

## **5. DECOMPRESSION SICKNESS**

*Richard E. Moon*

### **Rationale**

Decompression sickness (“bends”, DCS) arises from the generation of bubbles of inert gas in the tissues and/or blood in volumes sufficient to interfere with organ function (1-5), caused by rapid decompression during ascent from diving, flying, or a hyperbaric/hypobaric chamber. Bubble formation occurs when the speed of decompression exceeds the rate at which diffusion and perfusion reduce the tissue inert gas partial pressure. The resulting clinical manifestations include joint pains (limb bends), cutaneous eruptions or rashes (skin bends), neurological dysfunction (peripheral or central nervous system bends), cardiorespiratory symptoms and pulmonary edema (chokes), shock and death (6). Several mechanisms have been hypothesized by which bubbles may exert their deleterious effects. These include direct mechanical disruption of tissue, occlusion of blood flow, platelet deposition and activation of the coagulation cascade (7), endothelial dysfunction (8, 9) and capillary leakage (10-12), complement activation (13, 14) and leukocyte-endothelial interaction (15).

The diagnosis of DCS is made on the basis of signs and/or symptoms after a dive or altitude exposure (16). Manifestations most commonly include paresthesias, hypesthesia, joint pain, skin rash and malaise. More serious signs and symptoms include motor weakness, ataxia, dyspnea, urethral and anal sphincter dysfunction, shock and death (6, 16, 17).

Chest radiography prior to HBO<sub>2</sub> treatment in selected cases may be useful to exclude pneumothorax (which may require tube thoracostomy placement before recompression) and to exclude causes unrelated to diving for which treatment other than HBO<sub>2</sub> would be appropriate (e.g. herniated disc). However, imaging studies are generally not helpful (18, 19), and should not be relied upon to confirm the diagnosis of DCS or be used in deciding whether a patient with suspected DCS needs HBO<sub>2</sub>.

In addition to general supportive measures, including fluid resuscitation, airway protection, and blood pressure maintenance, the definitive treatment of decompression sickness is compression to suitable pressures greater than sea level. Improvement of decompression sickness symptoms as a result of compression was first noted in the Nineteenth Century (20). Recompression was first reported as a specific treatment for that purpose in 1896 (21). Oxygen breathing was observed to improve the signs of decompression sickness in animals (22). The use of oxygen with pressure to accelerate gas diffusion and bubble resolution in humans was first suggested in 1897 (23) and eventually tested in human DCS and recommended for the treatment of divers (24). The rationale for treatment with hyperbaric oxygen (HBO<sub>2</sub>) includes immediate reduction in bubble volume, increasing the diffusion gradient for inert gas from the bubble into the surrounding tissue, oxygenation of ischemic tissue and reduction of CNS edema. It is also likely that HBO<sub>2</sub> has other beneficial pharmacological effects, such as a reduction in neutrophil adhesion to the capillary endothelium (25, 26). The efficacy of administration of oxygen at increased ambient pressure (hyperbaric oxygen, HBO<sub>2</sub>) is widely accepted, and HBO<sub>2</sub> is the mainstay of treatment for this disease (27-30).

A wide variety of initial hyperbaric regimens have been described, differing in treatment pressure and time, partial pressure of oxygen and diluent gas. Although there are no human outcome data obtained in prospective, randomized studies for the treatment of diving related decompression sickness, broad principles that are generally agreed upon (26) include: (a) complete resolution is most likely to result from early hyperbaric treatment (17); (b) the US

Navy oxygen treatment tables (31) (and the similar RN and Comex tables), with initial recompression to 60 fsw (18 msw, 2.82 atm abs) have been the most widely used recompression procedures for decompression illness (DCI) treatment beginning at the surface, and have achieved a high degree of success in resolving symptoms if the delay to treatment is not excessive (17, 28, 32, 33).

Monoplace chambers were originally designed for the continuous administration of 100% oxygen and were not equipped to administer air for “air breaks”. For monoplace chambers of this type, tables are available for treatment of decompression sickness that are shorter than standard USN treatment tables (34-36). Retrospective evidence, using telephone follow up, suggests that such tables may be as effective as standard USN tables for the treatment of mildly or moderately affected patients (27, 37). However, many monoplace chambers are now fitted with the means to deliver air to the patient, and thus can be used to administer standard USN treatment tables.

