The Editor's Offering

In this issue we publish the provisional program for the 1997 Annual Scientific Meeting (ASM) to be held at the Quality Resort Waitangi on the Bay of Islands in New Zealand. For the first time an outside body has sponsored the travel costs of an overseas speaker at an ASM. Dr James Francis has been appointed Accident Rehabilitation and Compensation Corporation Professor for the meeting and all his travel and accommodation expenses have been covered by the New Zealand Accident Rehabilitation and Compensation Corporation. The Society is greatly indebted to the Corporation for its generosity.

With this issue we enclose an index of the Journal for 1996. Included in the index is a list of the key words used in the Journal index. The full index from 1971 to 1995 is now available on disc (Macintosh or Windows) for $Australian 10.00. Orders must specify the format required and be accompanied by payment. The Society accepts American Express, Bankcard (Australia and New Zealand only), Diners Club, MasterCard and Visa as well as cheques. If paying by credit card we need the card holder's name as on the card, the card number, expiry date and the amount to be charged.

The first part of Dr Michal Kluger's thesis *Implications of hyperbaric medicine for anaesthesia and intensive care* appears on pages 2-11. The second part will appear in the June issue. This is an important paper which members should bring to the attention of their friends and acquaintances in departments of Anaesthesia and Intensive Care. The Society was willing to allow Dr Kluger to offer the paper to *Anaesthesia and Intensive Care* provided that the SPUMS Journal would be given permission to reprint it after publication. Unfortunately, because of this condition, *Anaesthesia and Intensive Care* would not consider Dr Kluger's paper. Now only an educational campaign by SPUMS members is going to spread the word about the benefits of hyperbaric oxygen to those doctors working in hospitals which do not have hyperbaric units.

Dr Douglas Walker has been providing information about Australian diving-related deaths since 1972. His latest provisional report appears in this issue. The spectrum of causes runs from stupidity to unavoidable by way of deliberate suicide. The full series of Dr Walker's reports will be published in book form later this year.

This issue contains the papers on rebreathers delivered at the 1996 ASM in the Maldives. While the use of rebreathers in recreational diving is in its infancy, diving with enriched air nitrox and with heliox and tri-mix (helium, nitrogen and oxygen mixtures) is growing steadily. While most of this diving is done with open circuit there is a demand for rebreathers to extend time underwater by reducing the amount of gas lost on exhalation. However new dangers arise with the use of rebreathers, especially semi-closed systems. Divers using rebreathers need to have a full face mask to prevent them drowning while they go unconscious from hypoxia or oxygen toxicity. Navies have accepted this, but from the *Proceedings of Rebreather Forum 2.0*, which will be reviewed in another issue, it is clear that not all recreational divers have done so. Navies also insist on divers using rebreathers diving in buddy pairs on buddy lines so that an unconscious diver can have a better chance of survival. Such close contact diving finds few supporters in the recreational field. Semi-closed rebreathers are available for about $US 5,000 and upwards. One model is specifically designed for divers weighing less than about 75 kg.

Closed circuit rebreathers, using mixed gases, which have sensors to detect oxygen levels and control the addition of oxygen or diluent gas are much more expensive and should be safer. They can be if all the proper preparation and post-dive maintenance is done properly. These procedures add at least 2 hours to the dive. It is only the obsessional, or highly disciplined, diver, who can be relied upon to carry out the full maintenance and preparation, who should use this equipment. Discipline and much training in shallow water is required to become familiar with the problems which may arise and how to cope with them. Otherwise they can become an extremely expensive tomb. From the comments from manufacturers of closed circuit rebreathers reported in the rebreather forum they see no clear market for them to tailor their products to.

A major problem with all rebreathers is the efficiency of carbon dioxide removal. The US Navy (USN) has tested its absorbent to failure on many occasions at different temperatures using closed circuit mixed gas units. At identical work loads and temperatures there is a wide variation in the times taken to overwhelm the absorber's capacity. The USN's solution is to average the duration times and use that as the expected duration. As this is going to be longer than the shortest times found by experiment there is the probability that some divers are going to go unconscious from hypercapnia, hence the need for buddy lines. Rebreather safety would be much improved by using reliable CO_2 sensors as well as oxygen sensors. Unfortunately in September 1996 there was no CO_2 sensor available for rebreathers which had been extensively tested.

Loncar and Örnhagen describe a rebreather tester which can replace humans for most of the testing which should be done before a rebreather is released for sale. This includes testing with oxygen consumptions up to 3 l/minute. The machine can test the rebreather in deep water, in very cold water, for long durations and high work rates, all of which would put a diver in the water at risk.

ORIGINAL PAPERS

IMPLICATIONS OF HYPERBARIC MEDICINE FOR ANAESTHESIA AND INTENSIVE CARE PART 1

Michal Kluger

Summary

Hyperbaric medicine is becoming increasingly accepted as an important adjunctive therapy for many diseases. There are important considerations for anaesthesia and intensive care when interfacing with hyperbaric medicine. These include awareness of the indications for hyperbaric oxygen (HBO), physiological changes associated with HBO, potential complications and drug interactions. Awareness of these considerations will aid in the safe management of patients across these specialties.

Key Words

Anaesthesia, equipment, hyperbaric facilities, hyperbaric oxygen, hyperbaric research, medical conditions and problems, physiology, treatment and ventilators.

Background

Man's exposure to hyperbaric environments (breath hold diving) dates from at least 4,500 BC, and it is known that breath hold diving for sponge was a common and lucrative profession in Ancient Greece. Alexander the Great was reported to have descended in a glass diving vessel in 332 BC during the Battle of Tyre, one of the earliest recorded diving bells.[1] However the deliberate exposure to pressure for medicinal, non-diving purposes, was first described by Henshaw in 1664, when he constructed his *Domicilium*. This English clergyman and physician constructed a pressure vessel which could produce both increased (hyperbaric) or decreased (hypobaric) pressure to treat a variety of maladies using air as the breathing medium. Despite a belief that hypobaric exposure "cured" chronic diseases whilst hyperbaric pressure was better for acute diseases, scientific evidence was lacking.

Hyperbaric medicine's first contact with surgery and anaesthesia came with Fontaine's development of a mobile hyperbaric operating room.[2] Twenty-seven procedures were performed under moderate hyperbaric conditions using nitrous oxide as the sole anaesthetic agent. In addition the "normal" postoperative cyanosis was not seen due to the slightly higher partial pressure of oxygen in air at 1.25-1.3 atmospheres absolute (ATA). The diving equivalent of 1 ATA is 1 bar and the SI equivalent is 101 kPa. The father of pressure physiology, Frenchman Paul Bert, further developed hyperbaric nitrous oxide anaesthesia in the late 19th century, describing this in the treatise *La Pression Barometrique*.[3]

Hyperbaric air therapy continued to spread throughout Europe, Canada and the US as a panacea for a wide variety of illnesses. The complete loss of confidence in this type of treatment came with the public denigration of hyperbaric medicine in the North American medical community. Orville J. Cunningham, from Kansas City, used a hyperbaric chamber to treat the victims of the Spanish 'flu epidemic during World War I.[4] While its value in providing increased systemic oxygenation in pneumonias may have had some scientific basis, subsequent treatment for diabetes, hypertension, syphilis and cancers was at best naive and at worst fraudulent. Indeed, Cunningham postulated that cancers, diabetes and some arthritides were due to anaerobic micoorganisms, hence the efficacy of hyperbaric air. A grateful patient, who owned a large bearings firm, constructed a six storey, seventy-two room spherical hyperbaric chamber, complete with carpets and grand piano. Lack of scientific data to prove efficacy and minimal exposure to the greater medical community led an American Medical Association investigation bureau to conclude that hyperbaric medicine was "tinctured much more strongly with economics than with scientific medicine.[5] A hiatus remained in hyperbaric medicine until the middle of the 20th century.

Although Priestley discovered oxygen in 1776, it was not until 1937 that Benkhe and Shaw used hyperbaric oxygen (HBO), rather than air, to treat decompression illness (DCI). In the subsequent 30 years, carbon monoxide poisoning,[6] radiotherapy,[7] clostridial soft tissue infections[8] and osteomyelitis[9] were all treated with HBO. Once again surgeons were at the forefront in the development of hyperbaric medicine. Boerema, a Dutch cardiac surgeon, demonstrated that pigs which were exsanguinated, and had their blood volume replaced with saline to a haematocrit of 4%, could live with no problems for up to 15 minutes when subjected to 100% oxygen at 3 |atmospheres.[10] In the days prior to cardiopulmonary bypass, Boerema was able to perform complex cardiac operations, e.g. repair of Tetralogy of Fallot, under circulatory arrest and hypothermia. It was not clear however whether the real benefit from HBO was gained during the period of circulatory standstill, prolonging arrest time, or from enhancing oxygenation during the post-reperfusion phase.

As a direct result of Boerema's work, Professor Johnstone initiated the building of a hyperbaric facility at Prince Henry Hospital in Sydney in 1964. In an era of rapidly developing technology, extracorporeal oxygenation,

developed by Gibbon in 1953 and in wider use by the mid 60s, replaced HBO for cardiac operations. The requirement for surgery in a hyperbaric operating suite was soon relegated to that of historical interest only. Yet pioneering work by Dr Ian Unsworth allowed the unit to continue with new direction. The first recorded hyperbaric exposure in an Australian hospital occurred on 13th July 1970 at Prince Henry Hospital, with patient treatments taking place in early 1971 for carbon monoxide intoxication and gas gangrene. Over the next 25 years, formal units, mostly multiplace chambers, were formed in most Australian capital cities. New Zealand also developed hyperbaric centres on both North and South Islands.

TABLE 1

AUSTRALIAN AND NEW ZEALAND HOSPITAL HYPERBARIC MEDICINE UNITS IN 1996

State or Island	Town	Hospital	Address
South Australia	Adelaide	Royal Adelaide Hospital	North Terrace, Adelaide 5000
Northern Territory	Darwin	Royal Darwin Hospital	Rocklands Drive, Tiwi 0810
Western Australia	Fremantle	Fremantle Hospital	PO Box 480, Fremantle 6160
Tasmania	Hobart	Royal Hobart Hospital	PO Box 1061, Hobart 7001
Victoria	Melbourne	Alfred Hospital	Commercial Road, Prahran 3181
New South Wales	Sydney	Prince of Wales Hospital	High Street, Randwick 2031
New South Wales	Sydney	HMAS Penguin	RCC Facility, Balmoral 2088
Queensland	Townsville	Townsville General Hospital	Eyre St Townsville 4810
North Island New Zealand	Auckland	HMNZS Philomel	Naval Base, Devonport, Auckland
South Island New Zealand	Christchurch	Christchurch Hospital	Private Bag 4710, Christchurch

In Australia and New Zealand Specialist training in hyperbaric medicine is at present not structured. Historically, doctors with experience in diving and military medicine were at the forefront of hyperbaric medicine. Over the past years the majority of hospital hyperbaric facilities have been staffed by specialists in anaesthesia, intensive care or occupational and emergency medicine who have a sideline interest in hyperbaric medicine. Recently, independent units, with full time staffing, have allowed hyperbaric medicine to evolve into a distinct medical speciality; yet further work is needed to enhance the profile of hyperbaric medicine in the medical community. A recent review of physicians at a large United States teaching hospital showed that the majority of doctors never received any training in hyperbaric medicine in their undergraduate training. More importantly, these doctors may have had patients in their specialty who could have benefited from HBO treatment.[11] The Australian and New Zealand College of Anaesthetists (ANZCA) has approved a provisional fellow position in hyperbaric medicine to fulfil FANZCA requirements at the Royal Adelaide Hospital. Such positions have also been granted from the College of Emergency Medicine to Fremantle Hospital and the Royal Australasian College of Physicians at Alfred Hospital, Melbourne. Additionally, special interest group status is in the process of being sought from ANZCA.

Hyperbaric Medicine is not at present a registrable speciality in Australia, unlike in North America and Europe. The Diploma in Diving and Hyperbaric Medicine (DipDHM) is currently granted to doctors who have completed and passed a two week course in Diving and Hyperbaric Medicine, worked in a recognised Hyperbaric Unit for the equivalent of six months full time and submitted a thesis on one aspect of diving or hyperbaric medicine. This is submitted to SPUMS (South Pacific Underwater Medicine Society) for review by assessors. There are plans by the ANZHMG (Australian and New Zealand Hyperbaric Medicine Group) to develop a formal training program for hyperbaric medicine, but this is some time away. The growth of this field is reflected by the increasing number of national and international meetings devoted to hyperbaric medicine, along with national and international societies devoted to development and importantly controlled research into this area (Table 2). Hopefully the days of Orville J. Cunningham will never reappear!

Indications for HBO Treatment

An important rationalisation of the use of HBO came in the report of the committee on hyperbaric oxygenation.[12] This was a consultative document with the major US medical insurance companies (Blue Cross/Blue Shield) and Social Security (Medicare) along with an executive committee from the Undersea and Hyperbaric Medicine Society (UHMS). A list of those diseases which had sound basis for HBO treatment and those which were

TABLE 2

HYPERBARIC MEDICAL AND TECHNICAL ASSOCIATIONS
(in alphabetical order)

Organisations	Countries covered
Associacion Mexicana de Medicina Hiperbarica y Subacuatca, AC (AHMS)	Mexico and Central America
Australia and New Zealand Hyperbaric Medicine Group (ANZHMG)	Australia and New Zealand
Baromedical Nurses Association (BNA)	North America
European Undersea and Biomedical Society (EUBS)	Europe
Hyperbaric Technicians and Nurses Association (HTNA)	Australia and New Zealand
International Congress on Hyperbaric Medicine	International
Japanese Society for Hyperbaric Medicine (JSHM)	Japan
Societe de Physiologie et de Medicine Subacuatiques et Hyperbares de Langue Francaise (MEDSUBHYP)	France
South African Underwater and Hyperbaric Medical Association	South Africa
Undersea and Hyperbaric Medical Society (UHMS)	North America

investigative was formulated. These indications are under continual review and form the basis of practice in the majority of hyperbaric units in Australia and New Zealand.

Conditions commonly treated by HBO include; decompression illness, acute carbon monoxide (CO) poisoning, chronic osteomyelitis, osteoradionecrosis, problem wound healing (e.g. diabetic and/or arteriosclerotic wounds) and necrotising soft tissue infections.[12] There are other indications which would be considered as having considerable potential benefit, such as crush and reperfusion injury and compromised skin flaps. Finally there are occasions when HBO may be used in exceptional circumstances, or which are currently under study (e.g. thermal burns, exceptional blood loss anaemia (Jehovah's witnesses) or soft tissue sporting injuries).

TABLE 3

BENEFICIAL EFFECTS OF HYPERBARIC OXYGEN THERAPY

A **Reduction in bubble size**

B **Hyperoxygenation**
Vasoconstriction
Angiogenesis
Antibacterial (direct and indirect mechanisms, inhibition of toxin production and deactivation of toxins)
Osteoclastic stimulation
Fibroblast stimulation

Physiology of hyperbaric oxygen therapy

HBO is thought to exert beneficial effects through a variety of mechanisms (Table 3). Knowledge of the physiological responses to HBO are essential when dealing with patients who are critically ill, elderly or have significant cardiorespiratory disease. Key questions that need to be asked before any treatment include; can HBO benefit the patient and can it be associated with adverse physiological events? The answers to these questions can alter both anaesthetic and intensive care management of patients undergoing HBO therapy.

HEART RATE RESPONSE

Bradycardia is commonly seen during HBO treatment. Possible mechanisms include; direct pressure effect on pacemaker function, hyperoxia itself, increased work of breathing with dense gases or the effects of dissolved inert gases.[13] Bradycardia is also seen when 100% oxygen is breathed at normal (surface) pressure (1 ATA, 1 bar, 101 kPa).[14] Örnhagen studied hyperbaric exposure of isolated sinus node preparations from mouse, rat, guinea pig, rabbit and dog hearts.[13] All species showed a direct pressure related (up to 150 bar, 15,000 kPa) reduction in beating frequency in isolated pacemaker cells, which was not modulated by adrenergic or cholinergic agents. Studies in intact animal models at lower pressures suggest that this bradycardia may, however be mediated via vagal stimulation, baroreceptor activation following vasoconstriction and increased mean arterial pressure, or as a direct effect on chemoreceptors.[15,16,17,18] Over the course of a hyperbaric exposure the initial bradycardia will become less, but does not tend to return to baseline levels until the treatment is completed.[15] Unexplained tachycardia or even normalisation of heart rate have been

reported in the convulsive and pre-convulsive periods of central nervous system (CNS) oxygen toxicity.[19] In exposures up to pressures of 71 bar (7,100 kPa), which are not used clinically, a pressure induced bradycardia and reductions in P and T wave amplitudes were seen;[20] other conduction changes have been seen in commercial saturation divers.[21]

CARDIAC OUTPUT

Cardiac output has been shown to be reduced when breathing 100% oxygen at surface pressure (1 bar, 100 kPa) and during hyperoxic hyperbaric exposures.[14,23-27] This reduction in cardiac output may be oxygen tension dependent rather than due to the effect of pressure per se. Normoxic exposure to pressures up to 6 bar (600 kPa) using mixtures of helium, oxygen and nitrogen failed to demonstrate any reduction in cardiac output, contrasting with the hyperoxic data.[28]

Cardiac output is determined mainly by heart rate, preload, contractility and afterload. The heart rate response to HBO is usually bradycardia. HBO may affect preload as shown by an increase in haematocrit (Hct), possibly secondary to accumulation of interstitial transudate.[29,30] This may be mediated by the release of adrenalin, atrial natiuretic peptide and endothelin causing an increase in vascular permeability and leakage of fluid and albumin.[31] An increase in Hct of over 40% in a 6 hour study at 3 bar (300 kPa) was demonstrated by Amin,[30] an effect which may be increased in patients who have other causes of fluid loss, e.g. sepsis, burns, unhumidified ventilation, nausea, vomiting or impaired consciousness. Such haemoconcentration needs to be considered when assessing fluid requirements in the peri-treatment period. It can be worsened by repeated fasting for surgical debridements and interruptions of fluid administration during transportation.

Myocardial contractility has been shown to increase[32] or decrease[33,34] during HBO therapy. Myocardial contractility and left ventricular pressure have been shown to increase during exposures to 5 bar (500 kPa), even when the partial pressure of oxygen was maintained at the same level as before compression.[35]

There are few human studies which examine the haemodynamic responses to HBO at clinically relevant pressures. Pisarello[36] noted in a volunteer study that cardiac output decreased significantly during continuous oxygen exposure of 2-3 bar (200-300 kPa), but this effect recovered during the latter part of the hyperbaric exposure. A standard hyperbaric treatment profile with intermittent air breaks was examined by Pelaia.[37] Cardiac output, heart rate and stroke volume were all significantly reduced, while mean arterial pressure increased compared with that at 1 ATA. Cardiac output and stroke volume reduction rapidly reverted to control levels after cessation of HBO, but no change was noted during air breaks. Reduced cardiac output probably relates primarily to the combination of heart rate reduction and increase in systemic vascular resistance. However reduction in coronary blood flow[15] in combination with increased indices of contractility[35] may lead to negative supply/demand ratio with resultant cardiac ischaemia and loss of function.

PERIPHERAL CIRCULATORY RESPONSES

The peripheral circulatory response to 1 ATA oxygen in anaesthetised dogs was examined by Plewes.[14] Compared to an air control group, both heart rate and cardiac output fell by 14% and 7% respectively. There was no change in total peripheral resistance, however regional tissue beds showed a variable response to oxygen administration. Renal outer cortical and juxtamedullary blood flow fell by 20%, while there were no changes in overall splanchnic blood flow. Total cerebral blood flow was unchanged, but mesencephalon, vermis and hippocampal flows differed significantly from control values. Retinal blood flow was most markedly reduced, by 27% from the air control. Oxygen delivery may be significantly altered due to the balance of increased oxygen solubility combined with reduced tissue blood flow. Since this is calculated as the product of cardiac output and arteriovenous (A-V) oxygen difference, it may be unchanged in acute hyperoxia. In dogs at normal atmospheric pressure (1 bar, 100 kPa) the total oxygen delivery was 106 ml/min on room air compared with 103 ml/min when breathing 100% oxygen at 1 bar. The 20% increase in renal vascular resistance and reduction in renal blood flow seen at 1 bar (100 kPa) oxygen[14] was also demonstrated to occur in canine studies at pressures up to 4 bar (400 kPa).[38]

In unanaesthetised rats at 5 bar (500 kPa), heart rate and cardiac output fell by 21% and 14% respectively.[15] Organ blood flow, measured with the microsphere technique, fell in most organs, but was maintained at control levels in the kidney, liver and adrenals. In comparison with other studies oxygen delivery was significantly increased to the kidneys at 5 bar (500 kPa), from 1.26 ml O_2/min/g to 1.67 ml O_2/min/g. Importantly, in this study, right and left ventricular blood flow fell by 41 and 47% respectively with no change in myocardial performance, suggesting that myocardial ischaemia may be a possibility at hyperbaric pressures.

Studies looking at haemodynamic modifications during HBO have tended to focus on healthy control subjects. However, Muhvich examined regional blood flows in an antibiotic-treated septic rat model.[39] There was an overall reduction in renal, adrenal and myocardial blood flow from 1 bar to 2 bar. These alterations continued for 20 minutes after conclusion of the compression. Interestingly, there was no difference between control and “septic” rats. This may question the model, which even at 1 bar did not show any haemodynamic difference between the two groups.

Ten critically ill patients, who required invasive monitoring, were subjected to hyperbaric oxygen at 2.5 bar (250 kPa). There was an increase in oxygen delivery (DO_2), but no change was noted in oxygen consumption (VO_2) and oxygen extraction ratio, in contrast to the limited human data at 1 bar.[40]

PULMONARY CHANGES WITH HBO

Administration of HBO causes a significant increase in arterial and venous PO_2. Healthy subjects breathing 100% oxygen at 3.4 bar (340 kPa) showed raised PaO_2 (1,721 mm Hg, 2.26 bar or 226 kPa) and PvO_2 (424 mm Hg, 0.55 bar or 55 kPa) levels while $PaCO_2$ levels were marginally raised.[24] This latter effect was presumably secondary to the loss of CO_2 buffering capacity from reduced haemoglobin.

HBO does have effects on both pulmonary mechanics and vascular responses.[41] There is ongoing debate about the effect on alveolar-arterial (A-a) gradients during HBO therapy. Many studies have shown an increased A-a gradient[24,42] of up to 460 mm Hg (0.6 bar or 60 kPa) at 3.0 ATA. Flook,[43] in a porcine model, suggested that the shunt fraction (Q_s/Q_t) could increase to over 25% during hyperbaric exposure to 3 bar (300 kPa). This contrasts with other workers who have failed to show any increase in A-a gradient during HBO.[44] Interpretation of such data is difficult as there are significant differences in the experimental models. Data from uncontrolled human studies suggest that the measured PaO_2 is greater than predicted in patients with significant pulmonary disease. In contrast, patients with normal lungs have lower than predicted PaO_2 levels.[45] This has important implications for HBO therapy, as expected PaO_2 levels may not be achieved with the usual treatment regimens in some patients.

Animal studies have shown that the acute administration of HBO at 2.8 bar (280 kPa) induced significant increases in pulmonary vascular resistance and blunting of normal hypoxic vasoconstriction, while lung mechanics (static lung compliance, wet to dry weight ratio and surface tension) did not change.[29] These changes recovered after breathing air for 24 hours. Hyperbaric oxygenation significantly improves systemic oxygen supply; however this effect may be offset by pre-existing pathology (e.g. chronic airway disease, cardiac failure), disease processes (e.g. sepsis), drug therapy (e.g. vasodilator or vasoconstrictor therapy) or airway management (e.g. positive pressure ventilation and positive end expiratory pressure [PEEP]). Measurement of PaO_2 during treatment sessions are highly recommended to optimise therapeutic goals.

SUMMARY

There are many physiological responses which occur during HBO treatment. These have been studied widely, but interpretation is difficult due to differing animal models used in the studies, presence or absence of anaesthesia, variable pressure, duration and gas mixtures utilised and differing experimental methodology. However, some basic conclusions may be drawn. Heart rate reduction occurs frequently and may be significant in those patients with existing bradycardia or conduction delay. The increase in peripheral vascular resistance, leading to reduction in cardiac output, needs to be considered in those patients with marginal haemodynamic status or who are on vasoactive medications. The onset and offset of these cardiovascular changes can be anticipated, with appropriate vasodilator and vasoconstrictor therapy readily available. Direct invasive monitoring of all critical patients is mandatory, along with the recognition that HBO may not always mean improved tissue oxygen supply. Abolition of hypoxic vasoconstriction, increasing dead space and shunt all reduce theoretical gains from measured oxygenation. As there is potential for organ damage during HBO, ECG, arterial blood pressure, urine output and oxygenation (using pulse oximetry or arterial blood gases) should be measured in all critically ill patients and in those with marginal organ function. Further work is needed to investigate this in controlled human studies at pressures between 2 and 5 bar (200-500 kPa).

Administration of hyperbaric oxygen

In Australia and New Zealand the administration of HBO is most commonly carried out in dedicated multiplace chamber facilities. This contrasts with parts of Europe, United States and Asia where monoplace chambers are more commonly used.

MULTIPLACE CHAMBERS

In multiplace chambers (Fig 1, p 7) oxygen is breathed via a hood or demand flow BIBS (built in breathing system) apparatus. In cases where there is head and neck pathology, e.g. burns or radical surgery, a hood is often preferable. In other instances, e.g patient preference or claustrophobia, BIBS apparatus may be the better option. There are also rare circumstances in patients who have tracheostomies, or other anatomical problems, who require modification to their breathing circuit.[46] The multiplace chamber allows attendants to be present with the patients and so allows patient interventions which would be impossible in a monoplace chamber e.g. intubation, pleurocentesis and intravenous cannulation. It can also use treatment pressures greater than 3 bar (300 kPa), and often up to 6 bar (600 kPa). Other assistance can be easily obtained by pressurising staff to the treatment depth rather than requiring emergency patient decompression. However, multiplace chambers are more expensive to manufacture and maintain, and require attendants for each treatment, thus exposing them to risk, albeit small.

