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. Sensory deprivation and disorientation have caused diving fatalities, especially in caves.

Analyses of fatal \cite{1} and non-fatal accidents \cite{5} produce limited data, and consequently a multi-centre diving incident monitoring study is underway \cite{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.

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. 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.

SATURATION SYSTEMS

Diving accidents, coincident injuries or illnesses such as vestibular decompression illness, water-jet injuries and pseudomonas otitis externa often require medical intervention and a diving physician or medical
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 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 and accompanied by appropriate education.

Prolonged administration of oxygen will require interruptions for air breathing (air breaks) to retard pulmonary damage. 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, 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. Van Allen et al. showed that arterial bubbles did not embolise the brain of head-down dogs. These results have been confirmed in both cats and rabbits. 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; a significant arterial gas embolism probably occurs in decompression sickness by arterialisation of venous bubbles and a head-down posture will enhance this by increasing venous return; in small vessels bubbles travel with blood flow; 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. These observations must be balanced against the lethal deterioration seen in some divers suspected of arterial gas embolism when they have been sat up, 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. 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) 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. 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. Handling of the diver must be minimised to reduce the risk of cardiac arrhythmias. Maneuvers 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. Neither EAR nor ECC should be withheld; standard resuscitation routines should not be altered and resuscitation must continue until the diver is rewarmed.

Wet clothing must be removed, including woollen and synthetic garments which lose insulation when wet. 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 re-warming in the field is poorly tolerated, although bath temperatures can be rapidly increased once the diver is immersed; 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. Warming the inhaled air is advocated, but has not been shown to have any benefit over dry insulation alone in adults.

Intravenous rehydration should involve warmed fluids, but should not include dextrose solutions if brain injuries are suspected 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, infusion of warmed intravenous fluids, peritoneal lavage, extracorporeal blood warming and thoracotomy and bathing the heart with warm fluids. 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. Given the limited inside space, the solution has been to protect the vessel from the sun and 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.

Deep oxygen-helium saturation exposures are also liable to hyperthermic stress 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. 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 or pulmonary arterioles or indirectly by damaging endothelial cells, activating and causing focal accumulations of platelets and polymorphonuclear leucocytes, causing extravasation and haemoconcentration and activating a variety of plasma proteins including those of the coagulation system, complement proteins, kinins and lipoproteins. There is both theoretical advantages (oncotic and anticoagulant), a practical advantage has not been shown. Glucose-containing solutions should be avoided as they may lead to a worsening of neurological lesions. 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. 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, 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. 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. Hypoxia from drowning needs 100% oxygen administration and, if necessary, application of positive end-expiratory pressure (PEEP). If decompression illness is not suspected in near-drowning, then oxygen and intravenous fluids should be given to maintain normoxia and normotension.

**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. Bubbles do increase the permeability of the blood-brain-barrier but this is transient (several hours) and in air embolised dogs brain function and water content did not correlate well. 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. In contrast, there is usually a good correlation between brain function and blood flow after air embolism and consequently the fluid load should be adequate to maintain cerebral perfusion. Given that autoregulation of brain blood flow is lost after air embolism, 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 benefit99 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 Pg12 (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 anecdotal70,109 support for use in decompression illness treatment. However, heparin given either prophylactically or therapeutically to dogs with a decompression illness was not beneficial110 and other animal-models of the decompression illnesses have either shown no advantage for heparin111 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 prophylactically116 or therapeutically35,117 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 Israel120 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 m sw depth treatment), and retrieval to a recompression chamber. The Edmonds’ and a delay of greater than 24 to 36 hours will occur before supportable only if the above contraindications are absent.

Other essential that adequate communication with the definitive facility precede retrieval of the injured diver. Other measures such as resuscitation, posture, oxygen administration, thermal protection for the injured diver and the attendant; a cradle or chair that can be lowered to and kept at 9 m depth; fullface mask and dedicated oxygen delivery system; a thermal protection for the injured diver and the attendant; 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.

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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.

In the past 23 years a total of 2,404 U.S. recreational scuba fatalities have been recorded. In the first 10 years of fatality reporting, the average diver fatality per year was 123. Approximately half of all recorded recreational scuba fatalities occurred from 1970 through 1979, when both the dive industry and number of divers were smaller. Today, the average number of fatalities is approximately 104 deaths per year, 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. Figure 1 shows the total numbers of U.S. recreational diving fatalities by year, as reported by NUADC and DAN. 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. 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. 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.

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.