MANAGEMENT OF DIVING ACCIDENTS

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Summary

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

Introduction

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

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

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

Diver rescue

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

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

FREE-SWIMMING DIVERS

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

STAGES AND BELLS

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

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

SATURATION SYSTEMS

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

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

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

Cardiopulmonary resuscitation

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

Oxygen administration

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

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

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

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

Posture

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

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

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

Thermal balance

HYPOTHERMIA

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

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

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

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

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

HYPERTHERMIA

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

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

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

Fluid administration

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

DECOMPRESSION ILLNESS

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

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

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

PULMONARY OEDEMA AFTER NEAR-DROWNING

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

BRAIN OEDEMA

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

Drug administration

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

CORTICOSTEROIDS

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

ANTI-INFLAMMATORIES AND ANALGESICS

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

ANTICOAGULANTS

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

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

LIGNOCAINE

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

DIAZEPAM

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

Recovery to a definitive treatment facility

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

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

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

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

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

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

IN-WATER OXYGEN THERAPY

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

References

1 Edmonds C, Lowry C and Pennefather J. *Diving and subaquatic medicine (2nd edition)*. Oxford: Butterworth-Heinemann, 1992

2 Horne P. *South Australian diving fatalities 1950-1985*. Adelaide: Australian Underwater Federation, 1987

3 Edmonds C and Walker D (1989). SCUBA diving fatalities in Australia and New Zealand. *SPUMS J* 1989;19(3): 94-104

4 McAniff JJ. *United States Underwater Fatality Statistics 1986-1987*. Report URI-SSR-8920. Rhode Island: University of Rhode Island, 1988

5 Bennett PB and Moon RE. *Diving accident management*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1990

6 Williamson J, Acott C, Webb R, et al. Australia's Diver Emergency Service: Analysis of recorded usage over a 35 month period during 19871990. *SPUMS J* 1991; 21(1): 14-21

7 Acott C, Sutherland A and Williamson J. Anonymous reporting of diving incidents. *SPUMS J* 1989; 19: 18-22

8 Haydon JR, Williamson JA, Ansford AJ, Sherif S and Shapter MJ. A scuba-diving fatality. *Med J Aust* 1985; 143: 458-462

9 Arnoux G. Rescue and resuscitation of the unconscious diver. In: *Proc Commission of European Communities, Mine Safety and Health Commission, Congress on Medical Aspects of Diving Accidents.* Luxembourg: 1978

10 Marconi A and Zannini D. A technique for rescue and resuscitation of the unconscious diver. In: *Proc Commission of European Communities, Mine Safety and Health Commission, Congress on Medical Aspects of Diving Accidents.* Luxembourg: 1978

11 Meyers R and Bradley ME. An evaluation of CPR techniques for use in a diving bell. In: Bachrach AJ and Matzen NKM Eds. *Underwater Physiology VII.* Bethesda, Maryland: FASEB, 1981; 887-899

12 Farmer JC, Thomas WG, Youngblood DG and Bennett PB. Inner ear decompression sickness. *Laryngoscope* 1976; 86: 1315-1326

13 Humphrey VJ and Norman JN. *High pressure water jetting injuries. Safety report.* Houston, Texas: Oceaneering Int. Inc., 1980

14 Cox RAF. The use of drugs under pressure. In: Walsh JM Ed. *Interaction of drugs in the hyperbaric environment*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1980; 37-48

15 Cox RAF. *Offshore medicine*. Great Britain: Springer-Verlag, 1987

16 Sisman D. The professional diver's handbook. Houston, Texas: Gulf, 1985

17 Williamson J. *The marine stinger book (3rd edition).* Brisbane: SLSAA, 1985

18 Edmonds C. *Dangerous marine creatures*. Frenchs Forest, NSW, Australia: Reed Books, 1989

19 Sterba JA. Field management of accidental hypothermia during diving. In: Bennett PB and Moon RE, Eds. *Diving accident management.* Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1990; 88-116

20 Pearn J. The management of near drowning. *Brit Med J* 1985; 291: 1447-1452.

21 Danzl DF, Pozos RS, Auerbach PS, et al. Multicentre hypothermia survey. *Ann Emerg Med* 1987; 16(9): 1042-1055

22 Muizelaar JP, Van der Poel HG, Li Z, Kontos HA and Levasseur JE (1988). Pial arteriolar vessel diameter and CO_2 reactivity during prolonged hyperventilation in the rabbit. *J Neurosurg* 1988; 69: 923-927

