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Edited by John W. P. Leach

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psychiatric disturbances or psychological dysfunctions occurring in the pre-treatment stages. It has only recently been considered that there may be some long term, adverse, psychological effect on divers in all groups. Amongst other groups researching this topic is our own group from the University of Lancaster and preliminary results from our work suggests that there may be some measurable, psychological impairment in the long term. The actual significance of these changes, however, have to be very carefully assessed. It is one thing to find evidence of impairment or damage, it is another thing entirely to ascribe to it long lasting, serious effects upon an individual's ability to live a normal life.

It is often jokingly said that divers are not normal otherwise they would never, ever dive. The same may be said of mountaineers, surfboarders or water skiers, parachutists and so on. Diving is a high risk activity, both professionally and in sport, and any individual or group who seeks to participate has a duty to participate only on the most favourable terms. The way in which those terms can be met forms the subject of the discussions to come later.

LONG TERM HEALTH HAZARDS IN DIVING

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INTRODUCTION

Long term occupational or environmental health hazards are the subject of public and media interest. As doctors and scientists we often see the distortion of epidemiological evidence in order to prove a risk in a television documentary. Extrapolation from small numbers assumes the status of proven fact and can never be disproven by reasoned scientific opinion. Diving, like nuclear waste, has not been around long enough in order to be entirely certain about the long term effects of small cumulative doses. We all know that large doses of radiation and diving can permanently damage your health, but what may be the subtle effects in the longer term? Modern society requires the quantification of risk for informed personal decisions.

None of the standard texts on diving medicine contain chapter headings or index references on long term health hazards. Individual subjects such as dysbaric osteonecrosis may be covered, but I can find no overall review on this subject in the literature. Commercial diving in the 1970s pushed forward depth limits and markedly increased the number of commercial divers doing large numbers of deep dives and repetitive decompressions. This population is not yet old enough to identify any long-term degenerative diseases. If presenile demential or lung damage are long-term risks in diving we must plan our epidemiological surveys now in order to account for the problem of a small epidemiological population base.

KNOWN LONG-TERM HEALTH HAZARDS

Dysbaric Osteonecrosis

There are many causes of aseptic necrosis of bone, but dysbaric osteonecrosis was first observed in caisson

workers in 1911 by Bassoe in the United States.(1) The first report of a diver developing osteonecrosis appeared in 1941.(2)

Dysbaric osteonecrosis is usually symptomless in the early stages and hence the importance of early radiographic diagnosis. Lesions may appear in the head, neck or shaft of long bones as white fluffy areas on X-rays. Considerable skill is required to interpret and classify the X-rays. Histologically the bone shows areas of necrosis with areas of invasion and partial repair by connective tissue. Juxta-articular lesions may collapse causing bone to flake into the joint and produce symptoms. The shoulder joint is most commonly affected, but unfortunately there are no satisfactory prosthetic shoulder joints. The knees are usually spared and the hip joint can easily be replaced surgically. Unfortunately the histopathology of lesions has not explained their pathogenesis.

Many pathological theories have been suggested including bubbles blocking off end arteries, fat and marrow embolisation during decompression, intravascular blood coagulation and osmotic changes taking place during compression.(3)

Several epidemiological studies have been conducted throughout the world. Compressed air workers have always been found to have a much higher rate of dysbaric osteonecrosis than divers. Using the strict criteria of the medical Research Council Decompression Sickness Registry the rates of definite lesions has been found to be 17.6% in 2,534 compressed air workers and 4.2% in 5,015 divers.(3) A Royal Navy series of 350 divers also showed an incidence rate of 4%. Using M.R.C. criteria (4). In clinical terms we accept that dysbaric osteonecrosis has a low incidence in divers, but is related to the number of episodes of decompression sickness, depth of diving and age. Initially long bone X-rays were recommended by the Health & Safety Executive on an annual basis, but when the pick up rate became so small this was revised to the current recommendation of annually for mixed gas divers and every three years for air divers. Long bone X-rays were not officially recommended for divers who claimed to dive to depth less

than 30 metres, e.g. scientific divers and shellfish divers. The recommendations regarding the conduct of diving medical examinations are currently under review and long bone X-rays may be abandoned completely unless a diver gives a history of recent decompression sickness. Long bone X-rays are expensive to the diver and repeated irradiation of bone marrow has been questioned. However the X-ray dose is very small and well within currently accepted safety limits. There is a certain paradox that the one long term health hazard which we know anything about is about to be officially forgotten about.

Ear Damage

The ear comes under considerable physical stress in diving with repeated ear clearing, but the bone conducting mechanisms appear to stand up to this stress in the longer term. A high tone sensory neural deafness indicating damage to the cochlea has been reported in surveys of divers' hearing.(5) High noise exposure has been suggested as a mechanism and the diving medical examination continues to require audiometry. Engineering diving and offshore diving are high noise environments and ear muffs worn while on the surface will help protect against excessive noise exposure and long term damage to the hearing.

Round window fistula and cochlear decompression sickness can cause permanent unilateral deafness. Fortunately these conditions are rare and permanent hearing loss can be avoided by prompt recognition and treatment.

Nervous System

Decompression sickness and arterial gas embolism can both result in permanent damage to the central nervous system.(6) Permanent spinal paralysis and localised areas of damage within the brain are well documented sequelae of dysbaric illness.

THEORETICAL RISK AREAS

Dysbaric Osteonecrosis

Malignant change in the lesions of dysbaric osteonecrosis is a rare but important complication of a rare disease. It has mainly been reported in caisson workers.(7) There

have also been reports in divers.(8) It will probably present with a pathological fracture and appears to respond poorly to chemotherapy after amputation. Fortunately only 7 cases have been reported in Caisson workers and divers world wide. The tumour metastasises at an early stage and it is doubtful whether earlier diagnosis would affect the prognosis. In practical terms this is not a long-term health hazard which we should screen for in the older diving population.

Neurological Damage

As investigation techniques in neurology become more sophisticated we are uncovering more clinical evidence of previously unrecognised damage to the central nervous system following Type II Decompression Sickness. Nervous tissue is unique within the body because it can not regenerate and relies upon repair and reserve capacity for continued function after cellular damage. A classic case report by Palmer et al (6) described extensive spinal cord degeneration in a case of recovered spinal decompression sickness. The diver had been murdered 12 days after a neurological examination which showed a good functional recovery from decompression sickness. However histological examination revealed extensive damage to the dorsal and lateral columns of the spinal cord. This poses the obvious question that there may be unseen damage in neurological decompression sickness despite an apparent complete recovery. It could then be postulated that tables which have a high incidence of decompression sickness may cause subtle damage to the nervous system in the longer term. This subject is covered in more detail in Dr. James' paper which follows.

The High Pressure Nervous Syndrome was first described in 1965 during some deep oxyhelium trials at the Royal Naval Physiological Laboratory. The syndrome is characterised by hand tremors, myoclonic jerking, dizziness, nausea, vomiting and a decrease in manual dexterity and cognitive function. Changes in the electrical activity of the brain have been recorded and convulsions observed in experimental animals. Death has been observed in experimental animals if the compression is not halted.

High pressure nervous syndrome has been described on numerous deep trials since 1965, but the aetiology remains unclear. From the practical stand point HPNS can be provoked in any dive greater than 500 feet and is related to the speed of compression. The deeper the dive the slower the compression rate must be. The compression phase of very deep dives must take several days in order to ameliorate the affects. The symptoms resolve after 12 - 24 hours at stable pressure, but can be provoked again by further rapid compression or excursions from saturation storage depth. HPNS is regarded as the limiting factor for deep diving and the current world depth record remains at 2,250 feet during the Atlantas III dives in 1981 at Duke University. The observed pressure reversal effect on anaesthetic agents and the proposal of the critical volume hypothesis led to the development of trimix for deep dives.(9) Trimix has been shown to ameliorate the effects of HPNS but the disadvantage of reintroducing nitrogen into the breathing mixture is that it increases the gas density and hence the work of breathing and makes decompression sickness more likely during decompression.

Only a small percentage of working dives in the North Sea are conducted in depths over 500 feet. However oil exploration does require working dives at 1000 feet in order to cross the Norwegian Trench. Only a very small proportion of the total commercial diving population have ever experienced HPNS and any long-term neurological effects from HPNS remain a matter of debate and conjecture.(10) However, common sense would suggest that anything which causes convulsions and death in experimental animals is best avoided in man. Careful planning and physiological monitoring of dives deeper than 500 feet is required and we must endeavour to follow up individuals who have been deep divers in the longer term.

Hyperbaric Arthralgia.(11) It is a feeling of joint discomfort and popping noticed during the compression phase of both nitrogen oxygen and helium oxygen dives in all the major joints. The symptoms are not present when the joint is at rest and cause only mild discomfort. Osmotic fluid shifts within the joint have been suggested as the cause leaving insufficient sinovial fluid to lubricate the functioning joint. Again too little is

known about this condition in order to project any long-term effects. Like HPNS it is related to the speed of compression and common sense would suggest that it was best avoided in order to avoid long-term damage to joint surfaces.

The Lungs

Acute oxygen toxicity affects both the lungs and central nervous system.(12) The pathological effects of pulmonary oxygen toxicity include the destruction of both capillary endothelium and alveoli epithelium, atelectasis and impairment of gas exchange by interfering with surfactant production. Interference with production of brain neuro transmitters causes convulsions in CNS oxygen toxicity. Oxygen rich mixtures are used in order to aid decompression during surface decompression and for therapy in decompression sickness. There are a wide variety of individual responses in the development of oxygen toxicity which is difficult to predict. Divers themselves often ask if long-term exposure to hyperbaric oxygen will damage their lungs in the longer term, but there is no evidence present to suggest that this is a risk.

Blood

A variety of haematological changes have been noticed during and after dives. Haemoconcentration and a reduction in the platelet count are consistent findings. (13) A recent study of chromosomes in peripheral T lymphocytes in divers showed an unusual pattern of a few heavily damaged cells compared with non diving controls. (14) The study surveyed a 100 cell samples in 153 divers and 137 controls. There were 6 abnormal divers (3.9%) who included both air and heliox divers. A substantial amount of information collected by questionnaire at the time of the blood samples showed no significant correlation with the damage observed and diving history, past medical history or exposure to irradiation. The cause and clinical significance of the observed damage therefore remains uncertain. It is likely that these heavily damaged peripheral T lymphocytes which have undergone their terminal division will be lost from the circulation and destroyed by the normal homeostatic mechanisms. The number of cells affected is too small to have any significant effect on immunity. However, a

theoretical risk would be minimal chromosomal damage occurring by a similar mechanism to stem cells in the marrow. These could then produce an aberrant cell line which may be dangerous. Unfortunately we have no information on marrow cells or cells from the germ line.

FUTURE MONITORING OF POSSIBLE LONG-TERM HEALTH HAZARDS

Present medical supervision of divers concerns fitness to dive. At present there is no systematic attempt by any government or academic body to follow divers up in the longer term once they have stopped their active diving. Long-term follow up of occupational health risks is always difficult and entails large cohort studies or methods of tracing individuals at a later date via their National Health Service numbers. Some figures are being collected by the flagging of N.H.S. records and a notification system when that person dies. However, this is unlikely to provide the answers for many of our current epidemiological questions particularly regarding damage to the nervous system. The Health and Safety Executive must take long-term follow up of divers seriously and lay the foundations for future studies.

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DECOMPRESSION SICKNESS

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INTRODUCTION

The vulnerability of the nervous system in general and the spinal cord in particular was well recognised in caisson work using compressed-air, and in diving by about 1854, (Pol & Watelle). Paul Bert, the French physiologist, includes in his book La Pression Barometrique published in 1878, an account of the post-mortem examination of a Turkish sponge diver in which the substance of the cord flowed out when the meninges were cut. The substantial volumes by Heller, Mager and von Schrotter (1900) contain illustrations of the pathology of decompression sickness affecting human brain and spinal cord and similar material from exerimental work in dogs. The damage shown ranged from mild demyelination to gross infarction. In England, Boycott, Damant and J.S. Haldane (1908) recorded similar changes in their brilliant analysis of the problems associated with decompression using goats. In the Christmas day issue of the British Medical Journal of 1909, Blick, employed as a physician in the service of divers working in the pearling industry in Australia, documented 200 cases of paralysis of whom 60 died. He was only able to observe the gross changes at post-mortem examination. Particularly noteworthy were acute cases, where gross diapedetic haemorrhages involving both the grey and the white matter effectively transected the cord in the cervical region. An increase in blood-brain barrier permeability and petechial haemorrhages are also seen in cerebral decompression sickness, especially in aviators (Fryer 1959).

Decompression sickness was recognised by Bert (1878) to be due to the liberation of the gases dissolved in body

liquids under increased pressure. A second problem causing neurological symptoms was identified with the introduction of submarine escape training (Behnke 1932) as being due to tearing of the lung parenchyma (burst lung). This allows respired gas to enter the systemic circulation and is designated gas embolism.

PULMONARY BAROTRAUMA AND ARTERIAL GAS EMBOLISM

It is frequently asserted that only the brain is affected in this form of embolism, but reference to detailed accounts of the clinical features seen in casualties of submarine escape training (Mackay 1966), indicates that transient paraplegia and quadriplegia may sometimes by seen.

