

# 27 UNDERWATER EXPEDITIONS

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The increasing use of diving as a means of exploration in the underwater environment has resulted in a substantial rise in the popularity of underwater expeditions. Many such projects have additional scientific or ecological objectives and may involve large numbers of participants over many months or years. It is therefore unsurprising that most expeditions desire or require medical support.

Diving is an equipment-centred activity and, because of this, diving expeditions usually remain within closer reach of “civilisation” than many others. A notable feature of diving-related expeditions is that the variety of illnesses caused uniquely by hyperbaric exposure requires a recompression chamber for definitive treatment; this is not an item easily carried in a medical kit.

## **Fitness to dive**

Participants in a diving expedition may have diving qualifications from any of a number of training organisations, which have varying requirements for fitness to dive. In the UK the medical examination for commercial divers is much more comprehensive than for sports divers, reflecting the occupational nature of the risk. In recent years the UK’s Health and Safety Executive (HSE) has adopted a pragmatic risk assessment approach to diving regulations and fitness to dive; the standards required for a North Sea saturation diver are not necessarily those required of an underwater cameraman filming marine life in a tropical aquarium, although both may be employed as divers. A similar approach should be adopted for expedition divers with the obvious proviso that diving, for any form of reward, brings such diving within the jurisdiction of the HSE or analogous industrial health organisation. Some general standards apply, which may be modified according to the diving activity; these are shown in Table 27.1.

TABLE 27.1 GENERAL CONTRAINDICATIONS TO DIVING

Category	Condition	Notes
<b>Absolute</b>	Epilepsy	
	Cardiac disease	Ischaemic or arrhythmogenic
	Obstructive airway disease	COPD, emphysema, asthma
	Pregnancy	
	Middle-ear disease	
<b>Relative</b>	Insulin-dependent diabetes mellitus	In the context of an expedition
	Obesity	e.g. BMI > 35kg/m <sup>2</sup>
	Lack of physical fitness	e.g. $\dot{V}O_{2\max} < 40\text{ml/kg per min}$ or equivalent
	Psychiatric disease	Requiring medication (e.g. antidepressants)
<b>Temporary</b>	Previous penetrating chest wound/lung injury	Unless cleared by a diving physician
	Acute upper respiratory tract infection	Until resolved
	Tympanic membrane (TM) barotrauma	TM perforation 4 weeks, otherwise 5–10 days

Expert and impartial advice about fitness to dive, whether for recreational, expeditionary, commercial or military diving, can be obtained from the Institute of Naval Medicine, Alverstone, Gosport, Hants PO12 2DL.

### Diving activities and techniques

Most expeditionary diving has used conventional sport diving techniques (open-circuit scuba). As sport diving evolves to use more advanced procedures, such as Nitrox, helium-based mixed gas and rebreathers, expedition organisers increasingly seek to apply these techniques (often collectively called “technical diving”) for exploration purposes; indeed, many aspects of technical diving were developed by explorers for specific projects. The decompression advantages of Nitrox, the deep diving capabilities of Trimix/Heliox and the logistical benefits of rebreathers mean that expeditions are likely to use one or more of these techniques. Expedition medical officers should be familiar with at least the principles, if not the details, of their use.

#### Nitrox

Nitrox strictly means any gas mixture containing air and oxygen, but in practice is used for air enriched with oxygen (i.e. > 21% oxygen). The lower partial pressure of nitrogen and higher partial pressure of oxygen reduces decompression requirements

compared with air. Alternatively Nitrox can be used with air decompression tables or computers for an additional safety margin. The depth at which a Nitrox mix can be used is limited by the maximum safe partial pressure of oxygen that can be breathed with an acceptably low risk of central nervous system (CNS) oxygen toxicity (see below). For simple diving, mixtures of 25–35% oxygen are usually used, but higher percentages up to 100% oxygen may be used during decompression to speed up elimination of inert gas (nitrogen or helium).

