

FROM THE HBO₂ INDICATIONS MANUAL, 14TH EDITION:

CHAPTER 7

Hyperbaric oxygen for decompression sickness: 2021 updateRichard E. Moon, MD¹; Simon J. Mitchell MD²¹Departments of Anesthesiology and Medicine, Center for Hyperbaric Medicine and Environmental Physiology, Duke University, North Carolina U.S.²Department of Anaesthesiology, University of Auckland, Auckland, New ZealandCORRESPONDING AUTHOR: Richard Moon – richard.moon@duke.edu**RATIONALE**

Decompression sickness (DCS, “bends”) is caused by the formation of bubbles in tissues and/or blood when the sum of dissolved gas pressures exceeds ambient pressure (supersaturation) [1]. This may occur when ambient pressure is reduced during: ascent from a dive; rapid ascent to altitude in an unpressurized aircraft or hypobaric chamber; loss of cabin pressure in an aircraft [2]; and during space walks. In diving, compressed-gas breathing is usually necessary, although occasionally DCS has occurred after either repetitive or very deep breath-hold dives [3,4]. DCS can rarely occur in hyperbaric tenders after decompression from standard hyperbaric treatment at 2.2-2.5 atmospheres absolute (ATA) [5]. Although arterial gas embolism due to pulmonary barotrauma can occur after a dive as shallow as 1 meter, the threshold depth for DCS in compressed-gas diving is around 20 feet of seawater (fsw) [6]. DCS after a dive can be provoked by mild altitude exposure, such as a commercial aircraft flight [7,8], but without a preceding dive the threshold altitude for DCS occurrence due to acute altitude exposure is generally 18,000-20,000 feet [9,10]. Most cases of altitude DCS manifest as limb pain [11-13]. More serious manifestations of altitude DCS (e.g., neurological, cardiorespiratory DCS: “chokes”) have been described but typically at altitudes greater than 25,000 feet for periods exceeding one hour [2,14-18]. When neurological symptoms occur, cerebral manifestations seem to occur more frequently than after diving [19]. Acute neurological manifestations after altitude exposure to 5,000-8,000 feet in commercial aircraft have rarely occurred due to arterial gas embolism caused by expansion of pre-existing bullae [20].

Several mechanisms have been hypothesized by which bubbles may exert their deleterious effects. These include direct mechanical disruption of tissue [21], occlusion of blood flow, platelet deposition and activation of the coagulation cascade [22], endothelial dysfunction [23, 24], capillary leakage [16,25-28], endothelial cell death, complement activation [29,30], inflammation [31] and leukocyte-endothelial interaction [32]. Recent evidence suggests that circulating microparticles may play a pro-inflammatory role in DCS pathophysiology [33,34].

The diagnosis of DCS is made on the basis of careful evaluation of the circumstances of the dive (or altitude exposure), the presence of known risk factors, and the post-dive latency and nature of the manifestations [19,35-37]. DCS manifestations most commonly include paresthesias, hypesthesia, musculoskeletal pain, skin rash and malaise [19,35-37]. Less common but more serious signs and symptoms include motor weakness, ataxia, vertigo, hearing loss, dyspnea, pulmonary edema [38], bladder and anal sphincter dysfunction, shock and death [19,35-37]. Severe DCS may be accompanied by hemoconcentration, hypotension, and coagulopathy [27, 39]. Severe symptoms usually occur within one to three hours of decompression; the vast majority of all symptoms manifest within 24 hours, unless there is an additional decompression (e.g., altitude exposure) [19].

Investigations have limited value in diagnosis of DCS. Chest radiography prior to hyperbaric oxygen (HBO₂) treatment in selected cases may be useful to exclude pneumothorax (which may require tube thoracostomy placement before recompression). If the clinical presentation is not typical of DCS or notably inconsistent with the circumstances of the dive, neural imaging is occa-

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sionally useful to exclude causes unrelated to diving for which treatment other than HBO₂ would be appropriate (e.g., herniated disc or spinal hemorrhage). However, imaging studies are rarely helpful for the evaluation or management of DCS [40,41]. Magnetic resonance imaging is not sufficiently sensitive to consistently detect early anatomic correlates of neurological DCI [42,43]. Bubbles causing limb pain cannot be detected radiographically. Neither imaging nor neurophysiological studies should be relied upon to confirm the diagnosis of DCS or be used in deciding whether a patient with suspected DCS needs HBO₂.

