Decompression Sickness

Gregory Barefoot, PA-C

Decompression Sickness

Primary Training in Hyperbaric Medicine

Columbia, South Carolina

ON PROBABILITY

In the end..
Whether a bubble
Causes trouble
Or dwindles in size
Until its demise
Is ruled by chance
~ A happenstance

What is Decompression Sickness?

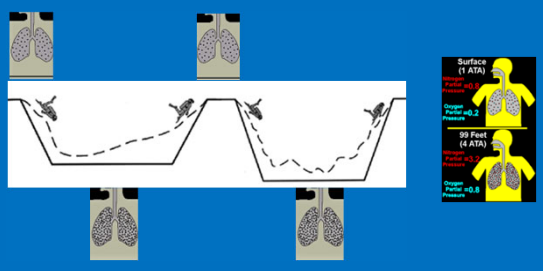
- Uncontrolled release of ***gas*** from tissues during or after surfacing with inadequate time for equilibration

What is Decompression Sickness?

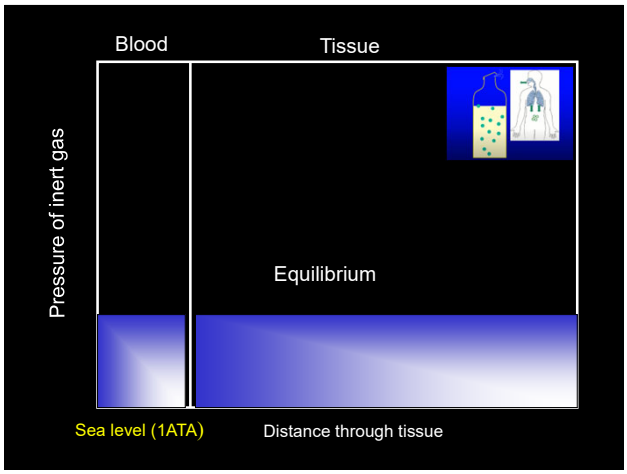
- Uncontrolled release of ***gas*** from tissues during or after surfacing with inadequate time for equilibration

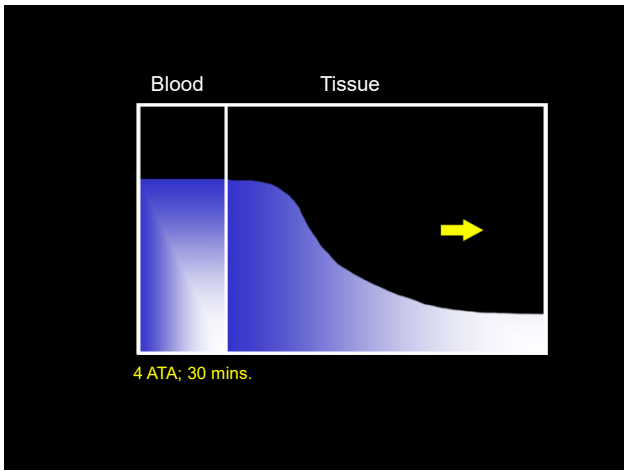
NITROGEN

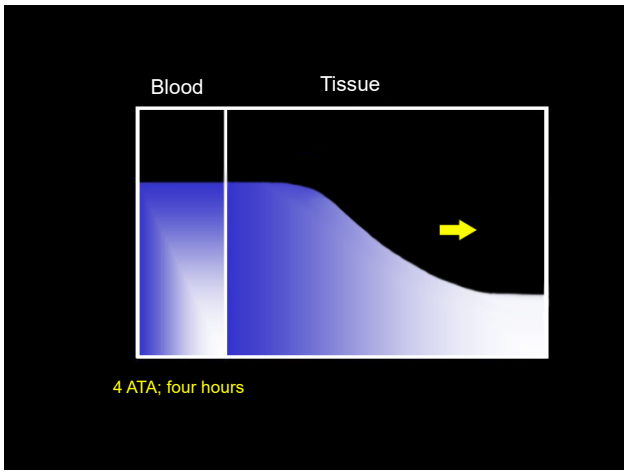
Nitrogen Absorption

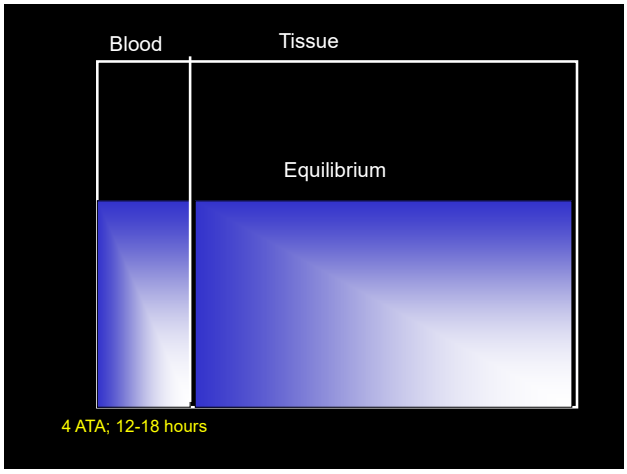


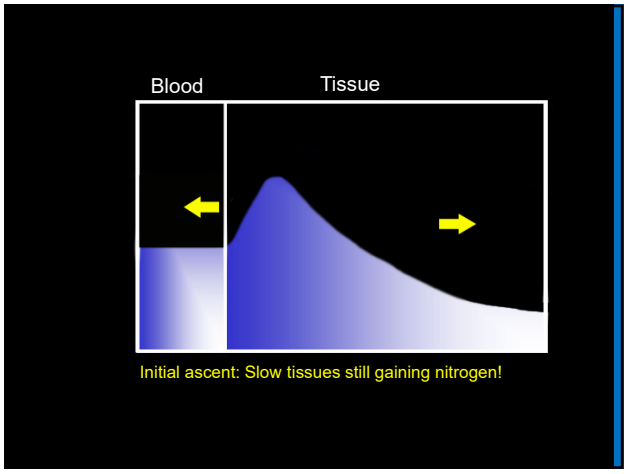
Nitrogen absorption is depth/time dependent

