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Edited by J. C. Partridge and S. I. Rogers

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Tc-99m HMPAO AND SPET: A NEW METHOD FOR IMAGING CEREBRAL PERFUSION DEFECTS FOLLOWING DIVING CASUALTIES

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DIVING TRENDS

Accepting that man is a land animal, rather than an aquatic animal, man's excursions into the oceans, throughout history, have been limited more by technology than by human physiology. It is only within the last hundred and fifty years or so that technology has begun to outpace us. Today, we are faced with a new breed of diver armed with higher pressure bottles, warmer suits and an array of new equipment designed to allow deeper and longer diving while keeping decompression to a minimum. We know that sport diving groups are changing the way they dive and nowhere is this more evident than in the new BSAC '88 decompression tables or in the new PADI "Wheel" for multilevel diving. We must be aware of these trends and be knowledgeable in our efforts to express a considered, cautious, alternative view.

To understand where we are going, it is often necessary to look back and see where we have been. We are well aware of an increase in the number of diving accidents over the last few years but of more concern is that the pattern of illness has changed as well. Studies throughout the years have divided decompression accidents into non-serious Type I (pain only) and more serious Type II (neurological/pulmonary) cases. Historically, the percentage of serious decompression illness was stated at around 30%. Different population groups, data interpretation, what constitutes a Type II lesion and differing exposures all make comparisons of these studies difficult. There is clearly, however, a changing pattern of diving and it is evident that the numbers, within recent years, have been changing. The 1988 statistics from the Royal Navy state that advice was given to 149 recreational divers with 110 resulting treatments. Eighty-six of these were diagnosed as neurological DCS, a total of 78%. In excess of 90% of the patients we treated last year displayed some

symptom of Type II decompression sickness. What does this trend towards more serious illness mean?

In 1947 Dr Al Benhke stated that "The chronic cerebral lesion is practically unknown, and symptoms pointing to acute focal injury of the brain are rare." We now have evidence that suggests every case of neurological decompression sickness, no matter how slight the symptoms or signs, involves damage to the brain. We are examining divers using a new method of looking at cerebral blood perfusion. Where blood goes, oxygen goes, and where blood flow is reduced, oxygen may be reduced and the result, we believe, is impaired function or death of tissue with resultant symptoms of decompression sickness. This method utilises the radiopharmaceutical Tc-99m HMPAO with Single Photon Emission Tomography (SPET).

Tc-99m HMPAO WITH SPET IMAGING

The development of a new imaging technique with Tc-99m labelled hexamethylpropyleneamine oxime (Tc-99m HMPAO) and single photon emission tomography (SPET) has allowed for *in vivo* imaging of cerebral perfusion defects secondary to diving accidents for the first time. Dysbarism presents in two major forms; barotrauma and decompression sickness (DCS). Barotrauma involves tissue damage as a direct result of a change in volume of entrapped gas, whereas DCS is a complex series of events initiated by formation of inert gas bubbles during or following decompression. The most significant form of barotrauma is "pulmonary over-inflation syndrome" which often lead to cerebral arterial gas embolism (CAGE), a potentially lethal complication. Decompression sickness presents in a wide variety of forms. Dysbarism, particularly DCS, while primarily a disorder of divers and compressed air workers exposed to increased pressures, can also occur in aviators as a result of decompression at high altitudes.

There is growing concern that dysbarism can lead to long term neurologic damage. Post-mortem studies of the spinal cord have suggested that significant pathological changes in the CNS have occurred even when a good recovery appears to have taken place (Calder, 1983; Palmer *et al.*, 1981; Palmer, 1990). *In vivo* studies have demonstrated little of pathological significance despite the use of the most advanced imaging methods e.g., CT, Radionuclide Imaging and Nuclear Magnetic Resonance Imaging, and despite well-defined clinical symptoms and signs of central nervous system involvement.

The search for an ideal agent to assess regional cerebral blood flow and thus allow for routine imaging of perfusion defects has been going on for some time. While some agents have existed, they have been difficult or expensive to use. The characteristics of an ideal agent are:

1. The molecule should be neutral and lipophilic to enable passive diffusion across the lipid bilayer.

2. The extraction efficiency of the molecule must be high.

3. Once trapped, the distribution of the molecule must remain effectively unchanged, at least over the time frame of the imaging procedure.

4. Clearance from brain tissue should be slow.

5. The radioisotope used should be continuously available and have physical characteristics suitable for high resolution gamma camera imaging.

6. The molecule must be easy to use and safe (Ell *et al.*, 1987).

Tc-99m HMPAO SCINTIGRAPHY

Hexamethylpropyleneamine oxime (HMPAO), marketed under the brand name Ceretec by Amersham International plc. in England, appears to meet the above demands.

When combined with Technetium-99m, an isotope used in 80% of all nuclear medicine procedures, HMPAO penetrates the blood barrier and becomes fixed in cerebral tissue with no significant redistribution (Volbert *et al.*, 1984; Ell *et al.*, 1985; Costa *et al.*, 1986). Its half-life within the tissues is in the order of 40 hours and imaging can be performed within minutes or can be delayed several hours following injection. Imaging gives a picture of cerebral perfusion pertaining at the time of injection rather than at the time of imaging. This has the distinct advantage that a diving casualty may be injected prior to recompression therapy and imaged hours later in the controlled environment of a nuclear medicine department. Single photon emission tomography (SPET) is performed using an orbiting gamma camera of the type found in most nuclear medicine departments. A sinogram is acquired using 360^{o} forward rotation, 64 projections on a 64 x 64 matrix and 20 second acquisition time per projection. The reconstructed image contains 32 axial slices. Repeat studies can be performed at weekly intervals to monitor changes in perfusion patterns. Using this technique, the Nuclear Medicine Department at Haslar Royal Naval Hospital, Gosport, England, has imaged over forty divers following dysbaric accidents, and perfusion defects, previously unseen in diving accidents, have been documented.

