ORIGINAL ARTICLES

DYSBARIC PERIPHERAL NERVE INVOLVEMENT

Carl Edmonds

General

Peripheral nerve involvement from dysbaric accidents encompasses both barotrauma and decompression sickness (DCS).

The significance of peripheral neuropathy in diving lies not so much in its serious nature (it really is not that serious in most cases), but in its diagnostic and therapeutic implications. Another difficulty is the lack of sufficient factual data, from which to draw definite conclusions.[1]

Conventional attitudes to neurological DCS

In the Bennett and Elliott text of 1982, it was stated that "The most sinister development following a decompression is the insidious onset of peripheral sensory and motor symptoms. Paraesthesiae may be noticed in a limb or possibly there might be a marginal degree of weakness. The peripheral sensory and motor lesions are thought to be due to bubbles in the spinal cord, but they do not always follow a typical segmental distribution and an isolated patch may occur in any limb. These or other deficits may not be explained by a simple anatomical lesion. Cutaneous "pins and needles" must always be considered as spinal cord DCS".[2]

Elliott and Kindwall[2] also stated that Type 2 DCS cases include all those of a more serious nature, with central nervous system deficits, peripheral neuropathy or respiratory involvement with or without hypovolaemic shock. Cranial nerve DCS were reported, as were 7th nerve lesions.

Webb Haymaker[3] in his 1957 review, which is still a productive source of research material, stated that symptoms referable to the peripheral nervous system (PNS) were common. He referred to paraesthesias and radiating pains involving the limb plexuses, trigeminal, facial and occipital nerves. Such pains may last for several days and persist after all other symptoms have disappeared. Oculomotor nerve palsy was also described.

In 1984 Goad[4] stated that "DCS involving the peripheral nerve system is sometimes seen as a patchy kind of deficit, predominantly involving the lower extremities. It is attributed to bubble formation in the myelin of peripheral nerves. It can be difficult to differentiate from an incomplete spinal lesion, but the distinction is important, since the spinal cord injury is potentially much more disabling if not recognised and if not treated appropriately (if in doubt treat the more serious injury)".

Sykes[5] described the problems with neurological DCS, "It can affect every structure within the nervous system and, to frustrate the most ardent of academic neurologists, frequently affects several sites making interpretation of symptoms and signs difficult. The onset of symptoms is usually rapid. The majority of symptoms of neurological DCS will become evident within an hour of surfacing".

In 1967, Panov and Elinskii[6] stated that isolated DCS attacks on peripheral nerves were not often encountered. As a general rule pain, without any well defined disturbance of sensitivity, was detected. The sciatic nerve was affected in 2 of their cases, the radial in one and the facial in one. The symptoms were always of short duration and cleared without a trace. One patient produced neuralgic symptoms (plus arthralgia and myalgia) and this appeared to be the basis on which an hysterical paresis of the lower extremity developed.

Chronic and sometimes severe pains, referable to either nerves, nerve roots or plexuses, may follow neurological DCS, when myelin sheaths may have be damaged. Spinal cord lesions may also be responsible for some of these cases.

DCS cases with peripheral nervous system lesions

LITERATURE SEARCH

Cranial nerve lesions were first reported many years ago. Pain persisting for some time was reported by Bauer in 1870.[7] In 1890 Catsaras[8] from France, also reported cranial nerve lesions as did Siberstern in 1895.[9] In 1904, Lie[10] and Parkin[11] both published cases in the German literature. In 1907 Klienberger[12] published a general review, then in 1909 Keays[13] also published a general review of the subject. In 1939 Gerbis and Koenig[14] published a case of oculomotor palsy in the German literature and in 1944 Donald[15] also published a case in the U.K.

RESEARCH PAPERS

In 1944 Gersh and Hawkinson[16] demonstrated the presence of bubbles in the myelin sheaths of peripheral nerves.

Jackson and Vanderwalt[17] assessed the possibility of Type 1 DCS being due to a bubble in the nerve lipid tissue, to explain the poor correlation between bubbles in the blood and Type 1 bends, and the propensity to DCS following nerve injury.

CLINICAL PAPERS

To select these I have used as evidence of peripheral nerve system (PNS) DCS, the reporting of patchy disturbances of sensation, paraesthesia, and neuralgias in DCS cases. The figures in parentheses are the numbers in each series.

Rivera[18] (935) described numbness or paraesthesia in 21.2%, although there was paralysis in only 6.1%. He described cranial nerve involvement in 0.2%.

