RECOMPRESSION TREATMENTS SHOULD BE TO A PRESSURE EQUIVALENT TO 18 m DEPTH

Richard E Moon

Key Words
Bubbles, decompression illness, hyperbaric oxygen, treatment

Bubble volume reduction

One therapeutic goal of recompression treatment of decompression illness (DCI) is a reduction of bubble volume. The higher the pressure the smaller will be the bubble volume, but high pressure is accompanied by practical problems of increased complexity and a higher probability of a treatment complication. Complications may include inert gas narcosis, DCI during the decompression phase or oxygen toxicity. Ultimately, the maximum treatment depth is governed by the chamber design. Thus, the choice of an initial treatment pressure must be a trade off between the opposing goals of maximising bubble compression and minimising the risk of treatment. Clinical experience has shown that recompression to a pressure equivalent to 18 m breathing 100% O_2 or to 50 m breathing 20-50% O_2 are safe.

Clinical observation

Until the 1940s a hodge-podge of empirical treatment tables were used. Recompression pressures were initially based arbitrarily upon either the depth of the dive or the pressure at which relief of symptoms occurred. Yarbrough and Behnke observed that in human cases, symptoms usually resolved at treatment pressures of 30 psig (2.04 atmospheres gauge, 20 m, 67.5 ft of sea water or 3 bar) or less, but that this treatment pressure was insufficient to prevent neurological damage in experimental animals.[1] They suggested using a short period of recompression to 165 ft (50 m, 6 bar) followed by the administration of 100% O_2 at 60 ft (18 m, 2.8 bar) and observed that this protocol effected complete relief of symptoms in 49 of 50 divers with bends.[1]

The use of O_2 in the treatment of bends was systematised within the US Navy in the 1960s. The method of administration of O_2 recompression was detailed in three reports.[2-4] Initially, it had been suggested that 100% O_2 should be administered at 33 ft (10 m, 2 bar), with further compression to 60 ft (18 m, 2.8 bar) if relief of symptoms did not occur within 10 minutes. However, based upon initial results, a prescribed trial of therapy at 10 m (2 bar) was abandoned in favour of immediate recompression to 60 ft (18 m, 2.8 bar).[2] The new O_2 tables produced a high rate of success. In his 1965 report, Goodman reported that treatment of bends using 100% O_2 at 60 ft (18 m, 2.8 bar) resolved 72 of 79 cases of DCS. Of those cases receiving a minimum of 30 minutes of oxygen breathing at 60 ft (18 m, 2.8 bar), for a minimum treatment time of 90 minutes, symptoms were relieved in 49 of 50 cases.[2] This led to adoption by the US Navy of Tables 5 and 6. These treatment tables, sometimes with minor variations, remain the standard in most hyperbaric facilities today.[5] Since their design and implementation there has been a large experience and a high degree of clinical success (see Table 1 in Moon[6]).

On the basis of historical experience, for the initial treatment of DCI almost all clinicians now use an initial compression to 18 m (2.8 bar) or deeper. To date, comparative studies of 18 m (2.8 bar) vs shallower initial compression have not been published. The only published data compared 14 m (2.4 bar) and 18 m (2.8 bar) for follow up treatment after initial compression to 18 m (2.8 bar). In this retrospective review, Wilson and colleagues from Melbourne, reported that of 50 divers who received 18 m (2.8 bar) follow up tables, 8 (16%) relapsed, compared with 6 of 15 (40%) whose follow up treatment was at 14 m (2.4 bar) ($P = 0.03$).[7]

Animal studies

Direct observation of intravascular bubbles during recompression therapy for cerebral arterial gas embolism was reported in 1967 by Waite, who observed bubbles in the cerebral circulation via a cranial window, following intracarotid injection of 1-7 ml of air in anaesthetised dogs. During recompression to 165 ft (50 m, 6 bar) he observed that of 6 animals, one had resolution of air at 60 ft (18 m, 2.8 bar), three had resolution at 80 ft (24 m, 3.4 bar) and two had resolution at 100 ft (30 m, 4 bar).[8] Gorman and colleagues observed that after injection of small volumes of air, if pial bubbles did not redistribute spontaneously, they could remain visible even after recompression to 11 bar (100 m).[9] While compression to 11 bar (100 m) or greater is usually impractical these particular data do not support the use of shallow recompression depths.

