Review articles

Adjunctive therapy for decompression illness: a review and update

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Key words
Decompression illness, decompression sickness, diving accidents, air embolism, first aid, treatment, review article

Abstract

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Therapeutic interventions may augment the outcome or provide temporizing support pending recompression. Effective measures include first aid (surface) oxygen therapy, fluid resuscitation, non-steroidal anti-inflammatory drugs and avoidance of fever. Lignocaine may also be effective, particularly for cerebral arterial gas embolism (CAGE). For critically ill patients with CAGE, animal studies do not support the use of hyperventilation. There is strong experimental evidence in animals for the efficacy of intravenous perfluorocarbon. When lower limb paralysis occurs, low molecular weight heparin is recommended to reduce the risk of venous thromboembolism.

Introduction

Adjunctive therapies had their origins in the nineteenth century during the early days of compressed air work. Prior to the advent of therapeutic recompression therapy a variety of concoctions and techniques were empirically used. These included ergot, morphine, atropine, alcohol, ginger and phlebotomy. Dr Andrew Smith, physician for the Brooklyn Bridge construction wrote the following:

"*On my recommendation a cup of good coffee was served to each man immediately upon leaving the caisson. It appeared to relieve, in a measure, the nervous prostration which marked the return to the open air; and possibly, by the effect which coffee is known to have, it may have done something, also to check the tendency to too rapid tissue-change.*

In some instances I have obtained the very best results from hypodermic injections of atropine at the seat of the pain; but in other cases it failed to procure relief, and, upon the whole, I consider it inferior to morphine."

"Starting from the theory already given as to the mode in which the disease is produced (a theory which was constructed wholly upon the observations of others), I was led to the idea that benefit would be derived from the use of an agent that would induce contraction of the capillaries, and thus correct the want of tone which I consider to lie at the foundation of the difficulty. For this purpose I proposed the use of ergot before I had ever seen a case of the disease. I reasoned that it would be useful, first, by contracting the vessels of the brain and spinal cord, and relieving their congested state; and secondly, by restoring tone to the superficial vessels, and thus imparting vigor to the circulation.

An extended trial warrants me in saying that the results have justified the theory. In my hands, though not always successful, ergot has certainly been very useful in a considerable number of cases. I have seen very severe pain completely relieved within half an hour after the administration of a drachm of the fluid extract. In other instances, unsteadiness of the limbs, which seemed about to usher in paralysis, has yielded promptly to one or two doses."

"But perhaps the best evidence of its usefulness is to be found in the preference for it of the night-porter, who had charge of the hospital at night, and who was instructed in the use of the few medicines employed, and treated such cases as occurred among the men composing the night gangs. Having both morphine and ergot at hand, he gradually, and of his own accord, almost abandoned the former, declaring that the ergot was more prompt and certain in relieving the pains. This from an intelligent, unprejudiced, non-professional source, is strong testimony in favor of the efficacy of the drug."[1]

After recompression therapy became routine, adjunctive therapies fell out of favour until the latter part of the twentieth century, when it became apparent that decompression illness was often accompanied by severe dehydration and can be complicated by thromboembolism, which is sometimes fatal. Additionally, because civilian diving accidents often occur far from recompression chambers, there has been an impetus to develop therapies that can provide temporary support during transport. Currently available adjunctive therapies are shown in Table 1.

Table 1
Adjunctive agents for decompression illness

First-aid oxygen
Fluid resuscitation and management of plasma glucose
Antiplatelet agents and DVT prophylaxis
Corticosteroids
Lignocaine (lidocaine)
Body temperature management
Manipulation of $PaCO_2$
Perfluorocarbons

First-aid (surface) oxygen (O_2)

Paul Bert published his observations on experimental animals with decompression sickness in 1878.[2] By direct observation, he found that oxygen (O_2) administration to dogs with decompression sickness caused resolution of intravascular bubbles. The value of first-aid oxygen (F_AO_2) has been confirmed by clinical experience and a systematic review of 2,231 consecutive diving accident reports.[3] F_AO_2 was often associated with complete relief of symptoms prior to recompression, and reduced need for more than one recompression treatment (Figure 1). Receiving early F_AO_2 (within four hours of surfacing) was more efficacious.