### *Helium (Trimix and Heliox)*

At depths beyond 30 metres, divers may become impaired by nitrogen narcosis. This becomes a substantial problem at depths greater than 50 metres and has led to the use of helium and oxygen mixtures (Heliox) and Trimix (a mixture of helium, nitrogen and oxygen) by sports divers wishing to dive deeper. Prevention of CNS oxygen toxicity means that the gas breathed on the bottom is low in oxygen and therefore is inefficient for decompression purposes; divers therefore typically breathe one or more different mixtures, often Nitrox, for decompression. This necessitates carrying additional cylinders on the dive unless they can be placed at a reliable retrieval point (e.g. a cave entrance). The breathing of a decompression gas at depth can cause acute oxygen toxicity with possibly lethal consequences (such as an underwater convulsion).

### *Rebreathers*

Traditional open-circuit scuba is inefficient, as all the gas breathed is wasted into the water despite having had very little of its oxygen used. The concept of recycling the gases via a reservoir ('counterlung') is as old as diving but until recently such units were available only for use by military divers. Rebreathers employ a canister of soda lime to remove waste carbon dioxide; additional oxygen is added to the breathing loop either at a constant rate (semi-closed circuit) or by a computer-controlled valve only as it is needed to maintain a constant partial pressure of oxygen (closed circuit).

Rebreathers have a number of advantages, the most important of which is their low gas usage; unlike open-circuit scuba this is largely independent of depth. It is quite possible to build a compact closed-circuit rebreather with an underwater duration of 24 hours *at any depth*. This enormous logistical advantage over open-circuit systems, an advantage that increases with greater depths, means rebreather use on advanced diving expeditions will inevitably become more common in the future. Other benefits are that the inhaled gas is already warmed and humidified, minimising respiratory heat loss, and the lack of bubbles – necessary for covert military operations, but also useful for underwater photographers and videographers.

The main disadvantage of rebreathers, apart from their expense, is that dangerous malfunctions can occur without the diver being aware of the problem (e.g. too low or high a partial pressure of oxygen) and which may cause diver loss of consciousness under water; without a full-face mask drowning is likely. Such risks are minimised in



Figure 27.1 *Decompression after a deep mixed-gas dive (A. Pitkin)*

the military setting by rigorous training and very high maintenance standards; most non-military users of rebreathers do not have the same level of training or technical support.

#### *Overhead environments (cave, wreck and ice diving)*

Most recreational dive training is based upon the premise that a diver who has a major problem can ascend directly to the surface. Inside a cave or wreck this is clearly impossible and therefore any problem that occurs must be solved under water. This requires training and additional equipment which is based on the principle of redundancy: any vital piece of equipment is backed up by an alternative so that a single failure under water will not pose an immediate threat to the diver's well-being. In cave diving, where caves may be penetrated under water for distances of several miles or more, elaborate and formalised techniques and equipment configurations have been evolved to minimise the chance of a problem becoming fatal. These include the "thirds rule" (using only one third of available gas for cave ingress, leaving a third for exit and a third for emergency use), maintaining a continuous guideline to the surface and having at least two back-up lights. Wreck penetration often uses these procedures but poses somewhat different hazards of entanglement and silting. Ice diving requires similar techniques but poses additional problems from the effects of cold on equipment and humans. Decompression diving tends to be the norm in all types of



Figure 27.2 *Cenote Carwash, a popular cave diving site in Mexico (A. Pitkin)*

overhead environment diving, and open-water dives with a significant decompression obligation may be regarded as having a “virtual” decompression ceiling, necessitating a similar approach to dive planning and equipment configuration.

## **MEDICAL PROBLEMS SPECIFIC TO DIVING EXPEDITIONS**

Participants in diving expeditions are exposed to general hazards related to being in or near the water (e.g. hypothermia, near-drowning) as well as those related exclusively to diving. These arise either as a result of exposure to increased ambient pressure under water or to the use of one of the many forms of underwater breathing systems available.

### **General hazards**

Hypothermia and near-drowning are dealt with in Chapters 25 and 29 respectively. Divers are vulnerable to trauma, particularly while at the surface and boarding a boat, especially in heavy seas. A small but persistent number of fatalities occur from propeller injuries to divers from surface craft. Hazards posed by dangerous marine life are covered in Chapter 20.