Improvement of decompression sickness symptoms as a result of recompression was first noted in the 19th century [44]. Recompression with air was first implemented as a specific treatment for that purpose in 1896 [45]. Oxygen breathing was observed by Bert in 1878 to improve the signs of decompression sickness in animals [46]. The use of oxygen with pressure to accelerate gas diffusion and bubble resolution in humans was first suggested in 1897 [47] and eventually tested in the 1930s for human DCS and recommended for treatment of divers [48]. The rationale for treatment with HBO₂ 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 central nervous system edema. It is also likely that HBO₂ has other beneficial pharmacological effects such as a reduction in neutrophil adhesion to the capillary endothelium [49,50]. The efficacy of HBO₂ is now widely accepted, and HBO₂ is the mainstay of treatment for this disease [19,51-56].

PATIENT SELECTION CRITERIA

Treatment is recommended for patients with a history of a decompression and whose manifestations are consistent with DCS. HBO₂ treatment is recommended for all patients with symptoms of DCS whenever feasible, although normobaric oxygen administration may be sufficient for the treatment of altitude DCS when neurological manifestations are absent, and for mild DCS (as defined below) following diving. For definitive treatment of altitude-induced cases that do not respond to ground-level oxygen, and for more serious cases of DCS after diving, HBO₂ remains the standard of care [53,54,57,58].

At a consensus workshop on remote treatment of mild DCS (limb pain, constitutional symptoms, subjective sensory symptoms or rash, with non-progressive symptoms, clinical stability for 24 hours or more

and a normal neurological exam), it was concluded that some patients with mild symptoms and signs after diving can be treated adequately without recompression [59]. Thus, although HBO₂ remains the preferred intervention in all cases of DCS, not least because DCS may recover more slowly without recompression [59], it is acceptable to treat cases fitting the mild classification with first aid measures (see below) alone if access to HBO₂ is logistically difficult or hazardous. Such decisions should be made on a case by case basis only and must always involve a diving medicine physician [60].

CLINICAL MANAGEMENT

First Aid. In addition to general supportive measures, including fluid resuscitation, airway protection, and blood pressure maintenance, administration of 100% oxygen at ground level (1 ATA) is recommended as first aid for all cases of DCS. Normobaric oxygen can be definitive treatment for altitude-induced DCS [51,52].

The following consensus guidelines for pre-hospital care have been developed by a group of international physicians organized by the Divers Alert Network [60].

- Normobaric oxygen (surface oxygen administered as close to 100% as possible) is beneficial in the treatment of DCI. Normobaric oxygen should be administered as soon as possible after onset of symptoms.
- Training of divers in oxygen administration is highly recommended.
- A system capable of administering a high percentage of inspired oxygen (close to 100%) and an oxygen supply sufficient to cover the duration of the most plausible evacuation scenario is highly recommended for all diving activities. In situations where oxygen supplies are limited and where patient oxygenation may be compromised (such as when drowning and DCI coexist) consideration should be given to planning use of available oxygen to ensure that some oxygen supplementation can be maintained until further supplies can be obtained.
- A horizontal position is generally encouraged in early presenting DCI, and should be maintained during evacuation if practicable. The recovery position is recommended in unconscious patients. The useful duration of attention to positioning in DCI is unknown. The head-down (Trendelenburg) position is no longer recommended in management of DCI.

- Oral hydration is recommended but should be avoided if the patient is not fully conscious. Fluids should be non-carbonated, non-caffeinated, non-alcoholic, and ideally isotonic (drinking water is acceptable).
- If suitably qualified and skilled responders are present, particularly in severe cases, intravascular rehydration (intravenous or intraosseous access) with non-glucose-containing isotonic crystalloid is preferred. Intravenous glucose-containing solutions should not be given.
- Treatment with a non-steroidal anti-inflammatory drug (NSAID) is appropriate if there are no contraindications.
- Other agents such as corticosteroids, pentoxifylline, aspirin, lidocaine and nitroglycerine have been utilized by suitably qualified responders in early management of DCI but there is insufficient evidence to support or refute their application.
- Divers should be kept thermally comfortable (warm but not hyperthermic). Hyperthermia should be avoided, especially in cases with severe neurological signs and symptoms. For example, avoid exposure to the sun, unnecessary activity, or excess clothing.

Hyperbaric oxygen. Recommended treatment of DCS is administration of oxygen at suitable pressures greater than sea level (hyperbaric oxygen). The choice of treatment table and the number of treatments required will depend upon: (a) the clinical severity of the illness; (b) the clinical response to treatment; and (c) residual symptoms after the initial recompression. 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 include: (a) complete resolution is more likely to result from early hyperbaric treatment [19,53]; (b) the U.S. Navy oxygen treatment tables [58] (and the similar RN and Comex tables), with initial recompression to 60 fsw (18 msw, 2.82 ATA) have been the most widely used recompression procedures for DCS treatment beginning at the surface, and have achieved a high degree of success in resolving symptoms [19,52,55,56,61,62]. Treatment at shallower depths (e.g., 33 fsw, 10 msw, 2 atm abs) can be effective as well, although published case series suggest that the success rate may be lower at treatment depths less than 60 fsw [55,63].