Measures other than bubble volume may be more appropriate, and to that end a series of experiments were performed in the 1980s at the US Naval Medical Research Institute on anaesthetised dogs with decompression sickness, using somatosensory evoked potential amplitude as the end point of treatment. In one study, the effect of PO_2 on outcome at 120 minutes after treatment was tested by recompressing dogs to 4 atmospheres gauge (5 bar) while breathing either one of a range of gas mixtures (see Fig. 1).[10] Using this short-term end point, the optimum PO_2 appeared to be between 2 and 3 bar (10-18 m).[10] A follow up comparison of two therapeutic PO_2 values, but at different depths, 60 ft (18 m, 2.8 bar) on 100%

oxygen and 66 ft (20 m, 3 bar) on 66% oxygen, failed to find a short term difference in outcome between 2.0 and 2.8 bar (10-18 m),[11] supporting the use of an initial recompression depth of 10-20 m.

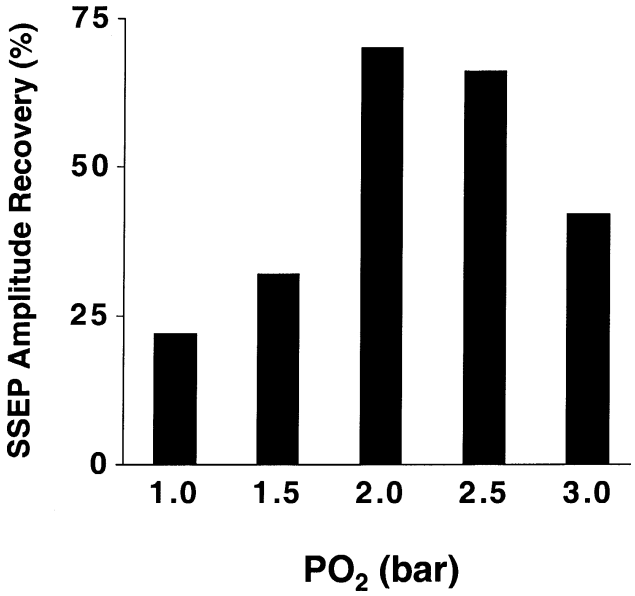

Figure 1. Recovery of somatosensory evoked potential amplitude after treatment of spinal cord decompression sickness in anaesthetised dogs. Therapy was administered at 5 bar, beginning 15 minutes after the onset of impaired neural conduction. The end point was 120 minutes after recompression. The optimum treatment PO_2 according to this model is between 2 and 3 bar. Data from Leitch and Hallenbeck.[10]

Other effects of bubbles

Bubbles have other effects besides mechanical obstruction or distortion. Most theoretical arguments regarding the appropriate pressure and gas composition to use for the treatment of gas bubble disease rest upon analysis of factors that augment gas volume reduction and absorption. This is based upon the thesis that bubbles cause tissue damage by their physical presence, either by occluding blood vessels and inducing ischaemia or, when they occur within the substance of tissue (autochthonous bubbles), by compression and distortion. A powerful argument in favour of this concept is the success of hyperbaric oxygen (HBO_2) in treating these conditions. Evidence has emerged within the last 20 years that bubbles can cause damage via a third mechanism.

