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(Accepted 7 June 1988)

Treatment of compressed-air decompression accidents

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Keywords: decompression sickness; barotrauma; divers; tunnellers

History

Following Sir Robert Boyle's observation of bubbles in live tissues in a famous exposure of a viper to a vacuum decompression in 1670, decompression illnesses in man were first recognized in the 19th century in tunnellers and miners.

In 1841, Triger[1] first described decompression illness in coal miners in pressurized shafts as caisson disease when large capacity compressors were developed to pressurize bridge building foundations to prevent flooding and numbers of men were exposed to raised pressures for prolonged periods.

In 1854, Pol and Watelle described the relation between symptoms and depth and duration of exposure, and speed of decompression[2]. In 1847, they also observed relief of symptoms by increasing ambient pressure and suggested gas bubbles might be the cause of the problem.

In 1872, Friedburg noted the similarity between severe decompression sickness and surgically induced arterial gas embolism, and Gal described the first case of neurological decompression sickness in a commercial diver (Elliott 1985, personal communication). Practical deep sea diving commenced in 1819 when Augustus Siebe designed the first diving dress. In 1878, Bert[3] analysed the blood gases and bubbles which formed in experimental animals with decompression sickness and identified nitrogen as the principal cause. He recommended recompression and elevated partial pressures of oxygen as therapy for decompression sickness. No procedure for standard decompression was developed until Heller, Mager and von Schrötter proposed, in 1900, a linear decompression rate of 20 min for each 33 ft (1 atmosphere absolute) which was successful for dives down to 160 feet of seawater.

Haldane was appointed by the Admiralty in 1905 to develop safe decompression procedures. They were published in 1908[4]. Subsequent air decompression tables for the next 50 years were based on these decompression procedures and later modified for divers[5]. The hazards of air diving without safe decompression were shown by Blick[6] who described 200 cases of decompression sickness, of whom 60 died in 1909.

Tunnellers had their own decompression tables and therapeutic procedures[7]. They now follow therapeutic procedures according to the Construction Industry's CIRIA code of practice (1982).

Decompression sickness

Following the earliest descriptions of decompression sickness (DCS) in 1872, there was a report of cases of 'divers' paralysis' in pearl fishers. Cases of slight paralysis were common after removal of diving dress but 'generally recovering completely'[8].

In tunnelling, the experience of the first Dartford Tunnel was recorded[9] which describes 685 cases of decompression sickness, 650 (94.9%) of which were 'simple Type 1 bends' and 35 (5.1%) having symptoms other than pain described as 'serious Type 2 symptoms'. All cases were successfully treated without permanent disability using minimum pressure required for relief of symptoms followed by slow decompression with occasional 'soaks' by holding at various depths during ascent. This method was considered more satisfactory than trying to force the bubbles wholly back into solution by use of high pressures.

The US Navy reviewed 20 years of experience of treating cases of DCS in 1964[10] and found an overwhelming preponderance of Type I as opposed to Type II DCS. This has now been entirely reversed with more serious symptoms being more common[11,12]. The apparently mild sensory symptoms may herald onset of spinal or cerebral DCS in sports divers[13]. This has been borne out over the last 23 years at this Centre where of a total of 406 cases recorded there were 80 Type 1 cases as opposed to 178 Type 2 cases. (The remainder were 54 cases of barotrauma from all causes and 94 cases subsequently proved to have 'non-dysbaric' illness).

Approach to therapy

The basis of therapy continues to be recompression, an elevated partial pressure of oxygen and use of certain drugs. Recompression reduces the gas volume (Boyle's Law) and tends to drive the gas back into solution (Henry's Law); if applied soon enough it should restore circulation. Addition of elevated oxygen partial pressure will increase inert gas washout, reduce tissue hypoxia and possibly oedema, and improve oxygenation.

