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안전을 향한 생각이 바른 가치를 만듭니다

1. Diving injury
2. Decompression Sickness
3. DCS pathophysiology
4. DCS manifestation
5. UHMS guideline

Diving injury

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Barotrauma

Pulmonary Barotrauma
Ear Barotrauma
Sinus Barotrauma
Other Barotrauma

Decompression Sickness

Decompression Sickness

Abnormal gas pressures

Inert gas narcosis
Hypoxia
Oxygen toxicity
Carbon dioxide toxicity
High-pressure neurological syndrome

Aquatic disorder

Drowning syndrome
Seasickness
Cold and hypothermia
Scuba divers' pulmonary edema

Diving injury

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TABLE 214-1 Summary of Barotrauma of Descent and Ascent

Barotrauma	Clinical Features	Treatment
Barotrauma of descent		
Otic barotrauma ("ear squeeze")	Pain, fullness, vertigo, conductive hearing loss from inability to equalize middle ear pressure	Decongestants, consider antibiotics
Sinus barotrauma ("sinus squeeze")	Pain over affected sinus, possible bleeding from nares	Decongestants, consider antibiotics
Inner ear barotrauma	Sudden onset of sensorineural hearing loss, tinnitus, severe vertigo after forced Valsalva	Head of bed up, no nose blowing, antivertigo medications, and urgent otolaryngology consultation as some surgeons advocate early exploration
Barotrauma of ascent		
Pulmonary overinflation syndromes (pulmonary barotrauma)	Dyspnea, chest pain, subcutaneous air, extra-alveolar air on radiograph; usually occurring secondary to rapid or uncontrolled ascent	Pneumomediastinum requires only symptomatic care and does not require recompression Pneumothorax requires drainage and does not require recompression (if recompression is instituted for treatment of arterial gas embolism, then the pneumothorax must be drained before recompression)
Arterial gas embolism	Neurologic symptoms occurring immediately after uncontrolled or rapid ascent or neurologic symptoms in the setting of pulmonary barotrauma	Airway, breathing, circulation, high-flow oxygen, IV hydration, immediate recompression (hyperbaric oxygen), consider adjunctive lidocaine Any neurologic symptom in the setting of documented pulmonary barotrauma must be treated as an arterial gas embolism

TABLE 214-2 Classification of Decompression Sickness (DCS)

Classification	Clinical Features	Comments
Type I: "pain-only" DCS	Deep pain in joints and extremities, unrelieved but not worsened with movement	Usually single joint, most commonly knees and shoulders
	Skin changes—mottling, pruritus, and color changes	Lymphatic obstruction can occur and takes days to resolve despite recompression therapy
Type II: "serious" DCS	Pulmonary ("chokes")—cough, hemoptysis, dyspnea, and substernal chest pain	
	Cardiovascular collapse can occur	
	Neurologic—sensation of truncal constriction, ascending paralysis, usually rapid in onset	More commonly affects the lower cervical and thoracic regions; may see scattered lesions Autonomic dysfunction may be seen
	Vestibular ("staggers")—vertigo, hearing loss, tinnitus, and disequilibrium	Usually occurs after deep, long dives
Type III: combination of DCS and arterial gas embolism	Symptoms of DCS II noted above plus a variety of stroke syndromes, symptoms, and signs	Symptoms occur on ascent or immediately upon surfacing Symptoms of arterial gas embolism may spontaneously resolve

Decompression Sickness

disease caused primarily by bubbles formed from dissolved gas in blood and/or tissue following a reduction in ambient pressure.