The force necessary to rupture the pulmonary membrane can be generated by a rapid ascent after breathing compressed gas from a depth of 1.75 metres of water. Death from cerebral gas embolism has resulted from the use of Self-Contained Breathing Apparatus (S.C.U.B.A.) in swimming pool training. Gas embolism is a common cause of death in amateur diving. The expansion in the volume of gas in the thorax caused by the reduction in pressure (Boyles Law), has been at a rate too rapid to be vented normally through the airway.

The exposure to compressed-air in submarine escape training is very brief and there is very little time for additional nitrogen to be dissolved in body tissues. Gas supersaturation is therefore unlikely to make a significant contribution to the volume of air liberated into the circulation by pulmonary barotrauma. In contrast, when a diver has spent time breathing at depth gas transported to the tissues from the ruptured lung following barotrauma will encounter considerable degrees of supersaturation. This may lead to a mixed picture with elements of both gas embolism and decompression sickness. Evidence of spinal cord dysfunction may be found if consciousness returns.

Clinical Features

Major arterial gas embolism is characterised by immediate loss of consciousness, often during the final stage of

the ascent and may be accompanied by severe generalised convulsions (Mackay 1966). There may also be hemiplegia, focal seizures, and many other forms of neurological disturbance, including those due to the involvement of pathways in the spinal cord. It is rare to see major symptoms more than ten minutes after surfacing, but Mackay (1966) records one case where convulsions occurred after a delay of about 45 minutes.

Large quantities of very small microbubbles always accompany injections and are visualised in the technique of contrast echocardiography even in the left ventricle without apparent ill-effects. (Meltzer et al 1980). However, neurological complications do occasionally occur, although only one case of detectable permanent damage has actually been recorded (Lee and Ginzton 1983). The patient presented with confusion and a facial nerve paresis. On examination there was a mild left hemiparesis, a right gaze preference and a constructional apraxia. Many cases of iatrogenic air embolism occur in clinical practice, although they are rarely recognised or treated, and a degree of air embolism always accompanies cariopulmonary bypass surgery (Pierce 1980).

Pathogenesis

When a large quantity of gas enters the systemic arterial tree it may occlude major vessels in the nervous system causing hypoxia and ischaemic infarction. This has been demonstrated by computerised axial tomography (Hwang et al, 1984).

In lesser degrees of embolism the gas occludes vessels of the order of 50 microns, (Babcock and Netsky, 1960). The bubbles do not produce infarction, but are associated with a disturbance of blood-brain barrier function (Waite et al 1967). This may be transient or it may lead to perivenous aedema, because of focal hypoxia and, as yet, undefined changes in endothelial permeability. The effects are critically dependant on the bubble size.

In uncomplicated air embolism the summation of the partial pressure of the gases contained in the bubble (N_2,O_2,CO_2,HO) (Dalton's Law of Partial Pressures) is well in excess of the tensions of the same gases in the surrounding tissue liquids, because the force of surface

tension is trying to crush the bubble. The tissues can therefore act as a sink for the gases in the bubble causing it to dissolve and disappear. In contrast, microbubbles in divers may grow encountering inert gas supersaturation in the tissues. In this case the bubble is acting as a sink for the tissue gas.

DECOMPRESSION SICKNESS

Because oxygen is toxic at significantly raised pressures a second gas must be added for respiration under hyperbaric conditions. In the case of compressed-air, the second gas is of course, nitrogen, but synthetic breathing mixtures using helium, neon and hydrogen have all been used. Gases dissolve in the respired gas (Henry's Law). The raised partial pressure of the second gas is reflected, after transfer across the pulmonary membrane: in an increase in the plasma gas tension which, in turn, is reflected at tissue level by an increase in the "inert" gas concentration.

At sea level an average adult man has about a litre of nitrogen dissolved in the tissues. Doubling the partial pressure of nitrogen by exposure to twice atmospheric pressure and allowing time for equilibration will double the amount of nitrogen dissolved (Henry's Law). A diver or compressed-air worker exposed to twice atmospheric pressure will eventually contain 2 litres of dissolved nitrogen, measured at normal barometric pressure. The nitrogen loading of a tissue reflects its fat content as the gas is about five times more soluble in fat than in water (Behnke and Yarbrough 1938). When ambient pressure is reduced and the sum of the dissolved gas tensions at tissue level exceeds the sum of the partial pressures of the gases being respired, a state of supersaturation is created providing energy for the liberation of gas phase from solution.

It is important to recognise that the utilisation of oxygen in metabolism by the tissues is not equalled by a rise in the level of carbon dioxide, because of its greater solubility. This effect has actually been demonstrated by a negative pressure in a rigid capsule implanted in living tissue, by Hills and LeMessurier (1969)

A reduction of ambient pressure which exceeds this partial pressure vacancy creates supersaturation at tissue level and gas phase may be formed. Symptomless bubbles can be detected ultrasonically in the pulmonary artery as they enter the microcirculation of the lung during routine decompressions, by using Doppler ultrasonic techniques (Spencer and Campbell 1968). This separation of gas from solution renders methods of calculation for decompression tables very imprecise and human trials cannot be avoided. The uptake of the nitrogen or helium by a given tissue is dependant on the blood supply and the flux or bulk movement of gas molecules. This is an area of controversy as the relevant importance of perfusion and diffision has not been defined for a given tissue. However it is clear that tissues like the retina and the cerebral cortex, which have a very large blood flow per unit mass of tissue, will equilibriate to a new plasma inert gas tension very rapidly, but other areas of the brain have a much smaller blood supply and a greater fat content. A range of perfusion zones is present in both the brain (Brownell and Hughes 1962) and the spinal cord (Hassler 1966). The arterial watershed territories are most often affected in cerebral decompression sickness (Brierley and Nicholson 1969) and in the spinal cord, where the expansion of tissue within the pia mater may reduce blood flow (Hills and James 1982).

Clinical Features

With the use of decompression tables now being almost universal in diving, fulminating decompression sickness is rare and the symptoms of nervous system involvement are usually insidious in onset. Serious decompression sickness is rare in helium and oxygen divers, but about ten air divers develop neurological decompression sickness each year in the North Sea from a working population of only a few hundred men. Many cases are not notified to the authorities. Typically transient cerebral symptoms such as dizziness, vertigo, blurring of vision and dysarthria may precede spinal cord features, which are often accompanied by back and girdle pain. Weakness in the legs, problems in co-ordination, urinary retention and sensory disturbances are common features and may simulate an infectious myelitis (Johnson 1957).

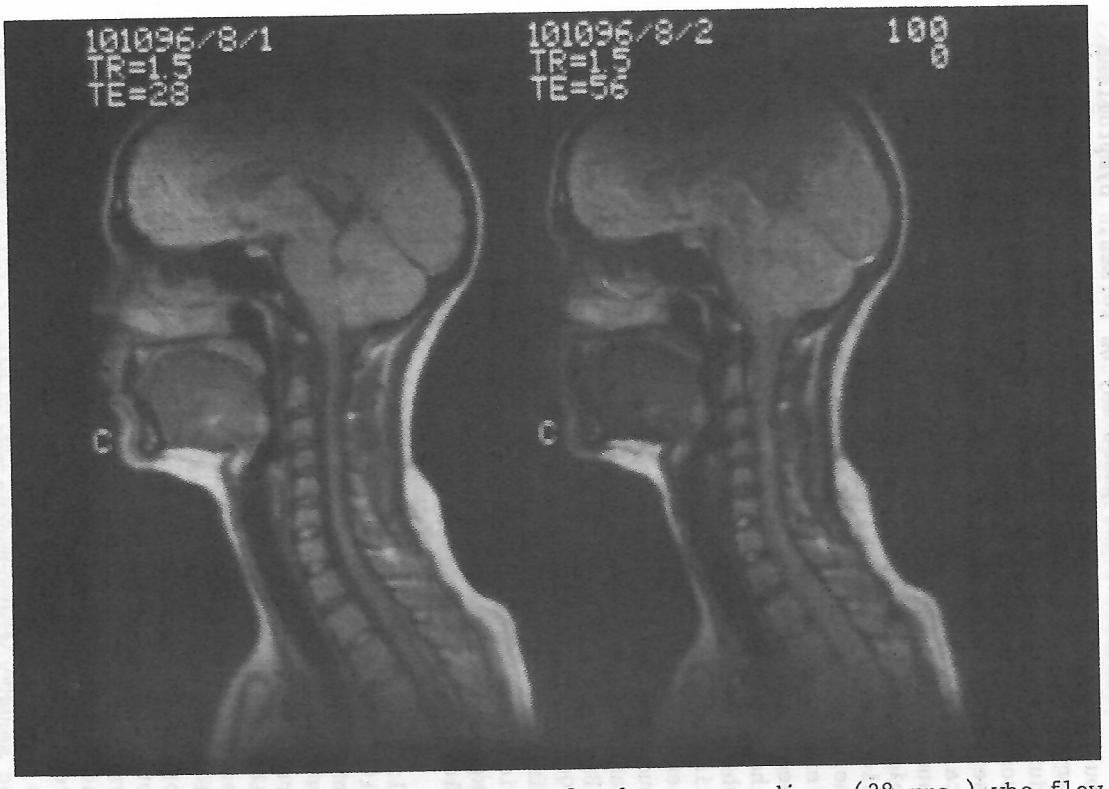

Fig. 1: Magnetic Resonance Images of a female amateur diver (28 yrs.) who flew about 11 hours after a dive to about 90ft (30m) for 40 minutes.

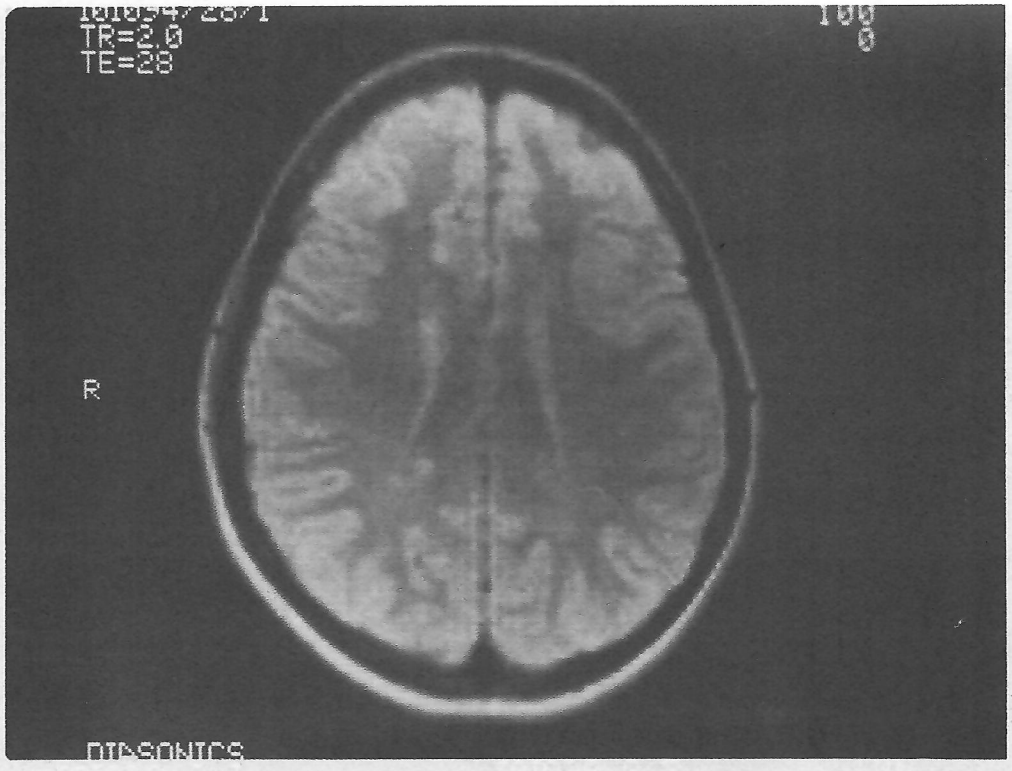

Fig. 2: Magnetic Resonance Images of a female amateur diver (28 yrs.) who flew about 11 hours after a dive to about 90ft (30m) for 40 minutes.

