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Thoughts on immediate care

Anaesthetists are being increasingly called upon to give immediate treatment for various life-threatening conditions in casualty departments and elsewhere. This feature of short papers by invited experts is designed to describe the proper management of patients who require immediate care before the opinion of specialists in a particular field can be obtained.

Treatment of the diving casualty

D. H. ELLIOTT AND R. DE G. HANSON

The diving casualty is a comparative newcomer to the accident departments of our hospitals but, with the current national increase in both professional and amateur diving, there is bound to be a continuing increase in this type of casualty. Many diving accidents either resolve spontaneously or their victims present as cases of drowning or acute hypothermia, but there are also some specific diving illnesses which may be seen in the emergency department of a hospital.

A sub-arachnoid haemorrhage or cardiac infarction may occur underwater just as easily as on land,[1] when confronted with a casualty who has recently been engaged in underwater activities a number of conditions specific to diving need to be remembered. In this review the intention is to concentrate on conditions specific to diving but to exclude hypothermia and drowning which have been dealt with earlier in this series.[2] Some conditions which are not immediately dangerous to life have been included to enable them to be differentiated from those which are more serious. An accurate history of the diver and the dive is invaluable to the diagnostician; some of the additional factors to be considered in this type of casualty are, the type of equipment used, the depth and duration of the dive, the frequency of the dives, and the stage of the dive at which the symptoms or signs appeared.

Some general problems of diving

Compression barotrauma

Barotrauma may occur when pressure, in accordance with Boyles Law, acts on the gas-filled cavities of the body. This compresses their contained volumes of gas on descent and thus compensation is needed either by the introduction of additional compressed gas or by replacement with tissue or tissue-fluids.

Ears. If, during descent, the pressure in the middle ear cannot be equalised through

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the pharangyo-tympanic tube with that in the external auditory meatus, otitic compression barotrauma may occur ranging in degree from injection to rupture of the tympanic membrane, possibly complicated by sero-sanguinous transudate in the middle ear space. Alternobaric vertigo[3–5] is a further complication which may be immediate or delayed. The vertigo may be initiated by the diver trying to equalise the pressure in the middle ear by means of forceful Valsava manoeuvre. Back at the surface this condition is usually transient but, if persistent, a history of difficulty with 'clearing the ears' may help to distinguish a possible round window rupture from the vertiginous manifestations of acute decompression sickness.

The sinuses. Like the middle ear, trouble occurs if their openings to the upper respiratory tract are blocked by such things as catarrhal swelling, polypi or deviated nasal septa.

As the diver descends the volume of air inside a sinus is reduced and the space may become filled with transudate. The pain is usually intense and may cause abandonment of the dive. During ascent the air re-expands and may make the pain worse or may cause a blood stained and occasionally muco-purulent discharge or even frank and profuse haemorrhage.

Equipment. The diver may suffer injury due to the effect of pressure on the gas contained in his equipment. If he is wearing a 'dry' suit and fails to equalise the pressure within it by means of gas from a suit-inflation bottle, then the reduction in volume of the gas inside the suit can give rise to a painful nipping of the skin. Marks are left on the skin in the form of red lines which should be distinguished from the diffuse skin rashes seen in some cases of decompression sickness. Similarly, if the pressure in the face mask cannot be equalised, oedema of the facial tissue and subconjunctival haemorrhage may occur. These conditions require no treatment apart from reassuring the patient that they are not serious and will resolve, but they need to be excluded in any differential diagnosis.

Lungs

As a breath-hold diver descends so the volume of gas in his lungs decreases. This causes a loss of buoyancy, and if he descends much beyond 40 metres the lung volume will become less than residual. If this happens he can suffer from a condition known as 'chest squeeze' when haemorrhage into the alveoli occurs to make up for the reduction of air volume. This has led to fatal results.[6]

Nitrogen narcosis

Nitrogen is a true anaesthetic agent and its narcotic properties can lead the inexperienced diver into danger at depths below about 40 metres. However, the effect is related to the partial pressure of nitrogen and either the diver will return to a lesser depth at which the narcosis is less significant, or he will become another drowning casualty.