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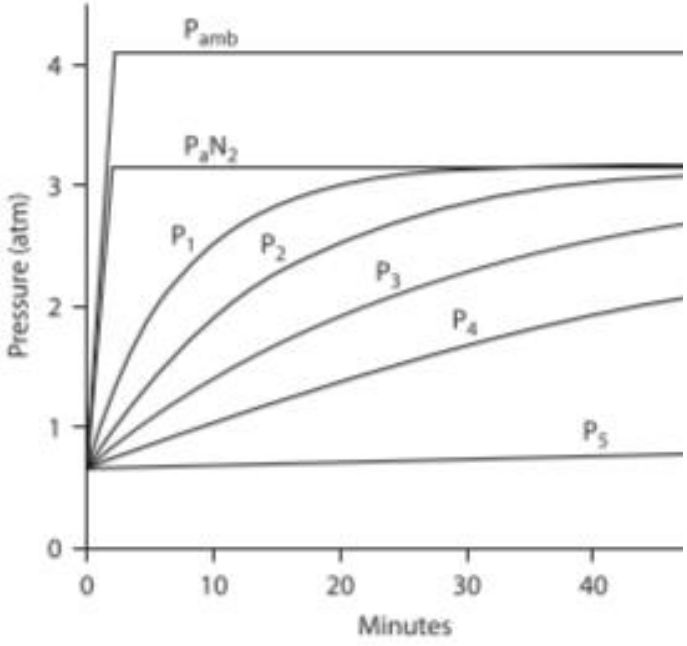
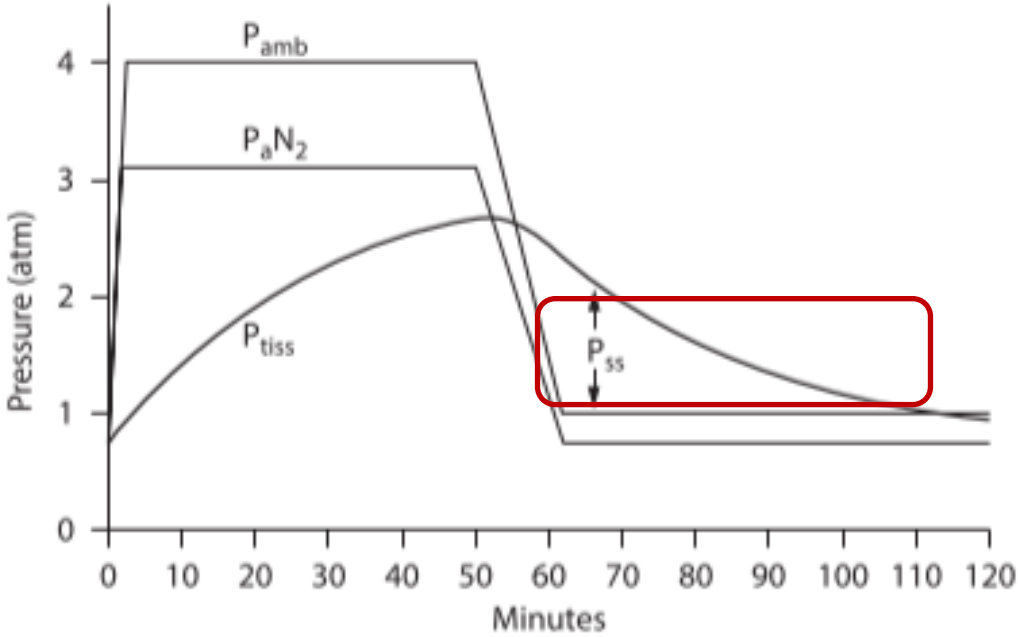
Pathophysiology

Uptake and elimination of inert gas

- Perfusion & Solubility
- Faster tissue: lung, brain, heart, kidney
- Slower tissue: bone, fat

