left wanting, e.g. a history of Guillain Barre Syndrome is "considered disqualifying" but "individual exceptions based on careful review, may be made". The advice on benign brain tumours concludes "One senior consulting neurosurgeon advises against diving after any of the above surgery".

The section on "Medications and Diving" covers only a third of a page and lacks any specifics. Even reference to ocular medications in the ophthalmology section refers the reader to a 1995 issue of *Survey of Ophthalmology*.

Some of the chapters are poorly laid out and specific conditions are lost in long screeds of text. The names could have been printed in a bold font for easy recognition. There is an index but it is very restricted, e.g. I read about conjunctival-dacryocystorhinostomies (Jones's Tubes) which was something new to me, but it is not listed in the index, so is difficult to find again.

The book purports to be about "medical examination" of sport scuba divers but no mention is made of a methodical and specific system for examining the diver, only a suggested medical history questionnaire is included as an appendix. The book would really be better titled "Lecture Notes on Medical Conditions Relevant to Scuba Divers".

Despite these problems the book contains much useful information and is worthy of a read to revise your knowledge but it is not recommended for doctors wanting to learn how to undertake sport diving medicals

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Key Words
Book review, diving medicals.

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THE DEVELOPMENT OF THE MINIMUM PRESSURE OXYGEN TABLES

Chris Acott

Abstract

The treatment of decompression illness (DCI) has been hindered by an incomplete understanding of the pathophysiology, the biophysics of bubble formation, inert gas uptake and elimination kinetics. Treatment protocols are based on minimal animal and human trial data and are to be found in military and government documents and so are difficult to review. This paper briefly traces the development of the recompression treatment tables up to the development of USN Tables 5, 5A, 6 and 6A.

Key Words
Decompression illness, history, oxygen, treatment.

Background

The treatment of decompression sickness (DCS) has been hindered by an incomplete understanding of the pathophysiology, the biophysics of bubble formation, inert gas uptake and elimination kinetics.[1,2]

The majority of the data relating to the development of recompression therapy is found in military and government documents and are difficult to review.[3] Reproduction of these various treatment guidelines and tables have varied with the edition of the Naval diving manual used or the particular Navy.[3] In 1978 Berghage prepared a report listing 67 different therapeutic tables used around the world.[4] Some of these were similar or identical, but were named differently, for example the Royal Navy (RN) 62 and the United States Navy (USN) Table 6.

Past therapeutic guidelines have been derived from various Naval protocols for example, in 1976 the Undersea Medical Society (UMS) issued guidelines for the treatment of offshore DCS which were similar to those used by both the RN and USN and are only relevant to military and commercial diving procedures.[5] All these guidelines reflect the view that DCS was an occupational disease confined to either military or commercial diving. There have been no treatment tables designed for recreational divers whose diving practises are totally different, being multilevel, multi-day and multiple dives per day.[6] Naval and commercial divers are treated immediately symptoms appear while with recreational divers there are inevitable delays to treatment.[6,7]

It is difficult to predict the response to recompression. There may be a group of patients who will respond to any recompression and another group who are refractory to treatment. The longer the delay to treatment the worse the initial response to treatment and the probably the poorer the outcome. However, what constitutes a delay to treatment has not been clearly defined.[1,2,6,7]

Data on outcome has largely been anecdotal and based on different variables. Many reports were derived from retrospective studies and often included non-medical opinion about the success of a treatment protocol. Decompression sickness has often been regarded as an accident, rather than a disease, consequently those treating cases have looked for someone, or something, to blame for its occurrence. Whether this mind set has affected the management and outcome of these cases is unknown.

Pathophysiology

Although Boyle[8] demonstrated bubble formation in living tissues in 1670 and Bert,[9] in 1878, showed that these bubbles were nitrogen, early attempts to explain symptoms following a reduction in ambient pressure included such things as reflex spinal cord damage caused by either by exhaustion or cold, frictional tissue electricity caused by compression, or decompression induced organ congestion and vascular stasis.[3,10-12] Even though all the salient clinical features of DCS were established between 1870 and 1910[8] the complete pathophysiology of DCS is yet to be defined.[1,2] The primary event is the formation of intravascular or extravascular bubbles, which can have mechanical (obstructive, disruptive or compressive), physiological or biochemical effects, however, the relative importance of these effects is still being determined.[6]