Other diving hazards

Equally insidious are cold exposure, muscular exhaustion and respiratory exhaustion. Unless these problems are dealt with by adequate training before the dive and knowledgeable supervision during the dive, they can contribute to panic and drowning.

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Underwater blast

If the patient has been in the water at the time of some underwater explosion then the possibility of underwater blast injury must be considered. Though dependent to a certain extent upon the position of the diver in the water, the principal injuries occur to the lungs and other air containing organs such as the alimentary canal. The treatment of such conditions[7-9] will not be considered further here.

Venomous marine animals

It is fortunate that there are few venomous animals dangerous to man found in the waters around Great Britain. However, in these days of increasing world-wide travel a brief mention of some of these animals would not be out of place. As a general rule marine toxins tend to be unstable, some even at temperatures as low as 0°C.[10] A clue to the type of envenomation may be given at the site of the wound. A stone-fish sting is generally found on the sole of the foot, a sting ray around ankle-level or higher and may be large and lacerated due to the fish's tail lashing up, and a sea snake bite—two small puncture wounds with little pain involved, in contrast to the other two in which pain is the major symptom.

The toxins found in the stings of stone-fish, sting rays and weever fish are heat labile. The relief given by applying heat to weever fish stings has been known for many years.[11,12] The treatment for sting ray injuries is well established[13] and consists of, washing the wound (since much of the venom can be removed this way), analgesics, attempting to remove the sheath of the sting (if it is visible in the wound), and immersing the limb in hot water for 30 to 90 minutes. If possible a constraining band should be tied directly above the wound site. This treatment could be used for the stings of other fish mentioned above and, in the case of stone-fish, it is also possible to obtain an antivenin.

Sea snake. In the case of the sea snake there may be a latent period of from 20 minutes to several hours before the onset of symptoms. This has led to some victims failing to associate the initial bite with the subsequent illness. Early signs include a sensation of thickening of the tongue, ptosis and a general 'aching stiffness' of the muscles. Pain is not a feature, this distinguishes the snake bite from the stone-fish and other stings. The treatment is the use of the appropriate antivenin.[14]

Jelly-fish, which have nematocysts for the capture of their prey, have toxins which give rise to severe pain. Severe envenomation by box jelly-fish may cause collapse and respiratory arrest.[15]

The tentacles can extend as far as 9 metres and, since these numerous strands are virtually invisible, a swimmer can be injured without knowing the cause. The outcome of the stinging is governed by the size and species of the jelly-fish, the amount of venom and the physical condition of the victim.

In severe cases there is little that can be done and death from shock and respiratory arrest may occur in minutes. Though studies have been carried out on sea-wasp venom[16] no antivenin is readily available for jelly-fish stings and treatment should be carried out as follows; the prevention of drowning, the treatment of shock with artificial respiration if needed, systemic absorption may be delayed by the use of tourniquets, removal of tentacles (while wearing gloves), and wash with sea water or, preferably, fix with alcohol or vinegar. Fresh water, or rubbing with sand, may trigger off more nematocysts. Meat tenderiser has a reputation for being effective in first aid.

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General supportive and analgesic treatment should be given.

Cone shells. No antivenin has been produced. Bites can lead to paralysis and death though usually the patients recover in about 6 hours.[17]

Octopus. The most dangerous is the tiny blue-ringed octopus whose small bite may be unnoticed by the victim. No antivenin has been developed and death occurs from respiratory paralysis. The only treatment is to ligature above the wound and give artificial respiration until after many hours the effect of the toxin wears off.