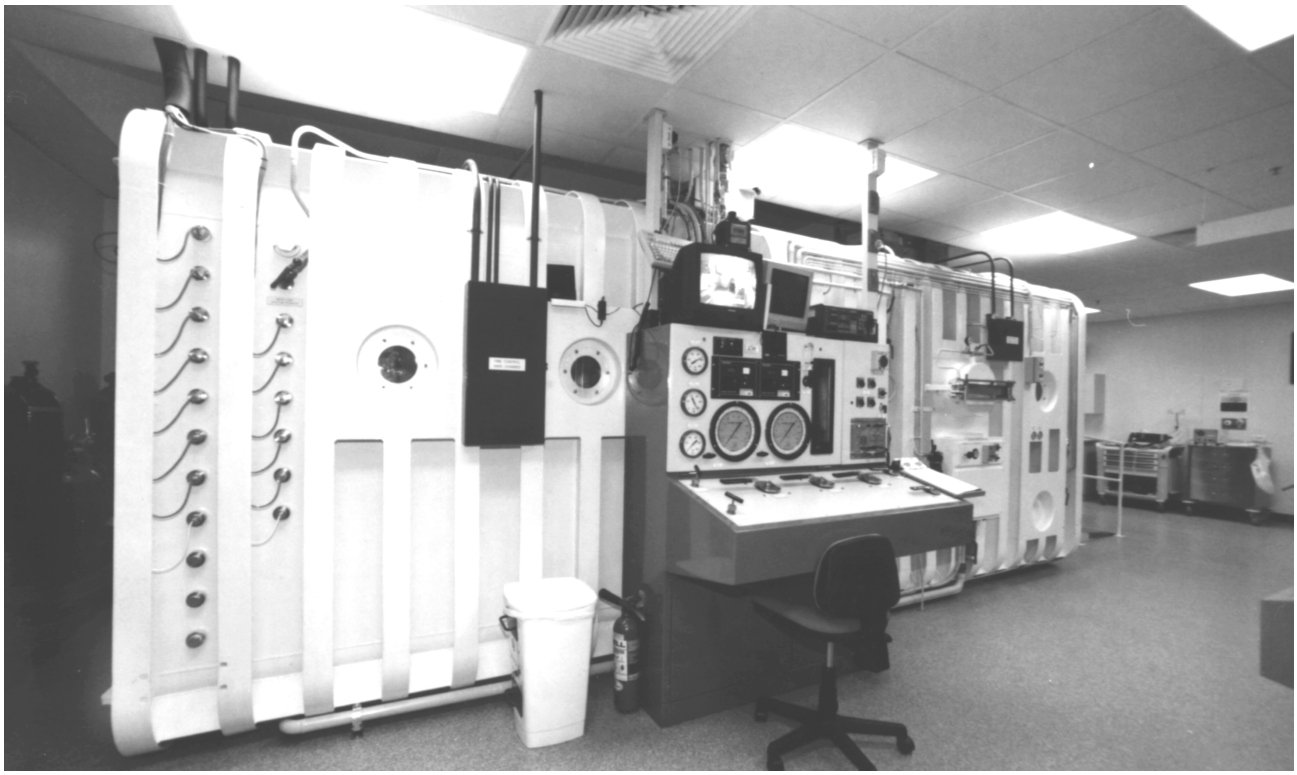

Figure 1. Multiplace chamber. (Royal Adelaide Hyperbaric Medicine Unit).

MONOPLACE CHAMBERS

Monoplace chambers (Fig 2, p 8) allow patients to breathe 100% oxygen from the environment. Air breaks can be given to co-operative patients who are able to use BIBS themselves.[47] They are cheaper than multiplace facilities, easy to use and do not require attendants. Their main drawback is lack of patient access if problems arise. These can be overcome, and some centres routinely treat unstable, critically ill patients in monoplace chambers. Finally, the additional risk of fire and explosion in a 100% compared to 21% oxygen environment, also makes monoplace chambers potentially riskier.

General

ATTENDANTS

It is essential that a trained attendant accompanies each patient during hyperbaric treatment sessions in a multiplace chamber. Ideally, these attendants should be the nurse treating the patient in the intensive care unit (ICU) or ward setting, so that continuity of care is maintained, although practically this may be limited by hospital staffing levels and nurse availability.

The risk of DCI in the patient undergoing hyperbaric oxygen therapy is rare. This is because patients usually breathe 100% oxygen for the duration of the treatment, apart from intermittent air breaks which minimise the risk of both CNS and pulmonary oxygen toxicity. DCI may become a problem in those treatment tables which use air as the breathing medium at depth, although these tables are rarely used nowadays. Attendants however do not breathe oxygen routinely at depth. Formal recommendations are made for some schedules used for diving problems (e.g. US Navy, Comex and other recompression schedules), but these are subject to many alterations. Moreover, the majority of HBO treatments are not standardised.[48] Finally, rates of compression, and more importantly decompression, are not uniform, thus potentially making the generation of in vivo bubbles possible with resulting symptoms and signs of DCI.

Anderson reviewed figures for DCI in 62 medical personnel who underwent 1,516 compressions.[49] Barotrauma was the most common adverse effect, affecting 47% of medical staff, though none required myringotomy or grommet insertion. Symptoms of DCI (extremity pain, pulmonary signs, retrosternal discomfort and dysaesthesias) occurred in 9 treatments (0.6%). Interestingly, there were also three episodes of homonymous hemianopia, one of which lasted for 10 weeks. Retinal field defects and cotton wool spots have been described in an attendant who breathed 100% oxygen at 2 bar (200 kPa).[50] Retinal vessels demonstrate marked vasoconstriction during hyperoxic exposure, an effect which shows great variability between individuals and in the same individual on different days. A more recent report from Baltimore reviewed 25,164 exposures.[51] The overall incidence rate of DCI in

Figure 2. Monoplace chamber. (photo courtesy of Dick Clarke, Carolina Hyperbarics).

attendants was 0.076%. Broken down by treatment pressure the incidence rate was as follows; 2.0 bar, 0.04%; 2.5 bar, 0.08%; 2.8 bar, 0.14%; 4.7 bar 1.68% and finally 6 bar, 5.71%. Figures from 1985 to 1995 at the Royal Adelaide Hospital Hyperbaric Unit indicate a similar rate. In 5,792 chamber runs, there were 4 reported cases of DCI, an incidence of 0.07%.

Inert gas narcosis (IGN) is well recognised in the diving literature as a consequence of exposure to inert gases (usually nitrogen) at depth with resulting neurological dysfunction. It has been suggested that there may be subtle neurocognitive effects of nitrogen at a partial pressure of 1 bar (100 kPa).[52] The relevance of IGN has been largely overlooked in the clinical hyperbaric literature. Attendants looking after critically ill patients require normal cognitive and psychomotor activity. Observation, interpretation and institution of corrective strategies may be affected by IGN. "Slight mania and euphoria" was self reported in 11 attendants out of 1,516 exposures at pressures above 2.5 bar (250 kPa).[49] Inert gas narcosis is a function of both depth and partial pressure of inert gas. Some hyperbaric units perform test treatments to acclimatise their attendants, making them aware of the effects of IGN by getting them to perform calculations and simple tasks at depth. There is some evidence from the diving literature that repeated exposure to depth "protects" against IGN. The presence of a trained nurse and physician outside the chamber also aids in the monitoring of both patient and attendant via close circuit television or direct observation.

BAROTRAUMA

Middle ear damage due to failure of equalisation is the most frequent complication of hyperbaric treatment. While it can be prevented in conscious patients who can auto-inflate their middle ear space, fluid retention, inflammation or Eustachian tube dysfunction can make this problematic.

Active auto-inflation during pressurisation led to acute severe hypotension (<40 mm Hg) in a patient with air embolism following a mediastinoscopy.[53] This patient had pulmonary artery occlusion, with the presumed mechanism of hypotension being reduced venous return following the Valsalva manoeuvre. An inadvertent breath hold by a study subject, produced hypotension (a drop from 120 mm Hg to 60 mm Hg) during decompression.[54] Again the proposed mechanism is that of reduced venous return from increasing intrathoracic pressure. Moreover, excessive equalisation can lead to rupture of the round or oval windows, resulting in tinnitus, deafness and vertigo. Equalisation problems are increased in the unconscious or sedated patient, when the first indication of a problem may be an unexplained tachycardia on compression which resolves suddenly after perforation of the tympanic membrane or bleeding into the middle ear cavity. There are two approaches to this latter situation. Firstly, emergency tympanotomy, with or without insertion of grommets. The second approach is to tolerate the middle ear bleeding, which will obliterate the air space and allow future compressions

to take place. However this may lead to problems with hearing impairment or infection, especially in the septic, immuno-compromised patient. The availability of ENT assistance and urgency of treatment often dictates which pathway is followed.

Theoretically, in patients who have an untreated or undiagnosed pneumothorax, HBO can be commenced before pleurocentesis is carried out. In many instances these pneumothoraces will resolve with 100% oxygen and compression alone.[55] In practice, unless there are exceptional circumstances, definitive management and stabilisation should be carried out before HBO. Chest tube insertion and connection to a Heimlich valve is the treatment of choice. Emergency pleurocentesis can be carried out in a multiplace chamber, although conditions may be cramped. It is not a viable option in a monoplace chamber; here a pneumothorax could be a life threatening situation, if decompression to ambient pressure was not possible before cardiac arrest occurs from a tension pneumothorax. However there have been no reports of such fatalities.

Other possible complications include sinus and dental barotrauma. Ventilatory impairment can occur in patients who have large amounts of intestinal, and especially gastric, air. Failure to relieve gastric distension has resulted in gastric rupture during decompression after a diving accident,[56] but has not as yet been reported following clinical HBO treatment. Nasogastric intubation should however be considered in patients who are sedated and ventilated, have had expired air ventilation or have a history of oesophageal anti-reflux surgery.

Skilled technical staff are required to prevent the ultimate barotraumatic insult, the uncontrolled decompression. Luckily this is rare, but is inevitably associated with mortality, often multiple.[57] As with other areas of modern medicine, treatment and crisis management algorithms can help prevent these disasters.

OXYGEN TOXICITY

Oxygen at high percentage or partial pressure has been demonstrated to have adverse effects on many body organ systems. The mechanisms of toxicity probably involve the production of reactive oxygen species (oxygen free radicals) which overcome the body's natural defence mechanisms. These highly reactive molecules include superoxide, hydrogen peroxide and hydroxyl ions. These molecules are produced in small quantities normally in the body, and can be dealt with by a variety of host defences which include avoidance of the univalent pathway, breakdown by enzymes (e.g. superoxide dismutase, catalase, glutathione peroxidase) and provision of natural anti-oxidants (e.g. vitamin A and C). However, under certain conditions, the defences can be overwhelmed, leading to alteration in membrane function, enzyme inactivation and subsequent loss of cellular and organ function. An in-house review carried out in the Royal Adelaide Hospital showed an overall incidence of CNS oxygen toxicity of 1.5 per 1000 treatments. This figure doubled in treatments at 2.8 bar or greater.

The incidence and severity of pulmonary and CNS oxygen toxicity can be reduced during treatments by the use of intermittent periods of lowered oxygen partial pressure (air breaks) during hyperoxia. Even brief (5 minute) air breaks can substantially extend the limits of oxygen toxicity. Hendricks showed that, using the decrease in vital capacity (VC) as an indicator of pulmonary oxygen toxicity, air breaks more than halved the decrease in VC seen with continuous hyperoxia.[58]

Experimental animal models have looked at various pharmacological methods of preventing the respiratory or CNS effects of hyperbaric oxygen. Strategies have included; vitamins A and E, prostacyclin analogues, intracellular hydroxyl ion scavengers (dimethylsulphoxide, dimethylthiourea, mannitol), chelating agents (desferrioxamine), lipid anti-oxidants (butyrated hydroxytoluene), arachidonic acid pathway modifiers (non-steroidal analgesic agents, magnesium), leukotriene inhibitors, inhibitors of nitric oxide synthetase and other synthetic analogues of natural defence mechanisms (catalase, superoxide dismutase), however none have been proven to be reliably effective in man nor have anticonvulsants, given for prophylaxis of CNS complications, been effective

HBO AND TUMOUR GROWTH

Finally, the question regarding cancer promoting effects of HBO needs to be addressed, for both patients and attendants. Theoretical risks of HBO therapy include; nourishing the tumour, immunosuppression and generation of oxygen free radicals, which are implicated in the genesis of some cancers. A review of the HBO literature has gone some way to answer this question.[59] Eleven animal, twelve human and one combined study were reviewed. Only two animal and three clinical human studies suggested a pro-cancer effect with HBO; all of these could be criticised on methodology and analytical technique. The conclusions drawn from available data suggest that HBO is not associated with an increase in tumour growth or metastases.

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The above paper is Part 1 of the thesis submitted for the Diploma of Diving and Hyperbaric Medicine which was awarded to Dr Kluger in 1996. At that time he was working in the Hyperbaric Medicine Unit at the Royal Adelaide Hospital, Adelaide, South Australia.

Part 2 will be published in the June issue of the Journal.

PROVISIONAL REPORT ON AUSTRALIAN DIVING-RELATED DEATHS IN 1993

Douglas Walker

Summary

Four snorkelling deaths, thirteen using scuba and four using a compressor supplied hose (hookah) were identified. No claim is made that all the fatalities have been identified. The deaths in the snorkelling group all occurred unobserved, although others were nearby at the critical time. Cardiac factors were implicated in two cases, epilepsy in one and one who drowned for no identified reason was possibly incompletely recovered from a recent viral illness. The causes of death in scuba divers included three possibly cardiovascular deaths, two shark attacks, two disappearances, two drownings with multiple adverse factors in the dive history, one with definite cerebral arterial gas embolism (CAGE), one with possible CAGE and depth related factors, one suicide and an unexplained death. The bodies of three of the victims were never recovered. Two of the hose supplied divers died from carbon monoxide poisoning, the third was victim of a shark attack and the fourth died from either CAGE or surface drowning.

Key words

Breathhold diving, CAGE, carbon monoxide, case reports, deaths, diving accidents, marine animals.

Snorkel user fatalities

BH 93/1

A group of four friends went spearfishing. After a time three of them decided to move to another area but first had to inform the other member of the group. Initially they misidentified him, signalling to a stranger who was diving near where they had last seen him. When they saw a spear gun on the sea bed they became anxious. Later they saw a snorkel at the surface and swam out. He was floating at the surface, face down, with froth coming from his mouth. He was quickly brought to shore but their resuscitation attempts failed. The sea was calm and visibility excellent. Autopsy revealed that his heart showed the changes of a primary cardiomyopathy. He was unobserved for only 10 minutes.

SPEARFISHING. SEPARATION. FOUND FLOATING. CARDIOMYOPATHY.

BH 93/2

Passengers on a cruise liner were offered an excursion to view the Barrier Reef. After a rough trip they reached a pontoon moored at a reef where they could view the reef from a glass bottomed boat and to borrow fins, mask and snorkels to swim in the area bounded by buoyed ropes between the pontoon and the reef. Buoyancy vests were available. The victim was, according to his wife, in good health, a good and confident swimmer and it is probable that he was snorkelling at the surface. He was on metoprolol tartrate (Betaloc) but no details of his medical condition are recorded. When he entered the water there were about 25 others swimming around, though earlier more had been in the designated area. There were two crew members appointed to watch the swimmers, though their task was made difficult because some were making short dives. His wife watched him for a time. When she next looked about 10 minutes later she was unable to see him and a search failed to find any trace of him. It is assumed that, for some unknown reason, he drowned silently and drifted away. His experience with using a snorkel is unknown.

SNORKELLING. SOLO AMONG OTHERS. SILENT DEATH. BODY NEVER RECOVERED. HISTORY HYPERTENSION. ON BETALOC. SUPERVISORS OF AREA SAW NOTHING UNTOWARD. GOOD SEA CONDITIONS.

BH 93/3

Two weeks after being struck by the Influenza A virus girl had recovered sufficiently to holiday at the Great Barrier Reef with her parents. She joined a dozen or so others to make an escorted snorkel viewing of a nearby reef. As they boarded the boat the supervisor counted them and collected their tickets but did not record their names. They were given a brief introduction to the use of a snorkel and fins during their trip to the dive location . Although she was said to be a good swimmer and to have used a snorkel previously, she chose to wear a flotation vest. Its buoyancy kept her on the surface. After reaching the anchoring area they all entered the water and swam, with their dive supervisor, about 20 m to a bommie where he described the corals and fishes they could see there. They were then free to swim about in the area, but first he asked them not to stray too far from him. He made a head count before leading them back to the boat and believed that all were present. The sea was calm with only a slight current. One of the group said that there seemed to be one person missing, so he decided to return to the reef area in case she was there. He saw nobody. After checking that she had not swum back to the beach or returned in another boat, he then conducted a wider search. He found her floating face down, mask in place, about 500 m from the bommie. Resuscitation attempts were unsuccessful. The autopsy showed no other cause for her death than drowning. She was described as having been “a cautious child, one who would never have put herself in danger” so she would not voluntarily have left the group, and the fact that her mask was in place was taken to indicate the absence of panic. The reason for her silent death cannot be known. Possibly

she inhaled water down her snorkel and suffered cardiac inhibition. Other than this occasion her experience with a snorkel is unknown.

RECENT ILL HEALTH. SNORKELLING IN CALM SEA. SILENT DEATH IN GROUP. WEARING BUOYANCY VEST BUT FLOATED FACE DOWN. DELAYED RECOGNITION OF ABSENCE. SUPPOSED GOOD SWIMMER. SNORKEL EXPERIENCE UNCERTAIN.

BH 93/4

Her intention had been to make a Resort Dive but when she filled in the medical history sheet she revealed that she had suffered an epileptic fit after a severe head injury 10 years before. She was told by the diving instructor that she could not be accepted for a dive in case a fit occurred during the dive. She declared that she was on regular medication and had never suffered a fit since this was started, but was still refused. However, as the ticket for the trip to the Barrier Reef included a statement that all passengers could borrow snorkel gear, the staff felt they were unable to refuse to supply her with this. The area for snorkelling was between the two moored boats and the reef. There was supervision by a member of the crew but viewing was difficult because the calm sea reflected the sunlight. The major safety factor was assumed to be the presence of a number of swimmers in the same area. Nobody noticed anything untoward and her absence was not noticed until, 2 hours later, a roll call was made. Six hours after she had entered the water her body was retrieved by a boat several kilometres away. A helicopter observer had seen her fins at the surface, with a dark shadow beneath and guided the boat. Had she failed to reveal her epilepsy history she would have been under the close supervision of the diving instructor on a Resort Dive Experience and her survival chances would have been better. It is assumed she suffered an epileptic fit and drowned.

POST-TRAUMATIC EPILEPSY HISTORY. REFUSED PERMISSION TO MAKE A RESORT DIVE. SILENT DEATH AMONG OTHER SWIMMERS. DELAY BEFORE HER ABSENCE NOTED. PROBLEMS IN SUPERVISION OF GROUP OF SWIMMERS.

Scuba Diver fatalities

SC 93/1

Three friends were snorkelling in a popular diving area. On returning to shore they looked down and saw a still figure on a concrete block below them. They dived down and found it was a scuba diver. There were no bubbles of air coming from his regulator and they found he was attached to the concrete block, which explained the failure of their attempts to bring him to the surface. Police divers had to use bolt cutters on the padlocked chain connecting him to the block. Suicide notes and the padlock's key were later found in the diver's car. He was known to be depressed, was receiving medication and had the support of friends but this proved insufficient to prevent the tragedy. He had made previous suicide attempts but on this occasion, he had taken great care to eliminate all possibilities of failure in his attempt. It is terrible to think of a person's state of mind who has arranged to wait to drown when his tank becomes empty. He had been aware that his body would be found as the area was frequented by divers.

EXPERIENCED DIVER. DEPRESSED. PREVIOUS FAILED SUICIDE ATTEMPTS. SOLO. CHAINED HIMSELF TO BLOCK TO ENSURE DROWNING. SUICIDE.

SC 93/2

Shark attacks on scuba divers are fortunately rare so it may be thought particularly unjust that this attack occurred on a honeymoon couple. They were both experienced scuba divers, the victim more so than his wife. It was a popular and frequently dived area and their first day's dive had been without incident. A large shark they had seen was thought to be a grey nurse and caused no anxiety. On the fatal day there were five divers on the dive boat which was close to some small rocky islets. The other divers formed a trio while the couple dived together. After an uneventful dive for 25 minutes at 21 m in good visibility they saw a large shark about 7 m from them when they were at 10 m. It was swimming away from them. They surfaced about 50 m from the dive boat, then the buddy remembered that they should have made decompression stops, so they descended to 9 m and after 3 minutes rose to 3 m. There the buddy looked round and saw a large shark approaching rapidly. The victim was a little behind and deeper than the buddy. The shark took him in its jaws and swam away. No blood was seen in the water after the attack. The buddy rapidly surfaced and cried out for help. Despite knowing that a shark attack had occurred, one of those in the boat jumped in to assist her while the boat was carefully but quickly brought to pick her up. At this time the other three divers were making a decompression stop. One of the divers, an instructor, made a courageous dive to see whether he could retrieve any part of the victim or his equipment. He saw a large shark in the area and then saw it swim about 3 m in front of him, so surfaced before he developed a need for in-water decompression. A large shark was seen by those in the boat before they left the area. Some fishermen later hooked a shark which vomited out the victim's torso before making its escape.

EXPERIENCED DIVERS IN POPULAR DIVING AREA. SUDDEN MID-WATER SHARK ATTACK DURING DECOMPRESSION STOP.

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and experience		Dive	Dive	Depth m (ft)		Weights	
		Victim	Buddy	group	purpose	Water	Incident	On	kg (lb)
BH 93/1	24	No training Experienced	No training Experienced	Group Separation before incident	Spear fishing	1.2 (4)	Not stated	No	Not applicable
BH 93/2	66	No training Experience not stated	-	Group Separation before incident	Recreation	21 (70)	Surface	No	Not applicable
BH 93/3	16	Some training Experienced	-	Buddy Separation before incident	Recreation	Not stated	Surface	No	Not applicable
BH 93/4	30	No training No experience	-	Group Separation before incident	Recreation	15 (50)	Surface	No	Not applicable
SC 93/1	35	Trained Experienced	-	Solo	Recreation	10 (30)	Surface	On	11 (24)
SC 93/2	31	Trained Experienced	Trained Experienced	Buddy Separation during incident	Recreation	24 (80)	3 (10)	On	Not stated
SC 93/3	29	Trained Experienced	Trained Experienced	Group Separation before incident	Recreation	37 (123)	Ascent	Off	Not stated
SC 93/4	34	Trained No experience	Trained Experienced	Buddy Separation before incident	Recreation	4.8 (16)	4.8 (16)	On	14.5 (32)
SC 93/5	43	No training Some experience	Trained Experienced	Group Separation before incident	Recreation	10 (30)	Surface	Off	Not stated
SC 93/6	34	Trained Experienced	Trained Experienced	Group Not separated	Recreation	75 (250)	75 (250)	On	Not stated
SC 93/7	34	Trained Experienced	Trained Experienced	Group Separation before incident	Recreation	10 (30)	5 (15)	On	9 (20)
SC 93/8	38	Trained Experienced	Training not stated Experienced	Buddy Separation before incident	Cray fishing	6 (20)	Not stated	On	Not stated
SC 93/9	43	Some training No experience	Trained Experienced	Group Separation before incident	Pupil	18 (60)	Not stated	Not stated	8 (18)

DIVING RELATED DEATHS IN 1993

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	Comments
None	Not applicable	Not applicable	Not applicable	Own	Shallow. Short separation from group. Cardiomyopathy. Silent cardiac death
None	Not applicable	Not applicable	Not applicable	Hired	Silent death in crowd. Drifted away. Never found. Hypertension.
Life jacket	Not applicable	Not applicable	Not applicable	Hired	Silent drowning in group. Drifted away. Recent “flu”.
None	Not applicable	Not applicable	Not applicable	Hired	In crowd. Floated face down. History of post-traumatic epilepsy.
Not inflated	Yes	Yes	Some faults	Own	Depression. Suicide.
Not inflated	Yes	Yes	Not stated	Own	Shark attacked after diver descended to make omitted decompression stop.
Not able to be inflated	Yes	Yes	Serious fault	Hired	Trio. Rapid descent. Rapid ascent hand over hand up anchor line. Buddies continued dive. Vest faulty. Overweighted. CAGE.
Not inflated	Yes	Low	No fault	Own	Asthma history. Just trained. Had had a panic attack during training.
Not inflated	Yes	Yes	No check	Own	No recent experience. Trio. Solo ascent. Coronary artery disease.
Buddy inflated	Yes	Yes	Significant fault	Own	Deep dive. Sudden unconsciouness. Possible nitrogen narcosis, CO_2 retention or O_2 toxicity. Possible CAGE.
Not inflated	Yes	Yes	Not stated	Own	Shark attack during descent, mid-water near seals.
Inflated	Yes	Yes	Some faults	Borrowed	No dives for 6 years. Separation. Found floating. Possible angina.
Not inflated	Yes	Not stated	Equipment lost	Borrowed	2nd open water dive. Drift dive. Separation. Body found 2 months later.

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and experience		Dive	Dive	Depth m (ft)		Weights	
		Victim	Buddy	group	purpose	Water	Incident	On	kg (lb)
SC 93/10	22	Trained Experienced	-	Solo	Recreation	15 (50)	Not stated	On	Not stated
SC 93/11	61	No training No experience	Trained Experienced	Group Not separated	Recreation	10 (30)	10 (30)	On	Not stated
SC 93/12	44	Trained Inexperienced	Trained Experienced	Group Separation before incident	Recreation	18 (60)	Surface	On	Not stated
SC 93/13	34	Trained Experienced	Trained Experienced	Group Separation before incident	Recreation	Not stated	Surface	Not stated	6 (14)
H 93/1) H 93/2)	32 27	No training Some experience	Trained Some experience	Buddy Not separated	Cray fishing	7.5 (25)	7.5 (25)	On	Not stated
H 93/3	27	Training not stated Experienced	Training not stated Experienced	Buddy Separation before incident	Work	12 (40)	12 (40)	On	Not stated
H 93/4	29	Trained Some experienced	Trained Some experience	Buddy Separation before incident	Netting fish	5.5 (18)	Ascent	On	Not stated

-Double fatality

SC 93/3

A diving holiday package was arranged by a dive shop in another State. The victim had trained elsewhere but was a member of the club and had dived with its members, though not with those making this trip. The local dive shop checked that they had certification, but not their experience level. It was a boat dive and although the boat owner held a dive master qualification he did not assume the responsibilities. There were seven divers and he left it to them to decide their dive groups, merely advising them not to exceed 33 m. The victim, being a stranger to the others, joined a buddy pair. He entered the water before his buddies, coming rapidly back to the surface because his air was not turned on. He then started his descent without waiting for his buddies. As they descended they could see him close to the sea bed, which was at 37 m, about 5 m from the anchor and swimming towards it. When they reached 22 m they met him ascending rapidly, hand over hand up the anchor line. They signalled to him to slow down. They observed no signs of panic and his breathing appeared normal. They thought he would reach the surface safely so continued their descent and their dive. He had waved his octopus regulator at them as they passed, what he meant by this is unknown.