Slark[19] (137) described disorders of sensation in 8%.

Kidd and Elliott[20] (250+) described sensory impairment in 25%, with motor impairment in only 13%.

Erde and Edmonds[21] (100) found that the incidence of clinical cerebral, spinal and PNS involvement in recreational divers was 24%, 22% and 11% respectively.

How, West and Edmonds[22] (115) described numbness, either alone or as part of an overall neurological complex, in over half of their cases.

Dick and Massey[23] (70) described limb paraesthesias in 34%, neurogenic limb pain in 9%, limb numbness in 56% and weakness in 23%. In only in 11 % was there an actual paralysis. The diagnoses were often delayed because symptoms did not fit the classic picture of major neurological dysfunction.

Todnem and colleagues[24] (156), found that divers had more central, peripheral and autonomic nervous system symptoms than controls. This correlated with DCS incidence.

In 78% of the 88 divers with DCS in the series reported to Gorman and his colleagues[25] there were overt neurological symptoms and signs, attributable to DCS, before treatment. Over half of the 19 divers initially considered to have had only Type 1 DCS demonstrated a neurological deficit after discharge. Seven had abnormal psychological tests and 5 had abnormal EEG records. This data suggests that most DCS incidents involve the nervous system.

The reason DCS cases do not conform to the conventional neurological disorders is probably because lesions occur in many different parts of the nervous system; cerebral, spinal and peripheral.

CASE HISTORIES

Case 1

Elliott and Kindwall[2] described how damaged tissues increased susceptibility to DCS affecting a peripheral nerve. A 25-year-old male diver was being investigated for epilepsy which developed while diving. A lumbar puncture needle caused an injury with muscular twitching of the right leg. Six days later he went to 15 m for 25 minutes and 2 hours afterwards he developed paraesthesia in the back of his right leg.

Two weeks later he dived to 54 m for 15 minutes, with appropriate stops, and 45 minutes after surfacing he developed anaesthetic patches down the back of his right thigh and on the knee. There was decreased sensation at the S1-S2 level. Recompression chamber treatment produced complete relief.

Case 2

A retired sheep shearer, aged 40, took up professional abalone diving. Following a spinal decompression accident he developed severe and protracted neuralgic pain in both legs, resistant to therapy.

Twenty minutes after surfacing he developed pain in the chest and paraplegia. He was then flown to Hobart, Tasmania, and treated in the recompression chamber and left with a fairly typical, almost total paraplegia (slight movement in one foot, urinary retention requiring catheterisation, poor bowel control and occasional erections). Afterwards he developed severe and progressive pains, especially in the left thigh and leg and then subsequently in the right thigh and leg. These have not improved over 5 years.

Case 3

In 1988, after repetitive daily diving in the Philippines, a 25 year old male dive master completed an uneventful dive for 30 minutes to 24 m and then decompressing for 10 minutes at 15 m. The next morning he noticed a slight twinge in the left elbow (with weakness), also numbness and tingling in the left arm and forearm. Subsequently pain developed in the left elbow and then progressed to severe pain throughout the whole of the left upper limb. Flexion gave some relief. He arrived at the USN recompression chamber at Subic Bay four days after the initial dive. A full neurological examination showed no significant abnormality, but a possible slight strength reduction was described.

The diver was taken to 18 m on 100% oxygen with almost complete relief of symptoms. He was observed for 12 hours after this. After a few weeks, severe “electric shock sensations” developed in his left arm and continued intermittently until he was seen in February 1989. Assessment by the Clinical Neurophysiology Department at Royal Prince Alfred Hospital, Sydney, indicated that the lesion was most likely in the brachial plexus on the left side, rather than an intrinsic cord lesion. By April, 1989, the symptoms had almost gone with only an occasional slight twinge or wave of sensation.

Case 4

A nursing sister, aged 28 years, had done 12 scuba dives. She dived for 10 minutes to 36 m on the Presid*ent*

Coollidge, at Santo Island. The next day she did a 46 m dive for 25 minutes, decompressing for 5 minutes at 6 m and 15 minutes at 3 m. Four hours after the dive an ache developed in her left shoulder. Nine hours later it changed to "shooting pains" and weakness from the left side of the head, neck and back to the left elbow and hand. These pains lasted a few seconds to a minute, and were associated with a constant ache. A very mild skin rash was noted along the arm and forearm for 24 hours

She noted inability to concentrate and difficulty in finding the correct words. "I felt spaced out." She was unable to read even short articles in newspapers. When she was flown to Australia two weeks after the incident, she had shooting pains in her left shoulder and upper arm, severe unrelenting headache, paraesthesia and alteration of sensation of the left arm and loss of power in the left arm.