CASE STUDIES

Case 1. A 33 year old male was performing a free in-water ascent from a depth of 18 metres during submarine escape training. At the surface, he noted weakness with unusual sensation in his right leg and dyspraxia of the right arm. He was treated by recompression to 50 metres at the scene of the incident with resolution of his symptoms within minutes. He was decompressed over 5 hours and admitted to hospital immediately upon completion of therapy. At that time, detailed examination of the CNS was negative as were an EEG,

ECG and cerebral CT scan. A CT Scan of the thorax showed old opical scarring and several intrapulmonary bullae. The diagnosis was cerebral arterial gas embolism (CAGE).

The Tc-99m HMPAO SPET scan obtained showed a hypoperfused area in two axial slices in the left fronto-parietal region. A second study, performed one week later, showed some improvement in perfusion in the left fronto-parietal lesion but also showed a new ischaemic area in the left posterior parietal region.

Case 2. An 18 year old male ascended from 28 metres during submarine escape training and was observed to hold his breath over the first 9-10 metres of the ascent. Upon surfacing he complained of tingling and weakness of his left arm. He was immediately recompressed to 50 metres with resolution of all symptoms within 15 minutes. Following decompression he was admitted to hospital where neurological examination was normal in all respects. The diagnosis was CAGE.

Trans-axial and coronal slices obtained from the SPET study performed showed two areas of diminished perfusion, one in the right frontal lobe and the other involving the right parietal region.

Case 3. A 47 year old female recreational diver undertook a series of dives to depths as great as 34 metres, over a period of several days, for which she had inadequate decompression. Following her final dive, she complained of stocking/glove paraesthesiae, nausea, vertigo, mild imbalance with slight weakness in her left leg, confusion and slightly blurred vision. She was treated by recompression to 18 metres and experienced complete relief of all symptoms. She was treated on a USN treatment Table 5 rather than on the required Table 6 and, despite a recurrence of several symptoms during decompression, was surfaced and released from care with a refusal from the chamber to treat her further. Symptoms continued for several days during which time she arranged air ambulance transportation to the UK. During the flight home she experienced a worsening of symptoms and signs and the aircraft was forced to fly below 300 metres to alleviate these. She was retreated upon arrival in the UK with resolution of her nausea and confusion but with little change in the parasthesiae. Recompression treatment was discontinued when she developed signs of oxygen toxicity. She was admitted to hospital with complaints of parasthesiae in a stocking/glove distribution. Neurological examination including EEG was normal. The diagnosis was neurological DCS.

Axial images obtained from the first study on Case 3 showed ischaemic areas in the left frontal and left parietal regions and in the right posterior parietal region. A second study, done one month later, showed continued ischaemia in all regions primarily affected with some extension of the ischaemic borders.

Case 4. This 23 year old female sports diver did two dives to a depth of 20 metres for a total of 42 minutes. She felt unwell

following the dives but attributed this to substantial alcohol intake and gastro-intestinal upset. She noted increasing lassitude, marked mental confusion, decreased vision and parasthesia developing across both feet over the next day. Examination 36 hours post-dive revealed a decreased sensation of the right foot along with generalised hyper-reflexia and mental changes involving memory and concentration difficulties. She was treated on a Royal Naval Table 62 with complete resolution of her symptoms. Forty-eight hours post-treatment she had a slight recurrence of symptoms and she was retreated on a modified Royal Naval table for 90 minutes at 18 metres. She again had complete resolution of symptoms without further recurrence. Follow-up examination was completely normal. The diagnosis was DCS with cerebral involvement.

Axial images revealed a large ischaemic lesion in the left fronto-parietal region with evidence of an infarct laterally. A follow-up scan one month later revealed minor resolution of the left fronto-parietal region ischaemia but confirmed the presence of an infarct in the region.

DISCUSSION

The ischaemic lesions demonstrated by HMPAO scanning in Cases 1 and 2 are compatible with the symptoms and signs produced in the right (Case 1) and left (Case 2) side of the body respectively. The location of the lesions in both cases supports a diagnosis of CAGE, almost certainly due to gas bubbles in the distribution of the left middle cerebral artery (Case 1) and the right middle cerebral artery (Case 2). Animal experience suggests that the site most prone to emboli is within the distribution of the middle cerebral artery (Pearson, 1984) and these findings would support this.

The lesions seen in Case 3 are consistent with a more scattered distribution of bubbles. This is consistent with either multiple small inert gas emboli reaching the cerebral circulation or secondary disruption of flow due to autochthonous bubbles in the tissue. A combination of these events may also have occurred. Any gas bubbles within the system in this case would appear to be a result of inert gas loading. There is, at least, indisputable evidence of bilateral cerebral involvement.

The lesion demonstrated in Case 4 is quite different in character from those demonstrated in Case 3. It is a discrete lesion consistent with a blockage of the anterior branch of the middle cerebral artery on the right side. It is virtually identical to the lesions noted in Cases 1 and 2, both of which were due to CAGE. A discrete lesion of this type is more consistent with an embolic phenomenon than with autochthonous bubbles arising in the cerebral tissues. Despite this, however, the latency of onset, the lack of difficulty during ascent and the type of symptoms would argue for the diagnosis of DCS rather than CAGE. It is possible that bubbles arising within the venous system in DCS may be later arterialized and cause a CAGE secondarily.

It is apparent from the recent studies performed on Case 1 and Case 3 that prolonged cerebral ischaemia may result from diving accidents despite apparently good recovery. Evidence obtained in these four cases lends support to the current concern over potential long term effects of diving and diving casualties. It will be of interest to ascertain how long the ischaemic phase lasts when further scintigraphy is performed at 3-4 monthly intervals, post incident, on each subject.

