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Some Occupational Hazards of Diving

Diving is considered to be a high-risk occupation: for instance there are said to have been some 26 deaths in the North Sea population of around 1000 divers during the last 4–5 years (Warner 1976, personal communication). A detailed analysis of these accidents must await completion of legal proceedings but it would seem that the majority were due to some failure of equipment or diving procedure. Only 2 were reputed to be due to decompression sickness and in 3 there was some underlying medical condition, not necessarily detectable at routine examination, which may have determined the fatal outcome. In several fatalities, however, the primary cause and contributory factors remain uncertain.

It is an objective of this paper to review the hazards to which divers are exposed while at work. Not all accidents have a fatal outcome and, besides the 'near-misses' who may be fit to resume work almost immediately, there are those incidents that leave some residual injury, most commonly a neurological deficit following decompression sickness. Additionally, there are those insidious occupational illnesses to which divers are exposed, the best-known example being aseptic necrosis of the subchondral bone of the head of the humerus and femur.

The hazards due to diving equipment failure and procedural inadequacies are largely beyond the reach of preventive medicine, even though the final outcome may be death by drowning. Nevertheless, there are many circumstances in diving when the possible reduction of hazard is primarily the responsibility of medical practitioners and physiologists. Such risks may rise from the direct effect of pressure upon the body or from the effect of the increased partial pressures of respired gases. These effects may be divided conveniently into those associated with the compression phase, those which are most commonly experienced while at maximum pressure and those which appear as a consequence of the subsequent decompression to normal atmospheric pressure.

Compression Phase

It is well understood that the air-containing spaces of the body are subject to Boyle's law while the tissues of the body are virtually incompressible. Failure to 'clear the ears' during descent can lead to otitic barotrauma and, if severe, a serosanguinous transudate in the middle ear or rupture of the tympanic membrane. Even

mild otitic barotrauma can be associated with 'alternobaric vertigo'. Since the diver is neutrally buoyant in the water and may lack any visible horizon vertigo can lead to a dangerous state of disorientation under water. Indeed, a diver who blew his nose while driving his car, after a dive in which he had some difficulty in 'clearing his ears', was considered negligent by the courts because the sudden vertigo associated with the Valsalva made him have a car accident.

Compression barotrauma can also give rise to pain in the paranasal sinuses and unhealthy teeth, but these are more of a nuisance than a hazard. Compression barotrauma of the chest is a hazard associated with only certain types of breathing apparatus but is not yet sufficiently rare to be regarded as a mere medical curiosity. It occurs when the breathing gas is supplied to the diver at a pressure less than ambient and is therefore not associated with those commonly used types of apparatus in which the pressure-regulating demand valve is incorporated.

At Depth

The dangers of euphoria due to the narcotic action of increased partial pressures of nitrogen are sufficiently well known not to be a hazard to the well-trained diver. The accepted safe limit for air diving is 50 m. To avoid narcosis at greater depth, helium, which is relatively non-narcotic, is the most common gas to be used in place of nitrogen as the inert gas diluent for oxygen in breathing mixtures. Nevertheless, impairment of performance and mental function is associated with the use of helium at very great depths even in the relative comfort and safety of the dry compression chamber. This so-called 'high pressure neurological syndrome' (HPNS) is especially associated with relatively rapid rates of compression to depths of greater than 250 m, and must be considered as one of the many possible contributory factors leading to diving accidents.

The neurotoxicity of oxygen is sufficiently well understood for the risk of an overt oxygen seizure in the water by a diver breathing an oxygen-rich mixture to be largely avoided. Such an oxygen fit would, almost inevitably, have a fatal outcome. However, it must also be appreciated that lesser inspired partial pressures of oxygen may also have an adverse effect and that the effects of oxygen upon, for instance, the cerebral circulation may act synergistically with other factors to produce what has been called the 'shallow water blackout'. The loss of consciousness by divers while at work under water is one of the occupational hazards in which research is currently being undertaken. The other factors which must be considered contributory are diving bradycardia (a reflex response to immersion), the retention of carbon dioxide (a problem associated with the effects of increased breathing gas density upon pulmonary function and considerably influenced by the design of the breathing apparatus) and acute hypothermia (a hazard of all swimming but one which is accentuated by the increased thermal transfer properties of helium at increased densities). With semi-closed breathing apparatus there is also an insidious risk of hypoxia since, in the presence of a carbon dioxide scrubber, there is no CO_2 build up while oxygen is consumed, and thus no respiratory warning when there is a failure of gas supply or when, during hard physical exertion, the oxygen consumption exceeds the rate of its supply.

In the water there is also a hazard from the presence of other marine animals. It is fortunate that there are few animals dangerous to man in the waters around Great Britain, but in warmer waters such hazards do need to be considered. Divers have been taken by shark and swallowed by a grouper. Venomous animals include the stone-fish, jelly-fish and sea snakes. Though antivenin is available for some sea snakes, in many cases of envenomation there is little that can be done beyond normal resuscitative procedures to prevent death.

