Decompression—a hazard of underwater sports

SURGEON-COMMANDER D. H. ELLIOTT, R.N., M.B.(Hons.), D.Phil.
Institute of Naval Medicine
Alverstoke

There are many types of accident which can happen to man underwater. Important for the doctor and particularly for the diver is a knowledge of the possible causes of underwater accidents but this is given fully elsewhere (Miles, 1966; Elliott, 1968). The medical practitioner who may be called to render assistance in such cases will find that the great majority require resuscitation or other treatments with which he is already fully familiar. Occasionally, however, the incident is one which requires a specialized medical knowledge without which the wrong therapeutic principles might be applied. In particular the hazards of decompression, namely decompression sickness and pulmonary barotrauma, are acute illnesses which require immediate recognition and treatment.

While at raised environmental pressure, the breathing gases of the self-contained diver must be supplied to him at that pressure so that the chest is not crushed (a squeeze). A demand valve regulated by the pressure of sea water acting upon it is commonly used to deliver compressed air to the diver at the pressure he requires. While at depth, the gases at raised partial pressures in the lungs will tend to dissolve in the blood and tissues in accordance with the laws of physics until an equilibrium is achieved. In fact the uptake of gases by some parts of the body is quite a slow process and full saturation of the tissues is not approached in the relatively short durations of underwater swimming by amateurs. During the return to the surface the gases at raised tensions in the blood and tissues will tend towards an equilibrium with the lower partial pressures of gases in the lungs. If the duration at any given depth has been sufficiently long the quantity of gas in the tissues may be so great that, on decompression, not all can be quickly removed at the lungs and some may be released as gas bubbles in the tissues and blood. Such is the basic theory of decompression sickness, incomplete but sufficient as a working hypothesis for clinical conditions.

Thus it follows that there are some dives from which it is safe to ascend directly to the surface and some, deeper or longer, from which a direct (no-stop) ascent would be hazardous because of a risk to the diver of subsequent bubble formation. The prevention of decompression sickness is therefore to ascend at a slow rate, usually by a series of short ascents between longer stoppages, allowing time for the excess gas in the tissues to be eliminated. The diver is able to look up the appropriate decompression stoppages in a book of diving tables. These are based on complex mathematical theories (vide Bennett and Elliott 1969), and are designed to include not just the average man but to be safe for the great majority. Even using the safest diving table an occasional case of decompression sickness will follow, and the deeper the dive or the longer its duration the greater this risk. Particularly hazardous are dives in strong tides which require hard physical work, a series of dives by one individual in the course of a day and dives in very cold water.

A quite different physical process is the cause of pulmonary barotrauma. During decompression the gases in the lungs must expand in accordance with Boyle’s Law, and this excess of gas is normally vented by natural exhalation. If for some reason, such as a blocked bronchiolo, the expanding gases are trapped they will burst into the mediastinum causing a surgical emphysema, through the pleura causing a pneumo-

J. ROY. COLL. GEN. PRACTIT., 1969, 18, 233
thorax or into the vascular system causing an arterial air embolism usually of the central nervous system.

There is a dangerous misconception that serious symptoms can follow only a dive deep enough or long enough to require decompression stoppages. From the outline of the underlying pathological mechanisms already given it is obvious that this is not so. On one occasion impaired consciousness followed a rapid ascent after one minute or so spent at a depth of no more than ten feet. It is therefore important to remember that any unusual incident which follows an inhalation of gases at raised environmental pressure, however brief or shallow, must be treated as air embolism or decompression sickness until proved otherwise.

At this point the distinction between 'snorkel-diving' and 'aqualung-diving' needs to be emphasized. Sports diving includes both activities. In snorkel-diving a simple J-shaped tube is used for the diver's ease of breathing when he is face downward on the surface. He then makes a breath-holding excursion to depth with his lungs full of air and returns to the surface for exhalation and more air. In aqualung-diving the diver takes with him a supply of compressed air in cylinders to be used for breathing while he remains underwater. It should be clear that decompression accidents can only happen to a man in the latter category, the aqualung diver, because the breath-hold diver cannot dive deep enough or long enough to acquire sufficient dissolved gas to cause decompression sickness and also, since he cannot return to the surface with more air than he started with, does not run the risk of bursting his lungs.

The prevention of decompression sickness is the responsibility of the diver who is taught to adhere to the published diving tables and who is advised, by bodies such as the National Underwater Instructors Association and the British Sub Aqua Club, on the correct technique for making a rapid emergency ascent. Although a decompression incident is often due to a failure of the diver to follow these recommendations, such foolhardy neglect is not the only cause and trouble may follow even when the decompression has been correct in every way.

**Clinical manifestations**

Although in other contexts it is customary to distinguish between two forms of decompression sickness, Type I in which only joint-pain is present and Type II in which serious symptoms are present, it is considered that in sports medicine such a distinction though convenient is not entirely justifiable. Both types require essentially the same treatment, immediate recompression. It is therefore important to avoid the implication that joint-pain is not serious, particularly as the presenting symptom of pain might well obscure or precede some more insidious and dangerous symptom elsewhere.