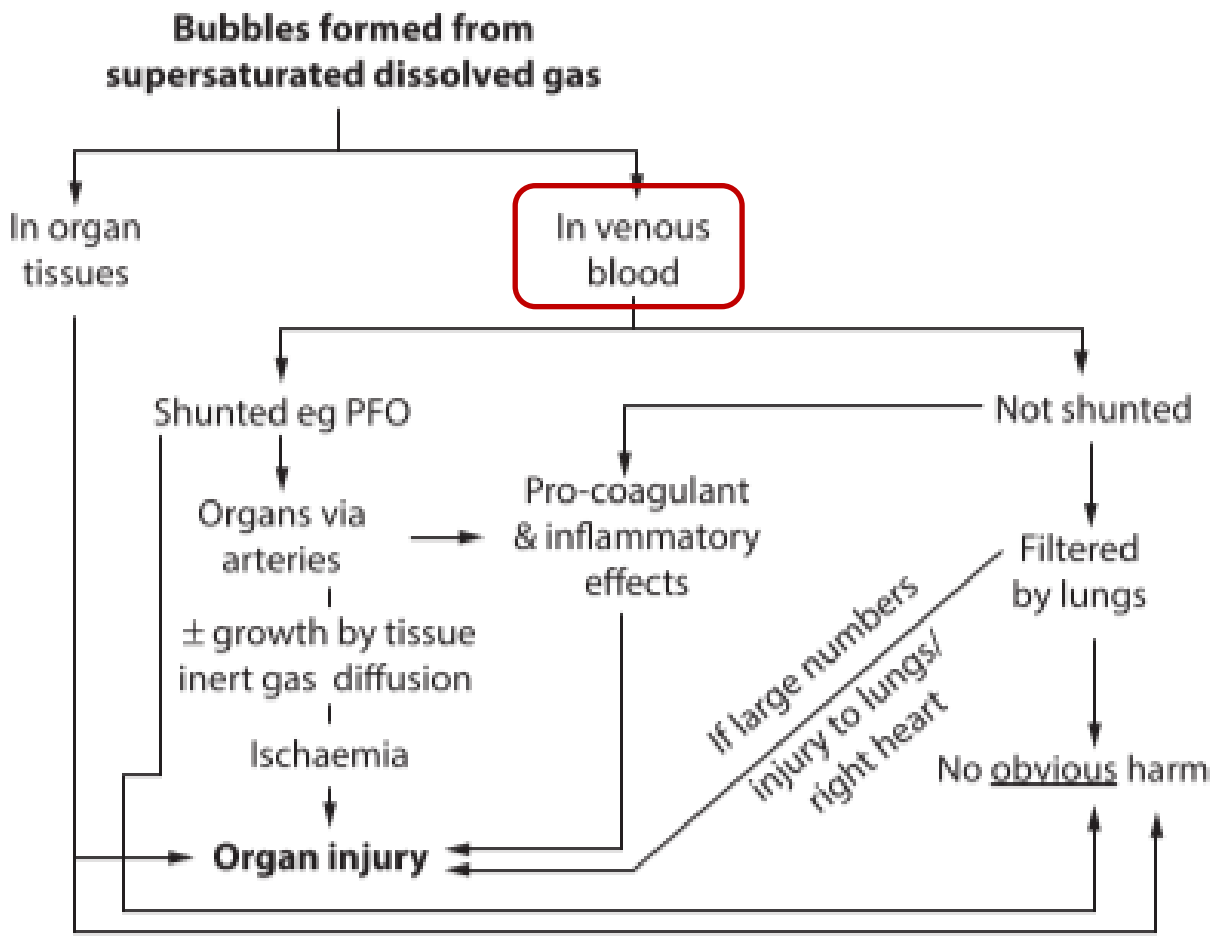




Pathophysiology
#bubble mechanism

- Direct mechanical disruption of tissue.
- Occlusion of blood flow, platelet deposition and activation of the coagulation cascade
- endothelial dysfunction
- capillary leakage
- endothelial cell death, complement activation
- inflammation
- leukocyte-endothelial interaction

Pathophysiology



Manifestation

Type I DCS

- Initially a designation for cases with musculoskeletal pain
- ‘pain only DCS’
- Modification -> include rash, lymphatic symptoms and mild neurological symptoms

Type II DCS

- Presence of neurological symptoms
- Patchy paresthesia vs gross motor dysfunction.

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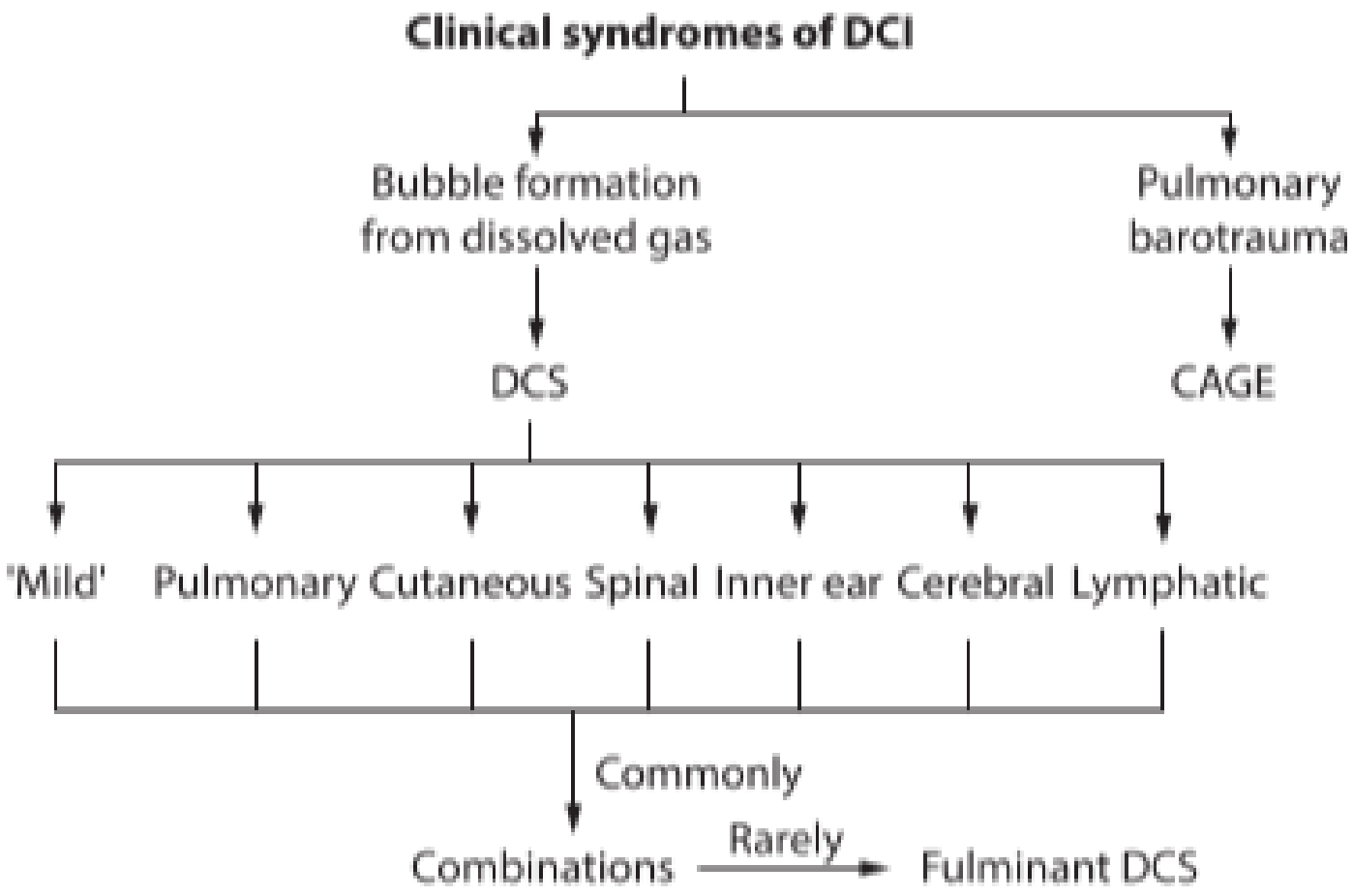
Manifestation