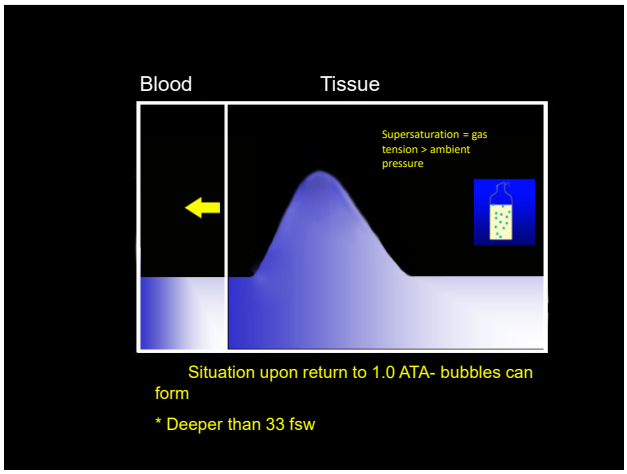








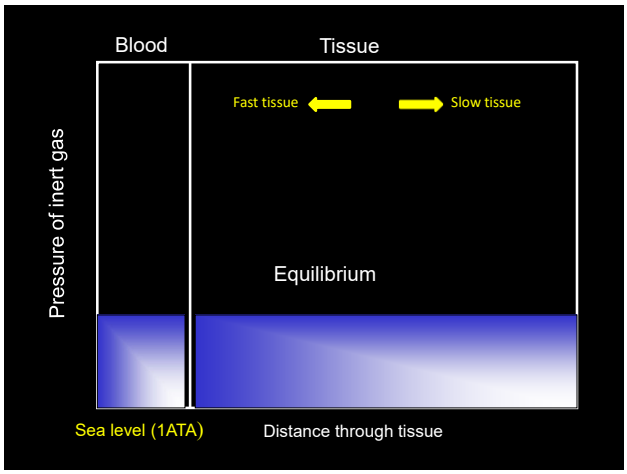




PRIMARY HYPERBARIC TRAINING

Predicting Gas Uptake and Elimination

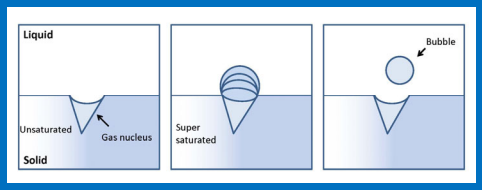
- Technology not available to measure tissue status
- Rely on mathematical algorithms to predict gas exchange
- Fast tissue
  - Lungs
  - Blood
  - Brain
- Slow tissue
  - Ligaments
  - Cartilage
  - Fat



Birth of a Bubble

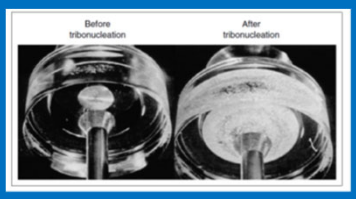
-volume of undissolved gas

- Gas nuclei – *de novo*



Birth of a Bubble

- Bernoulli Cavitation via tribonucleation



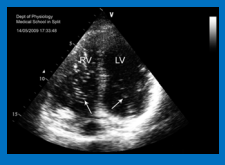
Birth of a Bubble

- Isobaric or inert gas counterdiffusion
  - If the gas that is diffusing into a tissue does so at a rate which exceeds the rate of the other leaving the tissue, it can raise the combined gas concentration in the tissue to a supersaturation sufficient to cause the formation or growth of bubbles

Doppler Studies

Grade	Definition
0	No observable bubbles
1	Occasional bubble
2	At least 1 bubble every 4 heart cycles
3	At least 1 bubbles every heart cycle
4	At least 1 bubble per cm^2 in every image
5	"White-out": single bubbles cannot be discriminated

Grade	No. Subjects	No. with DCS	DCS Incidence
0	110	1	1
1	27	1	4
2	18	3	17
3	17	6	43
4	5	4	80



Bubbles ≠ DCS

Spencer MP Johanson DC: 1974
ONR Tech Report N00014-73-C-0094

BUBBLE EVOLUTION AND EFFECTS

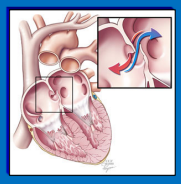
Bubbles form from super saturated dissolved gas

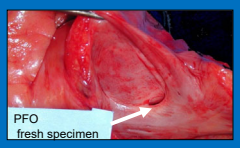
- Extravascular tissues
- Venous blood
  - Shunted
  - Not shunted
    - Filtered by lungs to expired air
      - No obvious clinical harm

BUBBLE EVOLUTION AND EFFECTS

Bubbles form from super saturated dissolved gas

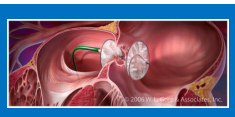
- Extravascular tissues
- Venous blood
  - Shunted — # 1. PFO
  - Not shunted → Filtered by lungs to expired air → No obvious clinical harm

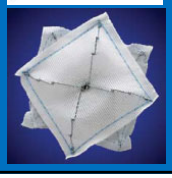




PRIMARY HYPERBARIC TRAINING

Effect of Catheter-Based Patent Foramen Ovale Closure on the Occurrence of Arterial Bubbles in Scuba Divers

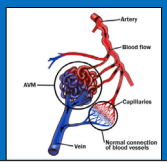


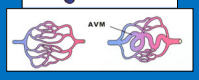


BUBBLE EVOLUTION AND EFFECTS

Bubbles form from super saturated dissolved gas

- Extravascular tissues
- Venous blood
  - Shunted — # 2. AVM
  - Not shunted → Filtered by lungs to expired air → No obvious clinical harm





Used with permission: Mayo Clinic Foundation for Medical Education and Research

BUBBLE EVOLUTION AND EFFECTS

Bubbles form from super saturated dissolved gas

- Extravascular tissues
- Venous blood
  - Shunted → High bubble volume accumulation
  - Not shunted → Filtered by lungs to expired air → No obvious clinical harm

BUBBLE EVOLUTION AND EFFECTS

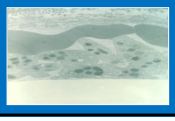
Bubbles form from super saturated dissolved gas

- Extravascular tissues
- Venous blood
  - Shunted → High bubble volume accumulation
  - Not shunted → Filtered by lungs to expired air → No obvious clinical harm

**
Inflammation
Platelet deposition
Platelet-endothelial interactions
Complement activation
Coagulation cascade
Capillary leakage
Endothelial dysfunction
Micro-particles

Cellular/vascular reactions **

Tissue Injury

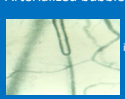


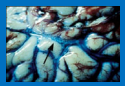
BUBBLE EVOLUTION AND EFFECTS

Bubbles form from super saturated dissolved gas

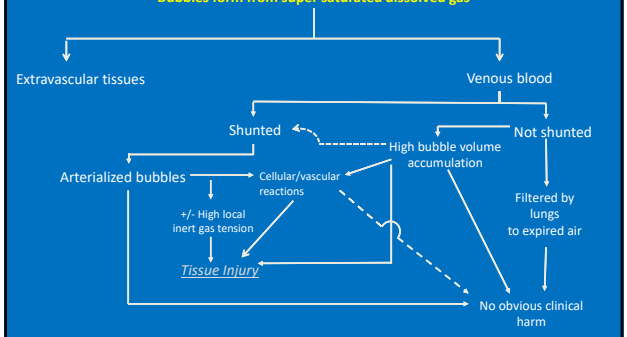
- Extravascular tissues
- Venous blood
  - Shunted → High bubble volume accumulation
    - Arterialized bubbles → Cellular/vascular reactions
    - +/- High local inert gas tension
  - Not shunted → Filtered by lungs to expired air → No obvious clinical harm

Tissue Injury

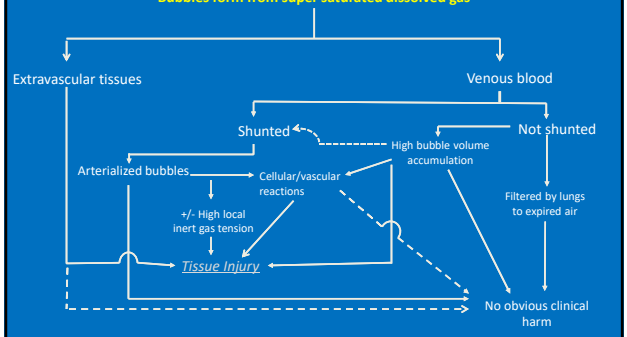




BUBBLE EVOLUTION AND EFFECTS



BUBBLE EVOLUTION AND EFFECTS



Incidence of DCS

- Recreational- 2.0-4.0 per 10,000 person-dives
- Commercial- 1.4-10.3 per 10,000 person-dives
- Scientific – 0.324 per 10,000 person-dives