Decompression

There are two decompression illnesses to which the diver is exposed during his decompression from depth to the surface. One is decompression barotrauma which may occur if the gases within a body cavity expand during the reduction of ambient pressure while some obstruction prevents their venting. The other is decompression sickness which follows a reduction of environmental pressure sufficient to cause the formation of bubbles from gases dissolved in the tissues.

Pulmonary barotrauma is the condition arising from the expansion of gases within the lungs during decompression and can lead to pneumothorax, mediastinal emphysema, pneumopericardium and, rarely, retroperitoneal gas. More seriously, the escape of alveolar gas into the circulation can cause cerebral arterial air embolism. The retention of expanding gases within a portion of the lungs is dependent only upon at least one breath of compressed gas having been breathed while at raised environmental pressure and, unlike decompression sickness, is not dependent upon the uptake of dissolved gas by the tissues. The relative over-pressure in the lungs need not be great; cases have occurred following ascent from as shallow as 2 m. It follows that a breath-hold diver avoids this hazard because he does not take a breath of compressed air while at depth and therefore during his ascent the expanding alveolar gases are no more than a re-expansion

of those inspired at the surface and compressed during descent.

The hazard is greatest if the subject holds his breath during decompression or, mistakenly, 'clears' his ears during ascent. Such errors should be avoided by previous training. The hazard also exists when the diver exhales correctly during decompression if there is some underlying pulmonary etiology which predisposes to localized air trapping. It is also possible that, at rapid rates of ascent, airway collapse in the apparently normal lungs may predispose to localized air trapping. Thus pulmonary decompression barotrauma is a hazard which is present during every decompression of the diver. In addition to the escape of pulmonary gas into the tissues and pleura, a consequence of barotrauma is cerebral arterial air embolism, a condition which, in the absence of a suitable recompression facility, may lead to permanent neurological injury or to death.

Decompression sickness following an exposure to raised environmental pressure is considered to be due to the evolution of bubbles from the gases which have been taken up by the tissues of the body while at depth. Thus the diver is not exposed to this decompression hazard until he has been at a particular depth for long enough to take up a sufficient quantity of gas into his body. Thus at each depth there is a duration of dive after which it is reasonably safe for the diver to return to the surface without running the risk of effervescence. This threshold is known as the 'no-stop curve' which shows that at depths less than 10 m a no-stop return to the surface after extended exposures is safe. This implies that there is an insufficient quantity of gas dissolved in the body of a diver who has 'saturated' at these depths to provide a pathological degree of bubbling. At 20 m the no-stop time is reduced to one hour, at 30 m to 20 min and, as shown by submarine escape trials, at 200 m the safe 'bottom time' is less than one minute. For greater durations of dive it is necessary for the diver to return to the surface at a predetermined rate of ascent calculated to allow the safe elimination of the dissolved gases. Thus the diver is exposed to the hazard of decompression sickness when he fails to adhere to the accepted safe decompression tables. Unfortunately, at greater depths, especially those at which helium is breathed, some decompression tables are themselves unsafe and an incidence of decompression sickness is inevitable even when the individual diver has adhered to these tables. Decompression sickness can also occur if the individual has adhered to a safe table but has some abnormal uptake or elimination of gas. Factors which may influence the safety of a decompression table include obesity, physical activity during the gas uptake phase, and conditions such as exposure to cold during the dive which will affect peripheral circulation and thus gas elimination. The immediate consequences of inadequate decompression range from the relatively mild cutaneous manifestations of decompression sickness, through limb pain to the more serious respiratory and neurological manifestations which can be fatal.

There is also the occupational hazard of aseptic necrosis of bone. The exact etiology of this condition is still unknown but there appears to be an association between bone necrosis and a history of some decompression hazard whether this be acute decompression sickness or an exposure to relatively unsafe diving tables. On the basis of the radiological criteria of the Medical Research Council Decompression Sickness Panel, bone lesions have been detected in about 5% of naval divers and 50% of some groups of civilian divers. Many of the radiological lesions are in the shaft of the humerus, tibia or femur and will remain symptomless. Clinically more important are the juxta-articular lesions which may be symptomless when detected but which may progress to subchondral collapse and secondary osteoarthritis of the shoulder or hip joints.

Prevention and Management

It is not the purpose of this paper to repeat the details of clinical care for these conditions, since this is readily available elsewhere (Royal Navy 1972, Bennett & Elliott 1975). More important, perhaps, is a brief consideration of the possible reduction of these occupational hazards of diving.

