COMMISSION OF THE EUROPEAN COMMUNITIES

MINES SAFETY AND HEALTH COMMISSION

CONGRESS

organised in association with the

EUROPEAN UNDERSEA BIOMEDICAL SOCIETY

and the

MEDICAL COMMITTEE OF THE EUROPEAN DIVING TECHNOLOGY COMMITTEE

on

MEDICAL ASPECTS OF DIVING ACCIDENTS

12/13 october 1978

LUXEMBOURG/KIRCHBERG - Jean Monnet Building - Avenue de Gaspéri

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CONTENTS

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LOSS OF CONSCIOUSNESS IN DIVERS - A SURVEY AND REVIEW

C.M.Childs

The altered conditions to which man is subjected during diving are not yet fully understood, and factors still exist which threaten the lives of divers. The signs and symptoms of decompression sickness, barotrauma, gas narcosis and toxicity can be recognised, and widely accepted forms of treatment meet with a great deal of success. Throughout the history of diving, however, and within every diver's repertoire, are stories of incidents whose causes are not obvious, and any successful treatment of many has been by luck rather than clinical judgement. In some such incidents consciousness is lost, in others unexpected signs or symptoms develop.

Loss of consciousness in divers is the end-point reached due to the operation of factors which, in other circumstances, cause only unexpected symptoms and difficulties. Evidence for the nature of these factors may thus be found by studying all problems which occur during diving.

The factors are not necessarily mysterious or unknown. Some of their effects in the altered environment of diving are known and some have already been implicated as the causes of difficulties during diving. The picture is not yet clear and this presentation will review present knowledge and hypotheses about factors which may be responsible for loss of consciousness underwater.

EXTERNAL FACTORS

1. Equipment Performance

Personal diving equipment, including gas bottles, back-packs, supply lines, valves and regulators has undergone close scrutiny with extension of the limits of diving. We still lack, however, much basic information necessary for equipment design even for diving within modest limits, and cannot set the physiological criteria for

design. In particular we require further information about peak oxygen requirements and the volumes of gas required by a diver working to perhaps supramaximal levels. We have some information about the effects of external breathing resistance on respiratory function but it is not certain that this yet allows satisfactory design of equipment for the extreme demands of operational use.

It has been suggested that, at low temperature and under pressure, the increased density of carbon dioxide may cause layering of the gas in the lower part of a diving bell. Entry of a diver into the bell through the bottom hatch would put him at risk as he breathes a relatively high partial pressure of carbon dioxide. The efficiency of scrubbers, their positioning within the bell, and gas movements in the bell are of critical importance in this potential hazard.

2. Breathing Gas Contaminants

The provision of clean air for SCUBA divers is straightforward, but instances of contamination, usually with hydrocarbons, still occur (Daenans, 1973). With pre-mixed gases for deeper diving, quality control should ensure purity but additional problems, such as residual freon or other cleaning agents in supply systems, still occur. We have now investigated four cases from the North Sea in the last three years where contamination has been suspected as the cause of unexplained incidents, but were unable to detect contaminants even by spectroscopy. A problem with deep diving is that even minute amounts of a contaminant become progressively more dangerous as depth, and thus the surface equivalence of the contaminant, become greater.

INTERNAL FACTORS

1. Diver Performance

This section includes principally the psychological effects of immersion, pressure, cold and stress and the likelihood of such mechanisms leading directly to diving accidents. Psychological factors may also lead indirectly to accidents by causing loss of perception, inattention and exaggerated stress responses. Some workers believe that psychologically unsuited candidates may be detected early in training, and have coupled this with descriptions of optimal physical characteristics for divers.

There is no doubt that diving can be very stressful both physically and psychologically. The physical demands may be met either by exclusion or by physical training. The psychological effects of diving, whether from immersion, confinement, cold or stress vary greatly between individuals; psychological responses are in many cases adaptive, improving rather than impairing performance. In some situations and with some individuals, however, psychological responses may become detrimental. It is possible that susceptible individuals may be identified but it may be equally valuable to identify the potentially pathological psychological effects which may occur during diving as it is these which may lead to accidents.

Diver Selection. Inherent in all diver selection tests is the assumption that suitable individuals conform more or less closely to a stereotype. Although this may be true it has been found impossible to define this stereotype, as physical, medical and psychological tests shows that divers do not differ significantly from the average for the population, apart from being rather fitter (Flemming, 1972).

Successful analysis of biography and physical fitness has been reported in relation to training for underwater demolition (Gunderson, 1972), but this prediction of training success largely excluded any assessment of psychological suitability. Definite and positive relationships have been shown between increasing age and

weight and the likelihood of decompression sickness (West, 1973) but the relationship was low and of limited value in prediction of problems. It must be concluded that, apart from the elimination from diving of individuals with some chronic illnesses, lack of stamina or physical fitness, and some types of pulmonary pathology, we cannot yet hope to improve diving safety by physical criteria which differ from those already applied. The psychological criteria are also difficult to define and an extensive study (Deppe, 1971), which attempted to predict success on the basis of diving skill, intellectual ability, task-orientation and emotional maturity, was unrewarding.

Defects in physical health and fitness could result in loss of consciousness by mechanisms to be described later. Psychological unsuitability or disturbance will not, of themselves, cause loss of consciousness; their effects are indirect, allowing other dangers to take effect because of inattention or distraction. It is therefore important that the effects of diving on psychological performances are appreciated.

Performance Changes During Diving. Much of this research has been done under simulated conditions, yielding valuable but limited results. There is no indication that oxygen toxicity affects performance until the actual convulsive stage is reached. The rate of onset of nitrogen narcosis is uncertain but its effects are probably insignificant at depths of less than 30 metres; the narcotic effects of oxy-helium mixtures have probably not yet been detected in man. Narcosis is more readily detected during complex tasks and it appears that the narcotic effect of a breathing mixture at pressure interacts with other stresses to produce a conbine performance decrement; the crucial variable seems to be the level of anxiety of the subject (Baddeley, 1971).

Visual performance, in terms of acuity, colour and visual field is reduced by physical factors underwater. It seems that attentional narrowing of vision limits the efficiency of peripheral vision (Baddeley, 1971). Loss of position sense and orientation occur due to lack of sensory information from proprioceptive and body surface pressure receptors. Combined with the visual errors, this leads to misinterpretation of the vertical, of distance, size, weight perception, orientation and movement.

In more general psychological terms there is evidence that danger and anxiety increase a diver's arousal level and influence performance by producing a narrowing of attention (Baddeley, 1972). While this may benefit a diver in trouble by concentrating his attention, it may seriously jeopardise his situation by making him less aware of peripheral dangers and threats. Cognitive function, including memory, reasoning and vigilance, may be affected during diving by perceptual disturbance, anxiety or attention narrowing, but there is evidence that the cognitive functions are not normally significantly depressed (Egstrom, 1973; Baddeley, 1975). Manual dexterity, however, is consistently reduced during diving, due partly to the same adverse factors but chiefly to the restrictions of diving dress and cold.

The less dramatic but nevertheless important, psychological effects of diving should be largely overcome or prepared for by training and experience. In two surveys of skin and SCUBA diving accidents (Schenk, 1972a; Schenk, 1972b) it was shown that 20% of SCUBA victims were on their first dive or first open water dive, and that the 16-25 year age group contained more than half the victims. In a study we carried out into non-fatal diving accidents more than half the divers were aged less than 25 years.

2. Direct Effects of Hydrostatic Pressure

The specific effects of increased hydrostatic pressure on man during diving are inevitably difficult to isolate from the effects due to, for example, gas narcosis, hypoxia, hyperoxia and hypercapnia. Despite much searching, only a few phenomena attributable to the direct effects of hydrostatic pressure have been observed, and at present it appears that the high pressure nervous syndrome is the most significant threat to diver safety directly attributable to pressure.

After initial suggestions that the high pressure nervous syndrome was due to the effects of helium it is now fairly well accepted that it is due to direct hydrostatic pressure effects on cell membranes. The high pressure nervous syndrome should not pose a threat to the safety of divers - all the effects known so far are reduced by slow compression and are reversible (Fructus, 1968). The high pressure nervous syndrome is not a problem until around 300 metres with a fast compression rate of 2 metres per minute (Fructus, 1971). At great depths other direct effects of pressure have been observed including on cell division (Marsland, 1968), bacterial reproduction (Albright, 1971), nerve function (Akers, 1973; Lemaire, 1973), and the effects of noradrenaline. Chemical equilibria and reaction rates, phase changes and effects at the receptor sites of many other chemical transmitters and hormones can be predicted, according to thermodynamic theory, to be progressively altered as pressure increases; even the volumes of amino-acid molecules are changed (Yayanos, 1971).

While it seems a long way from the molecular effects of pressure to loss of consciousness in divers, these effects are important. They are directly related to pressure increase and, being fundamental, affect every physiological mechanism in the body. It may be that minute disturbances of physiological functions such as neural and neuro-muscular transmission, metabolic reactions and rates, and hormonal responses become cumulatively significant even at modest depths.

3. Cold

Loss of manual dexterity due to cold accounts for the greater part of performance decrement, but with falling core temperature, attention narrowing may further impair performance to the point at which safety is threatened. Ultimately, if the core temperature continues to fall, the subject becomes comatose and dies, usually in a cardiac arrhythmia. A diver will initially lose fine manipulative hand movements, and performance of his task may suffer because of this and because of distraction due to discomfort. Distraction may cause him to ignore threats to his safety underwater and, finally realising he is in danger, he may be in further difficulty because of loss of power and dexterity in his hands. Recent work in Aberdeen (Auld, 1977) has suggested that cold may have other, perhaps more sinister, effects. It has been shown that the oxygen requirements of maximal shivering due to cold in dogs cause an increase in oxygen uptake of three times, and a threefold increase in cardiac output (Auld, 1977). Experiments on haemodilution in dogs (Norman, 1974) have shown, however, that high output cardiac failure follows such a sustained increase in cardiac output. The possibility exists that maximal thermal shivering may require an increase in cardiac output that can only just be met by a fit myocardium; if further stresses are applied, for example by frantic swimming efforts, or if the myocardium is unfit for any reason, high output cardiac failure may follow.

Two other facets of cold physiology have been reported which may have a bearing on diving. In rats it has been suggested (Pepelko, 1973) that hypercapnia may eliminate non-shivering thermogenesis, although this only occurred during inspiration of 10% carbon dioxide or greater. In another series of experiments it was demonstrated that cardiac activity was reduced in hamsters when exposed simultaneously to hypoxia and cold (Anderson, 1973). If substantiated in man these effects of high inspired carbon dioxide and hypoxia might be dangerous during

diving but there is no evidence at present that their effects are significant.

Clearly the problems of cold during diving could be avoided by prevention. Hot water diving suits are satisfactory although not perfect, but manual dexterity is still often impaired because of reluctance to wear gloves. Insufficient development of breathing gas heaters is one reason why they are not in widespread use, although it has been suggested that when respiratory heat loss exceeds 350 watts the diver is in danger because, even by wearing a heated suit, he is still losing more heat than he can generate or absorb. Due to malfunction, abuse or non-use of heating systems, divers still get cold, and sometimes into danger as the result. The detrimental and thus dangerous effects of mild hypothermia during diving are known, and there is circumstantial evidence that cold predisposes to decompression sickness, nitrogen narcosis and oxygen toxicity (Strauss, 1971).

4. "Diving Reflex", Cardiovascular Responses and Work Underwater

The most commonly reported cardiovascular response associated with diving is the "diving reflex". Bradycardia, manifest by a fall in heart rate immediately after face immersion, constitutes this reflex (Bove, 1973). In some subjects a second decline in heart rate is reported after about 140 seconds of breath-holding, continuing until breathing resumes and supposedly due to hypoxia developing during prolonged apnoea. It has been suggested that the bradycardia, also seen in diving mammals, is in man an atavistic reflex to conserve oxygen (Landsberg, 1972). Other cardiac arrhythmias have been reported during diving, but less consistently.

The magnitude of the reduction in heart rate is reported variously between 15% at atmospheric pressure (Hong, 1971) and 40% at 200 feet breathing air (Pilmanis, 1971). The bradycardia lasts for the apnoeic period or becomes more marked (Bove, 1973), but recovers within 50 seconds of the resumption of breathing (Pilmanis, 1971).

The mechanism of the response is not yet certain; there may be an element due to apnoea alone, but face immersion potentiates the bradycardia, especially in cold water (Moore, 1971). The magnitude of the reduction in heart rate may be reduced by prior oxygen breathing, and it is suggested that peripheral chemoreceptors may exercise some control (Elsner, 1971). Other workers (Dubois, 1971) suggest that the bradycardia is mediated only by cooling of the face and have demonstrated that it is not affected by arterial oxygen or carbon dioxide tensions, lung volume or intrathoracic pressure. An alternative suggestion is that the reflex is due to stimulation of normal sensory nerves in the face and nasopharynx, effective stimuli arising from cold, touch or even pressure. The possibility that pressure may initiate this bradycardia is interesting as, during diving, there is often a pressure differential between the face and the rest of the head because of failure of pressure equalisation in the face-mask.

In experiments, not underwater, to determine other cardiovascular changes during breath-holding (Hong, 1971), slight increases were demonstrated in cardiac output, stroke volume and arterial blood pressure, and it may be that these mitigate to some extent the loss in tissue perfusion due to the fall in heart rate.

To determine the combined effects of oxy-helium breathing, experiments were carried out by a group of workers (Hong, 1973) who showed that the bradycardia of face immersion was exaggerated after prior oxy-helium breathing. They could show no effect on the resting heart rate without face immersion but experiments with rats (Lin, 1973) have suggested that oxy-helium causes a bradycardia by a direct cardiovascular effect of helium. This effect requires further study as do the direct cardiovascular effects of pressure. A consistent bradycardia has been reported (Ardnashnikova, 1973) during a seven day saturation dive at 5 atmospheres breathing nitrox. In other experiments an increase in pressure, breathing

oxy-helium, caused progressive bradycardia in exercising subjects, as did increasing gas density at constant pressure (Flynn, 1972). It has been suggested that these effects are due respectively to a depressant effect of helium and a reflex change in heart rate because of increasing airway resistance. It may be, therefore, that increased gas density, helium and pressure itself have direct, additive cardiovascular effects. They all seem to induce a bradycardia although other cardiac arrhythmias have also been reported (Landsberg, 1972; Sem-Jacobsen, 1972).

Accomplishing physical tasks underwater may require the expenditure of considerably more energy than on the surface because of the constraints of clothing and equipment, water currents, weightlessness and the loss of leverage and a fixed working position. Experimental attempts to determine alterations in cardiovascular performance during exercise underwater have so far failed to discover any major discrepancies from normal except in the effect on the bradycardia due to increased gas density, helium or immersion. Heart rate under these circumstances fails to respond normally to the stimulus of exercise (Strome, 1971; Bergman, 1972; Flynn, 1972; Paulev, 1972). Some workers (Denison, 1972) conclude that horizontal subjects, breathing at eupnoeic pressures and working against mild and moderate loads in warm water show the same responses to this exercise in air. Others (Bergonzi, 1973) have used subjects exercising in dry chambers and shown no adverse cardiovascular effects during exercise down to 565 metres conceding, however, that increased gas density probably limits performance below this depth.

Such experiments, however, do not reflect the true demands of an open water working dive in cold water. It has not yet been shown that the direct cardiovascular effects of immersion, gas density, helium and pressure do not combine into a significant threatening influence on cardiovascular function.

5. Internal Homeostasis - Biochemical and Haematological Phenomena During Diving

Alterations have been observed during diving in the homeostasis of many biochemical, haematological and acid-base systems. Loss of body water, and plasma volume, rises in serum sodium, potassium and phosphate with coincident increases in urinary loss of sodium and potassium have been reported during immersion (Boering, 1972, 1972a, 1972b; Sode, 1972). These changes do not seem to be affected by pressure change although slight lowering of serum sodium levels has been reported in oxy-helium saturation diving (Sode, 1972). During the same dive slight decreases were reported in serum glucose, plasma insulin and cortisol levels, perhaps demonstrating a response to the stress of the environment.

The effects on acid-base balance during diving are difficult to assess because of the marked changes caused by alterations in inspired and metabolically produced carbon dioxide. There is, however, no evidence to suggest that, with proper control of breathing gases, acid-base status is adversely affected by diving.

There is no doubt that decompression accidents may lead to massive haematological disturbances. There is growing evidence that even safe decompression may lead to minor changes of the same kind, but no significant haematological disturbances have yet been attributed to pressure itself.

In summary, there is no evidence at present that disturbances in the homeostasis of biochemical, haematological and acid-base systems can result directly in diving accidents.

6. Respiration

In this section I shall discuss the influences on human respiration principally of increased gas density due to increased pressure. With air and other relatively

dense gases, diving encroaches upon the reserve of ventilatory capacity at any depth, and the capacity for physical exertion is progressively reduced beyond a certain depth even with optimal breathing apparatus (Lanphier, 1972). The use of oxy-helium mixtures restores much of this capacity and markedly increases the depth of useful activity but the full extent of this benefit is not yet precisely known.

The importance of increased gas density is that it increases the energy expenditure required for ventilation, and it has been suggested (Varene, 1972) that physical inability to achieve adequate ventilation will cause carbon dioxide accumulation within the body. This concept is confirmed in the finding (Fagraeus, 1973) of decreased ventilation with ambient pressure and gas density increase, and a concomitant rise in $PACO_2$ up to a mean value of 43 mm Hg at 3.0 ATA compared to 32 mm Hg at 1.0 ATA. The same workers point out, however, that compression in air to shallow depths results in moderate hyperoxia enhancing circulatory transport of oxygen in maximal exercise and a consequent increase in the aerobic potential of working muscles.

The detrimental effects of externally imposed mechanical resistance to breathing become even more significant with dense gases. It has been shown (Uhl, 1971) that when both inspiratory and expiratory resistances were added, during exercise and breathing a dense sulphur hexafluoride/oxygen mixture, subjects were stressed to their maximal tolerance. All subjects developed hypoxaemia and hypercapnia.

7. Hypoxia

A particular problem with hypoxia is that, when accompanied by normocapnia, the subject becomes unconscious without warning and without being aware of his danger. Normocapnic, or even hypocapnic, hypoxia can easily occur when a breathing circuit with carbon dioxide absorbent is used, or hypoxic breathing mixture is supplied to an open circuit breathing set.

Acute hypoxia, such as occurs when a diver's gas supply fails, is obvious because he can no longer inspire; this often causes immediate panic and an impulse to remove the mask and mouthpiece. This sequence of events has caused a number of incidents of loss of consciousness and some deaths.

It is clear that the mechanical functions of breathing are impaired during diving by a number of factors. It has been suggested (Chouteau, 1970) that the next stage of respiration, transport of oxygen across the alveolar membrane, is reduced under pressure. It was suggested earlier in this paper that oxygen requirements during strenuous diving while shivering may be very high indeed. Thus, for a number of reasons, even fit divers breathing normoxic mixtures may be close to hypoxia.

8. Hyperoxia

The signs and symptoms of hyperoxia are well recognised and their occurrence in routine diving is exceptional. There is no doubt that accidents have occurred because of C.N.S. effects; there is also no doubt that respiratory problems have occurred, especially during therapeutic tables with high partial pressures of oxygen.