Controlling Exposure

- Dive computers have supplanted dive tables
- Guidance based on determinants of risk
  - Pressure/time profile
  - Breathing media
- The unknowns
  - Timing and intensity of exercise
  - Thermal stress
  - Predisposition of the diver

At Risk Population






PRIMARY HYPERBARIC TRAINING

Decompression Sickness Risk Factors

the diver…

- Dehydration
- Recent soft tissue injury
- Poor physical (cardiovascular) health
- Obesity
- Increasing age
- History of decompression sickness
- Acute respiratory infection

PRIMARY HYPERBARIC TRAINING

Decompression Sickness Risk Factors

the dive…

- Temperature extremes
- High rates of physical exertion
- Repetitive diving; multiple ascent diving
- Exercise during/post decompression
- Elevated CO2 levels
- Rapid change in inspired inert gas

PRIMARY HYPERBARIC TRAINING

Classification of Symptoms

- Type I DCS (pain only)
- Type II DCS (serious symptoms)
- Type III DCS ? (DCS & CAGE)

PRIMARY HYPERBARIC TRAINING

Presentation-Type I Decompression Sickness

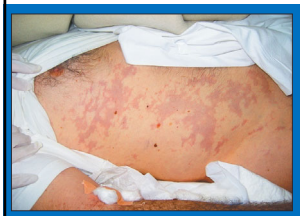
- Pain – on or near a joint(s) often described as an "awareness" of the join
- Fiber-glass like skin irritation
- Pitting edema (>5 hours)

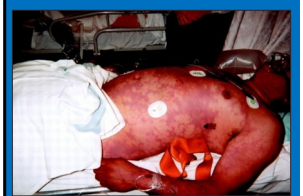
PRIMARY HYPERBARIC TRAINING

Presentation- Type II Decompression Sickness

- Central nervous system injury
- Cardiopulmonary symptoms- AKA the "chokes"; cough can be a bad sign
- Abdominal encircling pain
- Unusual fatigue, post dive
- Skin blotching *'cutis marmorata'* - blotching not itching. ?CAGE

PRIMARY HYPERBARIC TRAINING



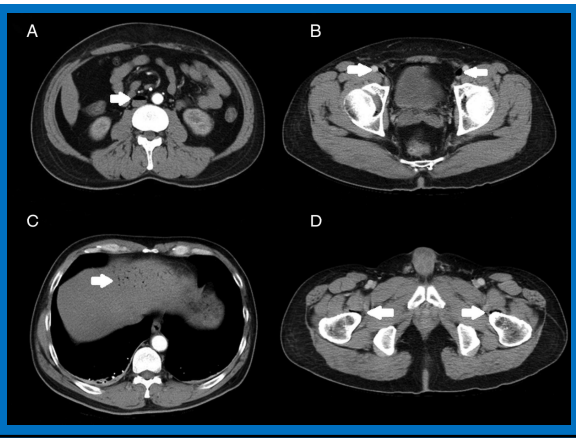


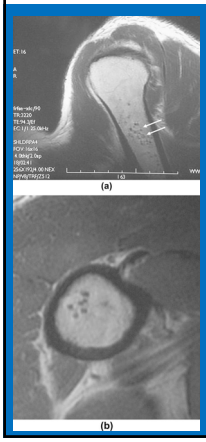
Medical Hypotheses

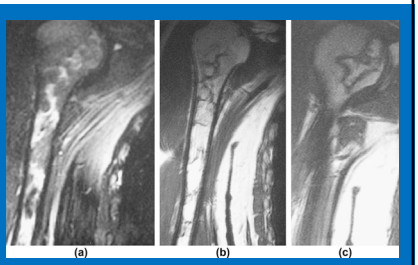
Cutis Marmorata skin decompression sickness is a manifestation of brainstem bubble embolization, not of local skin bubbles

IMAGES IN EMERGENCY MEDICINE

Systemic intravascular and extravascular air bubbles in type I decompression sickness







"The extent of these osteonecrotic lesions suggests that one should treat musculoskeletal DCS at once"

Stephant E, *et al.* 2008
Clinical Radiology 63:1380-1383

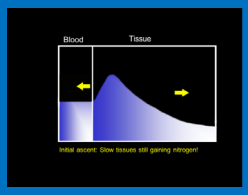
Symptom Onset Time

42% within one hour

60% with three hours

83% within eight hours

98% within 24 hours



USN Diving Manual, 2011

DCS unlikely to develop as new complaint > 24 hour S.I.

Imaging and Decompression Sickness

Chest x-rays may diagnose pneumothorax, pneumo- mediastinum, other pulmonary abnormalities*

*Aspiration; capillary leak syndrome secondary to cardiorespiratory DCS

Consensus Statement
45th UHMS Workshop, 1995

such screening of heightened importance with monoplace chambers

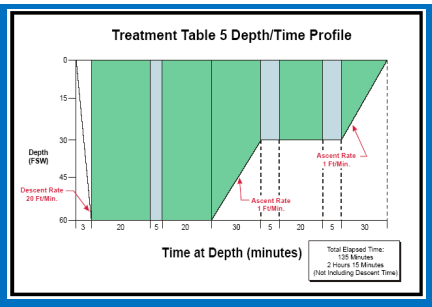
Imaging and Decompression Sickness

CT & MRI not consistently helpful in diagnosis & management

~ they may be indicated to differentiate neurological DCS from conditions of similar presentation

Consensus Statement
45th UHMS Workshop,1995

PRIMARY HYPERBARIC TRAINING

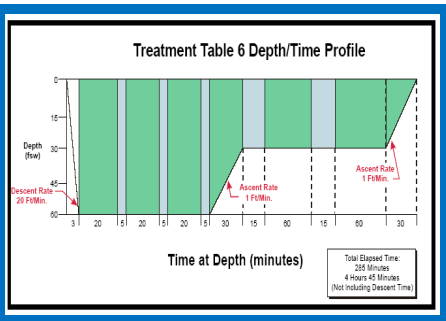


Indication: Type 1 DCS

Complaints c/w Type 1 *Neurologically intact* *Asymptomatic within 10 mins 60'*

Goodman MW Workman RD, Report No. 5-65; USN EDU 1965

PRIMARY HYPERBARIC TRAINING



Indication: Type 2 DCS

Plus Type 1 DCS not asymptomatic within 10 mins 60'

Goodman MW Workman RD, Report No. 5-65; USN EDU 1965

PRIMARY HYPERBARIC TRAINING

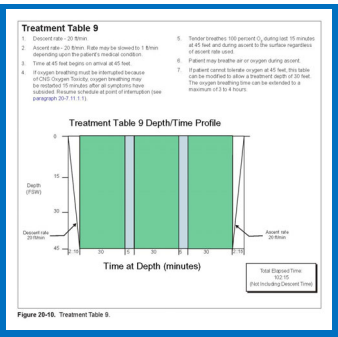


Figure 20-10. Treatment Table 9.

Indication: Incomplete resolution Type 2 DCS

U.S. Navy Diving Manual Revision 7 December 2016

PRIMARY HYPERBARIC TRAINING

054-142

Monoplace hyperbaric chamber use of U.S. Navy Table 6: A 20-year experience.