Manifestation
#mild DCS

The symptoms in the mild category are as follows:

1. Musculoskeletal pain.
2. Subjective sensory changes in a non- dermatomal distribution.
3. Constitutional symptoms such as fatigue and malaise.
4. Itch and rash of the superficial erythematous type.

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Manifestation
#mild DCS

Musculoskeletal pain →	'Deep boring ache' Proximal joints Usually early onset, but variable Back and abdominal pain is not mild
Constitutional symptoms →	Fatigue/malaise Usually early onset, but fatigue common after diving May be noticed the next day Cognitive dysfunction is not mild
Subjective sensory change →	'Patchy tingling' Non-dermatomal, may migrate Usually noticed after other sx Objective numbness is not mild
Rash/itch →	Erythematous rash Often on trunk but 'anywhere' Usually early onset Cutis marmorata is not mild

Figure 11.3 Symptoms of DCS that were designated "mild" by the 2004 remote DCS workshop[2].

Manifestation
#Pulmonary DCS

Cardiopulmonary DCS

'The chokes'

Rare and potentially fatal

Cough, short of breath, retrosternal chest pain, confusion, collapse

Large number of venous gas emboli

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#Cutaneous DCS

Cutis marmorata

Frequent association with other more serious manifestation

there is erythema accompanied by pruritus, and then the rash spreads irregularly and deepens in color. It develops a mottled appearance, with areas of pallor surrounded by cyanotic patches.

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#Spinal DCS

All levels of the spinal cord may be involved

thoracolumbar > cervical

Bilateral sensory change (distal -> proximal)

Weakness producing ascending paraplegia

Loss of bladder sensation

Loss of anal tone

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#Inner ear DCS

Vestibular symptoms: vertigo, nausea, vomiting, ataxia

Cochlear symptoms: tinnitus, deafness

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Manifestation

#Cerebral DCS

dysexecutive syndromes in which the diver complains of difficulties with concentration, memory, mood and other cognitive functions

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Manifestation
#lymphatic DCS

Localized subcutaneous swelling

Upper thorax and shoulder area

Not usually painful

Accompany with Musculoskeletal DCS

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UHMS guideline
#Rationale for HBOT

- 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
- reduction in neutrophil adhesion to the capillary endothelium

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UHMS guideline
#Patient selection criteria

- HBO2 treatment is recommended for all patients with symptoms of DCS whenever feasible
- Normobaric oxygen administration may be sufficient for the treatment of altitude DCS when neurological manifestations are absent, and for mild DCS following diving.

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UHMS guideline

#treatment table-USN table 5

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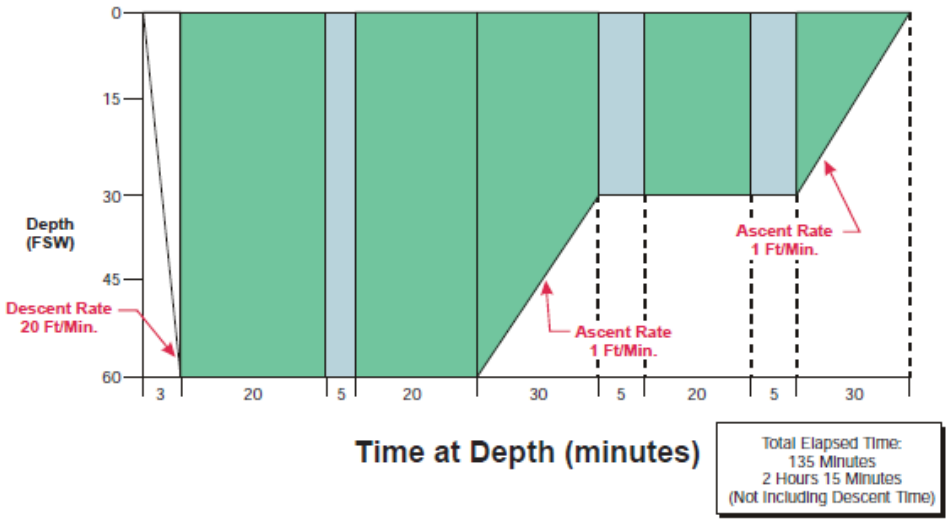
Treatment Table 5

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption (see paragraph 20-7.11.1.1)
5. Treatment Table may be extended two oxygen-breathing periods at the 30-foot stop. No air break required between oxygen-breathing periods or prior to ascent.
6. Tender breathes 100 percent O_2 during ascent from the 30-foot stop to the surface. If the tender had a previous hyperbaric exposure in the previous 18 hours, an additional 20 minutes of oxygen breathing is required prior to ascent.