Fluid resuscitation and management of plasma glucose

In 1871, Alphonse Jaminet, physician for the Mississippi Bridge project at St Louis, published his observation on urine specific gravity of men with and without decompression sickness.[4] Although he did not interpret the results in this context, retrospective analysis of his observations shows that urine specific gravity was significantly higher in the men with decompression sickness compared to those without (Figure 2). The higher specific gravity in symptomatic men is consistent with dehydration. Studies published in the middle of 20th century described haemoconcentration in cases of severe decompression sickness (Figure 3).[4,6] In 1964 Brunner published two cases of severe decompression sickness (DCS) after experimental dives with haemoconcentration. In those cases he used radioactive tagging to demonstrate that plasma volume was reduced in the face of normal packed erythrocyte volume and that the plasma deficit could be corrected with administration of plasma.[7]

A more recent paper has described the association of haemoconcentration with sequelae of decompression illness (DCI) (Figure 4).[8] Divers with neurological DCI with and without neurological sequelae one month after treatment and a series of control divers without symptoms were compared; the haematocrit was significantly higher in the divers with

Figure 1
First-aid oxygen and outcome in a series of recreational divers with DCI; * $P < 0.05$; ** $P < 0.001$

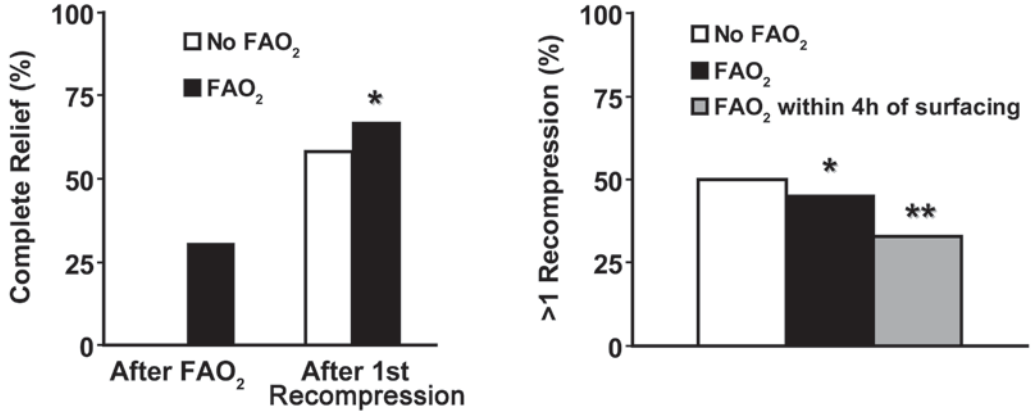


Figure 2
Urine specific gravity in compressed air workers with and without DCS (mean ± SD); * $P < 0.05$
Data plotted from observations by Jaminet[5]

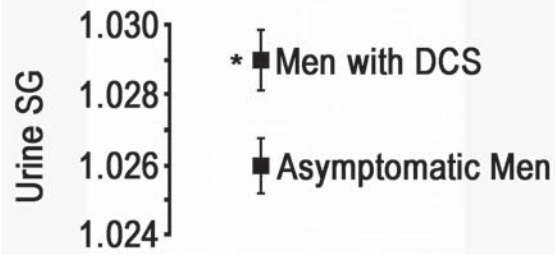


Figure 3
Plasma volume decrement in 7 cases of severe altitude-induced DCS (from Malette[6])