9. Hypercapnia

Adaptation to moderate inspired concentrations of carbon dioxide, from 3 - 4%, occurs in normal men who can tolerate continuous inhalation of 3% inspired carbon dioxide for at least one month and 4% for over a week. Levels of up to 2% in inspired air have not been shown to induce decrement in performance or normal physical activity (Lambertsen, 1971). Tolerance to increased levels of inspired carbon dioxide has been demonstrated by a number of workers (Clark, 1971; Clark, 1973) but they also showed that the tolerable levels are reduced by exercise. It has been shown (Florio, 1973) that divers do not in general show acclimatisation to carbon dioxide but it has been suggested following investigation of tolerance levels in divers that some individuals may possess an inate insensitivity to

moderate levels of hypercapnia. This may put them at risk from its narcotic properties during periods of high CO_2 production or inhalation without the victim realising the problem.

Forced airway closure as the result of excessive hyperventilation has been cited as a paradoxical cause of hypercapnia. Vigorous hyperventilation, whether voluntary or in response to exercise, shifts the airway equal pressure point until it ultimately moves inside the thorax and ventilation becomes inefficient. Carbon dioxide retention will then occur.

10. Inert Gas Narcosis

Nitrogen has long been recognised as a potent anaesthetic gas under pressure. Similar properties have been sought for helium but none found until recently; even those now detected are minor even at great pressure and can only be observed at cellular level.

Nitrogen is thought to exert its anaesthetic effect by altering the properties of cell membranes in the same way as other anaesthetics. Its effect is inevitable, although there is probably some habituation, and it is unpredictable both between individuals and in the same individual at different times. Nitrogen narcosis is known to be affected by interactions with carbon dioxide, hypoxia and hyperoxia, and by temperature (Schmidt, 1973). It is unlikely that nitrogen narcosis as a depth limitation to air or nitrox diving will be overcome, although protective agents have been suggested (Schmidt, 1973). It is a known problem, it has led to accidents, and will continue to do so when proper procedures are not followed.

PREDISPOSING FACTORS

1. Drugs and Alcohol

Drugs such as antihistamines, sedatives and anxiolytics, apart from social drugs

such as alcohol and marijuana, are so freely available and used now that their significance as drugs may be forgotten. These substances, and many others, may affect performace and render a diver liable to accident because of inattention, decreased arousal or just plain tiredness. If their use is allowed during diving the diver must be aware of side-effects, as must his colleagues both underwater and on the surface. There remains the problem of the diver taking drugs without the knowledge of his doctor or his colleagues.

Alcohol is not allowed at most diving sites in the North Sea and there is little evidence that this rule is broken to any extent. There may be problems in the first 24-48 hours following shore leave if a diver has been drinking heavily. Firstly, his attention and vigilance may be impaired and he may be feeling unwell. Secondly, he may be metabolically disturbed with the risk of episodic, dangerously low blood sugar levels due to reduced hepatic gluconeogenesis. A further danger of alcohol may occur with withdrawal after a period of heavy drinking. Some dependance may have developed in the diver and his whole attention may not be focussed on his work underwater.

2. Intercurrent Illness

Acute illnesses, unrelated to occupation and to the environment of diving, are as likely to occur in divers at work as in any other group of workers. Chronic illnesses, such as diabetes and epilepsy, which might cause loss of consciousness, should be detected during medical examination of the prospective diver.

Acute illnesses which can result in sudden loss of consciousness without death are very few. They include sub-arachnoid haemorrhage, post-epileptic coma, simple syncope and possibly meningitis although the onset of this condition is usually less dramatic. Another possible cause of diving accidents, although the condition seldom leads to unconsciousness, is an infective labyrinthitis.

In fatal diving accidents where all other causes have been excluded, acute illness resulting in death must be considered. In the general population 75% of sudden deaths are cardiac in origin, with 75% of these being due to coronary artery disease. Clearly divers are in a generally fit, young, age range, but the possibility of acute coronary insufficiency remains. A case of non-fatal coronary insufficiency in a diver at 110 feet has been reported (Owen, 1971). Other causes of cardiac deaths include congenital abnormality and cardiomyopathy, both of which should be discovered on medical examination and which are rare anyway, and myocarditis. So-called "minimal myocarditis", isolated groups of inflammatory cells scattered through the myocardium, has been reported in some pilots who died suddenly but the significance of the finding is questionable (Stevens, 1970). More significant, but very rare, would be the finding of extensive areas of inflammatory cell infiltration and change in the myocardium following a viral illness. Such a viral myocarditis could conceivably result in a fatal dysrhythmia and death in a diver underwater.

MISHAPS

1. Hyperventilation

The technique of voluntary hyperventilation before breath-hold diving has been reported as the cause of a number of diving accidents (Snively, 1972; Landsberg, 1972). Hyperventilation, carried out to maximise oxygenation of the blood, also lowers carbon dioxide levels so that hypoxia ensues before the carbon dioxide level rises sufficiently to stimulate the need for ventilation.

2. Dysbaric Illnesses

The clinical presentation of the dysbaric illnesses will be familiar to most of the audience who will be aware that sudden loss of consciousness underwater is not a common consequence of either air embolism or decompression sickness.

The effect of a thoracic squeeze has been reported as a cause of diving accidents (Sapov, 1972), but this problem has largely disappeared with changes in technique and equipment. It has been suggested that carotid sinus pressure from a wet suit or other apparel around the neck can impair cerebral blood flow (Shane, 1971). Although not always dysbaric in origin, disturbances in vestibular function have been reported as mechanisms for various syndromes associated with diving. The most likely consequence of disturbed vestibular function is vertigo, but nausea, vomiting, hearing and orientation disorders are also reported (Komordin, 1973). Possible causes of vertigo include caloric stimulation, external, middle or inner ear barotrauma, decompression sickness,abnormal gas pressures in the middle ear causing alternobaric vertigo, and counterdiffusion during changes of breathing and environmental gas composition. Disturbances of vestibular function can be so disabling that, although unconsciousness probably does not result directly, the diver may be in extreme difficulty underwater.

CONCLUSION

Loss of consciousness in divers is not common, but is clearly dangerous. Acute medical conditions which can cause sudden loss of consciousness allowing the diver no time to reach safety or alert his colleagues are very few. Suspect chronic medical conditions should be detected and excluded at pre-employment examination. Dysbaric illnesses might, on occasion, cause sudden loss of consciousness but in general their presentations are less dramatic.

There remain the conditions I have described which result from environmental factors, such as hypoxia, hypercapnia, narcosis and cold, from predisposing factors such as drugs and intercurrent illness, from the diver's physical and psychological performance and from various mishaps.

The physiological and pathological pathways leading to sudden unconsciousness in

man are few. The adverse factors which set a diver on one of these pathways towards unconsciousness are easily lost in the confusion of altered pressure, abnormal partial pressures of respiratory gases, hard physical exertion, cold, and the other environmental changes of diving. Continual critical recording and analysis of diving problems of all types is essential if we are even to approach answers to the questions raised by loss of consciousness underwater.

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RESCUE AND RESUSCITATION OF THE UNCONSCIOUS DIVER

G. ARNOUX

Mr. Chairman, Ladies and Gentlemen,

The exploration and development of oil resources in the North Sea over the past 10 years has created an intense build-up of diving activity unprecedented anywhere in the world.

Unfortunately, the toll of accidents has been correspondingly high. However, this massive concentration of men and activity has permitted studies, and identification of the main areas of hazard in diving operations.

Diving Regulations introduced over the past years have greatly improved the safety factors. However, incidents and accidents still occur. But it is our opinion that series of procedures, properly applied when the emergency arises, will prevent a diving incident developing into a fatal accident.

I must be the only non-medical speaker of the congress, and my point is not to tell Doctors how to treat diver/patients, but rather to highlight the additional specific problems arising in diving emergency situations and subsequent rescue operations.

We shall consider two types of situations -

First: The Rescue of a surface orientated diver from depths up to 50 Metres

and the second: The rescue of a bell diver from depths greater than 50 Metres

A surface orientated diver is directly attended, supplied and assisted from the surface. In case of emergency, he will have to reach the surface for safety. In his attempt, he will be not only exposed to the dangers specific to the water - such as asphyxia or drowning - but also to the dangers related to pressure - such as decompression accidents, barotraumas the worst being lung barotraumas.

One must also remember that the first phase of the rescue will be conducted on the surface by the only people available on the spot, divers/diving superintendents.

It is a common reflex when a surface supplied diver is in distress for all surface attendants to grab his umbilical line and haul him all the way to the surface as quickly as possible.

This type of reaction has been the cause of a certain number of fatalities in the past through lung barotrauma/embolism. On the other hand, somebody in the process of drowning at depth must be helped somehow.

The procedure we have developed is as follows:-

As soon as an emergency arises (in most cases this is realised through the diver/surface communications system),
the tender will start hauling the line in - assuming it is not snagged underwater - and simultaneously the stand-by diver will dive, following the umbilical of the diver in trouble.

This action of pulling the diver up while the stand-by diver is on his way down, will be continued only as long as the diver in trouble is heard to talk, breath or shout over the phone.

If these signs of breathing stop - almost certainly indicating loss of consciousness - then the hauling towards the surface must cease until the stand-by diver has reached the man, forced him to exhale, re-established a breathable supply of gas (through bail-out cylinder, pneumo line, etc.) and given the order to pull up.

The stand-by diver's main duty then will be to try and prevent lung barotrauma by ensuring the victim breathes out.

NOTE - On this specific point, a simple and 100% effective method forcing an unconscious man to breathe out underwater has still to be found.

Let us assume that we have past this stage ,that the diver in trouble has been brought to the surface and is now lying, unconscious, on the deck.

He could be unconscious for a number of reasons that can be summarised as:-

- onset of decompression sickness
- asphyxia/drowning
- lung barotrauma/cerebral embolism
- shock due to physical injury

It is difficult to speculate in advance on what type of physical injury might be involved, so for the purposes of this speech, I shall not discuss the possibility of injury for the time being.

The treatment required can therefore be categorised:

- onset of decompression sickness

  Obviously this can be treated only by recompression

- lung barotrauma/cerebral embolism

  Again quick recompression will be in order

- asphyxia/drowning

  Immediate resuscitation must be the course of action
  Transporting the injured diver to a possibly remote recompression/ decompression chamber would only delay this treatment.

We must also consider that there is a possibility of the diver suffering from a combination of these factors.

Our choice of action is thus going to be either:

Resuscitation on the spot

OR

Recompression (implying a certain delay during transportation to the chamber).

Our actions will be based on the diagnosis of what the trouble appears to be.

If the dive duration necessitates decompression or if we diagnose lung barotrauma or cerebral embolism we shall recompress.

If asphyxia or drowning during a short dive is diagnosed, we go for resuscitation.

NOTE :

- Applying resuscitation manœuvers to a man in need of recompression will not harm him in any way, unless the recompression is delayed by this action.

while

- Recompressing a man who needs resuscitation will not do him any harm unless the resuscitation is being delayed by this action.

Thus, the question is:

is our patient suffering from axphyxia/drowning or from lung barotrauma/cerebral embolism?

Though the answer to this question might appear simple to you members of the medical world, it is my opinion that it would still take you some time to make your diagnosis - and time is in short supply. Moreover, you will not be at the scene of the incident for several hours and it will be up to a diver/supervisor to make the choice.

- the diver/supervisor - therefore has to make a diagnosis based on a cursory examination and this presents several problems. For one obvious sign of lung barotrauma will be the presence of bloody froth at the mouth.

But a drowned patient will quite often have froth present in his mouth. If he has bitten his tongue during the emergency/recovery, the resulting sign will still be bloody froth.

CASE HISTORY No. 1

A short dive at 17 Metres. J.D. had not taken enough care of his umbilical, and his line, due to the flow of the water current, is not "clear". The gas connection (badly set up) on his mask disconnects while he is breathing heavily. He fails to switch to bail-out supply, panics, pulls his mask off in a panic reflex and passes out.

His buddy diver considers the difficulty of clearing his line and chooses to cut his colleague's umbilical to save time.

On surface, some time is spent lifting both men from the water to the deck. An examination indicates J.D. is not breathing and his heart is not beating.

J.D. is immediately transported to a surface chamber 60 yards away, across a very cluttered deck.

Three attendants are locked in with him and within minutes cardiac and respiratory functions are restored.

Medical help arrives within two hours of the accident. Pupils are found to react only sluggishly to light. J.D. dies in the chamber 26 hours after the accident.

Autopsy showed no evidence of lung barotrauma.

In this specific case, we must conclude that slow transportation did delay resuscitation maneuvers to a fatal extent.

We have seen that we have a choice between two courses of action, depending upon the identification of the state of either drowning/asphyxia or lung barotrauma/embolism. We have also seen that to distignuish immediately between these two states is very difficult, to say the least. So what shall we do?

We decide to react to the worst possible situation and assume the diver is suffering from an embolism.

This presents the problem of resuscitation delay due to transportation to a decompression chamber.

Therefore, we simply develop a system of applying resuscitation during transportation.

Artificial respiration is something that can be performed while the patient is being transported - providing he is in the right position and his air ways open - by walking along beside the stretcher, and holding an oral nasal mask over the patient's face supplied from a portable O_2 cylinder.

Cardiac massage, however, can not be performed while the stretcher is under transporation, or at least not effectively.

Thus a pattern has been developed allowing resuscitation to be performed in alternance with transportation.

The patient will be carried towards the chamber on a stretcher while an attendant walking along will perform artificial respiration by Ambu bag, or similar device, for a duration of 15 seconds,
then the stretcher will be laid on the deck and cardiac massage will be performed for a duration of 10 seconds.
Then transportation/respiration will be resumed for another 15 seconds.
The cardiac massage again for 10 seconds,
and so forth.
all the way to the chamber.

We are convinced that even if this procedure lengthens the tranpsortation time to the decompression chamber, hence a delay before treatment, the patient will still stand a better chance than if he had been carried more quickly but without proper resuscitation manoeuvres.

Let us consider now the rescue of a bell diver from his working depth.

This procedure will differ from the one involving the rescue of the surface diver in the fact that:-

- there is no lung barotrauma/embolism danger
- there is no decompression involved at the time of the emergency
- there is only one rescuer available
- the retrieving and reviving must be done within a diving bell implying, most of the time, a very narrow entrance and very little internal space.

The key person in this case will obviously be the bell attendant or bellman.

He will be on his own, and depending on whether he alone takes the right set of actions, the diver in trouble will survive or die.

The awareness of his responsibilities in this situation will add to the bellman's stress and some ill prepared men have been known to break down under such stress.

It is most important that this man is trained and, has experience of rescue techniques in advance. Also he must be convinced that the surface has faith in him, and will give him all the support, guidance and advice they can over the communications.

The presence of a TV camera looking into the bell is of immense value to the surface crew giving them a direct insight of the situation.

The first stage of the operation, to get an unconscious or otherwise endangered diver back to the bell, will be done either:-

-1) By pulling him by his umbilical all the way to the bell and through the trunking (assuming the diver and his line are free) Fishing him through the trunking could be tricky, depending on the point of attachment of the umbilical on the man, and of the size of the trunking.
Precious time has been wasted in the past because the man being rescued was jammed "across" the underneath of the trunking as his diving line was attached somewhere on his chest and not between the shoulder blades.

-2) If the man (or his line) is fouled and cannot be pulled back into the bell by the bellman, the bellman has to lock out, reach the diver try to ensure a gas supply is available, drag him all the way to the bell - being careful not to foul any of the umbilicals - and depending upon the size of the trunking, either swim directly into the bell, (partly flooded before hand) with the diver, or attach him outside, get in and then hoist the diver inside.

If the bellman locks out, this phase of the operation alone can last several minutes, adding to the critical shortage of time.

It must be noted that rising the level of water into the bell will help in two ways:-

-1) By making the diver lighter to pull through up the trunking

and -2) It will allow the bellman to swim back into the bell without having to climb into it unaided.

The system of piping we have installed as standard equipment in our bells will allow an automatic partial flooding of the bell, even while the bellman is out.

When the diver is eventually brought in by the bellman, it is most important that it is secured as soon as possible in order to prevent his from falling out again and give the bellman the free use of his hands.

CASE HISTORY No. 2

As M.B. is working outside the bell by 100 or so Metres, something goes wrong. He rushes back towards the bell, in the process he chose the wrong way, takes a loop of his umbilical around an obstruction, eventually reaches the bell, and tries to climb into the trunking.

He is prevented from succeeding because his snagged line is holding his back.

The bellman tries to help by pulling him in, without success.

There is a hoisting device present but the bellman does not make use of it.

In order to free the diver from his umbilical, he attempts to cut it. This not only results in stopping the gas supply to the diver but also lowers the water level into the trunking, thus adding the divers own weight to the resistance of his fouled line.

The bellman again attempts to help the diver by lifting him bodily, runs out of breath in the process, falls down on his knees on the verge of unconsciousness himself and lets go of the then unconscious diver who subsequently drowns.

Attaching the diver on the hoist should have been the very first action of the bellman in this case.

Having recovered the diver into the bell what shall be the next step?

It might surprise some of you, but you cannot lay a man onto the floor of a bell as I have heard a few doctors suggest because there is no floor in a bell.

All there is is an open trunking 24 to 32 inches in diameter and a narrow foot rest ridge half a foot wide all around it.

You might suggest closing the door and laying the man on top of it. This is not a bad idea but,

every second counts and it is pretty difficult, in some cases, to lift the man all the way through the trunking and have enough clearance to close the door.

CASE HISTORY No. 3

Having managed to bring the diver back into the bell, hanging on his harness, the bellman restores cardiac and respiratory functions. He then spends about 30 minutes simply attempting to winch the man all the way in before closing the door. He can not succeed and the bell is lifted up from 70 to 50 Metres where surface stand-by diver is able to help.

If the hoisting attmepts had taken place before the resuscitation attempts, I am very doubtful about the success of the enterprise.

Due to all these reasons, we are convinced that the only course of action, considering the shortage of time and the ergonomic design of most bells, is that

the man must be revived while hanging on his diving harness in a vertical position.

This is an unusual concept to say the least and I expect a shower of objections from this audience during discussion time.

The harness we are using provides an attaching point for both dive umbilical & a hoisting device located between the shoulder blades of the diver in order to facilitate the entry through the trunking.

It has also some leg loops or straps that provides pelvic support. A chest support only would hinder attempts to restore breathing functions.

This pelvic crutch support has a drawback; depending on the circumstances, a male patient might find it jolly painful. However, this particular aspect has been disregarded on the grounds that the hot water suits provide a pretty rigid protection, and that some pain in this area might help precipitating the return of consciousness.

In order to perform artificial respiration we must ensure the airways are clear.

The diver hanging on his harness is in a near vertical position, tilted a few degrees forward, his chin resting on his chest. This is far from the conventional position of having the patient lying on his back, neck extended, jaws drawn upward.

However, by just lifting his chin, we put the patient back into the position in which you and I are at this very moment.

In order to achieve this we use a "rigid collar" contraption. The opening at the front allows the checking of the carotid pulse.

A Goedel type tube or similar instrument is inserted into the mouth/throat.

During this sequence of events, the position of the man's head - tilted forward - allows the natural drainage of saliva, vomit, possible blood, etc.....from the mouth.

When it has been established that the airways have been cleared with the application of the rigid collar and Goedel tube, mouth to mouth respiration technique is used to restore breathing functions.