0141-0768/89/020074-06/$02.00/0

Recompression may be to the depth of relief of symptoms, to the depth of the original dive or to some arbitrary depth[14]. Most cases of decompression sickness occur following return to atmospheric pressure although occasionally they may occur under pressure.

Duration of treatment

In the early '70s in commercial diving, the maximum air treatment depth was 165 ft (50 m) and a maximum duration at that pressure was 2 h. In the Royal Navy and United States Navy, the tendency was to go to this depth for cerebral arterial gas embolism or for life-threatening serious symptoms. However, there was a feeling that if major resolution had not occurred at the end of standard therapeutic recompression, then there was nothing therapeutic about decompression, particularly if symptoms were worsening and alternatives to decompression were attempted.

Choice of table

In 1978, Berghage[15] prepared a report listing 67 different therapeutic tables used around the world. Some of these were similar if not identical. The doctor must familiarize himself with the therapeutic tables used by the client company or have detailed knowledge of an approved set of tables.

The European Undersea Biomedical Society issued guidelines in 1976 which were similar to those used as a basis for therapy by both the Royal Navy and the US Navy until recently.

The site of bubbles dictates the symptoms and there is also the possibility of problems if a silent bubble becomes dislodged and becomes symptomatic[16]. Gas may be either extravascular or intravascular. If it is intravascular on the venous side, it always moves into a larger vessel finally reaching the lung filter[17]. On the arterial side (as witnessed experimentally through a cranial window), vessels become progressively smaller until the larger bubbles ($>50\ \mu m$) appear to lodge. Dilatation of the vessel beyond the point of contact occurs and then the bubble moves on until it either lodges again at a bifurcation of the vessel or disappears. Bubbles may coalesce to form 'slugs' with length one and a half times the diameter. Oxygen tends to reverse this effect[18].

Disadvantages of deep compression

An arbitrary recompression to 50 m may be deeper than the original dive and more inert gas will be taken up by tissues. Subsequent decompression may introduce further hazards: therefore the advantage of the shallower tables with oxygen giving shorter therapy and quicker washouts, appears considerable. However, Shields[19] refers to oxygen exacerbating a pre-existing gas phase and quotes Royal Naval experience that recompression is more effective than oxygen for eliminating a pre-formed gas phase. Oxygen-induced decompression sickness is described by Donald[20]; other cases of development of new serious symptoms following recompression with oxygen are also known[19].

Oxygen therapy

Paul Bert first proposed the use of oxygen in 1878 although it had been tentatively suggested by Pol and Watelle in 1854. Behnke and Shaw[21] subjected 26 anaesthetized dogs to exposures of 1 hr 45 min at 5.4 atmospheres absolute (ATA) and surfaced them in 10 s. If they were not recompressed, they developed gas bubbles in cutaneous vessels. Recompression on air or oxygen to the same depth relieved all animals. Those who were recompressed on oxygen did not have recurrence on subsequent decompression suggesting that oxygen aided in prevention of recurrence.

In 1945 Van der Aue developed new treatment tables for the US Navy advising use of oxygen in some cases but giving air alternative versions using, in one table, oxygen at 60 ft for 30 min. These tables gave a 90% success rate but the longer air tables (USN 3 and 4) which were used in more severe cases, were less successful, albeit used for more complex and delayed cases.

In 1965, Goodman and Workman developed the minimal recompression oxygen breathing tables using air breaks to avoid convulsions. These were particularly effective if swiftly used and had a success rate of 88.6%. They also avoided adding further inert gas load but the tables must be used according to the classification of decompression sickness by Golding (1960) allocating cases with serious symptoms to the appropriate treatment for these symptoms.

In 1973, Saumarez, Bolt and Gregory[22] successfully treated a case of severe neurological decompression sickness with oxygen at the surface and with adjuvant drugs but without recompression. In the case of neurological decompression sickness following a deep heliox dive treated initially on air at 50 m with rapid deterioration[23], a switch to 5% oxy-helium and a further recompression to 93 m produced complete relief of symptoms. The oxygen had been reduced by more than 50% and Shields emphasizes the results of this greater pressure, whereas James endorses this use of heliox[24]. The original authors offer no single explanation.

