

FROM THE HBO₂ INDICATIONS MANUAL 14TH EDITION:

CHAPTER 7

Hyperbaric treatment for decompression sickness: current recommendations

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Rationale

Decompression sickness (DCS, “bends”) is caused by 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 any of the following:

- ascent from a dive;
- depressurization of a hyperbaric chamber;
- rapid ascent to altitude in an unpressurised 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 rarely DCS has occurred after either repetitive or very deep breath-hold dives [3,4]. 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) [5]. DCS after a dive can be provoked by mild altitude exposure, such as a commercial aircraft flight [6,7], but without a preceding dive the threshold altitude for DCS occurrence in unpressurized flight is 18,000-20,000 feet [8,9].

Several mechanisms have been hypothesized by which bubbles may exert their deleterious effects. These include:

- direct mechanical disruption of tissue [10];
- occlusion of blood flow, platelet deposition and activation of the coagulation cascade [11];
- endothelial dysfunction [12-13];
- capillary leakage [14-18];
- endothelial cell death, complement activation [19,20];
- inflammation [21]; and
- leukocyte-endothelial interaction [22].

Recent evidence suggests that circulating microparticles may play a pro-inflammatory role in DCS pathophysiology [23,24].

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 [25-28]. DCS manifestations most commonly include paresthesias, hypesthesia, musculoskeletal pain, skin rash and malaise [25-28]. Less common but more serious signs and symptoms include motor weakness, ataxia, vertigo, hearing loss, dyspnea, pulmonary edema [29], bladder and anal sphincter dysfunction, shock and death [25-28]. Severe DCS may be accompanied by hemoconcentration, hypotension and coagulopathy [17, 30]. 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) [27]. Altitude DCS has similar manifestations, although cerebral manifestations seem to occur more frequently [27].

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 occasionally 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 [31, 32]. Magnetic resonance imaging is not sufficiently sensitive to consistently detect early anatomic correlates of neurological DCI [33, 34]. 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.

KEYWORDS: decompression sickness; bends; hyperbaric oxygen

Improvement of decompression sickness symptoms as a result of recompression was first noted in the 19th century [35]. Recompression with air was first implemented as a specific treatment for that purpose in 1896 [36]. Oxygen breathing was observed by Bert in 1878 to improve the signs of decompression sickness in animals [37]. The use of oxygen with pressure to accelerate gas diffusion and bubble resolution in humans was first suggested in 1897 [38] and eventually tested in the 1930s for human DCS and recommended for the treatment of divers [39]. 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 CNS edema. It is also likely that HBO₂ has other beneficial pharmacological effects, such as a reduction in neutrophil adhesion to the capillary endothelium [40,41]. The efficacy of HBO₂ is now widely accepted, and HBO₂ is the mainstay of treatment for this disease [27,42-47].

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 [44,45,48-49].

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 [50]. Thus, although HBO₂ remains the preferred intervention in all cases of DCS, not least because DCS may recover more slowly without recompression [50] 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 [51].