It is worth re-emphasizing at this stage that many of the hazards to which divers are exposed are ones over which the medical practitioner and physiologist have little influence. Responsibility for the prevention of diving accidents also belongs to the diver himself and those who have trained him. The engineer is responsible for the design and functioning of the equipment upon which the life of the diver depends. Many accidents are a result of the interaction of several contributory factors, of which one might be bad seamanship, such as diving in too strong a tide; another might be bad engineering design of breathing apparatus, which permits a rapid build up of carbon dioxide when the diver is working particularly hard; another might be medical in that the diver is unfit to cope with this physical and mental stress; and a fourth might be inadequate training in that the diver might not know how to cope with such circumstances. In a similar manner the prevention of diving accidents requires close collaboration between the diver, his supervisor and his trainer, the design engineer,

the physiologist and the medical practitioner. It would seem that there are several critical places in the chain of events leading up to a diving accident at which members of this team could act. A diving accident does not necessarily begin at the site of diving operations and the reduction of the occupational hazards to divers should begin in the laboratory and training establishments ashore.

Once again confining ourselves to the medical and physiological aspects of this subject it is possible to consider what is being done to reduce these hazards.

The introduction of a statutory medical examination for divers in British waters has been coupled with the introduction of a category of medical practitioners who are 'approved' to conduct these examinations by the Director of Medical Services of the Health and Safety Executive on behalf of the Secretary of State for Energy. These practitioners should have attended a suitable course of instruction in underwater medicine and, for practical purposes, this means the Underwater Medicine Course at the Institute of Naval Medicine which is now fully booked into early 1977.

The medical examination of divers conducted by these 'approved' doctors is based upon the medical examination which has been used for the deep divers of the Royal Navy for many years. While one of the functions of the examination is to ensure that the diver is fully fit to cope with the physical demands of his work, another is to exclude any condition which might augment the hazards to these divers of his environment. One example is the exclusion as far as possible by history, examination, X-ray and pulmonary function tests of those conditions which might impede the venting of expanding alveolar gases during decompression.

The annual medical examination also includes a radiological survey of the bones and joints to detect the presymptomatic lesions of bone necrosis. Although the detection of a juxta-articular radiological lesion is indicative of a potentially disabling condition and will lead to the diver being declared unfit for further diving, this action is too late to be considered as a preventive measure. For that diver the hazard existed during the dives which he made during the preceding months and years.

One might consider that the place on which to concentrate in our pursuit of less hazardous diving is the site of the dive itself. While this may be true to a certain extent, for instance in the use of on-line monitoring of physiological function during dives of particular hazard, the greatest reduction in the occupational hazard which can still be made is probably in the research laboratory. For example, for bone necrosis one can identify several lines of research, such as: (1) Continued surveillance of the population of divers and collection of the details of their diving histories. (2) Statistical analysis of such data to look for associations which might provide clues about etiology. (3) Evaluation of bone scans and other possible methods of earlier diagnosis. (4) The possible production of bone lesions in an animal model. (5) The influence of different possible etiological factors. (6) The pathology of the developing lesion. (7) The management of the pre-symptomatic case, both clinically and in animals.

The other occupational hazards of diving can be reviewed in a similar manner and, indeed, each has been the subject of laboratory research for many years. The majority are very complex research topics which require the use of pressure chamber facilities. Thus the primary evaluation of new decompression tables is necessarily confined to naval and a few commercial diving laboratories in which a 'wet chamber' permits the subjects of the dive to swim and exercise in the water at raised environmental pressure under the watchful eyes of an experienced supervising staff and medical officer. Nevertheless, the evaluation of the results of such diving trials is not easy. The assessment of the success or failure of diving tables is based upon the incidence of decompression sickness occurring in the selected test dive. Unfortunately, the onset of decompression sickness is not a clearly defined criterion, but depends upon the pain or symptom threshold of the diver and upon the assessment of the doctor who is informed of that manifestation. The number of times that recompression has been required during a series of diving trials is thus a subjective measurement. The use of ultrasound to detect the presence of free gas as bubbles in the ciculation is no better. First, the interpretation of the sounds detected by such apparatus is also subjective, and also it seems that intravascular bubbles are present in the circulation of the apparently normal diver following decompression and do not necessarily indicate the later onset of acute illness. Since a dive to the greater depths may require several days of decompression, it is apparent that the evaluation of new diving tables is a lengthy and expensive process.

In a similar manner the problem of loss of consciousness of divers under water is a research project which requires laboratory study. Some on-site investigation of accidents and near-misses may give a better understanding of some possible etiological factors, but the majority of the work must be focused upon the interplay between impaired performance related to the physiological effects of the inspired gases, the design of the underwater breathing apparatus, the effects of

hard physical work upon blood-gas tensions and the role of acute hypothermia. Only after a lengthy examination of the interrelationships between these and other factors will it be possible to return to the site of diving operations with a diver-monitoring system that may provide on-line information capable of being interpreted meaningfully in the prevention of such accidents.

The extensive research programmes at present being conducted in this country and abroad are, of course, useless if the results are not communicated to those who need them – the divers, the diving supervisors and the training schools for divers. Publication in a scientific journal is not enough, a proportion of diving accidents might not have occurred had those organizing the dive been aware of relevant information which already exists.

Thus the occupational hazards to divers are numerous and there will always be some that will remain unavoidable, but much effort is still required at national and international level not only to coordinate current research into these extensive problems but to provide an appropriate centre for the collection, retrieval and dissemination of the acquired facts.

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