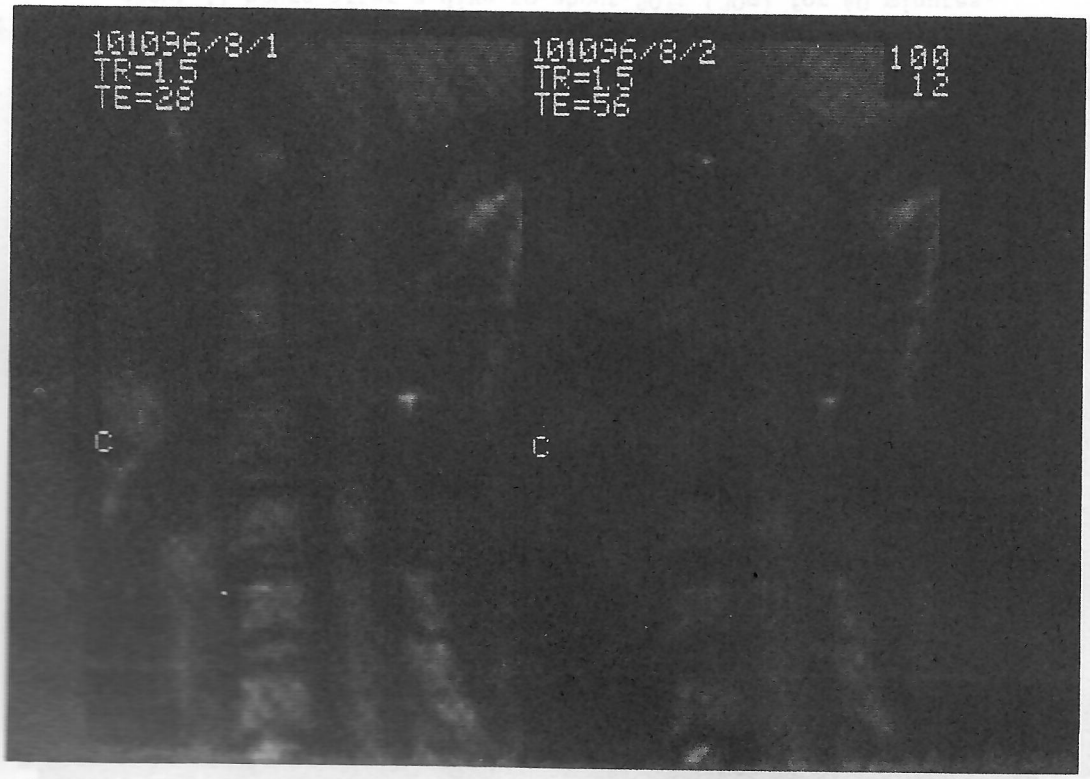

Fig. 3: Magnetic Resonance Images of a female amateur diver (28 yrs.) who flew about 11 hours after a dive to about 90ft (30m) for 40 minutes

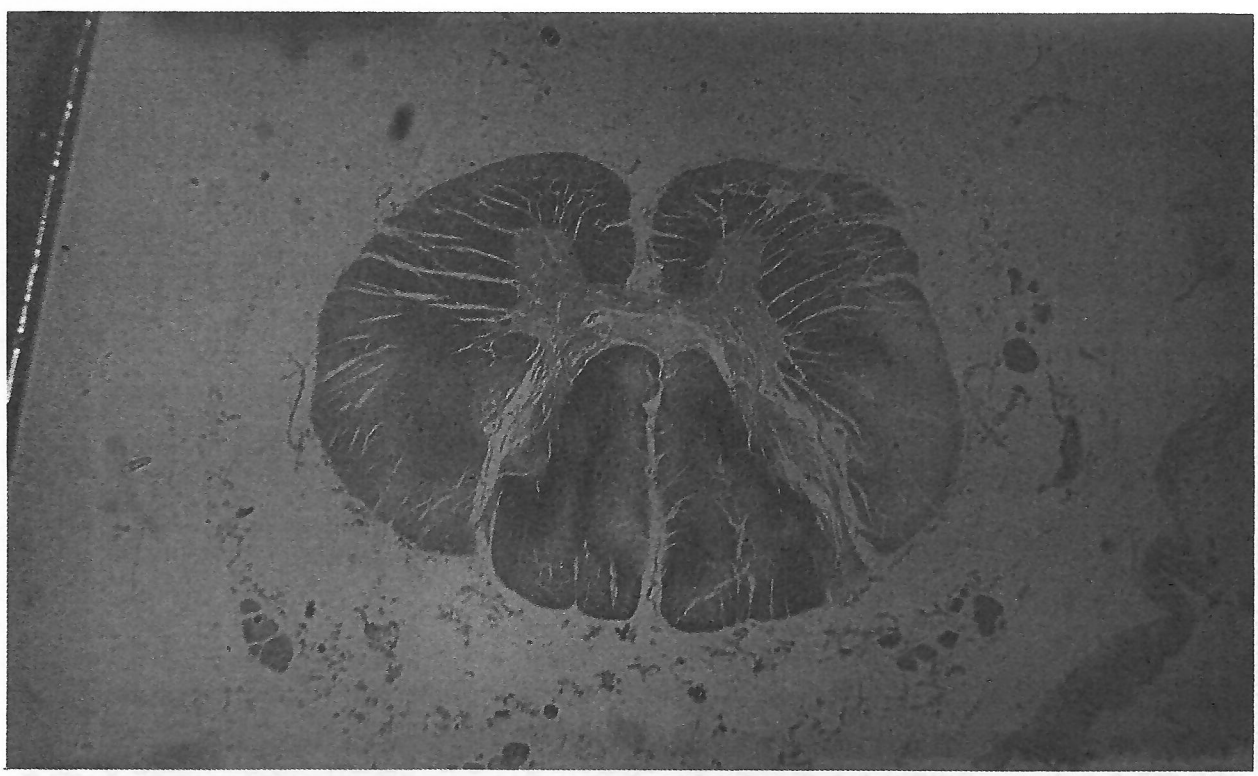

Fig. 4: Subacute damage to spinal cord following spinal decompression sickness (After Palmer et al 1981).

If neurological problems arise in helium and oxygen mixture divers, they are usually insidious in onset and the brain is most often involved, particularly the vestibular system. Sometimes there may be labyrinthine damage in divers, which is compatible with microbubble embolisation of the end organ (Money et al 1985). Brain stem damage causing accommodation and convergence insufficiency has been described in professional divers by Lieppmann (1981).

Aviators may also develop neurological decompression sickness (Fryer 1969). The cerebral manifestations dominate the clinical picture with loss of consciousness, focal seizures, bizarre paralyses simulating hysteria and even inappropriate behaviour. Most cases recover as pressure is increased with a reduction of altitude, but some continue to deteriorate at sea level and may die unless hyperbaric therapy is undertaken (Fryer 1969). Although paraplegia and quadriplegia have been described during flight, they have usually, but not always, (Hook 1958) been reversed as pressure is increased by descent.

Although the problem has almost disappeared from civil aviation with the introduction of cabin pressurisation, decompression sickness may be provoked by flying after diving at the modest cabin pressures equivalent to 6,000-8,000 feet ordinarily used. Figs. 1, 2 and 3 are the Magnetic Resonance Images of a female amateur diver, aged 28, who flew about 11 hours after a dive to about 90 ft. (30m) for 40 minutes. A left-sided hemiparesis and parasthesia developed during the flight, which is compatible with the lesion shown in the internal capsule. Despite making a neurological recovery, the demyelinating lesions are unchanged, including the lesion present in the cervical spinal cord. The neuropathology of this area is almost certain to be similar to the case of subacute damage reported by Palmer et al (1981). Fig. 4.

The problem is still of great importance in space exploration during extra vehicular activity, because the suit pressure is considerably lower than that used in the spaceship or space station (Hills 1985).

Pathogenesis

Mechanisms in neurological decompression sickness are

controversial and it is therefore important to emphasize the factors which are known. All significant decompressions, including those to below atmospheric pressure, cause systemic venous bubbles which can be detected ultrasonically in the pulmonary artery. In the case of deep long duration air dives, symptomless bubbles have been detected as long as 16 hours post-dive. It is known that in some divers these bubbles may cross the pulmonary filter and appear in the systemic arterial circulation (Powell and Spencer 1980). Arterial bubbles have been detected in some divers after small decompressions at extreme pressure (Brubakk 1981). The evidence suggests that transpulmonary passage of emboli is an extremely unpredictable event, which would explain the almost random nature of neurological decompression sickness.

If small arterial bubbles escaping pulmonary filtration are universally disseminated, areas of the nervous system of high fat content and which are poorly perfused will be at special risk (Boycott et al 1908). This mechanism would predict the vulnerability of the cord and especially the poorly perfused lower thoracic segments. It would also explain the frequency with which neurological symptoms are seen in divers with respiratory distress (the chokes). This condition is known to be associated with a large quantity of bubbles arriving in the pulmonary filter. Transpulmonary passage is greatly increased as the dose of emboli increases and in the presence of hypoxia (Niden and Aviado 1956).

A microbubble arriving in the spinal cord may encounter tissue supersaturation and therefore act as the sink for the gas, dissolved in the cord tissue and grow in size. The expansion of the cord and the development of cord oedema may cause major alterations in the mechanics of blood flow (Hills and James 1982). Variations in bubble size can account for the pathological findings which range from perivenous demyelination, associated with blood-brain barrier dysfunction, to ischaemic infarction and also the difference in clinical presentation between altitude and hyperbaric exposures (James 1982 a).

An alternative hypothesis (Hallenbeck et al 1975) relates the mechanism of spinal cord decompression sickness to

venous obstruction, in a way reminiscent of the now discredited theory of metastic cell dissemination to the nervous system via Batson's plexus (Henson and Urich 1982). The animal model used induced fulminating decompression sickness with the formation of large amounts of gas. This gas was redistributed in the experiments by recompression, which is a reliable way of inducing the transpulmonary passage of bubbles (Gait et al 1975). A major factor in the argument for this mechanism was the apparent absence of a spinal cord disease associated with failure of pulmonary filtration, but this point has been answered (James 1982b).

The hypothesis also cannot account for cerebral decompression sickness and there is growing evidence of subclinical cerebral damage in divers (Peters et al 1978). Delayed resolution of symptoms is associated with permanent cerebral damage evident on CT (computed tomography) scanning (Gerstenbrand et al 1980) and, as shown, by magnetic resonance imaging. This has been illustrated by the post mortem demonstration of extensive demyelinating lesions with relative preservation of axons in an amateur diver who had minimal signs and no significant residual symptoms following spinal cord decompression sickness (Palmer et al 1981). Illustration 4. (Courtesy of Drs. A.C. Palmer and I.M. Calder).

TREATMENT

The cornerstone of therapy is recompression in a hyperbaric chamber, which reduces the volume of the gas phase present and provides the driving force for its return into solution. Increasing the oxygen content of the gas used in therapy provides an additional concentration gradient for the removal of the offending gas and also relieves hypoxia, reduces oedema and C.S.F. pressure. Oxygen is also the only agent which causes vasoconstriction in the nervous system yet, at the same time, increasing tissue oxygen delivery (Jacobson et al 1963). Immediate recompression produces spectacular recovery, confirming the role of gas in the pathogenesis of the syndrome. The gas used has, because of availability generally been compressed-air. Predictably this gives good results in the gas embolism due to barotrauma, but is less reliable

in decompression sickness where the tissues have a high nitrogen loading. The trend in commercial diving is to the use of helium and oxygen mixtures (James 1981). The absence of nitrogen in the mixture results in the rapid elimination of the nitrogen and dramatically reduces the risk of the subsequent decompression. It is vitally important that treatment is started as soon as possible, a delay in treatment of as little as a few minutes may leave permanent disability. However astonishing results have sometimes been obtained in the treatment of comatosed patients with iatrogenic air embolism, even after a delay of as long as 29 hours (Mader and Hulet 1979).

Because haemoconcentration accompanies serious decompression sickness, intravenous fluid therapy is also important and high dose steroids, (Troiano et al 1984) and mannitol (Stefowski et al 1985) may assist in the reduction of focal oedema and restoration of normal blood-brain barrier permeability.

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POST-DIVE SYNDROME

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ABSTRACT

There is an anecdotal belief that commercial divers undergo some form of psychological change immediately following diving operations - a possible "post-dive syndrome". This change appears to affect both the diver's cognitive functioning and his personality. Fortunately, this change is often temporary with the diver recovering within 3 to 5 days - although this is not always the case. This paper reviews the evidence for the existence of psychological dysfunction following diving operations. (The reader is also referred to the paper by Frank Mee, Long Term Health Hazards: a Diver's View, elsewhere in this volume).

PSYCHOLOGICAL EFFECTS OF DECOMPRESSION SICKNESS

It is known that cognitive dysfunction can arise as a direct result of decompression sickness (DCS) in both divers and caisson workers. Rozsahegyi (1959) studied the effects of decompression sickness on caisson workers involved in the building of the Budapest underground railway. All patients who were unable to work for three or more days as a result of decompression sickness were clinically examined and 100 patients were subsequently followed up during a period of between two-and-a-half to five-and-a-half years after the event.

Rozsahegyi describes four types of CNS lesion occurring

as a result of decompression sickness, namely; (1) multiple focal injuries of the whole CNS, (2) multiple lesions to the cerebrum and upper brain-stem, (3) injury to the medulla, pons and cerebellum, and (4) spinal lesions. Of his follow up group a psychosomatic syndrome was seen in one-third of the cases of neurological DCS, with particular emphasis being placed on personality changes. Men who were quiet before injury became extremely irritable and uncontrolled. Pathological drunkenness and alcohol intolerance were also frequent. Rozsahegyi noted that this psychosomatic syndrome persisted after several years only in those cases in which the upper segments of the CNS were affected, particularly in group (1), those showing multiple focal lesions of the whole CNS. It was not found in group (4), viz. those with only spinal cord lesions. More than half the cases in group (4) showed no other symptoms several years after the event, as did half of group (2); but in groups (1) and (3) only one-fifth of the cases were without symptoms. The most frequent complaints were dizziness (49%), headache (46%) and increased irritability (40%). Rozsahegyi suggests a possible chronic progressive encephalomyelopathy occurring as a result of repeated decompression procedures. Although this progressive disorder could occur in the absence of decompression sickness, it was more common in individuals with a history of DCS.

