

RES MEDICA

Journal of the Royal Medical Society



The Diving Casualty, Aetiology and Management

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Adapted and revised from a dissertation read before the Society on Wednesday 1st February 1978

Abstract

The logarithmic increase in diving, both professional and amateur, is paralleled by an unprecedented rise in related morbidity and mortality. Improved breathing apparatus and protective clothing are enabling man to remain in water longer and to dive deeper than ever before. Many diving accidents are untreatable owing to the circumstances in which they occur: in a modern saturation diving system it may be impossible to bring a diver back to atmospheric pressure in less than several days — whatever the medical emergency. However, an increasing number of fatalities are occurring due to lack of knowledge of the basic signs and symptoms which a diver, surfacing obviously with great difficulty, may present.

This dissertation is an attempt to review and collate current work and knowledge of the medical problems presented by the underwater environment.

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ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)

Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, New Series No.2, 1980: 39-46

doi:[10.2218/resmedica.v0i2.929](https://doi.org/10.2218/resmedica.v0i2.929)

THE DIVING CASUALTY, AETIOLOGY AND MANAGEMENT

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The logarithmic increase in diving, both professional and amateur, is paralleled by an unprecedented rise in related morbidity and mortality. Improved breathing apparatus and protective clothing are enabling man to remain in water longer and to dive deeper than ever before. Many diving accidents are untreatable owing to the circumstances in which they occur: in a modern saturation diving system it may be impossible to bring a diver back to atmospheric pressure in less than several days – whatever the medical emergency. However, an increasing number of fatalities are occurring due to lack of knowledge of the basic signs and symptoms which a diver, surfacing obviously with great difficulty, may present.

This dissertation is an attempt to review and collate current work and knowledge of the medical problems presented by the underwater environment.

1. HAZARDS OF THE UNDERWATER ENVIRONMENT

Compression Barotrauma: barotrauma may occur on descent when external pressure, in accordance with Boyle's Law, acts on the gas-filled cavities of the body to compress their contained gas. Thus compensation is required either by the introduction of additional compressed gas or by replacement with tissue or tissue fluids. An effect produced by pressure change is that of "squeeze". Should a diver descend rapidly – for example as a result of falling from an underwater platform –

he may travel a great distance through the water before being checked by his lifeline or air-pipe. Thus although pressure within the respiratory system remains at the level of the working platform, external environmental pressure rises rapidly. The increase in external pressure and proportionate decrease in air volume, if there is a large pressure differential, will cause pulmonary oedema and haemorrhage. Other surfaces exposed to air will be subject to similar effects, and the pulmonary manifestations are often accompanied by subconjunctival haemorrhage and oedema. The resultant damage is more extensive, and the condition more dangerous, the nearer to the surface the fall occurs because of the greater relative pressure change.

Pulmonary Barotrauma: if an ascent is undertaken with lungs fully expanded, and air release is inhibited, then as surrounding pressures fall, the enclosed air will expand the lungs. This may tear lung tissue, and air may then leak into the connective tissues of the mediastinum. The interstitial emphysema so produced may rupture into the pleural cavity with the acute onset of pneumothorax. If, as is often the case, air is drawn into the pulmonary circulation, air emboli are produced.

Air Embolism: the most serious presentation is loss of consciousness, although convulsions, visual changes and spastic or flaccid paralyses are quite common, while less dramatic symptoms include vertigo and tingling in the extremities. Treatment of this condition is usually by immediate recompression of the patient: pressure both causes

much of the embolism to dissolve in the blood, and also causes it to shrink until it may slip past the point of obstruction. Obviously the patient must be repressurised within seconds if treatment is to be effective. Prolonged, slow decompression may be required to prevent the embolus reforming. Following decompression, the patient should be admitted to hospital for medical examination, and an E.E.G. immediately following admission may indicate a focal intracranial lesion. A lung scan should be performed, as this tends to be more informative than straight chest X-ray.

Pneumothorax and Interstitial Emphysema: the patient may complain of pain in the chest or abdomen upon leaving the water. This is commonly accompanied by a cough, epistaxis or slight haemoptysis. Treatment of these conditions presents a different problem. The interstitial emphysema is usually retrosternal and untreated but pneumothorax is drained through an underwater seal.