Outcome studies

Recompression therapy in commercial and military divers has been so successful that any controlled human studies have remained virtually non-existent.[7] The majority of the human studies have been analyses of case histories and outcome. These studies have several weaknesses: they are retrospective, the initial evaluation may be based on a non-medical opinion and subjective terms (i.e. substantial, relapse, recurrence or minor sequelae) are not clearly defined.[13] In addition, before 1985 the USN did not describe a complete neurological examination in its Diving Manual and so earlier reports would have underestimated the occurrence of central nervous system involvement and so an inappropriate treatment table may have been used.[14,15] Outcome data also vary widely because they are based on different variables (the population studied and the sensitivity of the assay) making any comparison between studies difficult. They therefore need to be viewed with an emphasis on the parameters measured. All studies concentrate on the treatment table used; there is little detail about resuscitation, particularly the prevention of secondary central nervous system damage due to an obstructed airway, or the adequacy of any fluid replacement (type, amount and by which route). In 1987 Green et al. reviewed 430 cases of decompression sickness treated by the RN (250 of these were considered to be serious and 180 were pain only) and found only 18 received intravenous fluid (the amount and type of fluid was not mentioned).[15]

The natural history of DCS may sometimes be spontaneous recovery or improvement. Some early studies report spontaneous remission of both pain only and serious symptoms. In 1870 Bauer published a report of 4 deaths in 25 paraplegic patients but the majority recovered within 1-4 weeks.[9] In 1872 Gal published a report in which paralysed patients either recovered over 5 days to 3 weeks or died from the septicaemic complications of bed sores or cystitis.[9] Both Woodward (in 1881) and Blick (in 1909) reported that the majority of pain-only cases and some of the neurological problems spontaneously resolved.[16] Recently, Green et al. reported spontaneous recovery or improvement in 8 cases of Type 2 DCS.[15]

Recent studies of the treatment of recreational divers have altered the data about successful outcome. These divers' dive profiles may be unknown or uncertain, there is a great variation in their medical and physical fitness and there is usually a considerable delay after the onset of symptoms before presenting for treatment. Delay before recompression treatment is thought to reduce success, however, the time period that constitutes a delay has not been clearly defined.[1,2,6,7] Recently, Lam and Yau, treating compressed air workers, suggested that the delay time should be measured from the conclusion of the dive, not from the development of symptoms, to the commencement of treatment.[17] In 793 cases they found that for every hour's delay, using their definition, there was an additional 0.04 bar pressure requirement for pain relief. Early treatment needs well-trained divers and compressed air workers able and willing to recognise early symptoms, an accessible hyperbaric chamber and a readily available team of treatment professionals. In addition, the period of delay after which no benefit from recompression and hyperbaric oxygen can be obtained is uncertain.[3]

Controversial issues

By 1939 recompression had become the accepted method of treatment but there was disagreement concerning its application.[18] Even today similar controversies exist: which treatment depth to be used in unresponsive or deteriorating cases,[19] what is the optimum pressure of oxygen to use and what diluent inert gas to use with oxygen.[3] The use of saturation therapy for non-responders or the repeated use of hyperbaric oxygen after initial treatment is still being debated, but there is no agreement on which hyperbaric oxygen table should be used.[20,21]

Development of the therapeutic tables

Decompression sickness was first described by Triger

in coal miners in 1841.[9,10] Recompression was first proposed as treatment in 1847 by Pol and Watelle, whose patients were coal miners working in compressed air to deepen the mine shafts.[22] Bert (1878), Moir (1889), Snell (1895) and Zuntz (1897) were other early proponents of recompression.[9,10,23,24] Heller et al. in 1907 and Keays in 1909 used recompression on an ad hoc basis.[10,25] Keays showed persistence of symptoms in 14% of caisson workers who were not recompressed compared to 0.5% in those who were.[25] Until 1912, when Ryan published the first treatment regime, the treatment of DCS had been on an ad hoc basis. Ryan suggested a return to 2/3rds the original pressure followed by a slow decompression.[10] In 1917 Levy advocated a return to the original pressure, again followed by a slow decompression.[10] Both regimes had limited acceptance. In 1924 the first standardised recompression tables were published by the USN. This recommended that the diver was rapidly recompressed to 45 pounds per square inch gauge (psig) (approximately 30 msw or 4 bar) with further recompression to 60 psig (approximately 40 msw or 5 bar) if there was no improvement. Decompression was started as soon as the symptoms resolved. The USN published another table before 1937 which recommended recompression to the depth of relief plus 1 atmosphere, decompression from this depth was the diving table air decompression schedule for that depth .[3]