**Decompression illness (DCI)**

This term covers clinical syndromes caused by intravascular or extravascular gas bubbles generated during ascent from depth. Breathing compressed gas at depth results in nitrogen (or helium) being dissolved in blood and body tissues at a rate determined by partial pressure gradient (i.e. depth), temperature and molecular weight (Graham's law). The time spent at depth obviously directly affects the amount absorbed. In addition delivery of gas to body tissues is affected by perfusion, so that lipid-rich and well-perfused tissues such as brain and spinal cord may have relatively large amounts of inert gas dissolved in them. When the diver ascends the gas in the tissues becomes 'supersaturated' and gaseous bubbles are formed, which may cause clinical effects in a range between none and physiological dysfunction severe enough to result in death, and which is usually referred to as decompression sickness (DCS). Traditionally DCS has been divided into type 1 (mild) and type 2 (severe) forms, a classification still used in the United States but falling into disuse in Europe where a descriptive classification is now favoured (Table 27.2).

**TABLE 27.2 DESCRIPTIVE CLASSIFICATION OF DECOMPRESSION ILLNESS**

<i>Manifestations</i>	Neurological pain Limb pain Girdle pain Pulmonary Cutaneous Lymphatic Constitutional	<i>Evolution</i>	Progressive Relapsing Static Spontaneously improving Resolved
<i>Inert gas load</i>	Depth/time profile Decompression obligation Gas mixture(s) used	<i>Evidence of barotrauma</i>	Lung Sinus Ear Dental

A different mechanism of injury is expansion of gas in the diver's lungs during a rapid ascent, which enters the pulmonary veins and is carried through the left side of the heart to the brain causing cerebral arterial gas embolism (AGE). In practice a distinction between this and the effects of dissolved inert gas (DCS) is very difficult and now both entities are commonly encompassed within the term decompression illness. This can be usefully divided into neurological (75%) and

**TABLE 27.3 COMMON CLINICAL SYNDROMES SEEN IN DECOMPRESSION ILLNESS**

<i>Clinical scenario</i>	<i>Cause</i>	<i>Management</i>
Rapid ascent to surface <ul style="list-style-type: none"> <li>• Loss of consciousness or seizure (may be within 10 min of surfacing)</li> <li>• Weakness of half the body (one arm and leg: hemiparesis)</li> <li>• Confusion/cognitive impairment</li> </ul>	DCI (cerebral arterial gas embolism)	<ul style="list-style-type: none"> <li>• Resuscitation: ABC</li> <li>• Give oxygen</li> <li>• Lie flat</li> <li>• Immediate recompression</li> </ul>
Rapid ascent to surface <ul style="list-style-type: none"> <li>• Breathlessness</li> <li>• Chest pain</li> </ul>	Pneumothorax	<ul style="list-style-type: none"> <li>• Give oxygen</li> <li>• Vent chest with intravenous cannula (or chest drain)</li> <li>• See Chapter 14</li> </ul>
Rapid ascent/omitted decompression <ul style="list-style-type: none"> <li>• Back pain</li> <li>• Weakness/numbness of legs</li> <li>• Loss of bladder control</li> </ul>	Neurological DCI (spinal cord)	<ul style="list-style-type: none"> <li>• Give oxygen</li> <li>• Give fluids</li> <li>• Urgent recompression</li> </ul>
After any dive <ul style="list-style-type: none"> <li>• Tingling/numbness of fingers (both sides, often asymmetrical)</li> <li>• Weakness, altered reflexes</li> <li>• Inco-ordination</li> </ul>	Neurological DCI	<ul style="list-style-type: none"> <li>• Give oxygen</li> <li>• Give fluids</li> <li>• Recompression as soon as possible</li> </ul>
After any dive <ul style="list-style-type: none"> <li>• Dull pain in/near the shoulder or elbow, often improved slightly by movement</li> <li>• Weakness/numbness of same arm or hand (easily overlooked)</li> </ul>	Limb pain DCI	<ul style="list-style-type: none"> <li>• Give oxygen</li> <li>• Give fluids</li> <li>• Recompression as soon as possible</li> </ul>
Possible difficulty equalising ears <ul style="list-style-type: none"> <li>• Vertigo, unsteadiness</li> <li>• Severe nausea and vomiting</li> <li>• Nystagmus (jerking movement of eyes)</li> </ul>	Inner-ear DCI or barotrauma	<ul style="list-style-type: none"> <li>• Give oxygen</li> <li>• Give intravenous fluids</li> <li>• Recompression as soon as possible</li> </ul>
After multiple or deep/long dives <ul style="list-style-type: none"> <li>• Extreme fatigue, sleepiness</li> <li>• Malaise, shivers</li> <li>• Nausea, anorexia</li> </ul>	Constitutional DCI	<ul style="list-style-type: none"> <li>• Give oxygen</li> <li>• Give fluids</li> <li>• Consider recompression</li> </ul>

