8.5.5(f) Diving medicine

Diving medicineChemical and physical injuries and environmental and occupational diseasesD. M. Denison

We often think of humans as able creatures that have conquered the globe, but almost three - quarters of the Earth's surface is covered by sea, which they invade with very little success indeed. Other mammals, such as dolphins, explore it with much greater mastery because they have various features, such as a highly streamlined shape, a much thicker layer of subcutaneous fat, a more mobile chest wall, and bronchioles reinforced all the way to the alveoli, which men are unable to engineer or match for themselves except by very cumbersome means.

Because this is so, human divers are exposed to many hazards that marine mammals avoid. Frequently it is too late or too impractical to give divers specific help once they are in trouble, so diving medicine is largely concerned with prevention. It depends upon a thorough understanding of the job a diver must do and the risks he runs in completing it. In this sense, almost every diving accident is a failure of education or equipment design. However, many well - trained and well - equipped divers come to grief when they are driven or drive themselves beyond limits that are clearly understood but expensive to maintain.

This chapter begins with a description of the environment, which is usually dark, cold, and too deep to stay in without risk for much time. After discussing some features of the sea, and the basic but minor problems of vision, hearing, dexterity, and mobility, it concentrates on the major hazards of air embolism, inert gas narcosis, oxygen toxicity, decompression sickness, the high - pressure syndrome, and the difficulty of keeping warm. It finishes with notes on the selection and general medical care of divers, and some observations on long - term health effects of diving.

THE CONTOUR OF THE SEA BED

Most people have a hazy idea of the contour of the sea bed (Fig. 1). Leaving a typical shore, the sea bed falls away gently, with a slope of about 1:50 until it is 200 to 300 m deep. This shallow stretch is the Continental Shelf. It then angles more steeply (roughly 1:15), the Continental Slope, to descend to vast flat expanses of soft mud, known as the Abyssal Plains, which lie at depths of 3 to 6 km. These are interrupted by occasional mountain peaks and deep chasms. One of the mountains is slightly taller than Mount Everest. The deepest point is just over 11 km below the surface.

Various deep currents, arising from differences in water temperature and salinity, take regular courses across the Abyssal Plains, welling up the sides of the Continental Shelves as mineral - rich streams. These supply the vegetable life that exists in the sunlit upper zone. The animals that feed on these plants, or on each other, become concentrated in these waters so that 80 per cent of the biological wealth of the sea lies in the top 0.2 km, mainly close to the Continental Shelves. Put together, these sites form an area equal to that of Africa, infinitely more fertile, and, as yet unfarmed.

At the surface, regular tidal currents vary widely in velocity from point to point as they are accelerated or slowed down by features of the shore. They often exceed the speed at which humans can swim (Fig. 2(a), so that it may only be practical to dive in slack water, i.e. for an hour or two each day. Similarly, waves are often sufficiently tall to prevent a diver being launched into the sea or recovered with safety (Fig. 2(b)). These surface phenomena rarely penetrate below 100 m or so, but tidal currents can be tunnelled along marine canyons, and springs of fresh water, or falls of cold ocean water, can carry divers in unexpected directions without them necessarily being aware that they are moving at all.

VISION

Most recreational diving takes place in clear warm waters at placid times of the year, but many tasks in professional diving, e.g. harbour work, hull inspections and repairs, pipe - line surveys, oil - rig work, and wreck salvage, occur throughout the year, alongside or beneath large obstructions, in waters where simply finding the task, let alone completing it properly, may be very demanding indeed.

There are many reasons why this should be so. During the middle of the day about 5 per cent of the light falling on the surface of the sea is reflected back to the sky, but, whenever the sun is lower, much more is reflected, so dawn arrives late and dusk comes early to the sea. The light that does penetrate the surface is quickly altered by absorption and scattering. On average, intensity halves with every 1 or 2 m of descent. In consequence, even in very clear water, it is effectively 'night' below 80 m. However, even in shallow water, for example in harbours, near estuaries, close to wrecks, and after storms, sea - water can be very turbid, blurring the boundaries of objects to such an extent that the diver may have to be within 15 cm or so of the objects to see them at all. In these circumstances, artificial illumination is often ineffective because of back - scattering.

Normally, some 65 dioptres of refracting power are needed to focus parallel rays of light on the retina, and two - thirds of this refraction occurs at the air - cornea interface. Under water, this boundary is lost unless some form of gas - filled goggles or mask are worn. If they are worn, the additional glass - water boundary makes objects seem 30 per cent bigger and nearer than they actually are. This distortion is easily adjusted to but very often the margins of the goggles or mask restrict the visual fields severely giving the diver a blinkered, as well as blurred, view of his surroundings.

HEARING AND BLAST INJURY

Under water, localization of sound is poor because binaural cues disappear. They are lost because sounds are transmitted almost five times as fast and many times more efficiently through water than air. So there is very little alteration or delay as sound passes from ear to ear. Localization is also affected by echoes bouncing from the air - water interface above and the water - ground boundary below. The loss of air - conduction alone raises auditory thresholds by 30 to 60 dB. The neoprene - foam hoods that keep the head warm raise them by a further 30 dB or so.