Treatment depth exceeding 60 fsw (18 msw). For the vast majority of cases of DCS, superiority of treatments at pressure exceeding 2.82 ATA or using helium as the diluent gas has not been demonstrated [64]. The speculative use of treatment schedules that deviate from U.S. 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.

Number of treatments. Most cases of DCS respond satisfactorily to a single hyperbaric treatment, although repetitive treatments (typically once daily) may be required depending on the patient's initial response. For patients with residual deficits following the initial recompression, repetitive treatments are recommended until clinical stability has been achieved. HBO₂ should be administered repetitively as long as stepwise improvement occurs, based upon clearly documented symptoms and physical findings. The need for such follow-up "tailing" treatments should be supported by documentation of the clinical evaluation before and after each treatment. Complete resolution of symptoms or lack of improvement on two consecutive treatments establishes the endpoint of treatment, typically no more than one or two treatments [19]. The optimal choice of recompression table for repetitive treatments has not been established. It is generally agreed that for tailing treatments, repetitive long treatment tables (such as the U.S. Navy Treatment Table 6) [58] are not justified, and it is typical to utilize shorter treatments (such as the U.S. Navy Table 5) [58] or even wound treatment tables conducted at 2-2.4 ATA for this purpose. Although a small minority of divers with severe neurological injury may not reach 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 five to 10 repetitive treatments for most individuals [65].

Time from symptom onset to hyperbaric treatment. Available data do not convincingly demonstrate superior outcomes in "rapid" versus delayed treatment [62,66]. For example, in two published series, time to treatment greater than 2,456 or 4,855 hours was as effective as earlier treatment. However, most series in recreational diving lack cases with extremely short symptom-to-recompression latency as comparators. In contrast, there are data from military experimental diving that suggest

immediate recompression is extremely effective in controlling symptoms [52,67,68]. As a general principle, timely treatment is preferred. Currently available data have not established a maximum time (hours or days) after which recompression is ineffective [68-74].

Monoplace chamber treatment. Monoplace chambers were originally designed for the continuous administration of 100% oxygen and not equipped to administer air for “air breaks,” which are incorporated in U.S. Navy treatment tables for DCS. For monoplace chambers of this type, tables are available for treatment of decompression sickness that are shorter than standard USN treatment tables [75-77]. 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 [51,78,79]. However, many monoplace chambers are now fitted with the means to deliver air to the patient and thus can be used to administer standard 2.82 ATA USN treatment tables [80].

Saturation treatment. For severe DCS in which gradual but incomplete improvement occurs during hyperbaric treatment at 60 fsw, saturation treatment may be considered if the hyperbaric facility has the capability. There is, however, no convincing evidence that such interventions are associated with a better outcome than other approaches.

In-water recompression. In-water recompression (IWR) of injured divers has been proposed as an emergency treatment modality if evacuation of a symptomatic diver to a hyperbaric facility cannot be performed in a timely manner. The advantage of IWR is that it can be initiated within a very short time after symptom onset. IWR breathing air has been used by indigenous divers, with a high reported success rate, although clinical details are scant [81]. There is anecdotal evidence that IWR using oxygen is more effective [82]; however, a major risk is an oxygen convulsion resulting in fatal drowning. IWR using oxygen has been discussed in the literature [60,82,83] and is described in the U.S. Navy Diving Manual [58]. Typical IWR oxygen-breathing protocols recommend depths no greater than 30 fsw (USN) or shallower [82]. Recommendations include a requirement that the diver not use a regular scuba mouthpiece but rather a full face mask, surface-supplied helmet breathing ap-

paratus or regulator retention strap (“gag strap”) [84]. Other requirements include the need for a tender in the water and the symptomatic diver to be tethered [82]. IWR is not recommended or may cause harm in the setting of isolated hearing loss, vertigo, respiratory distress, airway compromise, altered consciousness, extreme anxiety, hypothermia and hemodynamic instability.

In the absence of a sufficiently detailed case series from which risks and benefits can be assessed, IWR is not presently endorsed by the UHMS but was cautiously endorsed in a recent expert consensus for use by properly trained and equipped divers [60]. It should not be attempted without the necessary equipment, training and a full understanding of the necessary procedures.

Altitude DCS. The following algorithm has been used effectively by the U.S. Air Force [53,85].