In the 1930s the RN began using oxygen decompression in air dives to 300 fsw (90 msw or 10 bar). This procedure was based on animal experiments (12 goats), human chamber testing (10 divers) and actual naval dives (58 dives).[26]

By 1935 the USN air treatment tables were noted to afford relief only in mild cases so Behnke and Shaw began experimenting with the use of oxygen. They believed that oxygen should be used because it would create a maximum elimination gradient for nitrogen and afford immediate relief of bubble induced ischaemia.[27] Behnke proposed that reluctance to use oxygen had been due to a lack of conclusive experimental data on its efficacy, a lack of suitable facilities for administration and the fact that human tolerance was unknown. Behnke et al. conducted human oxygen tolerance studies using 12 divers. The data showed that 100% normobaric oxygen could be breathed for 6 hours without pulmonary symptoms while convulsions occurred after 3 hours at 3 bar (ATA) and 45 minutes at 4 bar.[27,28] They compressed 26 anaesthetised dogs to 5.4 bar for 105 minutes and then surfaced them in 10 seconds to produce severe cardiopulmonary and neurological DCS. The dogs were then recompressed to 3 bar (20 m) either breathing 100% oxygen or air. They choose 3 bar because of their human oxygen tolerance studies data and the postulated eleven fold increase in nitrogen elimination compared to air at 1 bar. Both groups initially responded well to recompression, but the dogs recompressed on oxygen had a better outcome with fewer recurrences of symptoms. They concluded that oxygen recompression to 3 bar had a better outcome than recompression using air, that 3 bar (20 m or 66 ft) was not an adequate pressure to reverse the CNS signs (paralysis) and that severe DCS caused plasma loss. They noted that the bubbles were eliminated within 1 hour in these dogs but concluded if this was to occur in humans 2 hours would be needed because man's circulation time is twice that of a dog. [28]

Their next experiments were designed to test the pressure needed to prevent or reverse the CNS signs/ symptoms ("to prevent paralysis").[27] Eight dogs were used in 15 experiments and again their model of severe cardiopulmonary and neurological DCS was used. These dogs were recompressed to 60 psig (5 bar or 40 msw) breathing a 50% nitrox mixture, or air if this was not available, oxygen was not used in decompression. In the first 8 experiments 1 dog failed to develop symptoms and so was not recompressed, 3 recovered, 1 partially responded, 2 failed to respond and 1 died following recompression. Overall the data from the 15 experiments showed that in 7 experiments the dogs survived (1 dog was used 4 times but failed to recover in the 4th experiment, 1 dog responded in 1 experiment but failed to respond in a subsequent experiment, 1 dog recovered function after 14 days having fully recovered on a previous experiment, 1 dog required 2 treatments); 6 dogs had an incomplete recovery; 1 died and 1 failed to produce symptoms in 1 experiment but failed to respond to treatment in another. From the data from these 2 groups of experiments Behnke and Shaw concluded that any serious symptoms would require a combination of rapid recompression and hyperbaric oxygen. These two groups of experiments are also important because they were the basis upon which subsequent treatment depth and oxygen pressure have are based .[27]

In 1937 Behnke and Shaw published their oxygen tables based on these animal data. For serious cases the maximum depth of 50 msw (165 ft or 5 bar) breathing either a 50% nitrox mixture or air followed by a decompression to 60 fsw (18 m, 2.8 bar) over 45 minutes where oxygen was to be breathed for 1-2 hours. The patient's response determined the time spent at 165 fsw (minimum 15 minutes and maximum 2 hours). One hundred and sixty five feet (165 fsw) was chosen because:

1 bubble shrinkage would be to 1/6th its surface volume;
2 pressure resolution of all bubbles was thought to require exceedingly high pressures and, by the time the serious cases were recompressed, tissue gas would have diffused into the blood stream limiting its capacity for any further absorption. They preferred to use oxygen at 3 bar for complete elimination of gas emboli.