The superior transmission of sound in water also increases susceptibility to blast injury. This is important, even in civilian fields, because plastic explosives are often used to free propellers from shafts or to ease buried objects from marine concretions, and the sort of detonation that would leave a human unharmed at 5 m range in air would be sufficient to kill at the same range under water. Damage is mainly due to abrupt decompression and recompression of the lungs, gut, and sinuses (see later). Gas - free tissues, such as the liver and spleen, are usually undamaged. The air - filled foam of some wet suits provides some protection.

DEXTERITY AND MOBILITY

Except for the surface waters of tropical seas, all of the oceans are too cold for individuals to stay long without insulation (Fig. 3). In air, they maintain body temperature at 37 °CC with minimal effort when the air temperature is 18 to 24 °CC, the zone of thermal neutrality. In water, this zone is higher and very narrow (35–35.5 °CC). Loss of tactile discrimination and manual dexterity are major problems for divers working in cold water. In general, there is a steep loss of discrimination once finger - skin temperature drops below 8 °CC, and there is a substantial loss of dexterity and grip - strength whenever the effective cold exposure of the forearm exceeds that of 20 °CC water for 30 min. Re - examination of Fig. 3indicates how easily such conditions are realized.

The most serious, and least tractable, problems of diving occur from working in a dense and viscous medium. Water is some 1300 times denser than air. Because it is virtually incompressible (the water at the deepest part of the ocean is 4 per cent denser than at the surface), ambient pressure rises linearly with depth. It does so, at very nearly 1 atm for every 10 m descent. Thus at 10 m depth, the surrounding pressure is 2 atm (one of water plus one of air), at 20 m it is 3 atm, and so on. Immersion in a fluid of this density makes the diver weightless and vertically unstable; it opposes any movement generating turbulent flow and exposes him to much higher pressures than on land. As water is also many times more viscous than air, it is useless as a ventilating fluid and opposes any body motion generating streamline flow.

When a diver enters water his body becomes 'weightless' because the density of sea - water, blood, tissue fluids, and the body as a whole are very nearly the same. As a result, his tissues are rehung on the skeleton, and blood and other fluids are displaced upwards, some 500 ml entering the chest, distending the large veins and the right atrium. Stretch receptors in the chest interpret

this signal as an excess circulating volume and promote diuresis until the central distension is reduced to its normal value. When the man emerges from the water the normal hydrostatic gradient of body materials is unopposed and blood drops away from the chest, leaving the man hypovolaemic. Any negative pressure - breathing exaggerates this effect (see later).

Because the immersed body is weightless it can be displaced vertically with ease. Although this permits the diver to poise himself anywhere or examine the full height of a submerged rig at will, it is a great disadvantage, because he can no longer use his body weight to apply leverage or torque, or to stay in place when a current is running. More importantly, any vertical movement can quickly become uncontrolled because of the positive feedback between depth and buoyancy. Because the gas in the chest and abdomen is compressible, the deeper a breath - hold diver goes, the denser he becomes and the more rapidly he falls. The higher he rises, the less dense he becomes and the faster he ascends. Such changes should be seen in the context of the man's actual buoyancy and swimming power.

Although the rises of cardiac output and alveolar ventilation, with effort, are the same in air and water, much of the effort is dissipated in simply moving the limbs through the medium, making most tasks more tiring, and much less efficient below the surface than above (Fig. 4). The maximum sustained thrust that a swimmer can develop underwater is about 5 kg, which is just enough to propel him at about 1½ miles an hour. An opposing force of 5 kg, i.e. that weight on a string over a pully, would immobilize him. If a normally built man takes a full inspiration he is about 2.5 kg positively buoyant and so requires half of his maximum swim - power to descend. If he breathes out to residual volume he is about 2.5 kg negatively buoyant and needs half his maximum swim - power to ascend. The chest wall is a floppy structure that can only maintain a pressure difference equivalent to 1 or 2 m of sea - water, so the gas in the lungs is at virtually the same pressure as the water surrounding the chest. Thus a breath - hold diver leaving the surface with a full lung and diving 30 m (4 atm absolute) will compress his lung from total lung capacity to residual volume, forcing the man to use half his aerobic capacity to overcome positive buoyancy at the start of the dive and the same amount of effort to ascend. If he is wearing insulating clothing (e.g. a foam ‘wet suit’, normal buoyancy about 8 kg) the total changes in buoyancy are very close to maximum swimming capacity, leaving little margin for controlling unexpected ascent or descent.

