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Treatment of decompression illness in the 21st century: a brief overview

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Abstract

(Mitchell SJ. Treatment of decompression illness in the 21st century: a brief overview. *Diving and Hyperbaric Medicine*. 2007; 37: 73-5.)
Definitive treatment of decompression illness usually involves recompression and the administration of hyperbaric oxygen. Little recent progress has been made in the development of recompression strategies. The 'short oxygen table' remains the mainstay, though variations in pressure, duration of treatment, and respired gas have their advocates. Other 'adjuvant' treatments are sometimes employed. Only one adjuvant treatment, a non-steroidal anti-inflammatory agent, is supported by directly relevant human data. The value of intravenous fluids is often self-evident in serious decompression illness, but benefit is less certain in mild disease. There are data of indirect relevance supporting the use of lignocaine in arterial gas embolism. Other agents such as nitric oxide donors and intravenous perfluorocarbon emulsions await testing in humans.

Introduction

Treatment of decompression illness (DCI) had its beginnings in the recompression of sufferers who simply returned to work under pressure. It evolved through the development of therapeutic recompression schedules utilising air breathing, and subsequently oxygen breathing. There has also been much interest in adjuvant strategies that are mainly pharmacological in nature.

Recompression

Despite the lack of any evidence beyond non-randomised studies, recompression and administration of hyperbaric oxygen remain the mainstays of modern treatment for DCI. Compression reduces bubble volume in accordance with Boyle's Law, and oxygen breathing hastens the elimination of inert gas. Remarkably little recent progress has been made in the development of recompression protocols. The so-called 'short oxygen tables' such as the US Navy Table 6, which were popularized in the 1960s, remain the most commonly used to this day. These treatments utilise oxygen breathing at a pressure (typically 2.8 ATA) chosen to optimise the 'oxygen window' whilst maintaining the risk of oxygen toxicity within acceptable limits.

Other parameters of recompression may be varied

Most practitioners recognise the utility of varying the duration of recompression in accordance with the severity and clinical response in individual cases. The most common approach is the prescribed 'extensions' to US Navy Table 6 (Royal Navy Table 62), which allow extended periods at both 2.8 and 1.9 ATA during decompression; but there are many variations, including controversial 'saturation' treatments for very serious and refractory neurological disease.[1]

There is also the option of compressing to greater pressures, at which oxygen breathing would not be appropriate. While there is supportive anecdote and some data from case series,[2] this option is also controversial. The previous fashion for an initial 'deep' spike (usually to 6 ATA) for serious DCI or those cases perceived to be caused by arterial gas embolism (AGE) has waned. This has occurred in the light of conflicting animal evidence on the effects of greater pressure on arterial bubble redistribution, the logistical and safety implications of these 'deeper' tables, and the lack of any convincing demonstration of significant benefit.[1] Nevertheless, 'deep' treatments still have their advocates,[2] and treatment protocols that specify escalating treatment depth in the face of refractory and serious disease are sometimes used at appropriately configured hyperbaric units.

In the treatment of DCI following air diving there are theoretical advantages in utilising helium as the diluent gas during periods where the F_iO_2 is lowered to minimise the risk of oxygen toxicity. This is especially so during 'deep' treatments at pressures greater than those where 100% oxygen can be safely used. There is some experimental evidence[3] and limited clinical data[4] in support of this practice, but it cannot be considered proven.

The somewhat extraordinary issue with all of these combinations and permutations of recompression strategy remains the near total absence of strong evidence to facilitate

objective choices. Notwithstanding the strength of anecdote, the same could be said for the efficacy of recompression *per se* in treating DCI. Indeed, in the absence of any controlled data the use of recompression and hyperbaric oxygen has evolved into an impregnable 'standard of care' for DCI of virtually any severity. It is therefore notable that a recent consensus of experts found that the final outcome for patients with mild DCI (strictly as defined in the consensus) is not compromised by not being recompressed.[5] This has significant implications for the management of mild or equivocal cases in remote locations where accessing recompression would involve very costly and inconvenient evacuation.