L. K. WEAVER

Medical Director Hyperbaric Medicine, Co-Director Shock-Trauma-Respiratory ICU, LDS Hospital; Professor of Medicine, University of Utah School of Medicine, Salt Lake City, Utah

INTRODUCTION

METHODS

PRIMARY HYPERBARIC TRAINING

Therapeutic Endpoints

UHMS: Until clinical exam reveals no further improvement in response to HBO

USN: Until no sustained improvement after two consecutive treatments

PRIMARY HYPERBARIC TRAINING

Adjunctive Therapies

Surface Oxygen

As soon as possible

As close to 100% as possible

As long as possible

General agreement that this improves overall outcomes

Adjunctive Therapies

Fluid Resuscitation

Frequently indicated, particularly in acute settings, given findings of:

- *hemoconcentration*
- *platelet and PMNL accumulations*
- *red cell clumping*
- *endothelial injury*

Glucose-free agents, unless hypoglycemia present

Moon RE Sheffield PJ, 1997 Aviat. Space Environ. Med. 68

Adjunctive Therapies

Steroids

Past widespread acceptance in neuronal cases despite little convincing evidence

data supporting reduction of cerebral edema comes from traumatic & vascular brain injuries, not DCS

Moon RE, 2000 SPUMS J; 30(2)

NSAID's

Large *(180pts)* RCT found Tenoxicam significantly reduced number of tx's necessary; no outcomes differences

Bennett MH, *et al.* 2003 UHM: 30(3): 195-205

Adjunctive Therapies

Anti-platelet Drugs

Limited evidence that agents such as aspirin modify platelet action on decompression-induced gaseous emboli

there are more arguments against its use (aggravate inner ear or spinal cord hemorrhage)

Adjunctive Therapies

Lidocaine

Neuro-protection thought afforded through several possible pathways

- *modulates leukocyte activity*
- *anti-inflammatory properties*
- *reduces cerebral metabolism*
- *decelerates of ischemic ion shifts*
- *lowers intracranial pressure*

Mitchell SJ, *et al.* 1999 Ann. Thoracic Surg. 67

Francis J, 2002 SPUMS J; 32 (2)

+1.919.684.9111
emergencies

+1.919.684.2948
general medical requests

Case Presentation

24 yo reports 3 day hx left shoulder pain. Onset secondary to multiplace chamber decompression

Inside tender for CO poisoning case; 2.5 ATA x 90, with numerous compression holds, TBT ~ 110 mins

Symptom Onset: Immediately upon arrival 1.0 ATA, soon associated with LUE weakness

Reported complaints, tx with reassurance

Subsequent History: Pain persisted, weakness remitting/relapsing

Flew to Columbia, SC, 24 hours post-complaint, from Texas, via Atlanta

Exacerbation of pain during flights, improvement on each descent .
Reported complaints two days later

Exam: WDWN muscular male NAD; HEENT WNL; Lungs CTA;
Neuro: cranial nerves II-XII , sensory/motor intact

Impression: Type 1 DCS; resolved Type 2; secondary to omitted stage decompression

Treatment: US Navy TT6; pt asymptomatic second O2 cycle.

Diagnosis

Type 1 & Type 2 decompression sickness, resolved

- *Failure to follow standard decompression protocol*
- *Failure to promptly diagnose*
- *Failure to promptly institute appropriate care*
- *Altitude exposure while symptomatic*

Exposed to hyperbaric environment prior to any formal training

SUMMARY

Depth threshold essentially > 33 fsw

Symptom onset within 24 hs. of decompression

Musculoskeletal, cardiopulmonary & CNS presentations

All medical evacuations/transfers on oxygen

No diagnostic test
~ history of dive, presentation & timing, diff dx, response to pressure

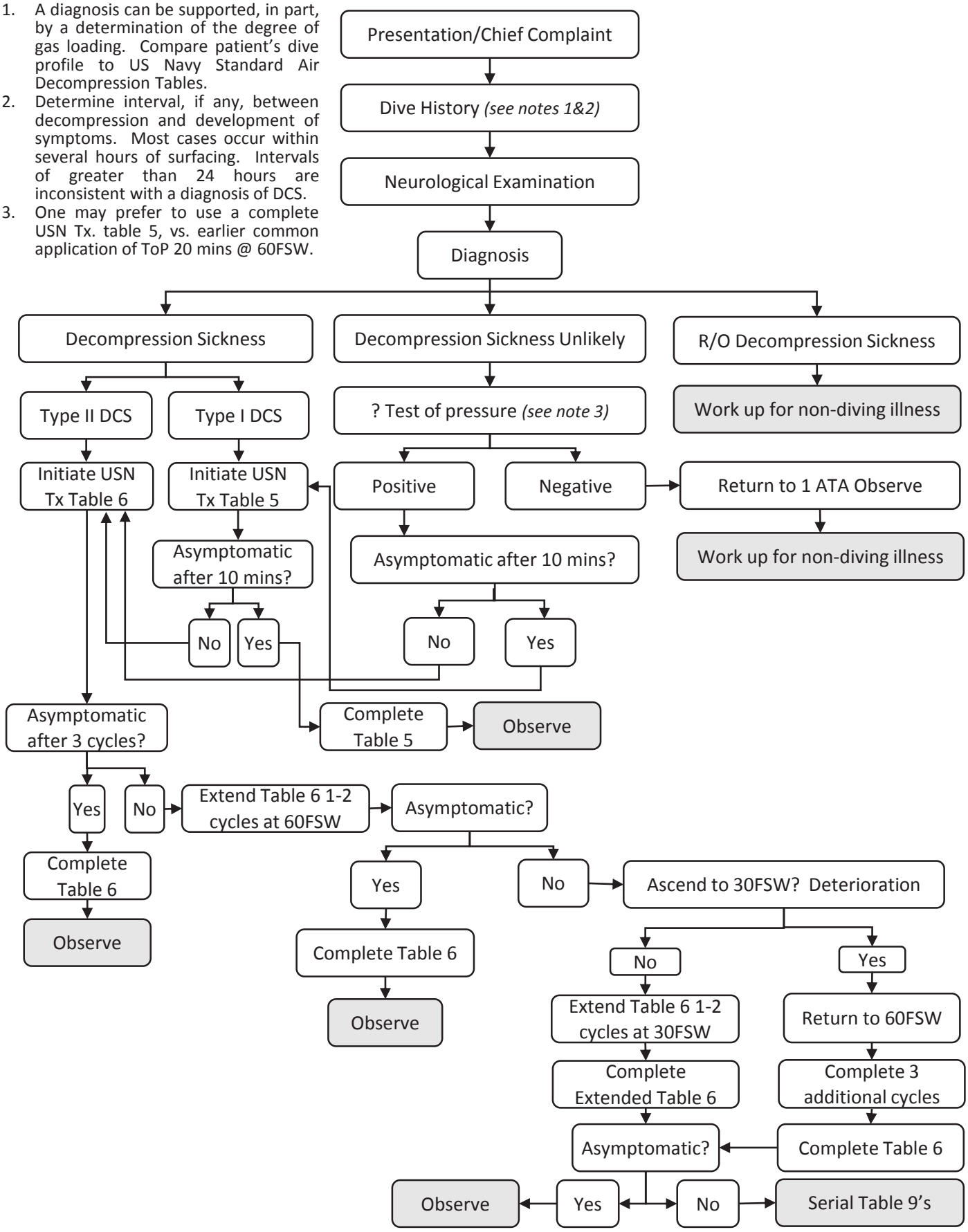
US Navy Treatment Tables 5, 6, & 9

DECOMPRESSION SICKNESS TREATMENT ALGORITHM

Patient presenting at 1 ATA

Notes:

1. A diagnosis can be supported, in part, by a determination of the degree of gas loading. Compare patient's dive profile to US Navy Standard Air Decompression Tables.
2. Determine interval, if any, between decompression and development of symptoms. Most cases occur within several hours of surfacing. Intervals of greater than 24 hours are inconsistent with a diagnosis of DCS.
3. One may prefer to use a complete USN Tx. table 5, vs. earlier common application of ToP 20 mins @ 60FSW.