Shields (1981) describes cases following nitrox saturation diving[19]. Following a therapeutic recompression on 100% oxygen another diver developed DCS at the surface despite breathing oxygen himself. A similar case was also treated successfully by recompression to 30 m using 40% oxy-nitrogen ($P_{O_2}=1.6$ ATA). He concluded that adequate recompression was more effective than administration of oxygen in eliminating pre-formed gas. He described another patient who on being recompressed on oxygen to 12 m for 20 min developed a right hemiplegia which recovered on decompression.

Shields describes another case with residual neurological signs (following inappropriate treatment of arterial gas embolism) of bilateral sensory and motor impairment in all limbs which was successfully treated by hyperbaric oxygen.

Finally, in a young woman with residual signs following arterial gas embolism, using 100% oxygen at 10 m made her markedly worse. She recovered following decompression.

The North Sea Medical Centre's cases have involved two patients who have had monoparesis, apparently induced at 60 ft breathing oxygen on a therapeutic oxygen table and which cleared after extending the table, in one case to 8 cycles of oxygen at 60 ft rather than risking either further recompression on air or decompression. It was felt that the metabolic utilization of oxygen reduced the separated gas which might have caused the problem. If the problem was due to the theoretical ischaemic cuff at the site of the lesion, then it is to be hoped that the oxygen flux would improve 'the ischaemic penumbra' in the nervous system causing the lesions.

Treatment with delay in recompression: adjuvant therapy

Fructus[25] quotes Wolkiewicz and Plant-Longchamps

Table 1. Medical treatment of CNS decompression sickness during transport to hyperbaric chamber

Step	*Treatment*	*Dose*
1	100% oxygen by mask	
2	Corticoids i.v.	
	hydrocortisone hemisuccinate or	1000 mg
	dexamethasone or	30 mg
	medrocortisone	160 mg
3	Aspirin i.v.	1000 mg
4	Dextran 40 i.v. infusion	500 ml

who devised a treatment protocol for 67 cases of decompression sickness with a delay of between 3 and 24 hours between onset and recompression. He stressed that even with complete resolution, recompression *must* be instituted (see Tables 1 and 2).

Recognition of a decompression problem with swift initial action is the key to a successful outcome. Oxygen should be given by mask at the surface immediately[26]. If the patient is conscious, copious oral fluids should be given; if unconscious, intravenous fluids should be given and the patient catheterized.

Intravenous fluid

It had been common to use dextran because of the positive benefits of reduction of red cell aggregation. In 1985 it was reported that the Committee on Safety of Medicines had received a total of 55 reports of anaphylactic reaction in patients given dextran, with 10 deaths. This group of reactions represented the largest reported total for any single drug and it was advisable to consider whether the benefit of the use of dextran in decompression sickness was likely to exceed the risk of anaphylaxis (James, personal communication).

In 1983, the FDA Drug Bulletin described 12 adverse reactions to dextran including 3 deaths occurring during the first half of that year. The British Drug and Therapeutics Bulletin[27] gave an incidence of 1 in 2000 mild anaphylactic reactions, and serious reactions in 1 in 6000 infusions. Leitch[28] stated that dextrans had not proved superior to crystalloid solutions in practice and that current theory would favour crystalloids.

Adequate general nursing care including attention to pressure areas is important for the paraplegic patient in a chamber from the very onset of therapy.

Corticosteroid therapy

It has been customary to use steroids in cases of serious decompression sickness without proof of efficacy but with the support from successful case reports[29,30].

More recent support has been given for use of glucocorticoids because of possible benefit in improvement of synaptic transmission in the injured spinal cord[31]; the doses would be considerably larger than those currently used.