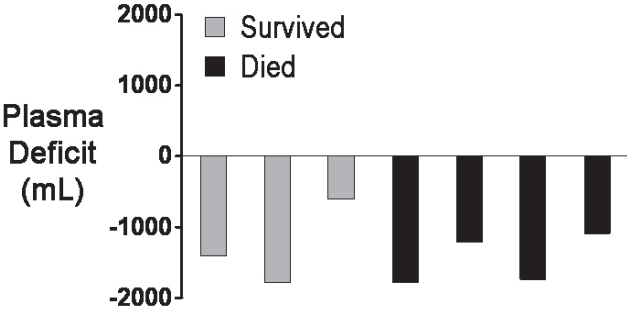


Figure 4
Haematocrit in divers with DCI; median, minimum and maximum haematocrit values are shown. Data plotted from Boussuges[8]

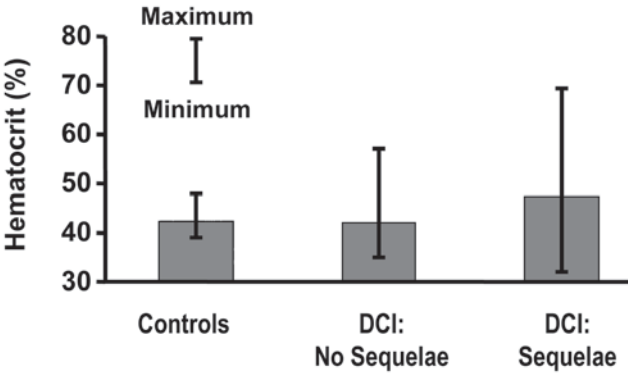
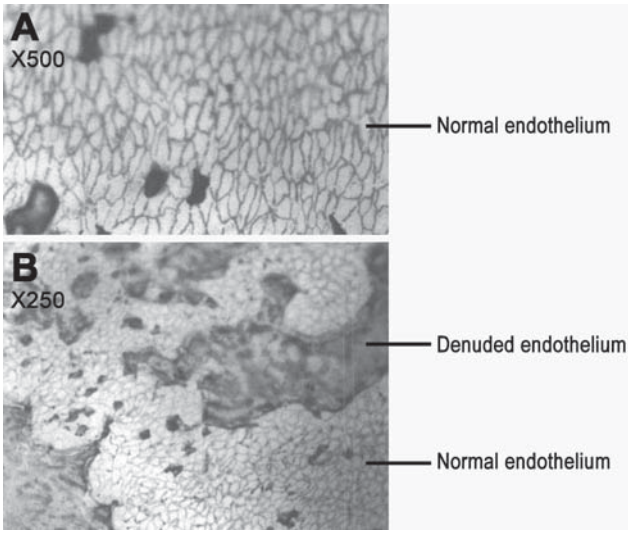


Figure 5
Light microscopy of endothelium from the pulmonary artery of a pig subjected to decompression-related venous gas embolism (VGE); A: normal endothelium; B: endothelium partially denuded after VGE; from Nossum[9], with permission



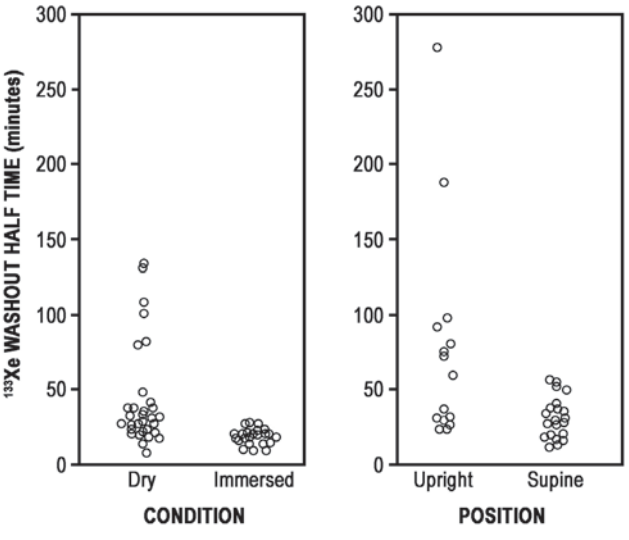
sequelae. The mechanism for haemoconcentration appears to be the result of bubble-induced endothelial damage (Figure 5) and extravasation of plasma into the interstitial space.