NOTE

It would be very helpful at this stage to have an automatic resuscitation machine similar to the minutman or motivus, or other models standardly available on the market. The bellman could simply strap the oral nasal on the face of the victim, open the valve and would have no further worries about respiration. The machine would fill the lungs of the patient at regular intervals with a breathable mixture, which would then be exhaled by the natural elasticity of the body.

The trouble with the machines on the market is that they are basically designed to work at atmospheric pressure with pure O_2. When put in Hyperbaric conditions and driven by heliox mixutres, they behave absolutely erratically.

Different firms approached with this problem have come up with propositions that were either not acceptable or, after some research, were abandoned.

Also, the prospect of a narrow market might have constituted a strong deterent.

Dr. Ian ROSS and Henry MANSON from ABERDEEN have developed a unit which appears to work perfectly in theory and which is now awaiting a generous sponsor to finance the construction of a prototype.

Having taken care of the respiration, let us assume that the heart has stopped. We now have to worry about cardiac massage.

Even if there is a real danger in having a non-medical, possibly ill trained, person attempting cardiac massage, by not attempting it we condemn the diver to death.

The standard way of applying cardiac massage, however, necessitates lying the patient on a flat, hard surface and raising his legs to allow the return flow of blood towards the heart.

Our patient hanging vertically on his harness is in the worst possible imaginable position for this purpose, but other alternatives would succeed only in making the situation worse.

Having accepted this, we try to prevent the pooling of the blood at the "bottom of the patient" by keeping his immersed as far as reasonably practicable.

Though I cannot express it in a scientific way, I am convinced that this procedure somehow helps the situation to a certain extent.

Some work has been carried out in Sweden, I believe, demonstrating a 30% increase in cardiac output of a submerged man.

I will also quote Dr. David ELLIOT in his book Physiology and Medicine of Diving page 136

QUOTE:

In the chapter Pulmonary Functions, effects of submergence

"when a diver is vertical, standing or sitting underwater, the forces acting upon him are quite complex."

he then goes on to say

"The pressure of surrounding water is different at every level subtended by the structures concerned. Buoyant force cancels the effect of gravity upon the abdonminal contents, whose weight ordinarily tends to pull the diaphragm down. Buoyant force clearly offsets the usual tendency of blood to pool in the lower portions of the body, so we can expect to find a larger-than-usual volume of blood in the great veins and pulmonary vessels."

UNQUOTE

A number of members of the medical profession, however, have told me that I was wrong and that maintaining the patient partially submerged does not help in any respect.

They might be right. But I do not think that this system of partially submerging a diver makes the situation any worse.

Moreover, an equal number of members of the medical profession have told me that I was right on this point.

I expect some strong and possibly contradictory views from the floor, when discussion time comes.

Coming to the practice of the cardiac massage itself, it is obvious that trying to apply some pressure on a man hanging on a rope will only make him swing. We have to provide therefore both the pressure and the support.

This can be achieved in several ways:

- by embracing the man from behind, circling his chest with both arms and applying a rhythmic pressure with both fists.
  This method is not very effective, it is very tiring and causes delay with the removal of the back-pack carried by the diver prior to the attmept.
- by facing the patient, right arm under his left shoulder, left arm holding his harness at his right shoulder level, and applying pressure with right shoulder. This method has been devised and developed in Italy by Professor ZANNINI from GENE.
  Though this method is easy to initiate quickly, it has the drawback of being tiring after a short period of time and of not allowing a good rate of compressions.
- by facing the patient, both arms passed underneath both of his shoulders and applying the pressure with the head.
  Though this procedure is unusual and difficult to imagine, it is most effective, not tiring and allows virtually any speed when squeezing.
- by facing the patient (if suspended at a lower level in the bell) holding both sides of the harness at shoulder level, and applying the pressure with the knee.

  This procedure is very similar to the previous one, only more tiring.

Gentlemen,

In this speech, I have looked at some of the basic problems we have encountered in rescuing and resuscitating divers in distress. It must be remembered that divers are working in an alien environment and therefore we have to resort to unorthodox techniques on some occasions instead of applying standard medical procedures.

Many of you may feel that we are not qualified to establish new such procedures and in a medical sense you are correct. However, our industry has gained great experience in diving practices in recent years and subsequently we have adapted our emergency techniques. We understand our "high technology" equipment; we have experience at working in this alien envoronment; we appreciate the stress exerted on bellman and surface crew when a diver becomes unconscious.

We therefore have to adapt standard medical procedures to suit this alien environment pooling our experience together with your knowledge. We must establish jointly more of procedures along the lines I have outlined in this speech to cope with emergencies and give divers training and in situ practice in these procedures.

ULTRASONIC DETECTION OF BUBBLES

by

Dr. G. MASUREL

After a brief historical review of ultrasonic detection of bubbles in hyperbaric conditions, the various techniques tested - absorption, distortion, reflection - and the results obtained are described.

The technique of reflection, based on the Doppler effect, is the only one to have been developed on a large scale and used in practice. The principle and the various ways of applying this principle (continuous emission, pulsated emission) in equipment design are explained. A description of how to use the Doppler detector is given :

Peripheral or precordial detection, examination at rest or after exercise, graphic and auditory analysis of the specific signal corresponding to the movement of bubbles.

There is a clear need to codify bubble quantities : the Spencer code based on the frequency with which the bubbles appear in each cardiac cycle is compared to another code which is based on selective analysis of the various components of the bubble signal.

This more detailed analysis makes it possible to minimize the subjective factor peculiar to auditory discrimination and provides the basis for determining the reliability of processing the signal by computer as is now possible.

Research workers soon expressed misgivings as to the safety aspects of using ultrasonic equipment in hyperbaric conditions, since it was feared that ultrasound could in fact generate bubbles. The tests carried out show that at the frequencies and acoustic powers used the risk of this eventuality is negligible even in a supersaturated environment.

The data on the quantity of bubbles in circulation obtained by Doppler detection have a twofold interest : theoretical and practical. They have illustrated that after exposure to hyperbaric conditions approaching the limits allowed by the schedules, circulating bubbles occur in several subjects. Provided that they do not exceed a given quantity, these bubbles are filtered via the lungs and are asymptomatic. They occur both in bounce diving and in saturation diving, in air and in helium. They come from the microcirculation, from the fast tissues in the case of short dives (muscles) or from the slow tissues (fatty tissues) in the case of saturation dives. However, the main point of detecting these bubbles during real or experimental dives is to be able to establish a relationship between the output of of bubbles and the occurrence of decompression accidents.

This relationship seems clearly established in the case of short bottom-time bounce dives in air or in helium. This is not the case as regards saturation dives, (especially in helium) : although the apparent output of bubbles during the first phase of decompression appears to be correlated with the occurrence of accidents, particularly vestibular accidents, the same is not true of the end of decompression which often involves Type I bends with few or no circulating bubbles.

These bends are obviously linked with the presence of stationary bubbles which cannot be detected by the Doppler techniques.

Since it is difficult to obtain sufficient Doppler detections to match the frequency of bends, it is proposed that standardized data banks be set up to permit rapid exchange of information between the various diving centres.

Summing up, when they reveal a critical output of bubbles, Doppler detection techniques provide data which enable the accident probability during bounde dives and during the first phase of a saturation dive decompression to be assessed. This information means that appropriate preventive measures can be taken. In addition, Doppler techniques are useful in assessing the state of overall supersaturation of the organism and are invaluable in checking the existing schedules and in compiling new schedules.

THE DIAGNOSIS OF DECOMPRESSION ILLNESSES

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Although the range of decompression illnesses is quite wide, it is intended in this paper to concentrate solely on decompression sickness and arterial gas embolism consequent upon pulmonary barotrauma.

Further, the title might more accurately be described as "possible aids to the diagnosis of decompression illnesses". This somewhat unwieldy title acknowledges that even with a full history and thorough clinical examination, it may be very difficult to reach a definitive diagnosis on which to base compression therapy. This is, of course, less of a problem where the distinction between decompression sickness and arterial gas embolism does not radically alter the initial therapeutic compression selected.

It would be conceit to discuss the minutiae of history taking and clinical examination but it is not conceit to stress the diagnostic value of both. To be able to reach an accurate diagnosis may not, as has been mentioned, affect the initial compression but it is certainly important when adjuvant and supportive therapy is considered.

It is intended therefore to discuss current and possible future aids to diagnosis which are additional to the history and examination. Some may be of use before therapy is instituted, others of use during therapy and some may be more applicable to situations arising during saturation diving.

Chest X-rays

In addition to the undoubted value of a CXR in determining the presence of pneumomediastinum and pneumothorax, which in themselves are not indications for compression therapy but may complicate the course of therapy, the possibility of X-raying the chest through a chamber port has been in use by the Royal Navy for some years at the Submarine Escape Training Tank at HMS DOLPHIN. More recently

such a facility has been developed for more general use by Comex Diving, Aberdeen.[1]

It should never be forgotten that a pneumothorax may arise de novo during decompression and a rather elegant adaptation of transcutaneous ultrasonic Doppler techniques has been described by JAMES[2] which allows accurate diagnosis of the presence or absence of pneumothorax. It basically depends on the modification of reflected ultrasound by gas interposed between the parietal and visceral pleura. This device works perfectly well under pressure and gives a potentially valuable diagnostic tool to the physician.

ULTRASONICS

Apart from research based on intrasvascular bubble detection using implanted ultrasonic Doppler transducers, a considerable amount of work has been done on precordially placed transducers. The subject is reviewed by EVANS[3] in The Physiology and Medicine of Diving and Compressed Air Work (Bennett and Elliott), and it is sufficient to say that the major difficulty is the poor signal to background noise ratio. Although satisfactory signal processing can not be far away, and the work NISHI[4] is but one distinctly promising approach, it is probably still true to say that a well trained pair of ears is the only really satisfactory way of detecting bubble events in real time, particularly when field use is considered. Nevertheless, PILMANIS[5] has reported favourably on field use of such "audio-interpretation" stressing that the period of one to two hours post-dive is the most valuable period for useful monitoring. Later events are more difficult to interpret by ultrasonic methods. Several classifications of frequency of bubble events measured precordially exist such as those of SPENCER[6] and POWELL[7] where grading is carried out with corresponding clinical actions are recommended. It must be said that the accent in this technique is on prophyllaxis but it is considered that it has a rather wider application than is currently accepted, particularly when signal processing has eliminated the problem of background noise.

Looking to the future, it is too early to say whether the work of RUBISSOW and MACKAY[8] and DANIELS[9], on ultrasonic imaging of bubbles,in situ,in limbs has a practical application outside research work and adding to the knowledge of the aetiology of decompression sickness.

Unfortunately, ultrasonics have little or no practical part to play in the diagnosis of arterial gas embolism, for the diver is invariably inaccessible when this potentially catastrophic event occurs. Unfortunate indeed, for it is relatively very easy to detect arterial gas emboli if they are passing through the carotid arteries, heading for the cerebral arterial circulation.

ENT INVESTIGATION

EDMONDS[10] states that "the relationship between inner ear disorders and decompression sickness is beset with conjecture and prejudice" and that "it is unfortunate that there are so few cases (ie inner ear barotrauma) which have been objectively assessed before, during and after treatment". These truisms still hold good far too often and if there is any question of otological involvement in decompression illness it should be assessed as carefully as time allows. It is fair to say that audiometry and caloric testing do not take up a great deal of time and that electronystagmography has been shown to be a practical proposition inside chambers. The latter could be a valuable diagnostic aid on these admittedly rare occasions, mostly excursions from deep saturation, when the diagnosis between vestibular decompression sickness and High Pressure Nervous Syndrome is difficult or even impossible. Also, simple motion sickness can be singularly troublesome to differentiate from vestibular decompression sickness.

Finally, inner ear barotrauma, resulting from rupture or fistula of the round or oval windows during compression, does not need further compression but does need urgent surgery if hearing is to be conserved. History, examination and an adequate ENT evaluation should resolve this problem.

HAEMATOLOGY

It seems prudent in cases of serious (as opposed to mild) decompression sickness to routinely take blood for haematocrit. Whilst in no sense diagnostic, such action is valuable in deciding how fluid replacement therapy should be carried out. This pre-supposes laboratory access which rules out certain occasions.

It would be valuable if some quick haematological test could differentiate between mild and serious decompression sickness, and arterial gas embolism. If the

events at the blood bubble interface are significant in determining the severity of decompression sickness, one possible test of value would be an assay of Fibrinogen Degradation Products (FDP). INWOOD[11] suggests that raised FDP levels are of significance in serious decompression sickness, as have other authors, and a reliable 2-minute slide test is available in the form of the Thrombo-Wellcotest*. which will detect concentrations of FDP in excess of 2 µg per ml. Certainly, such a test would be only of significant use if the result was to influence management of the case. It is suggested that perhaps this form of investigation might be worth looking at further but it has to be admitted that none of the many other possibly significant haematological changes described in decompression illnesses lend themselves to easy assay within a time limit that could influence the course of therapy.

MONITORING UNDER PRESSURE

The ability to monitor under pressure various functions such as ECG, EEG and ENG is demonstrably available and it is up to the physician to decide on their relevance to his conduct of therapy. There is certainly scope for standardisation of electrical penetrations in order that the physician going offshore may take his various monitoring aids with him in the sure knowledge that compatability exists.

However, none of the above monitoring is essentially of diagnostic value in terms of decompression illness.

POST-THERAPY INVESTIGATION

There is undoubtedly room for much improvement in the investigation of decompression illnesses following therapy, even if the therapy has been overtly successful.

Apart from uncomplicated mild decompression sickness, all other cases of decompression illnesses deserve full investigation following therapy. Such investigation ought to be concentrated on providing relevant information on the aetiology of the accident and prognosis with regard to future diving. The latter problem becomes increasingly important with the introduction of strict medical standards concerning fitness to dive and the potential loss of high earning power. Also, such assessment and investigation after initial therapy has not infrequently

indicated the need for further active hyperbaric oxygen therapy. The author has personal experience of two cases of vestibular decompression sickness which were ostensibly sign and symptom free following therapy. Immediate, full ENT investigation revealed a unilateral canal paresis in each case and further hyperbaric oxygen therapy was successful in achieving normality.

In conclusion, the physician treating decompression illness should bear in mind the various diagnostic aids available, particularly those capable of being adapted to use under pressure. In this last respect, it is hoped that through the aegis of the Diving Medical Advisory Committee to the United Kingdom Association of Offshore Diving Contractors, a standard range and form of electrical penetrations may be recommended for all chambers in the UK and the UK sector of the North Sea. If this can be achieved, a physician can be assured of compatability with any monitoring or diagnostic equipment he may care to use.

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THE USE OF OXYGEN AND PRESSURE AS INDEPENDENT VARIABLES IN THE TREATMENT OF DECOMPRESSION SICKNESS

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INTRODUCTION

The treatment of decompression sickness by the application of pressure implies two additional features; first, that the breathing mixture must contain at least 0.2 atm. oxygen together with some additional diluent gas, that is, the breathing mixture must be specified; secondly, compression implies subsequent decompression, which is not essential to treatment, but is essential for returning the patient to his customary environment. When, therefore, treatment is judged to be adequate one must expose the patient to the additional hazard of decompression, knowing that if this is not well-judged he may have a recurrence of signs or symptoms and be in a worse condition than that originally needing treatment. Decompression is not part of treatment but a necessary final step in completing treatment.

A complication, of course, is that the form of the decompression is related to the type of gas breathed and to the time spent at pressure; it is therefore quite natural to attempt to make decompression easier by reducing both the maximum pressure and the time at pressure and by increasing the amount of oxygen in the breathing mixture. Such an attitude is not wrong, but it is strictly irrelevant to the subject matter being presented, which is concerned with the evidence relating to the effectiveness of pressure and of oxygen in treating decompression sickness. In the interest of brevity the treatment of pulmonary barotrauma and arterial air embolism will not be discussed.

HISTORICAL PERSPECTIVE

A century ago, as a result of extensive research using a variety of animals, Bert (1878) reached the conclusion that the disorder, which we now call decompression sickness, was caused by the liberation of the gases of the blood under the influence of sudden decompression. Bert therefore not only

confirmed what had been suggested by Bucquoy as to the origin of symptoms, but he showed by experiment that recompression was beneficial and also recommended the combined use of oxygen breathing and recompression, in order to eliminate bubbles of gas from the blood. Treatment of decompression sickness was by no means as easy to achieve as it was to prescribe since the engineering techniques of the day allowed only moderate pressures to be attained in medical treatment apparatus, not usually more than 2 atm., and furthermore, current practice was to give a dose of oxygen during each treatment, usually not exceeding 30 liters.

The use of pressure in building the foundations of bridges was well advanced and since the invention of the caisson by Triger (1841) it had been used in France, Britain, Germany and North America. These enormous undertakings had led to a considerable body of experience, relating to thousands of exposures, of the harmful effects of decompression which were supplemented by the observations and scientific studies of Pol and Watelle (1854) who reported the death of a worker subsequent to decompression and seem to have been the first to use recompression by returning a worker to high pressure air. It is perhaps surprizing that these new engineering techniques seemed more readily transferable than the medical knowledge of the associated hazards; for we find Jaminet (1871), writing on the medical problems met during the construction of the bridge at St. Louis, prescribing a cordial, largely composed of Jamaica Rum, for the treatment of any workman taken sick after leaving the caisson. The first introduction of a "medical air-lock" for the specific treatment of cases of "caisson disease" was however in the U.S.A., by Smith (1873) and was used during the building of the Brooklyn Bridge at New York.

If the use of pressure was not widely known in the latter half of the nineteenth century, the same was not true of the use of oxygen, but prior to the construction by Junod (1834) of a copper chamber, oxygen and other gases could only be given at atmospheric pressure. Subsequently, the compressed air baths popularized by Pravaz (1837-38) and by Tabarie (1838) were used to treat anything from catarrhal deafness to pulmonary tuberculosis. If nowadays this seems strange it is relevant to note that the indications for treatment with oxygen and its limitations are still not generally understood, nor agreed. The variety of disorders being treated nearly 150 years later is no less and illustrates a theme to which we will return; namely the necessity for experimentation to precede rather than to follow clinical innovation. Kindwall (1977) has recently summarized the indications for the use of oxygen in clinical practice and includes the treatment of decompression sickness in the category (1) which comprises disorders for which oxygen is the primary mode of treatment. This choice is justified by reference to 100 years of experience rather than to any objective evidence.*

LOCAL PRESSURE

Although one generally thinks of compression as being of the whole body, Junod (1834) also made a chamber which could apply local pressure to the limbs. The use of such local pressure is discussed by Fryer (1969) who quotes the work of Fraser and Waters (1942) and calculates from their data that, for example, a hydrostatic pressure of 75-100 mm Hg has relieved leg pains. Anecdotal reports of Japanese divers using hot-baths to relieve the pain of limb-bends may represent the independant discovery of an effective treatment for the milder forms of decompression sickness.

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* In fairness to the authors it should be said that this work is only a summary and does not have room to present the detailed arguments.