Treatment Table 5 Depth/Time Profile



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UHMS guideline
#treatment table-USN table 6

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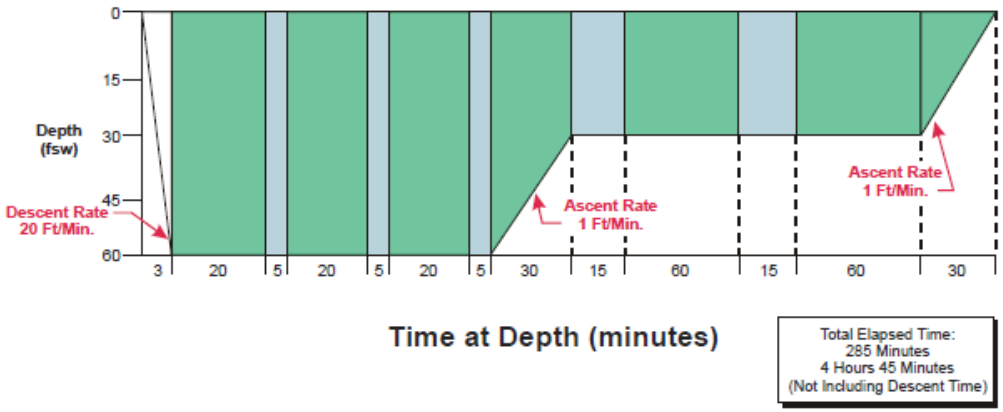
Treatment Table 6

1. Descent rate - 20 ft/min.
2. Ascent rate - Not to exceed 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster rates by halting the ascent.
3. Time on oxygen begins on arrival at 60 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, allow 15 minutes after the reaction has entirely subsided and resume schedule at point of interruption (see paragraph 20-7.11.1.1).
5. Table 6 can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air), or up to 2 additional 75-minute periods at 30 feet (15 minutes on air and 60 minutes on oxygen), or both.
6. Tender breathes 100 percent O_2 during the last 30 min. at 30 fsw and during ascent to the surface for an unmodified table or where there has been only a single extension at 30 or 60 feet. If there has been more than one extension, the O_2 breathing at 30 feet is increased to 60 minutes. If the tender had a hyperbaric exposure within the past 18 hours an additional 60-minute O_2 period is taken at 30 feet.

Treatment Table 6 Depth/Time Profile



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UHMS guideline
#treatment table-USN table 9

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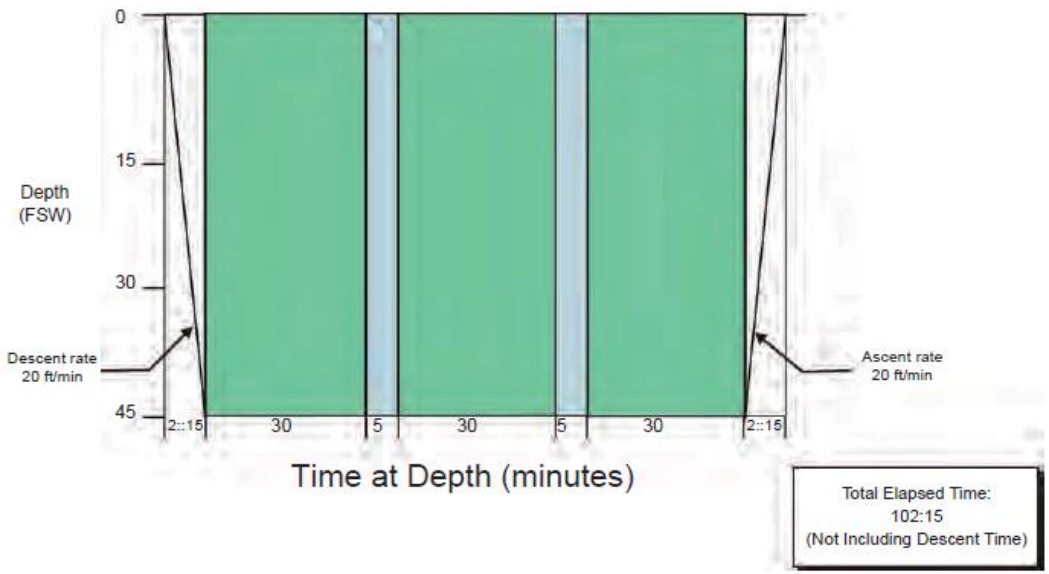
Treatment Table 9