Discharge Instructions for Decompression Illness (DCI)
After ONE HBO Treatment

- You have been treated for DCI. You should be sensitive to your condition over the next 1-2 days. If symptoms return, or you have any new complaints during this time, you must contact the chamber team at _____ or call the hospital operator to have the team instructed to contact you.

- You should not travel to higher altitude on foot or by airplane or car for at least 3 days.

- You should not scuba dive for at least 30 days, even if you feel well.

  DCI injuries are complex, and not yet fully understood. Injured tissues take time to return to normal, even though you may feel normal.

- When you do return to diving activities, you should be conservative. For example, avoid high risk dives and extend the surface interval time between dives.

- Risk factors that may have contributed to your injury are circled below:

Overweight	Out-of-shape	Increased age
Dehydration	Trouble breathing	After dive exercise
Recent physical injury	Strong ocean currents	Very cold temperature
High gas loading dives	History of DCI	Inexperience
Alcohol consumption	Poor buoyancy control	Multiple ascents
Altitude exposure after dive	Other _____	

- Recommended follow-up: ___ None

  ___ Pulmonary ___Neurology ___Cardiology Other: _____
  ___ No further diving until cleared by a diving medicine trained physician

. . . . . .

The above information has been explained to me, my questions are answered and I understand the instructions.

_____ _____

Patient signature Date/time

Treating Physician: _____ Discharge date/time: _____

Discharge Instructions for Decompression Illness (DCI)
After Multiple HBO Treatments

- You have been treated for DCI. You should be sensitive to your condition over the next 1-2 days. If symptoms return, or you have any new complaints during this time, you must contact the chamber team at _____ or call the hospital operator to have the team instructed to contact you.

- You should not travel to higher altitude on foot or by airplane or car for at least 7 days.

- You should not scuba dive for at least 90 days, even if you feel well.

  DCI injuries are complex, and not yet fully understood. Injured tissues take time to return to normal, even though you may feel normal.

- When you do return to diving activities, you should be conservative. For example, avoid high risk dives and extend the surface interval time between dives.

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Altitude exposure after dive	Other _____	

- Recommended follow-up: ___ None

  ___ Pulmonary ___Neurology ___Cardiology Other: _____
  ___ No further diving until cleared by a diving medicine trained physician

. . . . . .

The above information has been explained to me, my questions are answered and I understand the instructions.

_____ _____
Patient signature Date/time

Treating Physician: _____ Discharge date/time: _____

ADJUNCTIVE THERAPY FOR DECOMPRESSION ILLNESS (DCI): SUMMARY OF UNDERSEA AND HYPERBARIC MEDICAL SOCIETY GUIDELINES DECEMBER 2002

CONTENTS PAGE NUMBER

INTRODUCTION

The Adjunctive Therapy ad hoc Subcommittee was formed in 1998 to investigate and review therapies that could be used in addition to or in lieu of recompression therapy. In 2000 the subcommittee was formally changed to a UHMS standing committee. The Committee goals are (a) to review the available literature on treatment of decompression sickness and gas embolism and make recommendations for therapy based on the best clinical series, case reports, and animal studies available; (b) Place special emphasis in this review on the pre-recompression phase of treatment, which may be prolonged in civilian diving, certain military operations and in space; and (c) Make recommendations for specific animal and human trials that will study the most promising new treatment modalities or otherwise enhance our ability to treat dysbaric disorders.

In addition to experts in diving medicine, the Committee has sought input from clinicians and investigators in the neurosciences who have skills and interest in the treatment and investigation of cerebral and spinal vascular disorders and trauma. The guidelines listed below, patterned on the American Heart Association paradigm, are the result of two formal meetings and considerable electronic discussion. They are intended to be used in the pre-recompression and recompression phases of treatment, but are not intended to replace recompression treatment (currently the treatment of choice). The guidelines will be regularly reviewed and updated. A full printed volume to complement this summary will be available in early 2003.

ACKNOWLEDGEMENT

The UHMS is grateful to the Biomedical Initiatives Steering Committee of the United States Special Operations Command for supporting this effort.

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AMERICAN HEART ASSOCIATION GUIDELINES FOR CLINICAL EFFICACY

Class 1: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective

Class 2: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment

Class 2A: Weight of evidence/opinion is in favor of usefulness/efficacy

Class 2B: Usefulness/efficacy is less well established by evidence/opinion

Class 3: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful

Level of Evidence A: Data derived from multiple randomized clinical trials

Level of Evidence B: Data derived from a single randomized trial or nonrandomized studies

Level of Evidence C: Consensus opinion of experts

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UHMS GUIDELINES FOR ADJUNCTIVE THERAPY OF DCI

Aspirin		
	Class	**Level**
AGE (no significant inert gas load)	2B	C
DCS: pain only/mild	2B	C
DCS: neurological	2B	C
DCS: chokes	2B	C

NSAIDs*		
	Class	**Level**
AGE (no significant inert gas load)	2B	C
DCS: pain only/mild	2B	B
DCS: neurological	2B	B
DCS: chokes	2B	C

* The only evidence thus far available applies to the use of tenoxicam, a nonselective inhibitor of cyclooxygenase (COX). NSAIDs are not currently recommended for use in the field.

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Anticoagulants, Thrombolytics, IIB/IIIA Agents*		
	Class	**Level**
AGE (no significant inert gas load)	2B	C
DCS: pain only/mild	3	C
DCS: neurological	2B	C
DCS: chokes	2B	C
DCS with leg immobility (DVT prophylaxis)	1	A

* Routine therapeutic anticoagulation or use of thrombolytics or IIB/IIIA antiplatelet agents in patients with neurological DCI is not recommended, due to concern about worsening hemorrhage in spinal cord or inner ear decompression illness. Use of these agents may also be risky in combat divers who may be required to return to action after treatment of an episode of DCI.

Low molecular weight heparin (LMWH) is suggested for all patients with inability to walk due to leg weakness caused by neurological DCI. Enoxaparin 30 mg, or its equivalent, subcutaneously every 12 hours, should be started as soon as possible after injury.

If LMWH is contraindicated, elastic stockings or intermittent pneumatic compression are suggested, although their effectiveness at preventing DVT is probably less than LMWH.

Repetitive screening for DVT while withholding anticoagulants until clot is identifiable is a strategy likely to be less efficacious than routine LMWH administration.

These guidelines are extrapolated from observations in patients with traumatic spinal cord injury. Neither the efficacy nor the safety of these guidelines in neurological DCI has been specifically confirmed in patients with DCI. However, deaths have occurred in divers due to documented pulmonary thromboembolism. Furthermore, there is a recognized need for prophylaxis in traumatic spinal cord injury. Thus specific prophylaxis against DVT in spinal cord DCS has been assigned a 1A guideline.

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Surface O_2*		
	Class	**Level**
AGE (no significant inert gas load)	1	C
DCS: pain only/mild	1	C
DCS: neurological	1	C
DCS: chokes	1	C

* 100% O_2 administration can be safely administered for 12 hours with air breaks; thereafter, at the discretion of the receiving physician.