Adjuvant therapies

The well-recognised potential for incomplete resolution of DCI, especially the more serious forms, has led to much interest in adjuvant therapy that might improve outcome.

Intravenous fluid administration has long been considered important, and its necessity is self-evident in those rare severe cases that present with haemoconcentration and shock.[6] It is much less clear whether fluid administration is useful in milder cases. There are no relevant human data.

A fashion has developed for the use of intravenous lignocaine in the treatment of severe DCI. This is based on a series of *in vivo* experiments that demonstrated neuroprotection by lignocaine in arterial gas embolism (AGE) and in numerous other models of brain injury. These various studies are summarised by Mitchell.[7] In addition, two small randomised double-blind controlled trials demonstrated less post-operative neurocognitive impairment in humans given lignocaine during heart surgery.[8,9] Whilst there are some parallels between brain injury in cardiac surgery and diving, the extrapolation of these results to AGE in diving is drawing a long bow since in both studies the lignocaine was given prophylactically and clearly this cannot be the case in diving. Nevertheless, the Adjuvant Treatments Committee of the Undersea and Hyperbaric Medical Society considers the use of lignocaine to be potentially beneficial in cases of AGE presenting early for treatment.[10] A bolus and infusion regimen as per guidelines for an antiarrhythmic effect is recommended, and the infusion should be continued for 24 to 48 hours.

Administration of non-steroidal anti-inflammatory drugs (NSAIDs) has anecdotally been associated with quicker recovery from DCI. This issue was addressed in a randomised double-blind controlled trial of tenoxicam versus placebo (the only such trial of *any* intervention in DCI) involving 180 divers conducted at Prince of Wales Hospital in Sydney.[11] This study demonstrated no improvement in final outcome with tenoxicam but the median number of recompression treatments was significantly reduced in the NSAID group. Treatment of five patients with tenoxicam was required to prevent one hyperbaric treatment. NSAIDs are the only 'proven' adjuvant treatment for DCI.

There has been much speculation that potent steroids may improve outcome in DCI. There are no relevant human data, but a recent study of methylprednisolone administration in pigs with DCI demonstrated worse outcome with steroid treatment.[12] Steroids are not recommended in the treatment of DCI.[10]

One interesting and recent development has been the finding of reduced venous bubble formation after decompression in pigs[13] and humans[14] pre-treated with standard doses of nitrate agents. This line of research arose out of the hypothesis (which was subsequently disproved) that a reduction in bubble formation induced by appropriately timed pre-dive exercise might be a result of upregulation of NO production by endothelial cells. Pre-dive exercise appears to impart its benefit by a different mechanism, but the initial misattribution to NO effects has inadvertently sparked a new line of research that might be relevant to treatment of DCI. If the reported nitrate effect is confirmed in humans, and given the rapid kinetics of NO donation by nitrate drugs, it is perhaps only a matter of time before nitrates are tested in early presentations of DCI as a potential means of limiting ongoing bubble formation.

Perhaps the most exciting unrealised prospect for adjuvant therapy in DCI is the use of intravenous perfluorocarbon emulsions (PFCEs). These are chemically inert, water-insoluble aromatic or aliphatic compounds with fluorine substituted for all hydrogen atoms.[15] They have low surface tension and high solubility for gases. Oxygen is 100 times more soluble in PFCEs than plasma, and this has generated interest in their use as 'blood substitutes'. Their related ability to accelerate inert gas transport in blood has resulted in interest in their use to treat DCI – either in isolation or as an adjuvant to hyperbaric oxygen. Latson has summarised the numerous *in vivo* studies that have demonstrated significant benefit in both DCS and AGE.[15] Since that review there have been two further relevant studies that demonstrated benefit.[16,17] The lack of a reliable source of PFCEs licensed for human use has hampered human research in DCI to date.

A final recent development is that the field of DCI treatment now has its own Cochrane Review.[18] Although there are few studies suitable for inclusion in such a review at this point, it is hoped that future iterations will include randomised controlled studies of some of these promising prospects for adjuvant treatment.

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