23 Workman RD. Treatment of bends with oxygen at high pressure. *Aerospace Med* 1968; 39: 1076-1083

24 E.C.R.I. Pulmonary resuscitators. *Health Devices* 1989; 18: 333-352

25 Clark JM. The toxicity of oxygen. *Am Rev Resp Dis* 1974; 110: 40-50

26 Bardin H and Lambertson CJ. *A quantitative method for calculating pulmonary toxicity. Use of the "Unit Pulmonary Toxicity Dose" (UPTD).* Institute for Environmental Medicine Report. Philadelphia:

University of Pennsylvania, 1970
27 Van Allen CM, Hrdina LA and Clark J. Air embolism from the pulmonary vein - a clinical and experimental study. *Arch Surg* 1929; 19: 567-599
28 Atkinson JR. Experimental air embolism. *Northwest Med* 1963; 62: 699-703.
29 Gorman DF and Browning DM. Cerebral vasoreactivity and arterial gas embolism. *Undersea Biomed Res* 1986; 13: 317-335
30 Gorman DF, Browning DM, Parsons DW and Traugott FM. Distribution of arterial gas emboli in the pial circulation. *SPUMS J* 1987; 17(3): 101-115
31 Gorman DF. A proposed classification for the decompression illnesses. In: Francis TJR and Smith DJ, Eds. *Describing dysbarism.* Bethesda, Maryland: Undersea and Hyperbaric Medical Society, in press.
32 Adkisson GH, Macleod MA, Hodgson M, et al. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989; 8655: 119-122
33 Moon RE, Kisslo JA, Massey EW, Fawcett TA and Thiel DR. Patent foramen ovale and decompression illness. *Undersea Biomed Res* 1991; 18 (Suppl): 15
34 Butler BD, Katz J, Leiman BC, Waters RD and Sutton T. Cerebral decompression sickness: bubble distribution in dogs in the Trendelenberg position. *Undersea Biomed Res* 1978; 14 (2) Suppl: 125
35 Dutka AJ. Therapy for dysbaric central nervous system ischaemia: adjuncts to recompression. In: Bennett PB and Moon RE, Eds. *Diving accident management.* Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1990; 222-234
36 Gorman DF and Helps SC. Foramen ovale, decompression sickness, and posture for arterial gas embolism. *SPUMS J* 1989; 19 (4): 150-151
37 Stonier JC. A study in prechamber treatment of cerebral air embolism patients by a first provider at Santa Catalina Island. *Undersea Biomed Res* 1985; 12 (Suppl): 58
38 Edmonds C, Freeman P and Tonkin J. Fistula of the round window in diving. *Trans Am Acad Ophthalmol Otol* 1974; 78: 444-447
39 Freeman P and Edmonds C. Inner ear barotrauma. *Arch Otolaryngol* 1972; 95: 556-563
40 Golden F, Harvey GR and Tipton MJ. Circum-rescue Collapse: Collapse, sometimes fatal, associated with rescue of immersion victims. *J Roy Nav Med Serv* 1991; 77: 139-150
41 Farhi LE and Linnarsson D. Cardiopulmonary readjustments during graded immersion in water at 35°C. *Resp Physiol* 1977; 30: 35-50
42 Feldman KW, Motray JP and Schaller RT. Thermal injury caused by hot pack application in hypothermic children. *Am J Emerg Med* 1985; 3(1): 38-41
43 Lloyd EL. Airway warming in the treatment of accidental hypothermia: A review. *J Wilderness Med* 1990; 1: 65-78
44 Drummond JC and Moore SS. The influence of dextrose administration on neurologic outcome after temporary spinal cord ischaemia in the rabbit. *Anesthesiology* 1989; 70: 64-70
45 Lanier WL, Stangland KJ, Scheithauer BW, et al. The effect of dextrose solution and head position on neurologic outcome after complete cerebral ischaemia in primates: Examination of a model. *Anesthesiology* 1987; 66: 39-48
46 Shanks CA and Sara CA. Temperature monitoring of the humidifier during treatment of hypothermia. *Med J Aust* 1972; 2: 1351-1352
47 Shanks CA and Marsh HM. Simple core rewarming in accidental hypothermia with heat infusion, endotracheal intubation and humidification. *Brit J Anaesth* 1973; 45: 522-525
48 Lash RF, Burdette JA and Ozdil T. Accidental profound hypothermia and barbituate intoxication. A report of rapid core rewarming by peritoneal dialysis. *JAMA* 1967; 201: 269-270
49 Davies DM, Miller EJ and Miller IA. Accidental hypothermia treated by extracorporeal blood-warming. *Lancet* 1967; 1: 1036-1037
50 Pugh LG. Accidental hypothermia in walkers, climbers and campers. *Brit Med J* 1966; 1: 123-129
51 Gilligan JE, Gorman DF and Miller I. Use of an airborne tranportable recompression chamber and transfer under pressure to a major hyperbaric facility. In: Shields TG, Ed. *Proceedings of the XIV annual meeting of the EUBS.* Aberdeen: European Undersea Biomedical Society, 1988
52 *BR 2806.* London: Her Majesty's Stationary Office,
53 Flynn ET, Vorosmarti J and Modell JH. *Temperature requirements for the maintenance of thermal balance in high pressure helium oxygen environments. Report 21-73.* Panama City, Florida: US Navy Experimental Diving Unit, 1974
54 Chamberlain DA. Advanced life support: revised recommendations of the Resuscitation Council. *Brit Med J* 1989; 299: 446-447
55 Modell JH, Calderwood HW, Ruiz BC, et al. Effects of ventilatory patterns on arterial oxygenation after near-drowning in sea water. *Anesthesiology* 1974; 46: 376-384
56 Brierley JB, Brown AW, Meldrum BS and Tiche D. The time course of ischaemic neuronal changes in the primate brain following profound arterial hypertension, air embolism and hypoglycaemia. *Phys Soc* 1980; 59-60.
57 Butler BD and Hills BA. The lung as a filter for microbubbles. *J Appl Physiol* 1987; 47: 537-543
58 Hailer C, Sercombe R, Verrechia C et al. Effect of the muscarinic agonist carbachol on pial arteries *in vivo* after endothelial damage by air embolism. *J Cereb Blood Flow Metab* 1987; 7: 605-611
59 Obrenovitch TP, Kumaroo KK and Hallenbeck JM. Autoradiograph detection of ^{111}Indium-labelled