Levin (1975) also found a high incidence of neurotic and psychosomatic symptoms among divers with a history of decompression sickness. Kwiatkowski (1979) found a significantly higher proportion of pathological EEG records among professional divers, coupled with a distinct increase in pathological records correlating with number of years of diving. Furthermore, Hallenbeck (1978) reports correlations between repeated decompressions and the development of a progressive encephalomyelopathy.

Peters et al (1977) studied twenty divers, ten of whom had an unequivocal history of at least one episode of decompression sickness involving the central nervous system, while some professed more than one DCS trauma. The remaining 10 divers did not give a history consistent with CNS decompression sickness but had complaints

subsequent to various diving incidents, including brief episodes of hypoxia, phobic reactions following frightening experiences while diving, and other illnesses developed while diving but clearly unrelated (e.g. infectious hepatitis). Information obtained from the divers included historical data, neurologic examination (EEG) and neuropsychological examination. One diver did not undertake the neuropsychologic examination.

The neuropsychological test battery comprised the Wechsler adult intelligence scale (WAIS), Wechsler memory scale, Reitan trailmaking test, Reitan finger-tapping test, supraspan digit storage test and the Minnesota Multiphasic Personality Inventory (MMPI). Of the 19 divers who undertook the neuropsychological examination seven showed deficits consistent with cerebral dysfunction. Eight divers showed a normal neuropsychologic examination and these served as a control group. It was found that impaired divers performed significantly below the control group on both verbal and non-verbal subtests of the WAIS. Memory impairment was found on the Wechsler memory scale and the supraspan test. Forward digit span was also reduced, as was repetitive finger tapping. Comparison of MMPI profiles in impaired and control divers disclosed that the former group showed greater acute distress, depression, anxiety, somatic concern and disruption of cognitive efficiency.

Of the ten divers with a history of DCS, nine showed abnormalities on neurological and/or neuropsychological tests that suggest lesions of the higher central nervous system as opposed to being confined to the spinal cord. Eight of these divers had neurological findings compatible with multiple lesions usually affecting more than one area of the nervous system. Peters et al conclude that cerebral disturbance following decompression illness in divers is more common than has previously been appreciated.

Whereas Peters et al compared just two groups of divers (DCS versus non-DCS), Vaernes and Eidsvik (1982) took a further step and considered three groups; divers with a history of diving accidents, accident-free divers and a non-diving reference group. Their purpose was to investigate possible differences in specific central

nervous system functions between the three groups. The accident group consisted of nine subjects, the non-accident group consisted of 15 subjects while the reference group was based on data obtained by Matthews (1978) on 1,375 subjects.

Each subject was adminstered an extensive test battery including the Halstead neuropsychological battery, the Wechsler Adult Intelligence Scale, the Wechsler Memory Scale and the MMPI. Subjective symptoms were assessed by means of a structured interview.

The WAIS results were within the normal range for all three groups. Of the nine subjects in the accident group eight had one or more impaired scores on the Halstead battery. In other tests, five subjects had impaired scores on the Sandpaper test, eight on the Tactual Forms test, five on the Grooved Pegboard test and four on the Knox Cube test. All nine subjects scored below 1 standard deviation from the mean on some of these tasks.

On the subjective symptoms, eight out of nine reported a change in cognitive functioning after their accident. Most subjects reported impairment in memory as the major problem. Other problems mentioned included concentration problems, irritability, alcohol intolerance and aphasia.

Performance on the Wechsler Memory Scale by the accident group was below that for the non-accident group on all subtests. A significant difference was found between the two groups on the visual reproduction subtest.

Vaernes and Eidsvik argue that the low scores in the various tests by the accident group are indicative of impairment of complex spatial functions, and especially spatial memory, affecting both tactile and visual modalities. Their work further supports the views of Peters et al (1977) and Rozsahegyi (1959) that diving related CNS decompression sickness suggests lesions of the higher central nervous system levels and is not confined to the spinal cord. It is also becoming more apparent from the above studies that neurological and/or psychological manifestations of decompression sickness in divers is more common than was originally thought.

Edmonds (1984) reports cases of decompression sickness followed by discernable psychometric impairment, often with abnormal EEG patterns and occasionally with abnormalities in the brain scan. The following year, 1985, Edmonds and Boughton report a study in which they surveyed 30 compressed air abalone divers. They employed two separate psychometric tests, the ACER Higher Test WL and the Revised Visual Retention Test (Benton). The former is designed to measure general ability as revealed by performance on material of a verbal nature, while the latter is designed to assess visual perception, visual memory and visuoconstructive abilities.

Of the original 30 divers six had to be excluded for technical reasons. From the remaining 24 subjects Edmonds and Boughton found evidence of impairment in intellectual capacity in 11 (4 suggestive and 7 strongly suggestive), and one subject was placed in the 'questionable' category. It is argued that the results obtained strongly suggest a general impairment of intellectual capacity (i.e. a dementia) and that such a finding would not be expected in normal subjects. The divers showed no gross or obvious neurological manifestations.

Fifteen divers reported decompression sickness but this was found not to be significantly correlated with impairment of intellectual capacity. Similarly, there was only a slight age difference, and this was in the wrong direction.

PERSONALITY AND PSYCHIATRIC CHANGES FOLLOWING DIVING

There are a number of reports of personality changes which are alleged to take place in both divers and caisson workers. Rozsahegyi (1959) reports pathological changes in the personality of caisson workers as a result of decompression sickness. In particular, the tunnellers become extremely irritable and uncontrolled with pathological drunkenness and alcohol intolerance also frequently reported.

Biersner and Ryman (1974) studied the records of 4,720 U.S. Navy divers and compared them with a listing of all

Navy psychiatric hospital admissions between 1968 and 1971. They found that 83 Navy divers had been admitted as psychiatric cases. This was significantly higher than the admission rate for non-diving controls - divers = 17 per 1,000, controls = 7.5 per 1,000 ($t = 9.5$, $p<0.001$). Divers were also significantly more likely than controls to be admitted for situational maladjustment.

A more recent study of U.S. Navy divers by Hoiberg and Blood (1985) found quite the opposite. A comparison of 11,584 divers with 11,517 controls showed a significantly lower admission rate for psychiatric and stress-related disorders among divers than controls. Two possible explanations for this apparent reversal over a period of 11 years are that firstly, the medical and psychological standard for divers enlisted in the U.S. Navy have been tightened up. This could also be coupled with the fact that diving doctors have become more professional and are more informed on what to look for in a diver. Secondly, there is evidence (hinted at by both Biersner and Hoiberg) that the U.S. Navy Authorities were prepared to tolerate greater amounts of abnormal behaviour among their divers than from other Navy men. Hoiberg and Blood also suggest the possibility that divers do indeed live up to their image, reflecting a high level of toughness, hardiness and a high pain threshold. Kindwall (1975) reports that there are a number of phlegmatic individuals in the diving industry who, for example, "elect to 'walk off' symptoms of decompression sickness or treat them at home with classic remedy of aspirin and a few stiff shots of whiskey".

PSYCHOLOGICAL CHANGES FOLLOWING EXPERIMENTAL DEEP DIVES

There are increasing reports of temporary aberrations in personality following experimental deep dives. The two non-professional divers in the Norwegian experimental dive Deep Ex I reported that they were not interested in being involved as subjects in future dives and Tønjum (1983) reports that one subject had psychological distress with depression lasting 2-3 months after the dive and eventually sought out a psychologist for help. Vaernes (1983) reports post-dive neuropsychological changes from

pre-dive controls during both Deep Ex I and Deep Ex II. Observed changes included reduced motor tempo and hand grip strength, memory impairment and reduced scores on cognitive abilities (Halstead-Reitan). These results returned to normal within one year. There was also a high incidence of subjective problems, mainly depression, inactiveness and sustained attention problems. In some instances these problems lasted up to six months.

In America, at the end of the 1981 Atlantis III dive to 686m, one of the three divers reported difficulty in concentration, inattentiveness and difficulties in remembering simple things such as shopping lists and recipes. These symptoms were accompanied by feelings of acute anxiety and sadness. This man was a very experienced commercial diver who had previously made over 2,000 dives (Bennett, 1983).

After surfacing from a dive to 549m all six divers were reported to be suffering from a post-dive 'malaise' accompanied by complaints of absentmindedness lasting for several months, they could not remember things such as where they left their pay cheque or car keys. Three years after the dive all the divers underwent intense neuropsychiatric testing and no abnormalities were found (Thalmann, 1983).

After the British Deep Dive Series Torok (1981, 1983) reports divers having a pervading sense of fatigue, lassitude and lack of motivation amounting almost to lethargy. Torok argues that this may well be a decompression effect rather than hyperbaric pressure *per se*.

PSYCHOLOGICAL DYSFUNCTION AND COMMERCIAL DIVING

The above are, of course, rather exceptional deep experimental dives, but what of the routine commercial diving operations carried out everyday offshore? It is generally agreed amongst professional divers that it usually takes about three days to readjust to 'civvy street' after diving operations, and this view is strongly supported by their wives, girlfriends and close family members. Indeed, so noticeable is this change in

personal characteristics that one wife of a saturation diver contacted Dr. Alan Baddeley, at the Medical Research Council Applied Psychology Unit, to voice her concern. She had carried out a mini-survey of seven divers' wives and enclosed the results in the following letter which Baddeley published in the EUBS/NPD (1983) Proceedings (p.164-7):

> "With reference to our telephone conversation, I have listed below some of the physical and mental defects that seem to occur in divers after periods of saturation. This information represents the opinions of myself and six other divers' wives who were kind enough to respond to my inquiries. Notall of the ailments listed affected each diver. The number affected by the defect is shown alongside.
>
> | | | |
> |---|---|---|
> | 1. | Loss of memory | 4 |
> | 2. | Aggressive behaviour | 5 |
> | 3. | Short tempered | 5 |
> | 4. | Secretive | 3 |
> | 5. | Anti-social | 1 |
> | 6. | Slow reactions | 3 |
> | 7. | Tiredness | 4 |
> | 8. | Anxiety | 3 |
> | 9. | Inability to communicate | 2 |
> | 10. | Inability to concentrate | 4 |
> | 11. | Problems with eyesight | 4 |
> | 12. | Disinterest in appearance | 4 |
> | 13. | Derogatory effect of alcohol | 6 |
>
> The latter seems to be by far the most general complaint, the seventh diver is tee-total. We all felt that it is probably the most frightening aspect of all and one of the men involved has not dived for ten months, and yet according to his wife he was still unable to copy with the effects of alcohol."

At what must have been around the same time as Baddeley received the above letter, I carried out a small survey among the wives and girlfriends of the divers I was working with in the North Sea. No wife or girlfriend was contacted directly, rather the questionnaires were passed to them through the divers themselves. All the divers were volunteers and all agreed to take part. Thirty-two forms were issued of which thirty returned, either when the divers rejoined the ship or occasionally by post. The results of the survey are summarised in figs. 1 and 2. The most common observations made by the wives were: increased hostility (73%), memory impairment (53%), attention difficulties in concentration (37%), and a feeling of nervousness or being "strung up" (37%).

The results of this survey fit in remarkable well with the observations of the woman who wrote to Dr. Baddeley. In fact, there is a correlation between the items scored on the two surveys of $\underline{r} = .556$ $df = 10$ $p<0.05$.

Now, one must be careful about interpreting these results. Certainly the sample size is small for both groups ($n = 7$, $n = 30$), no control groups were studied, and the scientific methodology is not as rigid as it might have been under ideal conditions. But these were not ideal conditions, nor could they be. Also one is open to being taken to task over correlating two small samples such as these, and this is a fair criticism. Nonetheless, providing these caveats are borne in mind, then I believe the above surveys do begin to show a possible profile of behavioural aberration among divers immediately following diving operations - a possible 'post-dive syndrome'. This may have echoes of Biersner and Ryman's (1974) finding of increased situational maladjustment among U.S. Navy divers.