The man in the boat was surprised to see someone back at the surface less than 5 minutes from the beginning of the dive and became alarmed when the diver floated face up and failed to answer his call. He swam a line to the victim but, by the time he reached him, the victim was unconscious and not breathing. In-water CPR was started and, with the help of two nearby fishermen, he was put aboard the dive boat. Although alive when he reached hospital he never regained consciousness and died there later. He had been wearing his weight belt when he encountered his buddies during his ascent but it was absent when he was

DIVING RELATED DEATHS IN 1993 (CONTINUED)

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	Comments
Not inflated	Yes	Not stated	Equipment lost	Own	Solo unannounced dive. Sea conditions good. Reputedly cautious diver. Body never recovered.
Buddy inflated	Yes	Not stated	Not stated	Hired	Resort Dive. Requested ascent. Said was OK but acutely ill on pontoon. Acute heart pain. Died next day. Myocardial infarction.
Part inflated	Yes	None	No fault	Hired	At end of dive, solo return to boat on surface. Language problem. Unexplained death. Possible subarachnoid haemorrhage.
Not inflated	Yes	Not stated	Equipment lost	Hired	Advanced certificate after 9 dives. Buoyancy problems. Separation at surface at start of dive. Current. Body never found.
No vest	Not applicable	Not applicable	Some faults	Own	Untrained. Limited experience with hookah. Calm, hot, no wind. Dog knocked intake hose into boat. CO poisoning.
No vest	Not applicable	Not applicable	Some faults	Borrowed	Recent training. Little experience. Calm, hot, no wind. Dog knocked intake hose into boat. CO poisoning.
No vest	Not applicable	Not applicable	No fault	Employer	Shark attack in turbid water. Working on pearl farm, cleaning lines, shells.
No vest	Not applicable	Not applicable	Some faults	Own	Netting fish. Lost fin. Separation. Ascended as replaced fin. Surface cry, then sank. Possible CAGE.

reached at the surface. The rescuer attempted to inflate his buoyancy vest but failed. Subsequent examination showed there was a leak at the attachment of the inflator hose to the vest. An X-ray was performed before autopsy which showed a small left pneumothorax, some air in the left ventricle and some mediastinal emphysema. The autopsy showed that both ear drums were ruptured and that sinus barotrauma had occurred. His weight belt was described as "excessively heavy" but it was not recovered and its actual weight is not known. He probably descended uncontrollably rapidly, due to an inoperative buoyancy vest, suffering severe pain in his ears and sinuses. Failing to drop his weights he had to pull himself up the anchor line to return to the surface. It would be easy in such a situation to forget to breath correctly during the ascent and consequently suffer pulmonary barotrauma and CAGE.

TRAINED. POSSIBLY EXPERIENCED. TRIO. ENTERED WATER WITH AIR OFF. RAPID DESCENT WITHOUT WAITING FOR BUDDIES. THEN MADE RAPID ASCENT. PULLED HIMSELF UP ANCHOR LINE TO SURFACE. BUDDIES FAILED TO ACCOMPANY TO SURFACE. NEW WET SUIT. PROBABLY EXCESSIVE WEIGHTS. FAULTY BUOYANCY VEST. DITCHED WEIGHTS LATE IN ASCENT. UNCONSCIOUS AT SURFACE. PRE-AUTOPSY X-RAY SHOWED LEFT PNEUMOTHORAX, AIR IN LEFT VENTRICLE. CAGE. BAROTRAUMA EARS AND SINUSES.

SC 93/4

He had revealed his asthma history at his diving medical, but possibly played down its severity. On the basis of simple respiratory function tests (no provocation tests were performed) he was passed as fit. This decision

was undoubtedly influenced by his history of managing stress situations and involvement in triathalon competitions. His condition was known to his wife but not to his colleagues at work. In his short diving career he had acted calmly when he became separated during a drift dive and had to manage in a current. In contrast he had suffered an episode of panic hyperventilation at the surface during training which his instructor successfully managed. The victim's buddy was aware of his inexperience and took particular care to keep close to him at all times. They snorkelled out to a shallow reef, depth 3-4 m, and dived for about 33 minutes before the buddy decided it was time for them to return. About 7 minutes later the victim looked at his contents gauge before making a somewhat rapid ascent. The buddy had a 88 cu ft tank, the victim a 63 cu ft one, so the buddy had plenty of air at this time. It is assumed that the victim was down to 50 bar and believed this required surfacing, but the reading is unknown.

The surface conditions had deteriorated while they were under water so the buddy indicated they should return to the beach underwater. His signal was answered but he did not see the victim on the sea bed or when he returned to the surface. He heard a sound like a howl but saw nobody. He called out "Drop your weights. Inflate your vest". The waves limited his range of vision and he soon felt in need of assistance. His calls brought some divers who helped him to shore. A search was unsuccessful, although the victim's mask and snorkel were found. When the body was located next day there was sufficient air remaining to inflate his buoyancy vest and float the body. His weight belt was twisted round but whether this was a significant factor is unknown.

Autopsy showed the presence of thick, blood stained mucus in the trachea but no signs of pulmonary barotrauma or infection. There was evidence of some air trapping in the distal airways, due to plugs of thick brown mucus. He had a nebuliser fitted in his car which he used while driving to dives. Blood assays showed salbutamol (Ventolin) and pseudoephidrine hydrochloride (Sudafed) to be present. He was also reportedly using regular beclomethasone diproprionate (Becotide). The probable sequence of events was inadequate surface buoyancy in rough water, failure to inflate his buoyancy compensator coupled with failure to use his regulator or drop his weights. His respiratory tract changes may well have significantly reduced his capacity to exercise. Asthma was only one of several adverse factors.

TRAINED. INEXPERIENCED. ASTHMA HISTORY REVEALED AT DIVE MEDICAL. RAN IN TRIATHALONS. COLLEAGUES UNAWARE OF HIS ASTHMA. EPISODE OF SURFACE PANIC IN TRAINING. USED NEBULISER BEFORE DIVE. SYMPTOMS OF URTI TREATED BY "SUDAFED" BEFORE DIVE. SEPARATION AT SURFACE IN ROUGH WATER. FAILED TO INFLATE BUOYANCY VEST. FAILED TO DROP WEIGHT BELT. EXPERIENCED BUDDY FOUND SURFACE CONDITIONS SEVERE.

SC 93/5

Four friends decided to go diving, one reason being to provide a refresher dive for the victim who was untrained but had some past experience. He had not dived for some time because of ill-health. He had supposedly recovered from cancer of his spine and a back problem, but no details are recorded. One of the friends decided to fish from the rocks so was given the duty of keeping a watch and to assist them leave the water after the dive if requested. The two who were both trained and experienced took care to watch their friend during the early part of their dive at 5 m. They felt that he was competent so they gradually continued down to 10 m. After about 27 minutes he indicated that he wished to ascend. They were close to the agreed exiting area so continued with their dive while he returned to the surface. He showed no signs of panic or distress. The friend left fishing heard a call for help and then saw the victim holding onto a rock with waves washing over him from time to time. When the friend reached the victim, he was floating at the surface face up. His buoyancy vest was not inflated but his weight belt was off. The others heard the fisherman call out when they surfaced and together they managed to bring the victim onto the rocks. He failed to respond to their resuscitation attempts. At autopsy severe atherosclerotic changes were found in the left anterior coronary artery but no evidence of either old or recent myocardial infarction. The stress of his dangerous situation may have led to a severe angina or sudden arrhythmia, or inhaled water may have caused sudden cardiac inhibition. That he was out of training, separated from his buddies and in rough water in a rocky cleft were all adverse factors. He was described as "a heavy smoker, a bit overweight, but not fat".

UNTRAINED. PAST EXPERIENCE. NO RECENT DIVING BECAUSE OF ILL HEALTH. TRIO. ALLOWED TO MAKE SOLO ASCENT. BUDDIES CONTINUED DIVE. SURFACED SAFELY. ENTERED ROCKY CLEFT WITH ROUGH WATER. UNCONSCIOUS BEFORE REACHED. SEVERE LEFT CORONARY ATHEROSCLEROSIS. WATER POWER. PROBABLY CARDIAC DEATH.

SC 93/6

The six divers were all experienced in deep dives though only two had previously dived to 75 m, one being the victim. This was a dive on a deep wreck. One was to remain in the boat. When the first pair started their dive the others set up decompression bars at 6 and 3 m. As the trio descended they met the first pair ascending. On the wreck they tied a reel line to the anchor and then swam over the wreck. As the three divers began their return to the anchor,

in line ahead the tail ender saw that the middle diver (the victim), though he appeared to be finning, was making no progress. He assumed the victim had become snagged but when he touched him he saw he was unconscious. The regulator fell from the victim's mouth as he was turned over so the buddy replaced it. He appeared to breath shallowly but in a rapid hyperventilation manner. The diver attracted the attention of the leader and held the victim while the leader cut the reel line. Then the tailender let go and started to make his ascent. The leader grabbed the victim and replaced his regulator, but no further efforts to breathe were observed. At 13 m he put some air into the victim's buoyancy vest, ditched his weights and allowed him to ascend to the surface unattended. He then returned to 15 m to start the planned decompression. The man in the boat saw the victim break the surface and immediately jumped into the water. With the assistance of the first pair, who had completed their decompression stops, he got the victim into the boat. Their resuscitation attempts were unsuccessful.

A pre-autopsy X-ray showed a massive air presence intravascularly, with air in the heart and pulmonary vessels and also many other vessels. Some air was post mortem out-gassing but the total amount indicated probable air embolism. There were several adverse factors. Calculations showed that he had used far less air than would have been expected. His regulator was hard to breathe and had a partly inverted exhaust valve which would have caused a spray of water with each inhalation. He was wearing two tanks and the regulator on the second one also was misassembled, however he had not breathed from it. Oxygen toxicity, carbon dioxide retention and nitrogen narcosis could all have affected him and he was using equipment unsuitable for a deep dive because it required too much effort to breathe.

EXPERIENCED DIVER. DEEP DIVES. LOST CONSCIOUSNESS AND SEEN FITTING. TRIO. NITROGEN NARCOSIS POSSIBLE REASON WHY ONE BUDDY ABANDONED VICTIM. REGULATOR HARD TO BREATH. POSSIBLE WATER SPRAY ON INHALATION. LOSS OF CONSCIOUSNESS. PROBABLE NITROGEN NARCOSIS. POSSIBLE CARBON DIOXIDE RETENTION. POSSIBLE OXYGEN CONVULSION. PROBABLE CAGE DURING RAPID UNCONSCIOUS ASCENT.

SC 93/7

On a trip to view an island seal colony there were family members and children in addition to the three experienced divers, only one of whom had previously dived there. The victim had not dived during the past 18 months. The boat anchored more than 30 m from the island and they swam on the surface towards it. They descended, when near the island, to 10 m. Two had reached the sea floor and were watching the victim, who appeared to be equalising her ears at 5 m, when a shark was seen to take her across her body, let go of her, then swim away with her. They remained on the bottom for a time, then decided that it would be safest to exit onto the island. There were now no seals in the water around them and they had difficulty getting through the throng on the rocks. It took a little time to catch the attention of those on the boat, who were unaware of what had occurred. It was later reported that a diver hunting crayfish had encountered a shark here 2 months previously and discouraged it with his spear gun.

THREE DIVERS OFF SEAL COLONY ISLAND. GOOD VISIBILITY. SEPARATION DUE TO DIFFICULTY EQUALISING EARS. MID-WATER SHARK ATTACK. BODY NEVER RECOVERED.

SC 93/8

The employees of a firm had an outing to a resort island, the majority in one boat and three following in the boat owned by one of them. There was some surfing and swimming by all, then the owner of the private boat asked whether the victim would like to scuba dive with him. The owner had spare equipment with him so it is assumed that he was an experienced diver. It is known that the victim had been trained 8 years ago and dived regularly for 2 years but had not dived since. Visibility was good, sea conditions excellent and the water was shallow at the reef close to where the boat was anchored. The owner spent about 5 minutes exploring under a rock shelf at about 4 m, the victim remained outside looking for crayfish. When the owner emerged he was surprised not to see his friend. After a look around underwater he surfaced, but still could not see him. After another underwater check he climbed onto the bommie to obtain a better view. He saw an inflated buoyancy vest at the surface 70 m away, so went over in his boat. He found the victim floating, unconscious, face up and without his mask. After ditching the weight belt he managed to get him into the boat. This was difficult because there was now some breeze and a chop. CPR was unavailing. A check of his equipment showed the tank valve was incompletely open so that breathing would have required extreme effort. A history was later obtained that he had reported chest pains during the previous 3 months, but these had not been regarded as cardiac in origin. At post mortem the left descending anterior coronary artery was affected by atheroma, 60% occluded in places. While there is no evidence that he suffered an anginal attack or that arrhythmia had occurred, and no evidence of air embolism was noted, critical adverse factors were a combination of inexperience, separation and difficulty in obtaining adequate air.

TRAINED. SOME PAST EXPERIENCE. NO DIVING FOR 6 YEARS. BORROWED EQUIPMENT. TANK VALVE ONLY PART OPEN. SEPARATION. POSSIBLE ANGINA HISTORY. 60% NARROWING LEFT CORONARY ARTERY. SUDDEN DEATH.

SC 93/9

The 8 divers were all known to each other at work. One was a diving instructor, three were his pupils and the other four were trained. This was the third open water dive for two of his pupils but only the victim's second as he had aborted one dive because he became too cold. The boat was anchored in 8-9 m close to a drop off. The dive plan was for them to meet on the sea bed and then make a drift dive as a group, never to exceed 18 m. It was not to be considered as a part of the training course. There was no allocation of buddies. There was some current and they all descended at different rates so separation occurred. The instructor gathered four of them in one place and then swam towards the three pupils (two on the sea bed, the third still descending) and signalled them to follow him back to the main group. He thought one started to follow him but soon found he was alone so went back but found no sign of them. He assumed they had decided to dive as a trio and returned to conduct the planned dive with the four trained divers. The victim's absence was noted only after all surfaced and a head count was made. The two surviving pupils described how one had descended quicker, despite having ear equalisation problems, than his under-weighted friend. By the time they were both on the bottom there was no sign of the others and they never identified the drop off, finding themselves at 22 m at one time. They had ascended to the agreed 18 meters and drifted until down to 50 bar. Both groups had assumed the victim was safe with the other. The body was found floating 10 weeks later.

PART TRAINED. 2nd OW DIVE. GROUP DRIFT DIVE. NOT PART OF COURSE. NO BUDDIES ARRANGED. SEPARATION ASSOCIATED WITH INITIAL DESCENT. INSTRUCTOR FAILED TO ACT AS SUCH TO PUPILS. DROWNED.

SC 93/10

As a crew member of a boat taking divers out to the reef and a trained diver it was not against policy for her to go for a dive. Her experience is unknown, but she was described as being an excellent swimmer. An instructor was taking a group of divers on an advanced diver course and there were other divers in the water, but nobody was aware that she intended to dive or saw her enter the water or in the water. Her absence was not noticed until later and no trace of her or her equipment was ever found except for a small piece of her swimsuit. The water was very deep close to where the boat was anchored, too deep for searching. It is unknown why she dived alone or what happened to her. Although a shark attack is possible there is no evidence for this. Water conditions were good for diving when she disappeared.

TRAINED. EXPERIENCE UNKNOWN. SOLO DIVE. BODY NEVER FOUND.

SC 93/11

This was a well-conducted Resort Dive, undertaken off a pontoon moored at a reef. The diver's medical questionnaire revealed no ill health nor medications. The instructor took two divers to a maximum depth of 9 m. After 16 minutes the victim indicated that he wished to ascend, which they did in a normal manner. At the surface he stated he was "all right" but insisted he wished to return to the pontoon. The instructor partly inflated his buoyancy vest and assisted him to swim to the float at the end of the mermaid line attached to the pontoon. The victim part swam, part pulled himself the 20 m to the pontoon where his equipment was removed and he was assisted back on board. It was suggested that he should rest. Very shortly after this he became very pallid, sweaty, felt faint and sick and breathless, with noisy breathing. Some chest pain was also mentioned. He was placed on 100% oxygen. The Diver Emergency Service (DES) and the nearest hospital were contacted. The hospital sent a doctor by helicopter. His condition had so greatly improved with the oxygen that he was evacuated to the hospital for a period of observation and tests rather than because of his condition at the time. He made a good recovery from this episode of acute cardiac decompensation in hospital, but died there the next night from a cardiac arrhythmia due to an acute myocardial infarct. Ischaemic heart disease was noted. It is recorded he had been experiencing some anginal symptoms for about two weeks, indeed had felt an unusual weakness when walking that morning before he dived. It is believed he had a "dive medical" before being allowed to book the reef trip as he had indicated he intended to make a Resort Dive, but no copy of the report is available. It is unknown whether he was aware that his symptoms were due to angina.

RESORT DIVE. NO HISTORY OF ILL HEALTH. WELL CONDUCTED DIVE WITH CLOSE SUPERVISION. ASCENT WHEN ILL-DEFINED SYMPTOMS OF ILL HEALTH. ACUTE CARDIAC DECOMPENSATION AND SHOCK SYMPTOMS AFTER HE LEFT THE WATER. RESPONDED TO OXYGEN. REACHED HOSPITAL. DIED LATER. CARDIAC DEATH.

SC 93/12

Among the divers making a four day dive trip to visit some of the less accessible reefs were three from overseas whose experience was uncertain, though all were trained. The victim had obtained certification on an overseas holiday a year before and not dived since, so the instructor on the boat accompanied him during his first dive to check that he seemed competent. They made four dives on each of the first two days, the sea conditions perfect. However on the third day the visibility was poor. On the second dive of the day they dived as a trio. The two women seem to have managed the current they encountered underwater better than the victim. The dive leader twice left them on

the sea bed and surfaced to check their position. On the second occasion they started to follow her and, so poor was the visibility, collided with her as she was descending. On the third occasion they followed her to the surface as their air was becoming low. They found conditions had worsened and there were rain squalls. The two women signalled to the dive boat they wished to be collected but the victim decided to swim back to the boat rather than wait. By now there were some waves. The dinghy which collected them was on the line the victim took for his return but he was not seen. There was some initial delay due to language problems before they made it clear a diver was missing. An immediate surface search failed to sight him and an underwater search was organised. He was found, lying on the coral, at about 19 m, his mask full of blood, weight belt in position. The autopsy was unsatisfactory, no clear reason being offered for the blood in his mask and lungs, though it was suggested it was a result of aspiration of gastric contents. There was some blood at the base of the brain but its source and significance remains uncertain.

TRAINED. INEXPERIENCED. SURFACE LOW AIR. SEPARATION TO SWIM BACK TO DIVE BOAT. UNEXPLAINED DEATH. POSSIBLY ASPIRATION VOMIT SYNDROME. POSSIBLE SUBARACHNOID HAEMORRHAGE. FAILED TO INFLATE BUOYANCY VEST. FAILED TO DROP WEIGHT BELT. LANGUAGE PROBLEM. CHOPPY SEA DEVELOPED.

SC 93/13

A live-aboard dive boat carried 26 divers among whom there were four from overseas who required the assistance of the interpreter aboard. All held Advanced Diver certification, obtained after making a total of 9 dives, and they had subsequently made respectively 9, 22, 26 and (the victim) 20 dives, although the type of dives is unknown. The instructor gave a talk about the dive conditions and the interpreter was present to translate the talk to this foursome. How completely this was performed is doubtful as some of them believed their dive was to be as a group of four while others believed they were to form buddy pairs. After entering the water they swam in the wrong direction, to the stern rather than the bow, then held onto the mermaid line and adjusted the straps of their equipment. They were slow to leave the surface and the instructor was just about to go to them in the dinghy to offer assistance when the last one was seen to disappear from view. It was not until a subsequent roll call after the divers returned that anyone was aware that a diver was missing. The others described how the first two divers descended easily and watched the third slowly descending. He had waited for the victim, who appeared to be experiencing buoyancy problems, to join him. The visibility was poor, his buddy did not arrive and, hearing the dinghy's outboard motor overhead, he assumed the missing diver had returned to the surface and been retrieved. He therefore continued his descent and joined the others, believing it was intended to be a group dive. They continued the dive as a trio. Although an immediate and determined search was made no trace of either the diver or his equipment was ever found.

TRAINED. CERTIFIED ADVANCED DIVER AFTER 9 DIVES. SOME EXPERIENCE AFTER COURSES. SEPARATION AS DESCENDED. DESCENT DIFFICULTY DUE TO EXCESS BUOYANCY. CAREFUL WATCH ON DIVERS' WATER ENTRY. FAILED TO DITCH WEIGHT BELT. FAILED TO INFLATE BUOYANCY VEST. LANGUAGE COMMUNICATION PROBLEM. BODY NOT RECOVERED.

Hose supply divers

H 93/1, H 93/2

The owner of the compressor was untrained and had only recently bought it. His practice was to go diving, either with any available companion or solo, leaving the boat empty except for his dog. One trained diver, who had dived with him several times, advised him that he needed to make three changes to achieve a safe set up. He should never leave the boat unoccupied while diving, the air intake hose on the compressor should be fixed securely, on a pole, well above the boat, and the compressor's engine exhaust should be extended to reach over the side of the boat. This advice was ignored, a fatal error. The conditions were unusual for the area, with the sea glassy calm, no breeze and excellent visibility. A friend who had recently completed a scuba training course, and had dived a few times with scuba, was found to accompany him. Their failure to return was assumed to be due to their having run out of fuel, but when friends reached their boat it contained only an agitated dog. The compressor was cold as it had run out of fuel. They pulled up the single hose and found the two bodies still attached. They had died by drowning when they lost consciousness from carbon monoxide poisoning. It was the owner's habit to place the air intake hose, unattached, on the side of the boat. In the past it had occasionally been dislodged by the dog. On this occasion there had been no breeze to clear the exhaust fumes from within the boat so they would have been sucked into the compressor and contaminated the air supplied to the divers.

DIVER 1 UNTRAINED. FAILED TO ACT ON ADVICE TO MAKE HOOKAH SAFE. DIVER 2 RECENT SCUBA TRAINED. INEXPERIENCED. ENGINE EXHAUST INTO BOAT. AIR INTAKE HOSE NOT FIXED. DISLODGED BY DOG AND FELL INTO THE CARBON MONOXIDE POOL IN BOAT. NO BREEZE. CO POISONING.

H 93/3

Work for divers on a pearl farm is unromantic, cleaning the lines and shells of marine growths. The task is performed by pairs of divers working from small boats. The compressors were left unattended because the noise level was too high for anyone in the boat to tolerate. The debris causes the water to become turbid and attracts many fish, including tiger sharks. However these had never troubled the divers. The two divers were working on adjacent lines, supplied by the same compressor, when the buddy noticed he was short of air and had to use his bail-out bottle to surface. He checked that the compressor was working correctly then donned a fresh bail-out bottle and weight belt, intending to dive to continue his work. He found he was unable to descend any deeper than 3 m before he again experienced an inadequate air supply. Puzzled he called to the occupants of a nearby boat. They saw bubbles breaking the surface and when they pulled up the victim's hose they found he was missing, as was the end coupling of the hose with the regulator. Although aware that this almost certainly indicated a shark attack the buddy dived, using air supplied from the second boat, to see whether he could recover the body. He found evidence that an attack had occurred, damaged lines, but there was no sign of the victim. Some damaged parts of the equipment were recovered, the weight belt being still closed when found. A 2.5 m shark was caught 6 days later and found to contain a skull and a few vertebrae. Tests established they were the victim's. The buddy experienced air lack because of the free flow which occurred after the regulator was bitten off the air hose.

EXPERIENCED HOOKAH DIVER. PEARL FARM. CLEANING SHELLS AND LINES. SHARK ATTACK. NO PREVIOUS SHARK ATTACKS HERE. TURBID WATER.

H 93/4

A married couple had a salt water aquarium and held a licence to catch small reef fish for it. They left the boat unoccupied while they dived, the compressor unattended. Though both had obtained scuba training their experience with hookah is not recorded. They used a fine net to catch the fish, each one being disentangled and placed in a catch bucket in the boat as soon as possible. As they were returning to the boat with a fish the wife's fin came off. While she tried to replace it her husband returned to the boat. After placing the fish in the catch bucket he submerged again but was unable to see his wife. He returned to the boat and saw from underneath it that her air hose was leading out from the stern of the boat in the direction of the 1 knot current. About this time people in a nearby boat saw his wife surface and heard her call for help. By the time they reached her position she had sunk from view. Her husband, who was still underwater at this time, saw her slowly sinking. She was about 15 m (50 ft) distant, her back towards him, her demand valve hanging free. She was on the sea bed, weight belt on and mask off, before he reached her. He brought her up and CPR was commenced but she failed to respond. She had been underweighted for this shallow dive, drifting up while replacing her fin. It is unexplained why she was unable to remain at the surface. A formal finding of drowning was reached but it is possible she could have suffered a cerebral air embolism during her ascent through concentrating on her task and holding her breath. But there is no evidence that this occurred. The hookah was noted to supply inadequate air if two divers were working hard: this was not the case here. The air compressor was one designed to spray paint.

TRAINED. UNKNOWN EXPERIENCE HOOKAH. SEPARATION AFTER LOST A FIN. BUOYANCY CAUSED ASCENT WHILE REPLACING FIN. CALLED FOR HELP THEN SANK. MASK OFF. WEIGHT BELT ON. NO BUOYANCY VEST. POSSIBLE CAGE. SOME ADVERSE COMMENTS CONCERNING HOOKAH.

Discussion

The four deaths while using snorkels illustrate the impossibility of any effective supervision of a group of swimmers at the surface, particularly if some are making occasional dives. The fact that bodies appear to have drifted away unobserved underlines this fact. There is also proof that death can occur unobserved in a group where nobody is taking specific notice of anyone else. It was an example of the injustice of life that revealing a history of epilepsy placed the person in a less protected situation, as the instructor would have been observing her had she been in his Resort Dive group.

The scuba diver group of deaths contains examples of an unusually wide range of factors. There were two shark attacks (SC 93/2, SC 93/7), a highly unusual situation, and a suicide (SC 93/1) in addition to the more commonly identified factors. In three there was a proven or possible cardiac factor (SC 93/5, SC 93/8, SC 93/11) and in two an indisputable finding of CAGE (SC 93/3, SC 93/6) on X-ray before autopsy. There were three cases where the victim was either inexperienced or had not dived for a number of years (SC 93/5, SC 93/8, SC 93/12) and one where an instructor took three part-trained pupils on a drift dive without accepting that he had a duty of care (SC 93/9). To balance this, the instructor in case SC 93/11 did everything possible when incapacity struck his charge. Incomplete opening of the tank valve was a significant part in two deaths (SC 93/3, SC 93/8) and in eight there was separation (SC 93/3, SC 93/4, SC 93/5, SC 93/7, SC 93/8, SC 93/9, SC 93/12, SC 93/13), while in one the victim was alone and making an apparently safe dive (SC 93/10). The problems inherent in deep diving were illustrated in case SC 93/6 where the factors of nitrogen narcosis, probable carbon dioxide retention (due to the extra breathing effort required

because of the performance characteristics of the regulator and his low usage of air, either of which would encourage carbon dioxide retention) and possible oxygen toxicity may have been involved. It is possible that nitrogen narcosis, cold, dark and stress influenced the response of at least one of his buddies.

Three bodies were not recovered, one shark attack victim (SC 93/7) and two who simply disappeared (SC 93/10, SC 93/13)

The story of the occurrence of CAGE in case SC93/3 was a recapitulation of text book descriptions, a rapid ascent while breath holding. However case SC93/6 was unconscious and not breathing before being given a rapid, unattended buoyant ascent from 13 m. That this was enough to cause some pulmonary barotrauma is uncertain. Reports describing the recovering of an unconscious diver from depth are rare. Rarer still has been any discussion of the risk of causing pulmonary barotrauma while bringing an unconscious diver to the surface using different procedures. Possibly the present trend to deeper diving makes it important to address this matter.

There will inevitably be discussion on the importance of an active asthma history in case SC93/4. While this man certainly had well controlled symptoms, in that his work colleagues were not aware of his condition, he was on regular medication to maintain his activity level. However in the circumstances of this death it should be noted that he was very inexperienced, at the surface in rough water and had become separated from his buddy. While it is not known whether he attempted to dive to follow his buddy, he was certainly aware that he was in a low-air situation. He failed to inflate his buoyancy vest or ditch his weight belt, either action might have saved him. Whether there was an element of uncharacteristic panic cannot be known. The part played by some respiratory impairment due to his asthma cannot be estimated but it was certainly not the only significant factor in his death.