In the treatment of pneumothorax or interstitial emphysema, because of the possibility of air embolism, full emergency treatment must be given in all cases. If recompression is unavailable, the patient should be placed in either the left lateral or prone position with the body sloping such that the head is kept low, in an attempt to prevent any possible air emboli entering either the cerebral or the coronary circulations. The incidence of burst lung would be reduced by preventing from diving all those with existing lung pathology, for example emphysema or adhesions. If the subject is suffering from an attack of asthma, hay fever or acute bronchitis, he must not dive until resolution is complete.

Pressure Changes and the Ears: otitic compression barotrauma may occur, leading to rupture of the tympanic membrane (possibly complicated by con-comitant serosanguinous transudate in the middle ear) if the pressure in the middle ear space cannot be equalised with the external auditory meatus via the Eustachian tube. This may result in perforation and haemorrhage. Alternobaric vertigo is a further complication, which may be irritated by repeated attempts to equalise middle ear pressure by Valsalva manoeuvres. Many cases of difficulty in equalising pressure result from catarrh within the pharyngo-tympanic tube.

Another syndrome is that of "reversed ears". This can occur when air is trapped in the external auditory meatus, often as a consequence of a tight-fitting rubber hood. The increase in environmental pressure is transmitted to the middle ear by the pharyngo-tympanic tube, and thus the pressure in the outer ear is less than that in the middle ear. The drum tends to bulge outwards, and there is swelling of the lining of the meatus due to oedema. Pain is not a prominent feature, although minor haemorrhage may occur.

Hearing and Balance: any upper respiratory tract infection, or damage to the ear (e.g. hyperaemia or haemorrhage) must be allowed to recover before recommencing underwater activities. In addition, the sea may contain fine particles of sand which often irritate the outer ear with subsequent infection. Wright and Alexander have shown that prolonged exposure to water changed the healthy ear flora from Gram +ve cocci and diphtheroids to Gram -ve bacilli (particularly *Pseudomonas pyocyanea*) and that this change often preceded acute symptoms.

With regard to balance, the form of vertigo seen in decompression sickness is known as "staggers". The present hypothesis is that gas bubbles are trapped within the microcirculation thus restricting oxygenation of sensory organs within the inner ear.

Pressure and the Sinuses: upon descent, air volume is obviously reduced, and transudate may occupy the resultant space. In contrast to the previous condition, pain is often intense. With re-expansion of air volume during ascent, this may result in mucopurulent discharge or even frank haemorrhage.

Pressure and Bad Teeth: small pockets of gas (from fermentation of food debris) are often found round the roots of teeth. During compression, the gas "bubbles" are reduced in volume, the space being occupied by blood and tissue fluid. Again, re-expansion of air volume results in locally-increased pressure, and subsequent pain.

Pressure may also exert effects on the gas contained within the diver's equipment. If face-mask pressure cannot be equalised, there may be oedema of facial tissues, possibly extending to subconjunctival haemorrhage. Failure to equalise

pressure within a dry suit results in painful "nipping" of the skin. The marks left on the skin must be distinguished from the widespread skin rashes seen in some cases of decompression sickness.

2. HAZARDS FROM GASES

Inert Gas Narcosis: the noble gases, under pressure, may exhibit all the properties normally associated with anesthetic agents. As compressed air remains the most frequently used breathing "mixture", nitrogen is the gas most intimately and continuously associated with the poetically-termed "raptures of the deep". Paradoxically, euphoria is a not uncommon prelude to narcosis.

The signs and symptoms of nitrogen narcosis are as follows:

on descending to	45 m:	Euphoria
" " "	45-60 m:	Joviality
" " "	60-75 m:	Hysteria and numbing of the periphery

Beyond these depths depression, impaired neuro-muscular co-ordination and unconsciousness predominate.

Upon return to the surface, amnesia lasting several hours may follow nitrogen narcosis. Anxiety, alcohol, carbon dioxide retention and fatigue are proven predisposing factors, and ameliorating factors include frequent deep diving and anti-hallucinatory drugs.

When the pressure is released, the excess nitrogen leaves the tissue and recovery is complete. Thus nitrogen should be replaced by a less narcotic gas if dives in excess of 76.2 metres are anticipated. Helium, which is approximately one-eighth as narcotic as nitrogen, is used at these depths. Hydrogen and neon have also been used. However argon, krypton and xenon are more potent narcotics than nitrogen.

Oxygen: Problems are encountered with both hypoxia and hyperoxia:

(i) Increased oxygen tension — hyperoxia.

This is differentiated into acute and chronic forms.