Breathing equipment

Breath-hold diving

It is not sufficiently well known that the snorkel, a simple J-shaped tube, is a dangerous item of underwater breathing apparatus if not properly used. Hyperventilation before making a breath-hold dive can lead to a lessening of the carbon dioxide drive to respiration. At depth the carbon dioxide will build up, but the desire to breathe may be further suppressed in the excitement of chasing some exotic fish. During this time the decreasing percentage of oxygen is more than adequate because the depth of water increases its partial pressure but, as the snorkel diver returns towards the surface, the partial pressure diminishes leading to hypoxia, unconsciousness and, in these conditions, drowning.

Another hazard of snorkel diving, insufficiently appreciated by novices, is that the effectiveness of the chest as an integral part of the body's buoyancy is diminished by compression during descent. The majority of divers become negatively buoyant at about 5 metres depth and must realise that, in order to return to the surface, they cannot just float up but must make a positive swimming effort. Again, this could lead to panic and drowning.

Open circuit air

Self-contained breathing apparatus (SCUBA) using compressed air is commonly used and is relatively safe but although it is relatively simple, problems can occur. One is the contamination of the breathing gases with exhaust fumes giving rise to increased levels of carbon monoxide. It must be remembered that percentages of contaminants which may be tolerated on the surface result in an increase in partial pressures with depth and thus can give rise to serious problems in diving. It is as well to bear in mind the possibility of carbon monoxide poisoning in the case of an unconscious diving casualty. The diagnosis can be made either by analysis of the breathing gas or, probably more easily, by spectroscopic examination of the blood. Treatment should be as for other cases of carbon monoxide poisoning, including hyperbaric oxygen.

Another hazard is the danger of mechanical failure in the set which may give rise to asphyxia or drowning.

Closed circuit and semi-closed circuit breathing apparatus

This type of apparatus is probably used by amateur divers only in the confined spaces of limestone caves where the small size of oxygen equipment does not hinder exploration but professional divers often use closed-circuit or semi-closed circuit breathing

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apparatus. This equipment, using oxygen on demand (closed circuit) or a constant flow of oxygen, oxy-nitrogen or oxy-helium (semi-closed circuit), can be hazardous and the diver needs to have been well trained in its use. A partial pressure of oxygen exceeding some 1·8 bar (182·4 kPa) may lead to acute oxygen toxicity, a major epileptic convulsion which underwater may prove fatal. Conversely, the reduction of oxygen partial pressure in a breathing apparatus can lead to a hypoxia with no associated subjective respiratory warning because the soda lime eliminates carbon dioxide. The hypoxia could be due to a failure of supply or the diver working or swimming hard and requiring more oxygen than is delivered by the regulator.

The use of soda lime introduces another complication. If sea water leaks into the system the diver may inhale a caustic mixture of soda lime and sea water. This 'cocktail' can be dangerously disconcerting, especially since the diver is already in a hazardous environment. The treatment of this condition consists of removal of the breathing apparatus as quickly as possible. After this has been done the diver's mouth should be washed liberally with water or preferably with a dilute solution of vinegar or other weak acid. Exposed skin should be washed liberally with water and a drop of sterile liquid paraffin put into the victim's eyes. In all but the minor cases the diver should be kept under observation for any lung damage due to inhalation of the corrosive fluid.

Decompression accidents

There are essentially two kinds of dysbarism, or decompression accident.[18] One is a consequence of the expansion of gases in the lungs, in accordance with Boyle's Law, during the ascent of the diver through the water to the surface, and is known as pulmonary barotrauma. The other is decompression sickness which may be considered as due to bubbles formed from the gases which were dissolved in the tissues of the body during the time spent at depth. It should be appreciated that in addition to any mechanical effects of the bubble complex biochemical changes take place at the gas–blood interface. Manifestations of cerebral arterial air embolism which can follow pulmonary barotrauma are sometimes very similar to those of acute neurological decompression sickness. It seems to be insufficiently appreciated that whereas decompression sickness follows only a dive of sufficient depth or duration to acquire a critical load of dissolved gas in the body, pulmonary barotrauma and its neurological complications need no such threshold and have followed dives as shallow as 3 metres or with durations of less than 1 minute.