Hose supplied divers are always dependent for survival on receiving an adequate and wholesome supply of air. In the double tragedy (H 93/1, H 93/2) a combination of circumstances led to fatal carbon monoxide poisoning. It is especially tragic because it would not have occurred had simple changes been made to the equipment. The shark attack (H 93/3) occurred in low visibility where plentiful edible debris induced a feeding frenzy among bait fish. The shark is assumed to have failed to identify the diver as such. The last case (H 93/4) is difficult to explain but any differential diagnosis would include cerebral arterial gas embolism consequent on a floating ascent while concentrating entirely on the problem of replacing a fin.

It is hoped that examination of these case reports will lead to an increased awareness of the factors which cannot be disregarded by those wishing to dive safely.

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Project Stickybeak

Anyone with any information is asked to contact the author at the address given below. Confidentiality is guaranteed for all correspondence. The identification of diving-related deaths is the vital first step, and one in which readers can greatly assist and so play a part, in attempts to improve diving safety.

Dr D G Walker is a foundation member of SPUMS. He has been gathering statistics about diving accidents and deaths since the early 1970s. He is the author of the series of Provisional Reports on Australian Diving-related Deaths which have been published in the Journal covering 1972 to 1992. His address is P.O. Box 120, Narrabeen, New South Wales 2101, Australia. Fax + 61-02-9970-6004.

DIVING MEDICAL CENTRE
SCUBA DIVING MEDICAL EXAMINER'S COURSE

A course for doctors on diving medicine, sufficient to meet the Queensland Government requirements for recreational scuba diver assessment (AS4005.1) will be held by the Diving Medical Centre in 1997 at

Royal North Shore Hospital,
Sydney, New South Wales,
7th-9th June 1997 (Queen's Birthday Long Weekend)

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

Information and application forms from
Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road
Jindalee, Queensland 4047
Telephone (07) 3376 1056
Fax (07) 3376 1056

THE WORLD AS IT IS

A PROFILE OF THE QUEENSLAND OCCUPATIONAL DIVING INDUSTRY

David Windsor

Key Words

Diving industry, diver numbers, occupational diving

Introduction

This paper has been assembled from information supplied by, or solicited from, Associations, Organisations, Government Department and individuals known to be engaged in diving activities. Draft sector profiles or complete drafts were circulated to organisations and individuals from all sectors of the diving industry and 43 written responses were received and collated. It was widely agreed that the draft profile reflected each industry sector, the sum of which comprise the full spectrum of the Occupational Diving Industry in Queensland.

What is occupational diving?

Occupational diving is the activity, by which many dissimilar types of work are conducted in a fluid, non-respirable environment at pressures greater than 1 atmosphere. For some people it is a full-time profession and for others an adjunct to their normal duties. Occupational diving activities in Queensland have been classified as the following sectors:

Aquaculture, fish collecting and harvesting;
Construction;
Scientific (research) diving
Recreational
Film, television and stills photography
Marine aquariums
General occupational

Diving is not defined as an industry under the Australian Standard Industrial Classification (ASIC). The numbers of divers employed in the various occupational diving activities are shown in Table 1.

TABLE 1

DIVING EMPLOYEES

Industry	Part time	Full time	Total
Aquaculture		5	5
Aquarium collectors	58	117	175
Beche-de-mer	300	80	380
Pearl shell	10	20	30
Crayfish	100	60	160
Construction	70	30	100
Scientific	570	30	600
Recreational	650	1,350	2,000
Film, TV and stills photography	50	20	70
Marine aquariums	20	30	50
Police	15	15	
Transport Department		8	8
Queensland Rail		6	6
Other		20	20
Total	**1,828**	**1,791**	**3,618**

Occupational diving training

There is currently a number of instruments which relate to training in the Occupational Diving industry

AS 2815 (parts 1, 2, 3 and 4) covers the training of all occupational divers using compressed air. These standards, when developed, were primarily designed for "commercial" divers and were not intended to apply to other types of occupational diving. That said, the majority of the curriculum of parts 1 and 2 is relevant to all forms of occupational diving, specifically with regard to the physiology and physics of diving.

The Australian Diver Accreditation Scheme (ADAS), is a scheme which results in the issue of diver certification to AS 2815 and the quality controls put in place by the scheme mean that the certification is recognised internationally.

However to be certified to AS 2815 a diver does not require certification under ADAS. Some training establishments, principally in New South Wales (NSW) issue their own certification. Workcover in NSW accepts these certifications for Occupational Diving.

There are no formal Standards for Occupational Diving training other than AS 2815.

In the Recreational Diving industry, instructors and divemasters/dive supervisors are trained to individual standards developed by training agencies whose incomes are derived from the training of these occupational divers. There are no controls on the standard or quality of training of employees other than those quality assurance (QA) programs developed by the agencies themselves.

Operational Procedures

Worksafe Australia and Standards Australia have jointly developed a Draft National Standard for all Occupational Diving. This was sent out for public comment in December 1993. This comment is currently being reviewed and a set of Common Essential Requirements (CERs) which apply to all Occupational Diving is being drawn up by Worksafe. Standards Australia will then formulate a number of industry sector-specific Standards which will address all issues not covered by the CERs. At the current rate of progress it is not anticipated these will be complete before 1999.

AQUACULTURE, FISH COLLECTING AND HARVESTING

Aquaculture

There are some 250 aquaculture operations in Queensland. They are oyster farming, prawn farming, freshwater crayfish farming, barramundi farming and perch farming. They do not employ divers except on a contract basis on infrequent occasions.

One industry which is a potentially large employer of divers in the future is open water cage fish culture. This industry will employ divers on a similar basis to the tuna and salmon farms of South Australia and Tasmania.

Diving is the main method for collecting pearls, trochus shell, beche-de-mer (sea cucumber), rock lobster and live fish for the aquarium market. The harvesting of these marine organisms is primarily authorised by the issue of a licence or permit under the Queensland Fisheries Act 1976-89 or in the case of the Torres Strait, by Commonwealth Legislation allowing fishing and collecting to be carried out commercially under community fishing rights with no licence required for a dinghy (one diver and one driver).

Aquarium Fish Collecting

There are 67 permits to collect aquarium fish issued by Queensland Fisheries Management Authority (QFMA) and a number of these operators also hold permits to collect coral. There are about 14 self-employed divers operating full-time and a further 53 operators who can employ up to 2 divers per permit dependant on demand and weather conditions. There are in addition a further 8 permits issued to individuals for the collection of coral. This indicates an absolute maximum of 175 divers employed in the industry.

There are no training standards or operational procedures currently documented for this industry sector and a large percentage of collectors operate singly and without surface support. Equipment used is almost exclusively surface supply "hookah" (a petrol driven air compressor, usually with a small reservoir, supplying air to the diver through a long hose). The industry is made up of self-employed persons who solo dive and who employ divers who are required to solo dive. It is the firm belief of this industry that they would become commercially unviable if any standard requiring diving teams was to be imposed on them.

Beche-de-Mer

In 1995 , an exceptional year, over 1,400 tonnes of Beche-de-Mer were collected. 1,150 tonnes were collected by hand from reef flats at low tide in the Torres Straits and the remaining 250 tonnes were collected by divers. Collections to date indicate that only some 450 tonnes will be collected in 1996.

There are between 12 and 15 licensed operators at any given time. Each of these operators can use up to 6 collectors at any time. The vast majority of Beche-de-Mer collection by these operators is done by hand collection or breath-hold diving. Currently (May 1996) up to 300 part-time and 80 full-time divers work in this industry.

There are no training standards or operational procedures currently documented and a large percentage of collectors operate singly and without surface support using hookah diving equipment. They hold similar views to Aquarium Collectors

Pearl shell

This is a small industry sector with only 7 active vessels employing 4 to 6 divers each. Diving methods used to collect shell is almost exclusively hookah with some breath-hold diving. There are no training standards or operational procedures currently documented for this industry sector. Normally hookah is used almost exclusively. They hold similar views to Aquarium Collectors.

Crayfish

Torres Strait Islanders can carry out traditional fishing for their own use and can work commercially without a permit if they are collecting individually without equipment from one dinghy. All other fishing is carried out from vessels holding permits.

There are up to 300 Islander people involved in commercial fishing for crayfish. Of these, 200 do not use equipment and the remainder use hookah. Approx 30-40% of these islanders collect on a full-time commercial basis the balance (60-70%) collect on an irregular basis

There are usually 16-20 vessels with permits and these carry an average of 3 divers each. Most divers provide and maintain their own equipment and travel to the work-site aboard a vessel holding a permit. This allows the vessel's owner to classify them as self-employed. Currently 4-5 of the operators directly employ divers who collect from their vessels. A total of 60 divers operate from vessels holding permits.

Diving methods used include scuba and hookah. There are no training standards or operational procedures currently documented for this industry sector. They hold similar views to Aquarium Collectors.

CONSTRUCTION

There are currently 10 companies employing full-time construction divers. A further 35 self-employed divers also actively seek construction diving work in the industry. The main centres for construction diving operations in Queensland are Brisbane, Cairns, Townsville, Mackay, the Whitsundays, and Gladstone. The construction industry undertakes various activities including, underwater photography, jack-hammering, cutting and welding, pipeline repairs, chain and block work, as well as surveys for Lloyd's Insurance.

The Maritime Union of Australia has advised that it has a membership of approximately 100 employees actively engaged in construction-related diving in Queensland. The largest operator is located in Brisbane and employs up to 10 full-time divers.

Construction divers are trained to various parts of Australian Standard AS 2815 and work to the requirements of AS 2299.

SCIENTIFIC (RESEARCH) DIVING

Research diving is centred in and around Townsville in North Queensland, which is an international centre for tropical marine research. 65% of Queensland research divers live and work in North Queensland with most of the diving activity taking place on the Great Barrier Reef (GBR). There are approximately 600 research divers employed in Queensland. However, only a small proportion of these divers are engaged in full-time diving activities. Approximately half of these, are employed in Queensland State workplaces, with the remaining half being Commonwealth employees who are covered by the Occupational Health and Safety (Commonwealth Employment) Act 1991.

Research diving is carried out for a range of purposes, from pure scientific research and underwater archaeological excavation to the monitoring and survey of natural and cultural resources. Underwater work in research diving can include simple observation, note-taking, underwater photo/videography, direct measurement, sampling, specimen/artefact collection and manipulation of marine biota. Some project work can involve the use of light power driven equipment or assembly of lightweight structures such as fish traps and survey grids

There are currently a number of Codes of Practice drawn up by individual sections of the scientific diving community. None has been adopted by any regulatory authority. Recently when "construction" type diving has been conducted this has been done to AS 2299 using correctly trained and certified divers.

A wide range of training standards are used within this diving sector, ranging from minimum recreational standards to AS 2815 certification

RECREATIONAL DIVING INDUSTRY

A number of diving activities are undertaken within this section of the industry. These range from recreational diving instruction, to underwater "memory" video production. Employees diving in the recreational workplace are recognised as occupational divers.

A recent study undertaken by the Great Barrier Reef Marine Park Authority[1] shows that, in round numbers, 1,900,000 tourists visit the GBR each year, 1,300,000 dives are conducted on the GBR each year, furthermore 130,000 resort courses are conducted and 36,500 people taught to dive each year. The industry contributes over $450,000,000 to the Queensland economy.

From a number of studies and from information provided by this industry, it is calculated that the total number of diving instructors in the recreational area is between 600-700, of whom approximately 450 are employed full-time. The remainder are employed on a regular part-time basis. It is accepted that for each full-time diving instructor there are 2 divemasters, making a total dive personnel of approximately 2,000.

Diving instructors and dive supervisors in the recreational workplace are mainly employed by owners of dive operations who also market training packages supplied and supervised by a number of training agencies. A number of dive supervisors, instructors and divemasters are also employed by companies operating resort islands such as Heron Island and Lizard Island Lodge. These would total in excess of 100 employees.

Occupational divers in the recreational sector are trained and certified to recreational diving standards established by a number of training agencies (this includes

memory videographers etc). Non-employees are trained to AS 4005 Part 1.

All recreational diving at a workplace in Queensland is covered by the Code of Practice for Recreational Diving and Snorkelling at a Workplace (under review).

FILM, TELEVISION AND STILLS PHOTOGRAPHY

The film and television industry is of major importance to the Queensland economy. The total value of all film production in Queensland is accepted to exceed $200,000,000. Overseas film companies are attracted to Queensland predominantly because of the ease and relatively low cost of production, combined with the existing infrastructure, the good weather conditions, and, where underwater filming is required, the proximity to the Great Barrier Reef.

At the last count there were five individual production/service companies working in Queensland on a full-time basis who employ 12 full-time divers. They are concerned with the production of feature films, film documentaries and advertising. Staffing levels vary when a production is underway. For example, an average of 5-10 major productions such as “Flipper” and “Ocean Girl” are shot each year in Queensland. Up to a maximum 30 divers could be used in these features films for periods ranging from 2 weeks to 6 months (of which about 30 days would be spent in the water).

In addition approximately 30 documentaries are shot each year and between 3 to 5 divers may be used for periods ranging from 1 day to 1-2 weeks on each production. The total value of under water film production in Queensland is reliably estimated at $20,000,000.

A number of interstate and international companies visit Queensland for the production of television series or productions, commercials, and documentaries. These companies usually employ divers from the Queensland companies as well as their own staff.

Queensland television stations occasionally use their own staff for underwater filming of news and current affairs type programs. The numbers of divers used are minimal

There are currently no training standards in place in this industry. Some employees are trained to the requirement of the relevant part of AS 2815 for the work being undertaken. Others rely on training to recreational standards. It should be noted however that on numerous occasions the recreational diver training agencies have recommended against this practice as they maintain their standards are not suitable for the training of occupational divers other than those undertaking training or supervision of recreational divers.

A Code of Practice has been developed by the Underwater Visual Producers Association of Australia (UVPAA) but this has neither been implemented nor recognised by any regulatory authority to date. Diving methods used include scuba and surface supplied breathing apparatus (SSBA) which includes hookah.

MARINE AQUARIUMS

There are a number of marine Aquariums in Queensland such as Sea World or Underwater World. They employ divers for a wide range of tasks including research, fish collecting, underwater maintenance and public appearances in glass fronted display tanks.

A wide range of diver certifications (from recreational to AS 2815 Part 2) and procedures are needed to suit the various job specifications and risk assessment. Approximately 30 full-time and 20 part-time divers are employed in this industry.

GENERAL OCCUPATIONAL DIVING

Police Diving

The role of police divers is predominantly for underwater search and recovery of objects ranging from small articles of jewellery to vehicles, vessels and aircraft. Police are also required to recover the bodies of deceased persons and conduct investigations for the coroner into diving related fatalities.

The 15 members of the Queensland Police Diving Squad are based in Brisbane, but can be called on to perform tasks in any body of water in the State. Police diving work is currently performed using scuba equipment and is conducted in accordance with AS 2299-1992.

Transport and storage

The Harbours and Marine Division of the Transport Department operates a navigational aids section. Eight divers are employed in a mobile capacity along the Queensland coastline. They construct and repair beacons and buoys and search for underwater obstructions. They work to the requirements of AS 2299.

Queensland Rail employs 6 divers as timber bridge carpenters to perform underwater inspection and repair of railway bridges. Although they are based in Townsville they form a mobile unit working throughout the State. They dive in accordance with AS 2299, and are qualified to AS 2815

Part 2. Queensland Rail also undertakes some diving work on a sub-contract basis.

Communications

Austel and Telecom employ contract divers to conduct underwater work from time to time. These divers are trained to various parts of Australian Standard AS 2815 and work to the requirements of AS 2299.

Electricity, gas and water

Divers are used in the water supply and treatment industry to inspect sewage treatment plants, water reservoirs and dams. The majority are not directly employed by local authorities but are hired on a contract basis.

The Gold Coast City Council employs 7 occupational divers. These divers are trained to various parts of Australian Standard AS 2815 and work to the requirements of AS 2299.

Other occupational diving

Many of the major tourism operators on the Great Barrier Reef employ occupational divers either directly or under sub-contract to provide diving services. These divers inspect moorings, and vessel hulls as well as undertake other occupational diving related activities. Other diving activities in this field include the collection of golf balls from dams and water courses on golf-links.

Acknowledgments

Unfortunately space is not available for individual recognition of the 29 organisations, 6 Trades Unions and 27 people the author would like to thank for their input. This report would not have been possible without their help.

References

1 Windsor D. A study into the number of dives conducted on the Great Barrier Reef in 1994. *SPUMS J* 1996; 26 (2): 72-74

David Windsor is Secretary of Dive Queensland, a dive tourism body. He serves on Standards Australia and Worksafe Australia occupational diving committees. His address is PO Box 19, The Grange, Queensland 4051, Australia. Phone 07-3856-0717. Fax 07-3856-0686. E-mail windsor@mailbox.uq.edu.au .

AUSTRALIA AND NEW ZEALAND HYPERBARIC MEDICINE GROUP (ANZHMG)

ADDENDUM TO STATEMENT ON THE USE OF HYPERBARIC OXYGEN THERAPY AT SITES OTHER THAN PUBLIC HOSPITALS (OCTOBER 1995).

PHYSICIAN REQUIREMENTS AND TRAINING

Key Words

Hyperbaric facilities, hyperbaric oxygen, qualifications, treatment.

Introduction

This addendum is designed to further elaborate the position of this group with regard to appropriate training and qualifications for physicians who intend to practice Hyperbaric Medicine.

The ANZHMG wishes first to state that, as presently constituted, it has no regulatory or accreditatory status with regard to hyperbaric facilities or the physicians who may staff them. This paper represents the consensus view of this group and is offered as such in response to a number of requests for advice on these matters. It is our understanding that those matters are currently under review by appropriate State and Federal authorities.

Appropriate training for medical officers in "non-comprehensive" hyperbaric facilities

The question has arisen as to the appropriate training for physicians who wish to work with hyperbaric facilities of a more limited nature than those 24-hour facilities currently operating in public hospitals. Such facilities are proposed by a number of groups and a small number are already in operation in Australia. Typically they propose to limit treatment pressures to two atmospheres absolute (2 ATA, 2 bar or 101 kPa gauge pressure) and to treat only uncomplicated and non-critically ill patients. Proponents of such facilities argue that because of their limited nature, full training, as described in section 4 of our statement of October 1995,[1] is both inappropriate and impractical.

The ANZHMG acknowledges this argument and intends in this addendum to outline our view of the minimum requirements with respect to physician training and expertise in relation to such proposed non-comprehensive hyperbaric facilities.

Our view is outlined in the full statement of October 1995 with respect to the technical, operator, attendant or other minimum requirements for the safe practice of Hyperbaric Medicine. Specifically, we maintain that any

compression of patients for therapeutic purposes must be at the prescription of a suitably trained and registered medical practitioner. This practitioner remains medically accountable for the selection of patients for therapy and any consequences of such therapy.

Minimum requirements for medical practitioners prescribing hyperbaric therapy

1 Fully-registered as a Medical Practitioner by the appropriate Medical Registration Board.

2. Have successfully completed an approved introductory course in Hyperbaric Medicine.

The ANZHMG would consider appropriate the curriculum of an individual course which:

- Involved at least 30 hours of instruction.
- Involved formal assessment on completion.
- Involved some element of practical, 'chamber-side' instruction during patient treatment.
- Covered all of the following topics:
  * Physics, anatomy and physiology of compression
  * Oxygen and carbon dioxide toxicity
  * Hyperbaric chamber types
  * Breathing systems
  * Infection control
  * Mechanisms of action
  * Selection of patients for hyperbaric oxygen therapy
  * Assessment of patients for compression
  * Assessment of progress and end-point
  * Scientific basis for indications
  * Compression chamber safety and emergency procedures
  * Contraindications
  * Complications and management
  * Literature review and key papers
  * Record keeping and Quality Assurance.

Minimum standards for limited hyperbaric facilities

The ANZHMG supports the operation of a "limited" hyperbaric facility to deliver safe and appropriate hyperbaric oxygen therapy (HBOT) under the following definition of such a facility:

A *limited hyperbaric medicine facility* means a designated area that:

a is equipped and staffed to provide hyperbaric oxygen therapy at no more than 2 ATA (2 bar or 101 kPa gauge pressure) to non-attended patients, or no more than 2.5 ATA (2.5 bar or 151 kPa gauge pressure) to patients accompanied by an appropriately trained attendant, with compression in air and patient oxygen delivered by a built-in breathing system.

b is supported by:
  1 Appropriately trained and qualified technical and medical staff as defined in the ANZHMG statement on the use of hyperbaric oxygen at sites other than public hospitals (Oct. 1995) and addenda. (Attendant staff may be required in a multi-place facility and will also be appropriately trained as defined in the statement.) Such staff will be responsible for the conduct of treatment and maintenance of the facility. A suitably qualified medical practitioner will be responsible for the prescription, review of therapeutic response and discontinuance of HBOT.
  2 A suitably qualified medical practitioner who is immediately available at all times during patient treatment and has demonstrated skills in resuscitation. Where resuscitation skills are provided by a separate immediately available practitioner, this second practitioner should have some familiarity with the concept, conduct and complications of HBOT.

c has defined admission and discharge policies which exclude the treatment of patients who are critically ill, in need of emergency medical attention or likely to suffer complications during or due to therapy.

d has appropriate emergency resuscitation equipment immediately available and adequately maintained.

e has a defined relationship between the facility and an appropriate "comprehensive" facility for the provision of technical and medical support. In particular, there will be a demonstrated system for obtaining expert hyperbaric medical advice when required. Such advice should be obtainable both urgently and electively prior to treatment.

Michael Bennett
Hon. Secretary, ANZHMG
July 1996

References

1 Bennett M. Australian and New Zealand Hyperbaric Medicine Group Statement on the use of hyperbaric oxygen therapy at sites other than public hospitals. *SPUMS J* 1995; 25(4): 206-208

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.

THE 1997 ANNUAL GENERAL MEETING OF SPUMS

will be held at 1200 on Saturday 19/4/97 at the Quality Resort Waitangi, Bay of Islands, New Zealand

The following motions to be moved at the Annual General Meeting have been received by the Secretary.

From the Committee

That Dr David Elliott be elected a Life Member.

That Rule 3 Life Members, (b) be altered by replacing the word *five* in the last sentence by the word *eight*.

The new sentence would read: *The number of life members shall at no time exceed eight nor shall more than one such member be elected in the one financial year.*

From Dr Jim Marwood

That Rule 8 Annual General Meeting, (e) be altered by removing the words *of which notice has been given*.

The new rule would read: *The annual general meeting may transact special business in accordance with these rules*.

That Rule 11 Order of business at general meetings, (a) be altered by adding a new sub-section *(x) Any other business*.

That rule 12 Notice of meetings, (b) be altered by replacing the second *the* by *a special meeting*.

The new rule would read: *No business other than that set out in the notice convening a special meeting shall be transacted at the meeting.*

OBITUARY

John Noel Miller
1938-1996

John Miller was a revered and a highly respected colleague. He was probably Australia's first internationally famous diving physician. He died on the 4th October 1996 at the age of 57 after a tragic but courageous battle with cancer. He left his wife, Kay, and children. He also left a painful void in my life.

John was younger and brighter than I. He graduated with an MB, BS from the University of Sydney in 1963. Right from the start he was equally interested in clinical medicine and research. His interests covered respiratory

physiology, cancer research and psychiatry. He received the Sydney University prize for his work in the Research and Treatment of Alcoholism.

In the three decades that followed I often sat with John, usually in some exotic locale, an alcoholic beverage or two on the table, and reminisced over his first major research establishment, the Royal Australian Navy School of Underwater Medicine. He had every reason to be proud of his project.

In 1965 the Royal Australian Navy School of Underwater Medicine was little more than a sparse library, with contributions being dependent on the charity of Rex Gray (the Officer-in-Charge) and the benevolence of the Medical Director General. When Geoff Bayliss took over the School he realised the need to legitimise its position as an authority in this field. This was achieved initially by developing an instructional role but in the long term its research capability was the area in which it became famous.

The research plan and facilities were almost entirely designed by John Miller. When I joined the School in 1967 it already had the nidus of a scientific unit. With minimal expenditure and the judicious selection of scientific equipment, it was ready to take off as a clinical research unit. That little research establishment was able to cope with a series of demands and challenges, the results of which put the School of Underwater Medicine on the international map.

And it was all designed by John. He formulated the plan, proposed the equipment and even recommended some of the earlier projects.

John moved from Sydney to Kings College Hospital Medical School in London, the Royal Naval Physiological Laboratories at Alverstoke and then the Virginia Mason Research Center in Seattle. It was during these appointments that John became known internationally for his contributions to diving medicine.

Partly because he still did not have an officially recognised specialty, and partly because of his continued interest in diving medicine, he moved to the Departments of Anesthesiology at Duke University and the University of North Carolina. He was responsible for the development of the Duke University flow chart which was for many years the worldwide standard for treatment of decompression sickness.

He then accepted a post as Professor and Chairman of the Department of Anesthesiology in Mobile at the University of Southern Alabama.

His administrative talents were well recognised. He was Chairman of the Program Committee of UHMS, and acted on the Constitution and By-Laws Committee, the Publications Committee, and the Executive Committee. He was a medical consultant to four commercial diving companies and Medical Director of the Experimental Diving Program at the F G Hall Laboratory and for the Diver Alert Network (DAN).

He recently completed editing a text on Lung Function and Diving, which should be published later this year. He was completing the text immediately before his death.

One of the honours which John treasured was his invitation to be Guest Speaker at the SPUMS conference at Palau Tioman, in 1980. This was the final acknowledgment, from his Australian colleagues, of his international status.

John was a gregarious, fun-loving enthusiast in everything that he did, from building and flying his beloved biplane, *Skybolt*, to social and professional activities. In the field of diving medicine he will be remembered as one of the very few who could combine research and clinical acumen. To me he will be remembered as a dear friend.