Tc-99m HMPAO with SPET imaging affords the opportunity to observe *in vivo* cerebral lesions for the first time following insult due to dysparic related accidents. It is a potential tool for studying diving casualities and gaining new insights into the pathology involved, optimal treatment protocols as well as examining the long term neurologic effects of diving injuries.

To date, we have imaged over 50 patients who suffered some diving related incident. The results have been remarkably consistent and a clear pattern has emerged in divers scanned by this method. In cases of Type I decompression sickness, no brain lesions have been apparent. In all cases of cerebral arterial gas embolism (CAGE) and in all cases of Type II decompression sickness, no matter how slight the manifestation, cerebral lesions are present and correspond exceedingly well with the symptoms and signs present. This is true even in cases that would normally have been declared pure spinal cord disease.

In most cases the lesions have been remarkably persistent. We are pursuing long-term follow up but we theorise that many lesions persist for long periods of time or may become permanent. We are looking at a retrospective study to evaluate divers who suffered accidents 4 or more years ago.

Of interest is that four of the above cases have been evaluated as possible epileptics. One had a transient lesion, the others are in the process of re-evaluation following their diving accidents.

SUMMARY

We have been concerned as a medical community, that a great deal of long-term neurological damage may be caused by diving, and that it is not always apparent by our existing test procedures. Whether this subclinical damage affects the patient later in life remains a matter of speculation. What does appear to be certain, is that as technology advances and divers are urged to go deeper and stay longer, advancing technology in medicine will allow us to take a more critical look at the effects of this hostile underwater environment on man, the land animal.

Diving is a hazardous sport and a hazardous occupation. Few of us expect or desire the sport diving community to stop diving. We hope, however, that, as a community, they will

recognize the current limitations of human physiology. When faced with the possibility of a few extra minutes on the bottom or the possibility of reducing decompression by a few minutes they will balance the benefit against the potential cost. That cost, all too often is high. It may be a lifetime in a wheelchair, a life altering disability or simply a nagging reminder of a old injury. Whatever the cost, major or minor, it is important to remind the community that the vast majority of so called "accidents" that occur, are in fact, self induced injury that can be avoided with a reasonable degree of caution.

For the working diver the implications may be even greater. As techniques for examining the central nervous system become more sensitive, the question of short- and long-term neurological damage will make an even greater impact on the commercial industries. New standards of medical qualifications may become necessary. Baseline studies of the central nervous system may become the norm and follow up studies at various points in a divers career, or following an accident, may begin to document hitherto unappreciated neurological damage. As this damage is documented, the question then becomes whether the small degree of damage seen in some cases has any functional affect, either in the short- or the long-term health of an individual. These questions will undoubtedly be driven in part by medical-legal and occupational health considerations. The implications for the diving industry are enormous. As man's ability to dive ever deeper into the oceans develops, so does the appreciation of the dangers he faces. Now, more than ever, reasonable caution must be used whenever man, the land animal, enters the water.

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TARGET ORGANS IN DECOMPRESSION SICKNESS

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The purpose of this review is to demonstrate the wide range of organs in the body in which pathological changes have been found as a result of compression/decompression, lesions which result in decompression sickness (DCS) or decompression "illness". Investigations have been carried out on human and animal subjects, the latter studies indicating mechanisms whereby the lesions may arise. The work has involved collaboration with many people including Drs I.M. Calder and H.V. Hempleman.

Organs in man which are well known to be affected as a result of compression/decompression include the skin (erythema), bone (aseptic necrosis), spinal cord (degeneration of white matter) and the ear (the exact nature of the damage has not been established).

Investigations started in 1972 with Dr H.V. Hempleman at the Royal Naval Physiological Laboratory, at Alverstoke, looking into the pathological changes in the spinal cord of goats subjected to experimental compression/decompression (Palmer *et al.*, 1976).

Animals were usually compressed to a pressure equivalent to that exerted by 100 feet of sea water (100 f.s.w.), held at pressure for one hour and decompressed over 2.5 minutes. Out of 56 animals, 7 showed no clinical signs, 25 showed signs of Type I DCS and 24 showed signs of Type II DCS. In the last named group, 20 goats later proved to have lesions in the spinal cord. Surprisingly, 7 out of the 25 animals showing Type I DCS also had lesions in the spinal cord.

An additional group of 17 animals was subjected to the same compression/decompression routine but were killed within 20 minutes of surfacing. In these animals, bubbles were found intravascularly (in both arteries and veins) in most organs of the body. It could not be established whether these bubbles were present in these sites during life. In the spinal cord, the bubbles particularly affected vessels in the white matter (Fig. 1). Sometimes bubbles were also found extravascularly in the matrix of the tissue, but where this occurred these

spaces were usually associated with the histological remnants of a vessel, which may have disintegrated as a result of distension or as an artefact during the preparation of the tissue. Autochthonous bubbles were not a feature of this investigation.

In animals killed 48 hours after exposure, lesions occurred in the white matter of the spinal cord (Palmer *et al.*, 1978). These consisted of discrete foci of infarction, that is to say areas which had been deprived of blood supply and where the nerves and myelin had undergone the early stages of Wallerian type degeneration (Fig. 2). Vasogenic, proteinaceous globules occurred around adjacent blood vessels (Fig. 3) and in some vessels there were microthrombi, small clots lodged inside the vessels (Fig. 4). Ultrastructurally, affected vessels showed loss of endothelium and an aggregation of platelets embedded in the microthrombi (Palmer & Blakemore, 1980).

The size of the infarcts varied from animal to animal. Some only involved a small group of nerve fibres whereas others were widespread and affected a number of funiculi. One notable feature was the preservation of a circumferential margin of white matter, just beneath the pia matter (Fig. 5): this region receives a blood supply from vessels outside the cord.

Within 2 weeks of the original insult, a repair process had started which involved removal of tissue debris by lipid phagocytes followed by a reactive gliosis. In affected regions there was severe depletion of axons, although by using silver staining methods it was shown that a few axons survived.