An extended USN table 6 produced improvement in strength and complete removal of the headache. There was a slight alteration in sensation over the lateral aspect of the left upper arm, shoulder and left side of the neck. This was unchanged during the last two of three 2-hour hyperbaric oxygen (HBO) treatments at 9 m. There was some deterioration a few days after this and she was given a further 7 HBO treatments. During this time there was a considerable improvement in her ability to concentrate, but she still felt "snakey and irritable" and had late insomnia, anorexia, general tiredness and some degree of depression.

Examination showed her to be mentally alert and normal. Her left upper limb had reduced appreciation of light touch and pin prick over the whole of the arm and onto the trunk from C3 level to T4 level. Motor power was normal in the hand but there was winging of the scapula and slight weakness of other shoulder girdle muscles, the most seriously affected being the infraspinatus. Vibration, position sense and two point discrimination were normal. Her reflexes were symmetrical. Her neurologist reported "I have no doubt that she has neurological lesions resulting from decompression, with a spinal cord sensory abnormality, and evidence of a lesion of some nerves proximally in the brachial plexus."

The CT brain scan and EEG were normal. All the neurophysiological abnormalities were on the left side. Somatosensory evoked cortical responses revealed absent ulnar potentials from clavicle and cervical spine in the presence of normal median nerve potentials. There was evidence of a lesion of predominantly C8 input in the brachial plexus. Other electrophysiological studies revealed abnormalities in her arm and specifically an extensive denervation of the infraspinatus muscle. This must be the result of a lesion in the branch of the suprascapular nerve. Changes in other muscles tested, were minimal.

Initial psychometric studies revealed a high intelligence, but she had a problem in the initiation of tasks and suffered a visual memory deficit. Later testing was normal.

Four months later, the ache had progressed to a more intense pain down the lateral aspect of the arm and into the webbed space between the thumb and the first finger, the area supplied by the radial nerve, probable C6 root distribution. There was no change in the objective symptoms, with still a marked isolated weakness of the infraspinatus muscle and a sensory impairment in the same distribution as previously. The neurologist's assessment was: "She has pain as a residual of the injury which occurred to the brachial plexus and in the spinal cord from decompression sickness."

Many treatments were given for the pain over a six month period. These included Tegretol, Brufen, Voltaren, transcutaneous stimulation, stellate ganglion block and TENS. The last was of value.

Case 5

A male fish-farming diver, aged 17, performed hard work, with many ascents and descents, for nine hours each on two consecutive days to a maximum of 9-12 m (tidal), with only a 15 minute surface interval for lunch. After this he felt a little unwell, weak, tired, developed a slight headache, weakness in the legs and multiple joint pains which increased appreciably as he drove over the mountain. There were patchy areas of hypoaesthesia and a reduction of sensitivity to light touch and pin prick, over a large area on the left lower abdomen, anteriorly.

Treatment with USN table 6 gave considerable improvement during the second oxygen period. He was greatly improved by the end of the extended table. Power had returned and the hypoaesthesia had disappeared. After treatment he was given intermittent oxygen. The pains recurred on the following day. He developed a retrosternal and pleuritic chest pain with a reduction in the peak expiratory flow rate. He was not treated on the second day, however on the third day he was given a table 5 which relieved the joint pains. He was then free of these for another 8 to 10 hours, before they recurred. On the 4th day during the 3rd treatment, after two hours at 60 feet, the pains cleared. They recurred 24 hours later. On review one week later, there was still discomfort in one knee.

Barotrauma related PNS (Cranial) lesions

Cases occasionally present with cranial nerve lesions attributed to neuropraxis. This can be due to either implosive tissue-damaging effects during descent, or rising pressure in enclosed gas spaces during ascent, or both. The nerve damage varies greatly, often being transitory but occasionally long-lasting. These presentations are usually associated with the symptoms and signs of barotrauma

In the cases produced by ascent, there may be a delay of many minutes after the dive before symptoms and signs develop. The diver may be aware of a feeling of distension

of the gas space. The relief of this as gas escapes, may coincide with improvement in the neuropraxis. This suggests that the cause may be ischaemic, with a middle ear or sinus pressure in excess of the mean capillary perfusion pressure.