Problem areas in therapy

In 1976, a patient who had neurological decompression sickness with paraplegia was encountered who failed to respond to the then standard treatment initially with oxygen at 60 ft and then the deep air therapy table (165 fsw; 6 ATA: RN Table 62[32]); he steadily deteriorated during 'therapeutic' decompression and ascent was halted. Adjuvant therapy was given and a switch from air to heliox breathing mix was performed under guidance (Leitch, Pearson and Elliott, personal communication) to reduce the partial pressure of oxygen and to allow deeper recompression. Fears based on experimental evidence of counter-diffusion and worsening of symptoms fortunately proved groundless and he made some improvement, eventually walking successfully following further hyperbaric oxygen therapy daily.

In 1976, three other cases had been encountered at other Centres who had required deep recompression and extended time under pressure. Because of fears of oxygen toxicity, the reduction of the partial pressure of oxygen was arranged and here a nitrogen-oxygen mixture was used after slow decompression to 30 m on air. After stabilization for a considerable time on nitrox at 30 m, they were decompressed to the surface using air and oxygen with successful outcome. This was hailed as a success and proposals for emergency conversion of standard compressed air chambers into emergency saturation mode of therapy were made[33]. Three months later, one of the authors stressed that this was one further possible option but suggested it was a means of avoiding premature decompression by simply lowering oxygen tension and *not* proposed as curative[3,4]: 'Anyone who thinks that our aim was complete resolution of manifestations surely misunderstands our intent for experience has consistently shown the folly of such expectations'[34]. The article concluded that the use of helium-oxygen atmospheres may continue to have a place in treatment of commercial compressed air divers but was unlikely to be available for amateur divers in remote locations.

A review of problem areas in therapy

In the majority of cases of decompression sickness, if the patient is treated swiftly, there is rapid resolution. Most therapeutic tables start with a shallow oxygen recompression (18 m and 100% oxygen) with review

Table 2. Results of first aid during transport (53 patients)

Type of CNS decompression sickness		*Transport without treatment*		*First aid during transport*	
	No. of cases	*Unchanged*	*Improved or asymptomatic*	*Unchanged*	*Improved or asymptomatic*
Cerebral	23	8/8	0/8	3/15	12/15
Spinal	38	6/6	0/6	9/32	23/32
Mixed	6	0/0	0/0	3/6	3/6
Total	67	14/14	0/14	15/53	38/53
Percentage		100%	0%	28%	72%

after either 10 or 20 min[32,35]. In 1981, in review of 204 cases of serious decompression sickness over 20 years, the oxygen tables compared favourably with air tables[36] and were more effective after a 5 h delay than air tables. Where the short oxygen table (USN 5 or RN 61) was used inappropriately for serious symptoms, there was a high relapse rate.

In a comparison of recompression therapies in an animal model[37], the animals were divided into responders and non-responders to treatment, using somatosensory evoked potentials to measure spinal cord electrophysiological functions. The non-responders to therapy displayed a more rapid onset, a more severe insult pathologically, and more adverse physiological effects than the responders. Sykes[38] describes a pathology suggesting that autochthonous bubbles may play a more important role than was previously thought. These findings also suggest that new therapies should be directed at reducing ischaemic damage and protecting ischaemic neurones from further insult.

In 1981, the Diving Medical Advisory Committee commenced a revision of the 1976 guidelines on approach to therapy. A unified approach to cases was sought based on a search of the literature, case studies and reviews under a sub-committee chaired by Dr P B James.

James[24] outlined problem areas in therapy citing the rarity of neurological decompression sickness from heliox diving unless gas switches were being used on decompression, or in case of 'blow-up'. He gave 8 examples of death occurring where air had been used for recompression following heliox diving. He explains this as 'gas flux' (product of diffusion co-efficient and solubility) causing expansion of any extravascular separated gas phase of helium when breathing nitrogen. He proposed the use of heliox as a universal treatment gas in diving and that it might prove superior both to the use of pure oxygen at 2.8 ATA (18 m) and air in therapy.