As a matter of interest the group was divided into those divers who had just completed a period of diving on air ($n = 17$) and those who had been in saturation on oxy-helium ($n = 13$). The profiles were very similar for both groups although the mixed-gas divers showed noticeably more problems of memory, concentration, decision making and overall "slowness" than their air diving counterparts. The air divers, however, had more sleep associated

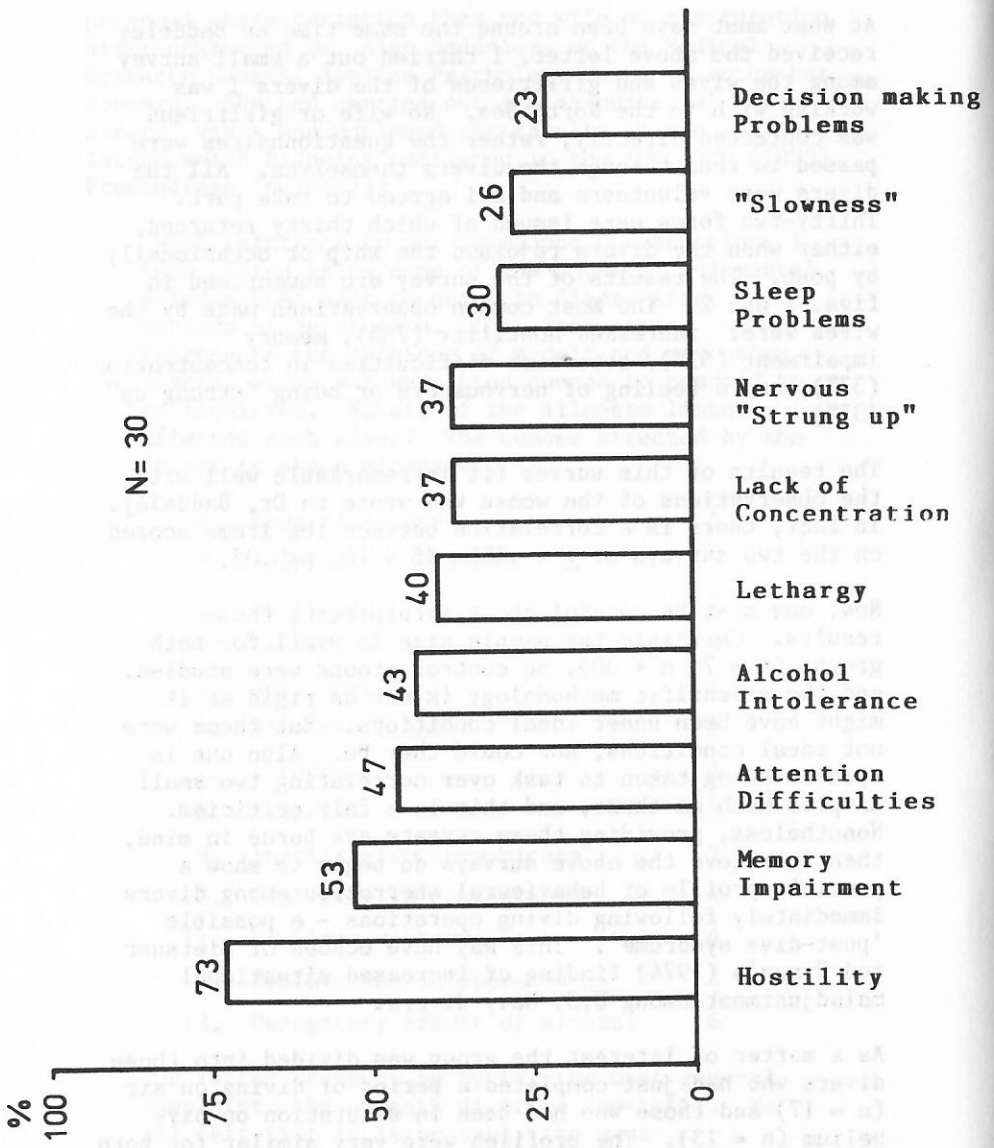

Fig. 1 Wives'/girlfriends' reports of personality changes in divers following diving operations.

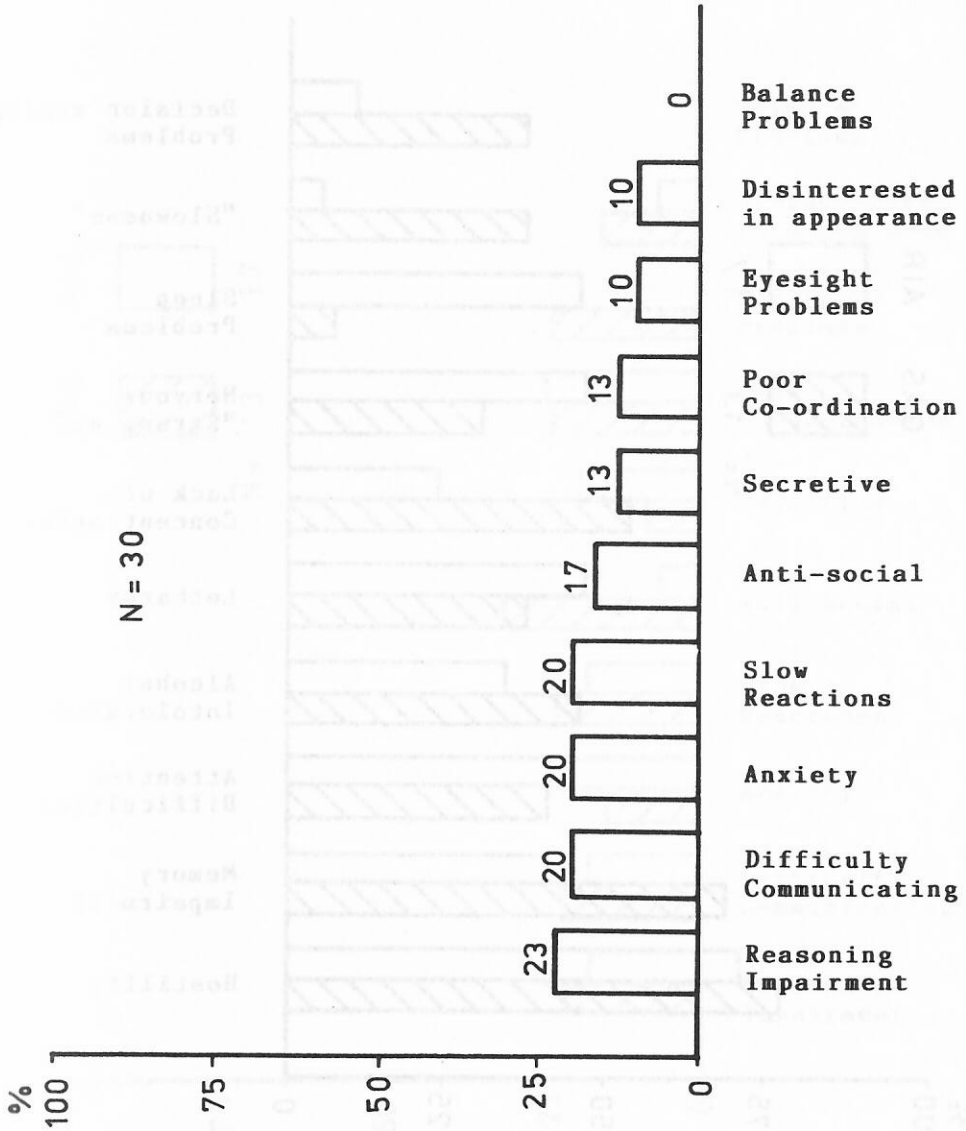

Fig. 1 (cont'd) Wives'/girlfriends' reports of personality changes in divers following diving operations.

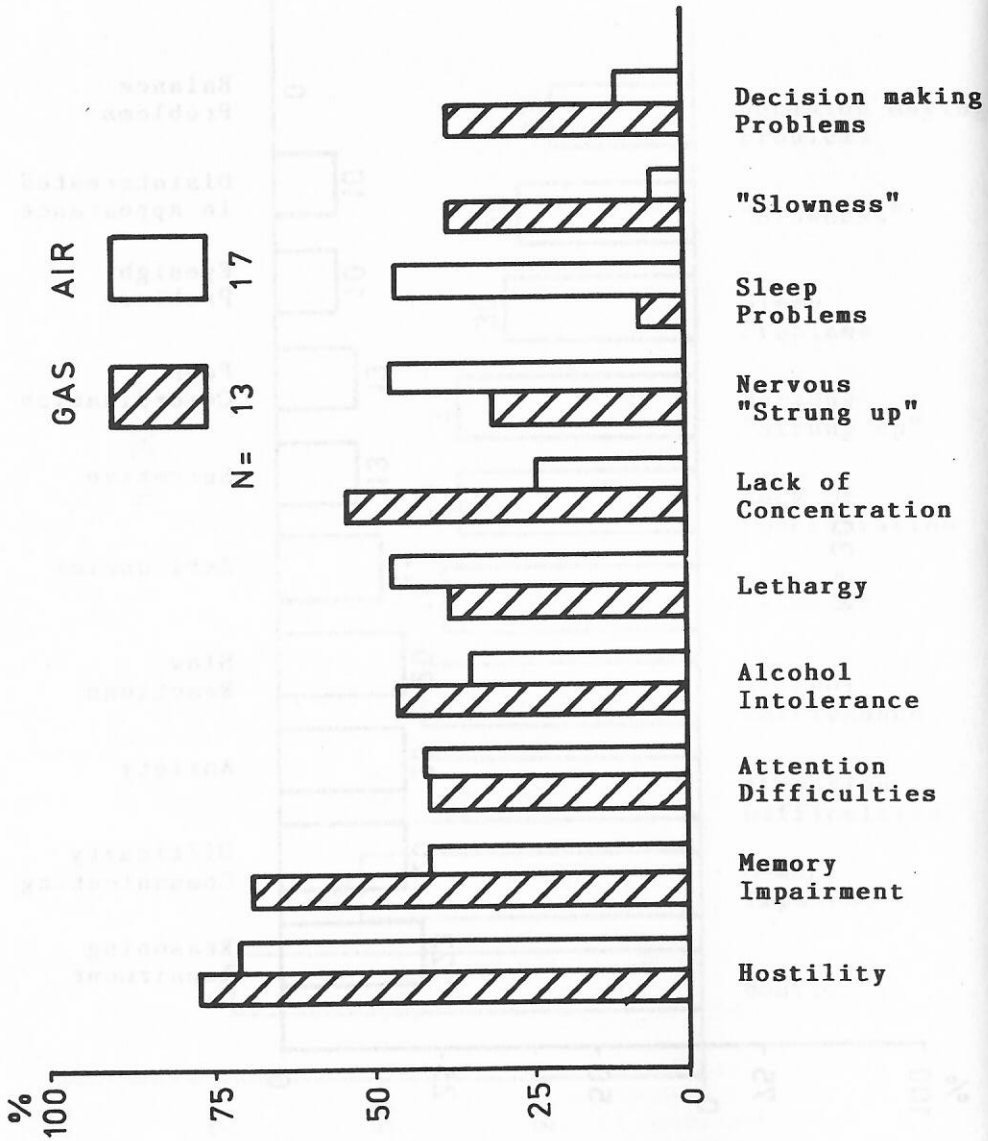

Fig. 2 Wives'/girlfriends' reports of personality changes in divers following diving operations (air diving v. mixed-gas).

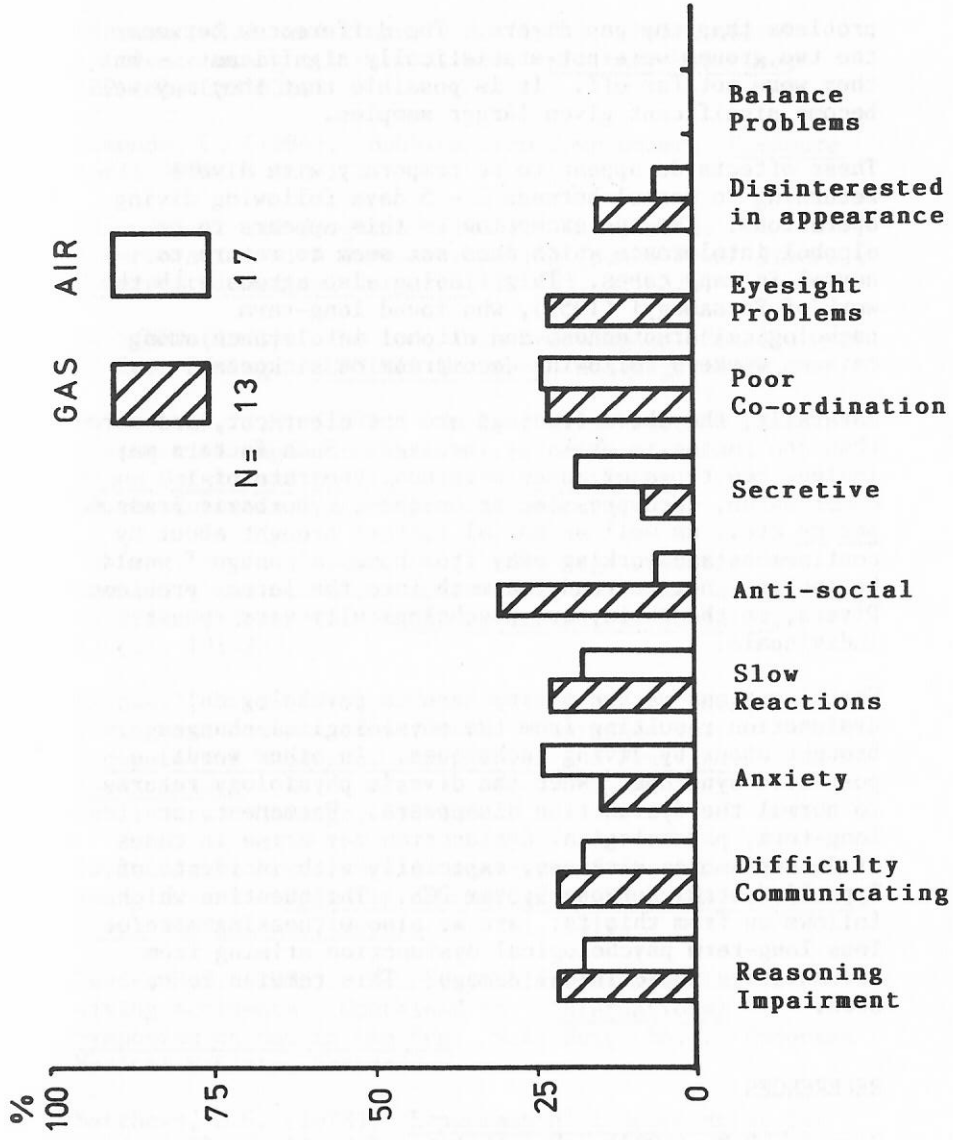

Fig. 2 (cont'd) Wives'/girlfriends' reports of personality changes in divers following diving operations (air diving v. mixed-gas).

problems than the gas divers. The differences between the two groups were not statistically significant - but they were not far off. It is possible that they may well become significant given larger samples.