MECHANICAL EFFECTS OF COMPRESSION AND DECOMPRESSION

When a breath - hold diver descends the gas in his lungs is compressed by the surrounding water, according to Boyle's law, as just instanced. On return to the surface the gas will re - expand to its original volume, with no risk of lung rupture. However, if a diver has access to fresh gas at any time after leaving the surface, he can fill his lungs with enough gas to burst them on ascent, unless they are adequately vented. The principles that determine whether the lung will rupture have been mentioned in detail elsewhere (see Section 17): the lung can be considered as a cluster of tubes and balloons with bursting pressure of about 75 mmHg (1 m of sea - water) and time - constants of emptying that are normally close to 1 s. The latter may be grossly prolonged by injury, inhalation of water, or by disease. Divers are taught to exhale continuously whenever they ascend, and to ascend no faster than the bubbles they exhale. If people with healthy lungs ascend in this manner, the alveoli have time to empty sufficiently and the risk of lung rupture is very low. The mechanics of lung rupture are not clear. Contrary to common sense it does seem that people whose lungs are small for their body size but otherwise normal are at greater risk, perhaps because the chest wall can exert greater traction on the small lung. It appears also that people with entirely healthy lungs can rupture them simply by taking a vigorous deep breath, as in lung - function testing. Such episodes of spontaneous pneumomediastinum are usually asymptomatic. However, should they occur underwater the trapped gas will expand, causing serious injuries. For this reason, it presently seems wise to suggests that divers should never take extremely deep breaths underwater.

Lung rupture usually occurs in divers breath - holding while making emergency ascents after losing their gas supply. Central tears of lung tissue lead to mediastinal emphysema. Peripheral tears cause pneumothoraces. In both cases, but more commonly the former, gas may also enter the circulation as air emboli. Whether it is in the tissue, pleural space, or bloodstream, the

escaped gas expands as the ascent continues, making matters worse. The victim usually notes dyspnoea, cough, or haemoptysis during the ascent, or no more than a few minutes later. If the tear is central he may also note dysphasia, voice change, or a sense of fullness in the throat or behind the sternum. On examination there may be surgical emphysema of the neck and upper chest, subcutaneous crepitus, increased cardiac dullness, or cardiac crepitus. If a pneumothorax has developed it is often accompanied by one - sided pleuritic pain, diminished respiratory movement, deviation of the trachea and apex beat, hyper - resonance, and distant breath sounds. If air embolism has occurred, there will be additional neurological signs. Patients with uncomplicated mediastinal and or superficial emphysema are treated by giving them pure oxygen to breathe, but they must never be positive - pressure ventilated. The oxygen accelerates bubble absorption by emptying the blood of inert gas so it is a more efficient sink for the gas in the bubble. After 2 or 3 days the emphysema will have subsided and the patient will be fit for release. Small pneumothoraces can be treated in the same way. If the pneumothorax is large it can be relieved in the ordinary way with a chest drain; however, if it is critically large at depth the patient should be recompressed immediately to reduce its size. This is rarely necessary for uncomplicated pneumothoraces. The chest drain must be clamped on compression and must be exposed to continuous suction at depth and during decompression.

If there is any evidence of air embolus, or any doubt about the diagnosis, the patient should immediately be laid semiprone, on his left side, with the head down and the buttocks up. He should be given oxygen, without positive - pressure ventilation, and be recompressed as soon as possible (details of recompression therapy are discussed below).

Lung rupture is the most worrying form of barotrauma (pressure damage) but others are more common. On descent, the diver may have difficulty in ‘clearing his ears’ because the mounting pressure on the outside of the drum is not balanced by an equal rise on the inner side, due to the valvular nature of the eustachian tube. This can be very painful, and may lead to drum rupture, but the strain on the drum usually resolves immediately on stopping the descent and ascending slightly. Sometimes the sinuses are affected on ascent or descent, in the same way. Pain on descent is always relieved by ascent. However, pain due to gas trapped in an obstructed space gets worse on ascent. Sometimes blood that has filled a sinus, and partly clotted during descent, is expelled in this manner as the diver returns to the surface. Occasionally, an ethmoid sinus may rupture into the cranial cavity. Usually, no specific treatment is needed for these injuries, but the diver is laid off diving until the cause has been established and he is fit to return. Pressure injuries also occur if the gas pressure inside the goggles, mask, or helmet does not keep pace with that outside. Sometimes, reversed - ear injury occurs because on descent the soft foam wet - suit helmet prevents a matching build - up of pressure in the external auditory canal, and the drum blows outwards.

INERT GAS NARCOSIS

Because the chest wall is a floppy structure, gas must be delivered to the diver at the same pressure as the water that surrounds him. This may be sent to him via an umbilical pipe from the surface, in which case it can flush through his helmet or face - mask continuously, which is wasteful of gas, but easily engineered, or it can supply a regulating valve that provides gas on demand only (surface - demand systems). Alternatively, the diver can take a self - contained underwater breathing apparatus (SCUBA) with him. This always feeds a demand regulator and rarely lasts for more than 1 h. Professional divers often use a combination of all three systems, i.e. a surface - demand supply for routine use, with a helmet - flushing capability for occasional comfort or emergency use, and a small, back - mounted gas supply ready in case the surface supply fails.