The revised approach to therapy commenced breathing 100% oxygen from the surface and alternative flow charts were developed for failure to respond (after 20 min) at the first stop with the options of deeper recompression on heliox mix if symptoms were not cured or were worsening.

The use of helium was favoured because of the existing safe decompression tables for helium and oxygen diving and because of the reduced incidence of serious decompression sickness noted in helium diving. In a number of cases this approach has been used where an alternative breathing mix with a reduced partial pressure of oxygen has been forced through circumstances to avoid pulmonary oxygen toxicity but the numbers are small. Because of the inherent conservatism towards any new therapy until proved experimentally and then in clinical trials, it is fair to say that these proposals have not been universally accepted. Comex Diving company uses heliox at 30 m as one treatment for serious symptoms. Recent case reports with good results have given further encouragement to use of heliox[39] on clinical grounds.

The pulmonary effects of helium and oxygen breathing during experimental decompression sickness following air dives were studied[40] and showed that helium and oxygen breathing caused a 22% increase in pulmonary vascular resistance, at 1 ATA, from which they concluded that breathing helium and oxygen during decompression sickness resulting from an air dive can intensify pulmonary vascular obstruction However, the animals were not recompressed but were kept at the surface.

James[24] has put the case for the use of heliox during recompression in serious cases of decompression sickness after air dives and Hills[16] in support of this explains the beneficial effect of heliox breathing on nitrogen bubbles in a lipid tissue by pointing out that the flux of nitrogen would be greater than that of helium whether the exchange of gas in the tissue is limited by perfusion or diffusion. In perfusion, the solubility of helium in blood being less than nitrogen leads to offloading from the tissues of an inert nitrogen load. Similarly, in the case of diffusion, the flux in lipid is greater for nitrogen than for helium. To examine this, bubbles in rat adipose tissue were studied at 1 ATA after an exposure to 3.3 ATA for 4 h. During air breathing at the surface, the bubbles grew throughout the observation period whereas during heliox breathing they shrank and eventually disappeared from view. When the breathing gas was switched back from heliox to air, while the bubbles were still visible, they started growing again whereas if they had disappeared when the breathing gas was changed, then they did not re-appear[41].

The deep air tables have already been shown to have significant initial failure rates (up to 25%[10]) and even to induce decompression sickness in the healthy accompanying tender on 11 occasions in 57 times of usage of the longest air table (USN 4), an incidence of 19%.

The conclusion was that heliox breathing is useful in the treatment of decompression sickness with nitrogen bubbles dissolved in lipid tissues such as the white matter of the spinal cord. The debate continues.

Arterial gas embolism

A distinction is drawn between the treatment of cerebral arterial gas embolism and serious decompression sickness. In the former it is customary to recompress to a greater depth (50 m) for between 30 and 120 min breathing either air or nitrox. If complete relief is obtained within 30 min, then the decompression to 18 m followed by a long oxygen table is customary. If relief is not obtained within 30 min, then a slower form of decompression (USN 4 or RN 64) may be used.

In a review of 88 cases of decompression barotrauma, Elliott[42] noted that all cases that required additional recompression were in the group that spent 60 min, or less, at 50 m. No patient who spent more than 60, or up to 120 min at depth subsequently required recompression for relapse.

Gorman[43] described increased cerebral blood flow in rabbit with pure oxygen exposure at 2.8 ATA after embolism as opposed to 6 ATA on air, and has since treated successfully a considerable number of cerebral arterial gas embolism victims on oxygen tables at 60 ft. This has advantages where there is delay or potential difficulty if problems are encountered in more remote locations at greater depths on the deep air tables. However, Leitch[44] says there is no reason, in a review of 20 years' Royal Naval experience, to change from the current initial recompression to 50 m for arterial gas embolism, before returning to 2.8 ATA (60 fsw) to complete the therapy.

Leitch also showed that none of the patients with cerebral arterial gas embolism treated with steroids, suffered from a relapse.