Carl Edmonds

SPUMS ANNUAL SCIENTIFIC MEETING 1997
PROVISIONAL PROGRAM (AS AT 23/2/97)

Workshops on board Tiger IV
(Departs 0700 and returns at 1730)

Sunday April 13th

Basic and Advanced Life Support revision sessions
CPR and intubation manikins, Computer Resusci-Annie
(Various instructors)
Oxygen First Aid equipment including Komesaroff semi-closed circuit and NZIG LSP resuscitator.
(Drew Richardson, Jeff Bertsch, David Komesaroff)
Aqua-Annie (Michal Kluger and Bob Ramsay)

Monday April 14th

Basic and Advanced Life Support revision sessions (continued)
Oxygen First Aid equipment (continued)
Aqua-Annie (continued)
Additional Basic First Aid principles
(Drew Richardson, Bill Day, Jeff Bertsch)
Poster presentations

Marine envenomation	Chris Acott
Diver identification devices	David Davies
Fluid therapy	Michal Kluger
Diving First Aid, does the diagnosis matter?	Mike Davis

Portable chambers and transfer under pressure
Des Gorman and David Youngblood
Analgesia for diving accidents David Komesaroff

Tuesday April 15th

Oxygen therapy equipment and manikins still available.
Diver Search and Rescue (Northland Emergency Services Trust representative, Mike Bennett, David Davies)
Posters on display (these will also be on display for the rest of the week in the Conference Centre)
Debate Chairman Des Gorman
"In-water oxygen recompression is a safe and proven procedure for use in remote areas"
For David Youngblood and Carl Edmonds
Against Chris Acott and James Francis

In the Conference Centre

Sunday April 13th
1900
****The biology of the Poor Knights Islands**. Wade Doak

Wednesday April 16th
1600-2000
Pathophysiology of Decompression Illness
Chairman Mike Davis
Pulmonary barotrauma: a new look at mechanisms
James Francis (**Accident Rehabilitation and Compensation Corporation Professor**)
A layman's guide to the vascular endothelium
Paul Langton
Mechanisms of cerebral injury in CAGE Des Gorman
Open chamber cardiac surgery: a clinical injury model of CAGE Simon Mitchell
Mechanisms of spinal cord injury in DCI James Francis
PFO and rapid onset/severe DCI: an update
Richard Moon

Thursday April 17th
1600-2000
Natural History of DCI Chairman Mike Bennett
Interpretation of gas in diving autopsies Chris Lawrence
Why divers die: a pathologist's view Rees Jones
DCI in recreational divers
UK experience James Francis
DAN USA experience Richard Moon
DES Australia experience Michal Kluger
NZ 1996 experience Simon Mitchell

Treatment of DCI. Part 1 Chairman Tony Slark
Clinical evaluation of the DCI patient Richard Moon
A scoring system for DCI severity Simon Mitchell
The origin of the recompression treatment tables
Chris Acott

** **These two lectures are presentations designed to be of interest to registrants and their families.**

Friday April 18th
0830-1630
Treatment of DCI. Part 2 Chairman Chris Acott
Recompression for DCI in recreational divers
Richard Moon
Panel discussion
"Is a consensus view on recompression procedures for DCI in recreational divers possible?"
Moon, Francis, Gorman and Mitchell
Adjuvant therapy for DCI Richard Moon
Pharmacology of lignocaine and NSAIDs: why might they have a role in DCI? Dave Cosh
The Slark Unit lignocaine trials: a progress report
Simon Mitchell
NSAIDs in DCI: a multi-centre study progress report
Mike Bennett
Critical incident stress debriefing Jeff Bertsch
Aftermath of recompression therapy: a case report
Peter Chapman-Smith
Lunch break

Other papers Chairman Guy Williams
Dive profile of a harbour diver (Poster) Prof Nashimoto
Tympanic membrane rupture in scuba divers
Noel Roydhouse
Diving and the lung Richard Moon
Immersion hypothermia in recreational diving
James Francis

Diving First Aid Workshop Chairman Des Gorman
First Aid teaching for recreational divers: what and why.
PADI Drew Richardson
SSI Bill Day
DAN Australia John Lippmann
The inadequacy of current First Aid James Francis
The GP's role in diving accident management
Peter Chapman-Smith
Evacuation methods in diving accidents Mike Bennett

1830
****The General Grant Expedition** Bill Day

Saturday April 19th
0830-1200
Diving First Aid Workshop Chairman Des Gorman
Oxygen therapy equipment: a theoretical overview
Mike Davis
Oxygen: how much is enough? Chris Acott
A proposed SPUMS protocol for First Aid Care of Diving Accidents Richard Moon

Formulation of SPUMS Policy on First Aid
Facilitator Des Gorman

1200
SPUMS ANNUAL GENERAL MEETING 1997
1730
Closing reception, entertainment and Maori Hangi.

LETTERS TO THE EDITOR

RECOMPRESSION FACILITIES AT PALAU AND CHUUK

Director Emergency Medical Services
US Naval Hospital, Okinawa
8/1/97

Dear Editor

I was very interested in the review of the recompression facilities in Palau and Chuuk by Dr Wong in the December 1996 issue. I was the senior Diving Medical Officer for the US Navy in Guam from 1990 through 1993, and treated many cases referred to our facility from these locations. The difficulties in arranging timely transportation and minimising delays to recompression were always accentuated by the remoteness and relatively primitive facilities available on these islands. It was most reassuring to hear that Palau now has a multi-place chamber, especially since their previous monoplace had proved to be somewhat unreliable during my tenure in Guam. Reviewing the statistics of diving accidents from 1993 to 1995, I suspect the apparent low number for 1993 may reflect that many DCI cases were still being referred to Guam for treatment.

I would like to correct one discrepancy regarding the chamber in Chuuk. This facility was being used in 1990, although it was used rarely since the assigned personnel had significant knowledge deficits regarding maintenance and proper recompression theory. I flew there, as an emergency, in October 1990 to treat a "decompensating" patient who had been undergoing a Table 4 that was discontinued when the chamber "ran out of oxygen". The US Navy sent myself, the Master Diver and 2 first class divers, with a supply of oxygen cylinders, to assist. Although still relatively new, the chamber was already in disrepair with improperly maintained compressors and leaking oxygen BIBS (built in breathing systems). The "operator" also believed that "bad air settles" and so switched oxygen cylinders whenever they were only half full. We spent one day doing maintenance and repair, the patient being quite stable and without overt signs of DCI on our arrival. I concur that it is a tragedy that this chamber remains unused, especially given the limit-defying profiles common in Chuuk, but until appropriately trained and knowledgeable operators are available it is safer for it to remain dormant.

My thanks to Dr Wong for providing an in-depth and timely update about Micronesia. It remains a divers' paradise and, with continued assistance, it will become a safer place for those of us who enjoy its waters and beauty.

William B Cogar LCDR, MC, USN

Key Words
Decompression illness, hyperbaric facilities, letter, treatment.

TRAVEL INSURANCE FOR DIVERS

201 Wickham Terrace
Brisbane
Queensland 4000
2/10/96

Dear Editor

Recently I went overseas with fifteen other divers to wreck dive at Vila and Santo. The dive company concerned advised members to take out travel insurance. While diving on the *SS President Coolidge* three experienced divers took electrically powered scooters to 69 m with a bottom time of 14 minutes. At approximately 0900, one of the divers (after ascending to 55 m) had difficulty breathing, became confused, took off his BC and tank, refused an octopus regulator and began convulsing followed by vomiting and coughing up blood. He remained unconscious and apparently ceased breathing.

He was taken to the surface over a period of approximately two minutes. His weight belt was dropped and his tank, BC and mask were left behind. No decompression was performed and the unconscious victim was given EAR on the surface. Breathing restarted in about two minutes. After assistance on the beach, the divers were placed in a utility vehicle and taken to the local hospital. No oxygen was available on the beach, as it was in the minibus picking up another group of divers. All three were treated with continuous oxygen and the near-drowned victim was treated with intravenous fluids, IV antibiotics and IV steroids.

Soon after I arrived at the hospital I rang the Hyperbaric Unit in Townsville and was informed that, with the permission of the insurance company, an appropriate aircraft would leave Townsville and arrive at Lugainville at midday local time. I next received a telephone call from Melbourne from a representative of the insurance company and medical recovery team requesting facsimile copies of the insurance certificates of all three victims plus my medical report. The representative was informed that I did not have access to the facsimile machine at the hospital and that the fax machine at the local hotel could not be operated by the staff on duty at that time.

The names, dates of birth and insurance numbers were passed to the representative, but this was insufficient to activate a recovery program and eventually the insurance documents were faxed to Melbourne. The matter was further complicated by one of the divers stating that his insurance was with an American company, Diver Alert Network (DAN), however he was also insured with the same Melbourne company as the other two divers. It was not until 1545 that I was informed that a pressurised aircraft would be leaving from Melbourne later that afternoon.

In the meantime a tele-conference was held with a representative of the insurance company and a doctor from the hyperbaric unit in Melbourne and a further tele-conference was held with DAN and their medical officer in the United States. As the airstrip at Santo normally closes at 1900, the acting medical superintendent drove to the airport to keep the control tower operational until the relief plane had arrived.

The medical team arrived at about 2345. The three divers, all on oxygen, were put in an ambulance where they waited for one hour as the medical relief team waited on a call from their Melbourne office. It was not until 0110 that the ambulance drove off to the airport. The time between the incident and the departure for Sydney was therefore about 16 hours. The reason they were sent to Sydney was due to the chamber nominated by the insurance company and the relief team.

The message to all divers outside Australia is that they should be careful to check with their insurance companies, before departure, as to where they will end up in the event of a diving accident. Obviously if one is diving in the northern Pacific region the closest chamber would be Townsville and the shortest distance for the aircraft to travel would also be Townsville or Cairns.

Fortunately all three divers recovered completely from decompression sickness as did the diver his near drowning. They returned to Brisbane, in a pressurised aircraft, four days after their admission to the hyperbaric unit in Sydney.

About two weeks after we left Santo two apparently experienced divers entered the SS President Coolidge at engine room level, became disorientated and were drowned. Their bodies were recovered the following day.

William Douglas

Key Words

Death, decompression illness, diving accident, legal and insurance, letter, rescue, transport.

DIVING FOR THE DISABLED

707 Oak Bluff Drive
Daphne
Alabama 36526, USA
8/1/97

Dear Editor

I would have to disagree with a number of comments made by Dr Marwood in his letter to the Editor.[1] First, "political correctness" has nothing to do with the struggle of minorities to find some measure of equality in our society. Secondly, those with physical disabilities have been among the last to seek redress from discrimination. This has been difficult because not only must we accept them as our human equals but in some cases we must make physical alterations in the environment to accommodate them.

Dr Marwood is concerned about a diver with a physical disability being paired with him or another able bodied diver in case a rescue is needed. At the Open Water level, even able bodied students are only taught basic rescue skills such as air sharing. This is a skill that must be performed adequately for anyone to receive certification. More difficult rescue procedures are reserved for advanced courses. Certainly, if one is paired with an individual, able bodied or otherwise, on a dive boat and there are reservations about that person's ability to perform the dive safely, then one has the responsibility to bring that to the attention of the divemaster. If one is uncomfortable being paired with a person with a physical disability, ask the divemaster to be paired with someone else.

The impression is given by Dr Marwood that paraplegics or double amputees are unsuitable buddies. What is overlooked is that these individuals, especially if they are wheelchair bound, often have tremendous upper body strength. Further, to go to the trouble of getting certified, they are usually extremely well motivated and have practiced their skills more diligently than the average dive student. The person who has a C-card but has not been diving for five years or the spouse who reluctantly took a scuba course would probably be a worse buddy than a disabled person who has kept in shape and dives regularly.

Dr Marwood suggests that a new level of qualification be recognised. Such a system has been adapted by the Handicapped Scuba Association (HSA) but it does not necessarily solve the problem. Consider those individuals who were fit to dive at the time of their training but subsequently developed a medical problem. It is safe to say that not everyone who has developed seizures, chronic obstructive airways disease, poorly controlled diabetes or even cardiovascular disease has thrown away their certification. In addition there is a growing group of divers who were certified and then sustained an illness or injury which led to a physical disability.

Certifying a physically disabled individual requires some extra work for everyone involved. It is important for the potential diver to discuss it with his physician. It is also important to search out a dive instructor who has expertise in training those with disabilities. Further, a discussion between the physician and instructor would be likely to prove beneficial.

As an instructor with HSA and a specialist in Physical Medicine and Rehabilitation, I can say unequivocally that there is no greater joy than seeing the sheer exultation on the face of a person who has left a wheelchair on the boat and experienced a freedom of movement that they had never thought to regain.

Terry J Brown

References

1 Marwood J. Diving for the Diabled. Letter to the Editor. *SPUMS J* 1996; 26 (4): 244-245

Key Words
Disabled, letter, training.

BOOK REVIEWS

BASIC DIVING PHYSICS AND APPLICATIONS
B R Wienke. Pp 320, illustrated, indexed.
Best Publishing Company, PO Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 14.95. Postage and packing extra.

Key Words
Book review, physiology and diving theory, mixed gas, thermal problems.

For those readers expecting a leisurely sojourn through basic diving physics, be afraid, as basic in the title refers to fundamental, and a run through the various chapters reveals a preponderance of mathematical formulae that would test even a 2nd year science graduate.

Bruce Wienke is a well known and respected scientist, who works in Los Alamos National Laboratory as Director of the Advanced Computing Laboratory. He has written extensively in the diving science literature, and is one of those people who seem to excel in all they attempt. He advises to DAN, Scubapro, owns a dive store, is a dive instructor trainer and national ski racing coach. However, as sometimes happens, such gifted people fail to recognise the intellectual limitations of others! This book unfortunately is a case in point.

The aim of the book, as stated by the author, is to target the dive instructor, hyperbaric technician, doctor, physiologist, chemist, and engineer amongst others. The author assumes that readers will know a large amount of theory before they read this book. Perhaps in the United States these groups have a similar exposure to physics. But this is not true of Australasia where these occupations are educationally diverse.

As a monograph the text tends to have a uniform style, has a few line illustrations, and many boxed examples. The initial chapters look at mechanical interactions, thermal interactions, pressure and density effects, gas and fluid kinetics, dissolved phase transfer, and free phase transfer, as an introduction to the following sections on compression and decompression, diving protocols and mixed breathing gases. The final sections on electromagnetic interactions, biophysics and modelling issues, statistics, geophysical and marine phenomena and supercomputers are included to illustrate the scope of mechanisms that allow the various mathematical models of bubble formation and DCI risk to be formulated.

The sections on the decompression models and diving protocols are by far the easiest to read, and relevant to the majority of interested divers and doctors. This section is pre-empted by the chapter on free and dissolved phase transfer which nicely clarifies diffusion and perfusion limited models, nucleation, cavitation and micronuclei. Having said this, there are many other diving textbooks which also describe these topics and Wienke's book does not offer any advantage over these standard texts. Although meant to be a book on basic diving physics, the author digresses into all fields of science. The section on compression and decompression includes "maladies", inert gas narcosis, pulmonary oedema etc. These are dealt with in a basic way, are simplistic and are out of place in such a book.

This book may serve as a valuable research reference text to those interested in fundamental diving physics, but its emphasis on mathematical derivations, imaginary numbers, partial differentials, operators, and other advanced mathematical descriptors mean that its readability is far from universal. Whilst there are a few titbits of information in general reading, the text fails to flow freely, is too specialised and lacks appeal to the majority of the target audience.

Michal Kluger

ARE ASTHMATICS FIT TO DIVE ?

Editor David H Elliott
Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Maryland 20895, USA. 1996.
RRP $US 20.00 plus postage and packing

Key Words
Asthma, book review, fitness to dive.

This report of the 1995 meeting in the FIT TO DIVE series run by the Undersea and Hyperbaric Medical Society (UHMS) shows that there has been a radical change in the attitudes of many of the members of the diving medicine establishment. This book should be read and digested by all diving doctors. It shows a wide range of views about the safety of diving with a history of asthma and active asthma. Unfortunately facts supporting this spectrum are few and far between.

Many speakers made the point that although common sense, logic and conventional wisdom all agree that asthma constitutes a serious and inescapable threat to the survival when scuba diving, there was a significant absence of documentation to support this belief. This was not to say that the condition was not able to adversely influence survival but it did indicate that hard and fast rules were inappropriate.

A few years ago few if any of the speakers would have said openly what they said here. There was even an absence of dissent when Dr Farrell stated that asthmatics should be tested when taking their regular medication (inhaled steroids) when estimating whether they could be accepted as fit to dive. There are two serious problems with this proposal, the diver cannot be monitored to ensure a perfect medication routine and the examining doctor might have trouble convincing a Court that the diver fully understood the potential risk he or she was accepting. However, any certificate which stated that a person has successfully completed a course of training could be similarly critically assessed. It is a pity that so many doctors have been eager to claim infallibility for their advice, and that so many lawyers are eager to deny that ultimately every person should accept at least some responsibility for their own actions. Particularly if they fail to follow advice.

There was general agreement that there is a lamentable lack of data on this subject. Dr Gorman informed the meeting that SPUMS was committed to developing a database even if others hung back. In response there was a commitment by Dr Moon on behalf of DAN and by Dr Farrell, the latter reporting a prospective study. There was, however, no actual agreement to set up such a databank. There was also the unresolved problem as to whether doctors should cease stating that anyone was Fit to Dive and use the wording that no medically adverse facts had been discovered, or that certain adverse factors had been identified and discussed and the following advice had been given. A real can of worms ! As was noted by Dr Elliott, rules come in black and white, but people come in shades of gray. This was a thoroughly useful workshop.

Douglas Walker

VENOMOUS AND POISONOUS MARINE ANIMALS: A MEDICAL AND BIOLOGICAL HANDBOOK.

Edited by John Williamson, Peter J Fenner, Joseph W Burnett and Jacqueline F Rifkin
University of New South Wales Press, Sydney, New South Wales, Australia.
ISBN 0 86840 279 6. 1996. 504 pages and 80 pages of colour plates. RRP $ 130.

Key Words
Book review, first aid, injuries, marine animals, toxins, treatment.

This is an impressive and erudite publication. The University of New South Wales Press calls this *a medical and biological handbook* which the Macquarie Dictionary defines as *a small book or treatise serving for guidance*. This weighs in at nearly four kilos! The editors have been far too modest. Although descended from the Marine Stinger Book (reviewed in *SPUMS J* 1991; 21 (2): 91) it is now quite definitely a textbook on jellyfish and other marine creatures which envenomate and poison humans. The colour plates are of high quality and excellently reproduced.

As with any multi-author book the writing is uneven and the same information is repeated in different sections. Tighter control by the authors would have avoided some verbosity and unnecessary repetition.

Immediately after the Foreword, by Struan Sutherland, is *In Case of Emergency*, an alphabetical list of the first aid treatment for specific species and then their medical treatments. Throughout the book the emphasis is on first aid treatment with medical treatment of the emergency in close pursuit.

A large proportion of the book (270 pages) is devoted to detailed study of jellyfish, their taxonomy, their stings and the appropriate treatment. To the average medico who dives and is interested in treating marine injuries this section is fascinating. One has to learn a new vocabulary to follow the relationships of the various cnidarians, the animals which have nematocysts. Dr Rifkin has written clearly and informatively, even if at times the italicised names overwhelm one. Luckily only a few jellyfish are known to have caused deaths, but the only reliable statistics about stings come from Queensland and the Northern Territory and there the availability of *Chironex fleckeri*

antivenom has prevented deaths in recent years. Throughout the book there is emphasis on the need for more knowledge about the various animals which sting. As it is, anyone stung on the New South Wales coast is likely to be diagnosed as stung by *Physalia*, simply because it is the commonly seen "stinger" and most jellyfish are difficult to identify even if they are seen. Mistaking the stinger could be the cause of the cases of "*Physalia*" stings which end up in Sydney hospitals with cardiovascular collapse.

The editors are very cautious about accepting reports of deaths, a number of these are rejected as "unconfirmed". As most venomous and poisonous marine animals occur in the tropics, where on the whole the medical services are not as well organised as in the Western World, which includes Australia, it is unlikely that reporting will be accurate or full. Third world countries are more accepting of early mortality and more fatalistic than Australia. However there are reports, mostly "unconfirmed" of jellyfish mortality in this book. The editors repeatedly emphasise the need for better reports and reliable statistics.

Echinoderms, molluscs and sponges get 40 pages and venomous vertebrates 58 pages. This section includes poisoning by eating marine creatures. Paralytic shellfish poisoning leads the field. Filter feeders concentrate saxitoxin in the dinoflagellates they feed on and humans who eat the shellfish get an effective dose which may kill. Ciguatera, caused by the concentration of another dinoflagellate toxin as bigger fish eat smaller fish, gets adequate advice for the early medical treatment. While in Australia deaths from ciguatera poisoning have not been confirmed there is a steady stream of people whose lives are drastically affected. However the physician looking for advice on the treatment of long continuing (longer than a fortnight) symptoms after ciguatera poisoning will find no help. In 1979 the SPUMS ASM was held in Port Vila in the New Hebrides (now Vanuatu). Dr Bowden of the British Hospital delivered a paper on Fish Poisoning which emphasised the vast range of presenting symptoms and mentioned at least one patient who died within two days from the consequences of his paralysis. Tetrodotoxin, found in puffer fish and blue ringed octopus, produces paralysis and requires artificial ventilation for some hours while the poison wears off. The editors correctly remind their readers that tetrodotoxin poisoned people, who have fixed dilated pupils and are not breathing have unimpaired consciousness if they are not anoxic and that this must be borne in mind. The reviewer is disappointed at the brief treatment of the third major fish poisoning, scombroid poisoning, but that is because he has suffered from it and remembers that it took much prednisolone and over two weeks to get back to normal.

Sea snakes and their venoms get brief mention, compared with jellyfish, although sea snake envenomation of fishermen in tropical waters has been known to be a problem for about 30 years and many have died.

A section on *International Toxic Marine Animal Occurrences* visits the Gulf of Oman, Portugal, and Japan. Once again it is clear that most people who are stung or envenomated do not recognise the animal which caused their pain and suffering.

Marine antivenoms, scuba diving injuries, mystery syndromes possibly associated with marine envenomation and research almost complete the list of contents. The International Consortium for Jellyfish Stings owes its existence to the editors co-operation and is a prime mover in attempting to clarify the picture, clouded as it is with many families of animals most of whom are invisible in the water.

The final chapter is about Platypus envenomation. Not really a marine animal, but certainly a very interesting one, which confirms that the energy behind this book comes from Australia.

One minor irritation is that the colour photographs are bound together near the middle of the book and not inserted in the chapters they illustrate. This leads to a lot of thumbing back and forth. As all the left hand colour pages are headed THE MARINE STINGERS REFERENCE BOOK it may be that they are to be used in another publication, which would explain them being bound together. However in such an important book, at such a price, the publishers have been penny pinching by not binding the colour plates in the chapters they illustrate.

The index should be upgraded for the next edition to include bold type for major headings. The present layout with all entries in the same sized type, with the heading only distinguishable by the fact that there is no page number against it and some inset entries below it, is confusing. The index is not user friendly. Looking up *Carukia barnesi* (see also Irukandji and Irukandji syndrome) produces a different set of page numbers from either of its "see alsos". A more serious complaint is that some of the entries in the index, e.g. *Chimaeridae* (Family), ecology, hot packs, hyperventilation and hypothermia, do not have any page numbers. These 5 examples came from five pages, about 1,400 entries.

Although the editors say that they want to attract the first aider and the medico wanting advice, it is the reviewer's opinion that the level of information provided is much above the needs of a first aider, as is the price. The third group of readers, those with a cosmopolitan appetite, will find the book of great interest.

This book should be in every medical library and in the consulting room of all doctors with seaside practices. The editors and contributors are to be congratulated on their hard work.

John Knight

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5TH ANNUAL SCIENTIFIC MEETING ON DIVING AND HYPERBARIC MEDICINE

Holiday Inn, Coogee Beach, New South Wales
August 29th and 30th 1997
will be hosted by
the Hyperbaric Medicine Unit, Prince of Wales Hospital,
on behalf of
the Hyperbaric Technicians and Nurses Association (HTNA) in conjunction with
the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) and
the Submarine and Underwater Medicine Unit (SUMU)
Royal Australian Navy

Friday August 29th will be devoted to hyperbaric medicine. Saturday August 30th will focus on diving related subjects. Drs Carl Edmonds and Des Gorman, well known specialists in diving medicine, are among an impressive list of speakers.

For further details contact

John Kershler or
Michael Talty, Conference Co-ordinator
Department of Diving and Hyperbaric Medicine
Prince of Wales Hospital
High Street, Randwick, New South Wales 2036
Phone (02)-9382-3881
Fax (02)-9382-3882

SPUMS ANNUAL SCIENTIFIC MEETING 1996

REBREATHERS : AN INTRODUCTION

David Elliott

Key Words
Equipment, mixed gas, nitrogen, oxygen, rebreathing.

Introduction

Recreational divers with money to spend turn to the latest technology. Current magazines for divers have numerous articles on rebreathers for deep cave and wreck exploration. Other rebreathers for use with oxy-nitrogen at shallower depths are available for the less adventurous diver. Although some may incorporate electronic sensors and controls, the basic principles of today's rebreather are the same as when they were first used, more than 100 years ago.

In 1880 a railway tunnel being constructed under the river Severn became flooded and attempts to shut an open sluice were unsuccessful. Standard divers were unable to get there because it was too difficult to drag 1,200 feet (364 m) of air hose that far. Mr Henry Fleuss of Siebe Gorman volunteered to try and reach it. Two years previously Henry Fleuss had designed and made the first self-contained rebreather.[1] His equipment included a copper cylinder, which was charged to 30 atmospheres with oxygen, and a scrubber which contained string soaked in caustic soda. Oxygen was let into the breathing bag "when needed". After several attempts to reach the open sluice, each of an hour or more duration, with the foreman of the labourers as his attendant, he decided to send the diver Alexander Lambert, because he was more familiar with the workings in the tunnel. On his second attempt Lambert managed to move some railway lines out of the way and completed the task of closing the sluice. His total dive on oxygen was 90 minutes at a depth of 40 feet (12 m; PO_2 2.2 bar). That railway tunnel between Bristol and Cardiff is still in use today.

A glance through the pages of Deep Diving and Submarine Operations [1] reveals that in the next 30 years or so there was a variety of designs for closed circuit rebreathers, including the use of sodium peroxide to both absorb CO_2 and generate oxygen, a principle still used in some coal mine escape apparatus. In 1912 a self-contained suit with a rigid helmet was devised for use with 50/50 oxy-nitrogen at 5 litres (surface equivalent) flow through an injector that also, on the venturi principle, sucked the helmet gas through the CO_2 absorber.

Subsequently it was the military in World War II[2] who developed closed circuit oxygen rebreather techniques for covert operations and also, at depths down to 55 m (180 ft), used oxy-nitrogen semi-closed circuit apparatus for acoustic mine clearance because of its low bubble noise.

In the early 1970's, to conserve expensive helium, a semi-closed rebreather was developed to use pre-mixes of helium, containing oxygen at less than 20%, for use out of a diving bell at depths below 50 m. Its operational use was limited by the need to ensure oxygen levels within the breathing bag that were neither hypoxic nor hyperoxic both at rest and when hard at work. To achieve this the flow rates of pre-mix had to be increased to a level that the set was no longer competitive vis-a-vis open circuit apparatus.

Meanwhile, largely inspired by the US Navy's SEALAB program, oxygen sensors had been introduced, enabling the rebreather to be developed as a closed circuit rig at depths at, and deeper than, the limit of semi-closed technology.

An introduction to rebreathers needs first to clarify the different categories of rebreathers (Fig. 1) and to consider their merits and disadvantages of their different gas flow systems. These and other important aspects of breathing apparatus performance and design are discussed in greater detail elsewhere.[3]

Closed circuit oxygen

This type of apparatus has a carbon dioxide scrubber and a simple counterlung or breathing bag which is full of oxygen from which the diver breathes. As the oxygen is consumed so more oxygen needs to be released into the breathing bag from the cylinder carried by the diver.

In Henry Fleuss' apparatus the oxygen was supplied "on demand" and replenished when the diver thought that the rebreathing bag was getting low. This is a dangerous procedure because, during the dive, dissolved nitrogen is being washed out of the body into the rebreather's closed system. As the bag diminishes in volume with the consumption of its oxygen, nitrogen comprises an increasing percentage of the bag's content. Unless more oxygen is released into it in good time, the point could be reached when the counterlung provides the diver with a hypoxic mix. Hypoxia is usually associated with a CO_2 build up but, with a CO_2 scrubber in the circuit, the diver could be quite unaware of the changes of inspiratory gas composition. The diver may pass gently into unconsciousness due to "dilution hypoxia" and death is likely to follow. This may happen at depth but can be precipitated by the fall of PO_2 occurring during ascent.