non-neurological DCI (25%). The former can cause permanent disability and should be treated aggressively with recompression therapy, even if this is delayed. The latter will resolve eventually without specific treatment although recompression will usually result in more rapid recovery. About a quarter of cases of neurological DCI will be left with a permanent neurological deficit even after treatment.

Symptoms and signs of decompression illness are extremely variable and may be bizarre; unexplained symptoms occurring after diving should be assumed to be due to decompression illness until proven otherwise. Sensory symptoms (tingling, numbness, particularly of the hands) are seen in 60% of cases; motor symptoms (weakness, inco-ordination) are present in 30% of cases and imply more severe disease. There are a number of common patterns of decompression illness which are shown in Table 27.3. *It is important to remember that the sine qua non of DCI is that it occurs during or after a decompression* (which may be a previous dive); symptoms appearing during the descent or bottom phases of a dive need an alternative explanation. A relatively common (15%) but easily overlooked feature of DCI is that of disordered thought or personality change, which may result in a diver inappropriately refusing treatment or evacuation.

First aid treatment of decompression illness is simple, but relies on recognising that there is a problem. The diver may be unable to self-diagnose the problem due to cognitive impairment. Concealment and denial are common because of the 'stigma' attached to DCI by many divers.

### *First aid treatment of DCI*

1. Resuscitation: ABC.
2. Administer 100% oxygen. This is most efficiently done with a demand regulator; constant-flow systems rarely deliver close to 100%, even when they incorporate a reservoir bag.
3. Keep the diver lying flat. This is most important for cerebral gas embolism, but is also useful in other forms of DCI.
4. Give fluids, oral if possible, otherwise intravenously. Immersion in water, cold and increased hydrostatic pressure all contribute to dehydration after every dive. Bubbles in the bloodstream cause blood vessels to become 'leaky' allowing further fluid loss from the blood into the tissues. If the diver is alert and able to speak give fluids orally; if there is any doubt about the airway nothing should be given by mouth.
5. Keep the diver comfortably cool, but not to the point of shivering. Most diving casualties have a degree of hypothermia, but in tropical regions conditions on the surface can get extremely hot; the diver should be kept as cool as is practicable.
6. Seek expert advice and consider evacuation to recompression facility.

Further treatment of a casualty with decompression illness depends on the nature of the problem and the logistics of transfer to a recompression chamber. It is essential to consider the possibility of urgent evacuation during the planning phase of a diving expedition, as few are fortunate enough to have a recompression chamber on site. Whether an individual casualty needs urgent recompression treatment can be a difficult decision; expert advice can and should be obtained 24 hours a day from the Royal Navy's Duty Diving Medical Officer at the Institute of Naval Medicine. *Any diver with neurological symptoms should be recompressed to minimise the chance of permanent neurological damage*; this applies even if symptoms have disappeared while breathing 100% oxygen. It is very common for symptoms to reappear hours or even days later and vital time for transport to a chamber may have been lost. The effectiveness of oxygen as a first aid measure does not mean that hyperbaric treatment is not necessary; this phenomenon of the "oxygen ostrich" has been responsible for poor eventual outcome in a number of DCI cases.

