ASPECTS OF UNDERWATER MEDICINE

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Two of the most interesting theoretical aspects of diving or working at increased ambient pressures are inert-gas narcosis and decompression sickness, both due, in different ways, to the behaviour of the so-called 'inert' gases dissolved in the fluids of the body.

The inert gases dissolve according to the gas laws so that the tissues of the body are in equilibrium with the alveolar gas pressures. At atmospheric pressure the P_{N_2} of the tissues is about 573 mm. Hg. and the total quantity dissolved is approximately 1 litre (B.T.P.).

If the ambient pressure is increased the quantity of nitrogen dissolved and the tissue P_{N_2} will be proportionately increased at equilibrium. However the process of achieving a fresh equilibrium after such an increase in pressure is not instantaneous. The quantity of gas dissolved after some finite time is described by an exponential function which, in this context, is usually defined by its 'half-time'; that is the time taken to reach 50% of the quantity dissolved at equilibrium (or 0.693 times the time constant of the exponential).

Inert-Gas Narcosis

Nitrogen

The degree of impairment due to narcosis has been shown by Barnard, Hempleman and Trotter (1962) to be approximately proportional to the partial pressure of the nitrogen breathed, but symptoms are not normally detected at pressures below 4 ats. abs. It has been recently suggested by Poulton, Catton and Carpenter (1964) that some impairment of performance may be found at 2 ats. abs.; however these findings await confirmation.

The symptoms are variable, but numbness of the lips and cheeks, increased excitability and euphoria are the most common manifestations. At increasing pressures excitement may give way to somnolence, judgement is markedly affected and at 10 ats. abs. most men are incapable of useful work and may even be stuporous. When the subject is decompressed the narcosis is relieved, but some subjects show amnesia for the time spent at pressure.

While there is no accurate estimate of the anaesthetic pressure for nitrogen, two divers became unconscious almost immediately when exposed to air at 31 ats.abs. (Keller, 1964).

Helium

Behnke, Thomson and Motley (1935) were the first to show that nitrogen was the cause of the symptoms described and Behnke and Yarbrough (1939) the first to demonstrate that these symptoms were absent if the nitrogen in the breathing mixture was replaced by helium. This finding had the immediate practical advantage that it allowed diving to be extended beyond 10 ats.abs. (300 feet of sea water). There is however another good reason for abandoning air and this is the toxic effect of oxygen at raised pressures. The accepted safe upper limit for the PO_2 in breathing mixtures is 2 ats.abs., which is reached at a total pressure of about 10 ats.abs. when breathing air. In order to dive deeper therefore, the oxygen percentage in the mixture must be reduced and the nitrogen replaced by some other inert gas.

Of the inert gases available to act as diluents for the essential oxygen, only helium, neon and hydrogen appear to be less narcotic than nitrogen.

Recent work by Bennett (1965) seems to show that neon is intermediate in its narcotic properties between nitrogen and helium. Regarding the position of hydrogen, although oxy-hydrogen mixtures have been used in diving by Zetterstrom (1948) it is not known whether hydrogen is more or less narcotic than helium or even whether it should be regarded as physiologically inert. This lack of information is partly due to the technical difficulties of handling potentially explosive mixtures of hydrogen and oxygen.

From this brief outline therefore it should be clear why oxy-helium mixtures are used in deep-diving and, also that, if no superior gas can be found the limits to which one can dive will depend upon the depth at which helium narcosis appears.

Recent observations at the Royal Naval Physiological Laboratory during the course of dives between 10 and 25 ats.abs. suggest that oxy-helium mixtures produce effects which are first detected at about 16 ats.abs.

The most noticeable sign is a coarse tremor, particularly of the hands, which tends to improve after about an hour. The divers may also report feeling weak, clumsy, nauseated or dizzy; feelings which also improve with time. Since these effects are different from those of nitrogen, both in their nature and in their behaviour with time, it is probable that they are due to a combination of factors. It seems likely however that at least part of the effect is due to the narcotic action of helium.

The precise mechanism by which inert gases produce these narcotic effects is not known but Miller, Paton and Smith (1965) show evidence which favours the 'lipid solubility' or 'non-aqueous phase' theories rather than 'aqueous phase' theories; and the evidence assembled by Bennett (1965) points to the synapses within the central nervous system as the sites of action.