The prevention of pulmonary barotrauma is by adequate venting of the expanding gases from the lungs. Nevertheless, cases do occur in which the correct drill for exhalation during rapid ascent seems to have been followed by the diver. In such persons it is possible that there has been some localised obstruction which has caused retention of gas within a portion of the lung. The history is often given that the diver had 'run out of air' while making an emergency ascent.

While there are some dives from which it is safe to ascend directly to the surface ('no-stop dives'), the prevention of decompression sickness from deeper or longer dives is to ascend at a slower rate, usually by a series of stoppages which are determined by diving tables, thus allowing time for the excess gases dissolved in the tissues to be eliminated. Such tables are based on complex mathematical hypotheses and are designed to be safe for the great majority of divers. However, an occasional case of decompression sickness will follow the use of even the safest diving table. Particularly

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hazardous are dives such as in strong tides which require hard physical work, a series of dives by an individual in the course of one day and dives in very cold water. It must be borne in mind that diving in mountain lakes introduces added complications of decreased atmospheric pressure which must be allowed for in the diving tables, and also that flying or mountain climbing too soon after diving may exacerbate decompression sickness from an otherwise safe dive.

Clinical manifestations

Decompression barotrauma. Characteristically the symptoms and signs of pulmonary barotrauma and its complications occur within a few seconds of reaching the surface, only rarely are manifestations delayed for more than a few minutes. The most common presenting sign is that of a sudden loss of consciousness but the particular signs and symptoms in an individual depend upon the site of the arterial air emboli and almost any neurological deficit can occur. There are occasionally some additional features following barotrauma such as pneumopericardium which would not be found in cases of decompression sickness. Pneumothorax, which may be bilateral, is one possible consequence of the overdistention of the lungs but it rarely causes the presenting symptom and may remain undetected until the expansion of gases within it cause dyspnoea some hours later, during the decompression from a therapeutic recompression. The escape of air into the mediastinum and the root of the neck can cause a subcutaneous emphysema, which is diagnostic of pulmonary damage.

Decompression sickness. Two forms of acute decompression sickness are described:

Type 1, in which only joint pain ('limb bends') and other relatively minor manifestations are present.

Type 2, in which there are more serious symptoms.

This classification has little merit unless it is remembered that about one-third of patients with acute decompression sickness have both minor and more serious lesions concurrently and, since the signs and symptoms of the more serious lesions may be relatively subtle, there is a danger that they may be overlooked in the presence of joint pain. It is also necessary to remember that the minor manifestations may precede the more serious ones. Thus there is a danger that an inexperienced practitioner might treat the patient incorrectly for his minor symptoms when he in fact requires a more vigorous therapy.

These minor symptoms include pain, which can be severe, in or around the synovial joints; skin rashes which take the form of patches of cutaneous vascular stasis with central cyanotic areas on the trunk, subcutaneous oedema, especially of the limbs; transient pruritus which is mild and multi-focal; a mild fatigue, which is disproportionate to the exertion of the recent dive and a malaise and anorexia which may not be noticed by the patient until relief is achieved some time later. In general the majority of cases occur within the first hour of surfacing but occasionally there are cases where onset may be delayed for over 24 hours.[19]

The more serious manifestations of acute decompression sickness are neurological, pulmonary and circulatory. Neurological decompression sickness most commonly affects the spinal cord and characteristically the patient in an emergency department will be paraparetic. However, any neurological deficit may be present: girdle pains of the trunk, monopareses; vertigo with nausea and vomiting ('staggers'); blurring of vision and other visual defects; migrainous headaches and mental disturbances.

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The pulmonary form of acute decompression sickness ('*chokes*') is quite distinct. The symptoms of breathlessness are attributed to the arrival of numerous small gas emboli in the lungs. Retrosternal pain, especially on attempting a deep inspiration, is pathognomic of this condition. Shallow rapid respiration with signs of hypoxia and venous congestion follow.