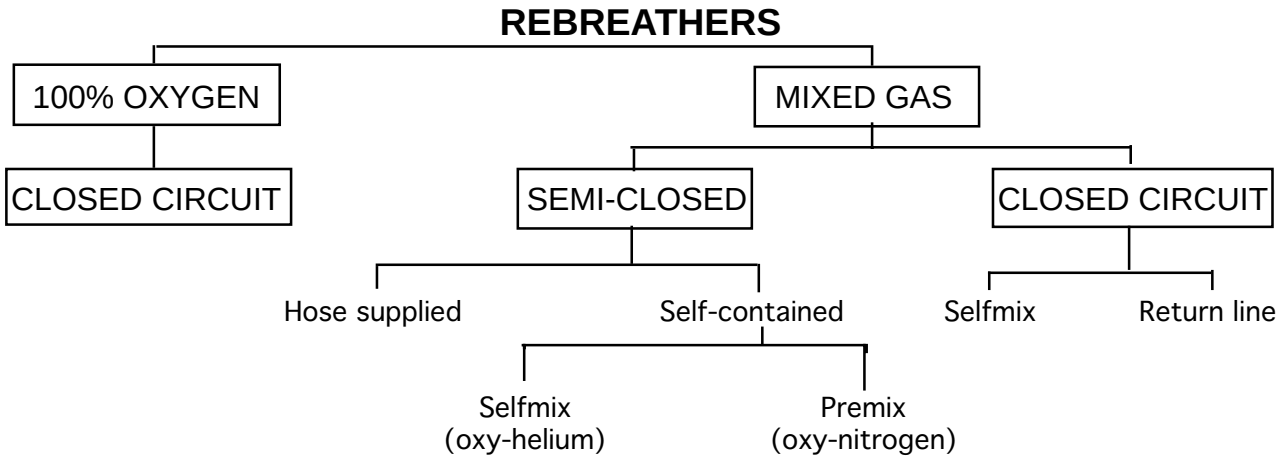

Figure 1. A classification of rebreathers.

To avoid this, an oxygen diver breathes for 2 minutes from the breathing bag while at the surface and then empties it and its contained nitrogen before recharging the counterlung with pure oxygen. Then the descent can begin. This one "nitrogen wash out" is sufficient for a 90-minute dive using a typical naval closed circuit rebreather.

Because breathing "on demand" can be particularly hazardous it is generally reserved for combat situations in which a lack of bubbles from the diver is essential. At all other times the diver is provided with a constant flow of oxygen into his breathing bag at a basic rate, usually a constant surface equivalent volume (constant mass) of 1.5 litres per minute ($l.min^{-1}$). This is achieved by an acoustic reducer, a tube within a brass plug which has the orifice engineered to allow only a relatively small mass flow of oxygen molecules through it. This flow rate reaches the speed of sound which means that, given a constant pressure of supply to the reducer, the flow remains constant regardless of the environmental pressure of the diver.

In spite of this constant mass flow, dilution hypoxia can still be a hazard. If, for instance, the oxygen bottles have perhaps leaked and they empty earlier than expected, the diver may be unaware that his counterlung contains an diminishing percentage of oxygen as he heads towards an anoxic death. Another problem can occur if the working or swimming diver exceeds 1.5 $l.min^{-1}$ without noticing that the bag is getting smaller. The diver is "beating the flow" and could be heading for hypoxia, especially if he fails to flush the counterlung with fresh oxygen before beginning the ascent with its associated drop in oxygen partial pressure.

As Alexander Lambert showed, this type of self-contained apparatus works, but the early oxygen divers were probably not fully aware of the many hazards to which they were exposed. As the particular hazards of hypoxia, hyperoxia, hypercarbia and "soda lime cocktail" became more recognised, rebreather designs and diving procedures were developed to reduce these risks. The hazards of oxygen neurotoxicity were recognised by naval authorities and so, notwithstanding the use of oxygen by some combat swimmers for short periods at greater depths, the use of oxygen sets was limited to 25 ft (7.6 m; PO_2 1.8 bar) when swimming with fins.

Semi-closed mixed gas

Of the many varieties of semi-closed rebreathers that have been made available to the diver, the designs most close to those now being introduced to recreational diving are those using an oxygen-rich nitrox pre-mix. These were developed from the closed circuit oxygen sets by the navies who needed them for defusing acoustic mines. The other types of semi-closed rebreather need to be mentioned briefly for clarification and completeness.

HOSE-SUPPLIED

The first semi-closed rebreather with a hose was a modification of the deep standard divers' open-circuit oxy-helium helmet. Within the large semi-rigid system of the helmet and associated dry suit there was no need to have a counterlung in the circuit. A certain amount of rebreathing occurred within the regular helmet which was supplied from the surface at a predetermined flow rate, sufficient to minimise the carbon dioxide build-up. In the deeper versions of the oxy-helium helmet, the constant flow of fresh gas to the diver was fed through a venturi which caused a proportion of the gas in the helmet to be recirculated through a soda-lime canister. This apparatus was used until the early 1970s.

Quite different is the commercial breathing apparatus which uses a hose to provide oxy-helium to a semi-

closed set with a counterlung. This, in its basic design principles, is very similar to the shallower oxy-nitrogen sets which require less gas and so do not require a hose. This oxy-helium semi-closed set necessarily uses oxygen percentages of less than 21% and so is used not from the surface but from a diving bell. To allow for the varying levels of oxygen consumption within a specific depth range, the flow rates have to be relatively high. This means that, at around 150 to 200 m, these semi-closed sets no longer provided cost savings over open-circuit oxy-helium demand breathing apparatus. Nevertheless there are several locations where these units are still operational.

SELF-CONTAINED SELF-MIX (OXY-HELIUM)

There is a semi-closed set which was developed for the Royal Swedish Navy and which is a "self-mix" unit. It has a constant oxygen flow rate and a separate helium supply which is increased with depth. With such a breathing apparatus the potential problems again relate to the varying need for oxygen during a dive. There is also a Canadian semi-closed breathing apparatus which is said to deliver a constant partial pressure of oxygen to a depth of 95 m but no technical reports have been reviewed and, as with any new apparatus, one would want to see rigorous manned testing at high work levels with oxygen monitoring before accepting it.

SELF-CONTAINED PREMIX (OXY-NITROGEN)

At shallow depths, in contrast to the hose-supplied and self-contained oxy-helium semi-closed rebreathers which need to have less than 21% oxygen at depth, the oxy-nitrogen semi-closed rebreathers use a pre-mixed gas of oxygen enriched air. The basic principles are very similar to those of the oxygen apparatus already described and, with a CO_2 scrubber in the system, require a constant flow into the breathing bag of a gas with a known O_2% and at a pre-determined rate.

The flow rate needs to be pre-set and there are two ways in which this can be done. The simplest is to set a sonic reducer, similar to that used for 100% oxygen flow, but at a higher set flow according to the mixture used. This can be illustrated by naval clearance divers breathing apparatus (CDBA) when used to its maximum operational depth of 180 feet (55 m) using 32.5% oxygen mix. The *constant mass flow* rate is set at 13 $l.min^{-1}$ and, for oxygen consumptions ranging from 0.25 to 2.5 litres per minute, this provides an oxygen PO_2 in the counterlung between 0.21 and 2.0 bar. This high partial pressure of oxygen will occur when the diver is at rest and was accepted at that time for operational use. It has since been reduced by defining a shallower maximum depth of use. In some circumstances, there can also be a risk from hypoxia due to an oxygen percentage which has provided an adequate partial pressure while at maximum depth, but which may be insufficient to maintain consciousness as the diver reduces his ambient pressure during ascent. So, for additional safety as when using closed circuit apparatus, the diver empties the counterlung and refills it before commencing the ascent. Although this procedure may raise the inspired PO_2 slightly above 2.0 bar, this is only transient and was, on balance, considered safer than the risk of hypoxia during ascent.

Equally ingenious, but slightly more complex to use, is the *constant ratio* semi-closed circuit principle.[4] This device was first used by the French Navy and is based on designing the breathing bag as a bellows system. Within the counterlung is a separate smaller (1:11) concertina bellows, the slave, which follows the movements of the main breathing bag precisely. Thus each of the bellows are filled at the same time when the diver exhales but, when he inhales from the main compartment, the contents of the slave (one eleventh of the previous exhalation) are discharged into the sea. The larger of the bellows is fed on demand from the pre-mix gas supply. Unlike the constant mass flow supplied semi-closed sets, the constant ratio set has a diminished endurance because of increased gas usage at increased depths. The calculation of inspired oxygen percentage is slightly more complex requiring also knowledge of the diver's ventilation coefficient: the relationship between oxygen consumption and minute volume, a relationship that could be different in those divers who are "CO_2 retainers". The mathematical and physiological principles are outlined elsewhere.[4] There seems to be no recreational application of this principle, yet.

The majority of the nitrox semi-closed breathing apparatus now being marketed to the recreational diver is based on a pre-mixed gas delivered by constant mass flow. They use the same principles already described for military use but avoid the CO_2 problems arising from the large dead space of pendulum breathing by having an inhalation and an exhalation hose, with the CO_2 scrubber in the loop circuit. Nevertheless, the selection of a percentage of oxygen for a particular depth range and of a constant mass flow rate for it inevitably leads to a compromise between the conflicting physiological needs of avoiding hyperoxia and hypoxia at different work rates. The biggest unknown, and a cause for concern if hypoxia is to be avoided, is the range of oxygen consumption to be encountered during the dive, particularly if, for a minute or two, it is necessary for the diver to expend maximum physical effort.

Thus, diving with semi-closed rebreathers introduces several hazards which are not encountered by those diving on open-circuit compressed air scuba. The potential consequences include dilution hypoxia, hyperoxia, hypercarbia and "soda-lime cocktail". The degree of risk to the diver from these hazards will be modified by the design, flow rates and other parameters of the particular set used and by the procedures which the diver should be

taught. These procedures are due to be considered in a later paper.

Closed-circuit mixed-gas

The self-contained and the hose-supplied closed-circuit mixed-gas apparatus are both closed circuit but are totally different designs. They share only the concept of recirculating the exhaled gas as a pragmatic response to the high cost of the helium which would be wasted if the diver were using an open-circuit demand breathing apparatus.

RETURN LINE CLOSED-CIRCUIT

Dating from the 1960s, the concept of a bell-mounted closed-circuit system for hose divers has proved attractive. The gas is supplied by hose from the bell to the diver on demand and after exhalation is returned through a necessary exhaust regulator and valve by a parallel hose to the bell where the CO_2 is scrubbed and the O_2 replenished. These "push-pull" systems lost their commercial battle to similar systems mounted not on the bell but at the surface. In the deck-mounted systems, a return line takes the exhaust gases from the diver via the bell to the system on board the diving support vessel where it is purified and returned to high pressure tanks for re-use. One reason for lengthening the circuit to the surface was a need for easy access by the deck crew to the system for maintenance. An advantage of these extended closed-circuit systems is that the diver breathes from a conventional demand valve during the dive and, provided the technology does not fail, should not be at risk from hypoxia, hyperoxia or hypercarbia.

SELFMIX CLOSED-CIRCUIT

Many versions of self-contained closed-circuit mixed-gas rebreathers have been developed in the past 25 years. They "self-mix" the respiratory gas from two gas bottles, one of inert gas, usually helium, and the other of pure oxygen. With continuously improving technology the earlier need to have an electronics engineer on hand to keep it going has given way to a remarkable reliability. A constant partial pressure of oxygen, around 0.7 bar, can be monitored by sensors and maintained at any depth. Duration is limited only by the capacities of the gas supply bottles and the duration of the scrubbing system. This type of apparatus has good breathing characteristics in the water and should maintain the inspired gas within defined physiological limits.

In the North Sea every diver must have a reserve "bail-out" gas supply so that he can get back to the bell if his primary breathing apparatus fails. In fact the duration of any diver-carried open-circuit system is likely to be limited to only a minute or two at great depths. In consequence self contained closed-circuit systems, such as the Rexnord, have been provided as bail-out systems for hose-supplied divers.

For the recreational diver a closed-circuit apparatus provides extended duration at any depth without the need to carry large volumes of gas. Reliable sets should provide reasonably warm breathing gas and few problems. These sets should be physiologically as safe as one could wish and only if the technology fails would the diver be exposed to the hazards of hypoxia, hyperoxia or hypercarbia. Only problems like the high pressure nervous syndrome (HPNS) and safe decompression, which are unconnected with the breathing apparatus, will limit their potential at the deeper recreational depths.

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TOLERATING OXYGEN EXPOSURE

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Key Words
Hyperbaric oxygen, oxygen, physiology, unconscious.

The fact that oxygen can be toxic is well known to divers, especially those practising the use of advanced techniques involving special breathing gases. Here oxygen tolerance techniques may be optimised in order to allow more efficient decompressions. Oxygen's toxicity is also a well recognised problem among the medical community, but in the latter case toxicity management techniques are not intended to be optimal or especially efficient; here the objective is to avoid making oxygen exposure, for the patient who needs it, become part of the problem.[1] This paper discusses some of the optimisation techniques.

The exact mechanisms of oxygen toxicity are gradually being worked out, but there is yet much to be learned.[2] We are notconcerned here with the mechanisms, because the methods of controlling oxygen exposure and of tolerating exposure to oxygen rely on empirical information rather than fundamental biochemistry or mathematical modelling. Although the mechanism at the cellular level is probably the same, we are concerned about two general manifestations of oxygen toxicity. For both of these categories, empirically derived procedures for use in managing exposure have been developed.

As this audience knows quite well the two categories of oxygen exposure are the toxicity that manifests itself in the central nervous system (CNS), and the whole-body or pulmonary toxicity. The two types of toxicity are distinct not only in their anatomical manifestations but also because of the "dose" of oxygen exposure required. CNS toxicity generally requires exposure to a level above about 1.6 bar (or atmospheres absolute) and may need only a few minutes exposure, while exposures for longer durations, hours or days, above about 0.5 bar may cause whole body toxicity. Management of both types of toxicity consists primarily of controlling the exposure and current procedures are entirely empirical. Other toxicities, such as to the eye, require longer and more intensive exposures than the two under consideration.

CNS toxicity

CNS oxygen toxicity may generally be seen as unconsciousness or incapacitation or may come on as a full blown epileptic-like convulsion. Lesser symptoms are important as warning signals but are not likely to be incapacitating. The convulsion itself is not particularly harmful, but the consequences of having a convulsion can be, especially for a diver in the water. People are occasionally injured when they convulse in a chamber. It is quite common to bite the tongue sometimes causing bleeding; this can be a misleading symptom in a rescue. In the case of a diver a convulsion underwater is extremely threatening because it can lead to drowning; one of the first reactions is an expulsive movement of the tongue, which will cause a mouthpiece to be spat out. For this reason divers pushing the oxygen exposure limits are well advised to wear a full-face mask or helmet to prevent loss of access to the breathing gas.

CNS toxicity requires a high level of oxygen exposure, and may occur after as little as a few minutes of exposure. Measured as partial pressure, the exposure level for CNS toxicity requires more than about 1.6 bars for a working diver, but a resting subject in a dry chamber may tolerate 2.5 or 3 bar for many minutes. Factors that increase susceptibility or reduce the tolerance threshold include exposure to an elevated carbon dioxide level, immersion, and both heat and cold.[3] An increase in brain blood flow could be a common element of all these factors. Exercise and breathing resistance due to equipment or dense gas all can cause CO_2 build up. Some individuals tolerate a higher level of CO_2 than normal and thus are at greater risk; these people are called "CO_2 retainers."

Pulmonary or whole body toxicity

The main manifestation of long term exposures to levels of oxygen not high enough to cause CNS toxicity is most commonly an effect on the lungs. This is marked by a substernal or chest pain and a feeling as if the lungs are burning (actually they are). There may be spontaneous coughing or difficulty in inspiring or exhaling a full breath without coughing. The symptoms become more severe with increasing exposure. From acute exposures this condition is regarded as being completely reversible, although from severe cases complete recovery can take a matter of months.[4] While the lung symptoms are the main focus and afford the method of monitoring this particular kind of toxicity, other symptoms are often seen that are not lung related. These are, in addition to the lung problems mentioned above, paraesthesias (especially numbness in fingertips and toes), headache, dizziness, nausea, effects on the eyes, and reduction of aerobic capacity. This has been described in detail by Sterk and Schrier.[5] Since this is more than a lung manifestation we feel that the term "whole body" is perhaps a better choice than just "pulmonary" toxicity[6] and "chronic" is not the right word here.

Intermittent exposures

Before discussing specific algorithms for keeping track of oxygen exposure it is important to mention the technique that is overwhelmingly the most important one

for tolerating oxygen, intermittent exposure.[8,9] Tolerance to all types of oxygen toxicity is increased by interrupting the exposure with periods of breathing a low oxygen mix. This is manifested as "air breaks" in the hyperbaric oxygen treatment of decompression disorders.[10]

Managing whole body toxicity

The story of how the methods of managing whole body toxicity have been developed is a fine illustration of the empirical nature of this practice. Almost all of the early work on this particular type of oxygen toxicity was performed at the University of Pennsylvania by CJ Lambertsen and colleagues.[11] A fundamental contribution of this laboratory was the unit with which low level oxygen exposure is measured.[12]

The parameter monitored to assess lung toxicity is vital capacity. Vital capacity is the maximum amount of gas that an individual can expire after a maximal filling of the lungs; it is reduced by excessive oxygen exposure. A mathematical "curve fit" to empirical data on vital capacity changes as a result of oxygen exposure yielded an equation that can be used to calculate a "unit pulmonary toxicity dose" (UPTD). A unit dose is one minute of exposure to a PO_2 of one bar. The empirical curve (Equation 1) accounted for differences in effect on vital capacity of exposures above and below one bar. The threshold for exposure effects is 0.5 bar, since exposures below this level have no measurable effect on vital capacity. The cumulative pulmonary toxicity dose or CPTD is the sum of UPTDs. A somewhat less intimidating term for the unit dose coming into use is the oxygen tolerance unit, OTU, defined by the same empirical equation:

$$OTU = t \left\{ \frac{(PO_2 - 0.5)}{0.5} \right\}^{0.83} \quad (1)$$

where t is the exposure time and PO_2 is the oxygen partial pressure in bar.

The unit toxicity dose was developed as an empirical measure of changes in vital capacity as result of oxygen exposure. With trained investigators and subjects vital capacity measurements can be quite reproducible, but it is fraught with quantitative hazards and requires careful monitoring.[13]

The original development of the pulmonary tolerance unit used a change in vital capacity as a measure of whether or not the dose was acceptable. A single exposure of 615 units was found to cause a 4% decrement in vital capacity, and this was regarded as the maximum tolerable for an ordinary operational exposure.[14] There was no overt provision in the UPTD/CPTD approach for dealing with recovery; in due course this prompted further empirical investigations.[5]

A project designated Repex had a requirement to manage whole-body oxygen exposure over an operational exposure period of a few days.[15] This resulted in a management algorithm that considers total exposure over a number of days so in effect takes recovery into account over the exposure period. It had been observed that an operationally acceptable daily exposure for a "fresh" diver was 850 OTUs. This method also takes into account the additional tolerance on the first few days of exposure of an individual who has not recently been exposed. Total exposure doses for two, three, or several days were determined, again empirically. The average daily doses get smaller with time and level out at 300 OTU/day (Table 1). The resulting data were put together into an upper limit "Repex" curve for exposure durations of one to 14 or more days shown in Figure 1.[6,15]

TABLE 1

INCREASES IN TOTAL OTU OVER 15 DAYS SHOWING EARLY TOLERANCE

Days	Average daily dose	Increase in Total OTU	Total OTU
1	850	850	850
2	650	550	1400
3	600	460	1860
4	520	240	2100
5	450	200	2300
6	420	220	2520
7	400	140	2660
8	350	140	2800
9	330	170	2970
10	320	130	3100
11	300	200	3300
12	300	300	3600
13	300	300	3900
14	300	300	4200
15	300	300	4500

The degree of "intermittency" of the exposures contributing the data to the Repex curve was not controlled. Most exposures used as data were more or less intermittent, however.

The Repex method provides an empirical method of predicting tolerance. Another approach to empirical control of whole body toxicity is that of Harabin and colleagues.[16] They produced an empirical predictive equation based on a large data base that estimates the reduction in vital capacity as a result of oxygen exposure:

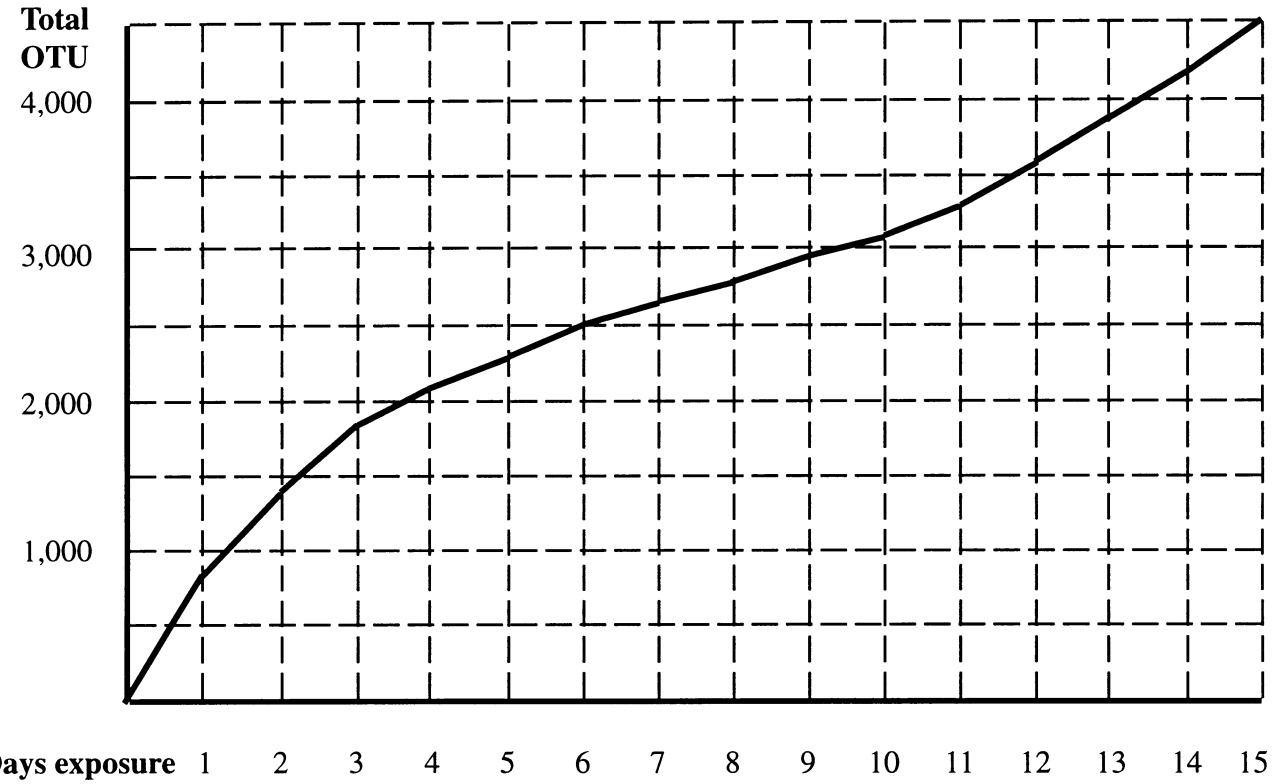

Figure 1. The allowable cumulative oxygen dose for daily exposures up to 15 days. The average daily doses are shown in Table 1. except for the first day and after day 11 the increment in total dosage is less than the daily dose due to tolerance which steadily decreases.

% VC drop = -0.011 (PO_2 - 0.5) t (2)
where t is time in minutes of the exposure, and PO_2 is the exposure level in bar.

The Harabin equation offers an attractive alternative if only vital capacity decrease is to be estimated. Because it is based on data from a wide range of exposures including some very long ones it thus takes recovery into account. A more complex exponential equation based on the same vital capacity data set has been derived recently by Arieli and associates; according to their analyses it gives a better fit.[17]

Managing CNS toxicity

Descriptions of the mechanism of CNS oxygen toxicity are not yet precise enough to permit predictive modelling or development of a "first principles" algorithm for managing exposure. Toxicity appears to be dose related, such that both level and duration of exposure are involved. A high degree of variability between individuals and even at different times in the same individual makes modelling CNS toxicity an imprecise art. Donald recently reviewed a lifetime of his work on CNS toxicity, and one major conclusion is that it is hard to predict.[3] Virtually all of Donald's data was from exposures where the individual was breathing pure oxygen, usually from a rebreather.

For many years there was only one recognised guideline on avoiding CNS toxicity during mixed gas diving (oxygen diving uses more liberal limits). This was a table from the US Navy Diving Manual.[18] This USN Oxygen Partial Pressure Limits Table has been widely reproduced and even incorporated into national law in some places. The table consists of a set of time limits, which are "allowable" exposure limits for various oxygen partial pressures. The values in the table are not expressly physiological, but are appropriate for the allowable exposure time of 30 min at 1.6 bar PO_2; they become excessively conservative for the next few exposure levels lower than 1.6 bar. There are a couple of other things wrong with this table as the sole means of managing CNS toxicity. It does not tell the user what to do if the exposure is not exactly on one of these PO_2 limits, and does not say what to do if part of the exposure is at one O_2 level and part in another. Nor does it provide a method of dealing with recovery e.g. after how much time and in what recovery situation can the exposure begin again.

To its credit, the USN did some additional targeted research and was able to replace this table in the 1991 issue of the US Navy Diving Manual.[10] As before, the new procedures allow somewhat more time for shallow water oxygen divers than for mixed gas divers. For mixed gas diving the Navy takes a fresh approach by setting a flat upper PO_2 limit of 1.3 bar; below this level there are no

time limits, and above this level emergency limits are set out in a chart that allows 30 min at 1.6 bar just as before and goes to a PO_2 level of 1.8 bar where 15 min are allowed; approval by the Chief of Naval Operations is needed for mixed gas diving at a PO_2 of greater than 1.3 bar.

In many situations there is nothing wrong with limitations that are more conservative than they need to be, but in some operational situations such limits can be a considerable handicap. One of these was the situation in undersea habitats. In normal surface-oriented diving with air it is almost impossible to get in a situation that will lead to central nervous system toxicity because of decompression limitations. However, when divers live in a habitat and make excursions with air as the breathing gas the bottom time can be more or less unrestricted; in such cases oxygen exposure while breathing air can become quite significant. This is true of both whole body and CNS toxicity. With the older USN chart as the only thing to go on, oxygen limits became somewhat frustrating for many of the scientists wanting to do extensive work from undersea habitats.

In an attempt to resolve this question NOAA sought the advice of a leading expert on oxygen tolerance, Dr C J Lambertsen. Lambertsen, in collaboration with others familiar with this problem, came up with a new set of oxygen limits. These are in Table 15-1 of the NOAA Diving Manual which came out in 1991, about the same time as the newer USN limits (Table 2). Recovery information is factored in by providing a 24 hour exposure limit as well as single exposure durations for specific oxygen partial pressures. The all-day limits take into consideration whole body exposure as well.