Nowadays, demand regulators and compensated outlet valves are well designed and there are few practical obstacles to providing the diver with gas at the right pressure. The composition of the gas to be breathed is a much more complex decision. Air can be breathed quite safely down to depths of 50 m, although tests of sophisticated cerebral function show there is already some impairment at 20 m. Below 50 m, mental deterioration becomes increasingly obvious, manifested by such actions as the diver offering his mouthpiece to neighbouring fish.

This condition is described as nitrogen narcosis or*‘l'ivresse des profondeurs’*. It is a specific example of the more general condition of inert gas narcosis related to inhalation anaesthesia. An

extension of the Meyer - Overton lipid solubility theory of anaesthesia supposes that the size or thickness of the nerve membrane, or some part of it, is dependent upon the size and number of gas molecules dissolved in it, and once the size or thickness of the membrane exceeds critical limits it cannot conduct an impulse. On descent breathing air, ambient and arterial nitrogen pressures rise, and more nitrogen dissolves in nerve membranes, making them thicker, until at a depth of about 50 m, interference in neural transmission becomes obvious functionally. Replacing the nitrogen molecules with smaller and less numerous helium molecules allows the membrane to shrink to an acceptable size and the impairment of function regresses (cf. replacing nitrogen molecules by bulkier xenon molecules, which is sufficient to cause anaesthesia at sea level). As would be expected, these changes in function develop within a few minutes and are rapidly reversible, because they depend purely on the process of passive chemical solution.

If men breathe an oxygen - helium mix, rather than air, they can descend to the lowermost parts of the Continental Shelves (730 m) without narcosis. Thus, nitrogen narcosis is a wholly preventable hazard in diving, which if it occurs by accident can be completely reversed within minutes, by ascent, and therefore needs no treatment. Unfortunately, helium is an expensive gas and there is an understandable reluctance for diving operators to use it. Medical advisers must oppose this and emphasize that it is dangerous for men to breathe air below a depth of 50 m or so.

HIGH - PRESSURE NERVOUS SYNDROME

At great depths, breathing oxygen ± helium mixtures, men show various neurological disturbances that are apparently not narcotic but are due to the direct effect of pressures on nerve tissues. This is thought to be so because very similar states can be induced in animals who are ventilated on fluorocarbon liquids containing normal sea - level quantities of oxygen and nitrogen or helium. It is also known that many organic and inorganic processes are disturbed by the applications of high barometric pressures *in vitro*. The high pressures are believed to alter the natural aggregations of water molecules, to increase the ionization of salts, oppose ionic bonding, liquefy gels, and cause various enzymes to fail. It appears to be the major obstacle to ambient - pressure diving much below the edges of the Continental Shelves. At present, men are able to make experimental dives to about 700 m. Various drugs have been investigated as modifiers of the syndrome, but its fundamental cause and protean manifestations suggest this depth will be close to the absolute limit to ambient - pressure diving.

OXYGEN TOXICITY

The hazards of inert gas narcosis, and of decompression illness (see below) could be avoided almost completely if people breathed pure oxygen when they dived. Unfortunately it becomes toxic to the lungs when the alveolar oxygen pressure exceeds half an atmosphere (5 m of sea water) and it becomes toxic to the nervous system when the alveolar, and the arterial, oxygen pressure exceed 2 atm (10 m of sea water). These effects are due to complex chemical interactions, rather than physical solution, and so take time to develop and reverse.

As mentioned elsewhere, respired oxygen is handled by a large number of enzymes and is used in very many ways. Some, such as the production and destruction of neurotransmitters and the synthesis of some steroid hormones, are critical. Increasing the amount of oxygen in simple solution not only affects the balance of some of these processes but also increases the risk of randomly forming the destructive superoxide ion. So, high - pressure oxygen affects body tissues in many ways.

It has two principal actions on the lung. Firstly, it promotes simple absorption atelectasis by replacing the relatively insoluble nitrogen in alveolar spaces. As their servant airways shut off due to transient obstructions, pulmonary blood flow rapidly removes the highly soluble oxygen and CO_2 causing the alveolar spaces to collapse, leading to linear regions of atelectasis that are most obvious in the well - perfused and more readily collapsed basal regions of the lung. Paradoxically, blood leaving the lung is then hypoxic due to admixture with blood perfusing the collapsed spaces.

Secondly, oxygen damages the lung by irritating its endothelial and epithelial surfaces. Some people suggest this is due to the extrapulmonary generation of a noxious agent because the endothelial damage appears first, but it is much more likely to be a direct action of alveolar

oxygen on these surfaces. At first there is an exudation of fluid and proliferation of macrophages and an interference with surfactant production. This damage is reversible. If the exposure is prolonged, fibrosis occurs and the lung is permanently scarred.

The time taken for symptoms to appear depends upon the dose. It varies from several hours at half an atmosphere P_{O_2} to a few hours at 2 atm. Above that pressure, the neurological sequelae overshadow the pulmonary damage that still occurs (Fig. 5).