Postural hypotension may be indicative of hypovolaemia due to fluid shift, secondary to a generalised increase of capillary permeability. Haemoconcentration may not be apparent at the time of onset of the pulmonary or neurological manifestations of decompression sickness, but the associated rheological changes will tend to exacerbate the consequences of intravascular bubbles.

Management

Provided that an accurate history of recent diving is obtained, the diagnosis of a decompression accident is usually obvious.

The specific treatment of decompression sickness or air embolism is by immediate recompression of the patient in a compression chamber, to reduce the size of the bubbles, followed by a slow decompression, to allow time for the safe elimination of the dissolved gases. Recompression should begin at the earliest opportunity. Experience has shown that if there is delay, the condition of the patient is likely to deteriorate and will become less responsive to any subsequent recompression.[20] Thus the primary task of the medical practitioner is that of ensuring the successful transfer of the patient to a recompression chamber without delay. There are only a limited number of compression chambers in the United Kingdom and each requires a team of experienced personnel to operate it. The whereabouts of the nearest operational chamber should be known to those responsible for planning diving operations but, *in case of emergency, a request for assistance may be made by telephone* (*Portsmouth 22351*), *to the Superintendent of Diving or, out of working hours, the Duty Lieutenant Commander, HMS VERNON.*

Transfer. If the transfer is made by helicopter it should not exceed 1000 ft (305 m) altitude. During the journey oxygen may be administered but nitrous-oxide/oxygen mixtures are contraindicated.[21] Analgesics such as morphia are contraindicated because of the possible onset of respiratory decompression sickness.

A venous sample for haematocrit and serum electrolytes should be taken at an early stage. In addition to any life supporting measures such as artificial respiration indicated by the patient's general condition, ancillary treatment may include low molecular weight dextran 500 ml 8-hourly, dexamethasone 12 mg, 8 mg 6-hourly, and heparin 2000 units 6-hourly. The latter dosage is intentionally below that needed for anti-coagulation and its effectiveness is considered to be related to its anti-lipaemic activity.[22]

In the absence of any recompression facility to which the patient can be transferred, a more complex intravenous therapy may be initiated as suggested by Saumerez, Bolt & Gregory,[23] a regime which may also be used during recompression provided that the compression chamber is close to a suitable laboratory.

If an indwelling catheter or cuffed endotracheal tube is used in a plegic patient, its balloon should be filled with water which will not change its volume during subsequent changes of environmental pressures. Similarly changes of air volumes within

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intravenous giving-sets must be anticipated. Attention to the pressure points of a plegic patient must also be remembered.

Recompression treatment. Upon arrival at a recompression centre the treatment of the case should be undertaken by persons trained and experienced in this type of casualty. The principles underlying the selection of recompression tables are dealt with elsewhere.[24]

Cerebral air embolism. The treatment of the neurological manifestations of cerebral air embolism is essentially the same as for those of decompression sickness, however, there is the rare case of pulmonary barotrauma with no associated neurological manifestations and this may be treated conservatively without recompression.

Pneumothorax. Pleurocentesis may be indicated for a tension pneumothorax. If the patient has been recompressed, the manifestations of this might not become apparent until much later during the course of the prolonged decompression. A Heimlich valve is more convenient for use in a compression chamber than an underwater seal bottle and can be improvised if necessary as suggested in an earlier article in this series.[25]

Summary

In a diving emergency a number of specific conditions are possible in addition to those which would ordinarily be considered in a life-threatening situation. There are many separate factors which, underwater, can lead by some unfortunate combination of circumstances towards death usually, but not always, by drowning.

The other major category of accidents are those resulting from the ascent of the diver to the surface. These must be considered as medical emergencies and the majority respond to immediate recompression. In the absence of readily available recompression facilities, various measures may ameliorate the condition pending transfer to a suitable compression chamber.

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