- Mild symptoms that clear on descent to ground level with normal neurological exam: 100% oxygen by tightly fitted mask for two hours minimum; aggressive oral hydration; observe 24 hours.
- Symptoms that persist after return to ground level or occur at ground level: 100% oxygen; aggressive hydration; hyperbaric treatment using (USN) Treatment Tables 5 or 6, as appropriate. For those individuals with symptoms consisting of limb pain only, which resolves during oxygen breathing while preparing for hyperbaric treatment, a 24-hour period of observation should be initiated; hyperbaric treatment may not be required.
- Severe manifestations of DCS (neurological, “chokes,” hypotension, or manifestations that progress in intensity despite oxygen therapy): continue 100% oxygen; administer intravenous hydration; initiate immediate hyperbaric therapy using USN Treatment Table 6. Recompression to 2 ATA (U.S. Air Force Table 8) has also been used effectively for altitude DCS [86].

Adjunctive therapy. Adjunctive treatments 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 [87] which is available on the Undersea and Hyperbaric Society website:

www.uhms.org/images/Publications/ADJUNCTIVE_THERAPY_FOR_DCI.pdf.

Table 1: Evidence-based review of adjunctive therapies for DCS
(from Moon [87] www.uhms.org/images/Publications/ADJUNCTIVE_THERAPY_FOR_DCI.pdf)

condition	surface O ₂ (I ATA)		intravenous fluid therapy		aspirin		NSAIDs		anti-coagulants*		cortico-steroids		lidocaine	
	Class	Level	Class	Level	Class	Level	Class	Level	Class	Level	Class	Level	Class	Level
AGE (no sig. inert gas load)	I	C	D5W [†] LR/crystalloid [‡] colloid ⁺	III C IIb	IIb	C	IIb	C	IIb	C	III	C	IIa	B
DCS: pain only/mild	I	C	D5W [†] LR/crystalloid [‡] colloid ⁺	III I I	IIb	C	IIb	B	III	C	III	C	III	C
DCS: neurological	I	C	D5W [†] LR/crystalloid [‡] colloid ⁺	III I I	IIb	C	IIb	B	IIb§	C	III	C	IIb	C
DCS: chokes (cardiorespiratory)	I	C	D5W [†] LR/crystalloid [‡] colloid ⁺	III IIb IIb	IIb	C	IIb	C	IIb	C	III	C	III	C

§ For decompression illness with leg immobility, low molecular weight heparin is recommended as soon as possible after injury (enoxaparin 30 mg or equivalent) subcutaneously every 12 hours.

† 5% dextrose in water.

‡ Lactated Ringer's solution, normal saline or other isotonic intravenous fluid not containing glucose.

+ Starch, gelatin or protein fraction with isotonic electrolyte concentration.

* Full dose heparin, warfarin, thrombin inhibitors, thrombolytics, IIb/IIIa antiplatelet agents.

EVIDENCE-BASED REVIEW

The use of HBO₂ for decompression sickness is an AHA level I recommendation (level of evidence C) (Table 1).

UTILIZATION REVIEW

Utilization review should occur after 10 treatments.