The fifth, or trigeminal, nerve may be influenced by gas pressure changes in the maxillary antrum with sinus barotrauma. The most common presentation is involvement of the maxillary division, especially the infraorbital nerve, which traverses the sinus.[26] Hypoaesthesia can be demonstrated for a variable time after the sinus barotrauma incident. It may involve the cheek, side of nose, lower eyelid, upper lip, maxillary teeth and gums. It may also be a cause of the pain from sinus barotrauma, referred to the upper teeth on the same side.

I have found only 4 cases described in the literature[1,15,26,27] and 2 cases of my own. Both of these did shallow dives, and the diagnosis was verified radiologically. CT sinus scan is superior to plain X-rays for this.

The seventh, or facial, nerve may be affected, causing "facial baroparesis", as it passes through the middle ear space. Recorded in both aviators and divers, it is more frequent following ascent, presents as a unilateral facial weakness similar to Bell's palsy, and tends to recur if diving continues. Paralysis of the facial nerve makes frowning impossible, prevents the eye from closing on that side and causes drooping of the lower lid (which may result in tears running down the face, because they do not drain into the nasolacrimal duct). The cheek is smooth and the mouth pulled to the opposite side. Whistling becomes impossible and food collects between the cheek and gum.

A metallic taste may be noticed at the start of the illness, as may impaired taste in the anterior part of the tongue on the same side, from chorda tympani involvement. Hyperacusis may be due to paralysis of the stapedius muscle.

A possible reason for an individual's susceptibility to this disorder can be found in the anatomy of the facial canal. This opens into the middle ear in some people and so shares its pathology.

A typical history of middle ear barotrauma is usually present, with all the conventional provoking factors . If there is an ascent barotrauma with a distension of the middle ear, this can sometimes be relieved by performing a Toynbee manoeuvre, pressure via the external ear, oxygen inhalation, decongestants or, if needed, recompression.

Before 1985, 16 cases of 7th nerve paresis from barotrauma had been reported. Eidsvik and Molvaer2[8] described another 5 cases, all developing within 10 minutes of surfacing. 3 episodes cleared when the middle ear overpressure was relieved by recompression. I have seen 4 cases[1], 2 of whom suffered a recurrence, and all were associated with middle ear barotrauma.

Eighth nerve lesions are less frequently attributed to DCS nowadays, except for the deep helium and saturation diving exposures. Certainly, of the many hundreds of cases of demonstrated inner ear damage seen by us, with high frequency or total sensory-neural hearing loss and/or vestibular abnormalities verified by the electronystagmograms (ENG), most are peripheral lesions associated with barotrauma. Inner ear DCS cases are well reviewed by Farmer[29], but central vestibular lesions demonstrated by the ENG and/or abnormal brain stem evoked auditory responses, are rare enough to be worthy of reporting.

CASE HISTORIES

Case 6

Donald[15] described the oft quoted case of a very experienced 38 year old Royal Navy Petty Officer who was said to have been "very resistant to bends".

In this instance he was exposed to a pressure of 40 feet (12 m) in the chamber, for up to 2 hours. Five minutes later he developed severe pain in the upper left premolar tooth. Donald considered the possibility of a concealed tooth cavity or a tooth abscess. Recompression to 18 feet (5.5 m) produced instant relief and he was surfaced over 30 minutes. The pain was less severe, but later increased and an area of anaesthesia developed over the left upper gum and teeth, reaching the midline.

The second recompression to 12 pounds per square inch (psi) produced relief of pain and he was decompressed at 8 minutes per pound. At 5 pounds the pain returned. At 0.75 pounds the anaesthesia returned. This was followed by a neuralgia, over the whole distribution of the second branch of the 5th nerve. Tender points were noted over the temporomolar and infra-orbital regions.

The 3rd recompression was to 13 psi with a decompression at 12 minutes per pound. The patient was then symptomless and the tenderness had disappeared.

There was no past history of ENT infections, trigeminal neuralgia or dental caries. He had previously suffered sharp dental pain in this premolar when exposed to temperature extremes, and there was a slight enamel erosion at the base near the gum.

The diagnosis made was DCS due to a bubble lodged in the second branch of the trigeminal nerve, without related subcutaneous anaesthesia present.