Steve Helps and Des Gorman, at the University of Adelaide, embolised anaesthetised rabbits and examined the fate of injected air (25-400 microlitres, compared with 1-7 ml injected in Waite's study[8]) through a cranial window. Surprisingly, the bubbles did not permanently occlude vessels, and usually remained visible for only a few minutes. Despite re-establishment of flow after the bubbles had moved distally, brain blood flow progressively decreased.[12,13] The passage of bubbles appeared to have caused a change in vascular physiology. Later experiments by the same investigators implicated neutrophils, as the blood flow reduction did not occur in animals made neutropenic before the experiment.[14]

These observations and insights provided by experiments on a model of myocutaneous flap ischaemia suggest an additional mechanism for the effect of HBO in gas bubble disease.[15-17] In these studies, blood flow after reperfusion was greater in animals treated with HBO_2 even when it was administered during total ischaemia, when blood flow to the flap was zero. Neutrophils have also been implicated in this process as, in control animals, neutrophils were observed to adhere to the endothelium in the microcirculation of the previously ischaemic flap, but not in animals treated with HBO_2. It has been hypothesised that these neutrophils may cause a reduction in blood flow either by mechanical obstruction to blood flow or by releasing mediators. In the flap model, HBO_2 appeared to inhibit neutrophil-endothelial adherence.

Whether neutrophil-endothelial interaction is important in human DCI is not known. However, studies

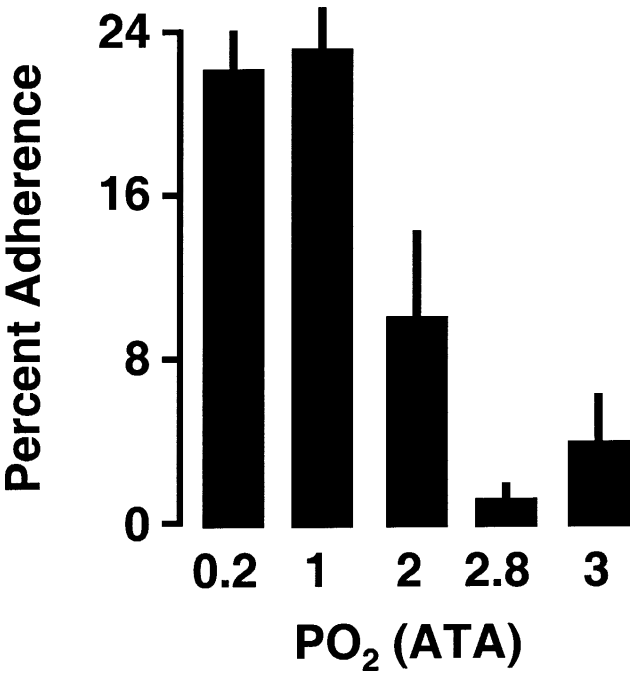

Figure 2. Human neutrophil adherence to nylon columns after 45 minute exposure to different partial pressures of oxygen ranging from 0.2 to 3 atmospheres absolute (ATA) (redrawn from Thom,[18] with permission). Maximum depression of neutrophil adhesion appears to occur at 2.8 ATA (18 breathing 100% O_2).

by Steve Thom in Philadelphia have demonstrated that after volunteers are exposed to elevated PO_2, their neutrophils are less adherent to nylon columns (see Fig. 2).[18] In those studies, maximum depression of neutrophil adhesion occurred at 2.8 ATA (18 m equivalent depth breathing 100% O_2).

The evidence linking inhibition of neutrophil adherence and clinical effectiveness of hyperbaric oxygen in DCI is at best indirect, but what information there is supports the use of 18 m tables.

Summary

It is conceivable that, under some circumstances, shallower recompression depths, or even surface treatment, may achieve a similar degree of success. But, because 18 m recompression is so successful, the burden of proof remains on the side of individuals suggesting a change in therapy. I conclude that the weight of clinical experience and some new insights into pathophysiology suggest that 18 m should remain the preferred depth for treatment of divers until proven otherwise.

AUDIENCE PARTICIPATION

David Doolette, Adelaide

In your last slide, the neutrophils were from normal volunteers. Have there been any studies from neutrophils that have been irritated with air bubbles beforehand?