Normal exposures are those involved in standard diving operations. A series of repetitive dives may be accumulated within a single limit. If the single limit is exceeded wait for 2 hours before diving again. If the day limit is exceeded wait for 12 hours.

The new NOAA limits were welcomed by habitat divers but especially by the technical diving community, divers whose decompression is limited in a major way by oxygen exposure. In retrospect, because there have been some oxygen toxicity incidents within the limits of this table,[19] it is best to use these limits conservatively and regard them as applying to a diver performing light work with little or no breathing resistance and thus a normal threshold to CNS toxicity. Many incidences of divers being affected within the limits of this table appear to be related to high workloads or breathing resistance or the like. The NOAA table is not based on a specific set of experiments but rather on the accumulated wisdom of experts in this field.

The structure of this table is just like the old Navy one in that there are limits specified as the number of minutes allowed given oxygen partial pressures. Again there is no provision for intermediate levels or multilevel diving, nor is there an algorithm for recovery.

TABLE 2

NOAA OXYGEN PARTIAL PRESSURE AND EXPOSURE TIME LIMITS
(from Table 15-1, NOAA Diving Manual 1991)

Oxygen partial pressure (PO_2) in bar	Maximum single exposure in minutes	Daily limit: Maximum total duration for any 24-hour day in minutes
1.6	45	150
1.5	120	180
1.4	150	180
1.3	180	210
1.2	210	240
1.1	240	270
1.0	300	300
0.9	360	360
0.8	450	450
0.7	570	570
0.6	720	720

The matter of operating at several PO_2s during an exposure or at values between these stated limits has been dealt with by a simple matter of lineal extrapolation. There is no specific physiological basis for this but likewise there is no meaningful physiological argument against it. All of these limits are empirical operational guidelines and they imply no particular physiology. A first approach was a computational method proposed by Kenyon and Hamilton[20] which called for a linear interpolation between exposure levels and limits, such that, for example, half the exposure time at a given limit would use up half the tolerance and the other half could be used some other way. This same approach was arrived at independently by a group of operationally oriented technical divers, which increments an “oxygen clock” as tolerance time is used up. These unpublished methods have worked well in practice.

Another somewhat arbitrary method of accounting for recovery has been proposed for dive computers.[21] This uses an arbitrary but quite conservative “decay” of the accumulated “oxygen clock” when oxygen exposure is low. This allows computation of decompressions over extended periods and multiple dives.

Harabin and colleagues at the U.S. Navy Medical Research Institute, using survival and likelihood statistics, have a mathematical model that predicts the benefit of intermittency when exposure is above a critical PO_2

threshold,[22] but it does not yet account for immersion and exercise.

Conclusion

Exposure to oxygen can be managed to minimise the operational cost of both of the major toxicities. In both cases it is a matter of staying below reasonably sound empirical limits. For CNS toxicity the limits can be interpolated, allowing oxygen to be used effectively for decompression. For whole body toxicity taking advantage of the initial tolerance at the beginning of an exposure can have equally beneficial effects for the kinds of operation that encounter this problem. For all exposures, tolerance can be increased substantially by keeping the exposures intermittent.

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SOME LIMITATIONS OF SEMI-CLOSED REBREATHERS

David Elliott

Key Words
Equipment, mixed gas, nitrogen, oxygen, rebreathing.

A rebreather has several features which make it attractive to the recreational diver but with it come additional hazards which must be understood if they are to be controlled. In most of the semi-closed oxy-nitrogen rebreathers made for the recreational diver, a pre-mixed gas is supplied at a pre-determined flow rate to a counterlung or breathing bag. The fresh gas is mixed there with the gas already present, much of which has just been exhaled and scrubbed of CO_2. Thus the diver breathes in from the counterlung and exhales through the scrubber back to the counterlung from which excess gas is vented at virtually the same rate that fresh gas is being supplied.

Calculation of the oxygen percentage in the counterlung is based on a simple formula which is independent of depth. In the steady state the percentage of oxygen in the breathing bag may be given quite simply by:

$$O_2\ \% = \frac{(O_2 \text{ flow} - O_2 \text{ consumed})}{(\text{Mixture flow} - O_2 \text{ consumed})} \times 100 \qquad (1)$$

As can be seen, this percentage is independent of depth and, once the supply flow rate has been set for a particular pre-mix, the only variable is that of oxygen consumption. The oxygen percentage is also independent of the volume of the breathing bag. The volume of the counterlung, or more strictly that of the whole breathing circuit including the lungs, will affect only the rate of change from one steady state of oxygen consumption to the next. The rate of change of oxygen content in the counterlung when the diver's work level changes can also be calculated[1] but, with a small circuit volume in relation to a respiratory minute volume for divers of around 20 l/min, this transient phase is brief in relation to the ability to sustain hard work.

Unlike open-circuit systems, in which the composition of the supply gas should be constant, and closed-circuit systems, in which the composition of the inspiratory gases is capable of being provided precisely, the semi-closed system is a dynamic system. The breathing bag provides the diver with gas the composition of which changes during the dive. Given a pre-determined flow rate to the breathing bag of premixed gas with a known composition, the formula above can be used in maintaining the oxygen range within predictable upper and lower limits. Thus the dominant variable during the dive is that of oxygen consumption and will be determined by activities ranging from minimal muscular effort (perhaps when composing a photograph) to maximum sustainable breathing capacity (in some life-threatening situation). Before examining the implications that varied activity may have for the gas composition inspired from the counterlung and the potential consequences of this for the diver, some basic assumptions need to be considered.

Minimal oxygen consumption

An oxygen consumption of around only 0.25 l/min is widely accepted as a lower limit. This value is therefore used to determine the highest percentage of oxygen that could be found in the counterlung, a percentage approaching that of the pre-mixed gas. The maximum allowable PO_2 can then be used to calculate the maximum depth permitted for that flow rate and mixture. In open circuit nitrox diving, the upper limits of allowable oxygen partial pressure have been reduced over the years to 1.5 bar for working hose divers in the North Sea and around 1.4 bar for recreational scuba divers. It is therefore disconcerting to calculate, from the data offered on one recreational semi-closed rebreather, a maximum oxygen percentage which, at the depth quoted, could have a partial pressure exceeding 1.7 bar.

High oxygen consumptions

The other extreme, the maximum sustainable oxygen consumption, is more difficult to predict. For a diver of average size and reasonable "fitness'", an O_{2max} of at least 3 l/min can usually be expected and is almost universally accepted.[2] For the elite athlete performing out of the water an oxygen consumption exceeding 7 l/min can be sustained.[3,4] It is also known that maximum voluntary ventilation (MVV) and maximum breathing capacity (MBC) are significantly reduced at raised environmental pressure,[2] and by as much as around 50% at 45 m. Nevertheless, for counterlung calculations the Royal Navy uses O_2 3 l/min and the U.S. Navy and at least one manufacturer use 2.5 l/min. Given also that apparatus for sport diving is not denied to exceptional athletes, the figure of at least 3 l/min for maximum sustainable O_2 should be used as the value appropriate for application to semi-closed apparatus at all depths.

An implication for the diver using apparatus set up in accordance with calculations based on oxygen consumptions lower than these extremes is that, when maximally exercising, the diver could well sustain an oxygen consumption greater than the volume of oxygen provided. One semi-closed rebreather currently available provides the diver with only 5 l/min of 40% oxygen according to its manufacturer.[5] These figures have since been confirmed by that manufacturer. That provides only 2 l/min of oxygen but even less than that is available for the

diver's use and, when oxygen consumption exceeds 1.25 l/min, the breathing bag oxygen will become less than 21%. The same apparatus, at a possible oxygen consumption of 1.75 l/min, with a constant mass flow of 5 l/min 40/60, will supply the diver with a PO_2 of 0.3 bar at its advertised maximum depth of 30 m. However, this would be achieved with only around 8% oxygen in the breathing bag which would mean, not only an equivalent air depth of 36 m, but also that it would not be a safe mixture for making the ascent. Although this particular breathing apparatus is claimed to be for only those divers weighing 198 lbs (90 kg) or less, maybe it should also be restricted to macro-photographers diving in swimming pools.

That example of 5 l/min seems particularly extreme because other manufacturers and several training agencies recommend double that flow rate for 40/60. Yet even these higher flows do not solve all the potential problems. In at least one design, an oxygen consumption 2.5 l/min (which is less than that used by the Royal Navy for its evaluation of breathing apparatus) can still be sufficient to bring counterlung oxygen content down below 21% and so reverse the advantages of using an "equivalent air depth" for decompression. Specifically with a 40% oxygen premix at the manufacturer's constant flow setting of 9.2 l/min, the formula (1) provides

$$\frac{(9.2 \times 0.4) - 2.5}{(9.2 - 2.5)} = 17.6\ \%\ \text{oxygen.}$$

The manufacturer's setting for 32% oxygen premix is 11.4 l/min. Perhaps the reader would like to calculate the oxygen percentage from that setting at a O_2 of 2.5 l/min or more. One conclusion might be that macrophotography and gentle swimming may be relatively safe with those settings, but the diver must not to get into a life-threatening situation which needs sustained hard work.

A manufacturer's response to my queries included the following:

> at the lower limit of technical tolerances a constant flow is guaranteed that creates a minimum O_2 content of 17% at a metabolic rate of up to 2.5 l/min.
>
> it is part of the training that in periods of higher workload and breathing, the diver needs to exhale through the nose in order to *(empty the breathing bag and)* make sure fresh gas is supplied through the bypass valve when inhaling the next time.
>
> for the calculation of EADs ... assume a constant O_2 consumption of 1.5 l/min.
>
> in case the diver encounters higher consumptions than estimated, we suggest the use of air-decompression tables.
>
> our ranges are only suggestions, the settings are the responsibility of the training organisations.

These answers raise yet more questions. Because decompression tables require to be entered at the deepest depth of the dive, how can one estimate the deepest EAD of the dive? Is it valid to estimate an average oxygen consumption? What would be the implications of an EAD which, using the conventions of the diving tables, should be based on 17% oxygen? In particular, as the actual EAD varies during a dive and sometimes, based on the quoted settings, may tend on some dives towards being deeper that the actual depth, how can a safe decompression ever be planned?

But enough has been said already to demonstrate that there are some uncertainties with the use of semi-closed circuit breathing apparatus. These need to be dealt with by the training agencies, perhaps at the price of increasing flow rates even though this reduces cylinder duration.* It is possible that there is sufficient padding in the decompression tables that these questions about unpredictable EADs and decompression are relatively academic, but the data needs to be collected and published. In the meanwhile, the active diver using semi-closed apparatus might prefer to plan on using the air decompression tables for the actual depth dived.

Evaluation of breathing apparatus

Once upon a time all new decompression tables and all new items of breathing apparatus were vigorously evaluated by a naval Experimental Diving Unit before being brought into service for the naval diver and, in due course, being released for public use. No longer is this process routine but rigorous testing of non-military equipment is still available if required. However, the recreational diving industry appears to be sufficiently confident in their designs that some items may never have been tested to their limits. A request to a particular manufacturer for data from manned testing on actual levels of oxygen in the breathing bag during hard work revealed that no such data was available. Wisely perhaps, some of the trainers using one semi-closed set have increased the flows and reduced the maximum depths for some mixtures. It is not known if such decisions are based on measurement or, more probably, intelligent guesswork and it is not known if the same safety factors are introduced worldwide by all training agencies. Also, it is not known, to the author at least, if similar safety considerations have been reviewed for all the versions of semi-closed sets that may appear on the market.

*** Footnote**

Since being sent a prepublication copy of this SPUMS presentation, one manufacturer has incresased their flow rates significantly and has also undertaken some manned testing of oxygen levels. A welcome step towards improved safety.

In contrast to the introduction of new naval equipment, a team of leading training agency officials and recreational instructors was convened some time ago for the first formal training program of a new oxy-nitrogen semi-closed rebreather. One would imagine that this group would comprise instructors who are focussed on diving safety and its evaluation but it is reported[6] that, in their spare time, some of them scuba dived solo on compressed air to 123 metres (400 feet). If this were so would *you* trust as safe a complex new breathing apparatus that is recommended by such an instructor? Validation demands appropriate laboratory evaluations by scientists and/or the military who are, and remain, independent.

Conclusion

More work needs to be done to confirm the safety of semi-closed breathing apparatus for recreational use. Gas samples for both O_2 and CO_2 from breathing bags at the O_2 extremes during shallow manned trials by exceptionally fit divers need to be taken at a laboratory experienced in diving physiology and analysed before settings such as flow rates are decided. A number of the claims made in the sport diving press and by the manufacturers about semi-closed rebreathers appear to be exaggerated, but the diving public is not sufficiently well informed to assess this. Diving doctors need to be aware of these problems and be prepared to educate if and when the agencies and manufacturers provide misleading statements.

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Dr David H Elliott was one of the guest speakers at the SPUMS 1996 Annual Scientific Meeting. He is Co-Editor of THE PHYSIOLOGY AND MEDICINE OF DIVING, which was first published in 1969, with the most recent edition in 1993 and is also the civilian consultant in diving medicine to the Royal Navy. His address is 40 Petworth Road, Haslemere, Surrey GU27 2HX, United Kingdom. Fax + 44-1428-658-678.
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TESTING THE PERFORMANCE OF REBREATHERS

Mario Loncar and Hans Örnhagen

Key Words

Equipment, mixed gas, oxygen, performance, rebreathing.

Abstract

The growing interest in nitrox- and tech-diving among recreational divers has created the demand for rebreathers. Compared with open systems, this breathing apparatus offers long duration, silent diving and, in some cases, decompression benefits. Some rebreathers are on the market, but many are designed and built by the divers themselves, with a possible increase in the risks for accidents caused by malfunction of the unit.

When rebreathers are approved for use today, only the work of breathing and the scrubbing capacity, using a CO_2-injection technique, are tested. We suggest the use of a respiratory simulator capable of extracting oxygen. The respiratory simulator, using catalytic combustion of propylene, also imitates other aspects of respiration such as CO_2, humidity and heat production. With the respiratory simulator standardised tests can be performed which, together with a limited number of verifying dives with divers, should offer good possibilities of revealing weak spots in rebreather designs.

Introduction

The growing interest in nitrox and so called "technical diving", has created an increasing interest in rebreathers to meet various demands from recreational divers. Sports diving associations such as PADI and CMAS have already issued special procedures for mixed gas or enriched air diving for open circuit breathing equipment.[1,2] It is likely there will soon also be procedures for rebreathers because closed circuits are needed to allow full use of the advantages with nitrox in scuba.

The rebreather is not a new invention. It has been used in military diving for a long time and today is also used as a bail-out system in saturation diving. The use of rebreathers by sports divers means that a technically more advanced apparatus requiring more sophisticated dive procedures has spread to a population of divers, who use a less efficient surface backup organization than professional divers, with a vast variation in educational background. Furthermore, a lot of "home made" designs and constructions are likely to be built and used by divers with the necessary skills, who find the rebreathers on the market too expensive.

In this situation we would like to present a test procedure that can be used for testing and approval of all kinds of closed and semi-closed breathing equipment.

Rebreathers

Rebreathers can be grouped into three main categories, closed, semi-closed and pure oxygen rebreathers. The distinction between the different types of rebreathers is based on the method of controlling the gas composition in the breathing circuit. Looking at complexity level of rebreathers, the oxygen rebreather is usually the least complex apparatus, based on either volume demand or constant mass flow with bypass. The semi-closed breathing apparatus can be simple and have a constant mass flow adjusted to the oxygen content of the supply gas, or be based on more sophisticated principles, e.g. supply gas additions in relation to the need of the metabolism as measured mechanically through the ventilation. Finally the highest sophistication can be found in the closed rebreather in which pressure and oxygen sensors, together with electronic control systems and valves, provide a constant PO_2 in the breathing gas. This complex scuba requires a higher degree of training and more maintenance than the other types.

The semi-closed rebreather with a pre-set gas mixture and fixed flow of supply gas will probably be the most frequently used rebreather for recreational diving, because of the less complex design and lower price compared with electronically controlled closed circuit rebreathers.

Compared with open systems, closed and semi-closed breathing systems offer long action duration, gas savings, and in some cases decompression benefits. In addition stable buoyancy and silent diving, which originally made the rebreather useful in military diving, is appreciated by underwater photographers and zoologists. These advantages are accomplished at the cost of the equipment being more complicated, more expensive and requiring a higher degree of user training. The complexity of rebreathers introduces risks that are not found in open circuit breathing equipment. In table 1 some of the major risk factors are listed.

TABLE 1.

MAJOR RISK FACTORS USING REBREATHERS

Problem	Possible cause
Hypoxia	
	Gas supply not opened or empty
	Wrong supply gas or setting of supply flow
	Failure of sensors, control circuit or valves
	Inappropriate purge procedures
Hyperoxia	
	Wrong supply gas or too deep dive
	Failure of sensors, control circuit or valves
Hypercapnia	
	Scrubber not filled or material worn out
	Inappropriate scrubber performance at low temp
	Scrubber flooded
Excessive work of breathing	
	Wrong type of scrubber material (granule size)
	Lack of maintenance
	Scrubber flooded
Caustic cocktail	
	Inefficient water trap (design or maintenance)
	Inappropriate use of mouth piece shut-off valve
Water entry	
	Leaks because of lack of maintenance or error in the assembly (e.g. missing gaskets)
Loss of breathing gas	
	Rupture of hose or bag. Technical failure

Although most rebreather designs have built in countermeasures to handle the problems listed, this is not the case with all, and one fears that the design of budget versions for recreational diving will lack these countermeasures. A test procedure should therefore be able to reveal the weak spots and help to improve both the design and the user's manual, to make the use of the rebreather safe and easy. This is most important when completely novel designs, "home made" equipment, or equipment from less well known manufacturers are to be tested and evaluated.

The importance of an adequate minimum oxygen partial pressure (> 20 kPa) to avoid hypoxic loss of consciousness, and a maximum PO_2, (usually < 160 kPa) to avoid oxygen convulsions is easily understood. Examples of accepted maximum PO_2-levels are shown in table 2 in which limits from different authorities are listed.[1-5] In military operations higher risks can be accepted and thus often a higher PO_2 is allowed, see fig 1.[3,6]

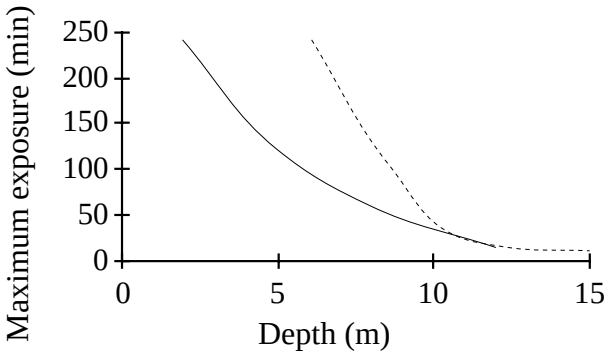

Figure 1. Maximum permitted oxygen partial pressure and maximum exposure time during oxygen diving in the Swedish Navy (solid line) and the US Navy (dotted line),

TABLE 2

MAXIMUM ALLOWABLE PO_2 IN MIXED GAS DIVING

Regulations	PO_2 [kPa]
Swedish Navy, nitrox	190
US Navy, bounce dive heliox	180
CMAS, mixed gas diving	160
UK commercial diving regulations	160
Norwegian commercial diving regulations	160
PADI, mixed gas diving	140
Swedish commercial diving regulations	140

TABLE 3.

THE CONSEQUENCES OF THREE DIFFERENT OXYGEN FRACTIONS IN THE INHALED BREATHING MIX DURING A 20 m DIVE USING NORDIC SPORTSDIVING TABLES (1995).[10]

FO_2	EAD	No stop time /N_2 load	N_2 load after 40 min dive	Surface interval to reach B
35%	14.7 m	85 / J	F	5:01
30%	16.6 m	60 / I	G	5:31
25%	18.5 m	40 / H	H	6:01

The frequent use of high oxygen partial pressures can also affect other organs and reversible changes can be detected in lungs and blood even if no diver performance decrement is observed.[7] It is thus highly recommended to follow and limit the accumulated daily "dose" of oxygen if high oxygen partial pressures are used .[8,9]

Less well understood is the importance of knowledge about PO_2 through the whole dive and thereby knowing the inert gas partial pressure. This allows a safe and optimal calculation of the nitrogen loading during the dive, and the need for surface intervals between dives and/ or decompression profiles. An example is shown in table 3.

Increase in the inspiratory PCO_2 can cause not only increased ventilation, dyspnoea and discomfort, but also jeopardise survival through effects on consciousness. For a summary of CO_2 effects on man during diving see.[11]

Test procedures

When evaluating open circuit demand breathing equipment, the work of breathing (WOB) and peak pressures have traditionally been the most important parameters.[4,12] The standard technique for testing the performance of open breathing equipment is with the use of a breathing simulator. This method is also appropriate for testing the WOB in closed and semi-closed breathing systems. Tidal volume is usually measured as the displacement of the breathing simulator piston. The pressure is measured as a differential pressure between the inside of the mouthpiece and a suitable reference point.[13] It is important that all measurements are made in water to include hydrostatic and hydrodynamic loads.

In open circuit demand breathing systems the inhaled gas fraction and consequently the gas partial pressures are well defined and directly depending on the dive depth. This is not the case in closed and semi-closed breathing apparatus. When evaluating a rebreather, besides the WOB, the performance of the carbon dioxide scrubbing system, the inhaled gas fractions and partial pressures have to be evaluated. The gas concentrations and partial pressures vary depending on the technique for adding gas to the rebreather, the oxygen uptake of the user and the ambient pressure.

Because of the consumption of oxygen from the circuit, the inhaled oxygen fraction is not same as in the supply gas. Theoretical models describing the behaviour of rebreathers are available and today the oxygen supply system is usually evaluated theoretically by calculations. In addition human test dives are made in experimental chambers where sampling lines for gas analysis can be

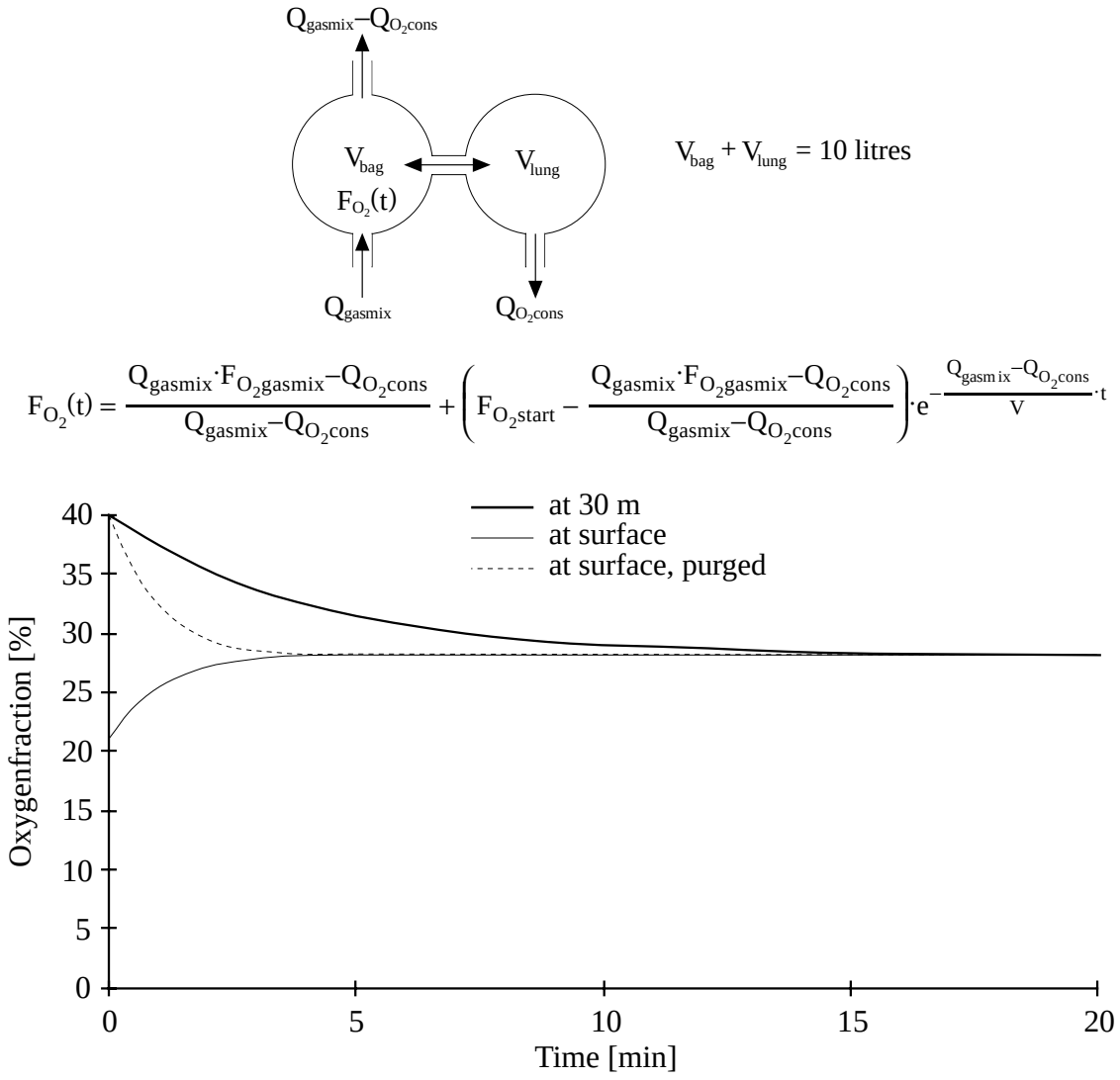

Figure 2. An example of a calculation of the oxygen fraction in a semi-closed rebreather at 0 m (dotted line) and 30 m (thick solid line) with a fixed gas mixture containing 40% O_2 added at a rate of 12 l/min (STPD) and an oxygen consumption rate of 2 l/min (STPD). The oxygen fraction when the rebreather-lung system is not purged at the start is illustrated by the thin solid line.

attached. In a design using a constant mass flow of a fixed gas mixture, it is possible to solve the equations explicitly. The equation and graph in figure 2 is an example of how to calculate the oxygen fraction in a rebreather with a constant mass flow of a fixed gas mixture:

When using semi-closed rebreathers with pre-set gas mixtures, incorrect use of gas mixtures and wrong settings of gas supply flow can impose hazards such as hypoxia and hyperoxia as illustrated in fig 3. Using air as supply gas in a constant mass flow rebreather, when no proper mixture is available, will undoubtedly lead to hypoxia if the supply flow is not set unreasonably high. Because of this risk it is important that the gas bottle connection in the rebreather is such that air bottles can not be connected to a rebreather designed for mixes of higher oxygen content.

Carbon dioxide can be added to test scrubber performance in a simulator test but no simple method for extraction of oxygen is used routinely today. Therefore divers are needed to verify the actual performance of the apparatus. Humans vary both intra- and inter-individually, which makes objective comparisons very difficult, and a large number of dives have to be performed to allow statistical analysis.

The use of divers when testing the equipment in extreme situations such as at great depth, low temperatures and long exposure times also imposes ethical limitations. We therefore suggest the use of a simulator, that can extract oxygen, deliver CO_2, heat, and water vapour for these tests.