(a) Chronic oxygen poisoning (low oxygen concentration prolonged over several

hours). The mechanism — which is not well documented — involves a severe inflammatory reaction within the lungs, often leading to pneumonia. General effects include fatigue, decrease in pulse pressure, slowing of pulse, evidence of vasoconstriction in the central nervous system and retinal vessels, and pleuritic chest pain.

The more common danger is that of:

(b) Acute oxygen poisoning, i.e. after brief exposure to high oxygen tensions (2.0 bars and above). The symptoms, in order of incidence, are: lip twitching, dizziness, nausea, choking sensation, dyspnoea and tremor — which may eventually progress to convulsions. Symptoms normally disappear within a few minutes on returning to air, and no further treatment is required. However, convulsions may cause the self-contained diver in open water to lose his mouthpiece, with possibly fatal consequences. In this syndrome:

- (1) as oxygen pressure is increased, the time of exposure before onset of symptoms is proportionately decreased;
- (2) tolerance to high pressure oxygen is greatly reduced if exercise is performed;
- (3) tolerance varies both between individuals and within the same individual from day to day. The prevention of oxygen poisoning is dependent upon the ability of the equipment to maintain the oxygen partial pressure with safe limits.

Diving on pure oxygen is now restricted to a depth of 8 metres.

(ii) Hypoxia:

In acute hypoxia, loss of consciousness may occur in a few seconds, the collapse often being preceded by severe convulsions. Although rapid recovery takes place if normal oxygen supply is restored before the intervention of anoxic brain damage, amnesia for events immediately prior to the hypoxic incident is a common sequel. Regrettably, unconsciousness may be the first symptom of hypoxia, and syncope in water will result in drowning unless there is immediate rescue. Another similar condition, seen in the subject who

hyperventilates and follows this by prolonged exertion, is latent hypoxia. The sudden loss of muscle tone (subsequent to the exercise) enhances peripheral pooling of blood, giving decreased venous return and reduced cardiac filling with consequent slight diminution in cardiac output. This may prove sufficient to precipitate unconsciousness in the already hypoxic subject.

Both hyperoxia and hypoxia usually result from malregulation of the closed circuit breathing apparatus, in which oxygen in the compressed air is recycled, carbon dioxide being removed by soda lime, thus permitting longer continuous periods under water.

Carbon dioxide: although carbon dioxide *per se* is not used for any specific purpose in diving, in many underwater accidents an increase in carbon dioxide tension is often beyond the control of the diver, and thus he should be able to recognise the preliminary symptoms and signs. These include increased ventilation, mental confusion, severe headache (possibly accompanied by vomiting), decreased blood pressure, lack of co-ordination and slowing of the pulse rate. If allowed to continue this leads to unconsciousness, paralysis of respiratory and cardiac centres, and death.

When man is under pressure the partial pressure of carbon dioxide in the lungs (which is determined by venous tension proportionate to the amount produced by the tissues) is approximately 40 mm Hg. i.e. equal to the normal value on the surface, provided that there is no carbon dioxide present in inspired air and that adequate ventilation is maintained. The percentage of carbon dioxide in alveolar air decreases with depth, but the partial pressure in relation to other gases remains constant. Any air supplied to the diver at depth, contaminated with CO₂, will raise the alveolar partial pressure and carbon dioxide contamination can be magnified greatly, possibly to a fatal outcome.

The use of soda lime introduced another complication. Should sea water leak into the system, the diver may inhale a caustic mixture. The breathing apparatus should be removed as quickly as possible and the diver's mouth washed liberally with a diluted solution of a weak acid, such as vinegar. Any exposed skin must be treated similarly and a drop of sterile liquid paraffin

placed into the victim's eyes. The diver should be examined at a future date for any residual lung damage which may have been caused by the caustic inhalation.

Other Gases: (i) carbon monoxide poisoning is an occupational hazard which is usually the result of failure to ensure that the air supply (pump or cylinder is free from traces of this gas. In many cases this is because the inlet of the compressor is situated close to the exhaust! The condition is often manifested by dizziness and fatigue, progressing to syncope. An important diagnostic feature is that lips and mucous membranes often appear bright red. Diagnosis is usually confirmed by examination of the inhaled gas or spectroscopic examination of the blood.