These effects do appear to be temporary with divers returning to normal between 3 - 5 days following diving operations. The one exception to this appears to be alcohol intolerance which does not seem to return to normal in many cases. This finding also agrees with the work of Rozsahegyi (1959), who found long-term pathological drunkenness and alcohol intolerance among caisson workers following decompression sickness.

Naturally, the above findings are not clear cut, and more than one factor is probably involved. Such factors may include the types of gases breathed, the rate of compression, decompression techniques, hyperbaric pressure per se etc., as well as social factors brought about by confinement and working away from home, although I would be inclined not to read too much into the latter problems. Divers, on the whole, are psychologically very robust individuals.

What I suggest we are seeing here is psychological dysfunction resulting from the physiological changes brought about by diving techniques. In other words, a post-dive syndrome. When the diver's physiology returns to normal the dysfunction disappears. Permanent, or long-term, psychological dysfunction may arise in cases of decompression sickness, especially with incidents of Type II Central Nervous System DCS. The question which follows on from this is: are we also witnessing more/or less long-term psychological dysfunction arising from accumulative sub-clinical damage? This remains to be seen.

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CHROMOSOME CHANGES IN DIVERS

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ABSTRACT

Examination of the chromosomes of T-lymphocytes from 77 air divers, 76 mixed gas divers, 75 oil industry controls and 52 other controls showed that in most cases divers have similar levels of chromosomal aberrations to members of the control groups. A few divers (2 air, 4 mixed gas) contain a few cells with many chromosomal aberrations (HDCs).

Resurvey of 6 divers, who originally had HDCs, after $3\,{}^{3}/_{12}$ - $5\,{}^{5}/_{12}$ years revealed that the HDCs had completely disappeared but that 2 other divers and 1 control individual did contain HDCs. While it is clear that HDCs occur both in divers and non-divers, the frequency of these cells may be higher in divers.

Various hypotheses are examined for the origin of HDCs. It is concluded that free radical attack or gene amplification events are the most plausible explanations. The possible significance of these cells for long term diver health is briefly discussed.

INTRODUCTION

Genotoxicology is the study of agents in the environment, including the work environment, which bring about alterations to the genetic information present in cells. Such alterations include gene mutation, chromosome breakage and exchange and changes in chromosome number. Any of these changes may occur in cells of the germ line or the soma but the consequences of their production may be quite different. Changes in the germ line may bring about reduced fertility due to cell killing or may result in an increase in inherited

SIMULATED DEEP DIVING AND COGNITIVE PERFORMANCE

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ABSTRACT

This paper describes the mental performance of human male divers during a series of eleven dives simulated in a pressure chamber. Eight of the dives were in oxyhelium to maximum depths that ranged from 300 metres of sea water (msw) to 540 msw. Our results showed that divers' mental abilities were impaired by about 15 to 20% at 300 msw. Impairments were greater with greater depths, and performance at 540 msw was impaired by as much as 80% in some cases. At 300 msw performance after 24 hours was better than that shown on initial arrival at this depth. At stable depths of 420 and 540 msw performance deteriorated over time. These results could not be explained by sleep loss or mood changes in the divers, the time of day that testing was carried out or by the long periods that divers are required to spend in a cramped environment.

One dive to 660 msw investigated claims by Bennet that an alternative mixture, TRIMEX (helium, oxygen and nitrogen) may prove beneficial. However in this dive performance was worse than that found in oxyhelium at equivalent depths.

One other 'control' dive is described. This involved a maximum depth of 61 msw in oxynitrogen, where the partial

pressures of these gases were identical to those in the deep TRIMIX dive. This dive suggested that the earlier results with TRIMIX could not have been due simply to the partial pressure of nitrogen involved. They were much more likely due to an interaction between the gas mixture and overall pressure. The 61 msw dive also showed that performance impairments at 55-60 msw in air are of the same order as those found at 300 msw in oxyhelium.

In open sea diving, errors of judgement could prove dangerous or even fatal. Performance in the open sea is unlikely to be any better than that shown in a pressure chamber. Our results therefore have important implications for maximising the safety of human divers in an extremely hazardous environment.

INTRODUCTION

In order to survive the diver must breathe a gas mixture that is at a pressure equal to the external pressure on his body. If the gas is pure oxygen, depths of 15 to 20 metres of sea water (msw) can cause oxygen poisoning leading to convulsions and even death. If a diver breathes compressed air, then at depths exceeding 30 msw he will show symptoms of nitrogen narcosis; a state similar to drunkenness produced by the nitrogen in air. For a trained diver, the maximum practicable working depth on air is around 50 msw (Shilling, Werts and Schandelmeier, 1976). This depth has been adopted in the Diving Operations at Work Regulations 1981.

Even at this depth, the increase in gas pressure results in an increase in gas density, causing resistance to gas flow in the breathing apparatus and lungs. If a diver is working hard at 60 msw, the resulting loss of respiratory efficiency can be quite distressing. In addition, decompression has to occur over a lengthy period to avoid 'the bends'.

The problems of narcosis, gas density and bubble formation can be significantly reduced by replacing the nitrogen in air with helium. The use of an oxygen helium mix has enabled commercial, open sea diving to depths of

around 180 msw, although even this gas mixture is not without problems. Rapid compression with oxyhelium can result in tremor, vertigo, nausea and a general impairment in performance; symptoms that are collectively known as the High Pressure Neurological syndrome (HPNS). The effects can be minimised by careful control of the compression profile where periods of compression are interrupted by lengthy 'holds' to enable recovery from the effects of HPNS. Under these conditions, a number of dives have been simulated in a pressure chamber to depths in excess of 500 msw. These depths require several days for safe compression and in the case of the deepest dives, several weeks for decompression.

It is therefore possible for a diver to reach considerable depths, while minimising the danger to his physiology. However, even if the severe physiological effects are minimised by such procedures, the diver may still show significant impairments in his cognitive abilities. Any difficulty in his ability to think clearly or remember instructions, could result in errors that may prove dangerous or even fatal under these extreme conditions. Therefore even if the diver is still alive on reaching maximum depth, he maynot remain so for long if his mental state is impaired. This paper reports the cognitive performance of a number of divers over a series of simulated dives ranging from 300 to 660 msw. The aim is to investigate the extent to which mental or cognitive performance is impaired at high pressure.

Design of performance tests

How can be best measure the mental state of a diver? One way would be to give him a task that he would be required to carry out during the normal course of his duties. However, the particular task chosen may not be sensitive to the effects of depth. Also even if we found that divers were impaired on such a task, it would be unwise to assume that such a result would apply to any other task that he might be required to carry out. The normal practice therefore is to choose tests that measure the mental skills that may underlie the many different jobs a diver is required to do. These tests can then be taken as indicators of mental state at any one time in a similar way that a thermometer serves as an indicator of one aspect of physiological state, namely temperature.

Generally, the tests will have been used by other investigators in a wide variety of applications, and we would be fairly confident that they measure as wide and representative sample of mental performance as is practicable.

Difficulties in Diving Research

Research in this area presents major problems of experimental design. Normally in such studies it is desirable to vary the order in which dives at a given depth take place. For example, divers may show an improvement in performance at depth because of prior practice on the tests used in earlier dives. However for reasons of safety, the diving programme must begin at a relatively shallow depth, with depth increasing on later dives if all goes well. Second, for practical reasons (the dive series took place over several years), and to minimise the risk to any one diver, it is not possible to have the same individuals tested at each depth or on every dive. Therefore there is a danger that any effects of differing depths may be due to differences between divers in their ability to cope with stress. Third, a given diving chamber can typically accommodate two or at most three human subjects on any one dive. This research is extremely expensive (in excess of £100,000 per dive), and the number of suitable chambers is small. Therefore the research involves very small numbers of subjects. This makes it difficult to predict whether our results could be applied to divers other than the ones we tested.

We have dealt with these problems in part by examining performance over a series of eleven dives, involving a total of 16 subjects (24 man-dives were completed in all). In some cases this has allowed comparisons between the same subjects on different dives and at different depths. This is a small sample given the complexity of the research questions involved, but is a large sample by the standards of diving research.

The dives reported were all carried out with the support of the procurement executive, Ministry of Defence, at the Admiralty Research Establishment Physiological Laboratory at Alverstoke, Hampshire. The psychological testing was only one of a number of studies for which these dives

were carried out. The dives also studied effects on the nervous system, haematology, respiratory physiology, metabolism and biochemistry. We had little or no control over the design of the dives and particularly during the early dives, we were permitted very little time during each dive to carry out the cognitive performance testing.

Initial Questions

This review is organised along the lines of specific research questions, and we have attempted to present these in a reasonably logical order. The first three questions are relatively straightforward and provide a basis for the remainder of the review.

(1) Despite the control of the symptoms of HPNS, are there nonetheless detectable impairments in cognitive performace when breathing oxyhelium at pressure? If so, which functions are impaired?
(2) Are there mood changes at high pressure and are these related to changes in performance efficiency?
(3) Are there alterations in sleep pattern at high pressure? If so, are these related to cognitive and mood changes at depth?

DIVES 1-5

Procedure

We attempted to answer these first questions by collecting information from a series of five dives, ranging in depth from 300 to 540 msw. This involved seven different subjects over ten man-dives. Results from these dives were originally reported by Lewis and Baddeley (1981).

Test battery Cognitive performance was measured using an extensive selection of tests. For the purposes of this paper we shall describe three of these namely adding, visual search and grammatical reasoning. The adding test involved presenting the divers with a sheet containing columns of five 2-digit numbers. The diver was required to add up the numbers in as many columns as possible over a 15 minute period. The visual search task involved presenting a sheet containing strings of random letters. Occasionally one letter was repeated, for example

GBAWKFFONDHT. The diver was to detect and mark as many repeated letters (FF in the example) as possible in two minutes. In the grammatical reasoning test, the diver was presented with short sentences followed by a letter pair. The diver had to assess whether the sentence described the order of the letter pair. For example: A FOLLOWS B - AB (false); B IS NOT PRECEDED BY A - BA (true). The remaining tests are described in Lewis and Baddeley (1981).

In addition two questionnaires were completed by the divers. One of these was concerned with the quality of sleep, and the other with their mood state. These questionnaires were completed every day from about one week before the dive through to about one week after the dive was completed.

During each of the dives, four testing sessions were used. The first session occurred prior to the dive and acted as a training session. Session two took place about one week before the dive. Session three took place approximately 24 hours after reaching maximum depth and session four occurred during the decompression phase at a depth where any impairments should be minimal.

Results and Discussion

Results from the cognitive performance battery suggested that there were indeed detectable impairments at depth in oxyhelium. The majority of our tests showed some impairment with depth and Figure 1 shows the results for the adding task. However, the effects were not shown by all of the tests. For example grammatical reasoning (shown in Figure 2) appeared to be unaffected at depth. In addition, even for those tests that were affected overall, it was clear that the individual divers varied widely, not only in their overall ability on the tests, but also in their sensitivity to the impairments associated with breathing oxyhelium at pressure.

Results from the questionnaires suggested that sleep quality did deteriorate, particularly during the compression phase, while mood varied very little during the dive. However, neither measure was related to the changes in cognitive performance during the dive.