For the vast majority of cases of DCS, superiority of treatments at pressure exceeding 2.82 atm abs or using helium as the diluent gas or using saturation treatments, has not been demonstrated. The use of treatment schedules that deviate from the US Navy oxygen treatment tables or published monoplace tables are best reserved for facilities and personnel with the experience, expertise and hardware necessary to deal with untoward responses.

While longer delays to treatment tend to be associated with incomplete resolution of symptoms, the data currently available have not established a maximum time (hours or days) after which recompression is ineffective (38-44).

The vast majority of cases respond satisfactorily to a single hyperbaric treatment. For patients with residual defects following the initial recompression, repetitive treatments are recommended until clinical stability has been achieved. HBO<sub>2</sub> should be administered repetitively as long as step-wise improvement occurs, based upon clearly documented symptoms and physical findings. Complete resolution of symptoms or lack of improvement on two consecutive treatments establishes the end-point of treatment. Although a small minority of divers with severe neurological injury may not reach a clinical plateau until 15-20 repetitive treatments have been administered, formal statistical analysis of approximately 3,000 DCI cases supports the efficacy of no more than 5-10 repetitive treatments for most individuals (45). In a group of 414 recreational diving accidents the median number of hyperbaric treatments was two, and only 6% of divers received more than 5 treatments (17).

Administration of 100% oxygen at ground level (1 atm abs) is recommended as first aid for all cases of DCS, and can be definitive treatment for altitude-induced DCS (46, 47). For definitive treatment of altitude-induced cases that do not respond to ground level O<sub>2</sub>, and for DCS after diving, HBO<sub>2</sub> remains the standard of care (30, 31, 48).

Adjunctive treatment such as first-aid oxygen administration, fluid resuscitation and, for patients with leg immobility, venous thromboembolism prophylaxis are indicated. These are discussed in detail in a separate monograph (49). A summary of current recommendations for adjunctive therapy is available on the Undersea and Hyperbaric Society website (<http://www.uhms.org>).

### **Guidelines for Use of HBO<sub>2</sub> in Decompression Sickness**

The use of HBO<sub>2</sub> for decompression sickness should be considered an AHA level I recommendation in spite of the absence of type I evidence (randomized controlled trials). Hyperbaric oxygen is the definitive treatment for this entity and has a history of many years of

effective and safe application. No other definitive treatments exist. All other treatments are adjunctive to hyperbaric oxygen.

### **Utilization Review**

The choice of treatment table and the number of treatments required will depend upon: (1) the clinical severity of the illness; (2) the clinical response to treatment; and (3) residual symptoms after the initial recompression. Depending on the patient's initial response, there may be repetitive treatments. Patients should be treated until clinical examination reveals no further improvement in response to the HBO<sub>2</sub> treatments. The need for follow up (“tailing” treatments) should be supported by documentation of the clinical evaluation before and after each treatment. Utilization review should occur after 10 treatments.

### **Cost Impact**

Only those people exposed to increased ambient pressure (divers or compressed air workers) or who suffer decompression sickness at altitude are affected. Therefore, the application of HBO<sub>2</sub> will be limited because there are relatively few individuals who develop this condition. HBO<sub>2</sub> is a treatment that usually provides resolution or significant improvement of this disorder that can otherwise result in permanent spinal cord, brain or peripheral nerve damage or death. It is therefore an exceptionally cost effective treatment.