Fluid Therapy*			
	Class		**Level**
AGE (no significant inert gas load)	D5W LR/crystalloid Colloid	3 2B 2B	C
DCS: pain only/mild	D5W LR/crystalloid Colloid	3 1 1	C
DCS: neurological	D5W LR/crystalloid Colloid	3 1 1	C
DCS: chokes	D5W LR/crystalloid Colloid	3 2B 2B	C

* For intravenous administration, lactated Ringer's solution or other glucose-free isotonic crystalloid is suggested, unless otherwise indicated. Patients who have been immersed for prolonged periods may require additional fluid because of immersion-induced diuresis.

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Corticosteroids*		
	Class	**Level**
AGE (no significant inert gas load)	3	C
DCS: pain only/mild	3	C
DCS: neurological	3	C
DCS: chokes	3	C

* Corticosteroids are not recommended for the treatment of decompression illness.

Lidocaine*		
	Class	**Level**
AGE (no significant inert gas load)	2A	B
DCS: pain only/mild	3	C
DCS: neurological	2B	C
DCS: chokes	3	C

* There is insufficient evidence to support the routine use of lidocaine for DCI, and it is not a standard of care. However, if it is to be used, evidence suggests that an appropriate end-point is attainment of a serum concentration suitable for an anti-arrhythmic effect (2-6 milligrams/liter or micrograms/milliliter). Intravenous dosing of 1 mg/kg then subsequent boluses of 0.5 mg/kg every 10 minutes to a total of 3 mg/kg, while infusing continuously at 2-4 mg/minute, will typically produce therapeutic serum concentrations. Use of more than 400 mg within the first hour could be associated with major side effects unless the patient is continuously monitored in a medical unit with the appropriate facilities and personnel. In the field, intramuscular administration of 4-5 mg/kg will typically produce a therapeutic plasma concentration 15 minutes after dosing, lasting for around 90 minutes. Experience with the use of lidocaine in other settings indicates that ataxia and perioral paresthesias are common. More serious toxic effects such as seizures can also occur.

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RESEARCH PRIORITIES FOR INVESTIGATION OF DECOMPRESSION ILLNESS DECEMBER 2002

Human studies

- Development of consensus and guidelines for diagnosis of DCI/severity
- Systematic search for and evaluation of outcome in cases of decompression illness not recompressed, and compare with conventional treatment
- Detailed clinical investigation of fresh serious DCI cases
- Perfluorocarbon trial
- Trial of surface O_2 vs. recompression for pain-only bends
- Anti-platelet therapy trial
- NSAIDs trial
- Lidocaine trial

Animal studies

- Development of small animal model neurological DCI with long term outcome
- Development of large animal model neurological DCI with long term outcome
- Use of acute animal model of neurological DCI to test interventions (e.g. lidocaine, perfluorocarbons, mild hypothermia)
- O_2 toxicity with perfluorocarbons

COMMITTEE PRIORITIES FOR 2003 AND 2004

- Organize and hold a workshop directed toward the development of consistent guidelines for diagnosis of DCI and assessment of its severity (2003)
- Organize and hold a workshop to re-evaluate expected results from human trials currently underway in stroke and head injury (2004)
- Update the adjunctive treatment guidelines in the light of new developments and feedback from the diving medicine community (2003 and 2004)

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CONSENSUS

PATHOPHYSIOLOGY

Decompression illness (DCI) includes both decompression sickness (DCS) and arterial gas embolism (AGE) that may result from a reduction in ambient pressure. While bubble formation, either in situ in tissues (autochthonous) or embolic, is widely accepted as the initiating event in decompression illness, secondary effects have also been described. Focal areas of spinal cord hemorrhage have been observed both in autopsy studies of fatal human DCI and in experimental DCI in animals. The cause of this hemorrhage is not elucidated at this time. It has been suggested that hemorrhage might occur as a result of recompression-induced shrinkage of autochthonous bubbles and leakage of blood from damaged vessels. Other possible causes include bubble-induced compression of venules or extravasation of erythrocytes due to bubble-induced damage to the blood–brain barrier.

The significance of these hemorrhagic areas is unknown. While it has been suggested that they may be one reason for failure to respond to recompression treatment, intraparenchymal blood in the CNS does not necessarily produce clinical effects, and these hemorrhagic areas may merely be a marker of the severity of the disease, rather than a cause of functional impairment.

Humoral mechanisms and activation of coagulation have also been described and are believed to contribute to the pathophysiology of DCI (3–5). There is also evidence that intravascular bubbles can precipitate endothelial adherence of leukocytes in the microcirculation (6,7).

In addition to immediate effects due to mechanical damage or depletion of intracellular energy stores from obstruction of blood flow by bubbles, mechanisms of delayed cell death which have been described in other forms of central nervous system injury may also be important in DCI. If cell death does not immediately occur, reperfusion of ischemic tissue can result in rapid recovery of cellular respiration and ATP synthesis and return of electrical activity, but be followed by increased production of oxygen free radicals leading to lipid peroxidation and other mechanisms of free radical injury. It is hypothesized that there is release of excitatory neurotransmitters such as glutamate, which may then promote neurotoxicity due to facilitation of entry of calcium into cells (8).

CLINICAL PRESENTATION

The spectrum of clinical presentation of DCI has been well described elsewhere (9,10). Individuals with rapid onset of symptoms or severe symptoms necessitate the most urgent and aggressive treatment. In order to facilitate optimal treatment, as well as epidemiological research, the workshop recommended that the following information should be obtained from patients with DCI:

- Presenting symptoms and signs
- Medical history
- Time of onset of symptoms
- Assessment of excess inert gas load (depth and bottom time of recent dives and altitude exposures)
- Breathing gas
- Pattern of evolution before treatment (progressive, spontaneously improving, static, relapsing)
- Response to treatment

DIAGNOSTIC TECHNIQUES

History and Physical Examination

History and physical examination, including neurological exam, should always be obtained, generally prior to initiating hyperbaric treatment. In patients with rapidly progressing symptoms physical examination should be adequate to make the diagnosis, but can be abbreviated in favor of rapid hyperbaric treatment. If symptoms are stable and not progressing the delay incurred by doing a complete neurological examination is not likely to alter the outcome.

Doppler and Ultrasound Imaging

While ultrasound can be used to detect moving intravascular bubbles, there is a poor correlation between the presence of such bubbles and DCI. Venous bubbles can frequently be detected in divers without symptoms, and divers without detectable bubbles can have DCI. The presence of venous bubbles without symptoms is not considered an indication for recompression treatment. Therefore these ultrasound techniques are not considered useful in the evaluation or treatment of DCI.

Electrophysiology

Electronystagmography (ENG) and audiography may be useful in distinguishing inner ear decompression sickness from inner ear barotrauma, for which the treatment is quite different. Inner ear DCI requires recompression, while inner ear barotrauma is managed with bed rest, avoidance of straining, and possibly surgical intervention. However, in the event the distinction cannot be made, and

it is felt that compression therapy may be of benefit, it should be carried out using appropriate precautions. These include tympanostomy, keeping the head elevated, and avoidance of conditions which may cause large excursions in CSF pressure (e.g., Valsalva maneuvers, straining, and breathing from a mask in demand mode requiring large pressure fluctuations vs. a free flow mask). When the clinical evidence is most compatible with the diagnosis of inner ear decompression illness, and hyperbaric therapy is planned, ENG can be performed after treatment.

Electroencephalography (EEG), somatosensory evoked potentials (SSEP), brainstem, auditory evoked responses (BAER), and visual evoked potentials have been discussed elsewhere (9,11) and are not generally useful in the evaluation and treatment of decompression illness.

