all the other divers' tanks as he stumbles aboard the inflatable. He unerringly targets the full fill of nitrox 36 first, and vents half the tank before he can fumble the valve shut again. His combination contents gauge console has more dials than a Klingon warship, and twice its firepower. With this lethal device he can level a RIBful of divers in one stumbling pirouette and still be ignorant of the reason for the carnage around him. Yet he is tolerated, and even encouraged, by his buddies to feel part of the team. Divers' natural affection for the underdog compels them to embrace explosively unpredictable idiots like the Sea-Squirt.

*Reprinted, by kind permission of the artist and of the Editor, from DIVER, the magazine of the British Sub-Aqua Club, 1997; 42 (12) December: 72-74.*

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