### **REFERENCES**

1. Harvey EN. Decompression sickness and bubble formation in blood and tissues. *Bull N Y Acad Med* 1945;21:505-36.
2. Haymaker W. Decompression sickness. In: Lubarsch O, Henke F, Rösste R, Eds. *Handbuch der Speziellen Pathologischen Anatomie und Histologie. Erkrankungen des Zentralen Nervensystems I*. Berlin: Springer-Verlag; 1957. pp. 1600-72.
3. Clay JR. Histopathology of experimental decompression sickness. *Aerosp Med* 1963;34:1107-10.
4. Francis TJ, Griffin JL, Homer LD, Pezeshkpour GH, Dutka AJ, Flynn ET. Bubble-induced dysfunction in acute spinal cord decompression sickness. *J Appl Physiol* 1990;68:1368-75.
5. Francis TJR, Mitchell SJ. Pathophysiology of decompression sickness. In: Brubakk AO, Neuman TS, Eds. *Physiology and Medicine of Diving*. New York, NY: Elsevier Science; 2003, pp. 530-56.
6. Elliott DH, Moon RE. Manifestations of the decompression disorders. In: Bennett PB, Elliott DH, Eds. *The Physiology and Medicine of Diving*. Philadelphia, PA: WB Saunders; 1993. pp. 481-505.
7. Philp RB, Schacham P, Gowdey CW. Involvement of platelets and microthrombi in experimental decompression sickness: similarities with disseminated intravascular coagulation. *Aerosp Med* 1971;42:494-502.
8. Nossum V, Hjelde A, Brubakk AO. Small amounts of venous gas embolism cause delayed impairment of endothelial function and increase polymorphonuclear neutrophil infiltration. *Eur J Appl Physiol* 2002;86:209-14.
9. Nossum V, Koteng S, Brubakk AO. Endothelial damage by bubbles in the pulmonary artery of the pig. *Undersea Hyperbaric Med* 1999;26:1-8.
10. Brunner F, Frick P, Bühlmann A. Post-decompression shock due to extravasation of plasma. *Lancet* 1964;1:1071-3.
11. Levin LL, Stewart GJ, Lynch PR, Bove AA. Blood and blood vessel wall changes induced by decompression sickness in dogs. *J Appl Physiol* 1981;50:944-9.
12. Boussuges A, Blanc P, Molenat F, Bergmann E, Sainty JM. Haemoconcentration in neurological decompression illness. *Int J Sports Med* 1996;17:351-5.
13. Ward CA, Koheil A, McCullough D, Johnson WR, Fraser WD. Activation of complement at plasma-air or serum-air interface of rabbits. *J Appl Physiol* 1986;60:1651-8.
14. Ward CA, McCullough D, Yee D, Stanga D, Fraser WD. Complement activation involvement in decompression sickness of rabbits. *Undersea Biomed Res* 1990;17:51-66.