Decompression Sickness (Dysbarism)

The term decompression sickness includes caisson disease, diver's 'bends' and similar conditions which occur in aviators. While the aetiology remains obscure, a fall in ambient pressure, i.e. a decompression, invariably precedes the onset; it is therefore an occupational disease almost entirely restricted to men of working age and unknown before the invention of the diving suit and the caisson in the first half of the nineteenth century.

The time of onset is variable; even in severe cases there seems to be a short latent period of the order of five minutes. In divers and caisson workers most symptoms come on within two hours following decompression; but in deep-diving numerous cases occur during decompression, (Barnard, 1966) and the onset may be delayed for many hours in caisson workers. The situation in aviators is slightly different since symptoms are usually caused by loss of cabin pressure when at altitude and the time of onset is related to the length of time for which oxygen has been breathed prior to decompression. A further point of difference is that symptoms in aviators are caused by decompression of the normal amount of nitrogen dissolved in the body at 1 ats.abs.; whereas in divers and caisson workers symptoms are caused by decompression of the greatly increased amounts of gas dissolved at raised pressures.

In spite of these differences it is possible to catalogue the types of decompression sickness without referring to the type of exposure which caused them. The classification of decompression sickness is however most unsatisfactory and that of Golding, Griffiths, Hempleman, Paton and Walder (1960) into two types, Type I cases being cases of 'bend' pain and Type II cases, all others, is no advance on the present odd collection of traditional names.

Minor Cases

This group is understandably the most difficult to diagnose, symptoms such as general malaise or sleepiness are common after diving and mild pyrexia may also occur. More definite however are itching of the skin, 'itches,' particularly of the exposed areas of the face and the backs of the hands; and mild aches and pains, 'niggles' varying in intensity and site but gradually improving. Apart from the signs due to lesions of the nervous system the only other signs are restricted to the skin. The commonest of these called 'marbling' frequently appears as florid red, white and blue marking on the skin. These markings seem to be due to dilated stagnant veins with large areas of arteriolar flare and small areas of contracted capillaries. That capillary damage occurs is shown by the presence of small petechial haemorrhage after the 'marbling' has faded.

A rare sign, which has occurred twice in the same individual, is induration of the skin. It occurred during an attack of a more severe type of decompression sickness and was fully developed within five minutes. The loose skin of the face and of the upper chest and back became swollen, firm, tender and hot. No crepitus could be felt, and the induration, which appeared to be due to an inflammatory reaction, gradually disappeared in the course of about ten days.

Local oedema may sometimes be seen in a limb which is the site of 'bend'-pain, but this usually disappears when the 'bend' is treated.

'Bends': The commonest symptom in divers and caisson workers is pain in the limbs. These pains vary from mild persistent aches to intense sharp pains causing sweating and distress. The pain is frequently localised in or near to a joint, usually to an area including the whole joint, but sometimes to a precise site such as the insertion of the ligamentum patellae.

Characteristically the pain increases in intensity from the time of onset and may be associated with transient pains in other sites. The intensity of the pain increases with exercise, it cannot be 'worked off,' although some relief may be obtained from a hot bath or from local pressure.

Abdominal pain (of a similar nature) also occurs and must be distinguished from the effects of expanded gas within the lumen of the gut, which gives rise to 'colicky' pain.

Cases of 'bends' rarely show any abnormality on physical examination, the diagnosis usually being made on the history alone; however one may find reduced tendon jerks on the affected side or a raised skin temperature over a painful joint.

'Chokes': This is characterised by substernal discomfort or pain and difficulty in taking a deep

breath. The attack is frequently 'triggered' by the first cigarette after a dive and consists of a paroxysm of coughing, the subject perhaps becoming cyanosed and needing urgent treatment. Cases of 'chokes' seem particularly liable to lead to shock especially if treatment is inadequate.

Nervous Disorders

Almost any type of nervous disorder seems to present at some time or another as a manifestation of decompression sickness.

Sensory symptoms are perhaps most frequent, patches of paraesthesia or anaesthesia, feelings of warmth or burning sensations and disorders of proprioception may be seen both together or separately. There is a tendency for sensory symptoms to precede any motor loss, but isolated lower motor neurone type paralysis may come on without any alteration of sensation.