Richard Moon

Not that I know of.

References

1 Yarbrough OD and Behnke AR. The treatment of compressed air illness using oxygen. *J Ind Hyg Toxicol* 1939; 21: 213-218

2 Goodman MW and Workman RD. *Minimal recompression oxygen-breathing approach to treatment of decompression sickness in divers and aviators. US Navy Experimental Diving Unit Report #5-65*. Washington, DC: US Navy, 1965

3 Bornmann RC. *Experience with Minimal Recompression, Oxygen Breathing Treatment of Decompression Sickness and Air Embolism*. Washington, DC: US Navy Experimental Diving Unit, Washington Navy Yard, 1967

4 Workman RD. Treatment of bends with oxygen at high pressure. *Aerosp Med* 1968; 39: 1076-1083

5 Moon RE and Sheffield PJ. Guidelines for treatment of decompression illness. *Aviat Space Environ Med* 1997; 68: 234-243

6 Moon RE. All divers with decompression illness require recompression. *SPUMS J* 2000; 30 (3): 149-151

7 Wilson M, Scheinkestel CD and Tuxen DV. Comparison of 14 and 18 metre tables on the resolution of decompression sickness (DCS) in divers. *Undersea Biomed Res* 1989; 16 (Suppl): 87-88

8 Waite CL, Mazzone WF, Greenwood ME and Larsen RT. *Cerebral air embolism I. Basic studies. US Naval Submarine Medical Center Report No. 49*. Panama City, Florida: US Navy Submarine Research Laboratory, 1967

9 Gorman DF, Browning DM and Parsons DW. Redistribution of cerebral arterial gas emboli: a comparison of treatment regimens. In *Underwater and Hyperbaric Physiology IX. Proceedings of the Ninth International Symposium on Underwater and Hyperbaric Physiology.* Bove AA, Bachrach AJ and Greenbaum LJ Jr. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987: 1031-1050

10 Leitch DR and Hallenbeck JA. Oxygen in the treatment of spinal cord decompression sickness. *Undersea Biomed Res* 1985; 12: 269-289

11 Sykes JJW, Hallenbeck JM and Leitch DR. Spinal cord decompression sickness: a comparison of recompression therapies in an animal model. *Aviat Space Environ Med* 1986; 57: 561-568

12 Helps SC, Parsons DW, Reilly PL and Gorman DF. The effect of gas emboli on rabbit cerebral blood flow. *Stroke* 1990; 21: 94-99

13 Helps SC, Meyer-Witting M, Reilley PL and Gorman DF. Increasing doses of intracarotid air and cerebral blood flow in rabbits. *Stroke* 1990; 21: 1340-1345

14 Helps SC and Gorman DF. Air embolism of the brain in rabbits pre-treated with mechlorethamine. *Stroke* 1991; 22: 351-354

15 Zamboni WA, Roth AC, Russell RC, Nemiroff PM, Casas L and Smoot EC. The effect of acute hyperbaric oxygen therapy on axial pattern skin flap survival when administered during and after total ischemia. *J Reconstr Microsurg* 1989; 5: 343-347

16 Zamboni WA, Roth AC, Russell RC and Smoot EC. The effect of hyperbaric oxygen on reperfusion of ischemic axial skin flaps: a laser Doppler analysis. *Ann Plast Surg* 1992; 28: 339-341

17 Zamboni WA, Roth AC, Russell RC, Graham B, Suchy H and Kucan JO. Morphological analysis of the microcirculation during reperfusion of ischemic skeletal muscle and the effect of hyperbaric oxygen. *Plast Reconstr Surg* 1993; 91: 1110-1123

18 Thom SR, Mendiguren I, Hardy K et al. Inhibition of human neutrophil beta2-integrin-dependent adherence by hyperbaric O_2 *Am J Physiol* 1997; 272: C770-C777