In the discussion Donald pointed out the unexpectedness of: bubble formation at such a shallow depth; bubble formation in a diver who was particularly resistant to bends;

bubble formation in a sensory nerve and bubble formation in a sensory ganglion. He suggested that if bubbles were in the sensory ganglia this might explain the more typical pain of other bends symptoms. He commented that the diver did sing during the original exposure, and as such this might be classified as the first reported case of operatic bends. The position of the bubble was considered an interesting problem for neurologists to solve.

It seems that the more likely possibility of sinus barotrauma, involving the maxillary division of the 5th cranial nerve, was not considered but dental barotrauma was.

Case 7

A 20 year old male carried out 3 short dives to 6-9 metres and had difficulty in equalising his left ear. He finally succeeded, with considerable effort. On ascent he developed pain in the left ear. He was aware of a full sensation in the left ear, together with loss of hearing. Pure tone audiometry revealed a left hearing loss of 60 db at 6000 Hz and 45 db at 8000 Hz. There was no problem with disorientation and an ENG was not performed at that time (November 1978).

He resumed diving and had another almost identical episode of left middle ear barotrauma of ascent, this time associated with vertigo and tinnitus, with a flat hearing loss of 60-90 db between 2000 and 8000 Hz An ENG showed a right beating nystagmus with eyes closed and increased when the head was positioned to the left. There was an absence of the caloric response in the left ear.

The provisional diagnosis was left middle and inner ear barotrauma, involving both cochlear and vestibular systems.

He still continued to dive! On 26.12.79 after a shallow dive he noted a severe pain in the left ear. He lost the movement of the muscles on the left side of the face, his eye remained open and his mouth was drooping.

The provisional diagnoses were 7th nerve palsy and inner ear barotrauma, both due to middle ear barotrauma of ascent.

Non-dsybaric PNS (Spinal) lesions

Divers may have hypoaesthetic or painful areas in the occipital region involving the posterior rami of the C2 and C3 , however in the cases that we have seen it has been related to a high cervical orthopaedic problem and not to DCS.

Headache, which can also with with upper cervical nerve lesions, is due to cervical spondylosis aggravated by excessive provocation, and may be confirmed by cervical spine X-rays. In these there may be loss of lordotic curvature, narrowing of inter-vertebral spaces, and osteophytosis in the lateral views. Many divers who develop this disorder are in the older age group or have a history of head and neck trauma. They often swim underwater with flexion of the lower cervical spine (to avoid the tank regulator) and their upper cervical spine hyper-extended (to view where they are going). This produces C2 and C3 compression and distortion of the cervico-cranial relationships; an unnatural posture aggravating underlying disease, which may be otherwise asymptomatic.

Local patches of hypoaesthesia have been frequently reported, throughout the diving literature. Nevertheless, there are occasional cases of misdiagnosis.

Brachial plexus lesions may be related to the use of standard dress diving equipment, when the weight of the helmet is taken directly on the supraclavicular region. This may be from mishandling the helmet or by having inadequate or incorrectly placed padding over the area between the neck and shoulder. It is more likely to be caused out of the water, when the weight is greatest. The standby diver was thus more prone to this disorder. The middle and lower cervical nerves are more likely to be involved i.e. the C 5-7, and this may be either temporary or permanent. Minor cases present with paraesthesia and numbness of the lateral aspect of the arm, forearm, thumb and adjacent fingers. Severe cases result in both motor and sensory paralysis over the affected nerve distribution. Rigid shoulder harnesses for scuba gear can also produce this.

More recently we have seen weight belt or crutch harness pressure produce meralgia paraesthetica, described in the diving medical literature some years ago. Scuba diver's thigh (meralgia paraesthetica) is neuropraxia of the lateral femoral cutaneous nerve of the thigh. The nerve is vulnerable to compression neuropathy by pregnancy, tight trousers, pelvic tilt, harnesses, low positioned weight belts and increased physical activity involving the thigh muscles (e.g. with fin use during diving) . It results in a numbness over the upper thigh, anteriorly and laterally. It usually clears up in a few months.

CASE HISTORIES

Case 8

An abalone diver, whose diving was between 4.5 - 6 m, had repeated bouts of severe pain, very localised, over the right occiput, later with hypoaesthesia. The area felt tender and hot and the pain was relieved by an ice pack. He observed that it developed when he moved his head in a certain position, while diving. This was when he twisted down and to the right to collect abalone. A photograph (Figure 1) demonstrates this provocative position. He was examined and investigated by a neurologist, who made the diagnosis of right occipital neuralgia. A local anaesthetic

injection around the right occipital nerve produced a numbness over the same area, with a diminution of his pain.