The distribution or extent of sensory symptoms is frequently difficult to correlate with any single lesion which could be postulated on the basis of the anatomical pathways, for instance, anaesthesia of the tips of all digits on both hands has been observed. This may of course be due to multiple lesions, which might be expected if the aetiological agent is gas-bubbles, but the distribution of such bubbles in a symmetrical manner is just as difficult to explain.

Hemiplegia and paraplegia are the most common severe neurological involvements but quadriplegia has also been reported. These paralyses show a peculiarity which may be of some value in differential diagnosis, in that recovery is usually much faster and more complete than is usual in other conditions. Many divers have made almost complete functional recovery following an incident which at the time might have been indistinguishable from complete transection of the cord.

Due to the protean nature of decompression sickness and the way in which symptoms may resolve without treatment, there may be real difficulty in distinguishing between cases of multiple sclerosis and decompression sickness, even if the history of diving is known. Diagnosis can be made if such patients are given a therapeutic test of response to pressure. The pressure must be adequate i.e. at least 6 ats.abs., the time at pressure needs to be at least two hours and the only conclusive result of the test is relief. This test is more likely to give information if it is used early, preferably within 24 hours of onset, but it is still worth trying after several days. Failure to respond may be due to the irreversibility of damage.

'*Cerebral Bends*': These are not common, but may present either as sudden loss of consciousness, perhaps followed by some degree of paralysis or as an alteration in mood, behaviour or personality.

The onset is normally soon enough after the decompression to make the diagnosis obvious.

'*Staggers*': Cases occur in which the labyrinth is effected, sometimes associated with a cochlear lesion on the same side. The patient presents with vertigo, nausea or vomiting, unsteadiness of gait and 'Rombergism' and there may also be some deafness. If treated soon enough recovery is complete.

Visual Disturbance: The development of temporary scotomata has been reported, particularly in aviators, by Flinn and Womack (1963). Such complaints are rare in divers, who however seem to get mild visual disturbances, similar to 'fortification spectra,' quite frequently. It is possible that raised oxygen partial pressures are responsible for these symptoms.

Avascular Necrosis of Bone

Although this disorder has been recognised as a risk of caisson working or tunnelling for many years it is only recently that large-scale surveys have been undertaken, notably by the Medical Research Council, in order to discover its incidence. The lesions are usually discovered on radiography and occur in the long bones particularly in the upper end of the humerus, femur and tibia in descending order of frequency. The lesions may be either in the shaft or in the head of the bone and they are frequently bilateral. They consist of discrete areas of necrosis surrounded by new bone. Lesions of the shaft may be asymptomatic while those of the head may present with secondary arthritic changes, or following collapse of the joint surface.

Post-Decompression Shock

This condition is more common in aviators but is sometimes seen in divers or tunnel workers. The likelihood of its occurrence seems to be related to the adequacy of treatment. It occurs in aviators after they have been recompressed back to atmosphere and should be treated by further recompression to greater pressures, while in divers or tunnel workers it is usually seen following unsuccessful attempts at treatment. Cases of post-decompression shock may occur in men with 'bends' treated with morphine or similar alkaloids, which although they relieve the pain do not prevent further damage. In early cases the pulse rate may be normal or slightly raised and the blood pressure within normal limits while sitting or lying. However there is usually marked postural hypotension so that the patient is unable to stand, but may feel normal when lying down. These symptoms seem to be due to gross haemoconcentration following fluid loss into the tissues and treatment is by recompression in the early stages or by a combination of recompression and intravenous infusion of plasma in more severe cases.

Incidence

Decompression sickness is, in theory at least, completely preventible. However, cases do occur and these supply the only information from which one can assess the degree of failure of a particular decompression procedure.

The current practice is to calculate crude incidence figures, defined as the ratio of the cases observed to the total number of exposures. This figure is of course quite satisfactory for an employer who needs to know how many man-hours are being lost due to decompression sickness, but it conceals relationships which might lead to a better understanding of the problem and a consequent improvement in the procedure.

Tunnel working in Great Britain produces an incidence of only about 1%, remarkably low in comparison with that in experimental diving, but this figure only represents those men who report for treatment. If the results of experimental diving are in any way comparable, then of all the men who have signs or symptoms, only about one third ask for or receive treatment. To pretend that these cases which are ignored represent successes is incorrect as is perhaps most clearly shown by recent studies of avascular necrosis of bone (M.R.C. 1966).