For mild cases (pain only) they recommended recompression to 60 fsw (18 m or 2.8 bar) breathing 100% oxygen for 1 hour followed by a 30 minute decompression. For unrelieved symptoms they suggested a prolonged stay

at 60 fsw (18 m or 2.8 bar) breathing oxygen for 2-3 hours in every 24. They limited the exposure to 3 bar oxygen in 24 hours to 3 hours.[27] These tables were not published or used by the USN because the USN Bureau of Medicine and Surgery decided that oxygen breathing in a chamber was not "sailor-proof". The risks of an oxygen convulsion and fire were considered too great for it to be used universally by the Navy.[3] However, these tables served a model for future recompression procedures.[3]

In 1939 Yarbrough and Behnke recognised that effectiveness of recompression was related to its prompt application and recommended that for 12 hours following a dive a diver "should not be further removed than 1 hour's travel time".[18] They based this maximum one hour delay on their own clinical and experimental data and data from Keays, Langlois and Bornstein.[18,25]

By now there was agreement that recompression was the treatment of choice, however, at that time there were 4 approaches to the depth to be used:

1 compression to the depth of relief;
2 compression to a depth greater than that required for relief;
3 compression to the depth of the original dive;
4 compression to a depth greater than the original dive.

Yarbrough and Behnke considered that, because the amount of gas in bubble form was unknown, options 3 and 4 (compression to the depth of the dive or deeper than the original dive) could be eliminated from therapeutic consideration. Their guide was the relief of symptoms, and 1 ATA (bar) was added empirically to completely restore circulation to any affected tissue. Using Behnke and Shaw's data[27] the minimum pressure for the treatment of mild cases (symptoms relieved at a depth less than 30 fsw (20 m or 3 bar) became 100 fsw (45 psig, 4 bar or 30 m) with oxygen being used from 60 fsw (18 m or 2.8 bar) if available. Decompression was staged, based on the Haldanian principle, so that the tissue gas pressure never exceeded the ratio of 2:1 compared to the environmental pressure. These tables became known as either the "short oxygen" (when oxygen was used) or "short air" table.[18]

In serious cases immediate recompression to 75 psig (6 bar, 165 fsw or 50 msw) breathing either a 50% nitrox mixture or air was recommended with oxygen being used from 60 fsw (18 m or 2.8 bar) if available. These tables became known as the "long oxygen" (when oxygen was used) or "long air" table. Again decompression was based on the staged Haldanian principle.

Time spent at 165 fsw was to be between 30 and 120 minutes. This was based on;

1 Behnke et al.'s previous clinical and animal data;
2 Borstein's recommendation of 30 minutes;[18]
3 and R H Davis's opinion that it was "useless to wait longer than 2 hours".[18]

If the treatment failed then the patient was immediately recompressed to the pressure of relief (this was usually found to be less than 3 ATA) and maintained for 12-24 hours followed by a slow decompression. This introduced the treatment concept of an overnight soak. These tables were published in the BUMED News Letter in 1944 .[3]

Development of tables 1 to 4

The long oxygen treatment table was used to treat 50 cases of "helium bends" and was successful in 49 patients.[18] However, when it was used to treat 10 air divers there was a 50% symptom recurrence rate with an overall failure rate of 30% in 30 divers treated. Even in human trials the long oxygen table failed in 6 out of 10 divers when tested. The shorter oxygen table, however, was successful in the 6 divers for whom it was used.

By 1945 it was apparent that these tables gave no better results than using the regimes of 1924. These failures led to Van Der Aue et al developing the USN Treatment Tables 1-4 (with Treatment Table 1A and 2A using only air).[29] Development of these tables involved subjecting healthy divers to a 130 fsw (39 m)/60 minute working dive and after a 30 or 60 minute surface interval using the treatment table under evaluation. If the treatment table did not prevent DCS it was modified until no DCS occurred. A total of 84 dives were made using 33 subjects, however, when Table 4 was tested, there were no preceding work dives and the 6 subjects tested all reported fatigue following exposure.[3,30] The short oxygen table became USN Table 1, the short air table was lengthened and became USN Table 1A, 60 minutes of oxygen breathing were added to the long oxygen table (which became USN Table 2) and the long air table was lengthened and became USN Tables 3 and 4. The RN developed their equivalent tables (RN 52, 53, 54, 55) a little later and these tables remained in use world-wide in both commercial and military diving for the next 20 years.[3,30]

In 1947 Van Der Aue et al. reported on the first 113 patients treated with these new tables.[31] Complete relief occurred on the initial compression in 107 cases, 4 had recurrences and 2 had residual problems. The initial success rate with tables 1-4 was excellent with an overall reported failure rate of 6%.