1. Descent rate - 20 ft/min.
2. Ascent rate - 20 ft/min. Rate may be slowed to 1 ft/min depending upon the patient's medical condition.
3. Time at 45 feet begins on arrival at 45 feet.
4. If oxygen breathing must be interrupted because of CNS Oxygen Toxicity, oxygen breathing may be restarted 15 minutes after all symptoms have subsided. Resume schedule at point of interruption (see paragraph 20-7.11.1.1).
5. Tender breathes 100 percent O_2 during last 15 minutes at 45 feet and during ascent to the surface regardless of ascent rate used.
6. Patient may breathe air or oxygen during ascent.
7. If patient cannot tolerate oxygen at 45 feet, this table can be modified to allow a treatment depth of 30 feet. The oxygen breathing time can be extended to a maximum of 3 to 4 hours.

Treatment Table 9 Depth/Time Profile



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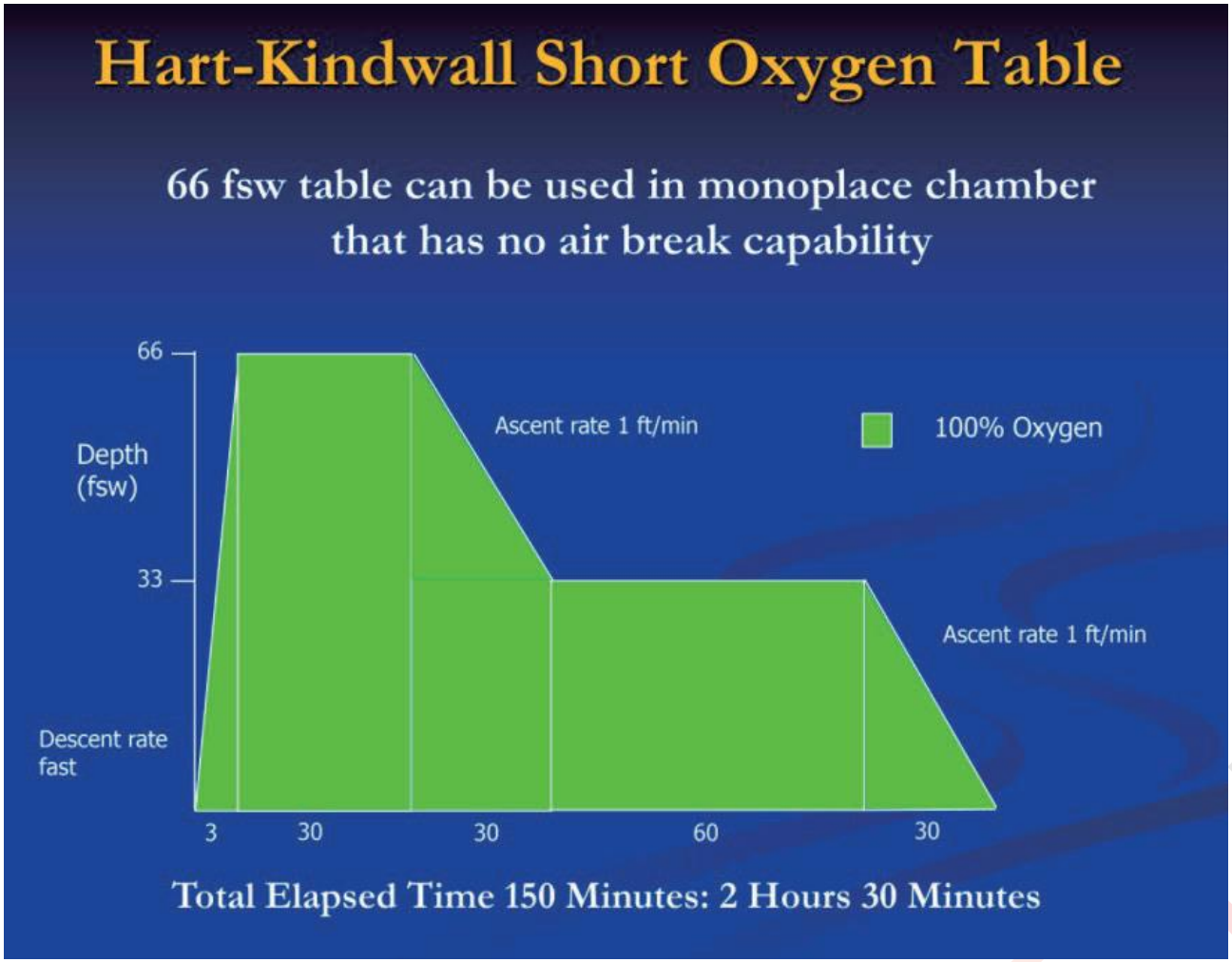
UHMS guideline

#treatment table-short oxygen table



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Review > Aviat Space Environ Med. 2006 Oct;77(10):1003-8.