Even if revised regulations for compressed air workers completely abolished all cases of 'bend' pain and yet produced avascular necrosis in 10% of the men at risk they would still be inadequate.

In calculating the incidence of decompression sickness one must therefore include all manifestations of decompression sickness, as far as they can be discovered. However, even if all cases could be recorded there is a further complication due to the factor of acclimatisation (Paton and Walder, 1954). The effect of acclimatisation is seen as a greater susceptibility to 'bends' in men when they are first introduced into compressed air compared with those who have been exposed repeatedly. Apart from other factors the effect of introducing new men will be to produce a fluctuating incidence of which the crude incidence represents the mean.

Since the pressure to which men are exposed varies throughout the construction of a tunnel, the decompression routine, which is determined by the maximum working pressure of each shift may alter from shift to shift. Now there is no certainty that the decompression routines for different pressures are equally safe, they tend in fact to become less adequate at pressures above 35 p.s.i.

Since in practice it is not possible to ensure that all men remain acclimatised, in order to have any clear idea of the effectiveness of a decompression procedure we must know the incidence of decompression sickness, for the particular routine in question, in unacclimatised men. This is the only

index which avoids the interference of factors other than individual differences in susceptibility.

Individual Susceptibility

The simplest situation in which to compare the susceptibility of individuals is in so-called 'no-stop' dives, which are dives followed by direct ascent to the surface. For dives deeper than about 33 feet of sea water (2 ats.abs.) there is an exposure time beyond which direct ascent is no longer possible with safety, and the maximum exposure at each depth which just allows direct ascent may be plotted as the no-stop curve (Fig. 1).

In all longer exposures ascent can be made to a point short of the surface but the decompression must be prolonged to avoid decompression sickness. These prolonged decompressions may consist of a series of 'stops' as in most diving schedules, or of a more or less continuous reduction of pressure as in the decompression procedures of tunnel workers.

The most detailed no-stop curve available is that produced by Eaton and Hempleman (1962) using goats as subjects (Fig. 2). This curve, although very similar to that for men, shows several curious features. The first of these is that there is a difference between the most susceptible and the most resistant animal of about 30 feet for both short deep dives and long shallow dives. The second is that an animal may alter its relative position within the sample, being more resistant at one end of the curve and more susceptible at the other. Thus the individual susceptibility of an animal is related to the type of exposure, but the variation in susceptibility between animals is constant and independent of the type of exposure.

Obviously if these findings can be applied to men they have a practical effect upon any selection procedure; since only exposures similar to working conditions are likely to produce comparable

FIG 1.—No-stop curve for men. After Albano (1961). The points plotted represent the threshold exposures for the production of decompression sickness when breathing air.

FIG. 2.—'No-stop' curve for goats. After Eaton & Hempleman (1962). The incidence of decompression sickness is shown for air exposures between 15 minutes and six hours.

results. This is particularly difficult in divers who need to work throughout a large range of depths and times and means in effect that there are no satisfactory ways of selecting those who might be more resistant to decompression sickness.

A further point arises from these experiments since it was found that for a constant time, increasing the depth of exposure led to a markedly skew distribution of individual susceptibility, most individuals being found near the lower limit as shown by the position of the 50% incidence. While it is possible to convert this into a more 'normal' distribution by plotting the incidence against the logarithm of the pressure, this is of little help in understanding the underlying causes of the situation.

One possible explanation is that the distribution only appears skew due to the suppression of sub-clinical or mild cases, which are difficult to diagnose in goats. Some support for this view is evidenced by the way in which an increase in the severity of exposure tends to alter the type of decompression sickness found in men, from mild itching or fleeting aches and pains towards true 'bends.' If only 'bends' were considered as a true end-point then all the premonitory symptoms would be suppressed in the way suggested.

FIG. 3.—Saturation curve for air derived from the no-stop curve for men.

Saturation

There are two common ways of talking about the uptake of gas, the first, describing the rate of uptake by the 'half-time,' has already been mentioned; the second in general use is to describe the quantity in excess of normal as a percentage of the excess gas dissolved at equilibrium, frequently called saturation.