COMPRESSION RATE

At first it is likely that rates of compression were set by factors such as the size of valves and the capacity of storage cylinders. In diving, compression was probably determined by similar factors such as pump-speed and the ability to handle life-line and supply hose. Whatever the reasons, the present position can be summarized as being; rapid compression for serious cases and more measured, arbitrary, compression rates for all other cases. Rapid compression for example at the Royal Navy Submarine Escape Training Tank, rarely exceeds 50 m/min, although experimental subjects have tolerated rapid compression to 19 atm. in 20 sec, which is approximately equivalent to 600 m/min (Barnard (1971)) but it is not suggested that such rates are necessary or desirable when treating patients. The diving company COMEX use a compression rate for treatment of only 2-3 m/min, whereas most European naval practice is to use standard rates of 10 m/min. In the U.S.A. the rate used is 25 ft/min (7 to 8 m/min).

It is probable that the rate of 25 ft/min was adopted after experience with cases in which recompression caused exacerbation of pain; although such cases are not common they were alluded to by Yarborough and Behnke (1939) and more recently by Leitch (1971). There is no known experimental evidence which can be used as a guide to the best compression rate, which must therefore remain arbitrary and may well be a matter of convenience.

TREATMENT PRESSURE

Discussing the pressure necessary for treatment Yarborough and Behnke (1939) considered the following four categories;

(a) sufficient pressure to relieve symptoms.

(b) employment of pressure greater than that required for relief.

(c) return to the pressure of the original dive.

(d) greater pressure than that corresponding to the original dive.

Their personal preference was to compress to the pressure of relief, but they eliminated options (c) and (d) for theoretical reasons which are not all clear nor convincing.

Historically there is not doubt that compression to working-pressure came first; only when separate treatment chambers became available was there any choice and predictably when there was a choice, there were those who expressed preferences for other pressures and for different durations of treatment. Ryan (1912) proposed compression to only two thirds of the working pressure and Pelton (1907) suggested that maximum pressure should not be held for longer than 5 to 10 minutes. Haldane (1907) quotes a case reported by Dr. Gould from the East River tunnels in New York which clearly shows that recompression to the pressure of relief was in use in 1906. The case is also of interest since it records the treatment, using compressed air, of a man paralyzed in both legs following exposure to 34 psi (3.3 atm) the patient was treated 45 minutes after the onset of symptoms and the pressure raised by stages over 15 minutes to 31 psi (3.1 atm). It is recorded that he "walked perfectly well and sensation normal at 30 pounds." Decompression took some 40 minutes, without recurrence of signs or symptoms. This case is an interesting one to compare with the treatment at similar pressures used in the Goodman and Workman (1965) tables, which in the shortest version (intended for cases with pain only) would have taken 135 minutes as against the total time of 57 minutes used by Dr. Gould.

A variation from the categories proposed was actually used by Yarborough and Behnke (1939) in which patients were taken to the pressure of relief plus an additonal atmosphere except, that a minimum pressure for treatment was arbitrarily set at 45 psi (4.1 atm). This should properly belong to

fifth category which is now by far the most common form of treatment namely;

(e) an arbitrary pressure. (a subset of (b)).

Compression to greater pressures than that of the original exposure is a late development arising on the one hand from experience with acute cases of decompression sickness in aviators, who were sometimes found to benefit from high pressure treatment (Donnel and Norton (1960) and on the other from the treatment of divers in whom symptoms occurred at depths greater than those used in normal treatment. Barnard (1967), using an empirical approach, found that in some of the cases occurring during experimental oxy-helium dives, compression to pressures greater than that of the original exposure was necessary to produce relief of symptoms. An additional treatment based on the use of a slightly increased maximum depth of 70 meters and using a continuous decompression approximating to a 1.3:1 pressure ratio drop over 5 hours was subsequently introduced into the Royal Navy Diving Manual as a direct result of the experience gained from these observations.

The method described by Griffiths (1969) extends this type of treatment to tunnel-workers. Due to the fact that serious cases tended to occur during locking-out from the work-site, these were treated by compression to working pressure. Cases with bend-pain only were compressed to working pressure plus 2 psi (0.14 atm) and if necessary to greater pressures in increments of 0.14 atm. if pain was not relieved after 15 minutes.

Zheifets Tetel'Baum and Rozov (1970) cite animal experiments by Yunkin (1969) and by Nazarkin and Yunkin (1969) which were said to show that an increase in the maximum treatment pressure increased effectiveness of treatment in animals with severe decompression sickness. Reference to the treatment tables used in the U.S.S.R. (Berghage, Vorosmarti and Barnard (1978)) shows that the maximum depth used for the treatment of severe cases

is 97 meters (10.7 atm) and the longest time at this depth is 1 hour. The breathing medium used at this depth is either air or an air-helium mixture.

It should by now be clear that all the items in the Yarborough and Behnke classification have been or are being used by physicians working in the field and that all seem to have some degree of success which is difficult to measure or to compare.

Evidence from statistical studies by Van der Aue, Duffner and Behnke (1947) Slark (1962) Rivera (1964) Moretti, Fontanesi and Ghittoni (1970) (1973) Berghage (1976) show that most cases of decompression sickness occur after a short surface interval. (This implies that few "subsaturation" diving schedules are grossly inadequate). During the development of new diving schedules or in other exceptional circumstances cases may occur at depth as has already been mentioned, such cases which necessarily have to be treated in an ad hoc manner supply valuable data since they amount to experiments in treatment.

TREATMENT OF DECOMPRESSION SICKNESS ARISING AT PRESSURE

The available data relating to the treatment of cases occurring at raised pressures has been compared in Tables 1 - 4. It will be seen that some of the data relate to oxy-helium exposures and treatment, the remainder to air dives followed by either air or oxygen treatments: Nevertheless, it seems clear that, independant of the gas being breathed, severe cases need significantly greater treatment pressures than do milder cases and in general, the latter seem to be effectively treated by increases in depths of less than 12 meters. A number of factors is probably responsible for the greater variability of the data relating to severe cases. These cases form a heterogeneous group, far more so than limb-bends, they are smaller in number (28 cases as against 64) and occurred during different types of experimental dives.

At present all that can be said is that the observed variability may be characteristic of such cases and that no reliable guide can be given for the pressure ratio or ΔP necessary for treatment.

ADEQUATE TREATMENT

Different authors have developed their own definitions of adequacy in treatment, for example Goodman and Workman (1965) refer to full treatment depth (60 ft) and the designated period of oxygen breathing, the presence of both constituting adequate treatment. Other such terms as 'optimum' or 'sufficient' pressure, reflect our lack of evidence to guide behaviour and also the two unresolved attitudes, "more is beneficial" and its converse that "more is harmful".

Whatever the theoretical reasons for supposing that all cases of decompression sickness should respond to compression in a similar manner, this hypothesis has rarely been tested. Barnard and Hanson (1973) studied six treatment depths from 0 to 80 meters following dives with mice to between 125 and 225 meters. All exposures used oxy-helium mixtures. There was a progressive and significant reduction in deaths with increasing treatment depth but only one of the 960 animals exposed developed symptoms after recompression to deeper than 15 meters. The authors concluded that for mice there appeared to be an optimum depth of between 15 and 40 meters, such pressures being sufficient to produce "cure" from any depth. The lack of a clear relationship between exposure depth and treatment depth is consistent with the theoretical discussion of Van Der Aue, Duffner and Behnke (1947) concerning the reduction in size to be expected from compression of spherical bubbles and would suggest that immediate recompression in mice produces a theoretical reduction in linear dimension, equivalent to from three quarters to one-half. It would be highly speculative to argue from these results to the effects in man, but it would

suggest that, in most cases, the amount of recompression necessary falls short of returning to the original pressure.

Heimbecker, Koven and Richards (1973) studied the microcirculation in hamsters and in dogs. Twenty dogs who had been decompressed were recompressed during observation of the microcirculation of the conjunctiva. Heimbecker et al. made the statement that "rapid recompression dramatically improved tissue perfusion" but presented no supporting data.

Both the treatment tables of Van Der Aue et. al. (1945) and Goodman and Workman (1965) substitute an arbitrary treatment pressure for an empirical end-point established for an individual patient. The statistical analysis of the results of such treatment tables may however be biased by the type of case presenting. Behnke (1942) for example, remarks that injury to the cerebrum is rare and that "Menieres syndrome was described not infrequently in the older reports." The greatly increased number of experimental dives over the past 15 years has resulted in an increase in the number of cases showing disorder of balance or hearing. (Coles (1973), Farmer (1973)). These hint at a relationship between the degree of adequacy of a diving schedule and the severity of decompression sickness produced. In other words, not only do different schedules produce a different incidence of decompression sickness but a different type of disorder. This point is well illustrated by the analysis of Goodman and Workman (1965) who noted an increase in severe cases related to grossly inadequate decompression over the period 1946-1964 which was correlated particularly with the failure rate of Van Der Aue tables 3 and 4.

DURATION OF TREATMENT BY PRESSURE

If it was not necessary to return to atmospheric pressure, and provided that the conditions of living at raised pressure were not in themselves

harmful, then it would be irrelevant to ask, how long should one stay at pressure? and we could allow the patient to continue his normal work once we had relieved his symptoms and established that there were no obvious residual effects.

In the real world, one must decompress, and there may be expensive and practical reasons why this time should be kept as short as possible. There have also been until recently equally practical reasons why treatment could not exceed some 2 hours at maximum pressure; since no decompression schedules were available for longer times which could be relied upon to be safe. The technique of saturation diving has however made available slow but reliable decompression from depths greater than those customarily used for treatment (Hanson, Vorosmarti and Barnard (1978) (Barnard (1967)). Experience with cases of decompression sickness during deep dives has also made it clear that the distinction between preventing decompression sickness and treating it is sometimes difficult to discern. In a series of dives already referred to (Barnard (1967)) the usual treatment was to return to the pressure of relief, stay at that depth for 10, 20, 30 or 40 minutes and decompress using the original dive schedule. A fuller discussion of the treatment of such cases is given by Hanson, Vorosmarti and Barnard (1978). In this latter series, time at treatment pressure varied from 45 minutes to 10 hours but no clear relationship could be seen between the time spent at the treatment pressure and the likelihood of the recurrence of symptoms. Fagraeus, Miller and Bennett (1977) have developed a treatment which uses a combination of a prolonged stay at 4 atm and 0.5 atm oxygen. Such a treatment is certainly logical for severe cases and preferable to the repetition of shallower treatment regimes, each of which involves a separate and possibly harmful decompression.

THE USE OF OXYGEN

The use of oxygen in addition to recompression for the treatment of decompression sickness was tentatively suggested by Pol and Watelle (1854) and again proposed by Bert (1878) either alone or mixed with hydrogen. As has been noted, oxygen treatment antedates the first cases of decompression sickness and was, no doubt, used if it was available. Hoff (1948) cites Knowles (1911) as using oxygen at atmospheric pressure, for he had no pressure chamber, to treat a case of decompression sickness in a caisson-worker from the Boulac Bridge across the Nile. Behnke and Shaw (1937) quote some unpublished observations made upon anesthetized dogs. Twenty-six exposures were made of 1 hour 45 minutes at a pressure of 65 psi (5.4 atm). Following this the animals were surfaced in 10 seconds. Unless the animals were recompressed, bubbles appeared in cutaneous vessels, the respiratory rate rose steeply and arterial oxygen saturation fell, the animals becoming cyanotic. This was described as a condition of "asphyxia with signs of shock." Recompression to 30 psi (3 atm) relieved symptoms whether the animals breathed air or oxygen. Subsequently, after 90 minutes at 3 atm and a further 30 minutes of decompression those animals which had received a second exposure to compressed air again showed signs of asphyxia and bubbles were seen at post-mortem examination. Animals recompressed upon oxygen by contrast recovered almost completely and did not show bubbles at post-mortem. These experiments clearly showed the value of not adding more inert gas during treatment, an indirect virtue of breathing oxygen, but also showed that recompression without oxygen was quite as effective in relieving symptoms as was recompression with oxygen. The principal value of oxygen therefore appears to be in the prevention of recurrence during decompression. A further 15 exposures using 8 dogs showed that in one case at least paralysis could develop

at 3 atm while breathing oxygen; Behnke and Shaw suggested that in order to prevent spinal cord damage it was necessary to go to a higher pressure. The treatments used in this series were diverse; one animal recovered breathing O_2 at 3 atm while another recovered on three separate occasions, with a combination treatment of 5.4 atm air followed by oxygen at 3 atm. The conclusion that "65 pounds is necessary to prevent or to arrest the progress of incipient paralysis" may be true but seems unsupported by the data. These authors concluded that oxygen inhalation combined with recompression comprised the essential treatment for decompression sickness and also recommended the use of oxygen-nitrogen mixtures for pressures at which O_2 would be poisonous.

The treatment tables developed by Van der Aue et al. (1945) which, with local modifications, have been largely adopted throughout the world, recommended the use of oxygen for some schedules but gave alternative air versions. Oxygen was breathed as deep as 60 ft (2.8 atm) for 30 minutes and for longer periods at shallower depths. These tables were more successful in treating cases of bend-pain having a success rate of about 90% (Rivera 1964). The longer tables, 3 and 4, used in more severe cases were less successful. Table 3 having an success rate of about 80% and Table 4 one of between 43% and 55%. A factor which may contribute to this difference is that such tables are most often used for the difficult, complicated or delayed cases and there seems to be a considerable body of experience, but little direct evidence, that treatment is most effective when given immediately and also perhaps that smaller pressures may be effective if used early. The theoretical arguments which are consistent with such a view stem from the observation that gas bubbles in blood initiate pathological changes (Sautet, Jullien, Leandri and Rampal (1961)). The present position concerning such changes has recently been reviewed by

Philp (1974) and there appears to be a qualitative difference between acute and chronic cases which is more significant than the site of decompression sickness; the usual classification upon which treatment is based.

Goodman and Workman (1965) introduced their minimal-recompression, oxygen-breathing approach in order to treat those severe cases for which the Van der Aue tables seemed inadequate. The maximum pressure used is 2.8 atm and the patient breathes alternately oxygen and air in order to reduce the risks of oxygen toxicity. This technique rests on the experiments of Kaufman, Owen and Lambertsen (1956) who showed that guinea pigs exposed to 3 atm of oxygen convulsed after 5.2 hours whereas those exposed to oxygen pressures equivalent to air breathing for 5 minutes in each 30, remained at pressure for 17.0 hours before convulsing.

Just like Behnke and Shaw, Goodman and Workman were concerned to hasten the resolution of presumed bubbles, to speed the elimination of inert-gas and to promote tissue oxygenation. The results of the treatment of 44 carefully documented "serious" cases, was a reduction in the recurrence rate when using Van der Aue tables 3 and 4 from 29-47% to a rate of 11.4% when using their own table. (2 cases with incomplete relief of symptoms and 3 recurrences). If the recurrences are regarded as being due to incautious decompression and therefore not relevant to treatment, one might say that from this sample 42 out of 44 (95%) are likely to be treated satisfactorily by a pressure of 60 feet (2.8 atm) and further by inference from Behnke and Shaw (1937) irrespective of whether or not oxygen is breathed. However, the three recurrences (7%) during the breathing of oxygen suggest that the initial treatment pressure (2.8 atm.) was inadequate for these cases. Albano (1972) reported the treatment of 27 cases of decompression sickness by the Van der Aue, Goodman and Workman and other treatment tables. He described his own

modification of existing treatment schedules which he called a 'mixed treatment', since it alternated periods of air and oxygen breathing but used greater pressures than the Goodman and Workman tables. Limb-bends were treated by compression to 4.0 atm and the most serious cases by compression to 6.0 atm following oxygen at 2.8 atm and the infusion of low molecular weight Dextran.

The physiological background of these tables was discussed and detailed case-histories were presented. The type of case presenting in Sicily seems to have produced a higher failure rate than that reported by Goodman and Workman above. In the experience of Albano, only 11 out of 22 cases responded favourably to a single treatment. Fructus, Cross, and Comet (1978) have collected data for 124 cases of decompression sickness in France, in many of which treatment was seriously delayed. In spite of 104 cases out of the total being classified as neurological cases, the overall failure rate of treatment was less than 8% and in no group was it greater than 15%. Fructus et. al. draw attention to some unpublished obervations of Wells (1977) which seemed to show that the infusion of Dextran" was more effective in reestablishing mesenteric circulation in decompressed dogs than was compression to 120 psi (9.2 atm). Even if this highly suggestive observation is confirmed it only makes a case for the intelligent use of medical techniques other than the application of pressure and does not weaken the experimental and observational evidence for the value of pressure. The authors of this paper supply an interesting survey of theoretical possiblilites which lead them to a rational course of treatment. They record experiences with 10 cases of decompression sickness relieved in 7 cases at 9-16 m (1.9-2.6 atm) and 3 others relieved at 20-30 m (3.0-4.0 atm). The opinion is put forward that in severe cases, when treatment is postponed, the pressure of relief becomes a dangerous mirage, a view which most would share and which supports

the separation of acute and chronic cases previously referred to. The tables used in treatment by COMEX include one with a maximum depth of 30 meters (4.0 atm) at which pressure oxy-helium mixture (50-50%) is breathed if this is available.

CRITERIA FOR TREATMENT

Decompression sickness is self-limiting, if not severe enough to be fatal and recovery may occur spontaneously. Saumarez, Bolt and Gregory (1973) have successfully treated a severe case with oxygen and numerous pharmacological agents with a satisfactory outcome in spite of the absence of a pressure-chamber. The first essential of treatment is adequate and timely diagnosis, but it is not uncommon to use a trial of pressure treatment in order to confirm the diagnosis. If such a trial is used as an alternative to diagnosis, there is a tendency to use the shortest treatment schedule and the lowest pressure in order to save time should the case not be one of decompression sickness. Rational treatment can only be given if we can identify the criteria by which to select the depth and duration of treatment. Van der Aue, et. al., (1945) used as their selection criteria, two categories, 'bends-pain only' and 'serious symptoms'. The commonly used classification of type 1 and type 2 introduced by Golding, et al. (1960) is a minor modification of the Van der Aue classification. One may try to derive treatment criteria from the outcome of treatment and although this is extremely difficult to interpret, Rivera (1964) found a correlation between early treatment and a successful outcome. Also relevant is the relationship which Doll and Berghage (1967) found between delay in instituting treatment and the pressure subsequently found necessary. Barnard (1965) suggested that success in neurological cases or 'chokes' appeared to be due to early diagnosis and treatment and Bayne (1978) in a series of 46 cases of decompression sickness in United States Navy divers has shown the effectiveness of the Goodman and Workman Tables if diagnosis

is early and treatment thorough. This review suggests that the primary criterion for treatment is whether the case is acute or chronic and only secondarily whether the symptoms, by whatever classification is preferred, are to be regarded as mild or severe.

RATIONAL TREATMENT

Bert (1878) was convinced, as a result of his own experiments in recompressing dogs, that one could ~~but~~ not be certain of cure; since the secondary effects of bubbles upon the tissues might have caused permanent damage. He therefore concluded that industry would have to depend upon preventive measures, such as slow decompression, rather than cure.

If we regard decompression as an exercise in preventive medicine then the cases which we have to treat represent failures of prevention: moreover, the incidence of failure is a index by which we can judge the relative merits of decompression schedules. The degree of inadequacy of a decompression schedule is also judged by the severity of the signs and symptoms produced, unfortunately many case reports give information as to the depth and time of a dive but are not able to give a full description of the decompression used. This, together with the small size of most samples, makes the comparison of different types of treatment carried out in different places and using different criteria of very little value in forming guidelines towards improved treatment.