Expedition organisers may raise the possibility of using a portable monoplace chamber (such as the Hyperlite) for treatment of DCI in the field. The main problem with these is that they do not allow access to the patient during treatment and this will be of greatest concern inpatients with severe DCI who should be treated as quickly as possible. This is the "portable paradox": cases that most need to be treated immediately are least suitable for treatment in monoplace chambers that can be used on site. Most experienced diving physicians would therefore advise continuing resuscitation with oxygen and fluids while awaiting evacuation to a multiplace chamber.

Similar considerations apply to in-water recompression. There may be situations where the potential benefit outweighs the risks but the hazards are not trivial and should be considered very carefully. The casualty is exposed to hypothermia, further dehydration and oxygen toxicity; additional symptoms can evolve which may not be noticed under water or which may be life threatening (e.g. loss of consciousness). In-water recompression has on occasion been used successfully in remote locations for divers with stable DCI, but the technique remains controversial.

Other treatments that may be of use in decompression illness include non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen or diclofenac. These can be very useful as an adjunctive treatment for limb pain DCI but, like aspirin, they should be avoided in neurological DCI where the antiplatelet effects could promote secondary haemorrhage in the spinal cord. Another drug that appears to have been useful in occasional cases of neurological DCI is lignocaine (lidocaine), given intravenously in the same doses used for ventricular arrhythmias. None of these experimental treatments should interfere with the proven ones, which are oxygen and fluids.

### **Nitrogen narcosis**

At high partial pressures under water nitrogen has effects similar to anaesthetic



Figure 27.3 A modern multiplace hyperbaric chamber (A. Pitkin)

agents, causing a variety of symptoms including tunnel vision, euphoria, apprehension, tinnitus, inability to carry out complex tasks, loss of co-ordination, drowsiness and eventually loss of consciousness. Hypercapnia (excessive  $\text{CO}_2$ ) and exertion increase the effects, which can vary considerably between divers and dive sites (worse in cold, dark, poor-visibility conditions). As the symptoms invariably resolve rapidly on ascent the main problem is the impairment of the diver's performance, which may lead to other problems such as DCI or near-drowning.

### **Hypercapnia (carbon dioxide poisoning)**

Carbon dioxide ( $\text{CO}_2$ ) is the main waste product of the body's use of oxygen, and is removed by breathing out of the lungs. A sensitive control mechanism exists to regulate breathing directly from the partial pressure of  $\text{CO}_2$  in the blood, which is thus kept remarkably constant, even during extreme exertion. The diver can also learn to override the 'urge to breathe' (as in free diving). A diver who is exerting hard and trying to eke out a limited supply of air through poor-quality breathing apparatus is at risk of hypercapnia. Rebreather divers whose absorbent is exhausted (more quickly in cold conditions) and cave divers breathing from exhausted air spaces in flooded caves can be exposed directly to high levels of carbon dioxide. The symptoms include headache, flushing, palpitations, drowsiness, and potentiation of both oxygen toxicity and nitrogen narcosis. It is often said that hypercapnia causes breathlessness: this

is *rare* in the diving scenario where the sensation of breathlessness appears to be inhibited by high partial pressures of oxygen. Under water the diver should stop exerting and abort the dive if symptoms do not resolve rapidly. At the surface symptoms will quickly disappear once the diver is breathing fresh air or oxygen.

### **Oxygen toxicity**

Oxygen can have toxic effects at the partial pressures encountered in diving, but oxygen toxicity rarely occurs using air. This is because the threshold for acute oxygen toxicity (about 1.6 atmospheres absolute, ATA) occurs at 66 metres, where nitrogen narcosis is likely to be a much more serious problem. Use of Nitrox mixtures means that this partial pressure may be reached at much shallower depths. The risk is increased by time of exposure, immersion in water, exertion and extremes of temperature. It is decreased by air breaks and avoidance of hypercapnia and heavy exertion. Acute oxygen toxicity mainly affects the central nervous system, causing visual disturbances, hearing disturbances, muscle twitching (especially in the face and diaphragm), nausea and convulsions; these may occur without any prior warning and have often been fatal when they have occurred under water. Rebreather divers may be at risk from malfunctions, inappropriate choice of gas mixture (semi-closed circuit) or rapid descents (closed circuit).