There are a number of manifestations which are to be considered as warnings of approaching decompression sickness. These early symptoms may not require treatment but should be the stimulus for immediate action to ensure that if recompression is required, there will be no unnecessary delay. Commonly described in this category are itches of the skin, especially of the trunk, and rashes due to cutaneous vasodilation with stasis. Another warning is a very mild pain in a joint (a niggle), a pain which reaches its maximum intensity within ten minutes of onset and soon begins to regress.

The joint pains (bends) of decompression sickness can be single or multiple. Their onset may be sudden or gradual. Most commonly in divers a shoulder is affected but the knees, hips and elbows are also common sites. Even the inter-phalangeal and temporo-mandibular joints have been affected.

Whether or not joint pains are also present, other symptoms may exist which are considered to be more serious because they may lead to a permanent disability. An alteration of sensation in a limb commonly heralds the start of more serious trouble.
DECOMPRESSION—A HAZARD OF UNDERWATER SPORTS

This might be a numbness or coldness of the feet or of any other part and may be associated with some muscular weakness. In such cases a wide variety of neurological lesions is found. At first they do not necessarily follow any particular anatomical distribution but many develop into a paraplegia with the level of numbness slowly ascending up the trunk.

Less common are other neurological forms of decompression sickness; girdle pains of the trunk; vertigo with nausea and vomiting (the stagggers); blurring of vision, scotomata and other visual defects; migrainous headaches; bizarre mental disturbances.

Quite distinct is the pulmonary form of acute decompression sickness (the chokes) in which the symptoms of breathlessness are attributed to the arrival in the lungs of numerous small emboli of gas which have passed through the right side of the heart. A sudden retrosternal pain, maximal or sometimes only present on deep inspiration, is pathognomonic of this condition which is considered to be a consequence of a grossly inadequate decompression. Shallow rapid respiration with signs of hypoxia and venous congestion follow and may lead rapidly to loss of consciousness and death. Typically there are no associated symptoms of joint pain but neurological lesions may be present.

Thus the symptoms of acute decompression sickness may be grouped conveniently into three common presentations; joint pain, symptoms of spinal or cerebral lesions and pulmonary manifestations. Although the onset in the majority of cases is within an hour of surfacing, a latent period of up to 12 hours is not uncommon and even longer times have been reported.

In contrast, the symptoms of pulmonary barotrauma occur usually within a few seconds of surfacing, rarely later than a few minutes. Characteristically the presenting sign is a loss of consciousness without warning but as the particular signs and symptoms depend on the site of the arterial air emboli almost any neurological deficit may occur. Thus the sudden loss of use of one arm could be the result of barotrauma or of decompression sickness and often the final diagnosis remains doubtful even in retrospect. Joint pain, however, is not found in cases of air embolism and, due to the nature of the injury, there are occasionally some additional features which would not be found in decompression sickness. Pneumothorax, which may be bilateral in severe cases, is one consequence of the overdistension of some portion of the lungs but it rarely causes the presenting symptom and may remain undetected until expansion of the gases within it causes dyspnoea during a therapeutic decompression. Detection of such a lesion in a compressed air environment is extremely difficult due in part to the alteration of the percussion note and breath sounds at pressure and not till a therapeutic pleurocentesis was needed would the diagnosis be confirmed. The escape of air into the mediastinum and the root of the neck, causing a subcutaneous surgical emphysema, is diagnostic of pulmonary damage. However the distinction between decompression sickness and barotrauma as the cause of any one incident has little practical significance in its early treatment.

Diagnosis

The diagnosis is usually obvious. The temptation to ascribe the symptoms of joint pain to some recent sports or other injury must be avoided as injured tissues are known to be a preferential site for the formation of bubbles.

When the presenting symptom is joint pain it is necessary to make sure that there are no symptoms of a neurological or pulmonary lesion as these would affect the course of treatment.

It is important to remember that in the early stages of the illness when it is most responsive to treatment there may be no abnormal physical signs. A detailed examina-
tion at atmospheric pressure is therefore contraindicated if it would delay the start of treatment.

Treatment

The treatment of decompression sickness and of air embolism is by immediate recompression, to reduce the size of the bubbles, followed by a slow decompression, to allow time for elimination of the gases. Such treatment can be given only in a pressure chamber. The old remedy of sending the diver down into the water again is to be discouraged strongly as the endurance of the diver and his air supply are unlikely to be sufficient for an adequate therapy. An inadequate therapy can make the diver’s condition worse.