Imaging

Plain chest radiography can be used to diagnose pneumothorax, pneumomediastinum, or the pulmonary abnormalites associated with aspiration or the capillary leak syndrome associated with cardiorespiratory decompression illness ("chokes"). In most cases it is not necessary to postpone recompression treatment in order to obtain radiographic studies. If pneumothorax is suspected, a chest radiograph can be helpful in making the decision to insert a chest tube.

While computed tomography (CT) and magnetic resonance imaging (MRI) of the brain can occasionally demonstrate abnormalities in cerebral decompression illness, these modalities have not been demonstrated to be helpful in the management of DCI. MRI of the spinal cord is insensitive for the detection of spinal cord DCI and has no present role in its management. CT and MRI may be useful to differentiate neurological DCI from other conditions with similar onset and clinical presentation such as intracranial or spinal hemorrhage. However, in general recompression therapy should not be delayed to perform a diagnostic imaging study unless there is strong clinical evidence indicating that a diagnosis other than DCI must be ruled out.

Examples of emission tomography (SPECT, PET) in patients with neurological DCI were presented at the workshop. While it has been suggested that abnormalities observed on SPECT imaging can be used to confirm the existence of CNS pathology in DCI, this hypothesis is not proven. Published evidence has indicated that ^{18}F-2-deoxyglucose PET, a technique with higher resolution than SPECT, is less sensitive than clinical evaluation in detecting abnormalities in neurological DCI (12). SPECT and PET scanning technology requires rigorous quality control for accurate image acquisition, processing, display, and interpretation. False positive readings can occur if artifacts or minor acceptable variations in tracer distribution are not recognized. Any statistically valid study incorporating emission tomography should include acquisition of images under carefully controlled conditions and interpretation of the images in a blinded fashion by two or more experienced radiographers or nuclear medicine physicians. Use of both SPECT and PET in the evaluation and treatment of DCI is presently investigational.

Neuropsychological Testing

Evidence was presented that neuropsychological abnormalities may be detected in neurological DCI (13). However, neuropsychological tests can be subject to artifacts related to the patient's fatigue, motivation, anxiety, and learning effects from extended testing. Moreover, administration and interpretation of neuropsychological tests require an experienced clinician. Further investigation is needed before routine administration of neuropsychological tests can be recommended. However, in the hands of experienced individuals neuropsychological testing may be a valuable adjunct in determining patient response to treatment and the need for follow-up treatments.

Laboratory Investigations

Measurement of hematocrit and urine specific gravity may be clinically useful for the quantification of dehydration. Hypoglycemia in a diabetic could be confused with cerebral DCI, and hyperglycemia can worsen CNS injury. Thus measurement of plasma glucose and electrolytes may be clinically indicated. Several participants reported instances of laboratory evidence of recreational (illicit) drug use in patients with DCI. Such drug ingestion may cause or contribute to altered mental status. Analysis of blood and urine for evidence of recreational drug use may provide clinically useful information in assessing any patient with altered mental status, and a patient with DCI should not be an exception. Elevated creatine phosphokinase (CPK) has been described in arterial gas embolism (14), suggesting that, in the collection of epidemiological data, CPK measurement might help to differentiate between AGE and DCS.

As a general principle, treatment of DCI with recompression should be administered as quickly as possible. Hyperbaric oxygen therapy should be delayed for a diagnostic test only if the results of that test might affect the therapy.

Initial Treatment Prior to Recompression

Patient Positioning

Head down position was recommended in the past for emergency treatment of AGE in order to prevent further embolization and to augment the clearance of bubbles impacted in cerebral arteries. There is little evidence that buoyancy plays any role in the distribution of arterial bubbles, thus head down position is unlikely to affect the likelihood of further embolization. In an experimental animal preparation arterial bubbles impacted in cerebral vessels allowing blood to displace air (15). However, head down position can augment cerebral edema (15,16). Appropriate positioning of the patient was discussed in a previous workshop, in which the consensus was that while a brief period of head down position might facilitate the clearance of air bubbles from the cerebral circulation after AGE, patients with DCI should be kept in a supine position (17) unless head down position is required to maintain an adequate blood pressure. To minimize the risk of aspiration of vomitus, unconscious patients who do not have airway protection by an endotracheal tube should be maintained in the lateral decubitus position.

Oxygen

Early administration of supplemental oxygen at the surface is beneficial for treating DCI. The optimal inspired concentration of oxygen is 100%. It was noted that in epidemiological surveys of recreational divers that less than one third of symptomatic divers received any supplemental oxygen, and less than 10% of symptomatic divers are administered concentrations approaching 100% (via a demand valve and tightly fitting mask). One can achieve 100% inspired oxygen via a tightly fitting aviator's mask attached to a demand regulator, DAN oxygen kit, closed or semi-closed rebreathing circuit, or a simple adaptation of an emergency oxygen delivery system by attaching a snorkel mouthpiece to an Elder valve. If such equipment is not available, supplemental oxygen can be administered via a loose fitting hospital mask. It is recommended that the need for inspiration of pure oxygen be emphasized among recreational divers and that equipment capable of delivering 100% oxygen, and training in its use, be more widely available.

The minimum duration of surface oxygen breathing is unknown but ideally it should be continued during the entire time required to transport the individual to a recompression facility.

Fluids

Intravenous fluid administration is a useful initial treatment. CNS injury may be exacerbated by hyperglycemia. Therefore, unless hypoglycemia is present, glucose-free fluids are recommended. CNS edema can result from hypotonic fluid administration. Thus, isotonic crystalloid fluids such as normal saline (sodium 154 mEq, chloride 154 mEq), Ringer's solution, Normosol-R (Abbott Laboratories, Abbott Park, IL 60064, USA: sodium 140 mEq, potassium 5 mEq, magnesium 3 mEq, chloride 98 mEq, acetate 27 mEq, gluconate 23 mEq), or similar solutions are recommended. There is no current evidence suggesting that colloidal fluids such as hetastarch or albumin solutions have any advantage over crystalloids for fluid resuscitation of patients with DCI. Although controlled studies of different rates of fluid administration have not been published, for a mildly dehydrated patient experience in other branches of medicine suggests that in the first hour 1,000 ml of intravenous fluid is a reasonable intake, followed by a constant infusion of twice the calculated maintenance requirement (approximately 1.5 ml $\cdot$ kg^{-1} $\cdot$ hour^{-1}). For severe dehydration or patients in shock (18), additional fluids should be titrated in order to normalize blood pressure and heart rate, and provide a urine output of 1–2 ml $\cdot$ kg^{-1} $\cdot$ hour^{-1}.

Oral fluids may also contribute to successful outcome. However, giving oral fluids necessitates interruption of oxygen administration and may contribute to vomiting in some individuals. Commonly available oral fluids (e.g., many carbonated beverages) also contain high glucose concentrations, which not only may contribute to hyperglycemia, but may also slow gastric emptying and delay the absorption of fluid. Such fluids should be avoided in patients with neurological involvement. Plain water has been suggested by some participants, however it can induce a reduction in plasma osmolality, inhibiting the secretion of antidiuretic hormone (ADH) and due to the increased urine output which is out of proportion to the increase in plasma volume may give a false impression of adequate rehydration (19,20). An ideal solution for oral rehydration in other clinical situations (e.g., diarrhea) been suggested as containing approximately 30–60 m*M* sodium, 70–150 m*M* glucose, and osmolality of around 240 mOsm/kg (21,22).