In view of spinal cord damage being present in some goats suffering Type I DCS, the question arose as to whether the same situation applied to divers. In collaboration with Drs I.M. Calder and J.T. Hughes, I have examined spinal cords from 13 divers. One had been paraplegic for 4 years after a severe Type II episode and there were severe lesions in the dorsal and lateral columns (Fig. 6), similar in nature to those seen in severely affected experimental goats (Calder *et al.*, 1989). Subpial myelin was also preserved. In a second case, an amateur diver suffered a spinal bend but made a good recovery after treatment (Palmer *et al.*, 1981). He returned to work and thought that he had made a total recovery. Four years later he was examined neurologically. He was noticed to be somewhat unsteady when standing on one leg and showed evidence of residual upper motor neuron deficits. He died 10 days later. When his spinal cord was examined there was clear-cut evidence of residual damage in both sensory and motor tracts (Fig. 7).

Subsequently, spinal cords from 3 amateur and 8 professional divers were examined (Palmer *et al.*, 1987). None of the divers had reported episodes of DCS and all had been actively engaged in diving until their demise, apart from one who had retired from diving 18 months earlier. When the spinal cords were examined, using the Marchi technique, degenerating myelin

was found in 3 professional divers, 2 of whom were saturation divers (Fig. 8). This result indicates that divers can be working without realising that their spinal cords are damaged. Brains from some 25 deceased divers are at present being examined.

Three other organs have recently been shown to be affected by DCS, the liver, eye and cochlea.

Rapid decompression in both animals and man can lead to the accumulation of proteinaceous, membrane-bound, spheroidal bodies in the spaces of Disse in the liver (Fig. 9). The origin of these has not been determined. The only other situation in which they have been reported previously has been in livers of patients dying from severe burn injuries (Langlinais & Panke, 1979).

In 1988, Polkinghorne and his colleagues reported vascular and retinal abnormalities in the fundus of the eye in a high proportion of the divers whom they examined. Although vision was not affected, the changes were directly related to the length of time the person had been diving. This research suggested that the changes may be an indicator of infarcts elsewhere, such as in the central nervous system.

Both balance and cochlear function in the ear in man are vulnerable to damage during or after compression. In collaboration with Dr M. Halsey and his colleagues at the Clinical Research Centre, Harrow, Margaret Wilkes and I have studied the ears from 4 minipigs which were compressed to 100 f.s.w. on 21 separate occasions and decompressed conservatively using Blackpool tables (Wilkes *et al.*, 1989). There were also 3 control animals. When hearing was tested by the brain stem auditory evoked potential method, 3 out of 4 experimental animals were deaf; the fourth was deaf in one ear. Severe damage was subsequently found in the organ of Corti using light and scanning electron microscopy (Fig. 10). The immediate cause of the changes was not established but barotrauma, DCS or the formation of bubbles within the endolymph may play a part.

In summary, pathological evidence shows that compression/ decompression in animals and man can lead to degenerative changes in diverse organs of the body. Those who dive should be aware of this evidence and realise that bodily functions can be compromised.

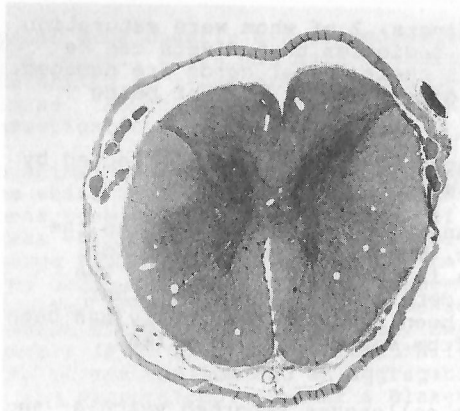

Figure 1. Transverse section from the spinal cord of a goat killed 8 minutes following decompression, having been held previously at a depth of 100 f.s.w. for one hour. Note the dilated vessels in the white matter, presumably distended with bubbles of gas. Haematoxylin and eosin, x 5.

Figure 2. Recent infarcts in the dorsal columns of the spinal cord from a goat, 24 hours after the onset of a spinal bend. Note the clear delineation of the boundaries of the vacuolated necrotic areas and the preservation of the subpial white matter. Haematoxylin and eosin, x 25. (Figure reproduced with permission from *Neuropathology and Applied Neurobiology*, **2**: p. 149).

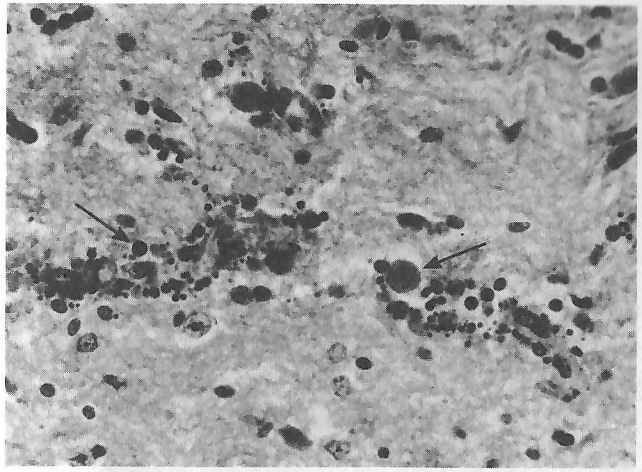

Figure 3. Perivascular proteinaceous globules in the dorsal horn from a goat 48 hours after decompression. The globules are an indicator of vasogenic oedema (leakage from a vessel). Picro-Mallory. x 180.