Delayed treatment of decompression sickness with short, no-air-break tables: review of 140 cases

Paul Cianci [1], John B Slade Jr

Affiliations + expand

PMID: 17042243

Abstract

Introduction: Most cases of decompression sickness (DCS) in the U.S. are treated with hyperbaric oxygen using U.S. Navy Treatment Tables 5 and 6, although detailed analysis shows that those tables were based on limited data. We reviewed the development of these protocols and offer an alternative treatment table more suitable for monoplace chambers that has proven effective in the treatment of DCS in patients presenting to our facility.

Methods: We reviewed the outcomes for 140 cases of DCS in civilian divers treated with the shorter tables at our facility from January 1983 through December 2002.

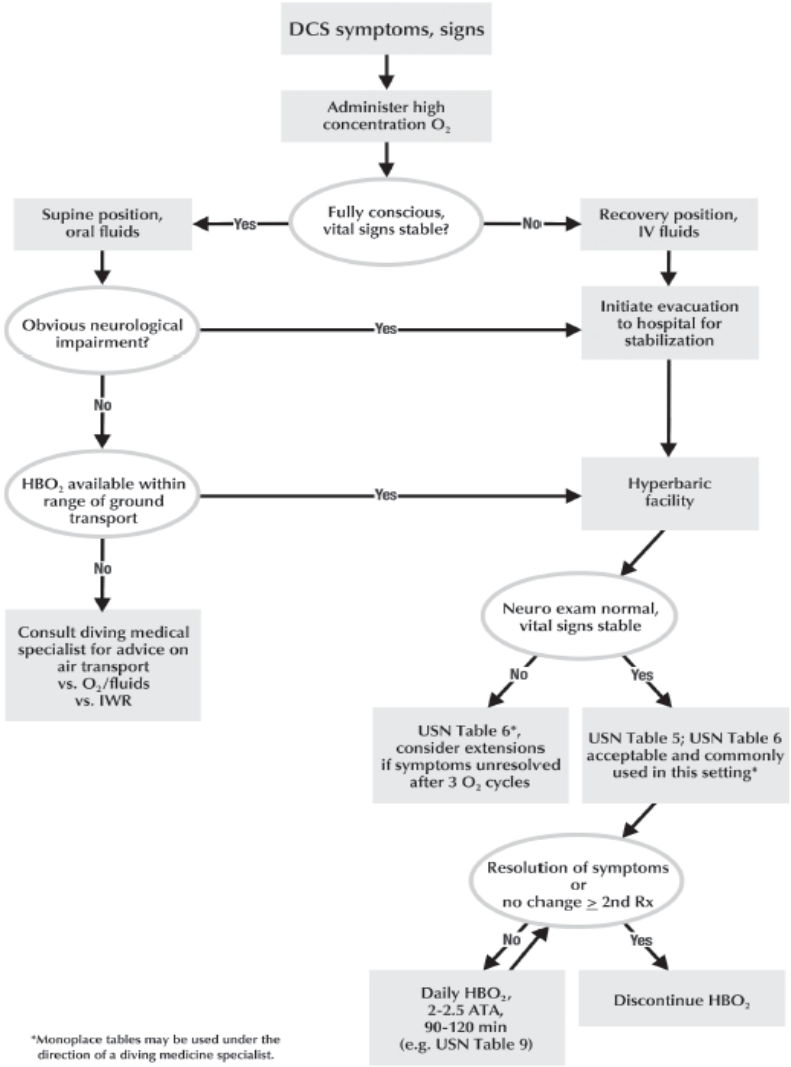
Results: Onset of symptoms averaged 9.3 h after surfacing. At presentation, 44% of the patients demonstrated mental aberration. The average delay from onset of symptoms to treatment was 93.5 h; median delay was 48 h. Complete recovery in the total group of 140 patients was 87%. When 30 patients with low probability of DCS were excluded, the recovery rate was 98%. All patients with cerebral symptoms recovered. Patients with the highest severity scores showed a high rate of complete recovery (97.5%).

Discussion: Short oxygen treatment tables as originally described by Hart are effective in the treatment of DCS, even with long delays to definitive recompression that often occur among civilian divers presenting to a major Divers Alert Network referral center.

UHMS guideline

#Flowchart for DCS symptoms

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



*Monoplace tables may be used under the direction of a diving medicine specialist.