If a diver has any symptoms that suggest oxygen toxicity, he or she should stop any exertion and either ascend or change to a gas mixture with a lower oxygen content. For closed-circuit rebreathers this may entail flushing the loop with diluent. Rescue of a diver suffering a convulsion under water is rarely ultimately successful unless the diver is using a full-face mask. The initial 30 seconds of a convulsion typically comprises a tonic phase where the diver is rigid and has a closed glottis; decompression to the surface during this phase should be avoided as it is likely to cause arterial gas embolism. During the clonic phase (rhythmic jerking movements) the diver can be surfaced and resuscitated. It should then be assumed that the diver has suffered arterial gas embolism and should be recompressed as a matter of urgency; in the meantime oxygen administered at the surface is unlikely to cause further problems.

Pulmonary oxygen toxicity, whilst occasionally seen during hyperbaric oxygen treatment of a diving casualty, is not a problem in self-contained diving.

### **Hypoxia**

There are few situations where a diver is exposed to hypoxia, but like oxygen toxicity it can cause sudden loss of consciousness under water without warning. Deep divers use mixtures with oxygen fractions of less than 12% at great depths which if breathed at the surface could cause hypoxia. Almost any mixture used in self-contained diving will be safe to breathe at depths of 10 metres or more. Of more concern are rebreather divers whose variable oxygen consumption (by exertion) must be matched by oxygen added to the loop at a steady flow rate in semi-closed systems and according to

directly measured loop partial pressure in closed-circuit systems. Hypoxia is notorious for its insidious effects on mental function; lack of insight is characteristic. Other features are euphoria, loss of fine motor control and unconsciousness. These are likely to occur when the inspired partial pressure of oxygen is less than 0.1ATA. Like oxygen toxicity loss of consciousness under water due to hypoxia may easily be fatal. Treatment once on the surface is with oxygen, or air if oxygen is unavailable.

### **Barotrauma**

The volume of an enclosed gas-filled space varies inversely with pressure (Boyle's law). If air-containing spaces within the body are not equalised with the pressurised breathing gas on descent they will be compressed ('squeezed'). In practice this causes problems only with normally rigid cavities such as the middle ear and facial sinuses, although abnormal air spaces such as under dental fillings can also be very painful.

**Middle-ear barotrauma** affects the tympanic membrane and is extremely common. Even mild eustachian tube dysfunction may lead to painful stretching of the tympanic membrane and if the diver continues to descend it will perforate. Characteristically this results in a sudden disappearance in the pain and sometimes a salty taste in the throat (blood or salt water). Cold water entering the middle ear may cause temporary vertigo. On examination the tympanic membrane will appear reddened and usually the perforation can be seen. A fluid level, sometimes haemorrhagic, is often present behind the tympanic membrane. Simple non-perforating barotrauma heals within a few days, but a diver with a perforation should not dive again for at least 4 weeks.

**Inner-ear barotrauma** causes similar but more severe symptoms. High pressures in the skull from overenthusiastic Valsalva manoeuvres can rupture the round window of the cochlea (the organ of hearing), resulting in leakage of perilymph and damage to the cochlea and vestibular apparatus (organ of balance). Vertigo is severe and usually associated with a hearing loss; these symptoms continuing after a dive are a sinister sign and expert advice should be sought. Occasionally the inner ear is directly affected by decompression illness and distinguishing this from inner-ear barotrauma can be impossible. One other cause of transient vertigo that can occur during ascent is due to the eustachian tubes venting air at different rates (alternobaric vertigo). It always disappears within minutes.