Recompression should begin at the earliest opportunity. Experience has shown that if recompression cannot begin immediately the condition of the patient is likely to deteriorate and become less responsive to treatment. Every case of decompression sickness, even if no more than a mild pain in just one joint, should therefore be regarded as a medical emergency. Air embolism is an emergency of such urgency that the training of divers in the technique of making a free ascent without breathing apparatus is permitted only where there is a suitable recompression chamber and team to operate it at immediate notice within a few yards of the point of surfacing. Nevertheless, treatment of some cases has been successful when it has begun as late as five days after the onset of symptoms.

From the point of view of the medical practitioner first called to render assistance, the task is primarily that of ensuring the successful transfer of the patient to a recompression chamber without delay. If the journey is made by helicopter it should not exceed 1000 ft altitude. During this transfer, oxygen may be administered but drugs such as morphia are contraindicated as they obscure the clinical features so much as to make the later management under pressure very difficult. Serious cases may be complicated by an increased permeability of the capillary wall to plasma proteins causing a hypovolaemic shock for which intravenous plasma is indicated.

Recompression chambers are available at only a few centres. In the United Kingdom most belong to the Ministry of Defence (Navy) and those that are owned by commercial diving companies are unlikely to be used for the treatment of sports casualties. The details of the recompression treatment schedules are to be found in the Royal Navy Diving Manual and this, together with the essential team of men required to operate the chamber, would be available at these sites. Any assistance requested by National Health Service or other sources would, however, be subject to the availability of a chamber and to other Service requirements. The medical officer responsible for treatment would reassess the condition of the patient when he has been recompressed to the depth of full relief of his symptoms. Often, when breathing oxygen, this is at a pressure equivalent to that exerted at a depth of 60 feet in sea-water (2.8 atmospheres absolute) and if the relief of pain is prompt and complete the subsequent decompression takes no more than a few hours. In more difficult or serious cases a pressure equivalent to a depth of 165 feet (6 atmospheres absolute) is reached at which relief usually occurs. It is at this particular depth that a detailed physical examination is required if the diver has any residual symptoms. For reasons of oxygen and nitrogen toxicity it is not possible to increase the depth by more than another 60 feet or so for very long and therefore it is the management of the case which does not respond at 165 feet that requires particularly careful attention. At this stage the medical responsibility should have passed to a doctor with special experience of this illness.

During the lengthy decompression from the maximum therapeutic depth it is important to reassess the patient’s condition at each stoppage in order to make sure that no insidious new loss of function has occurred. Any relapse or recurrence must be
treated by further recompression to the depth at which the symptoms are fully relieved. It may also be necessary to catheterize the bladder, to give intravenous fluids or to perform a pleurocentesis. When the patient has derived maximum benefit from recompression and has reached atmospheric pressure once again it is essential that he remains in the immediate vicinity of the chamber for up to 24 hours so that he may be promptly recompressed if trouble recurs.

A patient who has had even the mildest of symptoms suggestive of decompression sickness should not dive for at least another five days. If he has had neurological decompression sickness or pulmonary barotrauma he should be advised that he must give up diving and seek some other sport. If any neurological symptoms or signs persist after treatment it is a reassuring feature of decompression sickness that continued improvement can be expected for some months.

REFERENCES


---

**ACCOMMODATION AT COLLEGE HEADQUARTERS**

Temporary residential accommodation for members and associates and their families is provided at college headquarters. This building, overlooking Hyde Park on one side and Princes Gardens on the other, is central and easily accessible.

The charges, including breakfast, are as follows:

<table>
<thead>
<tr>
<th>Type of Accommodation</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>For single rooms</td>
<td>£2 10s. 0d. per night</td>
</tr>
<tr>
<td>For double rooms</td>
<td>£4 5s. 0d. per night</td>
</tr>
<tr>
<td>For flatlet (bed-sitting room for two, bathroom and dressing room)</td>
<td>£6 per night or £36 per week</td>
</tr>
<tr>
<td>For self-contained flat (double bedroom, sitting room, hall, kitchen and bathroom)</td>
<td>£42 per week</td>
</tr>
</tbody>
</table>

Children under the age of 12 years cannot be admitted, and dogs are not allowed.

Members and associates may, subject to approval, hire the reception rooms for meetings and social functions. The charges for these are:

<table>
<thead>
<tr>
<th>Type of Room</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long room (will seat 100)</td>
<td>25 guineas for each occasion</td>
</tr>
<tr>
<td>Damask room (will seat 50)</td>
<td>15 guineas for each occasion</td>
</tr>
<tr>
<td>Common room and terrace</td>
<td>15 guineas for each occasion</td>
</tr>
</tbody>
</table>

A service charge of 10 per cent is added to all accounts to cover gratuities to domestic staff.

For the convenience of members, four car ports, outside 14 Princes Gate, have been rented by the College and may be hired, at a cost of 10s. 6d. per 24 hours.

Enquiries should be addressed to the Administrative Secretary, The Royal College of General Practitioners, 14 Princes Gate, London, S.W.7. (Tel. 01-584 6262). Whenever possible bookings should be made well in advance.