Without in any way criticizing or underestimating the skill and judgement of the many physicians who have developed effective treatment methods; we should all recognize that these methods are often based upon unproven theories and almost non-existant experimental evidence; moreover, even accepted clinical methods of such as controlled clinical trials have not been used in a discipline

in which individual physicians must often work in professional isolation away from any hospital facilities.

In the course of a discussion on the ethics of hyperbaric research (Young (1973)) Surgeon Vice Admiral Sir James Watt, the Chairman, made the point that the absence of facilities for animal experimentation was no excuse for substituting human experimentation. A review of the literature of the last ten years showed that there were more than 60 papers which could be described as case-reports but only 4 papers which contained experiments relating to the treatment of decompression sickness. If treatment schedules are modified, without prior experiment, physicians may be open to the criticism that they are using patients as experimental subjects. In order to avoid such charges we should attempt both to collaborate in clinical trials and to test in the laboratory the hypotheses relating to treatment. Using existing standard schedules (Barnard (1972)) outlined an approach which is shown diagrammatically in Fig. 1. In summary, acute cases should be compressed to 2.8 atm breathing oxygen from a mask. If relief is not complete within 10 minutes compression is continued to 6 atm using air. If relief is complete after 10 minutes the treatment is completed on the Goodman and Workman schedules. If relief is not complete at 6 atm after a further 30 minutes, treatment is completed on the Royal Navy (RN) equivalent of the Van der Aue table 4 (RN Table 54); whereas if relief has occurred prior to 30 minutes; decompression follows the equivalent to Van der Aue Table 2 (RN Table 52). For chronic cases the evidence seems to favour a more conservative compression, which in the past would have meant taking the patient to greater depths for longer times. In this instance, the treatment of choice, the one which appears to be conservative, is the longer of the Goodman and Workman oxygen tables (RN Table 62) without regard

to whether the symptoms are classified as limb-bends or as serious symptoms.

In summary, the primary method of treatment for decompression sickness is the application of pressure and an important adjunct is the use of oxygen; however the usefulness of physical methods of treatment seems to decline the longer a patient is left untreated, presumably due to the initiation of pathological processes which are no longer susceptible to pressure. It is possible to devise a rational approach to treatment using existing schedules but these may not be optimal and although present evidence is scanty, the approaches being pursued by Albano (1973), Fagraeus et al. (1977) and Fructus, et. al. (1978) may offer treatment superior to existing methods using pressures between 4 and 6 atm, for longer than 2 hours at maximum pressure and using mixtures of oxygen with one or more inert gases.

It is to be hoped that in the near future we will have at our disposal a system of treatment based on sound theory, upon firm experimental evidence, and extensive clinical trials; flexible enough to suit the many different types of case which will continue to occur as a result of our efforts to understand the aetiology of the disease and to achieve its prevention.

TABLE 1

Relationship of the pressure at which symptoms were reported to that found necessary for relief for cases of decompression sickness occurring above 1 atm.

Barnard (1967) Oxy-helium dives and treatments.		
	Mild cases (1)	Severe cases (2)
Mean onset Pressure P_1 (atm)	3.39 ± 1.98 (n = 17)	7.08 ± 3.19 (n = 6)
Mean pressure of relief P_2 (atm)	4.23 ± 2.41 (n = 17)	12.52 ± 3.36 (n = 6)
Mean ratio $\frac{P_2}{P_1}$	1.28	2.23
Kidd and Elliott (1969) Air dives followed by air or oxygen treatment.		
	Mild cases (1)	Severe cases (2)
Mean onset Pressure P_1 (atm)	1.24 ± 0.14 (n = 17)	1.41 ± 0.28 (n = 17)
Mean pressure of relief P_2 (atm)	1.87 ± 0.36 (n = 17)	2.52 ± 0.42 (n = 17)
Ratio $\frac{P_2}{P_1}$	1.51	1.81

TABLE 1 Cont.

Hanson, Vorosmarti and Barnard (1978) Oxy-helium dives and treatments.		
	Mild cases (1)	Severe cases (2)
Mean onset Pressure P_1 (atm)	3.65 $\pm$ 2.35 (n = 30)	4.60 $\pm$ 1.54 (n = 5)
Mean pressure of relief P_2 (atm)	4.32 $\pm$ 2.65 (n = 30)	6.38 $\pm$ 1.51 (n = 5)
Mean ratio $\frac{P_2}{P_1}$	1.29	1.47

TABLE 2

Unpaired 't' test for difference between treatment ratios

	Mild cases (1)	Severe cases (2)
Barnard (1967)	1.28 ± 0.18 n = 17	2.23 ± 1.62 n = 6
	t = 2.47 d.f. = 21 0.05 > p > 0.02	
Kidd and Elliott (1969)	1.51 ± 0.22 n = 17	1.81 ± 0.3 n = 17
	t = 3.36 d.f. = 32 0.01 > p > 0.001	
Hanson, Vorosmarti and Barnard (1978)	1.29 ± 0.44 n = 30	1.47 ± 0.42 n = 5
	t = 0.87 d.f. = 33 not significant	

TABLE 3

Unpaired 't' test for difference between

Δp necessary for treatment

	Mild cases (1)	Severe cases (2)
Barnard (1967)	0.84 ± 0.75 n = 17	5.44 ± 3.34 n = 6
	t = 5.51 d.f. = 21 p < 0.001	
Kidd and Elliott (1969)	0.63 ± 0.29 n = 17	1.11 ± 0.33 n = 17
	t = 4.45 d.f. = 32 p < 0.001	
Hanson, Vorosmarti and Barnard (1978)	0.68 ± 0.67 n = 30	1.78 ± 0.93 n = 5
	t = 1.68 d.f. = 33 not significant	

TABLE 4

Estimates of 95% confidence limits for recompression ratio or Δp (metres) necessary for treatment of decompression sickness occurring at pressure

	Ratio		Δp	
	Mild (1)	Severe (2)	Mild (1)	Severe (2)
Barnard (1967)	1.17-1.39	0.37-4.09	4-12 m	13-96 m
Kidd and Elliott (1969)	1.38-1.64	1.2-1.97	5-8 m	10-13 m
Hanson, Vorosmarti and Barnard (1978)	1.13-1.45	0.89-2.05	4-9 m	5-31 m

TABLE 5

RN Treatment Schedules

(RN Diving Manual HMSO London 1972)

TABLE 61

OXYGEN RECOMPRESSION THERAPY

GAUGE DEPTH (*metres*)	STOPPAGES/ ASCENT (*minutes*)	ELAPSED TIME (*hours and minutes*)	RATE OF ASCENT (*metres/minute*)
18	20 (O_2)	0000–0020	—
18	5	0020–0025	—
18	20 (O_2)	0025–0045	—
18–9	30 (O_2)	0045–0115	3 m in 10 mins
9	5	0115–0120	—
9	20 (O_2)	0120–0140	—
9	5	0140–0145	—
9–0	30 (O_2)	0145–0215	3 m in 10 mins
Surface		0215	

TABLE 62

OXYGEN RECOMPRESSION THERAPY

GAUGE DEPTH (*metres*)	STOPPAGES/ ASCENT (*minutes*)	ELAPSED TIME (*hours and minutes*)	RATE OF ASCENT (*metres/minute*)
18	20 (O_2)	0000–0020	—
18	5	0020–0025	—
18	20 (O_2)	0025–0045	—
18	5	0045–0050	—
18	20 (O_2)	0050–0110	—
18	5	0110–0115	—
18–9	30 (O_2)	0115–0145	3 m in 10 mins
9	15	0145–0200	—
9	60 (O_2)	0200–0300	—
9	15	0300–0315	—
9	60 (O_2)	0315–0415	—
9–0	30 (O_2)	0415–0445	3 m in 10 mins
Surface		0445	

TABLE 52

AIR RECOMPRESSION THERAPY

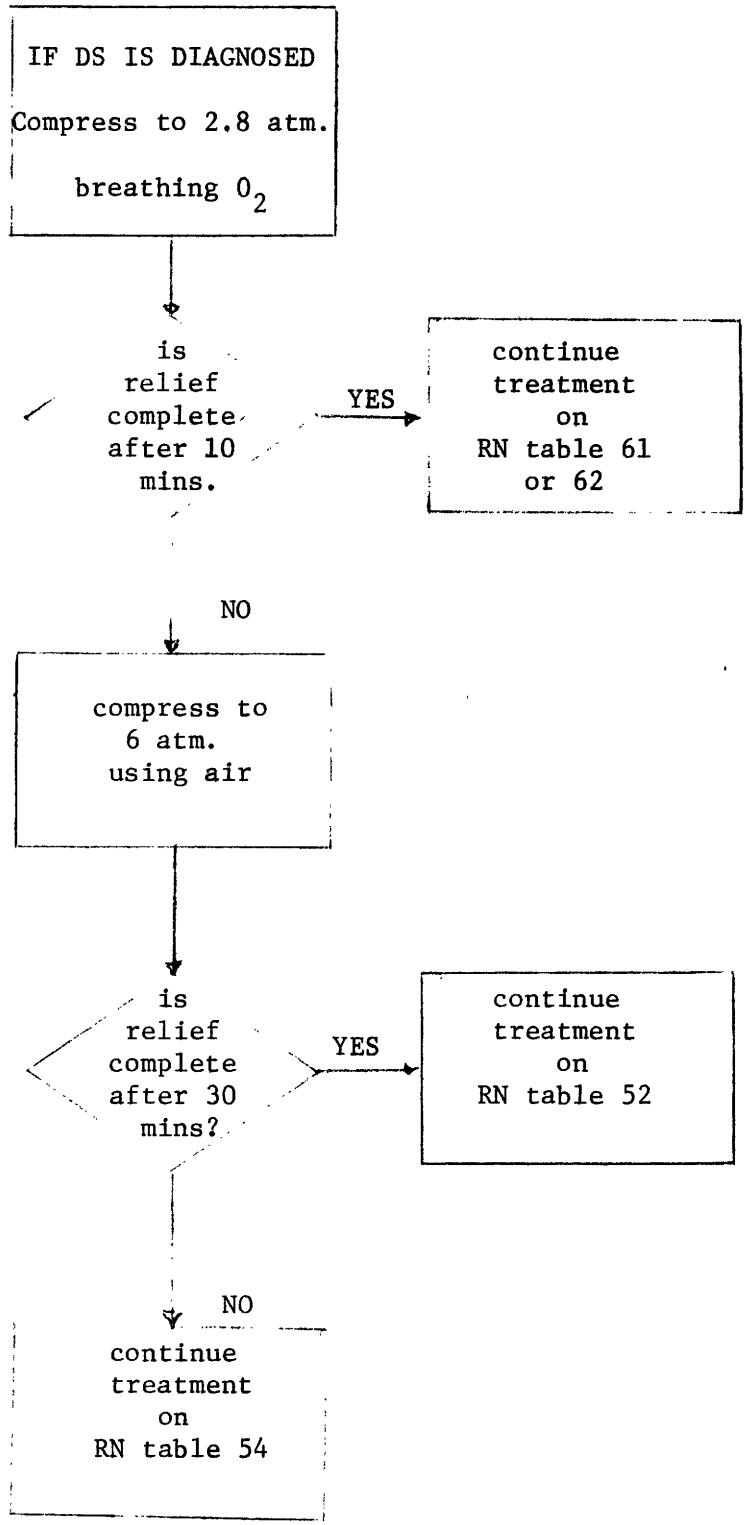
GAUGE DEPTH (*metres*)	STOPPAGES (*hours (h) and minutes (min)*)	ELAPSED TIME (*hours and minutes*)	RATE OF ASCENT
50	30 min	0000–0030	5 minutes between stoppages throughout
42	12 min	0035–0047	
36	12 min	0052–0104	
30	12 min	0109–0121	
24	12 min	0126–0138	
18	30 min	0143–0213	
15	30 min	0218–0248	
12	30 min	0253–0323	
9	2 h	0328–0528	
6	2 h	0533–0733	
3	2 h	0738–0938	
Surface		0943	

TABLE 54

AIR RECOMPRESSION THERAPY

GAUGE DEPTH (*metres*)	STOPPAGES (*hours (h) and minutes (min)*)	ELAPSED TIME (*hours and minutes*)	RATE OF ASCENT
50	2 h	0000–0200	5 minutes between stoppages throughout
42	30 min	0205–0235	
36	30 min	0240–0310	
30	30 min	0315–0345	
24	30 min	0350–0420	
18	6 h	0425–1025	
15	6 h	1030–1630	
12	6 h	1635–2235	
9	11 h	2240–3340	
	1 h (02)	3340–3440	
6	1 h	3445–3545	
	1 h (02)	3545–3645	
3	1 h	3650–3750	
	1 h (02)	3750–3850	
Surface		3855	

Figure 1.



Flow diagram for the use of the Royal Navy treatment tables for acute cases of decompression sickness (after Barnard 1972).

(vid. Table 5)

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DRUGS FOR THE TREATMENT OF DECOMPRESSION SICKNESS

by

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INTRODUCTION

Therapeutic compression using oxygen enriched mixtures or pure oxygen is still the main factor in treating decompression sickness. However, the symptoms associated with the presence of gas bubbles and which constitute decompression sickness can be treated by drugs.

This drub therapy is difficult to classify, since decompression sickness is very polymorphous, there are few cases and each treatment centre's experience of each clinical form of the sickness is limited.

Few clinical tests have been carried out and the real effectiveness of the various substances is consequently difficult to assess.

Treatment patterns are frequently based on the approaches of different schools of thought.

The selection of symptomatic medicines has been based mainly on physio-pathological data obtained for the most part from animal experiments. Delving a little into the historical background reveals that medical conclusions came relatively long after the physio-pathological discoveries : platelet and gas bubble interaction (the existence of platelets around the bubbles and of free platelet aggregates) was discovered as early as 1942 (Jacobs and Stewart), yet the first therapeutic trials on anti-aggregating agents were not conducted until over 20 years later.

For a very long time the only recognized treatment was therapeutic compression in air.

The beginning of drug treatment dates back to 1961 with the use of heparin by Barthélemy and Laborit.

Subsequently, therapeutic trials have proliferated and great strides have been made in the past five years.

Since 1976, however, the body of experience built up has meant that one can choose from a large number of available medical products and at a meeting of the EUBS (European Undersea Biomedical Society) held in London in February 1976, the usefulness of many products was questioned.

The purpose of this paper is to take stock of how the problem has evolved since this 1976 meeting.

PHYSIO-PATHOLOGICAL DATA

Here is a brief reminder of the now familiar physio-pathological pattern of decompression sickness.

The very presence of gas bubbles in the blood will hamper micro-circulation which is of capital importance particularly to the spinal cord (disorders due mainly to venous stasis), to the lungs (the accumulation of bubbles halted by the pulmonary filter impedes alveolo-capillary diffusion), and to the brain (gas bubbles in the arteries and arteriolae).

The result of this microcirculation obstruction is tissular anoxia.

However, these disorders are severely compounded by the phenomena arising from the existence of blood/gas interfaces around the bubbles.

Plasma protein molecules (albumin, fibrinogen, γ-globulin) adhere to these interfaces by means of their hydrophobic poles. These plasma proteins will become denatured, while the plasma lipids also adhere to the bubbles or produce fat embolisms.

The blood platelets adhere to the bubbles and also to the lesions of the vascular endothelia caused by the irruption or passage of micro-bubbles. Platelet aggregates will fall away, circulate and, in conjunction with the red blood corpuscles, result in widespread intravascular coagulation. The aggregated platelets then salt out their components : serotonin, histamine, ATP, ADP, lysosome/enzymes and phospholipids.

Platelet phospholipids activate the Hageman factor, resulting in hypercoagulation.

Kininogens are activated in kinin, including bradykinin. A plasma factor also originates : the SMAF (smooth muscle active factor).

The SMAF, serotonin and bradykinin lead to bronchostenosis and also vasoconstriction, which aggravates microcirculatory disorders.

These various plasma components also lead to increased vascular permeability in the form of a plasma loss, hypovolaemia and an interstitial oedema (in the lungs and mainly in nerve tissue). In the event of serious neurological damage, shock occurs which differs only slightly in its symptoms from, for example, septic shock.

Loss of plasma and hypovolaemia merely aggrevate microcirculatory disorders which generate cellular anoxia.

One symptom thus seems to prevail over the others : microcirculatory disturbance.

PRESENT BASIS OF DRUG THERAPY

This symptomatic treatment should comprise :

- re-establishment of the blood volume,
- prevention of interstitial oedema,
- anti-aggregating treatment,
- hypocagulating treatment,
- vasodilators.

Lastly, an agent is added to forestall nervous oxygen toxicity since high partial pressures are used in recompression.

It has now become accepted practice to begin this treatment at the scene of the accident.

Perfusion of a plasma expander solution is therefore carried out, the most common being low molecular weight Dextran or Dextran 40.

A corticosteroid which can be injected in large doses is used to prevent shock and oedema. For instance, 10 - 12 mg of Soludecadron is used or 1 g of Hydrocortensyl intravenously.

Aspirin is widely used in France as an anti-aggregating agent. In the event of serious neurological damage, 1 g of lysine acetylsalicylate can be injected intravenously.

If the accident is less serious, ingestion of 1 g of aspirin dissolved in a 1/4 l of water may suffice.

An anticoagulant will be used at this stage of treatment only if transportation to a hyperbaric treatment centre is slow (over two hours) ; in this case heparin is administered subcutaneously.

During transportation to the hyperbaric centre, perfusion of 500 ml of Dextran continues while the patient breathes pure oxygen at normal atmospheric pressure. At the hyperbaric centre, these various forms of treatment are continued or resumed simultaneously with recompression.

A vasodilator and also diazepam (valium) can be added. Intravenous heparin can be commenced or can be substituted for subcutaneous heparin after monitoring the situation as regards coagulation.

Admittedly, these methods of treatment have not always conclusively proved their effectiveness on decompression sickness and the results are

very often given the benefit of the doubt.

APPRAISAL OF PRESENT TREATMENT METHODS

The various forms of therapy should correspond to physio-pathological knowledge and their effectiveness - or, at least, their harmlessness - should have been clearly established.

On the basis of these criteria, we shall now take a look at the various products proposed.

1. Plasma expander solution

A normal blood volume must be restored if microcirculation is to be re-established.

In order to achieve this, the main products at our disposal are :

- Dextrans (of the Rheomacrodex type)
- Gelatin plasma volume expander (Plasmion, Haemacel)
- Electrolytic solutions (lactated Ringer's solution).

Pure electrolytic solutions cannot be justified since they remain only a short time in the vascular system and migrate towards the extravascular system where they could provoke oedema.

Gelatin plasma volume expanders increase the plasma volume but only by their own volume, as their oncotic potency is low (comparable to that of plasma). They possess a lower viscosity than Dextrans. Migne (Anesthésie-Analgésie-Française 1976, pp. 33-34) considers that they do not affect coagulation, although certain authors have shown that Haemacel intensifies coagulation. They do not act as anti-aggregating agents.

Dextrans have an average molecular weight of between 30 000 and 80 000. Dextran 40 (Rheomacrodex), which has a molecular weight of 40 000, is the most commonly used in France.

Dextrans have a higher oncotic potency than gelatins and the greater their molecular weight the higher the potency. Consequently, for greater extravascular water inflow and longer-lasting action a high molecular weight (70 000 - 80 000) Dextran (Dextran 70 or Dextran 80), which has a high oncotic potency and which passes at least through the capillary membrane, is in theory to be recommended. Experiments have shown that following local administration of bradykinin or histamine, the capillaries remained impermeable to high molecular weight Dextrans while the others passed through easily.