Oxygen is of fundamental importance in the treatment of this condition, although — depending on the circumstances — artificial resuscitation may be a preliminary requirement. Oxygen plus added carbon dioxide is preferable to pure oxygen, as the CO₂ increases the ventilatory drive. Severe hypoxic brain damage is indicated by failure to respond to this treatment within one hour.

(ii) Compressed air may also be contaminated by oil vapour. Oil fumes, under pressure, irritate the membranes of the lungs: in mild cases this may merely precipitate a slight cough, but if exposure to such contamination is prolonged, lipid pneumonia may result.

3. DECOMPRESSION:

There must be a complete reversal of processes by which adaptation to depth is achieved if the subject is to return safely to the surface. Decompression sickness effectively encompasses many diverse features, including any abnormality which is the direct result of environmental pressure. The commonest presenting symptom is severe arthralgia — frequently flitting in nature. Any abnormality often depends on the site of air emboli, and theoretically a wide spectrum of neurological deficits is possible, but for practical purposes the most common presenting sign is sudden loss of consciousness.

Pneumothorax, a frequent consequence of lung overdistension, may well remain undetected until dyspnoea is experienced during decompression from a therapeutic recompression.

(i) Acute Decompression Sickness is sub-divided into Types I and II. The former includes peripheral manifestations and the latter, the central and potentially more dangerous effects.

- Type I
- (a) Pain is the most common feature, ranging from mild pain in the limbs (the "niggles") to a very severe joint pain (the "bends"), and is frequently accompanied by generalised fatigue.
 - (b) Skin involvement tends to be widespread and includes transient pruritis, subcutaneous oedema — especially of the limbs, and areas of cutaneous vascular stasis with central cyanotic areas, particularly related to the trunk.
 - (c) Fatigue, malaise and anorexia.

Type II (a) Pulmonary effects, which divers refer to as "the chokes", include substernal pain especially on inspiration, acute dyspnoea, and cough. These frequently precede collapse, shock and asphyxia. The pulse becomes feeble and increasing cyanosis develops.

- (b) Neurological decompression sickness shows characteristic effects on the spinal cord, the patient commonly becoming paraparetic. Other neurological deficits encountered in this condition include vertigo, visual defects, monoparesis, mental disturbances, migraine and girdle pains of the trunk.

- (c) Postural hypotension may result from the fluid shift which occurs secondarily to the increased capillary permeability. Although haemoconcentration (due to relative hypovolaemia) may not be obvious, the associated rheological changes will almost certainly exacerbate consequences of intravascular pockets of gas.

(ii) Chronic Decompression Sickness: The more extreme cases, involving the spectrum of hemi-

plegia or paraplegia, condemn the victim to years of disability. Recently, attention has been directed to aseptic bone necrosis, which may result from chronic decompression sickness. This may in turn cause joint deformity and arthritic changes. It is assumed that the condition is caused by minute gas bubbles with surrounding platelet aggregation acting as micro-emboli in the end-arteries of bones.

Diagnosis is normally made on the symptoms and signs already mentioned; however this may only be satisfactorily confirmed by further subjecting the patient to increased pressure, when the symptoms should disappear. Essential treatment for this condition — or air embolism — is to reduce the size of "pressured" air bubbles by immediate recompression to an adequate depth, followed by slow decompression. Any delay in instituting such a procedure will render the patient less responsive to subsequent recompression. In the absence of any recompression facility, a more complex regime of intravenous therapy — as suggested by Saumarex, Bolt and Gregory — may be commenced: however the efficacy of such treatment has not yet been determined.

During recompression, changes in air volumes — for example, within intravenous giving sets — must be anticipated. In the same context, the balloon of a cuffed endotracheal tube must be filled with water to prevent volume changes paralleling those of external pressure. Nitrous oxide and opiate analgesics are contraindicated during the transfer of the patient: however, oxygen can be safely administered. Ancillary treatment includes Dexamethasone, Dextran, and Heparin (the effectiveness of the latter being related to its anti-lipaeamic activity).

4. IMMEDIATE CARE OF THE DIVING CASUALTY

Many cases of drowning are complicated by concomitant acute onset of hypothermia. Both salt- and fresh-water drowning — although physiologically distinct — have similar consequences, i.e. acidosis, hypoxaemia and hypercapnia.

Immediate effects of the inhaled fluids include an acute inflammatory reaction in the alveolar capillaries and subsequently accumulation of a plasma-rich exudate within the alveolus. Areas of

atelectasis may result if the inhaled water destroys normal surfactant. In addition, pulmonary or cerebral oedema may ensue, and the risk of pulmonary infection is greatly increased when the water is polluted.