15. Helps SC, Gorman DF. Air embolism of the brain in rabbits pre-treated with mechllorethamine. *Stroke* 1991;22:351-4.
16. Francis TJR, Mitchell SJ. Manifestations of decompression disorders. In: Brubakk AO, Neuman TS, Eds. *Physiology and Medicine of Diving*. New York, NY: Elsevier Science; 2003. pp. 578-99.
17. Divers Alert Network. Report on Decompression Illness. *Diving Fatalities and Project Dive Exploration*. Durham, NC: Divers Alert Network; 2003.
18. Warren LP, Djang WT, Moon RE, Camporesi EM, Sallee DS, Anthony DC. Neuroimaging of scuba diving injuries to the CNS. *AJNR* 1988;9:933-8.
19. Reuter M, Tetzlaff K, Hutzelmann A, Fritsch G, Steffens JC, Bettinghausen E, et al. MR imaging of the central nervous system in diving-related decompression illness. *Acta Radiol* 1997;38:940-4.
20. Pol B, Wattelle TJJ. Mémoire sur les effets de la compression de l'air appliquée au creusement des puits à houille. *Ann Hyg Pub Med Leg* 1854;2:241-79.
21. Moir EW. Tunnelling by compressed air. *J Soc Arts* 1896;44:567-85.
22. Bert P. *Barometric Pressure (La Pression Barométrique)*. Bethesda, MD: Undersea Medical Society; 1978.
23. Zuntz N. Zur Pathogenese und Therapie der durch rasche Luftdruckänderungen erzeugten Krankheiten. *Fortschr Med* 1897;15:632-9.
24. Yarbrough OD, Behnke AR. The treatment of compressed air illness using oxygen. *J Ind Hyg Toxicol* 1939;21:213-8.
25. Zamboni WA, Roth AC, Russell RC, Graham B, Suchy H, Kucan JO. Morphological analysis of the microcirculation during reperfusion of ischemic skeletal muscle and the effect of hyperbaric oxygen. *Plast Reconstr Surg* 1993;91:1110-23.
26. Martin JD, Thom SR. Vascular leukocyte sequestration in decompression sickness and prophylactic hyperbaric oxygen therapy in rats. *Aviat Space Environ Med* 2002;73:565-9.
27. Kindwall EP. Use of short *versus* long tables in the treatment of decompression sickness and arterial gas embolism. In: Moon RE, Sheffield PJ, Eds. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996. pp. 122-6.
28. Thalmann ED. Principles of US Navy recompression treatments for decompression sickness. In: Moon RE, Sheffield PJ, Eds. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996. pp. 75-95.
29. Moon RE, Sheffield PJ. Guidelines for treatment of decompression illness. *Aviat Space Environ Med* 1997;68:234-43.
30. Moon RE, Gorman DF. Treatment of the decompression disorders. In: Neuman TS, Brubakk AO, Eds. *The Physiology and Medicine of Diving*. New York, NY: Elsevier Science; 2003. pp. 600-50.
31. Navy Department. *US Navy Diving Manual*. Revision 4. Vol 5 : *Diving Medicine and Recompression Chamber Operations*. NAVSEA 0910-LP-708-8000. Washington, DC: Naval Sea Systems Command; 1999.
32. Ball R. Effect of severity, time to recompression with oxygen, and retreatment on outcome in forty-nine cases of spinal cord decompression sickness. *Undersea Hyperbaric Med* 1993;20:133-45.
33. Ross JAS. *Clinical Audit and Outcome Measures in the Treatment of Decompression Illness in Scotland*. A report to the National Health Service in Scotland Common Services Agency, National Services Division on the conduct and outcome of treatment for decompression illness in Scotland from 1991-1999. Aberdeen, UK: Department of Environmental and Occupational Medicine, University of Aberdeen Medical School; 2000 27 April 2000.
34. Kindwall EP. Decompression sickness. In: Davis JC, Hunt TK, Eds. *Hyperbaric Oxygen Therapy*. Bethesda, MD: Undersea Medical Society; 1977. pp. 125-40.
35. Hart GB, Strauss MB, Lennon PA. The treatment of decompression sickness and air embolism in a monoplace chamber. *J Hyperbaric Med* 1986;1:1-7.
36. Elliott DH, Kindwall EP. Decompression sickness. In: Kindwall EP, Whelan HT, Eds. *Hyperbaric Medicine Practice*. Flagstaff, AZ: Best; 1999. pp. 433-87.
37. Bond JG, Moon RE, Morris DL. Initial table treatment of decompression sickness and arterial gas embolism. *Aviat Space Environ Med* 1990;61:738-43.
38. Workman RD. Treatment of bends with oxygen at high pressure. *Aerosp Med* 1968;39:1076-83.
39. How J, Chan G. Management of delayed cases of decompression sickness--3 case reports. *Singapore Med J* 1973;14:582-5.
40. Erde A, Edmonds C. Decompression sickness: a clinical series. *J Occup Med* 1975;17:324-8.
41. Kizer KW. Delayed treatment of dysbarism: a retrospective review of 50 cases. *JAMA* 1982;247:2555-8.
42. Meyers RAM, Bray P. Delayed treatment of serious decompression sickness. *Ann Emerg Med* 1985;14:254-7.

43. Curley MD, Schwartz HJC, Zwingelberg KM. Neuropsychologic assessment of cerebral decompression sickness and gas embolism. *Undersea Biomed Res* 1988;15:223-36.
44. Rudge FW, Shafer MR. The effect of delay on treatment outcome in altitude-induced decompression sickness. *Aviat Space Environ Med* 1991;62:687-90.
45. Vann RD, Bute BP, Uguccioni DM, Smith LR. Prognostic factors in DCI in recreational divers. In: Moon RE, Sheffield PJ, Eds. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society: 1996. pp. 352-63.
46. Kimbrell PN. Treatment of altitude decompression sickness. In: Moon RE, Sheffield PJ, Eds. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society: 1996. pp. 43-52.
47. Krause KM, Pilmanis AA. The effectiveness of ground level oxygen treatment for altitude decompression sickness in human research subjects. *Aviation Space & Environmental Medicine*. 2000;71:115-8.
48. Moon RE. Treatment of decompression sickness and arterial gas embolism. In: Bove AA, Ed. *Bove and Davis' Diving Medicine*. Philadelphia: WB Saunders: 1997. pp. 184-204.
49. Moon RE, Ed. *Adjunctive Therapy for Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society: 2003.