The Dextran solution has a greater viscosity than a gelatin expander solution and this could constitute one drawback to using Dextran. In actual fact, however, it is not really a problem since the plasma dilution achieved by Dextran is greater and viscosity is fairly low.

Dextrans have been criticized as harmful to kidney function ; however, there is a risk only if kidney function is poor, which is not the case of divers, who are young and healthy adults.

Lastly, Dextran offers a considerable advantage in the event of decompression sickness, in that it has a marked anti-aggregating action. It decreases platelet sensitivity to ADP (in patients suffering from thromboembolism and undergoing continuous treatment). Dextran also reduces the adhesiveness of platelets, brings down the plasma rate of factor VIII and fibrinogen without increasing bleeding time (Childs, Ah-See, Arfor, 3rd EUBS Congress 1977). Nevertheless, the dose must be lower than 1.5 g/kg weight, i.e. 1.5 l of Dextran 70.

Dextran has proved its effectiveness both in experiments (re-establishing micro-circulation in animals suffering from decompression sickness) and in the treatment of humans (Cockett's observations).

We would therefore opt for Dextran as an expander solution because of its high oncotic potency and its anti-aggregating properties. In practice, choosing between Dextran 40 and Dextran 70 is somewhat academic since Dextran 40 solutions have a concentration of 10 % and Dextran 70 solutions 6.5 % (the stronger concentration in the case of the former offsets its slightly lower oncotic potency).

Administering excessive amounts of Dextran must be avoided, as this could aggravate the pressure in pulmonary circulation and the stasis upstream. The volume used must be in proportion to the gravity of the accident.

A slow intravenous injection of 500 ml of Dextran the first day is, in our opinion, appropriate for decompression sickness of average gravity and can be administered systematically even at the first aid stage if there are neurological symptoms.

A recent paper at the annual scientific meeting of the Undersea Medical Society held in Seattle in May 1978 highlighted the usefulness of introducing an expander solution in conjunction with recompression treatment, irrespective of the type of expander solution used.

Wells and coworkers have studied the effects of recompression alone, of an expander solution alone, or of the two combined, in re-establishing microcirculation in dogs suffering from decompression sickness. The results show that recompression alone has very little effect, and 7 cc/kg of fluid injected intravenously re-establishes microcirculation if administered in tandem with recompression, while 400 cc/kg are necessary if administered in isolation. However, there are no indications as to whether a colloidal solution is preferable to a simple electrolytic solution of the lactated Ringer type.

2. The use of large doses of corticosteroids

This has become an accepted part of the treatment of decompression sickness. Nevertheless, the effectiveness of corticosteroids in treating shock is apparently questioned. In the case of septic shock, for example, Robin and Le Gall (Revue du Praticien 1975, 25, 11, 841, 848) consider that the beneficial effect of the corticosteroids injected in large doses is by no means clearly established. They write that : 'in series properly examined no significant improvement was noted in cardiac output, arterial pressure, peripheral resistance, or terminal congestive heart failure'. They note that corticosteroids are effective in protecting lysosome membranes when they are injected in advance as a preventive measure, but have no effect once shock has occurred.

Moreover, Darragon and coworkers (in 'Réanimation et Médecine d'Urgence, 1976'ed., l'Expansion scientifique, 1 vol. Paris, pp. 51-56) observe that much -and often controversial- material has been written on the use of corticosteroids in the treatment of shock, leading to contradictory results, and that hormonotherapy as a substitute in treating shock would be useful only in the event of acute adrenocortical insufficiency. The beneficial effects using strong doses has been clearly established only as a preventive measure since it protects the cell and lysosome membranes. Its effects on survival in man have never been proved by random investigation.

On the other hand, the side effects of using strong doses of corticosteroids are known (depression of the immunization mechanisms, aggravation of cardiac arrhytmia and necrosis during hyperadrenalism). Consequently, there seems to be no justification for maintaining strong doses of intravenous cordicosteroids in the treatment of decompression sickness.

3. Anti-aggregating agents

There is doubt here, too, as to the real effectiveness of the anti-aggregating agents known at present.

Naturally, it is desirable to prevent platelet aggregation (which aggravates microcirculatory disorders) and especially to stem the dissemination of the aggregates. The four most familiar anti-aggregating agents are aspirin, dipyridamole and its derivatives, clofibrate and sulfinpyrazone (or Anturane).

In vitro tests on platelets illustrate the action of these agents. Tests carried out ex vivo, i.e. on blood samples taken from patients treated using a substance considered to be anti-aggregating, do not provide a clear-cut illustration of their action. Monitored random tests carried out as part of the ultimate yardstick -clinical appraisal - have so far failed to provide solid proof of the effectiveness of the substances in question.

In the case of decompression sickness induced in animals and in human subjects, certain products have revealed some degree of effectiveness

with regard to the fall in the number of platelets. In particular, Philp and Ackles have successfully tested RA 233 and VK 744, derivatives dipyridamole. For our part, we have successfully tested a Diamicron derivative on rats.

On the other hand, experimental tests with aspirin which is widely used in France, have proved negative on animals (Broussole) and on man (Philp). Nevertheless, clinical results in the treatment of decompression sickness appear to be very good in a study of 100 such cases presented by Wolkiewiez at the Journées Méditerranéennes des Accidents et du Trafic in Nice (June 1978). It would seem that the usefulness of aspirin in treating decompression accidents can therefore be upheld.

It should be administered preferably in a form having a pH buffer or in alkaline form (Aspegic). In any case, there will be a beneficial anodyne effect.

New generations of anti-aggregating agents, in particular those derived from prostaglandin X (PGX) are being developed.

Studies should be undertaken on these powerful anti-aggregating agents, since there is a potential risk. Preventive treatment carried out on miniature swine led to earlier and more numerous osteoneocrosss (Stegall, Slichter, Smith, Hacker, Circulation vol. 49 50 1974, III 286). The onset of osteoneocrosis is even more rapid when an anti-aggregating agent and an anticoagulant agent are at work together. Administering the anti-aggregating agent as a preventive measure hampered the physiological role of the platelets which is to fill in the little gaps in the capillary endothelium, particularly in the bony tissue, since these vascular gaps are thrombogenic.

4. The use of anticoagulants

Heparin has been used in the treatment of decompression sickness since Barthélémy's first observations. He demonstrated that this treatment had no dangerous side effects, but reduced the gravity of decompression sickness. Its effectiveness could be connected with its anticoagulant action, but also with its role as a plasma clearing factor (anti-lipaemic) and to its vasodilatory, anti-exudative and anti-adrenergic properties.

Philp's experience proves that it is anti-lipaemic factor which is at work. Administering the substances to rats suffering from decompression sickness, he compared the effects of heparin, an anticoagulant (dicoumarin), and an anti-lipaemic agent (PDHA).
The results showed that dicoumarin had no effect (although the prothrombin rate was multiplied by 5, whereas the other two products had the same degree of effectiveness.

In our view, the use of heparin in anti-lipaemic, active and safe doses can be retained until very precise clinical experiments prove otherwise. Subcutaneous administration of heparin (a 0.25 g dose of Calciparine) is, in our opinion, advisable at the first aid stage in the event of a serious case of decompression sickness if transportation to the hyperbaric centre takes over two hours.

5. Use of vasodilators

The physiopathological arguments in favour of their use are very convincing.

Prof. Le Mouël (Toulon) considers the clinical results (not yet published) of treatment of barotrauma of the internal ear to be very good.

Nevertheless, the use of this treatment in identical cases was criticized at the London meeting in 1976 by Farmer : he maintained that it was not certain that vasodilation of the face did not entail vasoconstriction of the internal ear as a result of irregular circulation. No experiments have so far proved or disproved the effectiveness of vasodilators in this case. A series of experiments are planned at the CERB in Toulon in conjunction with the ENT Department of the Hôpital d'Instruction des Armées Sainte-Anne (Prof. Le Mouël).

Generally speaking, only very active vasodilators can be injected intravenously and can be used only under strict haemodynamic monitoring by an intensive coronary care department. Vasodilators are arterial dilators (e.g. pentolamine or hydralizine), venous dilators (nitroglycerin) or mixed (sodium nitroprusside). All these products are dangerous to use in a pressure chamber, even in a hyperbaric centre.

The most common vasodilators, the effectiveness of which is somewhat unclear and the precise site of action unknown, should not be used in treating decompression sickness.

However, increasing importance is being attached to nitroglycerin as a venous vasodilator (since it rapidly lowers pulmonary capillary pressure and decreases pulmonary oedema). Despite the fact that it requires stringent haemodynamic monitoring, there is no doubt that research into the effects of nitroglycerin in treating decompression casualties should be carried out.

6. Use of diazepam

This is undoubtedly the substance which offers the best protection against the convulsions provoked by hyperoxia.

The risks of convulsions at 2.8 ATA oxygen must be known ; they are overlooked in some quarters, but tests have been carried out in others.

No risk is involved in using diazepam (valium). At most it will tranquillizethe patient and provide him with better protection against a hyperoxic crisis. It should therefore be on hand in the hyperbaric centre when high partial pressures of oxygen (e.g. above 2.5 ATA) are used.

CONCLUSIONS

Which drugs are left and which should be retained for treating decompression sickness where the patient is hyperoxic and undergoing recompression ?

The expander solution (Dextran in particular) is the most important part of this treatment, since it can re-establish microcirculation which, when adversely affected, can lead to anoxia. Dextran acts simultaneously as a plasma expander, an anti-aggregating and an anti-lipaemic agent. It should be administered in moderate doses (500 ml/day).

None of the anti-aggregating agents known at present (apart from aspirin, thanks to apparently beneficial clinical results), vasodilators or corticosteroids administered in strong doses, have proved their effectiveness and harmlessness and their use should, in our opinion, be discontinued. On the other hand, heparin (0.25 g Calciparine subcutaneously at the first aid stage or 500 mg/day heparin intravenously) should be retained.

Valium should also be used in the hyperbaric centre.

However, further research on anti-aggregating agents and vasodilators is needed.

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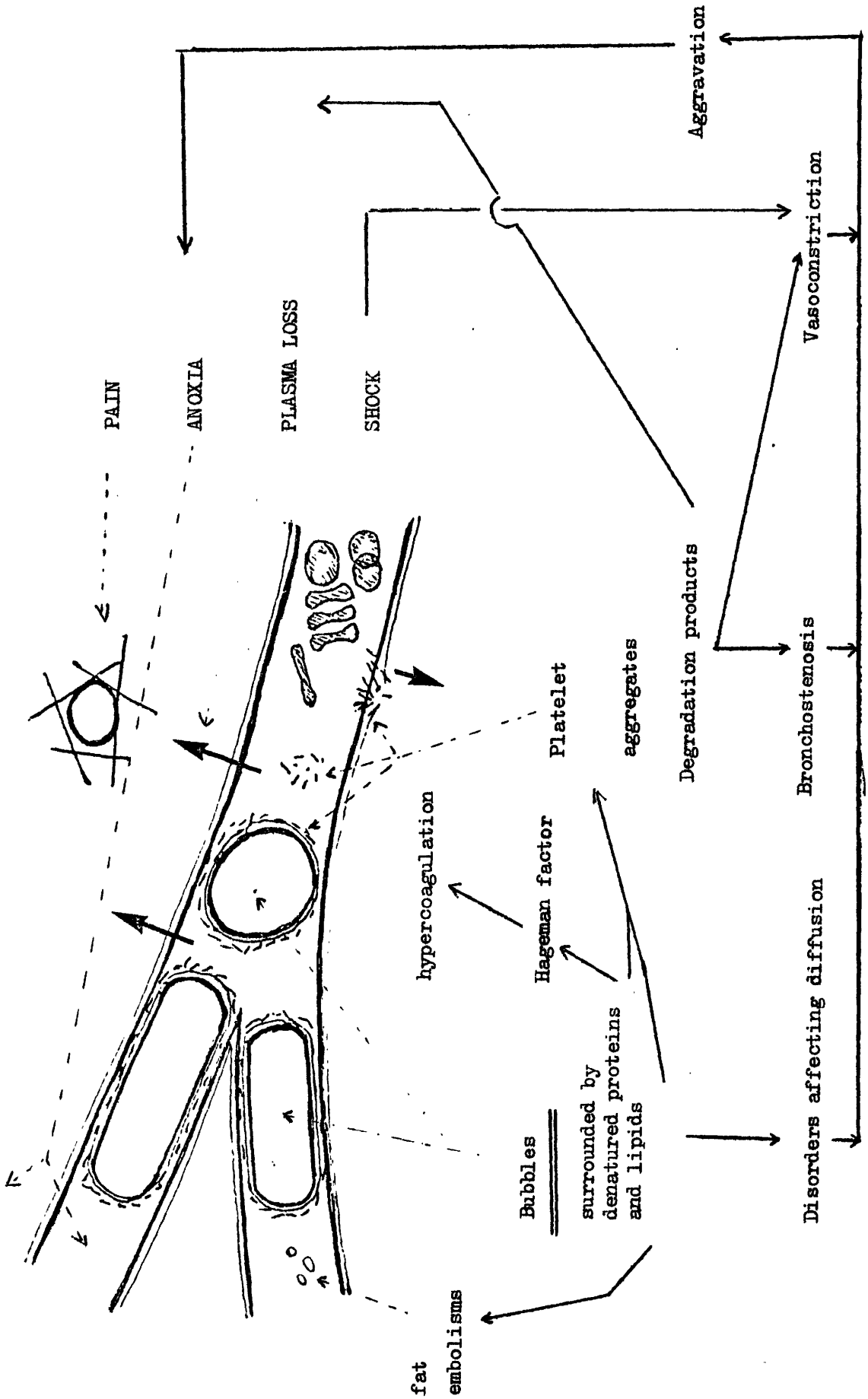
PAIN
ANOXIA
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Degradation products
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Hageman factor
Bubbles
surrounded by
denatured proteins
and lipids
Disorders affecting diffusion
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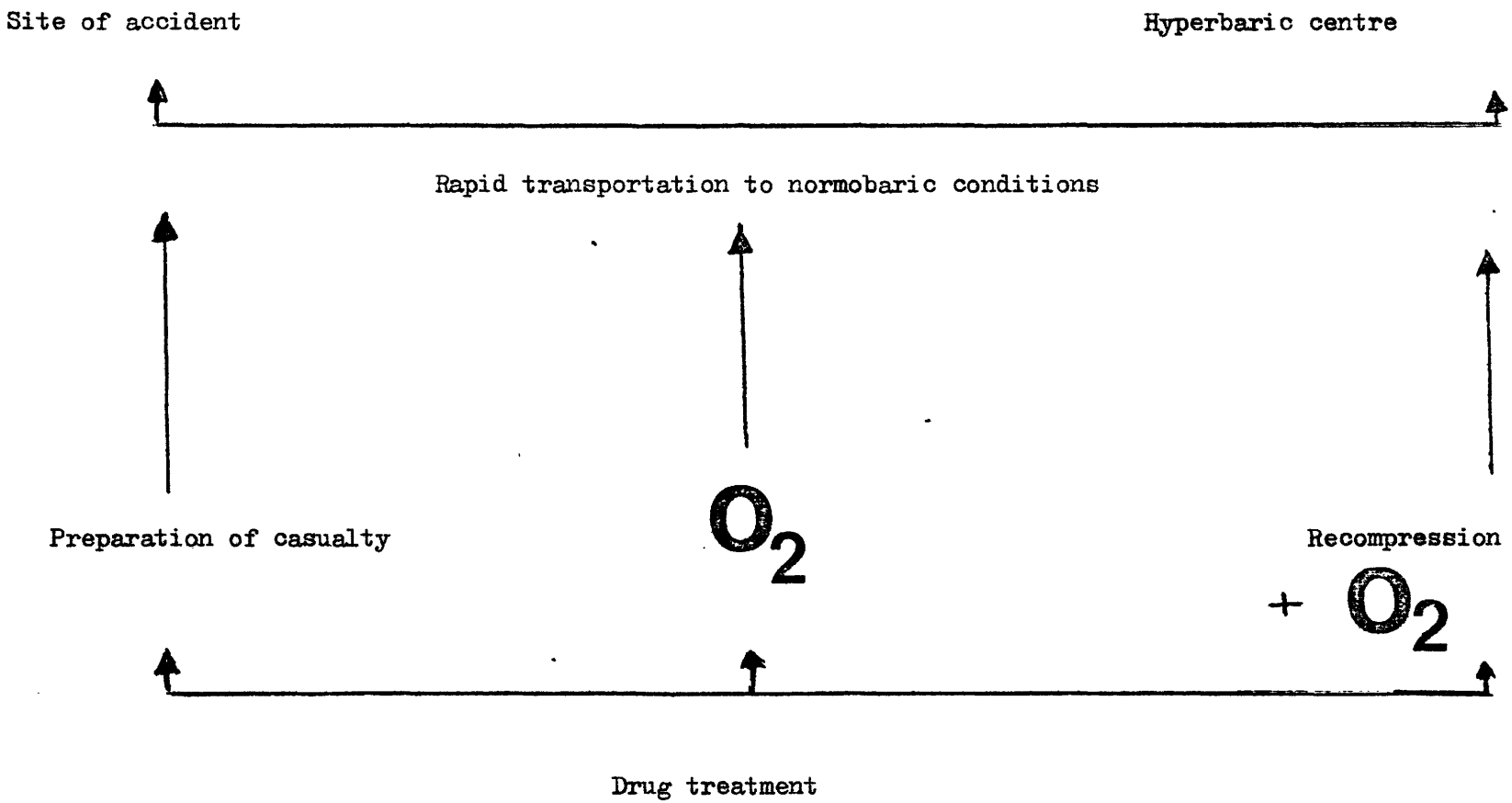
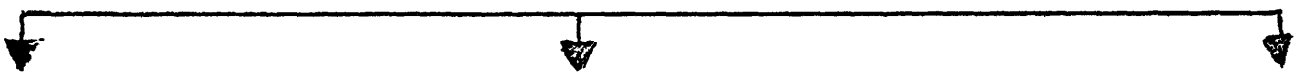

Site of accident
Hyperbaric centre
Rapid transportation to normobaric conditions
Preparation of casualty
O_2
Recompression
+ O_2
Drug treatment

DRUG TREATMENT



FIRST AID

expander solution
corticosteroids
subcutaneous heparin
anti-aggregating agents?

TRANSPORTATION

expander solution

HYPERBARIC CENTRE

Immediate treatment

expander solution
corticosteroids
heparin
vasodilators
anti-aggregating agents?
valium
other treatment

Follow-up treatment

ANAESTHESIA IN A HYPERBARIC ATMOSPHERE

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ANAESTHESIA IN A HYPERBARIC ATMOSPHERE

INTRODUCTION

Anaesthesia and hyperbarism have been closely linked from the start. Just a century ago, Paul Bert drew attention to the possibility of achieving protracted insensibility by increasing the ambient pressure and using nitrous oxide, and, as early as 1879, in Paris, Fontaine had a hyperbaric chamber built in which he could perform surgical operations.

Since the development of saturation diving and work on the seabed entail the risk of accident or illness possibly requiring surgical measures at depth, can anaesthetic techniques keep abreast of the substantial progress made by diving?