UHMS guideline

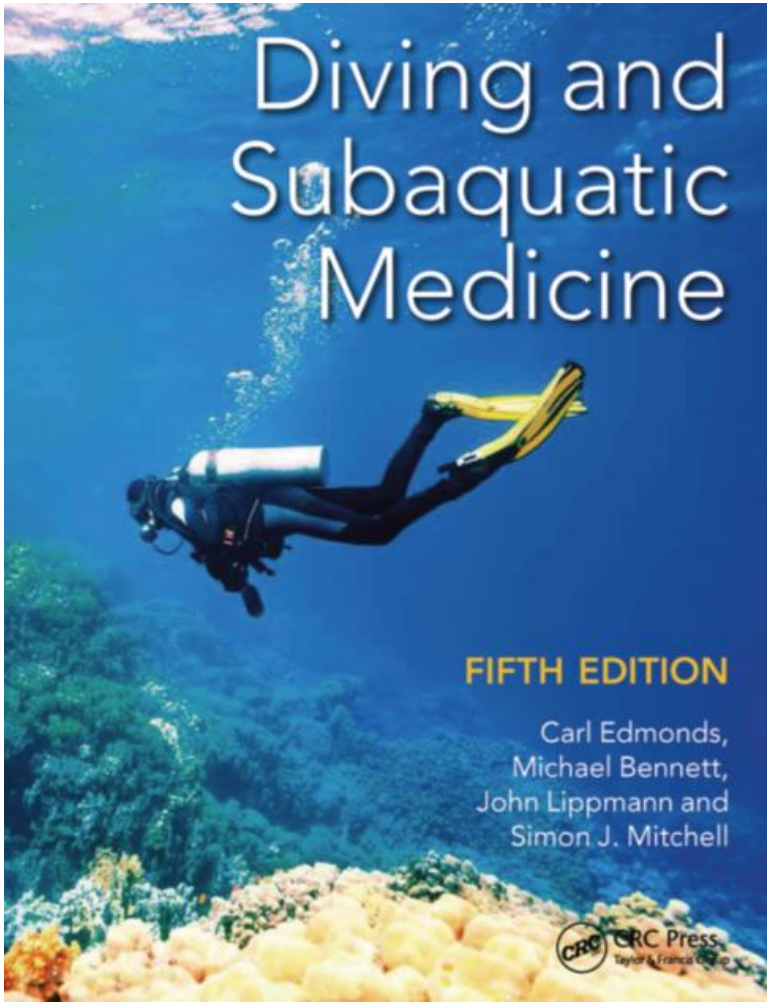
#adjuntive therapy

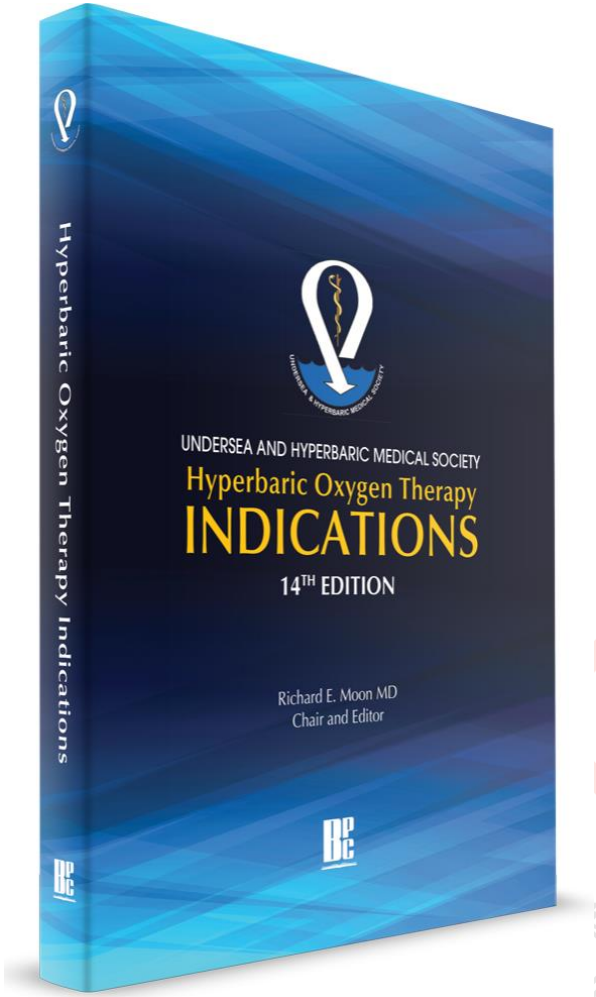
Table 1. Evidence-based Review of Adjunctive Therapies for DCS
(from Moon:[78] www.uhms.org/images/Publications/ADJUNCTIVE_THERAPY_FOR_DCI.pdf)

	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‡ IIb Colloid‖ IIb	C	D5W† III LR/crystalloid‡ I Colloid‖ I	C	D5W† III LR/crystalloid‡ I Colloid‖ I	C	D5W† III LR/crystalloid‡ IIb Colloid‖ IIb	C
Aspirin	IIb	C	IIb	C	IIb	C	IIb	C
NSAIDs	IIb	C	IIb	B	IIb	B	IIb	C
Anti-coagulants*	IIb	C	III	C	IIb§	C	IIb	C
Cortico-steroids	III	C	III	C	III	C	III	C
Lidocaine	IIa	B	III	C	IIb	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.

Further reading





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