The major aims of treatment of such a condition must include correction of acid base imbalance, rewarming, and — most important — restoration of acceptable levels of ventilation and circulation.

After clearing the mouth and oropharynx, artificial resuscitation should be performed as soon as possible, and, in pulseless patients, this should be accompanied by closed chest cardiac massage (although the mere restoration of adequate ventilation will often result in a satisfactory cardiac rhythm). One must *not* perform closed chest cardiac massage on the hypothermic patient. Ventricular fibrillation commonly follows the slightest mechanical irritation in hypothermia victims, and in such patients the most successful regime includes prevention of further heat loss plus maintenance of ventilation. More recent forms of immediate treatment are:

- (i) The "Space Blanket", which is an aluminium coated plastic sheet, intended to prevent further heat loss. This has several disadvantages. It prevents only radiation heat loss (not that by convection or conduction) and the patient may also lose a great deal of both core heat and moisture by normal breathing, a facet of temperature exchange totally unaffected by the space blanket.
- (ii) A modern, and controversial, item of equipment in this sphere is one which does not yet even possess a name! This "apparatus" allows the patient to breathe CO_2 through a soda-lime open circuit, resulting in the inhalation of both moisture — an essential requisite in these situations — and heated air thus directly acting upon core temperature. The equipment has not been fully tested, and complications discovered to date include inflammation and sloughing of pharyngeal epithelium; however the extent of usage required before this arises is not known.

When the patient arrives in hospital, an intravenous line should be set up using 8.4% NaHCO_3

to correct the acid base imbalance; normally 150 mEq is given, then 70 mEq every subsequent ten minutes until adequate ventilation and circulation return. An E.C.G. is taken on arrival, and if the patient is in ventricular fibrillation D.C. counter shock is applied (unless, as already stated, the patient is hypothermic).

The treatment of hypothermia in hospital is controversial. Myocardial rewarming can be accomplished by means of cardio-pulmonary bypass, although this is obviously limited in its practical potential. The most widely-used technique is probably immersion in a hot bath at 45°C with the limbs *out* of the bath, always ensuring that the water remains at that temperature; there is an unfortunate tendency to place the patient in the bath forgetting that as his temperature increases, that of the surrounding water will proportionately decrease, thus reducing the efficacy of the treatment. Present forms of anti-arrhythmic therapy have proved ineffective in the treatment of cardiac arrhythmias resulting from hypothermia. These usually revert to normal within 12 hours of restoring core temperature.

Other forms of management include peritoneal dialysis with warmed fluid; breathing heated humidified air; enemas with warmed fluid; and instilling warm water into the stomach. Deeply hypothermic patients may suffer little anoxic brain damage during periods of circulatory standstill: at 37°C the maximum safe period is about 3 minutes at 32°C 8-10 minutes and there is one documented case of a patient with a core temperature of 15°C surviving after 1 hour of circulatory arrest. Return of spontaneous respiration obviously depends on the amount of anoxic brain damage present.

Patients should be intubated and ventilated with 100% oxygen. Normally the oxygen is given by IPPV, however recently PEEP has been found to be effective in preventing pulmonary oedema, and this is now being increasingly used. In non-hypothermic patients who are being ventilated but do not have an acceptable heart action, an intracardiac injection of 2-4 ml. 1/10,000 adrenaline and 10 ml. of CaCl_2 should be given.

Thus the problems of initial treatment, both immediate and subsequently in hospital, have been

briefly considered. The later management of the patient includes problems of "secondary drowning" and a condition resembling the respiratory distress syndrome. Secondary drowning is characterised by tachycardia, anoxaemia, fall in pulse pressure then in overall blood pressure, a rapidly increasing pulmonary oedema and changes in cardiac rhythm. Both the respiratory distress syndrome, in this instance, and secondary drowning are treated by early ventilatory support accompanied by concurrent circulatory support with the infusion of up to one litre of plasma (under central venous pressure control). Methyl prednisolone is usually given to counteract any

pulmonary or cerebral oedema secondary to anoxia.

It can be seen that the underwater environment may encompass additional features, within the emergency situation, to those commonly encountered on the surface. All possible aspects of this form of accident must be thoroughly investigated: later complications often result from omissions in initial investigation, rather than inherent difficulties in subsequent treatment regimes.

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