platelets in brain tissue sections. *Stroke* 1984; 15: 1049-1056

60 Hallenbeck JM, Dutka AJ, Tanishima T, et al. Polymorphonuclear leukocyte accumulation in brain regions with low blood flow during the early postischemic period. *Stroke* 1986; 17: 246-253

61 Helps SC and Gorman DF. Air embolism of the brain in rabbits pre-treated with Merchlorethamine. *Stroke* 1991; 22: 351-354

62 Persson LI, Johnasson BB and Hansson PLA. Ultrastructural studies on blood-brain barrier dysfunction after cerebral air embolism in the rat. *Acta Neuropathol (Berl)* 1978; 44: 53-56

63 Hallenbeck JM, Bove AA and Elliott DH. The bubble as the non-mechanical trigger in decompression sickness. In: Ackles KN, Ed. *Blood bubble interactions*. DCIEM Report 73-CP-960, Downsview, Canada: Defence and Civil Institute of Environmental Medicine, 1973

64 Ward CA, Koheil A, McCulloch D, et al. Activation of complement at the plasma-air or serum-air interface of rabbits. *J Appl Physiol* 1986; 60: 1651-1658

65 Ward CA, McCulloch D, Yee D, Stanga D and Fraser WD. Complement activation involvement in decompression sickness of rabbits. *Undersea Biomed Res* 1990; 18(1): 51-66

66 Childs CM, Ah-See AK and Arfors KE. Dextran administration in the treatment of diving accidents. *Med Aeronaut Spat Med Subaquatique Hyperbare* 1978; 17(65): 81-84

67 Cockett ATK and Nakamura RM. Treatment of decompression sickness employing low molecular weight dextran. *Rev Physiol Subaquatique Med Hyperbare* 1968; 1: 133-134

68 Wells CH, Hilton JG and Hall CB. Microcirculatory effects of intravenous fluid resuscitation in dysbarism. *Undersea Biomed Res* 1978; 5(Suppl): 18

69 Bove AA. The basis for drug therapy in decompression sickness. *Undersea Biomed Res* 1982; 9(2): 91-112

70 Catron PW and Flynn ET. Adjuvant drug therapy for decompression sickness, a review. *Undersea Biomed Res* 1982; 9(2): 161-174

71 Menasche P, Pinard E, Desroches A-M, et al. Fluorocarbons: A potential treatment of cerebral air embolism in open heart surgery. *Ann Thorac Surg* 1985; 40: 494-497

72 Lynch PR, Krasner LJ, Vinciquerra T and Shaffer TH. Effects of intravenous perfluorocarbon and oxygen breathing on acute decompression sickness in the hamster. *Undersea Biomed Res* 1989; 16: 275-281