Oxygen interferes with nervous tissue in a manner that is not yet understood, perhaps because it disrupts many processes rather than one. It manifests itself by epileptiform convulsions that are sometimes, but not always, preceded by aura such as twitching of the face and hands. Any convulsion underwater is potentially fatal and must be avoided. There is a safe latent period, during which oxygen can be breached without any detectable harm to the central nervous system. This time is inversely proportional to depth (i.e. to P_{O_2}). However, the time and threshold level can vary widely in the same individual from day to day. Retention of CO_2 and exertion lower the threshold and shorten the latent period. Like nitrogen narcosis, oxygen toxicity is a preventable hazard in diving. Divers should not breathe oxygen at partial pressures greater than 2 atm except under observation in the decompression chamber. Diving mixtures that are to be breathed for several hours should maintain inspired P_{O_2} between 0.5 and 1.0 atm. Those that are to be breathed for longer periods should maintain inspired P_{O_2} between 0.2 and 0.5 atm.

DECOMPRESSION ILLNESS

Oxygen toxicity, nitrogen narcosis, and the high - pressure nervous syndrome set depth limits to diving of 10, 50, and 500 m, respectively, and so are physicochemical obstacles to invading the sea. By contrast, the major medical hazard to diving, decompression sickness, is a physicochemical obstacle on returning from the sea. It occurs because, during any dive, extra inert gas, usually nitrogen or helium, goes into passive solution in the body. On ascent, as the ambient pressure falls, this gas can come out of solution in an uncontrolled way, forming bubbles in the circulation and within tissues. As the ascent continues, these bubbles increase in size and number, blocking blood vessels, and distorting or rupturing cells. On re - descent the bubbles contract and are eventually resorbed. If the first or the subsequent ascent is slow enough, few if any bubbles are formed, the extra gas diffuses into the bloodstream and out of the lungs easily, and the diver reaches the surface unharmed.

Experiments by Haldane at the turn of the century, which have been confirmed subsequently many times, suggest bubble formation occurs whenever ambient pressure falls below about half the total pressure of inert gas in solution. It follows that it is normally safe to ascend from dives to 10 m or less, without hesitation, however long they have endured, and to ascend without stopping, from any deeper dive that has been too brief to take this critical mass of gas on board. For any other dive, return to the surface must be delayed so the ratio of pressure of gas remaining in solution: ambient pressure never exceeds the critical value.

More recent work indicates that the critical value varies from tissue to tissue, and may not reflect a simple ratio of the dissolved nitrogen and ambient pressures, but could be determined by the absolute differences between the two pressures. Because, in general, it is unethical to explore these issues in man, and species differences in susceptibility to decompression illness are very marked, further understanding is most likely to come from accurate epidemiological observations in very large numbers of divers. The development of inexpensive miniature depth - time recorders that can easily be attached to a diver's weight - belt should be very helpful in this regard.

A vast amount of experimental work has been done to determine the safe limits to ‘no - stop’ diving and the depth - time profiles that have to be followed on returning to the surface after any longer dive. The time - limiting curve for ‘no - stop’ diving is shown in Fig. 6. It represents the time taken to accumulate 2 atm of dissolved gas in the tissues at the depths shown. The rate of gas accumulation in tissues is determined by the ratio of the solubility of the gas in that tissue to the speed of its blood flow. Because the tissues vary widely in these respects, the body behaves as if it were made up of a series of compartments, the ‘fast’ ones having short time - constants of inert gas uptake, and the ‘slow’ ones having long time - constants. The fast tissues such as working muscle dominate the time - course of safe short dives. The slow tissues such as body fat determine the safe ascent rate from long dives. Knowledge of safe practice is tabulated in a series of lengthy decompression schedules, which vary somewhat from one country to another,

and are to be found in any textbook of diving medicine. After long (saturation) dives, the ascent is very slow and can take several days.

After relatively brief, shallow descents, divers can return to the surface slowly, stopping for a few minutes at scheduled depths. To do this they normally ascend up a marked shot - line. It is not practical or safe to ascend from deeper or longer dives in this manner, so a submersible recompression chamber is sent to the bottom to collect the diver and return him to the surface in warmth and comfort, or the divers make a limited number of stops in the water and then quickly ascend to the surface and immediately enter a deck - mounted chamber to be recompressed and then decompressed slowly and safely. Usually they are recompressed to 20 m and then given oxygen to breathe intermittently, to accelerate inert gas excretion.

About 1 per cent of dives conducted to authorized schedules, and many badly conducted dives, lead to decompression sickness. This can take two forms, skin irritation or limb pain only (type 1 'bends'), or any other serious manifestation (type 2 'bends'). Skin irritation and mottling alone is treated by oxygen inhalation at the surface and does not require recompression. It is the mildest form of decompression sickness. All other cases should be recompressed as soon as possible. This is the only effective treatment. The object is to reduce the size of existing bubbles and prevent the formation of new ones, before irreversible infarction and oedema have occurred.