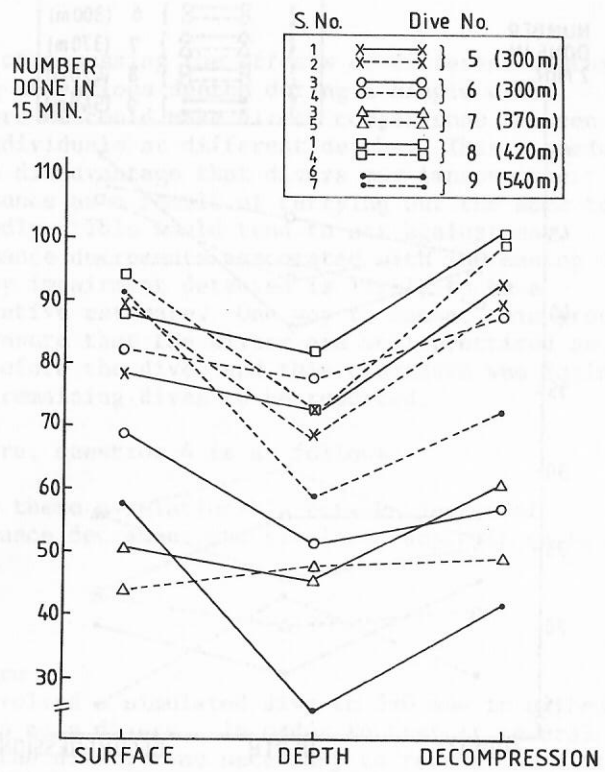

Figure 1. Adding performance in Dives 1 - 5.

However, neither measure was related to the changes in cognitive performance during the dive.

It appears then that we have fairly clear answers to our first three questions. Yes, there are decrements in performance with depth. Mood state is largely unaffected although sleep quality does appear to suffer. Finally the performance decrements cannot be explained by changes in sleep quality or mood.

The next stage was to look at the performance decrements in rather greater detail. In particular, there were impairments on most tests when the data were averaged across all ten man-dives. Obviously it would be desirable to have some indication as to the impairments

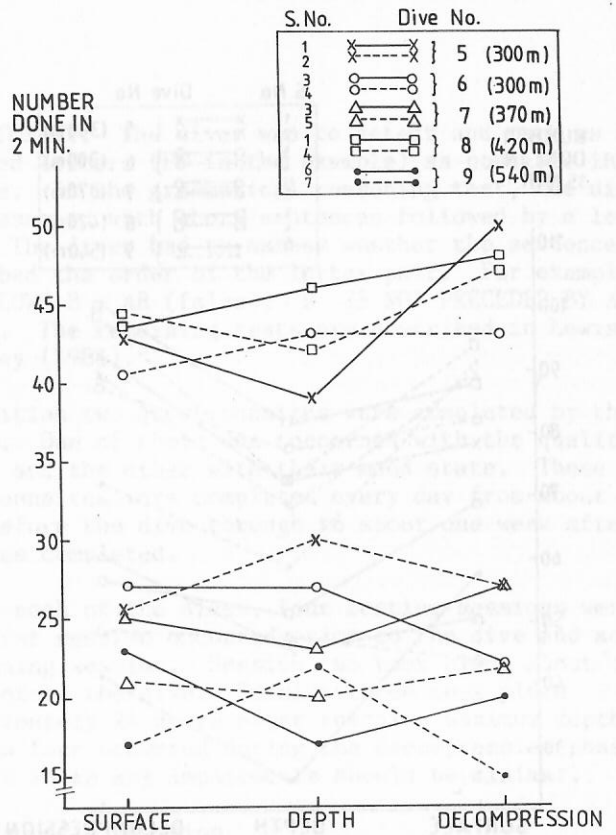

Figure 2. Grammatical reasoning in Dives 1 - 5.

associated with different depths. It is possible to gain a general impression from this first series of dives, and an analysis across dives suggested that impairments on some of our tests, including adding, varied with depth. However, Figures 1 and 2 show that individual divers differed widely in the extent to which they were affected at depth. These 'individual differences' may underlie any between-dive differences and as such, it would be unwise to place too much weight on these comparisons.

In Dives 1 to 5 we had available only a limited time for testing during each dive. However, since it appeared that our preliminary results were likely to have important implications for diving, in the subsequent dives the time available for testing was rather more extensive. This allowed for a more sophisticated

method of assessing the effects of different depths by testing at various depths during a single dive. Therefore we could make direct comparisons between the same individuals at different depths. This procedure has the disadvantage that divers may improve their performance as a result of carrying out the same tests repeatedly. This would tend to act against any performance decrements associated with increasing depth. Thus any impairment detected is likely to be a conservative estimate. One way to combat this problem is to ensure that the divers are well practiced on the tests before the dive, and this procedure was followed in the remaining dives to be reported.

Therefore, question 4 is as follows:

(4) Is there a relationship between degree of performance decrement and simulated depth in oxyhelium?

DIVE 6

Procedure

This involved a simulated dive to 540 msw in oxyhelium, with two male divers. In order to test at several points during the dive it was necessary to reduce the size of the test battery. This was done so as to ensure that the modified battery would continue to test a wide range of cognitive abilities, but still allowing comparisons with a subset of the tests used in the earlier dives. The mood and sleep questionnaires were also included.

The divers were tested at 300 msw and 420 msw during compression, at maximum depth of 540 msw and at 420 msw and 300 msw during decompression. In addition, surface control testing took place before and after the dive, and during the last stages of decompression. This dive was first reported in Logie and Baddeley (1985).

Results and Discussion

Results for the adding test and grammatical reasoning test are shown in Figures 3(a) and 3(b) respectively. As before, there are individual differences in each diver's sensitivity to the effects of pressure on any

particular test. However, the general pattern across divers is fairly consistent. It appears that neither test was dramatically affected at 300 msw. On initial exposure to 420 msw, neither test is particularly affected. However, with increased exposure to this depth, both tests show a steady decline. Compression to 540 msw shows a further decrement, and it appears that continued exposure for several days at this depth resulted in a substantial impairment.

As before, sleep and mood changes were unrelated to changes in cognitive performance.

Unlike Dives 1-5, the grammatical reasoning test was affected in Dive 6. One possible reason may be the increased sensitivity of using a profile of depth against performance, a conclusion that supports the use of this procedure. However, by no means all the tests showed such consistent decrements. For example one diver was largely unimpaired at any point in the dive on the visual search task (see Figure 3(c)).

These data supported our earlier results in showing impairments at depth in oxyhelium. It is now clear that on most of our tests there is some relationship between degree of decrement and depth. It is also clear that the length of time at a given stable depth can also affect performance. There are a number of confounding factors in any comparison between dives, because of differences between dive procedures. It is encouraging therefore that some consistency appears across dives.

DIVE 7

One potentially important factor in these dives is the length of time spent in the relatively cramped space of a diving chamber ('caging'). This may be several weeks in the case of the deeper dives, and it may have had an effect on performance independently of any effects of depth. This is unlikely to account for all of the decrements shown since performance during the later stages of the dive (during decompression) is generally better than that at maximum depth, which occurs only a few days after the start. However, there may be some

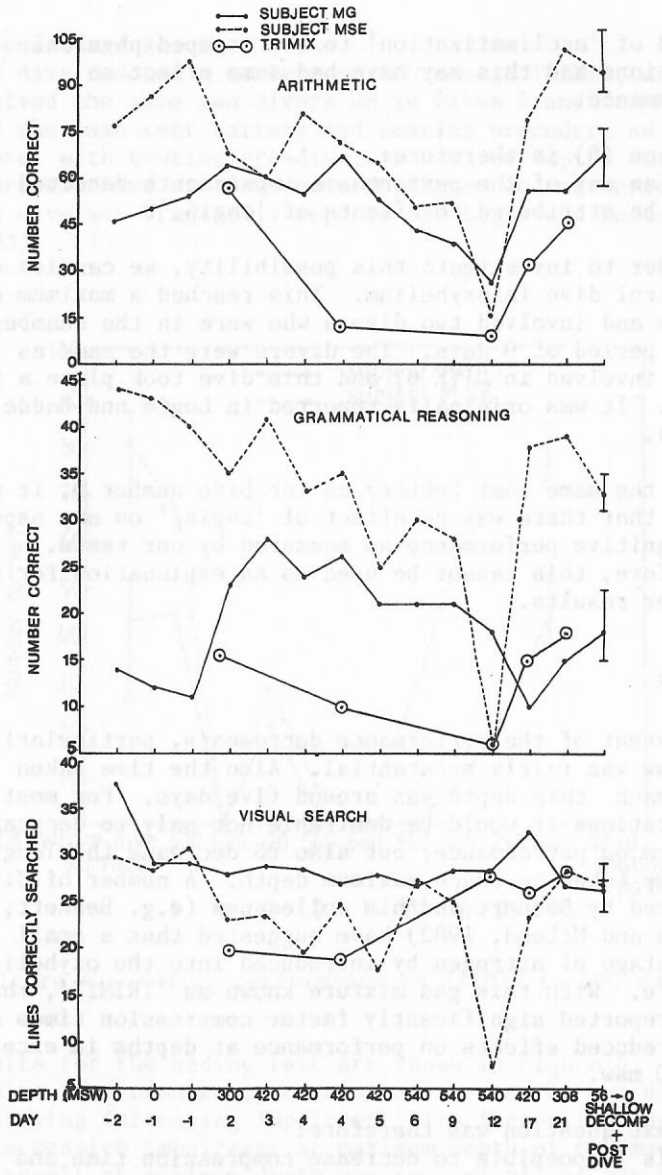

Figure 3. Performance in oxyhelium and Trimix to 540 msw for (a) Adding, (b) grammatical reasoning and (c) visual search.

period of 'acclimatization' to the cramped physical conditions and this may have had some effect on performance.

Question (5) is therefore:
(5) Can any of the performance impairments detected at depth be attributed to effects of 'caging'?

In order to investigate this possibility, we carried out a control dive in oxyhelium. This reached a maximum of 6 msw and involved two divers who were in the chamber for a period of 9 days. The divers were the same as those involved in DIVE 6, and this dive took place a year later. It was originally reported in Logie and Baddeley (1985).

Using the same test bettery as for Dive number 5, it was clear that there was no effect of 'caging' on any aspect of cognitive performance as measured by our tests. Therefore, this cannot be used as an explanation for our earlier results.

DIVE 8

The extent of the performance decrements, particularly at 540 msw was fairly substantial. Also the time taken to reach this depth was around five days. For most applications it would be desirable not only to decrease effects on performance, but also to decrease the length of time taken to reach maximum depth. A number of dives reported by Bennett and his colleagues (e.g. Bennett, Coggin and McLeod, 1982) have suggested that a small percentage of nitrogen by introduced into the oxyhelium mixture. With this gas mixture known as 'TRIMIX', they have reported significantly faster compression times and much reduced effects on performance at depths in excess of 600 msw.

Our next question was therefore:
(6) Is it possible to decrease compression time and ameliorate the decrements associated with depth, by using a helium-oxygen-nitrogen mix (TRIMIX)?

Procedure and Results
This dive reached a maximum of 660 msw in TRIMIX, and involved the same two divers as in Dives 6 and 7. We used the same test battery and testing procedure as before, with testing pre-dive, at various depths during compression and at these same depths on decompression. This dive was originally reported in Logie and Baddeley (1983).

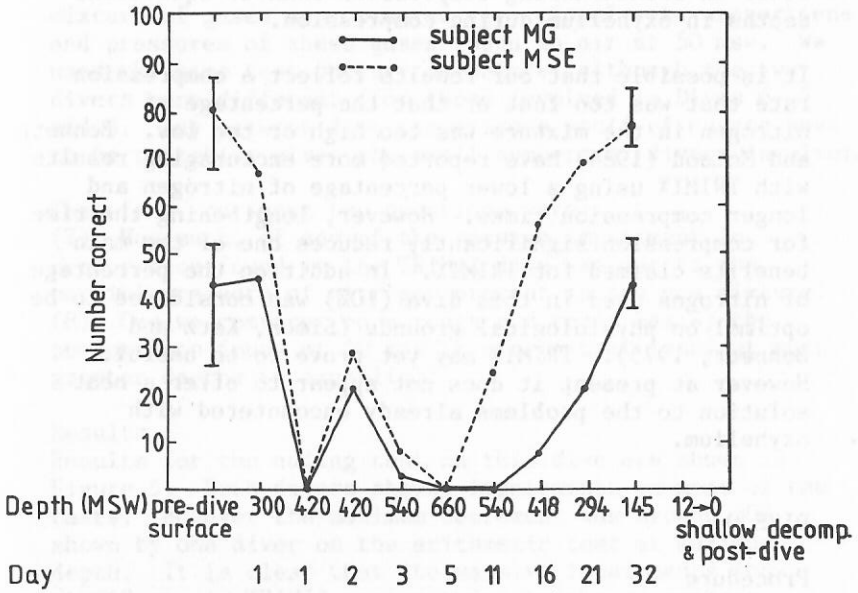

Figure 4. Adding performance in TRIMIX to 660 msw.

Results for the adding test are shown in Figure 4. Results were remarkably consistent across the two divers in showing increasing impairment with increasing depth, and a massive impairment at maximum depth of 660 msw. The result at maximum depth was true of all of our tests. Indeed one subject was unable even to attempt the tests.

It would be useful to have a direct comparison between

performance in oxyhelium and in TRIMIX at equivalent depths. Mean data for the two divers in TRIMIX are shown in Figure 3 for arithmetic grammatical reasoning and visual search. It is clear that performance in TRIMIX was noticeably worse than in oxyhelium for adding and grammatical reasoning at 420 msw and 540 msw. Only after several days at 540 msw in oxyhelium did performance deteriorate to the levels found after 24 hours at 540 msw in TRIMIX. With the visual search task, the difference between gas mixtures was less marked, but was nonetheless lower in TRIMIX during compression than at equivalent depths in oxyhelium during compression.