Evidence that fluid administration may be helpful in DCI is suggested by a study by Balldin and Lundgren in which inert gas washout (^{133}Xe) from the anterior tibial muscle is facilitated by immersion in water and the supine position (Figure 6).[10] Both head-out immersion and supine position were associated with faster Xe washout, consistent with the expected greater tissue perfusion. These data support the use of fluid resuscitation in patients with DCI in order to facilitate inert gas washout. Immersion causes redistribution of blood from venous capacitance vessels to the central circulation, equivalent to a blood or fluid transfusion, and hence increased tissue perfusion.

The accumulated experience over the last 130 years strongly supports the use of fluid resuscitation in decompression illness. Hypotonic fluids, such as dextrose-water or half normal saline, reduce plasma osmolality and can cause swelling of the central nervous system.[11] Moreover, elevation of plasma glucose can augment CNS injury.[12] Therefore, fluids that contain glucose should be avoided. Ideal fluids include isotonic fluids, either colloids or crystalloids. For severe cases large volumes are often required. In one patient with severe DCI treated at our facility, reduction in his haematocrit from 66% to 48% required 12 litres of crystalloid and 1 litre of starch solution. Aggressive fluid resuscitation is not recommended for patients with isolated cerebral arterial gas embolism (CAGE), either diving-related or iatrogenic, unless there is concomitant haemoconcentration.

If oral fluids are used for resuscitation, their ideal composition for rapid absorption and maintenance of electrolyte balance has been determined. The ideal oral fluid for resuscitation of a dehydrated patient should contain 30–60 mM.L^{-1} sodium, have an osmolality of around 240 mOsm.kg^{-1} and a glucose concentration of 70–150 mmol.L^{-1}.[13] The addition of glucose facilitates water absorption from the gut, and in non-diabetics causes no significant rise in plasma glucose. Some fluids (e.g., Gatorade® in the USA) are close to ideal but most commercially available fluids, such as juices or carbonated beverages, are far from ideal. However, in the absence of the ideal fluid, water or other beverages can be used. Protein-containing fluids such as milk are not recommended because they delay absorption of fluid from the gut. Beverages containing caffeine or alcohol are not recommended because of their tendency to promote diuresis.

Figure 6
^{133}Xe washout half times from the anterior tibial muscle in human volunteers. Graphs drawn from the data published by Balldin & Lundgren[10]



Anti-platelet agents and deep vein thrombosis (DVT) prophylaxis

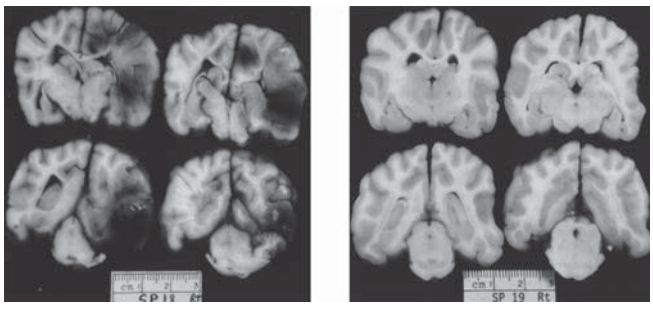
Evidence that bubbles in divers may activate platelets has suggested the use of aspirin and dextran to inhibit clot formation in symptomatic divers after decompression.[14–17] Retrospective anecdotal evidence supporting the use of aspirin, oxygen, corticosteroids and dextran was first published by Fructus.[18] To test this hypothesis, Mike Bennett and colleagues performed a randomized double blind controlled study of the non-steroidal anti-inflammatory drug (NSAID) tenoxicam (a non-selective cyclooxygenase inhibitor) versus placebo. The results of the trial indicated that although there was no long-term outcome difference in divers who received tenoxicam, the number of recompressions required to reach a therapeutic plateau was reduced by one (two treatments versus three treatments in the placebo group).[19]