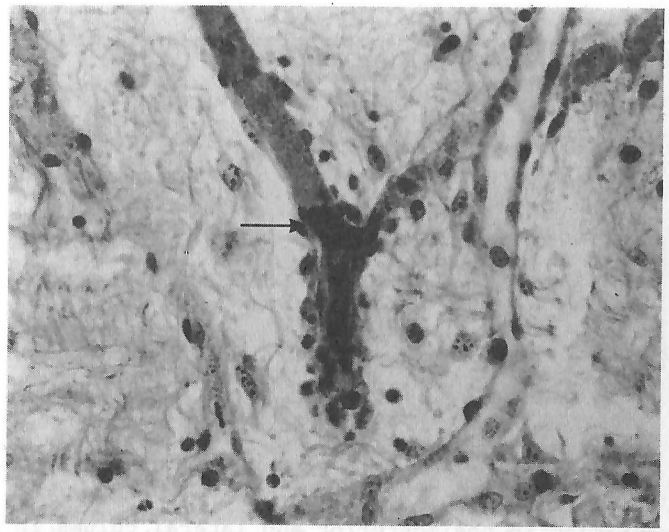

Figure 4. Microthrombus (clot) present at the bifurcation of a vessel in the spinal cord of a goat, 48 hours after the onset of a spinal bend. Picro-Mallory, x 180. (Figure reproduced with permission from *Undersea Biomedical Research*, **5**: p. 283).

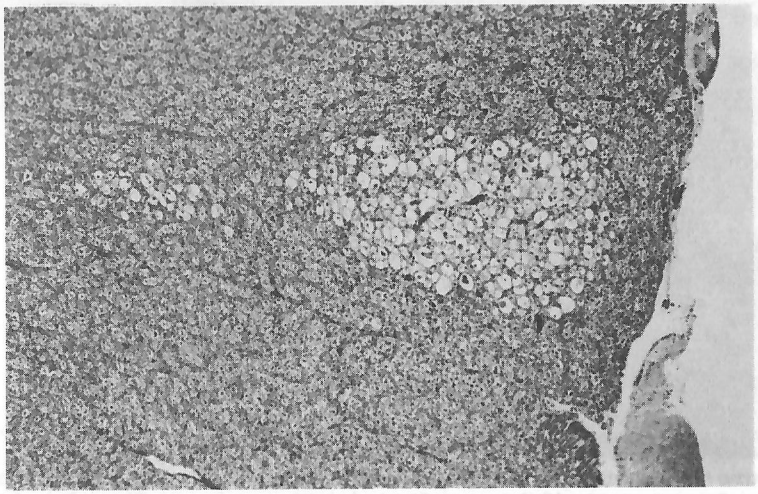

Figure 5. Wedge-shaped infarct in the spinal cord of a goat which showed temporary paralysis of the hind-legs. Within the infarcted area there is early Wallerian type degeneration of the white matter. Note the preservation of subpial myelin. Haematoxylin and eosin, x 27. (Figure reproduced with permission from *Undersea Biomedical Research*, **5**: p. 280).

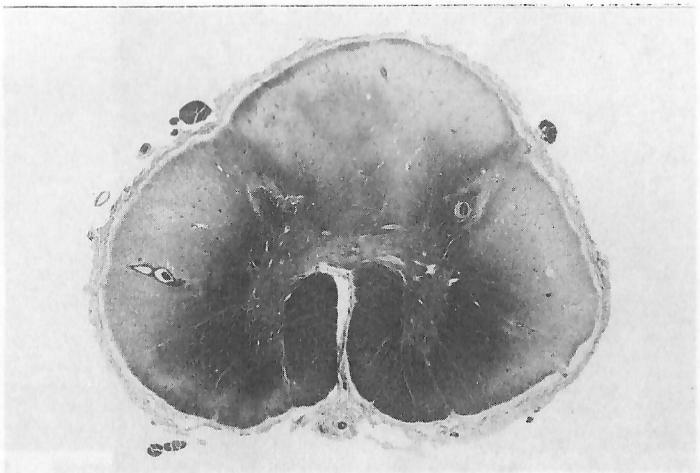

Figure 6. Degeneration in the posterior and lateral colums in the cervical cord of a man who survived for 4 years as a paraplegic after an incident of Type II decompression sickness. Note the preservation of the grey matter and of the subpial myelin. Methasol fast blue, x 7. (Figure reproduced with permission from *Paraplegia*, **27**: p. 54).

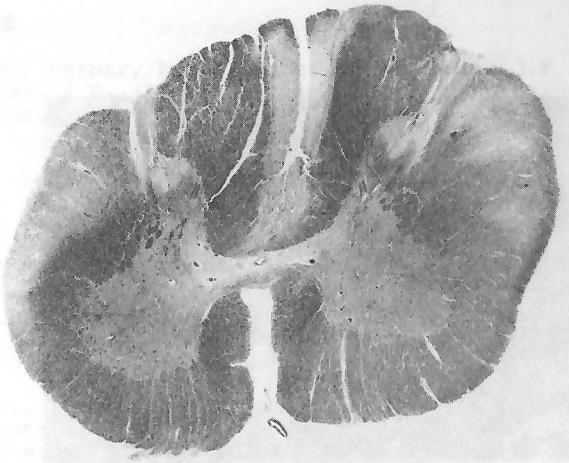

Figure 7. This is a transverse section from the cervical cord from a man who "recovered" from signs of spinal paralysis 4 years previously after an episode of decompression sickness. There is loss of myelin from posterior and lateral tracts. Methasol fast blue, x 6. (Figure reproduced with permission from *British Medical Journal*, **283**: p. 888).