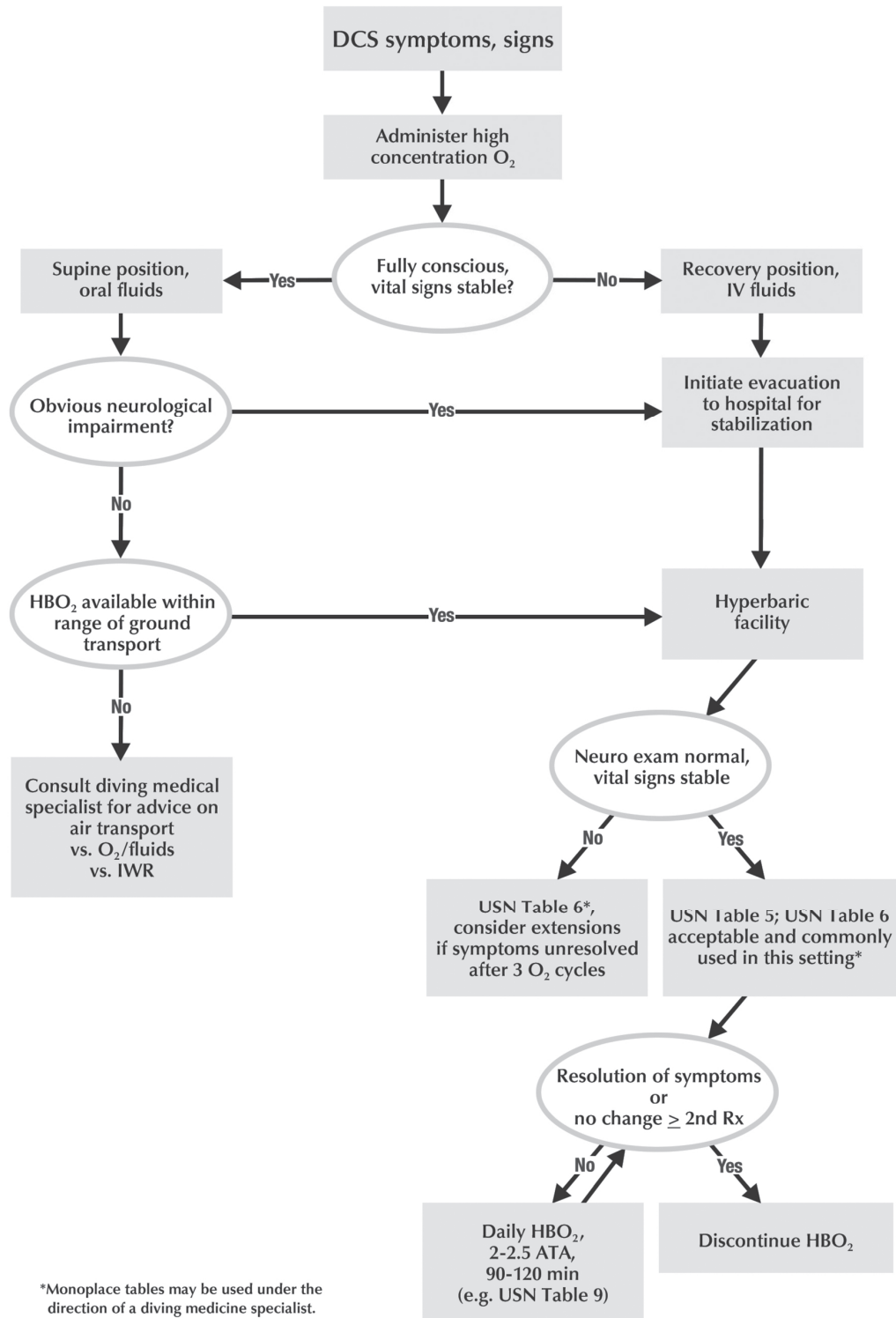
COST IMPACT

Only those people exposed to increased ambient pressure (divers or compressed-air workers) or who experience decompression sickness at altitude are affected. Because there are relatively few individuals who develop this condition, the use of HBO₂ will be limited. HBO₂ is a treatment that usually provides resolution or significant improvement of this disorder that can otherwise result in permanent damage to the spinal cord, brain or peripheral nerves or, in some cases, death, and is therefore an exceptionally cost-effective treatment. ■

Note:

Since its original publication (Moon RE, Mitchell S. Hyperbaric treatment for decompression sickness: current recommendations. Undersea Hyperb Med. 2019;46(5): 685-693) – which was based on the UHMS book Indications for Hyperbaric Oxygen Therapy, 14th ed. – this guideline has been updated with additional information on altitude decompression sickness and recently published studies.

Figure 1. Flowchart for DCS Symptoms
 Details of management are described in the text.