73 Spiess BD, McCarthy RJ, Tuman KJ, et al. Treatment of decompression sickness with a perfluorocarbon emulsion (FC-43). *Undersea Biomed Res* 1988; 15: 31-37

74 Zhang L-D, Kang J-F and Xue H-L (1989). Ethanol treatment for acute decompression sickness in rabbits. *Undersea Biomed Res* 1989; 16(4): 271-274

75 Eckenhoff RG and Olstad CS. Ethanol and venous bubbles after decompression in humans. *Undersea Biomed Res* 1991; 18(1): 47-52

76 Simcock AD. Sequelae of near drowning. *Practitioner* 1982; 222: 527-530

77 Conn AW, Montes JE, Barker GA and Edmonds JF. Cerebral salvage in near drowning following neurological classification by triage. *Can Anaesth Soc J* 1980; 27: 201-210

78 Neuman TS. Near drowning. In: Bove AA, Davis JC, Eds. *Diving medicine*. Philadelphia: Saunders, 1990; 105-114.

79 Dutka AJ, Hallenbeck JM and Kochanek P. A brief episode of severe arterial hypertension induces delayed deterioration of brain function and worsens blood flow after transient multifocal cerebral ischaemia. *Stroke* 1987; 18: 386-95

80 Helps SC, Meyer-Witting M, Reilly PL and Gorman DF. Increasing doses of intracarotid air and cerebral blood flow in rabbits. *Stroke* 1990; 21: 1340-1345.

81 Helps SC, Parsons DW, Reilly PL and Gorman DF. The effect of gas emboli on rabbit cerebral blood flow. *Stroke* 1990; 21: 94-99

82 Hossman K-A and Fritz H. Coupling function, metabolism, and blood flow after air embolism of the cat brain. *Adv Neurol* 1978; 20: 255-262

83 Oaks DD, Sherck JP, Maloney JR and Charters AC. Prognosis and management of victims of near drowning. *J Trauma* 1982; 22: 544-549

84 Pearson RR and Goad RF. Delayed cerebral edema complicating cerebral arterial gas embolism: case histories. *Undersea Biomed Res* 1982; 9: 283-296

85 Hills BA and James PB. Microbubble damage to the blood-brain barrier: relevance to decompression sickness. *Undersea Biomed Res* 1991; 18(2): 111-116

86 Nishiomoto K, Wolman M, Spatz M and Klatzo I. Pathophysiologic correlations in the blood-brain-barrier damage due to air embolism. *Adv Neurol* 1978; 20: 237-244.

87 Hallenbeck JM, Leitch DR, Dutka AJ and Greenbaum LJ Jr. The amount of circumscribed brain edema and the degree of postischemic neuronal recovery do not correlate well. *Stroke* 1982; 13: 797-804

88 Fishman RA. Brain edema. *New Eng J Med* 1975; 293: 706-711

89 Gastant JL, Michel B, Sabet Hassan S et al. Electroencephalography in brain edema. *Electroencephalography and Clinical Neurophysiology* 1979; 46: 239-255.

90 Marmarou A, Takagi H and Shulman K. Biomechanics of brain edema and effects on local cerebral blood flow. *Adv Neurol* 1980; 28: 345-358

91. Paulson OB. Intracranial hypertension. *Anaesthesiology* 1972; 36: 1-3

92 Penn RD. Cerebral edema and neurological function: CT, evoked responses, and clinical examination. *Adv Neurol* 1980; 28: 383-394

93 Rapoport SI and Thompson HK. Osmotic opening of the blood-brain barrier in the monkey without associated neurological deficits. *Science* 1973; 180: 971

94 Fritz H and Hossmann k-A. Arterial air embolism in the cat brain. *Stroke* 1979; 10: 581-589

95 Simms NM, Kush GS, Long DM, Loken MK and French LA. Increase in regional cerebral blood flow following experimental arterial air embolism. *J Neurosurg* 1971; 34: 665-671

96 Fructus X. Treatment of serious decompression sickness. In: Davis JC, Ed. *Treatment of serious decompression illness and arterial gas embolism*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1979; 37-43

97 Kizer KW. Corticosteroids in treatment of serious decompression sickness. *Ann Emer Med* 1981; 10: 485-488

98 Francis TJR and Dutka AJ. Methylprednisolone in the treatment of acute spinal cord decompression sickness. *Undersea Biomed Res* 1989; 16: 165-174.