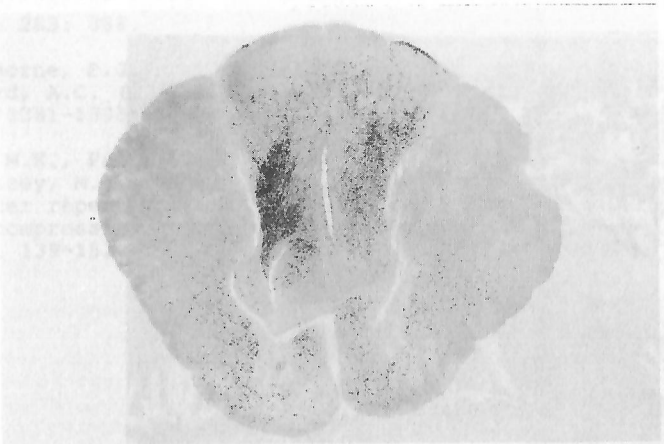

Figure 8. Marchi positive degeneration (staining black) in afferent fibres of the posterior columns, in fibres of Lissauer's tract and also scattered in the lateral and anterior columns. The subject was a professional diver who was active until the time of death. Marchi, x 5. (Figure reproduced with permission from *The Lancet* **ii**: p. 1366).

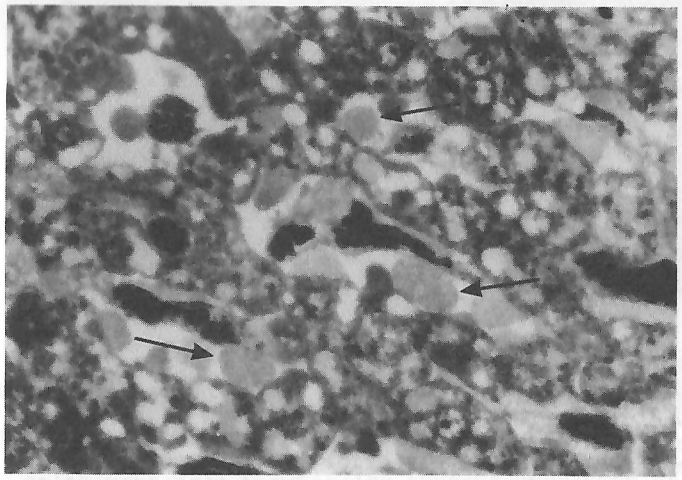

Figure 9. Spheroidal, membrane bound, proteinaceous bodies in the spaces of Disse in liver of a goat killed within a short time of decompression. The origin of these bodies has not been established. Resin embedded, toluidine blue, x 50.

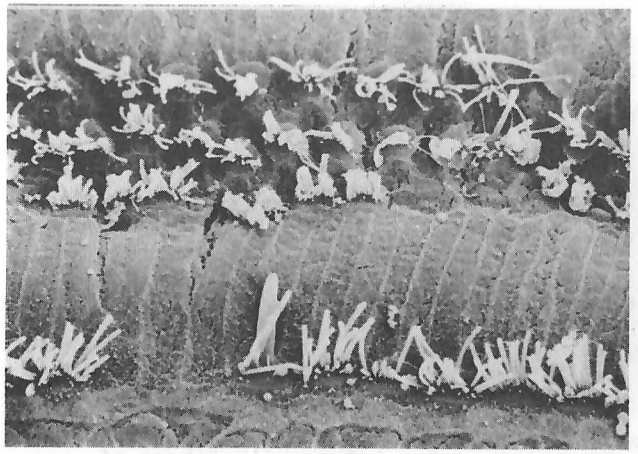

Figure 10. Scanning electronmicroscope picture from experimental deaf minipig showing missing inner (lower row) and outer hair cells (upper three rows), fused and giant stereocillia and disorganisation of normal stereocilia pattern. x 900.

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ANALYSIS OF DECOMPRESSION ACCIDENTS IN AMATEUR DIVERS

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ABSTRACT

The analysis is in two parts. The epidemiological analysis consisted of identification from British SubAqua Club incident statistics and surveys the trends in numbers and types of cases of decompression sickness. The data suggests that although there has been an increase in decompresson sickness (and gas embolism) in the last decade, the increase is largely related to increased numbers of divers and increased numbers of dives conducted. The ratio of type 1: type 2 decompression illness has altered little. Although deep diving, repeat diving and rapid ascents were implicated in causation, in many cases decompression procedures appear to have been correctly followed.

The clinical analysis involved investigation of individuals who suffered decompression sickness by means of contrast echocardiography and in some cases pulmonary investigations. The contrast echocardiography was performed in a blind, controlled manner. Of divers who had neurological symptoms within 30 minutes of surfacing, 65% had an intracardiac shunt on contrast echocardiography and some of the remainder had a probable pulmonary cause for their symptoms. In those with late neurological symptoms, those with joint decompression sickness or normal control divers the incidence of shunt was significantly lower. Those with early symptoms had usually followed recognised safe decompression procedures whilst those with late neurological symptoms had followed decompression profiles which could be considered provocative.

INTRODUCTION

In absolute terms, amateur scuba divers are more frequently affected by decompression sickness than any other group, but amateurs are the group about which least published epidemiological data is available. Recent developments in diving technology have made it possible for amateurs to dive deeper and longer. This theoretically increases the risk of decompression sickness. It is therefore important to determine whether these technological advances have increased

numbers or altered the types of cases of decompression sickness. Data from British SubAqua Club records were analysed to investigate this possibility.

In addition, we have previously reported our observation that suggests that paradoxical embolism of venous gas bubbles via intracardiac shunts can produce early neurological symptoms in divers (Wilmshurst *et al.*, 1986; 1989). Others have reported in a large uncontrolled trial, that an association between decompression sickness and intracardiac shunts exists (Moon *et al.*, 1989). In this study we investigated individuals who had suffered decompression sickness to determine possible aetiological factors. Investigations included contrast echocardiography, which was performed in a blind controlled manner to determine the incidence of intracardiac shunt.

METHODS

EPIDEMIOLOGICAL ANALYSIS

The data derived from:

1. Membership returns of the British SubAqua Club for the years 1981-1988.

2. Cases of decompression sickness and gas embolism reported to the British SubAqua Club Incident Panel for the years 1981-1988.