About half of all cases involve the central nervous system, most often the lower cervical, the thoracic, or the upper lumbar segments of the spinal cord. Visual disturbances and other cerebral signs are seen often also. Many others involve limb pain, commonly of the shoulders or elbows in divers and of the knees and hips in tunnel workers. A minority of victims experience sudden chest pain, dyspnoea, and cough, believed to be due to bubbles in the pulmonary circulation. Symptoms appear minutes to hours after the end of a dive. Some of them are due to bubble formation directly—others are believed to be secondary to clumping of red cells.

If the symptoms are of limb pain only, the patients are recompressed to 20 m on oxygen. Providing the pain disappears within 10 min of recompression, they are then decompressed over a period of 135 min. If the pain persists, the decompression time is doubled. If there is any evidence of central nervous or chest involvement, at the time of initial diagnosis or subsequently, the patient is immediately put semiprone on the left side, with the buttocks raised, recompressed to 20 m on oxygen and decompressed over at least 4.5 h. Sometimes it is necessary to recompress the patient briefly to 50 m or so to relieve persistent symptoms or signs. Some people give low molecular - weight dextran to combat the clumping of red cells and heparin to reduce any lipaemia from tissue disruption. It is important to emphasize that the only effective treatment is recompression, and that this may still be beneficial even if it cannot be achieved for several hours.

THE PROBLEM OF KEEPING WARM

The general problems of survival in cold water are described inChapter 8.5.5(c). However, some difficulties are peculiar to divers. They insulate themselves in one of three ways: by cramming as much clothing as possible beneath a supposedly impermeable layer of rubber or canvas sealed at the neck and wrists (a 'dry' suit); by wearing an open - cell foam 'wet' suit that contains air at the surface but immobilizes water freely percolating in; or by wearing a closed - cell suit that consists of gas - filled plastic bubbles. All three suits rely on some gas for insulation and thus are buoyant and compressible. Typically a wet suit will provide some 8 kg of buoyancy, i.e. contain some 8 litres of air. As the diver descends, this gas compresses, making him heavier and colder. The loss of insulation is very noticeable. At depths greater than 50 m it is compounded by the need to breathe helium, which has a high thermal conductivity especially when it is compressed. In fact, the conductivity of helium is so high that gas - filled habitats for saturation diving have to be kept at temperatures close to 30 ℃C to achieve thermal neutrality. When divers emerge from the habitat they lose heat rapidly through the respiratory tract and through the 'air' filled insulation, which now contains helium. It is often necessary to provide them with personal heating systems (e.g. piped hot water).

ATMOSPHERIC PRESSURE DIVING

In classical diving, men invade the sea breathing gas at ambient pressures which rise linearly with depth. These men are able to deploy most of their sensory and motor skills but are very

seriously limited by cold and the chemical consequences of breathing gases at high pressures. Two of these, oxygen toxicity and inert gas narcosis, restrict the choice of breathing mixtures. A third, decompression sickness, greatly reduces the maximum permissible rate of ascent. Divers cannot be launched or recovered when wave heights exceed a few feet, are unable to swim against currents greater than 1½ knots and are usually too cold to work for longer than 90 min. The long times spent in controlled ascent cannot usually be employed profitably, but detain expensive equipment, sometimes in the face of worsening weather. At present, it is difficult to imagine that classical divers will be able to descend much lower than 500 m. This gives them access to the whole of the Continental Shelves but not to the Continental Slopes or the Abyssal Plains beyond.

There are three other ways in which one can explore the sea. First and most safely, one can send down unmanned sensors and manipulators. Almost all of our present knowledge has been obtained in this way. Many of the exploratory devices are dropped at one point as spot samplers to measure local values of particular variables, which they record and transmit from the bottom or on return to the surface. Others can be towed with a fair degree of vertical and lateral control relaying similar information along a cable. Some can be envoyed remotely under sonar or an internally programmed control, storing or transmitting information as they go. As investment pressure rises, more of these will be fitted with manipulators to perform specific tasks.

Unmanned devices do not imperil human life, may be designed to work without a bulky pressure protection, should be able to move freely to and from great depths, and have the important advantage that in need they can be instructed to wait at the bottom indefinitely until surface conditions are favourable for their recovery. Their serious disadvantages are cost, lack of dexterity, and inflexibility of mission purpose. (To get these in perspective, it may be helpful to consider as a parallel the problems of exploring and redeveloping a dry continent, if it could only be done with towed or remote robots operated from night - flying helicopters.) There are bound to be considerable technical improvements in manipulators, sensors, power sources, and programme controls. Similarly, more marine engineering and mining plants will be designed to be built and maintained by telechiric devices. Nevertheless, at present, no one can envisage a time when submarine robots with anything like human capabilities could be constructedfor economic sums.