99 Gorman DF. Arterial gas embolism as a consequence of pulmonary barotrauma. In: Desola J, Ed. *Diving and hyperbaric medicine*. Barcelona, EUBS, 1984; 348-386

100 Bean JW. Factors influencing clinical oxygen toxicity. *Annals N.Y. Acad Sciences* 1965; 117: 745-755

101 Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone in the treatment of acute spinal-cord injury. *N Engl J Med* 1990; 322: 1405-1411

102 Hallenbeck JM, Leitch DR, Dutka AJ, Greenbaum LJ and McKee AE. Prostaglandin I_2, indomethacin, and heparin promote postischemic neuronal recovery in dogs. *Ann Neurol* 1982; 12: 145-156.

103 Kochanek PM, Dutka AJ, Kumaroo KK and Hallenbeck JM. Effects of prostacyclin, indomethacin and heparin on cerebral blood flow and platelet adhesion after multifocal ischemia of canine brain. *Stroke* 1988; 19: 692-699.

104 Philp RB, Gowdey CW and Prasad M. Changes in blood lipid concentration and cell counts following decompression sickness in rats and the influence of dietary lipid. *Can J Physiol Pharm* 1967; 45: 1047-1059.

105 Cockett ATK, Pauley SM and Roberts AP. Advances in treatment of decompression sickness: an evaluation of heparin. In: Fructus X, Ed. *Third international congress on hyperbaric and underwater physiology*. Paris: DOIN Editeurs, 1972; 156-160

106 Laborit H, Barthelemy L and Perrimond-Trouchet R. Action de l'heparine dans le traitement des accidents de decompression. *Aggressologie* 1961; 2: 229-236

107 McCormick JG, Holland WB, Brauer RW and Holleman IL. Sudden hearing loss due to diving and its prevention with heparin. *Otolaryngol Clin N Am* 1975; 8: 417-430

108 Philp RB. The ameliorative effect of heparin and depolymerised hyalurinate on decompression in rats. *Can J Physiol Pharmacol* 1964; 42: 319

109 Kindwall EP and Margolis I. Management of severe decompression sickness with treatment ancillary to recompression: Case report. *Aviat Space Environ Med* 1975; 46: 1065-1068

110 Reeves E and Workmann RD. Use of heparin for the therapeutic prophylactic treatment of decompression sickness. *Aerosp Med* 1971; 42: 20-23

111 Inwood MJ. Experimental evidence in support of the hypothesis that intravascular bubbles activate the haemostatic process. In: Ackles KN, Ed. *Symposium on blood-bubble interactions in decompression sickness. DCIEM Report 73-CP-960*. Downsview, Canada: Defence and Civil Institute of Environmental Medicine, 1973; 171-200

112 Elliott DH, Hallenbeck JM and Bove AA. Acute decompression sickness. *Lancet* 1974; 2: 1193-1199

113 Palmer AC, Blakemore WF and Payne JE. Decompression sickness in the goat: nature of brain and spinal cord lesions at 48 hours. *Undersea Biomed Res* 1978; 5(3): 275-286

114 Waite CL, Mazzone WF, Greenwood ME and Larsen RT. *Cerebral air embolism. 1. Basic studies. US Navy Submarine Medical Centre report No. 493*. 1967

115 Landolt JP, Money KE, Topliff EDL et al. Pathophysiology of inner ear dysfunction in the squirrel monkey in rapid decompression. *J Appl Physiol: Respir Environ Exercise Physiol* 1980; 49: 1070-1082

116 Evans DE, Kobrine AI, LeGrys DC, et al. Protective effect of lidocaine in acute cerebral ischaemia induced by air embolism. *J Neurosurg* 1984; 60: 257-263

117 Evans DE, Catron PW, McDermot JJ, et al. Therapeutic effect of lidocaine in experimental cerebral ishaemia induced by air embolism. *J Neurosurg* 1989; 70: 97-102

118 McDermott JJ, Dutka AJ, Evans DE and Flynn ET. Treatment of experimental cerebral air embolism with lidocaine and hyperbaric oxygen. *Undersea Biomed Res* 1990; 17(6): 525-534

119 Kindwall EP. Decompression sickness. In: Davis JC and Hunt TK, Eds. *Hyperbaric oxygen therapy*. Bethesda, Maryland: Undersea Medical Society, 1977; 125-140

120 Melamed Y, Shearman D, Wiler-Ravell D and Kerem D. The transportable recompression chamber as an alternative to delayed treatment in serious diving accidents. *Aviat Space Environ Med* 1981; 52: 480-484

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REFLECTIONS ON DIVER FATALITY AND SAFETY STATISTICS

Drew Richardson

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

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

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

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

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

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

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

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

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

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

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

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