REFERENCES

1. Mitchell SJ. Decompression sickness: pathophysiology. In: Edmonds C, Bennett MH, editors. *Diving and Subaquatic Medicine*. 5th ed. Boca Raton, FL: Taylor and Francis; 2015. 125-140.
2. Hundemer GL, Jersey SL, Stuart RP, Butler WP, Pilmanis AA. Altitude decompression sickness incidence among U-2 pilots: 1994-2010. *Aviat Space Environ Med*. 2012;83(10):968-974.
3. Kohshi K, Wong RM, Abe H, Katoh T, et al. Neurological manifestations in Japanese Ama divers. *Undersea Hyperb Med*. 2005;32(1):11-20.
4. Schipke JD, Gams E, Kallweit O. Decompression sickness following breath-hold diving. *Res Sports Med*. 2006;14(3):163-178.
5. Kot J, Lenkiewicz E, Lizak E, Goralczyk P, Chreptowicz U. Spinal cord decompression sickness in an inside attendant after a standard hyperbaric oxygen treatment session. *Diving Hyperb Med*. 2021;51(1):103-106.
6. Van Liew HD, Flynn ET. Direct ascent from air and N₂-O₂ saturation dives in humans: DCS risk and evidence of a threshold. *Undersea Hyperb Med*. 2005;32(6):409-419.
7. Freiberger JJ, Denoble PJ, Pieper CF, Ugucconi DM, Pollock NW, Vann RD. The relative risk of decompression sickness during and after air travel following diving. *Aviat Space Environ Med*. 2002;73:980-984.
8. Vann RD, Pollock NW, Freiberger JJ, Natoli MJ, Denoble PJ, Pieper CF. Influence of bottom time on preflight surface intervals before flying after diving. *Undersea Hyperb Med*. 2007;34(3):211-220.
9. Webb JT, Pilmanis AA, O'Connor RB. An abrupt zero-pre-oxygenation altitude threshold for decompression sickness symptoms. *Aviat Space Environ Med*. 1998;69(4):335-340.
10. Webb JT, Kannan N, Pilmanis AA. Gender not a factor for altitude decompression sickness risk. *Aviat Space Environ Med*. 2003;74(1):2-10.
11. Ryles MT, Pilmanis AA. The initial signs and symptoms of altitude decompression sickness. *Aviat Space Environ Med*. 1996;67(10):983-989.
12. Balldin UI, Pilmanis AA, Webb JT. Central nervous system decompression sickness and venous gas emboli in hypobaric conditions. *Aviat Space Environ Med*. 2004;75(11):969-972.
13. Ercan E, Demir AE, Sabaner E, Toklu AS. Incidence of decompression sickness in hypobaric hypoxia training. *Undersea Hyperb Med*. 2020;47(2):203-210.
14. Adler HF. Neurocirculatory Collapse at Altitude. USAF School of Aviation Medicine. Project Report. Randolph Field; 1950.
15. Hornberger W. Decompression sickness. *German Aviation Medicine World War II*. Vol. I. Washington, DC: Department of the Air Force, US Government Printing Office; 1950. 354-394.
16. Berry CA, King AH. Severe dysbarism in actual and simulated flight; a follow-up study of five cases. *U S Armed Forces Med J*. 1959;10(1):1-15.
17. Rayman RB, McNaughton GB. Decompression sickness: USAF experience 1970-80. *Aviat Space Environ Med*. 1983;54(3):258-260.
18. Weien RW, Baumgartner N. Altitude decompression sickness: hyperbaric therapy results in 528 cases. *Aviat Space Environ Med*. 1990;61:833-836.
19. Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377(9760):153-164.
20. Moon RE. Hyperbaric treatment of air or gas embolism: current recommendations. *Undersea Hyperb Med*. 2019;46(5):673-683.
21. Francis TJ, Griffin JL, Homer LD, Pezeshkpour GH, et al. Bubble-induced dysfunction in acute spinal cord decompression sickness. *J Appl Physiol* (1985). 1990;68:1368-1375.
22. Philp RB, Schacham P, Gowdey CW. Involvement of platelets and microthrombi in experimental decompression sickness: similarities with disseminated intravascular coagulation. *Aerosp Med*. 1971;42(5):494-502.
23. Nossum V, Koteng S, Brubakk AO. Endothelial damage by bubbles in the pulmonary artery of the pig. *Undersea Hyperb Med*. 1999;26(1):1-8.
24. 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;86:209-214.
25. Malette WG, Fitzgerald JB, Cockett AT. Dysbarism. A review of thirty-five cases with suggestion for therapy. *Aerosp Med*. 1962;33:1132-1139.
26. Brunner F, Frick P, Bühlmann A. Post-decompression shock due to extravasation of plasma. *Lancet*. 1964;1:1071-1073.
27. Boussuges A, Blanc P, Molenat F, Bergmann E, Sainy JM. Haemoconcentration in neurological decompression illness. *Int J Sports Med*. 1996;17:351-355.
28. Levin LL, Stewart GJ, Lynch PR, Bove AA. Blood and blood vessel wall changes induced by decompression sickness in dogs. *J Appl Physiol* (1985). 1981;50:944-949.
29. 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* (1985). 1986;60:1651-1658.
30. 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.
31. Little T, Butler BD. Pharmacological intervention to the inflammatory response from decompression sickness in rats. *Aviat Space Environ Med*. 2008;79(2):87-93.