He continues abalone diving to shallow depths, with advice not to hyperextend his cervical spine.

FIGURE 1

PNS lesions due to oxygen toxicity

Oxygen toxicity may produce a variably persistent bilateral peripheral neuropathy, as may many of the fish poisons and marine toxins.

Donald[30] when investigating oxygen toxicity, showed that bursts of potentials in the EMG were related to facial twitching and were unrelated to EEG abnormalities. Chvostek's sign inferred hyperexcitability of the facial nerve. At these levels of oxygen (over 3 ATA), however, paraesthesia was a rarity, conflicting with the earlier observations of both Bornstein and the British Navy.

Becker-Freyseng[31], described neurological symptoms of oxygen toxicity in man. With Clamann in 1938-1940 in Berlin, he breathed 100% oxygen at 1 ATA. During the second day, prickling and numb sensations in the fingertips developed. By the third day this had spread to the feet and also produced transient pains in the knees. The paraesthesia lasted two weeks.

Brue[32] and Balentine[33] independently demonstrated that animals could avoid death from respiratory oxygen toxicity by intermittent exposure to 100% oxygen at 1 ATA, but they then died of a neuromuscular paralysis. The first histological changes were in the dendritic mitochondria in the spinal cord, 24-72 hours after exposure.

Recurrent or delayed symptoms from DCS

Gorman et al[25] showed that the apparent clinical morbidity amongst a group of DCS divers when they were discharged (3.4%) was considerably lower than the frequency of abnormalities demonstrated one week later. The natural history was for complete remission, eventually. There was a highly significant improvement in clinical neurological examinations and EEG performed one month after the treatment compared to those at one week.

Symptomatology, especially neurological and neuropsychological, often continues, develops or extends after the successful treatment of divers with DCS, so that it can only be picked up a week or more later. Such symptoms include

1 recurrence of the original symptoms, but less severe.
2 minor aches and pains, paraesthesias.
3 symptomatic (endogenous) depression
4 neuralgic pains or, uncommonly, "hysterical" symptoms
5 dysbaric osteonecrosis

Apart from the first, these symptoms are not improved with recompression or HBO. The reason for subjective neurological sensations in the weeks following therapy, is conjectural. Peripheral or central neurological damage from DCS or even from oxygen, may take time to mend.

Unless it is appreciated that these symptoms do persist, even after adequate treatment, there is the danger of excessive repeated HBO exposures, exhausting to the patient and demoralising to all. Some patients currently receive 8 to 10 treatments for purely subjective symptoms!

For this reason we introduced a criterion of "an acceptable clinical result" as the reasonable aim of recompression therapy, instead of a true "cure" which is not all that frequent in the delayed treatment of recreational divers. A "cure" is most readily achieved as the diver emerges from the chamber, and is commonly maintained only by avoiding neurological investigations and any follow up!

Symptoms recurring after flying

Another problem that we have encountered on many occasions is with exposure to altitude following recompression therapy for DCS. Initially, when people flew back to the mainland or to their own country, one or two weeks after

treatment, it was common for them to notice general, subjective neurological symptoms such as numbness and paraesthesia, during the flight. This would then result in multiple recompressions or even saturation treatments.

I have not been impressed with the value of recompression therapy for such delayed and minor aviation induced symptoms. The following causes are as plausible as the assumed bubble pathology:

1. Mild hypoxia, hypocapnia and respiratory acidosis (aviation effect) on an irritable or partially demyelinated or damaged nerve.

2. Anxiety and hyperventilation are common in mildly agoraphobic personalities during plane travel and are also caused by an awareness that aviation exposure can produce a recurrence of DCS.

It is our policy to give aggressive oxygen recompression therapy.[34] To ensure that there are no bubbles left, we follow this with daily oxygen sessions for at least a week before flying. I advise patients not to be too concerned with minor sensations, such as paraesthesia, noticed during aviation exposure but to phone me before consulting with their local hyperbaric centre, if they wish to avoid unnecessary recompression therapy. This advice may conflict with "established wisdom".

For a recurrence of symptoms during aviation, similar to those previously experienced and treated, or with organic signs of DCS, I endorse active and enthusiastic recompression.