**Pulmonary barotrauma** (affecting the lungs) is the most serious form and can be fatal. If gas in the lungs is not able to vent freely during ascent it can escape through the delicate lung tissue: into the pleural cavity, causing a pneumothorax (chest pain, breathlessness); into the mediastinum, causing pneumomediastinum (central chest pain, voice change, neck swelling); and, most seriously, into the bloodstream, causing gas embolism to the brain and sometimes the coronary arteries that supply the heart with blood. Typically the diver has made a rapid ascent and is unconscious on surfacing or loses consciousness shortly thereafter. Convulsions are common. Less se-

vere embolism causes stroke-like symptoms with one-sided weakness (hemiparesis) and speech/language difficulties. Treatment is discussed above along with other forms of decompression illness.

### **Carbon monoxide poisoning**

Carbon monoxide poisoning is now rare since most divers are aware of the problem of compressor air intakes being close to or downwind of an exhaust. The symptoms appear at depth and can be bizarre. Jacques Cousteau's account of his near-fatal dive in the Fontaine de Vaucluse in *The Silent World* is characteristic of the disorientation, loss of sense of time, inco-ordination, headache and vomiting that carbon monoxide poisoning causes. The 'cherry-red' coloration described by some authors is almost never seen. Treatment is with 100% oxygen and persisting symptoms are an indication for hyperbaric oxygen treatment.

### **Expert advice**

Diving diseases are complex and can be difficult to manage even by experts with long experience in the field. Expedition medical officers are urged to make use of the excellent service provided by Royal Navy diving physicians at the Institute of Naval Medicine in Alverstoke, Hampshire. At least one Royal Navy diving physician is available 24 hours a day, 365 days a year, for advice on management of a diving accident anywhere in the world. Contact them on:

+44 7831 151 523

Less urgent enquiries (for example on fitness to dive) can also be made through the above number or by letter to:

Senior Medical Officer (Diving Medicine)  
Institute of Naval Medicine  
Alverstoke  
Gosport  
Hants PO12 2DL

## **ADDITIONAL MEDICAL SUPPLIES FOR DIVING EXPEDITIONS**

### **Base camp medical kit**

Additional items to those listed in Chapter 3:

Oxygen with mask and tubing  
0.9% saline 500ml × 4

## EXPEDITION MEDICINE

Intravenous fluid infusion sets  
Intravenous cannulae 18g × 4 (e.g. Venflon)  
Inflatable splints  
Plaster of Paris bandage  
Velband  
Chest drains, tubing and Heimlich valves  
Urinary catheter and drainage bag  
Laryngoscope and batteries  
Endotracheal tubes  
Airways  
Artery forceps  
Suction catheter and apparatus  
Syringes 10ml, 20ml and 50ml  
Nasogastric tube  
Aneroid sphygmomanometer and BP cuff  
50% dextrose injection  
Ventolin inhaler and spacer device  
Diazepam injection  
Ketamine injection

### **Dive site/mobile camp medical kit**

Additional items to those listed in Chapter 3, Table 3.3:

Auroscope  
Stethoscope  
Sleek adhesive tape (two rolls)  
Sutures:  
    0/0 black silk on hand needle  
    3/0 Dexon  
    5/0 nylon  
Syringes 2ml and 5ml  
Injection needles and cannulae (assorted)  
Sofratulle – dressing for a wide range of infected lesions  
Fusidic acid cream 30g – topical antibacterial cream for skin infections  
Miconazole cream 30g – topical antifungal cream for feet infections  
Betnovate cream 30g  
Neutrogena hand cream  
Antacid tablets  
Cinnarizine tablets 15mg – anti-sea sickness medication  
Buccastem 3mg – anti-sea sickness medication  
Glyceryl trinitrate spray

Lip salve  
Calamine cream  
Space blanket

**Emergency injections box**

Adrenaline (epinephrine) 1 in 1,000 solution  
Atropine 600mcg/ml  
Dexamethasone 4mg/ml, 20mg/ml  
Benzylpenicillin 600mg vial  
Hydrocortisone 100mg/2ml vial  
Metoclopramide 5mg/ml  
Chlorpheniramine 10mg/ml