3. Anonymous questionnaires sent randomly to ten percent of the British SubAqua Club membership in 1976, 1980 and 1986.

The average number of dives necessary to give rise to each case of decompression sickness was derived for 1981 and 1986 from the formula:

No. of dives giving rise to each bend =

$$\frac{\text{Total no. of BSAC divers X average no. of dives conducted by each BSAC diver}}{\text{Total bends in BSAC divers}}$$

CLINICAL ANALYSIS

Four groups of divers were studied.

1. Those who had had early neurological decompression sickness, defined as onset of neurological symptoms within 30 minutes of surfacing from a dive (49 episodes in 43 divers).

2. Those who had had late neurological decompression sickness defined as onset of symptoms more than 30 minutes after surfacing from a dive (36 episodes in 34 divers).

3. Those who had joint or limb bends (21 episodes in 19 divers). Eleven divers who had limb bends also had neurological symptoms (3 early, 8 late onset).

4. Normal divers (n = 91) who had never had any form of decompression sickness, but had been at risk of developing it. This was judged from the fact that they had either been the asymptomatic diving buddy of one who developed decompression sickness or the fact that they had performed over 100 uneventful dives.

Each diver underwent contrast echocardiography to look for an atrial intracardiac shunt. Images were obtained with a Hewlett Packard HP 77020AC/AR. The contrast material was 5-6 ml 0.9% sterile saline containing microbubbles produced using the two syringe and three-way tap method (Lechat *et al.*, 1988). The contrast material was injected into a left antecubital vein via a 21 gauge butterfly needle. The passage of contrast was assessed visually by two observers without knowledge of the subjects' history. If contrast was not seen to pass spontaneously across an intracardiac shunt, a two second valsalva manoeuvre was performed at the peak of right heart opacification. (The short valsalva manoeuvre is similar to the ear clearing manoeuvre performed by some divers). The valsalva was released suddenly. This procedure was performed six times. In addition the effect of a cough performed at the time of maximal right heart opacification was studied twice. If no right to left shunt was seen after these manoeuvres the study was considered to be negative.

Each of the divers who had had early neurological symptoms also had a standard chest X-ray.

Divers gave informed consent to the tests which had the approval of the hospital ethical committee.

RESULTS

EPIDEMIOLOGICAL ANALYSIS

Table 1 shows the data on episodes of decompression sickness and cerebral gas embolism reported to the British SubAqua Club from 1981-1988. (Prior to 1980 the information available was not adequate for scientific analysis). The number of cases of decompression sickness has risen progressively during that period, although the exceptional number of cases in 1984 at least partly reflects the good diving weather experienced that year.

The percentage of the cases which had neurological involvement (cerebral gas embolism and type 2 decompression sickness, whether or not also associated with type 1 symptoms) has altered little. A large proportion of those developing decompression sickness had adhered to "safe" decompression profiles as laid down on their decompression tables (or computers) and should thus have had a low theoretical risk of decompression sickness.

Table 1. Annual reported incidence of decompression sickness and cerebral gas embolism

Year	1981	1982	1983	1984	1985	1986	1987	1988
Total number of cases of decompression sickness or cerebral gas embolism.	30	36	38	72	57	52	69	89
% of cases with neurological involvement.	70	69	71	74	79	75	73	82
% of cases without neurological involvement.	30	31	29	26	21	25	27	18
% of cases who adhered to safe decompression procedures.	-	-	-	-	30	50	42	47

The British SubAqua Club's survey of members showed that the average number of dives performed per year by each member has increased progressively. The number was 17.3 in 1976, 18.6 in 1980 and 21.8 in 1986. Thus in 1981 one case of decompression sickness occurred for every 20708 dives by the British SubAqua Club and in 1986 there was one bend for every 21308 dives. (Because of under reporting of episodes of decompression sickness this method of analysis under-estimates the risk of bends from diving, but it is hoped the error will be consistent from year to year, allowing analysis of trends).

CLINICAL ANALYSIS

Table 2 shows the prevalence of atrial intracardiac shunts in divers in each group. (This is the preliminary report of data from an uncompleted trial).

The prevalence of shunt was significantly higher in those who had had early neurological symptoms than in the other groups.

The chest X-rays of the divers with early neurological symptoms and shunts were normal. Four of the remaining divers had pulmonary lesions which could have resulted in barotrauma and hence cerebral air embolism.

Table 2. Prevalence of Atrial Intracardiac Shunts in Divers

	Normal divers	Limb bends	Late neurological symptoms	Early neurological symptoms	Total no. with decompression sickness
No. studied with contrast echocardiography	91	19	34	43	85
No. with shunt	22	3	8	28	38
% with shunt	24	16	23	65*	45

* Significantly different from other groups.

N.B. Some divers had limb bends and neurological bends - see text.

Early neurological bends followed single dives on 39% of occasions and repetitive dives on 61% of occasions. Late neurological bends and limb bends were less often associated with single dives (19% and 10% respectively).

Dive-related risk factors for decompression sickness (missed decompression stops, rapid ascents, post-dive ascent to altitude, dives deeper than 50 m, repetitive deep dives ($\geq$ 40 m) and frequent dives (> 3/day)) were implicated in the majority of late neurological bends (78%) and limb bends (86%). When early neurological bends occurred, there were usually dive-related risk factors if the diver had neither shunt nor lung disease (67%) but rarely were there risk factors if a shunt was present (27%). No risk factors were present in those divers with early neurological symptoms and lung disease.

It was particularly apparent that those with symptoms occurring soon after surfacing frequently had a greater neurological deficit than those with symptoms of late onset.