We shall first define the scope of our study, after which we shall examine the material, pharmacological and physiopathological implications of life at pressure which could modify the techniques of anaesthesia.

Part 1: THE SCOPE OF THE STUDY

I - Anaesthesia is not particularly easy to define (67).

Etymologically, it means the loss of sensation and its prime objective is to suppress the feeling of pain.
Anaesthesia can be achieved by direct and local medicinal depression of nerve conduction: this involves the various techniques of local, loco-regional or spinal (block) anaesthesia.
It can also be induced by drugs which have a more general effect on the organism as a whole and in particular on the central and peripheral nervous system; this is general anaesthesia. This does not necessarily imply a state of unconsciousness. Modern anaesthetic techniques include all means - we shall, however, avoid discussing electrical anaesthesia and anaesthesia induced by acupuncture or hypnosis - which enable a patient to undergo an 'aggressive', usually surgical, intervention without damage or discomfort, and to derive benefit therefrom, while giving the surgeon the maximum

freedom to perform his task. This preparation makes it necessary to choose between a variety of therapeutic actions to shut off:

1 - all feeling, particularly the feeling of pain: this is analgesia;
2 - all reflexes by seeking to induce a state of neurovegetative calm: this is neurovegetative protection;
3 - all muscular tonus in order to achieve 'muscle relaxation';
4 - and lastly, all consciousness by seeking to induce either sleep ('narcosis') or at least a certain imperturbability as regards the operation undergone (ataraxia). In order to shut off these various reactions a single drug ('general anaesthetic') or a number of more elective drugs in combination, e.g. analgesics, neuroleptics, curares, narcotics, etc. may be used. These drugs can be introduced into the organism via the lungs (anaesthesia by inhalation), parenterally (usually intravenous) or via the the digestive system.

II - Hyperbarism at first sight appears easier to define: it begins when the ambient pressure is above 1 bar!

In actual fact, the concept of hyperbarism is essentially a biological one (25): a pressure is hyperbaric for a living being when it is above the local atmospheric pressure at which this being is in a state of equilibrium. In practice, however, in the very general case of low altitudes, hyperbaric conditions begin when the ambient pressure is above 1 bar, 'pressure at sea level'.

For our purposes here, a distinction seems necessary between the following:

1 - moderate hyperbaric conditions (up to 3 - 4 ATA) in respect of which there exists a long-standing experience in the field of anaesthesia (50);
2 - high pressures (up to 20 - 25 ATA): progress in diving techniques make it essential as of now to study the possibilities of anaesthesia at these depths;
3 - very high pressures: although we are still at the stage of experimenting with animals, the evident interaction between the effects of anaesthesia and those of pressure is of great interest to both anaesthesiologists and to diving specialists.

III - Anaesthesia at hyberbaric pressures may be advisable in several circumstances:

1 - First, to enable a surgical operation to be carried out on a sick or injured person in case of necessity. It may be necessary to maintain the hyperbaric atmosphere in order to operate on an injured diver, in which case the medical team will be required to descend to his level (46). Let us take another example: since hyperbarism can facilitate surgical or other treatment, the creation of a hyperbaric atmosphere may prove useful. In this way, for instance, hyperbaric oxygen treatment can be applied so as not to hold up the debridement of a gas gangrene or to permit more prolonged circulatory arrests during heart surgery as Boerema did in Amsterdam in 1956.

2 - A second circumstance is of historical interest: hyperbaric pressure can facilitate the use of a mild gaseous anaesthetic by permitting high partial pressures of this anaesthetic without risking hypoxia.
In this context one thinks of Paul Bert who, as early as 1878, drew attention to the "possibility of achieving protracted insensibility using nitrous oxide ... and the harmlessness of this anaesthetic".
Xenon, a gas which is comparable from the point of view of its anaesthetic potency, was used in identical circumstances by Pittinger in 1955 (59).

3 - Lastly, the scope of this study can be broadened to consider other uses of drugs which traditionally were used in anaesthesia, e.g. to forestall or reduce the toxic effects of the high partial pressures of oxygen or the onset of high pressure nervous syndrome. For our purposes, i.e. principally anaesthesia for surgical purposes, we shall simply mention these problems in passing.

Part 2: EFFECTS ON THE EQUIPMENT NEEDED FOR ANAESTHESIA

The equipment needed to administer anaesthetics or to ensure safety during the operation will, once introduced into a hyperbaric chamber, be subjected to a rise in ambient pressure and this could affect its performance (31 - 59 - 69 - 74).

These effects arise mainly from the application of the physical laws governing gases.

I - The laws of the compressibility of gases of Boyle, Mariotte, Charles and Gay Lussac:

Caution must be exercised with regard to variations in the volume of the bags on the end of endotracheal tubes or self-retaining catheters, of the permanently inflated rims of certain anaesthetic masks, and also with regard to certain surgical drains. The bags should be filled preferably with a non-compressible fluid, a liquid e.g. isotonic saline serum.

There are certain technical requirements for carrying out perfusions: there must be air intakes above the upper limit of the liquid; care must be taken at the decompression stage to avoid the risk of air embolism originating in the drip bottles.

Similar precautions are necessary as regards the use of bottled substances whenever these are vacuum sealed.

When performing spinal anaesthesia, it is essential to avoid introducing air into the spinal canal; in the case of peridural anaesthesia (49), the liquid madrin technique should be used to reach the peridural space and a catheter should be left in position during decompression in the interests of safety.

II - The laws governing the dissolution of gases in liquids

Gases dissolve in liquids, the quantity being subject to Henry's law and speed to Haldane's laws. The quantity dissolved in an organism can be substantial depending on the partial pressures of the gases and the duration, irrespective of whether these gases are respiratory or anaesthetic.

In order to forestall untimely ill-effects on the operating team (27) working inside the pressure chamber, it is necessary to study the problem of ventilating the hyperbaric chambers and eliminating nuisances, particularly the gases exhaled by the person under anaesthetic (oxygen - CO_2 - water vapour and anaesthetic gases or fumes).

When gaseous or volatile anaesthetics are used, it is considered that the minimal ventilation inside the chamber should be raised from 85 litres/minute and per person present (5 m^3/hour) to 285 litres/minute/person (17 m^3/hour), which would be enormous in terms of both quantity and cost (25 - 87).

A simple solution to this problem is to use an "inhaler-exhaler" enabling the gases exhaled by the patient to be vented outside the working area. This exhaler should be compatible with direct connection to, and the expiratory flow rate of, an artificial respirator.

III - The density of gases increases under pressure: these changes in density will modify their resistance to the flow of the fluids and also the readings of certain measuring instruments. They also favour turbulence, in which case flow resistance no longer depends on viscosity, which is unaffected by hyperbaric pressure, but on the density of the fluid: roughly speaking, the resistance is proportional to the square root of the density. In addition, the anaesthetist must take account of the different ways of expressing a gaseous flow, either in "molar flow" (number of molecules per unit of time, expressed in volume-normobar/minute), i.e. the volume which the gas would occupy at a pressure of 1 bar, or in "volume per unit mass" or in "voluminal flow".

The resistance to the flow of the gases in the anaesthetic circuits will rise, which in turn will increase in the same proportion the ventilatory effort of a patient breathing spontaneously.

The setting of the flow meters will be altered, particularly the rotameters: calibrated for 1 ATA, they show, when under pressure, readings higher than the true values (31).

The same is true of the calibration of certain vane-type respirometers, particularly the Wright model.

On the other hand, it is significant that the pressure reducers are unaffected when they are situated inside the pressure chamber, whether their valves are upstream or downstream, unless, of course, the feed pressure falls below the ambient pressure in the operating area.

Suitable artificial respiration requires ventilation which is constant volume-wise, which presupposes a molar throughput proportional to the pressure.

One solution is to place the respirator outside the pressure chamber (51 - 52). The simplest solution, however, is to use suitable respirators inside the chamber with controls within easy reach of the anaesthetist.

We generally use the Celog 2, a model which adopts a system of distribution and pneumatic timing based on the use in combination of

computerized pneumatic cells. In these circumstances, however, its performance is modified and imposes limitations on its use on adults at 4 and even 3 ATA (16).

The Bird Mark 7 pressure reducer has been tested in our department (22). A relative throughput pressure of 3.5 bars must be kept up in order to maintain, for a given initial calibration, constant tidal volumes and inspiration timing at hyperbaric pressure. In order to maintain a frequency and a constant I/E ratio it is necessary to adjust the wheel which regulates the expiratory pause. The use of this respirator is restricted to hyperbaric atmospheres of 3 ATA maximum.

The respirator pressure reducer is of great theoretical interest since the tidal volume is determined by the height of a mobile tray; this height cannot be changed by the pressure. The throughput must, of course, remain at 3 bars relative and the modified values of the flow meters must be taken into account (26).

As far as I know, respirators have not been tested at above 4-5 ATA and in a helium atmosphere.

The quality of the gases supplying these respirators (pure oxygen or gaseous mixtures) will be determined by the conditions of their use and according to the toxicity of the high partial pressures of oxygen (36).

IV - The combustibility of a material increases as the partial pressure of an oxygen atmosphere increases.

Most materials become combustible in pure oxygen under pressure.

Nitrous oxide under pressure becomes an excellent comburent. The limits of inflammability of certain anaesthetic gases or vapours can be lowered. Certain anaesthetics considered as non-flammable can become inflammable under pressure (32-59).

The risk of ignition and explosion means that all fatty substances (even for dressings), grease and oil must be eliminated; the hydraulic fluid of certain operating tables must also be examined. Silicone is a possible alternative. Electric bistouryes, electric motors and cauterization systems must not be used; no source of static electricity can be tolerated (e.g. certain plastics, certain synthetic fabrics) (1-87).

Endotracheal intubation can also give rise to certain problems unless the system of illuminating the blade uses cold light.

V - Effects on the evaporators

It is known that the pressure of saturated vapour varies as a function of temperature but is not affected by ambient pressure; it does not follow the ideal-gas laws (24).

As the partial pressure of saturated vapour is thus independent of barometric pressure, the PP/BP ratio moves towards 1 when the ambient pressure drops (in altitude); the reverse occurs in a hyperbaric atmosphere.

At 20°C the saturated vapour pressure of halothane is 243 mmHg, which means that its 'concentration' at 1 ATA is therefore around 32%, but only 10.7% at 3 ATA. Naturally, this simple example proves that all calculations must be based on partial pressures and that all calibrations on a percentage basis, which could be misleading, must be abandoned.

It should also be pointed out that when a gaseous current removes the saturated vapour from the surface of the volatile liquid, this vapour reforms but since this forced evaporation has disturbed the equilibrium, a certain quantity of heat - known as latent vaporization heat - has to be supplied

Otherwise, the temperature of the volatile liquid drops, the corollary of which is a change in the evaporation conditions and a fall in the saturated vapour pressure. This explains why thermostatically controlled calibrated evaporators have been developed for anaesthesia purposes.

In theory, therefore, the quantity of anaesthetic supplied by an evaporator of this kind will remain the same whatever the ambient pressure (calculating in terms of partial pressure and not of percentage!). In actual fact, experiments carried out in a pressure chamber have revealed appreciable differences in the performance of Fluoteo type calibrated evaporators. The reasons are far from clear (31 - 72).

Any evaporator intended for use in performing 'volatile anaesthesia' in a hyperbaric chamber should therefore be tested, even though the monitoring of the clinical symptoms remains the prime factor in carrying out these anaesthesias (61).

VI - Other problems involved in carrying out anaesthesia in a hyperbaric atmosphere (69)

- a source of aspiration. Electrical aspirators must not be used. A portable aspirator equipped with a foot-operated portable Ambu could be used, but is inadequate and unsuitable for the purposes of an operation. Aspirators using the Venturi system will in a pressure chamber consume an inordinate volume of gas. A fairly simple solution is to exploit the difference in pressure between the inside of the chamber and the external barometric pressure using a sluice valve; a flow meter can regulate the throughput and therefore the aspiration power; the liquids aspired can be collected in an intermediate bottle;
- monitoring, particularly ECG and EEG, is often necessary when working in an oxygen flow, since 'anaesthetic' and 'tranquilizer' drugs can mask convulsions caused by hyperoxia. The measuring instruments remain outside the pressure chamber; there are connecting panels for banana plugs which are situated inside the operating area and connected to the outside by an armoured cable.
- blood gas analysis, the blood samples taken for this purpose cannot be taken outside the pressure chamber since the gases in solution would be released as soon as the ambient pressure dropped, whence the need to have equipment suited to the hyperbaric conditions inside the chamber.

In order to analyse the exhaled gases, Spence proposed that they be drawn off through a fine tube to a specially calibrated infrared analyser (74).

Part 3: THE PHARMACOLOGICAL EFFECTS

The pharmacology and pharmacokinetics of the drugs used in anaesthesia can be affected by an increase in ambient pressure and by their use in a helium atmosphere.

There may be changes in their physico-chemical properties, their diffusion space, their site of action, their toxicity (70), or their catabolism: enzymatic systems and excretion pathways.

Very little is known as yet in this context (69-82).

I - Gaseous or volatile agents

We shall immediately eliminate from our study all products which obviously cannot be used in hyperbaric conditions because they are too toxic and have already been abandoned in normobaric conditions or because they are inflammable or explosive: Cyclopropane - anaesthetic ehters - halogens of the traditional type (Chloroform - ethyl chloride - trichlorethylene, etc.).

1) - Nitrous oxide N_2O

Its anaesthetic potency is low: very high partial pressures are needed to achieve narcosis (1 400 mmHg for a dog - 800 mmHg for a human). An enormous amount of gas is thus dissolved in the organism and may increase certain risks. It makes decompression more tricky; this gas spreads easily into closed body cavities; it heightens the potential risk of anoxia by diffusion as the patient come out of anaesthetic (60). It apparently remains chemically inert, but its harmlessness at high pressures is far from established: cycostatic effect with depression of the spinal cord and of spermatogenesis; teratogenetic effect in animals; the presence of nitric oxide through failure to separate at the manufacturing stage could lead to pulmonary oedema or the formation of methaemoglobin (48). Being a comburent, its use as a carrier gas for another anaesthetic vapour or to enable the FiO2 to be reduced is not to be recommended; it extends the inflammability area of Halothane.

2) - Halothane or fluothane

This is a fluorinated halogen and its use does not involve too many problems up to 3 ATA; it is neither inflammable nor explosive in pure oxygen from 1-4 ATA (32). Brown and Morris nevertheless consider that certain precautions must be taken, viz. not exceeding 2.5 ATA and not using N_2O as a carrier gas (18). Its stability is apparently not affected by hyperbaric pressures; on the other hand, its agressivity to certain materials (rubber - plastics - copper) could well be increased. The azeotrope mixture fluothane ether is obviously not permitted (37).

3) - Methoxyflurane or penthrane

This is a halogenated ether and its non-flammability is not absolute; however, it becomes flammable at 1 ATA only in high concentrations and

at high temperatures. At partial pressures of 15 mmHg and 1-4 ATA in a pure oxygen atmosphere, penthrane is neither inflammable nor explosive. It has a fairly high degree of toxicity, particularly to the kidney, but this degree in hyperbaric conditions is not known.

4) - The other inert gases: Argon (28) - xenon, etc.

At 1 ATA, for example, 80% xenon provides rapid but very mild anaesthesia with minimal side effects; its effects wear off in 3-5 minutes. Xenon has been used on monkeys in hyperbaric conditions; the anaesthesia produced is sufficiently profound with aproea and areflexia; EEG activity appears to be less reduced than in the presence of other agents (59).

Moreover, it should be noted that, when the pressure rises, nitrogen also produces effects which are reminiscent of narcosis. The least that can be said is that choosing a gas or a vapour is a delicate task considering: the equipment which has to be placed inside the hyperbaric chamber, since most of these drugs are not truly inert gases and that their biotransformation in hyperbaric conditions and in helium atmospheres is not known; and also considering the risks thrown up by the saturation of the various tissues by one or two additional gases because of the differences in solubility between the gases thus introduced and the gases which may be expelled by the organism (nitrogen if the patient was recovered at the surface, helium if he was recovered at depth, etc.).

Possible worsening of the second gas effect with the risk of significant variations in the volume of the closed body cavities (or in the prevailing pressure inside these cavities); of anoxia by diffusion in the case of recovery from anaesthesia during decompression (60) and, lastly, possible heightening of the risk of bubbles. The significance of these problems of counterdiffusion and second gas is perfectly illustrated when a subject is forcibly made to inhale a gas in a different environment; counterdiffusion can facilitate the formation of bubbles which can be detected in the inferior vena cave of an animal whose hindquarters are placed in a different gaseous environment.

Percutaneous elimination of gaseous or volatile anaesthetics cannot be neglected in hyberbaric conditions. Known since Waters and Orcutt in 1933, percutaneous elimination is directly proportionate to the

partial pressures of the gas in solution; wholly secondary in normal conditions (60) this is not so in the hyperbaric chamber. In Toronto, the above-mentioned American authors were able to demonstrate substantial elimination of inert gases; in this way anaesthetic gases could cause atmospheric pollution inside the pressure chambers.
Lastly, in the attempt to obtain an "anaesthetic" effect at depth, the use of these gases or vapours are, to the mind, debatable à priori: in these conditions, these gases deviate too far from the so-called ideal gases and the laws relating thereto can no longer be applied. At very high partial pressures, there are very significant deviations from Henry's law. The partition coefficients may vary (75).
The foregoing considerations impose great caution when extrapolating from our normal practice to abnormal conditions of use.

II - Drugs used parenterally

Here, too, we are largely ignorant as to the effects of hyperbaric conditions and helium, the evolution of acute toxicity and variations in effectiveness using the customary therapeutic doses. Studies are under way, but the results are incomplete and sometimes conflicting depending on the experimental animal used.
For instance, pentobarbital used on a mouse in hyberbaric conditions with air at 2 - 8 ATA has a protracted effect (41), but this is no longer so when used on rats, cats or guinea-pigs.
With a partial pressure of helium of 19.2 ATA and of oxygen of 0.2 ATA the acute toxicity of pentothal, ethanol, xylocaine, aspirin and morphine does not change (70). The toxicity and effectiveness of cardiotonic glucosides are not altered.
The length of pentothal narcosis is appreciably reduced in helium-oxygen atmospheres and this reduction seems to be directly linked with the increased partial pressure of the helium. The quantity of thiopental needed for induction is substantially increased at 20-30 ATA in a helium atmosphere (79).
The pharmacological action of morphine appears to be modified: at 11 ATA the anti-diuretic effect disappears; at 21 ATA in a helium oxygen atmosphere there is a drop in the excretion of free morphine and a fall in analgesic effectiveness (80).

In practice, up to 3-4 ATA, the action of most drugs used intravenously for anaesthesia does not seem to be changed in any way which is clinically detectable and most narcotics, tranquillizers, neuroleptics, analgesics and curares appear to have been used without any significant adverse effects. One important point is their repercussions on the patient's breathing: by adding a depressive effect to the increase in his ventilatory effort, their use may necessitate artificial (manual or mechanical) respiration. As for the rest.....?

III - "Outlook for the future": anaesthesia and very high pressures

Experiments have shown that there are extremely interesting interactions between hydrostatic pressure and the action of anaesthetic:

1 - High pressures can affect the performing of anaesthesia by modifying its structure and thus the properties of certain proteins; as a result, the affinity of haemoglobin for oxygen varies. Denaturation of the protein is unlikely as it would require over 1 000 ATA. Separation of molecular chains by molecules of inert gas has been referred to (15).