32. Helps SC, Gorman DF. Air embolism of the brain in rabbits pre-treated with mechlorethamine. *Stroke*. 1991;22:351-354.
33. Thom SR, Yang M, Bhopale VM, Huang S, Milovanova TN. Microparticles initiate decompression-induced neutrophil activation and subsequent vascular injuries. *J Appl Physiol* (1985). 2011;110(2):340-351.
34. Yang M, Kosterin P, Salzberg BM, Milovanova TN, Bhopale VM, Thom SR. Microparticles generated by decompression stress cause central nervous system injury manifested as neurohypophysial terminal action potential broadening. *J Appl Physiol* (1985). 2013 Nov;115(10):1481-1486
35. Elliott DH, Moon RE. Manifestations of the decompression disorders. In: Bennett PB, Elliott DH, editors. *The Physiology and Medicine of Diving*. Philadelphia, PA: WB Saunders; 1993. 481-505.
36. Francis TJR, Mitchell SJ. Manifestations of decompression disorders. In: Brubakk AO, Neuman TS, editors. *Bennett & Elliott's Physiology and Medicine of Diving*. New York, NY: Elsevier Science; 2003. 578-599.
37. Mitchell SJ. Decompression sickness: manifestations. In: Edmonds C, Bennett MH, editors. *Diving and Subaquatic Medicine*. 5 ed. Boca Raton, FL: Taylor and Francis; 2015. 141-151.
38. Zwirowich CV, Müller NL, Abboud RT, Lepawsky M. Non-cardiogenic pulmonary edema caused by decompression sickness: rapid resolution following hyperbaric therapy. *Radiology*. 1987;163:81-82.
39. Trytko B, Mitchell SJ. Extreme survival: a deep technical diving accident. *SPUMS J*. 2005;35:23-27.
40. Warren LP, Djang WT, Moon RE, Camporesi EM, et al. Neuroimaging of scuba diving injuries to the CNS. *AJNR Am J Neuroradiol*. 1988;9:933-938.
41. Reuter M, Tetzlaff K, Hutzelmann A, Fritsch G, Steffens JC, Bettinghausen E, Heller M. MR imaging of the central nervous system in diving-related decompression illness. *Acta Radiol*. 1997;38(6):940-944.
42. Gempp E, Blatteau JE, Stephant E, Pontier JM, et al. MRI findings and clinical outcome in 45 divers with spinal cord decompression sickness. *Aviat Space Environ Med*. 2008;79(12):1112-1116.
43. Chung JM, Ahn JY. Relationship between clinical and radiologic findings of spinal cord injury in decompression sickness. *Undersea Hyperb Med*. 2017;44(1):57-62.
44. 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;2:241-279.
45. Moir EW. Tunnelling by compressed air. *J Soc Arts*. 1896; 44(May 15):567-585.
46. Bert P. *Barometric pressure (La pression barométrique)*. Bethesda, MD: Undersea Medical Society; 1978.
47. Zuntz N. Zur Pathogenese und Therapie der durch rasche Luftdruckänderungen erzeugten Krankheiten. *Fortschr Med*. 1897;15:632-639.
48. Yarbrough OD, Behnke AR. The treatment of compressed air illness using oxygen. *J Ind Hyg Toxicol*. 1939;21:213-218.
49. 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;91:1110-1123.
50. Martin JD, Thom SR. Vascular leukocyte sequestration in decompression sickness and prophylactic hyperbaric oxygen therapy in rats. *Aviat Space Environ Med*. 2002;73(6):565-569.
51. Kindwall EP. Use of short versus long tables in the treatment of decompression sickness and arterial gas embolism. In: Moon RE, Sheffield PJ, editors. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996. 122-126.
52. Thalmann ED. Principles of US Navy recompression treatments for decompression sickness. In: Moon RE, Sheffield PJ, editors. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996. 75-95.
53. Moon RE, Sheffield PJ. Guidelines for treatment of decompression illness. *Aviat Space Environ Med*. 1997;68:234-243.
54. Moon RE, Gorman DF. Treatment of the decompression disorders. In: Neuman TS, Brubakk AO, editors. *Bennett & Elliott's Physiology and Medicine of Diving*. New York, NY: Elsevier Science; 2003. 600-650.
55. Hadanny A, Fishlev G, Bechor Y, Bergan J, Friedman M, Maliar A, Efrati S. Delayed recompression for decompression sickness: retrospective analysis. *PLoS ONE*. 2015;10(4):e0124919.
56. Chin W, Joo E, Ninokawa S, Popa DA, Covington DB. Efficacy of the U.S. Navy Treatment Tables in treating DCS in 103 recreational scuba divers. *Undersea Hyperb Med*. 2017; 44(5):399-405.
57. Moon RE, Gorman DF. Decompression sickness. In: Neuman TS, Thom SR, editors. *The Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders Elsevier; 2008. 283-319.
58. Navy Department. *US Navy Diving Manual. Revision 7. Vol 5: Diving Medicine and Recompression Chamber Operations*. NAVSEA 0910-LP-115-1921. Washington, DC: Naval Sea Systems Command; 2016.
59. Mitchell SJ, Doolette DJ, Wachholz CJ, Vann RD, editors. *Management of Mild or Marginal Decompression Illness in Remote Locations*. Durham, NC: Divers Alert Network; 2005.
60. Mitchell SJ, Bennett MH, Bryson P, Butler FK, et al. Pre-hospital management of decompression illness: expert review of key principles and controversies. *Diving Hyperb Med*. 2018;48(1):45-55.