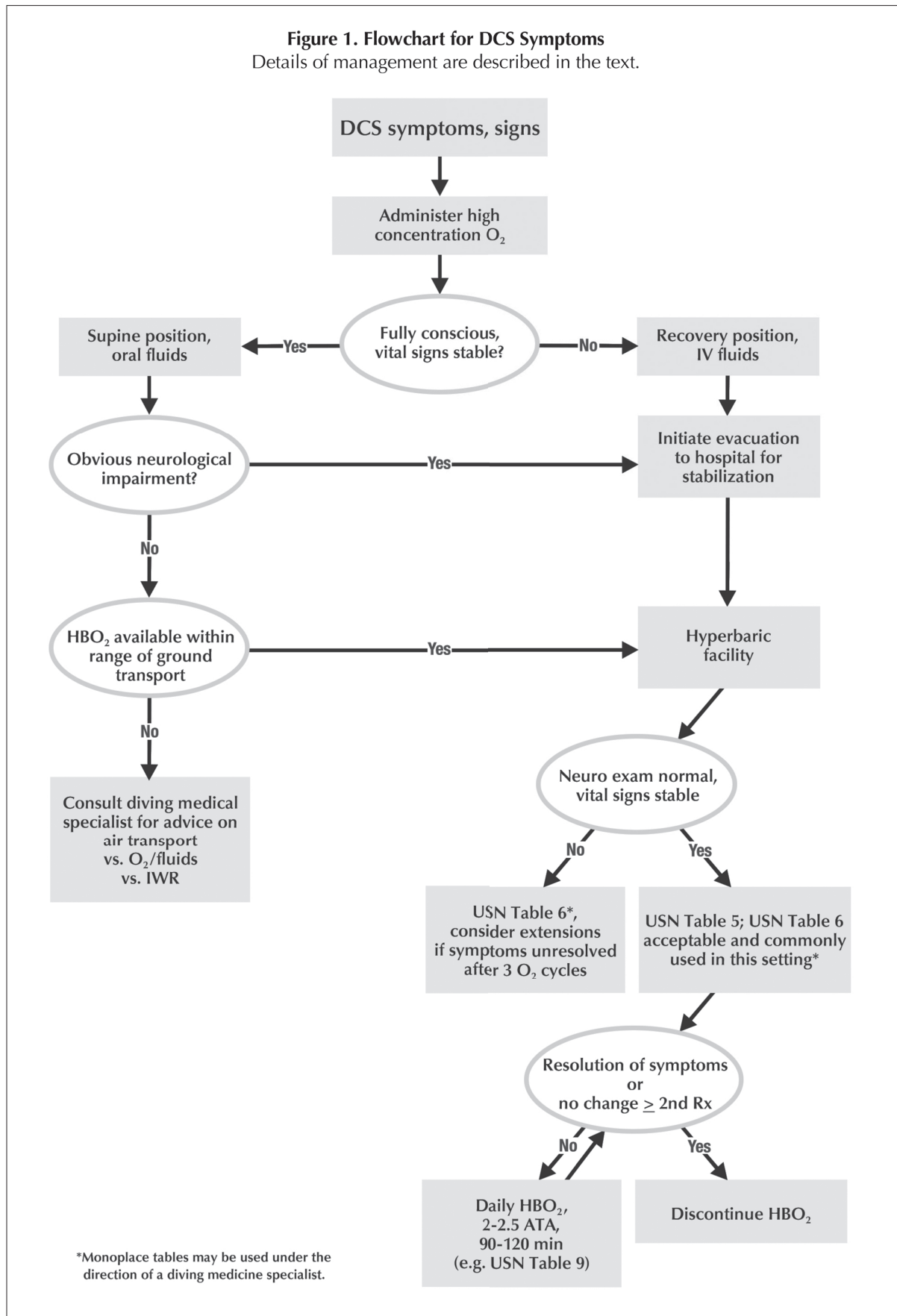
Clinical management

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 atmosphere absolute [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 [51].

- 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 an electrolyte-containing oral resuscitation fluid such as WHO oral rehydration solution or Pedialyte™ (but 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 nonsteroidal anti-inflammatory drug (NSAID) is appropriate if there are no contraindications.

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



- 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 the following: (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 the following: (a) complete resolution is more likely to result from early hyperbaric treatment [27, 44]; (b) the U.S. Navy (USN) oxygen treatment tables [49] (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 [27, 43, 46, 47, 52, 53], Treatment at shallower depths (e.g., 33 fsw, 10 msw, 2 ATA) can also be effective, although published case series suggest that the success rate may be lower at treatment depths less than 60 fsw [46, 54].

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 [55]. The speculative use of treatment schedules that deviate from the 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 to two treatments [27]. 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 Table 6) [49] are not justified, and it is typical to utilize shorter treatments (such as the U.S. Navy Table 5) [49] 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 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 five to 10 repetitive treatments for most individuals [56].