Secondly, men can enter the sea in pressure - resistant vessels that can be towed, powered via a cable, or moved under their own steam. All of the present submersibles are fitted with porthole transparencies, most have additional sensors, and some have primitive manipulators. They can search large areas and some can perform simple tasks. Their present disadvantages are weight, restricted vision and manoeuvrability, bulk, poor manipulator performance, limited operability (most cannot be launched or recovered when wave heights exceed 1 m), high capital and operating costs, and the need for a large and specially equipped surface support vessel.

Thirdly, men can invade the sea in armoured diving suits, a relatively cheap weightless but warm second skin capable of resisting high ambient pressures but putting less restriction on touch, vision, and dexterity than the larger devices. These leave the diver with many of his sensorimotor skills and give him vertical freedom, greater depth range, and very reduced ascent times.

Although such suits were tried and found wanting in the 1930s, they are now easier to make and the need for them has risen greatly. At present they have a working depth limit of about 500 m and are able to ascend and descend freely. The inhabitant remains dry and warm, breathing air at atmospheric pressure throughout. When he surfaces, someone else can descend immediately using the same suit. It is likely that, in the future, much deep - sea diving will be conducted in this way.

THE SELECTION AND MEDICAL CARE OF DIVERS

Divers have a physically demanding job and often must work in sites remote from any medical aid. They must have a high exercise tolerance and be free of any active or latent condition that could erupt while they are away from medical help. In addition, specifically, they must not have any condition that could imperil their own lives or those of their would - be rescuers when they are under water.

In Britain all commercial divers have to pass an annual medical examination conducted by a doctor competent in diving medicine. If the diver is found to be unfit to dive and disagrees with the decision of the examiner he has the right of appeal to an independent tribunal set up by the Health and Safety Executive. The codes of safe diving practice have been summarized in the British ‘Health and Safety—Diving Operations at Work Regulations’, which took effect in July 1981. These regulations have been nationally and internationally accepted.

In principle, all divers are expected to be physically able and mentally stable people, free of conditions such as epilepsy and ill - controlled diabetes or asthma. They should not be addicted to alcohol or any other drug and they should not have a history, or any other evidence, of obstructive lung disease, ruptured eardrums, or aural surgery. Divers who are generally fit to dive should not be allowed to do so when they have chest, upper airway or ear infections, or when they are overweight (because obesity predisposes to decompression sickness). Neither should they dive while taking any medication that could impair their ability to think clearly or orientate themselves in space correctly.

Unresolved issues

There are several unresolved issues in diving medicine: the nomenclature of the disorders associated with decompression, the management of diving accidents, the role of lung function tests in assessing fitness to dive, the significance of patent foraman ovale in divers, whether people with mild asthma should be allowed to dive, and whether there are any long - term effects of ‘safe’ diving on the health of divers. These are discussed below.

THE NOMENCLATURE OF THE DISORDERS ASSOCIATED WITH DECOMPRESSION

The two principal hazards of ascent are lung rupture and the evolution of bubbles in blood and tissues (previously known as decompression sickness). On the whole, lung rupture manifests itself within a few minutes of its occurrence, but ‘decompression sickness’ usually takes several minutes to hours after decompression to become obvious. However, there are exceptions to both rules that can lead to errors in diagnosis and, more importantly, errors in treatment. Many authorities now believe that it is wiser to describe all disorders associated with decompression as decompression illness, defined by the site, acuity, and rate of progress of symptoms before recompression. This system of nomenclature is described in one of the references given at the end of the chapter.

THE MANAGEMENT OF DIVING ACCIDENTS AND EMERGENCIES

Several diving emergencies, for example hypoxia, oxygen toxicity, carbon dioxide poisoning, nitrogen narcosis, hypothermia, various ear, nose, and throat disorders, and underwater blast injury, do not require treatment by recompression, but two of them, acute decompression illness and carbon monoxide poisoning, do. In these two, recompression can be life saving. The Royal Navy's Institute of Naval Medicine at Alverstoke, near Gosport in Hampshire, maintains a 24 - h diving medical watch throughout the year (telephone 01705 818888) to provide advice on diagnoses, treatments, and availabilities of recompression chambers. Their advice is also summarized in a freely available report listed at the end of this chapter.

THE ROLE OF LUNG FUNCTION TESTS IN ASSESSING FITNESS TO DIVE

Military and commercial divers are obliged to have annual medical examinations for ‘fitness to dive’, and sports divers are strongly urged to do likewise. In the past, one of the most stringent requirements of this check - up was that for acceptable spirometry, in particular for an acceptably high forced expiratory volume in 1 s/forced vital capacity **(FEV_1/FVC)** ratio, because of the fear that an obstructed lung was more liable to rupture. It has now become clear that worry is not well founded. The purpose of the medical check is two - fold. Firstly to determine whether the candidate is fit to swim in swift currents and rough waters at remote sites and only secondly to discover whether there is any bar to him or her also diving. In principle anyone who has more than a slightly reduced lung function (FEV_1, FVC, or carbon monoxide transfer) or aerobic capacity will not be fit enough to cope with emergencies in water and they should therefore be

advised not to swim except in calm and supervised sites. Diving is often a relaxed affair, but people normally dive in pairs so that in the case of an emergency one diver can get the other out of trouble. At that stage, diving and survival at the surface can be very strenuous indeed. The prime purpose of the medical check is to determine that the candidate is capable of that activity.