61. Ball R. Effect of severity, time to recompression with oxygen, and retreatment on outcome in forty-nine cases of spinal cord decompression sickness. *Undersea Hyperb Med.* 1993;20:133-145.
62. 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.
63. Goodman MW, Workman RD. Minimal recompression oxygen-breathing approach to treatment of decompression sickness in divers and aviators. Washington, DC: US Navy Experimental Diving Unit Report #5-65; 1965.
64. Bennett MH, Mitchell SJ, Young D, King D. The use of deep tables in the treatment of decompression illness: the Hyperbaric Technicians and Nurses Association 2011 Workshop. *Diving Hyperb Med.* 2012;42(3):171-180.
65. Vann RD, Bute BP, Uguccioni DM, Smith LR. Prognostic factors in DCI in recreational divers. In: Moon RE, Sheffield PJ, editors. *Treatment of Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996. 352-363.
66. Gempp E, Blatteau JE. Risk factors and treatment outcome in scuba divers with spinal cord decompression sickness. *J Crit Care.* 2010;25:236-242.
67. Rivera JC. Decompression sickness among divers: an analysis of 935 cases. *Mil Med.* 1964;129:314-334.
68. Workman RD. Treatment of bends with oxygen at high pressure. *Aerosp Med.* 1968;39:1076-1083.
69. How J, Chan G. Management of delayed cases of decompression sickness--3 case reports. *Singapore Med J.* 1973;14(4): 582-585.
70. Erde A, Edmonds C. Decompression sickness: a clinical series. *J Occup Med.* 1975;17(5):324-328.
71. Kizer KW. Delayed treatment of dysbarism: a retrospective review of 50 cases. *JAMA.* 1982;247(18):2555-2558.
72. Meyers RAM, Bray P. Delayed treatment of serious decompression sickness. *Ann Emerg Med.* 1985;14:254-257.
73. Curley MD, Schwartz HJC, Zwingelberg KM. Neuropsychologic assessment of cerebral decompression sickness and gas embolism. *Undersea Biomed Res.* 1988;15:223-236.
74. Rudge FW, Shafer MR. The effect of delay on treatment outcome in altitude-induced decompression sickness. *Aviat Space Environ Med.* 1991;62:687-690.
75. Kindwall EP. Decompression sickness. In: Davis JC, Hunt TK, editors. *Hyperbaric Oxygen Therapy*. Bethesda, MD: Undersea Medical Society; 1977. p. 125-40.
76. Hart GB, Strauss MB, Lennon PA. The treatment of decompression sickness and air embolism in a monoplace chamber. *J Hyperb Med.* 1986;1:1-7.
77. Elliott DH, Kindwall EP. Decompression sickness. In: Kindwall EP, Whelan HT, editors. *Hyperbaric Medicine Practice*. Flagstaff, AZ: Best Publishing Co; 1999. p. 433-87.
78. Bond JG, Moon RE, Morris DL. Initial table treatment of decompression sickness and arterial gas embolism. *Aviat Space Environ Med.* 1990;61:738-743.
79. Cianci P, Slade JB, Jr. Delayed treatment of decompression sickness with short, no-air-break tables: review of 140 cases. *Aviat Space Environ Med.* 2006;77(10):1003-1008.
80. Weaver LK. Monoplace hyperbaric chamber use of U.S. Navy Table 6: a 20-year experience. *Undersea Hyperb Med.* 2006; 33(2):85-88.
81. Farm FP, Jr, Hayashi EM, Beckman EL. Diving and decompression sickness treatment practices among Hawaii's diving fisherman. Sea Grant Technical Paper UNIHI-SEA-GRANT-TP-86-01. Sea Grant Technical Paper. Honolulu: University of Hawaii; 1986. Report No.: UNIHI-SEAGRANT-TP-86-01.
82. Doolette DJ, Mitchell SJ. In-water recompression. *Diving Hyperb Med.* 2018;48(2):84-95.
83. Pyle RL, Youngblood DA. In-water recompression as an emergency field treatment of decompression illness. *SPUMS J.* 1997;27:154-69.
84. Dituri J, Sadler R, Siddiqi F, Sadler C, et al. Echocardiographic evaluation of intracardiac venous gas emboli following in-water recompression. *Undersea Hyperb Med.* 2016;43(2): 103-112.
85. Dart TS, Butler W. Towards new paradigms for the treatment of hypobaric decompression sickness. *Aviat Space Environ Med.* 1998;69(4):403-409.
86. Butler WP, Topper SM, Dart TS. USAF treatment table 8: Treatment for altitude decompression sickness. *Aviat Space Environ Med.* 2002;73(1):46-49.
87. Moon RE, editor. *Adjunctive Therapy for Decompression Illness*. Kensington, MD: Undersea and Hyperbaric Medical Society; 2003. ◆