References

1 Edmonds C. Peripheral neurological abnormalities in dysbaric disease. In *Describing Decompression Illness 42nd Undersea & Hyperbaric Medical Society Workshop.* Francis and Smith Eds. Bethesda, Maryland: UHMS, 1991.

2 Elliott DH and Kindwall EP. Manifestations of decompression disorders. Chapter 18 in *The Physiology and Medicine of Diving*. Bennett PB and Elliott DH (Eds), 3rd Edition. London: Bailliere Tindall, 1982.

3 Haymaker W (1957). Decompression sickness. In *Key Documents of the Biomedical Aspects of Deep-Sea Diving.* Bethesda, Maryland: UMS, 1983; IV-15-(107-179).

4 Goad RF. Diagnosis and treatment of decompression sickness. Section 6 in T*he Physicians Guide to Diving Medicine*. New York: Plenum Press, 1984.

5 Sykes JJW. Neurological decompression sickness. In *Case Histories of Diving and Hyperbaric Accident*. Charles L Waite (Ed). Bethesda, Maryland: UHMS, 1988.

6 Panov AG and Elinskii MP. In *Zh Neuropatol i Psikhiatr* 1967; 12: 1791.

7 Bauer L. Pathological effects upon the brain and spinal cord of men exposed to the action of a largely increased atmospheric pressure. *St. Louis Med & Surg* 1870; 7: 234-245.

8 Catsaras M. *Recherches cliniques et experimentales sur les accidentes survenant par cemploi des scaphandres*. Paris. Lecrosnier et Babe, 1890.

9 Silberstern P. Aur Kasuistik der Caissonbrankheit. *Wein Klin Wschr* 1895; 1306-1307.

10 Lie HP. Veranderungen in dem Nervensystem beim plotzlichen Ubergang vom hohen zum normalen Barometerdruk. *Virchows Arch* 1904; 178: 142-156.

11 Parkin A. Caisson disease, or compressed air illness. *Univ Durham Coll Med Gaz* 1904; 4: 81-88.

12 Kienberger. Uber Luftdruckerkrankungen beim Bau der Grunen Bruke. *Konigsberg i Pr Hyg Rdsch* 1907; 17: 447-451.

13 Keays FL. Compressed air illness, with a report of 3,692 cases. *Cornell Univ Med Coll, Dept Med* 1909; 2: 1-55.

14 Gerbis H and Koenig R. *Drucklufterkrankungen (Caissonkrankheiten)*. Leipzig: Georg Thieme, 1939.

15 Donald KW. A case of compressed air illness. *Brit Med J* 1944; 1: 590-591.

16 Gersh I and Hawkinson GE. *Research project X284. US Navy Medical Research Institute Report No 1.* 1944.

17 Jackson JH and Vanderwalt JG. *S African Med J* 1981; 59(26): 928.

18 Rivera JC. *Decompression sickness among divers: An analysis of 935 cases. USN EDU Research Report 1-63.* 1963.

19 Slark AG. *Treatment of 137 cases of decompression sickness. MRC Royal Navy Personnel Report 63/1030*. 1962.

20 Kidd DJ and Elliott DH. Decompression disorders in diving. Chapter 25 in *The Physiology and Medicine of Diving;* Bennett PB and Elliott DH Eds., 2nd Edition. London: Bailliere Tindall. 1975.

21 Erde A and Edmonds C. Decompression sickness; a clinical series. *J. Occup Med* 1975; 17: 324-328.

22 How J, West D and Edmonds C. Decompression sickness in diving. *Singapore Med J* 1984; 17(2):92-97.

23 Dick APK and Massey EW. Neurological injuries in sport divers. *Undersea Biomed Res* 1984; 11 (1 Supp): 19

24 Todnem K, Nyland H and Skeidsvoll R et al. Nervous system involvement from occupation diving. *Undersea Biomed Res* 1990; 17(Supp); 126.

25 Gorman DF, Edmonds CW, Parsons DW et al. Neurological sequelae of DCS: a clinical report. 9t*h International Symposium of Underwater and Hyperbaric Medicine.* Bethesda, Maryland: UMHS , 1987.