THE SIGNIFICANCE OF PATENT FORAMINA OVALES IN DIVERS

On most, possibly almost all, asymptomatic decompressions, many bubbles can be detected in systemic venous bloodstreams. These bubbles are normally trapped in the pulmonary vessels and are resorbed unnoticed. However, about one - quarter of the normal population has a slightly patent foramen ovale, raising the possibility that, in them, some of the systemic venous bubbles might escape through to the left ventricle, bypassing the lung filter, and continuing on to cause cerebrospinal gas embolisms. This thought is reinforced by the observations that as many as 80 per cent of the victims of cerebrospinal gas embolism have patent foramina. However, it is by no means clear whether that is cause or effect. Although systemic venous bubbles are common, arterial gas embolisms are rare. Bubbles have complicated inflammatory interactions with vessel walls, and any significant load arriving in the lung could trigger pulmonary vasoconstriction, raising right heart pressures and opening a previously ‘closed’ foramen. Presentevidence is equivocal and does not appear to justify excluding one - quarter of the population from diving, when gas embolism is a rare event.

SHOULD PEOPLE WITH MILD ASTHMA BE ALLOWED TO DIVE?

It has been the custom to bar asthmatics from diving because of the fear that they were more likely to rupture their lungs on fast ascents. However, very many mild asthmatics are known to have completed very many dives without ill effect. It seems there is a case for relaxing this requirement slightly, but three questions prevail: is the candidate (i) fit enough to cope with a diving emergency, (ii) liable to exercise - induced asthma, and (iii) likely to bronchoconstrict on the inhalation of salt water? Perhaps the most reasonable view at present is to allow a candidate with very mild asthma to dive, providing they can demonstrate a stable and essentially normal spirometry over a 2 - month period, they do not have exercise - induced asthma, and they do not constrict abnormally to a saline aerosol.

DOES ‘SAFE’ DIVING HAVE ANY LONG - TERM EFFECTS ON THE HEALTH OF DIVERS.?

As mentioned earlier most ascents are associated with the appearance of many bubbles in systemic venous streams, but these usually do not cause symptoms, and so such dives are considered to be ‘safe’. Decompression tables that have been established experimentally define the boundaries beyond which more than 2 or 3 per cent of divers will experience symptoms of decompression illness. Such dives are ‘unsafe’. There is no doubt that the neurological or other sequelae to unsafe dives often fail to resolve completely, but these are regarded as the consequences of unsafe practices. More recently, diving physicians have been asking whether ‘safe’ dives lead to insiduous, cumulative damage. Although the damage is slight or subclinical, there is definite evidence now that they do. Autopsies on asymptomatic divers with no history of acute decompression illness have revealed their brains and spinal cords contain considerably more microinfarcts than non - diving controls. More importantly, radiographs of the long bones of divers and caisson workers show increasing numbers of aseptic infarcts in a sizeable minority (up to 11 per cent). The incidence is higher in those with a history of decompression illness than those without, but occurs in many with no such history. The infarcts can occur after a single decompression, but their incidence rises with age, depth, and diving intensity. Those in the shafts of bone are asymptomatic, but those at juxta - articular surfaces can be severely disabling. They are more common in caisson workers than divers, but are even seen in professional breath - hold divers, such as the *ama*of Japan, in whom the dissolved gas burden must be light. The aetiology is unknown, but gas embolism is the favoured explanation.

There is also good evidence that commercial diving, especially saturation diving causes the lung's total and vital capacities to expand, its FEV_1:FVC ratio to fall, and its pulmonary capillary blood volume, as judged by carbon monoxide transfer, to fall. The effects are slight but definite and may be cumulative. The expansions in lung volumes are attributed to training effects of breathing compressed gases for long times. The fall in FEV_1:FVC ratio is mainly due to the rise in

FVC but there are hints of additional small - airway damage. The fall in pulmonary capillary blood volume appears to be due to transient episodes of hyperoxia during saturation diving procedures, but may also be associated with the influx of bubbles from systemic veins on ‘safe’ decompressions.

It is also known that commercial divers develop a mild degree of high - tone deafness, currently attributed to the noise of gas flows within their helmets.

Conclusion

Diving is a sometimes very vigorous activity that demands a high degree of mental and physical fitness. It exposes people to several physical and chemical challenges that are reasonably well understood. Because it takes place remote from medical help, there is a strong emphasis on prevention of illness by the following of safe practices. The safe practices have been developed empirically on the non - appearance of symptoms in brief trains of dives. There are now indications that these practices may not be quite as safe as first thought, but the cumulative effects are generally slight.

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