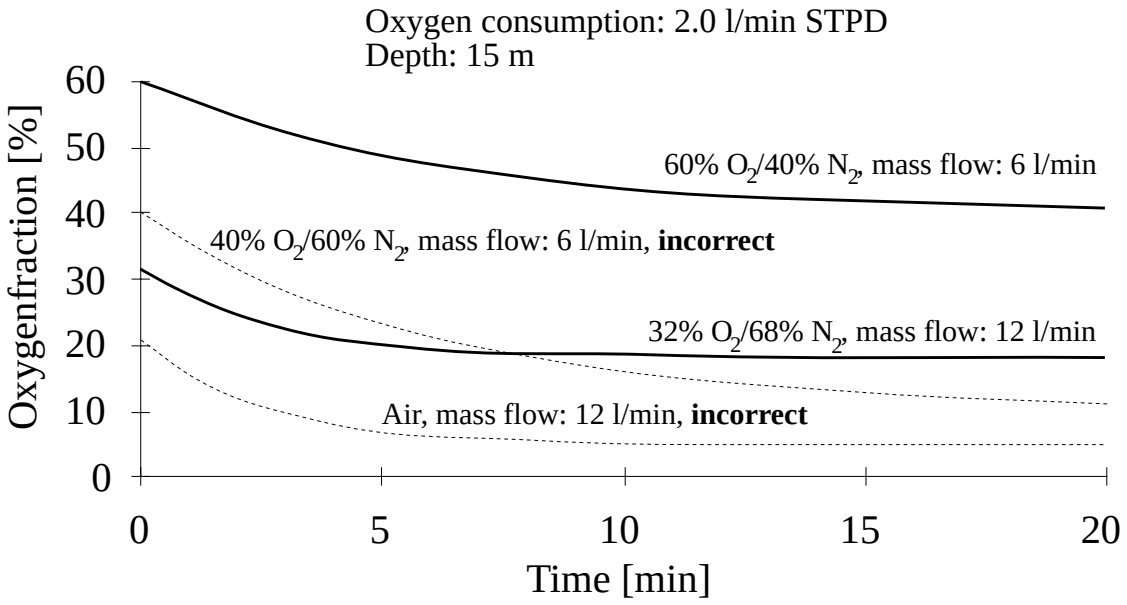

Figure 3. An example of the oxygen fraction in a semi-closed rebreather at 15 m and an oxygen consumption rate of 2 l/min with four fixed gas mixtures. The mixtures are added at two different rates, 6 l/min (STPD) and 12 l/min (STPD). The two mixtures 60% O_2 at 6 l/min (solid line) and 32 % O_2 at 12 l/min (solid line) do not produce hypoxia. The other two, 40% O_2 at 6 l/min (dotted line) and air at 12 l/min (dotted line) will produce hypoxia and are labelled incorrect.

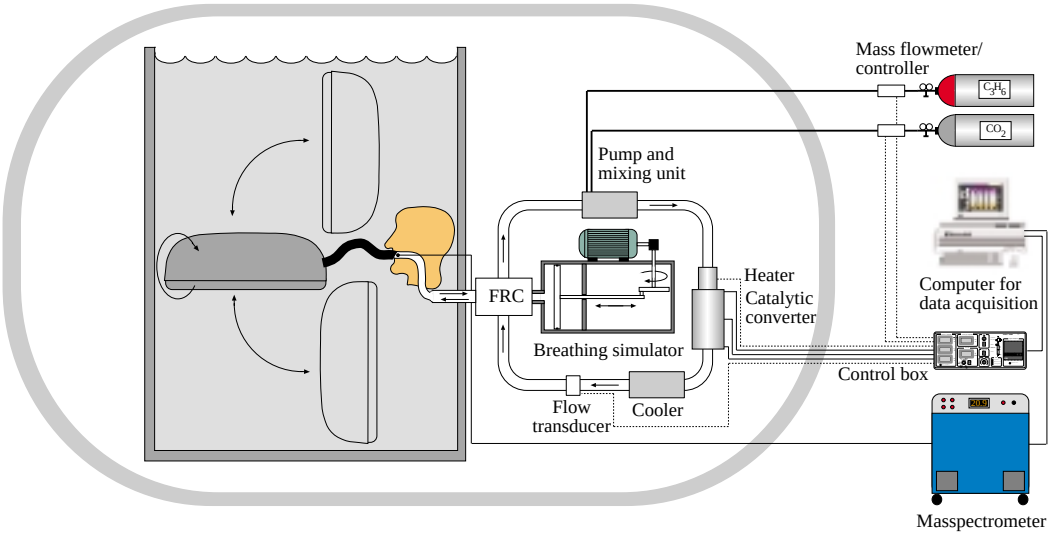

Figure 4. A schematic figure of the respiratory simulator. To the left is shown how the apparatus is tested in four different attitudes.

Improved test procedure with respiratory simulator

To overcome the shortcomings of the present testing methods for rebreathers a respiratory simulator (FOA respiratory simulator) incorporating both the ventilatory and the metabolic components of the human respiration has been developed.[14] The respiratory simulator uses catalytic combustion of propylene gas resulting in an oxygen consumption directly proportional to the flow of fuel added. The V_{CO_2}/V_{O_2} (respiratory quotient) with the gas used is 0.67, which makes addition of extra CO_2 necessary. This makes it possible and easy to vary the "respiratory quotient" from 0.67 to over 1, which can be an advantage in some situations. The internal volume of the unit is small, ≈ 1.5 litres, and this makes it possible to have a volume of the whole system comparable to the functional residual capacity of humans of different size. This is important when the exact gas composition is measured during changes in ambient pressure and when simulating breath-holding or other changes in the breathing pattern.

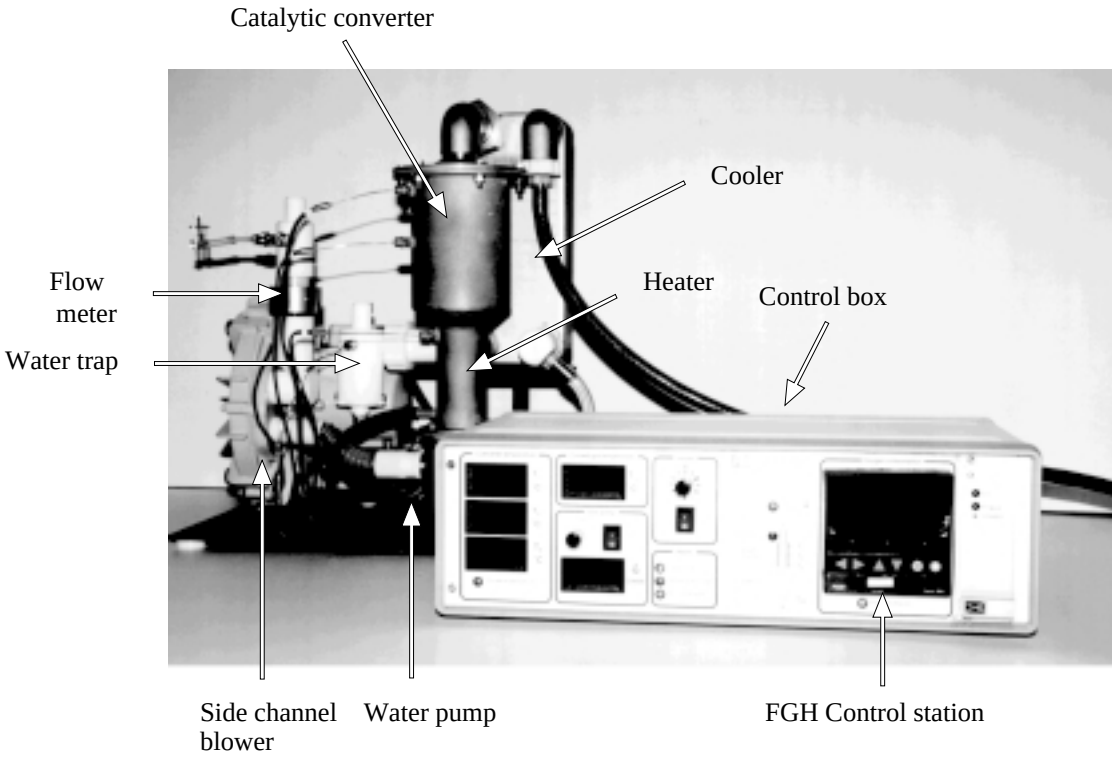

Figure 5. A photo of the metabolic simulator. For size reference, the control module is 48 cm (19") wide.

Advantages using the respiratory simulator:

- Objective and reproducible measurements of gas concentrations and time constants in closed and semi closed breathing apparatus under different diving conditions.
- Ability to test the equipment under extreme test conditions without exposing divers to risks.
- Man and time saving procedures because no diving is involved.

Figure 6 shows screen dumps from the data acquisition system, showing the high degree of similarity of inspiratory/expiratory gas contents between the simulator (to the left) and human (to the right). Since the "metabolic process" continues also when the breathing machine is stopped, it is possible to simulate breath holding with the respiratory simulator (not shown in the graph).

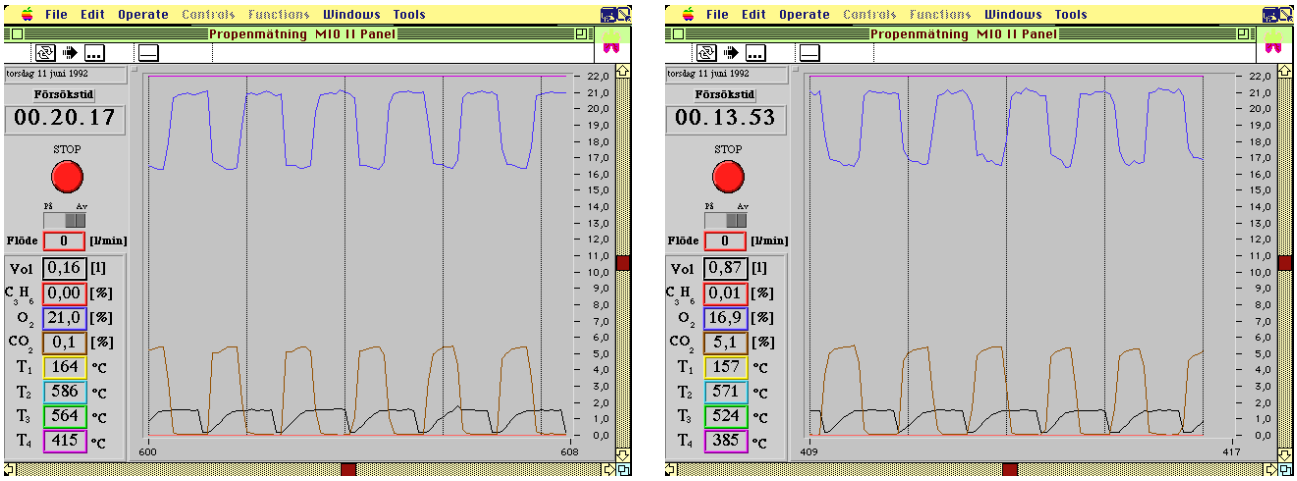

Figure 6. Recordings of O_2 and CO_2 during respiratory cycles of the respiratory simulator (left) and a human (right).

Suggested test protocol

We suggest that a testing procedure for rebreathers, should include:

- Work of breathing (WOB), peak pressures and static loading test in at least four different attitudes (head up, head down, prone and supine).
- CO_2 scrubbing capacity test (time until P_iCO_2 0.5, 1.0 and 1.5 kPa)
- Tests of O_2- and CO_2-fractions and partial pressures and also their rate of change during simulated dives to, and ascent from, the maximum approval pressure during at least two different oxygen extraction rates (rest and 3.0 l/ min [STPD])

With the respiratory simulator all of the above mentioned tests can be performed in the same dive thus saving time, effort and money. A limited number of verifying dives with human subjects should also be performed after the unmanned testing.

In figure 7 is illustrated how the suggested test procedure was used in a quality assurance process to verify the function of an improved version of the Interspiro semi-closed breathing apparatus for mine clearance.[15] In the graph are some of the measured parameters illustrated during a wet 57 m dive using the FOA respiratory simulator in the wet pot of the chamber system at the Swedish Navy Diving Centre. The oxygen fraction is the average inhaled fraction from one rebreather. From the graph it is seen how the oxygen fraction is slightly reduced at the highest work load near the surface, but stays well above the minimum 20%. During the rapid compression the breathing bag and lung volumes are filled with supply gas and the highest oxygen partial pressure is recorded when the bottom is reached. A 44 min decompression should follow the 25 min bottom phase if a human performed the test dive. With the simulator, ascent to surface can be done directly, thereby saving time. The real advantage is in the fact that once the equipment is installed, one person can manage several tests during a day, which is impossible if divers are involved.

Conclusion

To allow extensive tests of rebreathers at reasonable cost and manpower, a respiratory simulator capable of consuming oxygen has been developed. It is our recommendation that in the future rebreather approval should include tests of the oxygen delivery system to assure oxygen partial pressures are within acceptable limits in addition to other important parameters.

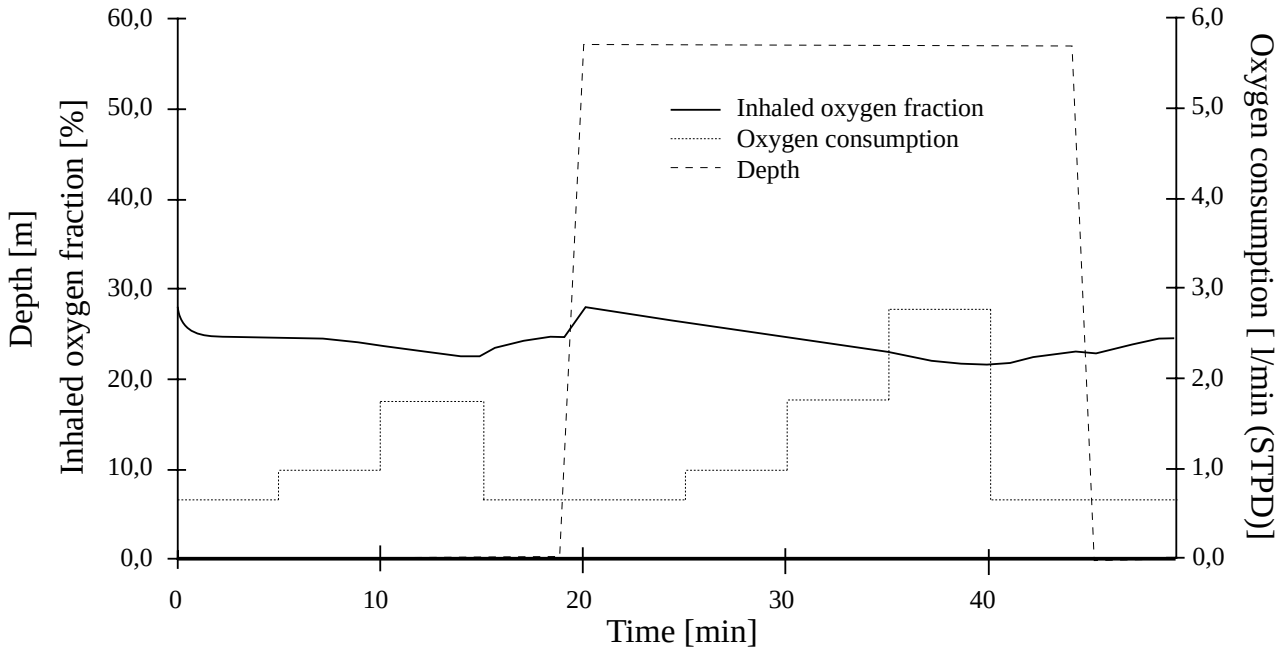

Figure 7. An example of a test of an Interspiro DCSC rebreather using the described respiratory simulator. The inhaled oxygen fraction (solid line) as a function of oxygen extraction (dotted line) and pressure (broken line) is shown over the 50 min test period. The minute ventilation at different oxygen extractions follow the Norwegian Petroleum Directorate (NPD) rules for corresponding CO_2 productions (NPD 1991).

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REBREATHER PHYSIOLOGY

RW (Bill) Hamilton

Key Words
Equipment, mixed gas, oxygen, physiology, rebreathing.

A diver breathing on open-circuit apparatus "throws away" a great deal of perfectly good gas and this "waste" increases with increasing depth. A rebreather recovers and reuses much of this inert gas that would otherwise be lost; it removes the CO_2 and replaces the oxygen consumed.

The basic characteristics of rebreathers in general, a bit about their history and the problems of semi-closed rebreathers have been discussed by Dr Elliott.[1,2]

Rebreather essentials

Only a small amount of the air a person inhales on each breath is actually used by the body. Virtually all of the nitrogen and most of the oxygen is exhaled with a little CO_2. A rebreather enables most of this exhaled breath to be reused and must have a few essential components. These are a breathing loop with valves to control the flow direction, a counterlung or breathing bag, a canister to absorb CO_2 and some way to add gas when the volume in the breathing bag decreases. Valves maintain the flow in a constant direction and breathing pushes the gas through the canister.

For diving a rebreather must have a compliant volume, a space that can expand by the same volume that the diver exhales and inhales on a breath. As a result the total gas volume does not change appreciably, so buoyancy does not change during breathing. Usually it is the diver's

breathing which moves the gas around the circuit. Valves direct the flow in all but the to-fro types.

A rebreather should have low breathing resistance and protect against excessive heat loss. It should have high reliability, and perhaps redundancy, appropriate size and weight, a manageable degree of complexity in both use and maintenance (i.e. be "diver proof"), reasonable cost, upkeep and maintenance.

For military purposes it might be silent, bubble free and non-magnetic. The lack of bubbles can also be important to photographers and naturalists studying marine wildlife. Oxygen and oxygen-controlled rebreathers produce no bubbles during use at a constant depth, but all rebreathers must vent some gas on ascent. Semi-closed rebreathers make few bubbles, perhaps 15 to 25% of the equivalent open-circuit scuba diver; the bubbles usually come out of the backpack instead of the mouthpiece, so are less noticeable and not so noisy.

Oxygen rebreathers

The simplest category of rebreather for divers is the pure oxygen rebreather. This unit is filled only with oxygen; it adds oxygen when the gas volume in the bag is reduced below a selected volume. Oxygen rebreathers are depth-limited because oxygen becomes more toxic as pressure (depth) increases. Because they are fully closed and do not release any bubbles during level swimming they are popular for military use.

An older British oxygen rebreather uses to-and-fro or pendulum gas flow where the diver breaths directly through the canister; this design is still in use. The diver rebreathes the dead space so these units tend to cause CO_2 build up and unconsciousness. The to-and-fro design does help conserve breathing gas heat, which can be an advantage, and they are less costly, simpler to operate, and in that sense more reliable.

Fully closed rebreathers with oxygen control

The most sophisticated and effective rebreathers are fully closed units with oxygen control. The oxygen level is controlled electronically and usually several sensors are used for redundancy. These units carry both oxygen and diluent gas (an inert gas with a small amount of oxygen), and add whichever gas is needed. The more modern ones have computers and do sophisticated control and logging of many things in addition to adding gas.

A fraction of oxygen should be added to the inert diluent gas to make it breathable or at least survivable in case the diver breathes the diluent gas only. These units allow oxygen to be set at a given PO_2 and held throughout the dive. US Navy (USN) rebreathers (Mark 15 and 16) are "hard wired" to maintain a PO_2 of 0.7 atm (acceptable range 0.6 to 0.9 atm), but other more modern units allow the PO_2 level to be selected. Oxygen-controlled rebreathers usually make no bubbles except during ascents. Considerable experience has been accumulated in military use of this type of rebreather.

Semi-closed rebreathers

Dr Elliott has dealt with these rebreathers and their problems on pages 48-50.[2]

Physiological aspects of rebreathers

There are physiological consequences of breathing on a rebreather that are different from diving with air. Some of these are due to the nature of the gas mixture, others due to the mechanical aspects of the rebreather itself. There can be major physiological concerns to the diver if the rebreather is used beyond its design limits or in the event that it does not function properly. It is advisable for rebreather divers to be acquainted with these factors.

Respiratory exchange and lung ventilation

Exhaled gas (when breathing air) is mostly oxygen and nitrogen but it has less oxygen and also contains some carbon dioxide. About 0.8 as much carbon dioxide is exhaled as oxygen consumed. This difference, the ratio of CO_2 produced to O_2 consumed, is called the respiratory exchange ratio. A volume of gas, with low or no CO_2, much greater than that needed for metabolism has to be breathed to ventilate the lungs sufficiently to remove CO_2. At increased pressures the number of molecules of oxygen in a breath is proportionally more, but the amount of gas required to meet the body's metabolic needs does not change significantly with depth.

Effects of breathing gas disturbances

ASPHYXIA

If a person breathes air in and out of a closed bag the bag will accumulate CO_2 and will become depleted of oxygen. In a short time, which depends on the size of the bag and how hard the person is working, the CO_2 will become excessive, causing shortness of breath (dyspnoea), and the oxygen deficient, causing unconsciousness, and if this continues will inevitably lead to death.

With an 8 litre bag, as in some rebreathers, a constant oxygen consumption of 0.5 l/min and constant CO_2 production of 0.4 l/m, in 2 minutes the PO_2 will be down to

0.09 bar (9%) and the PCO_2 will be about 10%. This would be extremely stressful but most people would probably still be conscious. In 3 minutes the PO_2 will be down to 0.02 bar, low enough to cause unconsciousness and very soon death. The PCO_2 will be about 16%, enough to be extremely distressing and narcotic. Although some people might become unconscious from this level of CO_2 it is not life-threatening. Low oxygen is the dominant and dangerous factor in "closed bag" asphyxia. The principle is valid here, but this is a simplification of what would happen.

HYPOXIA

If the bag has a device that will remove CO_2 repeated breaths would deplete the oxygen, but no CO_2 would accumulate. The person would be unlikely to experience severe dyspnoea, and might not be aware of the shortage of oxygen until too late (unconsciousness occurs), but the respiratory minute volume (RMV) would begin to increase due to hypoxia. In about the same time he would become unconscious and eventually die from hypoxia. There would be very little discomfort and he might feel rather euphoric and unconcerned about the situation; euphoria is a typical and characteristically dangerous aspect of hypoxia.

The symptoms of hypoxia with rapid onset (a few minutes) are dizziness, dimness of vision or "tunnel vision," paraesthesia and tingling, numb lips, difficult speech, breathlessness, followed soon by collapse and unconsciousness. These symptoms can be loosely related to decreasing inspired partial pressures or sea level percentages; these are quite variable with individuals and circumstances. In general oxygen levels above 0.16 bar or 16%, have no noticeable effects except loss of night vision; 14 to 12% or 0.14 to 0.12 bar causes tingling, numb lips, tunnel vision and slight increase in RMV; 10 to 9% or 0.1 to 0.09 bar produces difficult speech, dizziness and for some collapse is imminent; leads to unconsciousness and death.

HYPERCAPNIA

A person breathing from a bag filled with oxygen, which has the oxygen replenished as needed but which allows the CO_2 to accumulate would experience mild dyspnoea which would become more severe with each succeeding breath. Eventually the person would become unconscious. As the CO_2 level increased the person would feel considerable circulatory changes, would feel a flush over the body, would begin to have a headache, and might have a convulsion. There is no shortage of oxygen, it is hypercapnia, a build-up of CO_2**.** A level of up to 30-40% CO_2 is survivable, but well before this level the individual would become unconscious; beyond this level the individual would have serious problems.

HYPERVENTILATION LEADING TO HYPOCAPNIA

Another disturbance is hypocapnia, a reduction of the CO_2 level in the body. CO_2 controls ventilation, which is the only way it can be reduced. Excessive ventilation can wash enough CO_2 out to have physiological effects. These resemble those of hypoxia which can make immediate diagnosis quite difficult. The symptoms are dizziness, paraesthesias and tingling, numb lips, difficult speech, and confusion; in addition there may be a "tetany" or muscle tension and twitching, especially of the hands. Hyperventilation can be triggered by low oxygen (which stimulates breathing), but anxiety is a commoner cause in diving.

HYPERVENTILATION LEADING TO HYPERCAPNIA

Divers are often known to "hyperventilate." The term hyperventilation is used to describe rapid breathing, but in some cases it is not an excess ventilation of the lungs, as the name implies, but rather an excessive ventilation of the dead space, snorkel or rebreather, with inadequate ventilation of the lungs. If the person is exercising this can lead to a rapid CO_2 build-up. Apparent hyperventilation that is in fact inadequate can happen if the breathing rate increases while the effective depth of each breath decreases. This is a natural response when breathing against a resistance and stimulated to breathe (e.g. by exercise). The diver may make a great effort to ventilate, but because the breaths are too shallow the result is ineffective. This is most likely to happen when the diver is distracted and only aware that more ventilation is needed. It is more likely to occur in a to-and-fro type rebreather which adds some dead space. This and other things that can cause a build-up of CO_2 that can lead to loss of consciousness.

HYPEROXIA

Oxygen is essential in breathing gas for body metabolism, but too much of it can cause oxygen poisoning. The physiological effect of oxygen is a function of its partial pressure (the product of oxygen fraction and pressure), so the ideal fraction of oxygen in a diver's breathing mixture depends on the depth. The remaining space has to be filled with an inert gas usually called the diluent gas**.** Almost any rebreather that has an oxygen supply component is capable of delivering excess oxygen and the important danger is CNS oxygen toxicity. There are various algorithms for avoiding this, but a good rule of thumb is not to allow the PO_2 to exceed 1.4 bar, or even better, 1.3 bar.

Dealing with breathing gas disturbances

For almost all situations the prescribed action is to abort the dive, switching to the open-circuit backup breathing system if possible. In almost all cases it will be beneficial to reduce the work load. It is good practice in being prepared to deal with emergencies to think about the different things that can go wrong before they happen.

Decompression disadvantages and advantages of rebreathers

Accepting that higher oxygen levels are beneficial to decompression, rebreathers can work both ways with regard to the efficiency of decompression.

Semi-closed rebreathers have two problems. First, they can have a variable level of oxygen which makes predicting the optimal decompression quite difficult. Decompression may have to follow "worst case" presumptions with a significant loss of efficiency.[3] The more important effect is increased diver activity causes the oxygen levels to go down. Activity tends to accelerate the circulation and causes the diver to take on more gas, resulting in a greater decompression obligation. Just when the diver needs the better decompression, which would result from a higher PO_2, the oxygen is lower.

On the other hand, oxygen-controlled rebreathers can be efficient. Decompression can be almost as efficient as it gets with a constant, optimal PO_2 level (a useful setting is 1.3 to 1.4 bar). By maintaining this level throughout the dive the maximum advantage of oxygen is achieved .[4,5] Special decompression tables or a computer are needed. Some state-of-the-art computer-controlled rebreathers also include a decompression computer. This can add to decompression efficiency since it can know the PO_2 continuously as well as the time and pressure profile.

Design factors

Several aspects of rebreathers that are an inherent part of the design can have physiological impact. Among these are breathing resistance, the relative location of the counterlung, and the scrubber. Morrison and Reimers provide a good review of the mechanics and physiology of rebreathers.[6]

References and additional reading

1 Elliott DH. Rebreathers: an introduction. *SPUMS J* 1997; 27 (1): 39-42

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