Leg immobilization due to spinal cord DCS is likely to promote deep vein thrombosis and thromboembolism.[20] Therefore, while routine full anticoagulation is not indicated, prophylactic measures such as low molecular weight heparin are recommended when significant leg weakness is present.[21]

Corticosteroids

In addition to their anti-inflammatory effects and ability to reduce brain volume, corticosteroids may have antioxidant properties. However, several studies of corticosteroids in animal models of decompression illness have revealed no advantage in short term outcome.[22–25] Nevertheless, some human outcome studies have suggested that a loading dose of methylprednisolone 30 $mg.kg^{-1}$ with a 23-hour infusion at 5.4 $mg.kg^{-1}$ per hour has resulted in improved long-term outcome in humans with spinal cord trauma.[26] Unfortunately, the outcome scales used in this study and others performed by the same group are difficult to interpret, and the improvements in outcome may be marginal.[27] A more recent trial did not find a benefit for methylprednisolone in acute spinal cord injury but did report more infections when it was used.[28] The challenges of performing outcome studies in acute spinal cord injury have recently been discussed.[29] A review of the use of high-dose methylprednisolone in DCI has resulted in a recommendation that corticosteroids not be used in DCI.[21]

Figure 7
Brain slices from dogs in which injury was induced by inflation of an epidural balloon; left – brain of a normothermic dog; right – brain of a dog cooled to a core temperature of 31°C (see text for details); from Pomeranz, with permission[38]



Lignocaine (lidocaine)

Lignocaine (lidocaine) has been examined in various forms of DCS, particularly CAGE, in animals and found to have beneficial effects.[30,31] Case reports in which administration of lignocaine appeared to be beneficial for DCS and gas embolism and systematic reviews provide support for its administration to patients.[30–33] Randomized outcome studies in diving-related CAGE are virtually impossible to perform because of the rarity of the condition. However, open-heart surgery may be a reasonable surrogate, as arterial bubbles are commonly observed during these procedures. A randomized study of lignocaine in open-heart surgery was associated with a significant improvement in long-term outcome.[34] This has been followed by two other studies demonstrating improvement in outcome with lignocaine after cardiopulmonary bypass.[35,36] Lignocaine is a relatively benign drug when given intramuscularly to provide a short term effect or when administered via infusion when it can be monitored. It therefore remains an optional therapy that may provide some benefit, particularly in CAGE.