2 - Reversal of the anaesthetic effect by very high pressures: this seems to have been confirmed in the case of most anaesthetic drugs and most animals, including mammals (2 - 6 - 7 - 8 - 23 - 39 - 41 - 44 - 47 - 52 to - 64 - 83).

3 - Anaesthesia seems to counteract certain deleterious effects of pressure, improving the high pressure nervous syndrome (10 - 63) or by enabling animals to survive at far greater depths: mice for which pressures of 100 - 120 ATA in a helium atmosphere are fatal, survive at 272 ATA if N_2O is added to the breathing mixture.

The study of these interactions is extremely interesting in that they have a twofold interest, theoretical and practical.

1 - Theoretical since they permit us to gain an insight into the dynamics of narcosis; a single site of action for anaesthesia and pressure has been referred to. Mayer and Overton had noted a certain correlation between anaesthetic potency and solubility in fats. Mullins put forward the theory of "critical volume": the onset of anaesthesia corresponds to the dilation, above a critical volume, of a hydrophobic region by absorption of the molecules of an inert substance. When the initial volume is restored by

means of a sufficient hydrostatic pressure, it is claimed that the anaesthetic effect wears off (11 - 55 - 56 - 57). The mechanism could be an interruption of ion exchange (3) across this hydrophobic region the nature of which remains unknown: the cell membrane itself or the intra-cellular enzymes (52 to 53 - 58 - 68 - 81).

2 - Practical: for diving specialists these experiments entail the study of certain gaseous mixtures inhaled in order to improve tolerance at depth (10 -62 - 38). In the context of this study anaesthetists, for their part, are at a complete loss as to what would happen should they seek to obtain an anaesthetic effect at depth: it might be necessary to increase the therapeutic doses, but the toxic effects of these drugs might perhaps become intolerable before the anaesthetic level needed to perform an operation is reached.

Part 4: PHYSIOPATHOLOGICAL EFFECTS

The consequences of life at pressure are by no means negligible for the patient or for those assisting him, particularly the medical team (15-17), and some of the possible repercussions on anaesthesia and surgery should be mentioned briefly.

1 - Disorders arising from variations in the gas volume of the organism's various closed cavities

- Barotrauma of the ears and the sinuses especially during rising pressure. Equilibration of the middle ear may be difficult, even impossible, if the patient is already unconscious before being placed in hyperbaric conditions. Preventive bilateral paracentesis has been suggested (9).

- At decompression, painful intestinal dilatations (diver's colic) if the patient has swallowed gases or if these have been introduced into the stomach during artificial respiration using a mask. This is why a gastric catheter should be used.

- Account must also be taken of the gaseous effusions provoked by the closing of the surgical wound; in the mediastinum, the pleural or peritoneal cavities, the subcutaneous tissue should also be examined (73).

2 - Respiratory disorders

- The increase in ventilatory effort caused particularly by the increase in the density of the respiratory gases and a fall in lung compliance may be poorly tolerated by the patient or casualty.

- Gas exchanges are adversely affected:
by the deterioration of ventilatory function (both as regards volumes and flow rates) by an aggravation of the heterogeneity of the gas and blood distributions (changes in the ventilation/perfusion ratio): as from 2 ATA, Gordon MacDowal has shown that there is an increase in the alveolo-capillary gradient for oxygen, P (A-a) O_2 mainly as a result of shunt (31).

by disturbances of alveolo-capillary diffusion, studies of CO uptake confirm that disturbances in carbon dioxide and oxygen transfer occur at depth (31 ATA).

A certain degree of hypercapnia and tissular acidosis is common in hyperbaric conditions (and especially in the case of OHB; this state of acidosis can alter the metabolism and the therapeutic effect of certain anaesthetic drugs. Mention could also be made of the significant increase in heat loss through the upper airways (subsequent to the increase in the density of the gaseous mixtures), the problems of humidifying these gases and reducing the anatomical dead space caused by bronchostenosis and by the inflammatory oedema which may occur with hyperoxia.

All things considered, mechanical artificial respiration seems particularly advisable for performing general anaesthesia in a hyperbaric atmosphere. However, artificial respiration causes per se several disorders: respiratory (fall in compliance, degeneration of surfactant, intensification of the heteregeneity of the ventilation-perfusion ratio, etc.), haemodynamic (fall in cardiac output), neurocrine and metabolic (65-66).
This illustrates the importance of artifical respiration which is well-controlled as regards the choice of gaseous mixture insufflated (particularly FiO_2 as a function of pressure), as regards the pressure schedule for insufflation and exsufflation, and as regards its frequency and flow rates: alveolar hypo-and-hyperventilation also seem dangerous à priori.

3 - Oxygen toxicity

This very interesting biological problem to which an adequate solution has still to be found, does not concern us here (12 - 20- 29 - 36).

It can nevertheless be pointed out that the toxic effects of oxygen on cell metabolism are limited by very effective defence mechanisms (21) and that in the pathogenesis of this toxicity it is important to take account of the damage to the cell environment as a whole (circulatory, neurocrine, neurotransmitter). Anaesthesia can disrupt these natural defence mechanisms (e.g. by counteracting the vasoconstriction of hyperoxia).

Oxygen toxicity calls for very strict control over its partial pressure in the gases inhaled or insufflated by artificial respirators.

In order to maintain a normal and constant volume of alveolar ventilation, and in order to avoid hypercapnia (which is conducive to oxygen toxicity), a carrier gas must therefore be chosen. The use of nitrousoxide could be envisaged subject to the reservations which we have made, while notrogen is in our view to be excluded and helium will be selected in most cases.

Let us not forget that several anaesthetic drugs (narcotics - neuroleptics - local anaesthetics (47), various tranquillisers and particularly the derivatives of diazepam) can mask the external clinical symptoms of oxygen toxicity (4 - 5 - 19 - 33 - 35 - 72 - 77 - 84). Unfortunately, suppression of the external symptoms does not necessarily mean suppression of the toxic effect. Diazepam provides adequate protection against oxygen convulsions, but the gasps are only delayed and their irreversible nature, when they do occur, means that caution should be exercised and electro-encephalographic monitoring carried out during general anaesthesia in hyperbaric conditions.

4 - Narcosis by inert gases

All inert gases apart from helium have proved able, at pressure, to depress the central nervous system. It is felt in certain quarters that this could also apply to helium at sufficiently high partial pressures (14-28).

a) - This fact calls for a sound choice of the mixtures inhaled by the medical team which must remain fit to carry out its work. These mixtures are well known by diving specialists

b) - Could use be made of this property of inert gases to carry out "pressure narcosis" on patients or casualties?

Let us not forget that this was Paul Bert's first idea.

Nevertheless, it is hard to say. The mechanism of this narcosis is still not altogether clear. It would perhaps be preferable to speak of inert gas neurophysiological syndrome which for some gases means narcosis. Reference is sometimes made to a neuro-intoxication with excitation of the reticula and hypothalamus (the latter particularly with nitrogen, but this does not permit us to extrapolate as regards all the inert gases) (13).
Narcosis is merely one - and the least important - of the elements of general anaesthesia. Would the conditions provided by this technique be sufficient to give the surgeon adequate comfort to perform his task? What effect would this have on the effectiveness of the other drugs necessary in anaesthesia? These are questions which cannot be answered for the time being.

5 - High pressure nervous syndrome

This generally occurs at below 250 metres. It can obviously be harmful to a diver, but it can also make a surgical team compelled to dive quickly less efficient, if the gravity of the accident means that the saturation diver has to be reached as quickly as possible (63).
How to speed up the descent of the medical team without impairing its efficiency is a problem which also besets deep-diving experts.

6 - The dangers of decompression

Doppler ultrasonic detection of circulating bubbles has made it possible to prove the existence in a healthy man, even in the case of dives considered as safe, of asymptomatic bubbles. The onset of clinical symptoms varies greatly according to the persons involved in, and the circumstances of, the dive. There seems to be a relationship between the output of bubbles and the occurrence of accidents.

The existence of the "bubble" produces a number of pathological phenomena, anomalies of coagulation resulting in the formation of platelet aggregates, fat embolisms, etc. (17), which cause various circulatory and haematological disorders the consequences of which may be serious on a patient under anaesthetic and subject to the vasomotor effect of anaesthetic drugs, especially as anaesthesia can be performed on a patient in a state of exhaustion or even shock subsequent to a saturation dive. Haemodynamic disorders resulting from shock and the upheaval which this generates in local circulation may make it difficult to move the bubbles formed. If they develop locally, these can have extremely serious effects on the various parenchymas concerned.

The lung's capacity to filter the circulating bubbles can be affected by artifical respiration at intermittent positve pressure. At the moment of insufflation, there is a drop in the instantaneous pulmonary blood flow which is no longer suited to alveolar ventilation. Insufflation at positive pressure and raising the alveolar pressure compresses the capillaries (65 - 66(and this obstacle to pulmonary circulation can adversely effect the lung's capacity to discharge the circulating bubbles.

Finally, is it not possible that the use of anaesthetic gases facilitates the formation of bubbles? Their solubility coefficients are variable; their elimination speed is not known; a decompression schedule based on the usual tables could in this case become dangerous.

The principle of using different inert gases alternately could perhaps be applied to research into anaesthesia at depth. Since an inert gas is given off from the tissues at the same time as another is absorbed, perhaps with no reciprocal action, an attempt could be made to free a gas from a "slow"tissue at a speed greater than that of the uptake of a second gas by the same tissue.

It today appears advisable, following a long anaesthesia induced by inhalation, to bring the patient out of anaesthetic while still at pressure and to commence ascent after eliminating the majority of the anaesthetic gas; the use of a helium-oxygen mixture can facilitate the 'flushing' of the organism.

The use of Doppler ultrasonic detection on the right ventricular area by means of a precordial probe is to be recommended, despite

its limitations: it detects only the bubbles in circulation and its operator has to be trained (34).

7 - Lastly, the effect of hyperbaric conditions on the various parenchymas, particularly on the activity of the hepatic enzyme systems which interfere in the catabolism of drugs, should not be underestimated. This brings us back to the problems of changes in the pharmacological effects of drugs in a hyperbaric atmosphere (30).

CONCLUSIONS

Anaesthesia in a moderate hyperbaric atmosphere - up to 3-4 ATA - poses problems which are mainly of a technical nature and relatively easy to solve.

Beyond this level, it is new ground which is only just beginning to be explored.

It is extremely interesting, like everything relating to "life at pressure", and close attention will have to be given to this field as of now, for the increasing frequency of saturation diving and work at depth will necessarily confront us with patients or casualties who will have to be treated "at pressure".

Unfortunately, this paper constitutes no more than a brief review of the problems which could arise. Only a series of experiments can provide tentative solutions.

The Case for A Mobile Intensive Care Unit

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Before the case can be made for a mobile intensive care unit it is necessary to define the unit and then, in the present discussion, suggest the part which it would play in managing the critically ill diver at depth.

Mobile intensive care currently implies the provision of intensive care for a critically ill patient during the journey to hospital or between one hospital and another. In the present discussion, however, mobile intensive care implies the transportation of staff and facilities to a patient who cannot be moved to hospital to provide high-grade medical care until it is possible to convey him to hospital. This means in fact that provision of the facilities to establish an intensive care unit in a remote place and the unit must be capable of functioning in a self-sufficient manner for several days. The areas which must be covered are summarised in the first slide:

Slide 1

Mobile Intensive Care Unit

1. Equipment for resuscitation, anaesthesia, surgery, radiology, biochemistry and bacteriology.
2. Staff skilled in medicine, surgery, anaesthesia, radiology, physiotherapy, nursing and medical technology.
3. Self-sufficiency in power, communications and administration.
4. Total versatility to enable all varieties of crises to be dealt with efficiently.

Part of the equipment and some of the staff may enter the pressure chamber and part of the unit may remain outside but the disposition of

the various parts of the unit must be sufficiently flexible to deal with the particular problems of any patient wherever he may be or whatever adjacent facilities are already available.

The medical problems of divers vary greatly in complexity but will not often require the services of a transportable intensive care unit. On the other hand in an illness involving major trauma the events of the first few hours may determine immediate or ultimate survival of the patient and the intensive care unit will always have a time and distance problem. For this reason it is necessary to examine the priorities which are required in such a case and eventually to place the intensive care unit in perspective.

During the past few years there has been a great deal of discussion about the management of the critically ill diver at depth and in Britain the Diving Medical Advisory Committee have produced a list of priorities which are necessary if this complex problem is to be managed efficiently. These are listed, in order of importance, on the next slide:

Slide 2

Priorities for Management of Sick Divers in an Offshore Pressure Chamber

1. Training of Divers in First Aid.
2. Clear and precise communications.
3. Trained doctors willing to go off-shore.
4. Mobile Intensive Care Unit.
5. Transfer under pressure facility.

To cope with the initial phase of the illness it is essential to train divers to look after themselves and at the same time to establish a clear line of communication to a medical centre so that there is free interchange of information and advice between the divers and a doctor during the time that medical help is on the way. The training of the

divers must therefore be heavily weighted towards clinical examination and the transmission of the detailed type of reliable information which the doctor at a distance needs. The quality of the doctor's advice will always depend upon the precision and reliability of the information with which he is provided. Of equal, but not greater, importance in diver training is instruction in practical manoeuvres such as cardio-pulmonary resuscitation, chest intubation, intravenous therapy etc. If communications are good and the diver is able to provide such reliable information that the doctor has a clear, clinical picture of the distant patient, then the authority to employ these potentially dangerous manoeuvres can be shared by the doctor and the diver who must undertake them.

Diver training in first-aid will always be necessary for safe diving even when the problem is minor, and the third priority of freely available, properly trained doctors, willing to attend off-shore is also mandatory for safe diving. High grade paramedic training of divers and the existence of a medical communication centre is only necessary for the seriously ill diver in an offshore situation and the medical component of this must be regarded as part of the package of what has come to be known as the transportable intensive care unit.

The fifth priority - some form of transportation under pressure - is a concept which has caused much confusion and argument. It is important to distinguish at the outset between the hyperbaric lifeboat, which is intended for use in the evacuation of healthy divers from the pressure chamber of an offshore installation, which is in danger of destruction and the hyperbaric ambulance, which is available for the transfer of the critically sick diver to a large pressure chamber on land, where it would be more convenient to administer the specialised care which he may require. The points which emerge in this connection are as follows:

1. At the present time there is no large hyperbaric installation in a

major hospital and thus the use of the hyperbaric ambulance does not obviate the need for the transportable intensive care unit, which is just as necessary if the chamber is 10 miles from a hospital as where the chamber is in the middle of the North Sea.

2. The hyperbaric ambulance presently available is so small that it would not be possible to undertake the emergency treatment which may suddenly become necessary to save the life of the critically ill patient. The critically ill diver has thus a better chance of survival if the specialists are taken to the off-shore chamber, and remain there until the condition becomes stable. Only then can transfer under pressure be contemplated.
3. It is not medically acceptable for the critically ill diver to be transferred under pressure until he has been critically assessed by medical specialists. In practical terms the hyperbaric ambulance will not allow the doctor to be with his patient more speedily because the ambulance has the same problems of time and distance to overcome as the doctors.

It is for these reasons that the hyperbaric ambulance has been placed fifth in the order of priorities for the facilities needed to manage the critically ill diver at depth. There are many non-critical illnesses which it would be more convenient to manage in a large pressure chamber ashore and where it would be more comfortable and safer for the patient. An efficient form of transfer under pressure would be most useful in this situation and also in the case where there was a problem of diagnosis eg, chest or abdominal pain. This is the place of the hyperbaric ambulance and it could be used appropriately here, just as it would not be appropriate in serious, unstable life-threatening conditions.

In fact there can be little argument about the necessity for the first three priorities, which are basically required in any area where commercial diving takes place to any extent. The argument centres around the fourth and fifth priorities because they are expensive and

will not often be needed. Basically we need both but it is necessary to use them together and for the correct indications. I think the order is correct because if there was only one available, the life-saving potential of the medical back-up would be greater if a mobile intensive care unit was in existence.

In economic terms one of the problems inherent in establishing a transportable intensive care unit is that such a facility will not be required often, and recent diving history shows this to be true. Since diving operations will always require a group of doctors to provide immediate care, however, it would be reasonable to provide one highly-equipped first class transportable intensive care unit and to make it available to those doctors, providing immediate care, who may have use of its services. Since immediate care would already be provided the time and distance problem of second-line medicine becomes less important and the unit could be made internationally available. In addition, transfer under pressure cannot be contemplated unless there is a mobile group of specialists to deal with the resuscitation and stabilisation problem off-shore and with the difinitive therapy in the shore-based chamber.

Again, in economic terms, the main problem in setting up a mobile intensive care unit is the provision of high-calibre staff. In Aberdeen, it is proposed that such a unit should be established within the Institute of Environmental and Offshore Medicine and in association with the Grampian Area Health Board. The relationship of the various areas involved with an off-shore installation is summarised on the next slide.

Slide 3

The mobile intensive care unit will be administered from the Accident and Emergency department of Aberdeen Royal Infirmary, where the patient will ultimately be admitted for hospital care. The off-shore installation is linked through the occupational health service

provided by Off-shore Medical Support with that company's organisation of doctors, who provide immediate care for the diving population, which Aberdeen supports. If the doctors require the mobile intensive care unit, it will be mobilised by the Accident and Emergency department and the staff will partly come from the hospital and partly from the teaching and research divisions of the Institute of Environmental and Offshore Medicine who are teaching and researching the practical problems of diving medicine already. In this way staff, basically employed for other purposes, will be readily available to man the mobile intensive care unit on the infrequent occasions, when it is required. A problem which remains to be solved if the unit is to be available to practice on an international basis is that of medical registration to practice in another country: in the E.E.C. this is possible under "Prestation de Service" but Norway, for example, is not a member of the community.

Although the mobile intensive care unit is only really necessary for the management of a critically ill diver during saturation diving, it could be used for other purposes and these may make it more easy to persuade medical authorities to provide the facility which is certainly necessary for safe saturation, off-shore diving. The other uses are summarised on the next slide:

Slide 4

Other Uses of Mobile ICU or Parts of the Unit

1. Topside injury where extrication surgery may be needed.
2. Off-shore disaster: To re-inforce medical facilities aboard an Emergency or Stand-by vessel.
3. Remote Community: Serious illness or injury in geographically remote area eg, mountain rescue, sports diving.
4. Disaster in a remote area: To re-inforce the medical facilities in remote areas following a disaster eg, Shetland.
5. Diving onshore: Serious illness or injury in a shore-based hyperbaric chamber.

6. Helicopter ditching: To manage the rescued in transit.
7. Readily available specialist opinion off-shore.

Following detailed discussions in many forums over the past few years a system has gradually emerged to allow for the management of the critically ill diver in saturation diving and I think I have given the consensus view. Within this system there is a requirement for a transportable intensive care unit, and the establishment of such a unit is the only means available to offer the critically ill off-shore diver the best chance of survival. The unit is likely to be required most infrequently but it would be reasonable to set up one unit of high quality and make it freely available on an international basis. Although its main use would be in off-shore saturation diving other situations can be identified where such a unit would be of value.

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