DISCUSSION

The epidemiological analysis showed that although the incidence of decompression sickness (and gas embolism) in amateur scuba divers is increasing in the United Kingdom, the increase appears to be a reflection of the increased number of divers and greater numbers of dives performed. There was no change in type of decompression sickness encountered. 70-80% of cases of decompression sickness in amateur scuba divers is neurological. This is a higher percentage than in individuals who get decompression sickness as a result of their occupation (Erde & Edmonds, 1975).

The observation that a high proportion of divers with bends had decompression sickness despite apparently safe decompression profiles is supported by the observation that most of those with early neurological symptoms had performed a "safe" decompression profile and appear to have developed symptoms as a result of paradoxical gas embolism across an intracardiac shunt. Those with late neurological symtoms and those with pain-only decompression sickness had a low incidence of shunt. They were usually affected because they had performed provocative repeat diving. Experienced divers who had never had decompression sickness also had a low incidence of shunt.

The prevalence of cardiac lesions likely to allow shunting is known, from necropsy studies, to be about 25% in normal individuals (Hagen *et al.*, 1984).

These observations have a number of implications. Since numbers of divers and amount of diving performed in the United Kingdom is likely to increase, the incidence of decompression sickness is also likely to increase. At the present time, the provision of facilities for treatment of cases of decompression sickness is saturated. It is planned that some of these facilities will close. This will lengthen the delay in treatment experienced by affected divers and will reduce the chance of complete recovery.

The observation that a common condition is implicated in causation of serious neurological decompression sickness despite adherence to safe decompression profiles has implications for diver selection and design of decompression algorithms.

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The second group of studies explored the possibility that similar mechanisms may be responsible for the damage seen in sickle cell disease and divers. Sickle cell disease is caused by a mutation in the haemoglobin gene which causes the protein to denature in certain conditions. This makes the red blood cells become 'sickle' shaped and more stiff than normal red blood cells. The infarction in sickle cell disease occurs because these sickle cells cannot deform enough to pass through the capillaries. In our laboratory we exposed preparations of red and white blood cells to pressure and have found that in certain conditions their stiffness can increase as much as three fold. In red cells the stiffness observed under pressure reverses on decompression or when the partial pressure of oxygen is increased. In the case of white blood cells, particularly granulocytes, the increased stiffness can last for more considerable periods which suggests some degree of chemical change or activation is involved in their reaction to pressure. The reaction to oxygen partial pressure perhaps explains some of the beneficial effect of oxygen to bends.

HYPERVENTILATION, FLUOROCARBONS AND DECOMPRESSION SICKNESS

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Hyperventilation is generally dangerous in diving because it prolongs breath-holding times, constricts the blood vessels of the brain and spinal cord, and allows dangerous degrees of hypoxia to develop, especially during subsequent ascents. However, in the brief profound compression and subsequent decompression of a submarine escape, it may offer an advantage by limiting the uptake of inert gas by nervous tissues. Once bubbles have formed in vessels or tissues, they take a long time to resorb because blood is so poor a solvent of inert gas. A case can be made for replacing blood by a good solvent after a dive in which Type II bends of cerebral gas embolism are suspected. The newer fluorocarbon emulsions look promising in this regard. They might also be worth giving before a prolonged (saturation) dive, to reduce the decompression time several-fold. Arguments and evidence for these points will be presented.

DESIGNING NEW DECOMPRESSION TABLES FOR RECREATIONAL DIVING: THE BS-AC'88 DECOMPRESSION TABLES

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The overall risk of decompression sickness (DCS) depends on a set of PROFILE PARAMETERS and on a set of PROCEDURAL PARAMETERS which determine how to find the required decompression stops

both for a given ideal single rectangular dive profile and also to allow for departures from it.

Long-established tables assume a remarkably wide variety of values for the PROFILE PARAMETERS. The PROCEDURAL PARAMETERS, therefore, must be the more important element in assessing the incidence of DCS in the recreational diving sector. The need to review procedures has increased significantly with the advent of decompression meters which have highlighted various practices that are not properly allowed for on old-style tabular decompression tables and what rules are given are too easily and too often ignored.

The BS-AC'88 Decompression Tables have been designed with the primary aim of introducing easy-to-apply yet comprehensive and safer procedures which can coexist with a decompression meter which, it is anticipated, with eventually replace tabular diving tables. Unambiguous advice is given on how to deal with those practices peculiar to recreational diving which are known to increase the DCS risk.

The biophysical basis of the decompression model is described and the risk of DCS on the BS-AC'88 Tables is assessed and compared with the risk on several other recreational diving tables. The Swiss decompression meter curves are also presented for a conventional rectangular and several non-rectangular dive profiles and the PROFILE PARAMETERS are compared with the USN and BS-AC'88 Tables for rectangular profiles at depths of 18 m, 30 m and 48 m. It is shown that despite the shorter TDT to arrival at 0 m on the deeper dives the new PROFILE PARAMETERS of the BS-AC'88 Tables actually reduce the DCS risk below that of the USN and similar tables.

Careful consideration was given to the special problems of mounting a suitable trial of a recreational decompression table using recreational divers. The incidence of DCS on recreational diving tables is already very low (about 0.1%) and the formal testing of too small a sample of both divers and divers would probably be dismissed as unrepresentative and the practical and financial constraints of mounting a more substantial trial is obviously prohibitive.

After a thorough assessment of all the factors it was decided that the actual incidence of DCS was likely to be LOWER on the Tables than on other tables and therefore a small formal trial (say 100 man-dives at 48 m for 20 minutes) would be statistically inconclusive. Instead a 4-month period of informal open-sea proving trials was undertaken by a group of BS-AC National Diving Committee members and National Instructors. It was completed successfully without any problems and the Tables were approved by the BS-AC for release to its members in August 1988. It is estimated that in the past six months, several thousand dives have been done using the Tables. The new decompression procedures have been shown to be practical and worthwhile and the overall simplicity of use has been acclaimed by divers and Diving Officers alike, and Instructors have found the principles of safe decompression very much easier to explain using the new Tables.