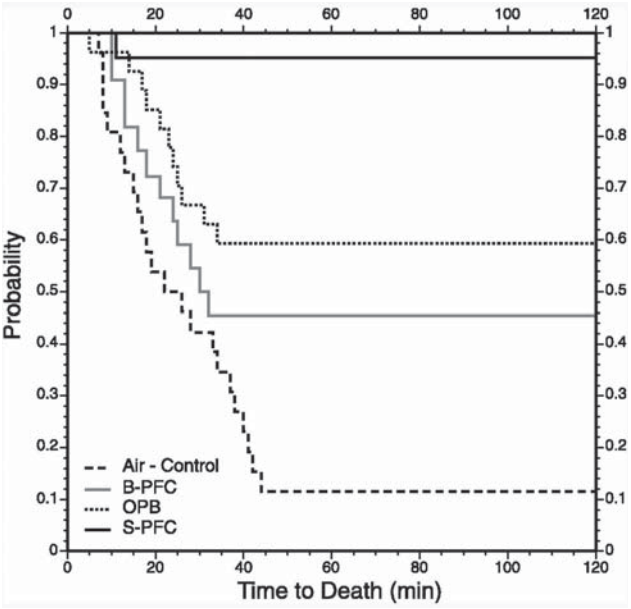
Body temperature management

Induced hypothermia has been observed to be beneficial in numerous animal models of brain injury.[37] Figure 7 shows brain slices from dogs in which injury was induced by inflation of an epidural balloon to maintain an intraventricular pressure of 62 mmHg for 90 minutes. On the left is the brain of a normothermic dog who developed brain death. On the right is the brain of a dog in which cooling was instituted 15 minutes after balloon inflation. A core temperature of 31°C was maintained for 5 hours, after which a core temperature of 35°C was maintained until 7–62 hours post insult; standard resuscitative maneouvres and anesthesia were maintained throughout.[38] Conversely hyperthermia is detrimental. Two hypothermia studies have demonstrated improved outcome after global ischaemia during cardiac arrest.[39,40] Although there is no evidence as yet that hypothermia is useful for other forms of brain injury,[41] there is evidence in humans that hyperthermia is detrimental.[42,43] Therefore, although hypothermia has not been embraced as a therapy for neurological DCS it is strongly recommended that fever in the setting of DCI be aggressively treated.[21]

Manipulation of arterial PCO_2

Hyperventilation and the resulting hypocapnia generally cause cerebral vasoconstriction and a reduction in intracranial pressure. CAGE can cause brain swelling and

Figure 8
Probability of death in pigs after decompression from 5 atmospheres in a dry hyperbaric chamber for 22 hours. Animals were randomized to one of four groups: air and saline (Air-Control); oxygen pre-breathe and saline (OPB); OPB with intravenous PFC given at depth (B-PFC); OPB with PFC given after surfacing (S-PFC).
Reproduced from Dainer[49], with permission



increased intracranial pressure,[44] thus, it has often been standard practice to induce hyperventilation following severe CAGE in an attempt to maintain cerebral perfusion and oxygenation. However, studies by Van Hulst have failed to show any evidence of improved cerebral perfusion pressure as a result of hyperventilation.[45] Moreover, brain glucose is lower and brain lactate is high in hyperventilated animals.[46] These studies support maintaining arterial PCO_2 within the normal range in patients with CAGE.

Perfluorocarbons

A new modality for treatment of DCS consists of perfluorocarbon (PFC) emulsion. These substances have a high solubility for both oxygen and inert gases. Administration of PFC emulsions in animal models of gas embolism or DCS have demonstrated improvement.[47–50] In particular, administration of PFC after decompression from air saturation in pigs has resulted in reduced DCS and mortality (Figure 8).[48,49] Animals were randomized to one of four groups: air and saline; O_2 pre-breathed (OPB) for 9 minutes before decompression at an inspired concentration >90% and saline; OPB with intravenous PFC (Oxygent, Alliance Pharmaceutical, San Diego, CA) 6 $mL.kg^{-1}$ given at depth; OPB with PFC given after surfacing. OPB combined with PFC administered after surfacing was associated with the greatest reduction in death rate due to DCI. While these substances are not yet commercially available and have never been tested in settings other than saturation-related DCS in animals, they hold great promise for use in acute DCS and CAGE. One caveat is that after administered PFC emulsions brain PO_2 may be increased, and hence CNS oxygen toxicity more likely during subsequent hyperbaric oxygen treatment. Human and animal studies are planned to address this issue.

Summary

In summary, administration of intravenous or oral fluid to maintain intravascular volume, blood pressure and urine output are recommended in addition to surface oxygen for all cases of decompression illness. NSAIDs, aspirin and lignocaine remain options. DVT prophylaxis in the form of low molecular weight heparin is recommended for patients in whom there is reduced mobility. $PaCO_2$ should be maintained within normal limits when treating critically ill patients with CAGE. Perfluorocarbons may be available within a year or two and may have a significant impact on outcome, particularly when recompression is not immediately available.

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