26 Newman T, Settle H, Beaver G and Linaweaver PG. Maxillary sinus barotrauma with cranial nerve in-

volvement. *Aviat Space Environ Med* 1974; 46: 314-315.
27 Garges LM. Maxillary sinus barotrauma. *Aviat Space Environ Med* 1985; 56: 796-802.
28 Eidsvik S and Molvaer OI. Facial baroparesis. *Undersea Biomed Res* 1985; 12(4): 495-463.
29 Farmer JC. Ear and sinus problems in diving. In *Diving Medicine.* Bove AA and Davis JC (Eds). Grune and Stretton 1989.
30 Donald KW. Oxygen poisoning in man. *Brit Med J* 1947: May 24: 712-717.
31 Becker-Freyseng H. *German aviation medicine World War II, Vol 1.* Reprinted by the Surgeon General of the US Air Force 1971.
32 Brue F, Guillemonot R, Brerl D and Broussolle B. *Bull Medsuphyp* 1972; 8 Nov: 709.
33 Balentine JD. Pathological observations on rats fatally exposed to oxygen. In *5th International Conference on Hyperbaric Medicine* 1973; 102-107.
34 Edmonds C, Lowry C and Pennefather J. *Diving and Subaquatic Medicine*. 3rd Ed. London: Butterworths, 1992. In Press.

Dr Carl Edmonds FRACP, Dip DHM, is a Director of the Diving Medical Centre, North Shore Medical Centre, 66 Pacific Highway, St Leonards, New South Wales 2065, Australia.

A BUBBLE MODEL FOR REPETITIVE DIVING

Bruce Wienke

Abstract

Within the critical phase hypothesis in a bubble model, we have shown that reduced tissue tensions are necessary for multi-level and multi-day diving (multi-diving). Deep repetitive and shallower multi-day exposures are affected directly by the model. Within nucleation theory deeper-than-first dives are also affected. Sets of multi-diving fractions, accounting for micronuclei excitation and regeneration, reduced bubble elimination in repetitive diving and coupled effects on tissue tension, are discussed. Multi-diving fractions are simple multiplicative factors reducing permissible tissue tensions used in tables and decompression meters. These factors restrict repetitive diving over short time spans, deeper-than-previous and continuous multi-day diving, compared to standard algorithms using a fixed slow compartment. Within the model, fast compartments, controlling deeper exposures, are affected the most, and slower compartments, controlling shallower exposures, are least affected.

Introduction

Validation of decompression schedules is central to diving and testing of no-stop and saturation schedules, with requisite analysis,[1-10] has progressed. Repetitive, multi-level, deeper-spike, and multi-day diving cannot claim the same validation, though some programs are breaking new ground. Application of present models in these latter cases has apparently produced slightly higher bends statistics that in the former ones, as reported in DAN newsletters,[7] and discussed at workshops,[5,6] and technical forums. Perceived problems associated with multi-diving might be addressed by reducing critical tissue tensions, particularly as they drive bubble excitation and growth beyond permissible levels in bubble models.[11-18]

Accordingly a model, called the reduced gradient bubble model[18] (RGBM), has been developed which reduces permissible tissue tensions in repetitive diving. The need for this reduction arises from the lessened degree of bubble elimination over short repetitive intervals, compared to long surface intervals, and the need to reduce bubble inflation rate through smaller driving gradients. Deep repetitive and spike exposures feel the greatest effects of gradient reduction, but shallower multi-day activities are also affected. Single daily (bounce) dives have long surface intervals to eliminate bubbles within the critical phase hypothesis, while repetitive diving must contend with shorter intervals, and thus reduced time for bubble elimination. Theoretically, a reduction in the bubble inflation driving force, namely, the tissue tension, holds the inflation rate down. The concern is bubble growth driven by dissolved gas, and a certain limiting volume for all bubbles, called the critical volume, before symptoms develop.

Within the RGBM three reduction factors, addressing bubble regeneration, deeper-than-previous excitation of nuclei, and shorter repetitive time spans for bubble elimination, are discussed and applied to some marginal exposures. First, we consider two repetitive dives to 36 m (120 fsw) for 10 minutes with a 2 hour surface interval, repeated for three days. Three repetitive dives to 36 m (120 fsw) for 10 minutes with 2 hour surface intervals, on one day, is known to cause bends in roughly three out of four cases, according to Leitch and Barnard,[19] so the exercise is not academic. As a second application, the hazardous repetitive profile reported by Edmonds,[5] three repetitive dives to 44.5 m (147 fsw) for 5 minutes, with 1 hour surface intervals, is also treated. Meter predictions of no-stop limits are contrasted with predictions of the RGBM, and the reductions in time limits are quantified. A brief description of some theory, then the applica-