These tables were not subjected to a further review until Slark in 1962 and Goodman in 1964 published studies which showed failure rates of 24-47%.[32,33] In these series, recreational divers accounted for almost 46% of the initial treatment failures. There were no reported failures of Tables 3 or 4 when used promptly on naval divers.

These reports led Goodman and Workman to begin a series of treatments based on moderate pressures of 100%

oxygen. They believed that these "minimal pressure" oxygen tables relieved ischaemia without further exposure to inert gas and provided a maximum gradient for inert gas washout. They also tried to convince diving physicians that deeper treatments were not better because of the decrease in bubble volume vs radius changed little at depths deeper than 18 m (2.8 bar) and that deeper treatments were paralleled by an additional inert gas uptake.[34] Goodman defined what he considered to be the fundamental aspects of the treatment of DCS:

1 compression to reduce the bubble volume and radius to decrease any tissue reaction;
2 relief of focal ischaemia caused by endothelial irritation.[33]

Assuming the strength of Tables 3 and 4 was the 33 fsw (10 m or 2 bar) soak, they began conducting trials at this depth. The patient was compressed to 33 fsw (10 m) breathing oxygen. If all the symptoms were relieved after 10 minutes the patient completed an extra 30 minutes and then decompressed. If relief was not obtained after 10 minutes then the patient was further compressed to 60 fsw (18 m). They treated 150 divers (110 military and 40 civilian) with this regime. The 5 divers (4 military and 1 civilian) treated at 33 fsw had recurrence of their symptoms and had to be treated again.[34] Retrospective statistical analysis of their data showed that oxygen breathing time and depth were related to the treatment adequacy (the minimum adequate exposure time was 30 minutes with a 90 minutes total treatment time). As a result the 33 fsw (10 m) treatment was abandoned and all divers were recompressed to 60 fsw (18 m). Alternation of oxygen breathing with air for periods of 5 to 15 minutes was introduced to reduce the risk of oxygen toxicity. USN Tables 5 and 6 were developed from their data.[34]

Arterial gas embolism

Arterial gas embolism is a relatively recent diving disease. It was clinically defined in the 1930s after the beginning of submarine escape training.[10] Following the development of USN Tables 1-4 it was either treated on Table 3 or 4 which meant a 22 or 38 hour stay in the chamber irrespective of the patient's inert gas burden. Before the development of Tables 3 and 4 gas embolism was treated on the same protocols used for serious DCS.

In 1967 Waite and Mazzone began to re-evaluate the treatment of gas embolism.[35] In a series of experiments they embolised 14 dogs, observing bubble behaviour through a cranial window. Eleven dogs were successfully embolised, 8 were embolised at 1 bar and 3 at 2 bar. Five of the dogs embolised at 1 bar were not treated. Two died within 20 minutes and 3 survived with residual problems. The other 3 dogs embolised at 1 bar were treated with a dive table of 170 fsw (52 m) for 10 minutes with staged decompression. The 3 embolised at 2 bar were surfaced and then treated with the same protocol. All the treated group survived. It was noted that all the bubbles disappeared between 3 and 4 bar with no bubble reappearance following decompression. Because of the prejudices of the USN Bureau of Medicine and Surgery only 6 bar (50 m or 165 ft) had to be used in any treatment tables.

Waite et al. at the USN Submarine Medical Center later modified USN Tables 5 and 6 for the treatment of cerebral arterial air embolism in submarine escape trainees. These tables were called Tables 5A and 6A.[35]

Table 5A was later abandoned in 1976 because it did not allow enough time to assess if there has been any resolution of symptoms.[36] The diver would also not have had enough time to adjust to the thermal stress, noise and narcosis of a rapid compression. These would also interfere with the attendant's assessment.[37]

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