The application of experimental therapies for cerebral ischaemia to the treatment of cerebral arterial gas embolism was reviewed in 1985 by

Dutka[45]. He concluded it would probably be impossible to develop a universally effective and easily administered therapy or even a combination or sequence of therapies that would be applicable to all cases. Immediate recompression and use of some increased partial pressure of oxygen was the most effective and the most easily administered therapy. Secondary deterioration should probably be treated with recompression or extended time under pressure as a first step, although the depth and partial pressure of oxygen remained unresolved. The danger of neuronal cell loss due to the toxicity of free oxygen radicals was mentioned. The usual cardiorespiratory resuscitation including defibrillation probably required management at the surface before recompression. Intravenous fluid should include some glucose (but not dextrose to avoid hyperglycaemia). Corticosteroids are currently recommended as a standard adjunct to recompression although their benefit in the acute stage is not proven.

Conclusion

Prevention through safe practice and early recognition and treatment are the most important issues today. In tunnellers, large numbers of cases, albeit mainly mild, occur. Lo and O'Kelly[46] report 2033 cases of decompression sickness and this affected 29% of the workforce. Medical selection and supervision is also important.

In diving, a review of commercial diving in the North Sea[47] reported a low total incidence of cases with disproportionately high number of neurological cases, related to the severity of the hyperbaric exposure and use of hot water diving suits.

The practising diving emergency physician must acquaint himself with the treatment schedules used by those for whom he is responsible, most of which have some proven record of success. Clearly, there remains much room for development of further methods of treatment.

Acknowledgments: This paper is based on the collective experience of Dr I K Anderson, Dr H A Evans and partners at Gorleston from 1965 onwards. I am grateful to Dr T G Shields and Dr I M Calder for reading the manuscript and to Ms Adele Southall for her assistance in its preparation.

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(Accepted 7 June 1988)

Long-term sequelae of diving

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Keywords: divers; osteonecrosis; neurological sequelae; decompression sickness

In the last few years there have been an increasing number of publications in the scientific literature which allege or report neurological and other permanent deficits in divers as a consequence of their occupation. In turn this has led to a number of articles in publications such as *The Economist* (8th August 1987), the *Sunday Times* (7th April 1985) and *New Scientist* (8th October and 24th December 1987) with the titles 'Divers at risk in lethal shortcut', 'Hidden cell damage puts divers in peril' and 'Diving disease linked to brain damage'.

Are these allegations true? If so, what is the nature of the problem? I will attempt to put these emotive issues into context, to demonstrate what is known, what is indeed new and, more importantly perhaps, what now needs to be done.

Hydrostatic pressure affects every cell in the immersed body. Changes in many biochemical and physiological processes occur during compression, depth exposure and decompression. These changes may be of no clinical significance and the vast majority are transient. Permanent effects are known to occur in several bodily systems but, for the purposes of this review, only two will be considered: the central nervous system, because it is now in the media spotlight, and juxta-articular bone because of the Medical Research Council Decompression Sickness Panel's experience with this over the last 30 years. It is that success that provides the precedent for a confident approach to the perceived central nervous system problem. Manifestations of permanent neurological damage due to diving have been known for more than 100 years but these are the sequelae of acute spinal cord decompression sickness. What is new is the suggestion that subtle lesions may occur in the absence of any reported history of decompression sickness. This concern, that neurological damage may occur 'silently' in members of the professional diving community, is growing regardless of the paucity of the evidence. The postmortem finding of demyelination in the spinal cord of a diver that was greater in extent than would be expected from his previous examination by a neurologist[1], together with some evidence on microscopy of damage in the brain of another diver, confirms that this is an issue that must not be ignored.

Turning to osteonecrosis, a major long-term consequence of diving, we find an example in which there were similar alarmist reports in the press ('Bone rot disease cripples divers'). Work by members of the MRC Panel has put into perspective the bone necrosis problem for compressed air workers and divers. Many

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