It is possible that our results reflect a compression rate that was too fast or that the percentage of nitrogen in the mixture was too high or too low. Bennett and McLeod (1984) have reported more encouraging results with TRIMIX using a lower percentage of nitrogen and longer compression times. However, lengthening the time for compression significantly reduces one of the main benefits claimed for TRIMIX. In addition the percentage of nitrogen used in this dive (10%) was considered to be optimal on physiological grounds (Simon, Katz and Bennett, 1975). TRIMIX may yet prove to be useful. However at present it does not appear to offer a neat solution to the problems already encountered with oxyhelium.

DIVE 9

Procedure

The gas mixture in the TRIMIX dive was made up such that at the maximum depth of 660 metres (around 65 times atmospheric pressure), the majority of the pressure was due to oxyhelium, and 0.4 bar (atmospheric pressure =1 bar) was oxygen. Some 10% (around 6 bar) of the pressure was made up with nitrogen. It is not clear whether the decrements found in TRIMIX were due to the pressure of nitrogen alone or some combination of the overall pressure and the presence of nitrogen.

In Dive 9 the gas mixture was designed to equate the pressure of nitrogen with that used in the TRIMIX dive. As such this next dive (Logie and Baddeley, 1985) involved a maximum depth of 61 msw (around 6 bar) in a mixture of oxygen and nitrogen.

This dive also had a secondary purpose. As we mentioned in the introduction, the extent of performance impairment using compressed air at depths around 50 or 60 msw are relatively well known (Shilling, Werts and Schandelmeier, 1976). These have been used to establish the 50 msw recommended maximum on air. Therefore it would be useful to compare the performance decrements obtained in oxyhelium at depths on air. At one stage in the dive the mixture of gases approximated very closely the proportions and pressures of these gases found in air at 50 msw. We used the same test battery as before, although the two divers were different from those involved in Dives 6, 7 and 8. Any between dive comparisons would of course have to be tentative given the small numbers of divers involved.

Therefore, our next two questions were:
(7) How much, if any of the massive impairment in performance found in the TRIMIX dive was due to the partial pressure of nitrogen present in the gas mixture?
(8) Can we compare (on a rough and ready basis) the performance found at 50 msw in air to that found at much greater depths in oxyhelium?

Results

Results for the adding test on this dive are shown in Figure 5. Both divers showed impairments on most of the tests. However the maximum decrement was around 30% shown by one diver on the arithmetic test at maximum depth. It is clear that the massive impairments shown at 660 msw in TRIMIX were not due to the equivalent partial pressure of nitrogen at that depth.
At the simulated depth equivalent to 50 msw in air, neither diver showed impairments on any of our tests. In the oxyhelium dive (Dive 6) we detected very little decrement at around 300 msw, with noticeable and increasing decrements at greater depths. However we should remember that these dives involved different individuals and therefore any direct comparisons should be treated with some caution. Particularly in view of the large differences in sensitivity shown by different individuals on the same dive.

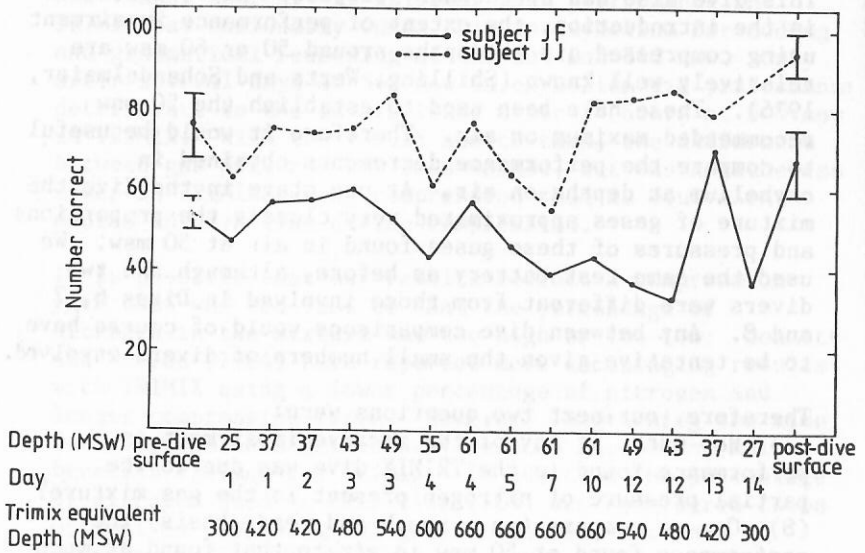

Figure 5. Adding performance in oxynitrogen to 61 msw.

DIVES 10-11

Introduction

In all of the dives that we have reported so far, we have concentrated on the effects of simulated depth on performance, with very little consideration of how a given depth was achieved. Typically, compression consists of periods of compression followed by 'holds' at a given depth. The hold is intended to allow recovery from any effects of HPNS that may have been induced by compression. Thus for example, Dive 1 in this series consisted of a fast compression rate of 1 m/min, followed by a hold at 225 msw for one hour before further compression to 300 msw. Dive 2 was achieved with the same compression rate but with a hold of 24 hours at 225 msw.

Decrements appeared in both dives. However, the individuals who took part in those dives differed widely in their sensitivity to the effects of depth and it was not entirely practical to make meaningful comparisons of performance between these dives because of the large individual differences in performance. Also, only one testing session at depth was possible in these earlier dives.

Therefore it would be useful to examine in a more systematic fashion, the effects on performance of the compression profile: the combination of compression rate and 'holds' at intermediate depths. The relatively shallow depth of 300 msw involves rather less risk to the divers and is flexible enough to allow systematic variation in compression profile in comparatively short dives.

Duration of exposure to a given depth is a further important aspect of the dives we have reported. It was clear from Dive 6 that an exposure to 540 msw for around 6 days resulted in greater impairments in performance than shorter exposure to this depth. It would be useful to know whether exposure duration has similar effects at somewhat shallower depths in oxyhelium, where only small decrements have been observed.

Therefore our final questions in this review are:-
(9) How is performance at depth affected by the profile of compression?
(1)) Does exposure duration affect performance at depths where only small decrements are detected?

Procedure

Two dives were carried out. Both dives involved a maximum depth of 300 msw in oxyhelium, and three divers were involved in each dive. One diver took part in both dives. Dive 10 involved a compression rate of 5 m/min with 'holds' at 120m for 2 hours, at 180 m for 15 hours overnight and at 240 m for 2 hours, giving a total compression time of about 0.82 days. Maximum depth was maintained for 2.36 days. Dive 11 followed the same compression profile. However maximum depth was maintained for 5.9 days.

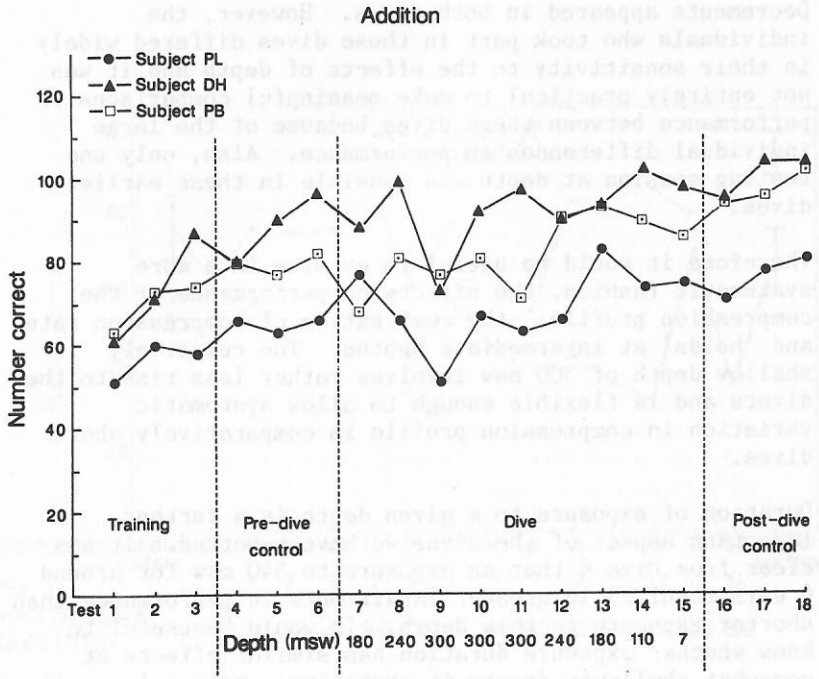

Figure 6. Adding performance in oxyhelium to 300 msw, in Dive 10.
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A more detailed report of these dives is given in an unpublished report by Williams (1986).

Results

Results for the adding test are shown in Figures 6 and 7 for Dives 10 and 11 respectively. As previously there are large differences in the results for different divers. However in Dive 10, two out of three divers showed decrements on arrival at 300 msw, with a suggestion that some recovery occurs after 24 hours. In Dive 11, only one diver showed performance impairment on arrival, with recovery 24 hours later. Only one diver in Dive 11 appeared to show an increasing impairment with continued exposure to 300 msw.

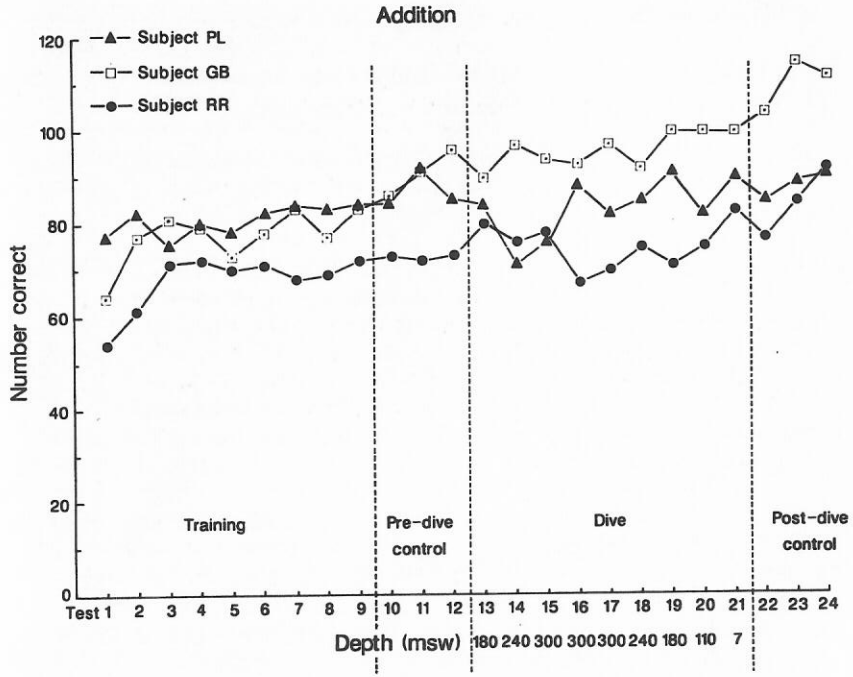

Figure 7. Adding performance in oxyhelium to 300 msw, in Dive 11.
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Dives 1 and 2 involved performance tested 24 hours after arrival at 300 msw. In those dives, decrements were of the same order (15% to 25%) as those obtained on arrival at 300 msw in Dives 10 and 11. In the later dives, a period of 24 hours was sufficient to allow recovery in some divers. However, at least one diver in Dive 11 did not show this benefit. These between dive comparisons must of course remain very tentative. Nonetheless it appears that the particular compression profiles chosen may make a difference to the level of performance at depth. Also, the effects of exposure duration at 300 msw appear to be rather different from those found at 420 msw and 540 msw in Dive 6.

A number of studies (eg. Folkard 1975) have shown that cognitive performance may vary as a function of the time

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PREDICTING TARGET DETECTION DISTANCES IN VISUAL SEARCH TASKS UNDER WATER

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ABSTRACT

The prediction of visual performance in dynamic viewing conditions has been viewed as an engineering problem that can always be solved if adequate input data are available. Consequently there is a temptation to modify mathematically the simple model that exists for static viewing conditions, until observed and predicted performance match. Experiments were conducted with experienced divers in a swimming pool and a laboratory (using an aquarium) in order to determine the detection distances of various targets when the target or diver was in motion. The data did not agree with predictions calculated by incorporating the specified corrections to the basic model. The data are discussed in relation to the potential use of visibility models in search operations.

INTRODUCTION

Visibility is an important factor to be considered when planning an underwater search. For example, knowing the maximum distance at which divers can detect an object might help to minimise the spatial overlap between successive search sweeps or lines of a search grid. Considerable advantage could be gained if the visibility could be predicted without first having to send divers into the water. A model for doing this has been available for many years (Duntley, 1960), but has been little used, partly because it left unsolved the problem of how to measure some of the necessary optical characteristics of the water. Recent research has shown that this difficulty can be overcome (Emmerson, in press). The model was primarily designed for use with static observers and targets, but it was considered possible to