The obsession with the time taken to 'saturate' which is found in diving literature derives from a preoccupation with the no-stop curve and its possible interpretations, particularly with the first assumption in attempts to derive a 'saturation curve' from the meagre data.

Inspection of a more complete curve shows that it appears to be asymptotic and that a dive to 33 feet may apparently be prolonged indefinitely without any increase in risk. The basic assumption is that this particular dive represents the condition of saturation and hence that all shorter dives on the curve represent different degrees of partial saturation.

To calculate a saturation curve from these data a further assumption must be made, namely that the onset of decompression sickness is caused by the presence of the same number of gas molecules in every case. Without knowing what this number is we can express it as the quantity of excess gas dissolved after a saturation dive to 33 feet, or more simply as 33 footsworth. The percentage saturation is then given by the simple expression:

$$\% \text{ Sat}^n = \frac{33 \times 100}{\text{Depth in feet}} \quad (\text{Fig. 3})$$

This is an extremely roundabout way of deriving an uptake curve for inert gas: it is obviously simpler to measure the uptake and elimination directly. However since we are not quite sure how a certain quantity of gas acts to produce a certain degree of risk, we must try to correlate the information given by these two different methods.

The first difficulty in such an attempt is that there seems only to be general agreement by the various authors as to the order of magnitude of the time constants concerned. Lundin (1953) refers to 'the conflicting results obtained by different investigators using the nitrogen elimination during oxygen breathing as a means to establish the role of nitrogen in producing decompression sickness.'

The problem seems to arise from the fact that any desaturation curve is not a simple exponential but can be represented as the sum of several exponentials. It is usual to analyse an experimentally produced curve by semilogarithmic analysis into two or more exponentials with different time constants. In spite of the fact that some authors, notably Jones (1951), analyse the elimination curve into four or five components, it seems that the accuracy of the measurements and the large errors involved in lung-rinsing, make an analysis into more than two or three components of limited value.

If however one takes as a crude measure of such a curve the half-time for the total curve, then for nitrogen elimination this time is between 60 minutes and 100 minutes, whereas the saturation curve, obtained from a very limited number of values, gives a half-time of about one hour. This is perhaps as good agreement as one could expect from the data available. What is of perhaps more interest currently however is the rate of uptake and elimination of helium.

Behnke and Willmon (1941) gave some measurements which showed that helium was eliminated much more quickly than nitrogen, the crude half-time lying between 15 and 25 minutes for men at rest. While recent experiments at the Royal Naval Physiological Laboratory would predict a similarly rapid rate of uptake by inference from no-stop dives, there are some findings of Duffner, Snyder and Smith (1959) which are in direct contradiction, since they imply that helium and nitrogen are exchanged at the same rate.

It seems that this situation is unlikely to be resolved until further work on the uptake and elimination of inert gases give more reliable information.

Treatment

There is still no satisfactory basis on which to calculate decompression procedures whether for normal use or for use as treatment tables when decompression sickness develops. Even the pre-

ceding discussion on the uptake of inert gas does no more than attempt to put into figures what Paul Bert (1878) put in words, that decompression sickness is caused by the presence of inert gas, which it is thought produces bubbles of free gas if the decompression is too severe. A discussion of bubbles has purposely been omitted since the evidence is no more precise than the original suggestion. However the greatest argument in favour of bubbles as the aetiological agent is the response to pressure of cases of decompression sickness.

The general treatment for all cases of decompression sickness is recompression. The decompression schedules used after taking the patient to pressure are nearly all modifications of those proposed by Van der Aue, White, Hayter, Brinton, Kellar and Behnke (1945). These involve returning the patient to an arbitrary pressure of 4 or 6 ats.abs. according to the type of case. A second method, which is in many ways preferable, is to return the patient to the pressure of relief. This method was in use for many years and is still used in tunnel workers; it has also been used with success for men developing symptoms, at greater pressures than 6 ats.abs. for which no ready-made procedures exist. Implicit in this method is that a pressure of relief can always be found, that is to say pressure cures, and as a result of the accumulated experience of the last century it can be said that this is so, but that delay in starting treatment or inadequate recompressions are the commonest causes of failure.

Instances of death due to decompression sickness still arise; there is therefore a need for improved treatment, but if we knew enough to make treatment completely effective we should have solved the problems which would make prevention certain.

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