Time from symptom onset to hyperbaric treatment. Available data do not convincingly demonstrate superior outcomes in "rapid" versus delayed treatment [53, 57]. For example, in two published series, time to treatment greater than 2,447 or 4,846 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, which suggest immediate recompression is extremely effective in controlling symptoms [43, 58-59]. 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 [59-65].

Monoplace chamber treatment. Monoplace chambers were originally designed for the continuous administration of 100% oxygen and were 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 decompressionsickness that are shorter than standard USN treatment tables [66-68]. 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 [42, 69-70]. 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 [71].

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 while breathing air has been used by indigenous divers with a high reported success rate, although clinical details are scant [72]. There is anecdotal evidence that IWR using oxygen is more effective [73]. However, a major risk is an oxygen convulsion resulting in fatal drowning. IWR using oxygen has been discussed in the literature [51,73,74] and is described in the U.S. Navy Diving Manual [49]. Typical IWR oxygen-breathing protocols recommend depths no greater than 30 fsw (USN) or shallower [73]. Recommendations include a requirement that the diver not use a regular scuba mouthpiece but rather a full face mask, surface-supplied helmet breathing apparatus or regulator retention strap (“gag strap”) [75]. Other requirements include the need for a tender in the water and the symptomatic diver to be tethered [73]. 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 [51]. IWR 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 [44,76].

- **Mild symptoms that clear on descent to ground level with normal neurological exam:** 100% oxygen by tightly fitted mask for a two-hour 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 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 therapy 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 (USAF Table 8) has also been used effectively for altitude DCS [77].

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 [78], which is available on the Undersea and Hyperbaric Society website at: (www.uhms.org/images/Publications/ADJUNCTIVE_THERAPY_FOR_DCI.pdf).

Evidence-based review

The use of HBO₂ for decompression sickness is an AHA level I recommendation (level of evidence C). A number of adjunctive therapies have been used for the treatment of DCS (Table 1) and discussed in the Report of the Decompression Illness Adjunctive Therapy Committee of the Undersea and Hyperbaric Medical Society [78]. These guidelines can be accessed via the internet at: www.uhms.org/images/Publications/ADJUNCTIVE_THERAPY_FOR_DCI.pdf.

Utilization review

Utilization review should occur after 10 treatments.

Table 1. Evidence-based review of adjunctive therapies for DCS

CONDITION								
	AGE (no significant inert gas load)		DCS: pain only/mild		DCS: neurological		DCS: chokes (cardiorespiratory)	
	Class	Level	Class	Level	Class	Level	Class	Level
surface O ₂ (1 ATA)	I	C	I	C	I	C	I	C
intravenous fluid therapy	D5W [†] III LR/crystalloid [‡] I Ib colloid ^{!!} I Ib	C	D5W [†] III LR/crystalloid [‡] I Ib colloid ^{!!} I Ib	C	D5W [†] III LR/crystalloid [‡] I Ib colloid ^{!!} I Ib	C	D5W [†] III LR/crystalloid [‡] I Ib colloid ^{!!} I Ib	C
aspirin	I Ib	C	I Ib	C	I Ib C		I Ib	C
NSAIDs	I Ib	C	I Ib	B	I Ib	B	I Ib	C
anticoagulants*	I Ib	C	III	C	I Ib§	C	I Ib	C
corticosteroids	III	C	III	C	III	C	III	C
lidocaine	I Ia	B	III	C	I Ib	C	III	C

(Table from Moon:78 www.uhms.org/images/Publications/ADJUNCTIVE_THERAPY_FOR_DCI.pdf)

§ 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.

Cost impact

Only those people exposed to increased ambient pressure (divers or compressed air workers) or who suffer decompression sickness at altitude are affected. Because there are relatively few individuals who develop this condition, the application 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 spinal cord, brain or peripheral nerve damage or death, and is therefore an exceptionally cost-effective treatment.



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