Some commonly available beverages have appropriate glucose and electrolyte concentrations. One way of preparing an appropriate solution is to mix one part orange or apple juice with two parts water and adding half teaspoon salt to 1 liter of the mixture (35 fluid ounces). Individual judgment must be used in administering oral fluids. Provided the patient is conscious and not nauseated or

vomiting, 1,000 ml or more of fluid can be administered in the first hour, with subsequent fluid intake as needed to a urine output of 1–2 ml · kg^{-1} · $hour^{-1}$.

Hyperbaric treatment should not be delayed in order to administer fluids. Intravenous access can be established after the start of recompression. Alternatively, oral fluids can be administered during air breaks.

Pharmacotherapy

There is evidence from a prospective clinical trial that early intravenous administration of high dose (insert dosing regimen) methylprednisolone (MP) to patients with spinal cord trauma improves long-term neurological outcome (23). There are no similar data for individuals with DCI. Use of MP in this manner can be considered an option, although its use for this indication is still investigational. There are no current data examining the propensity of high dose MP to alter the threshold for pulmonary or CNS oxygen toxicity or for masking their early symptoms.

Studies of arterial gas embolism in experimental animals (24–27) and a case report of DCI (28) suggest a possible beneficial effect of intravenous lidocaine. Since a randomized human trial is ongoing, no specific recommendation is made.

No specific recommendation is made regarding the use of anti-platelet agents such as aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs). Since publication of the previous workshop (17), no new evidence has been published on their possible contribution to DCI treatment. They can be effective in treating the pain of DCI, thus if they are used before or during recompression, assessment of the effect of hyperbaric treatment can be confounded.

Similarly, there is no evidence that heparin or other anticoagulants are beneficial in treatment of the primary manifestations of DCI. However, patients with leg weakness due to DCI may be at risk of deep venous thrombosis and fatal pulmonary thromboembolism (29). Therefore, some anti-DVT regimen, which could include low dose heparin, is recommended for such patients.

Adjunctive agents worthy of investigation in DCI include specific glutamate receptor antagonists such as *N*-methyl D-aspartate (NMDA) receptor antagonists, a-amino-3-hydoxy-5-methyl-4-isoxazole propionate (AMPA) receptor antagonists, glycine receptor antagonists, 1-aminocyclopentyl-trans-1,3-dicarboxylic acid (t-ACPD) receptor antagonists, and free radical scavengers. A clinical trial of tirilazad, a 6-amino steroid ("lazaroid") in CNS injury is in progress (30). Thus far, use of calcium channel blockers such as nimodipine, in cerebral injury models other than DCI, has been disappointing. Workshop participants are unaware of any human or animal trials of such agents in DCI.

Thermal Control

Decompression illness can be complicated by hypothermia, requiring rewarming of the diver. Conversely, hyperthermia has been shown in animal models of CNS injury to worsen neurological outcome (31). Thus, fever should be aggressively treated and chamber temperature controlled to comfortable levels whenever possible.

DEFINITIVE TREATMENT: ALTITUDE DCS

The following algorithm has been used effectively by the US Air Force:

Symptoms that clear on descent to ground level with normal neurological exam

One-hundred percent oxygen by tightly fitted mask for 2 hours minimum; aggressive oral hydration; observe 24 hours.

Symptoms that persist after return to ground level or occur at ground level

One-hundred percent oxygen; aggressive hydration; hyperbaric treatment using USN Treatment Tables 5 or 6, as appropriate. For individuals with symptoms consisting only of limb pain, 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 symptoms of DCS, including neurological symptoms, "chokes," hypotension, or symptoms that progress in intensity despite oxygen therapy: continue 100% oxygen; administer intravenous hydration; initiate immediate hyperbaric therapy using USN Treatment Table 6. Oxygen should be continued during transport to a recompression chamber.

Any recurrence of symptoms

Restart 100% oxygen; initiate hyperbaric therapy (even if symptoms resolve on surface oxygen). Oxygen should be continued during transport to a chamber.

Guidelines for use of USN Treatment Table 5 in treatment of altitude DCS

All three of the following must apply: 1) Symptoms consisting only of joint pain (normal neurological exam before treatment); 2) onset of symptoms within 6 hours of return to ground level; 3) symptoms must resolve within 10 minutes of reaching 60 fsw (18 msw). *Note*: USN Treatment Table 6 is used in all other cases. In Canadian

experience USN Treatment Table 5 has been associated with an unacceptably high recurrence rate and has resulted in a preference for USN Treatment Table 6 in all cases requiring recompression.

It must be emphasized that these guidelines apply only to DCS experienced during de novo altitude exposure without a pre-exposure dive. If a preflight dive has been made, the case must be managed as a diving-related DCI.

Experience in treating altitude DCS in experimental subjects who have pre-breathed 100% oxygen prior to altitude exposure suggests that, for symptoms that resolve during descent, one hour of post-exposure 100% oxygen breathing may be adequate.

For DCI occurring in space during extravehicular activity (EVA) the treatment protocol calls for recompression to initial cabin pressure and administration of oxygen. Although the procedure works well for altitude DCI as described above, since there have been no reports of EVA-related DCI during space flight in either the US or Russian space programs, the efficacy of this treatment in space is unknown.

Return to flying

Patients whose symptoms resolve, and in whom the exam is normal, can return to flying in 72 hours.

DEFINITIVE TREATMENT: DIVING RELATED DCI

Pressure and Oxygen

As recommended in previous workshops (1,2), definitive treatment of diving related DCI incorporates compression and administration of breathing gas with elevated partial pressures of oxygen. Rapid administration of pressure and oxygen is paramount, particularly in severe disease that manifests shortly after surfacing.

A wide variety of initial hyperbaric regimens have been described (Table 1). Current treatment options differ in level of treatment pressure, time under pressure, partial pressure of oxygen, and the diluent gas.

At present the apparent differences in protocol and procedures between different hyperbaric centers may be a result of their local circumstances (such as the availability of trained staff), the local style of diving (deep occasional or shallow multi-day) leading to different presentations and, perhaps most important, the immediacy of or delay before recompression (contrast delays greater than 12 hours in some locations with the requirement to have a chamber on the site in some naval and commercial diving).

There are no human outcome data obtained in prospective, randomized studies comparing the various regimens. However, the following principles are agreed upon:

- Complete resolution is most likely to result from early hyperbaric treatment.
- Since their introduction in 1965 the US Navy oxygen treatment tables with initial recompression to 60 fsw (18 msw) have been the most widely used and studied recompression procedures for DCI treatment beginning at the surface. Other procedures such as the ones used by the Royal Navy and COMEX follow the same general principles of pressure and oxygen breathing. Review of the effectiveness of USN oxygen treatment tables shows a high degree of success in resolving symptoms if the delay to treatment is not excessive (32). For symptoms occurring after surfacing from a dive, the "gold standards" against which other treatment tables are compared are USN tables, and the closely related RN procedures.
- The use of tables with a shallower or deeper initial treatment depth should be reserved for facilities and personnel with the experience, expertise and hardware necessary to deal with untoward responses.
- Under certain circumstances symptoms treated immediately may respond to pressure alone (e.g., air embolism and other blow-ups, and symptoms arising in the course of planned decompression).
- While there is an inverse relationship between delay to treatment and complete resolution of symptoms, the data currently available have not established a maximum time (hours or days) after which recompression is ineffective. In the absence of an altitude exposure, the onset of symptoms greater than 24 hours after a dive are unlikely to be caused by DCI (10).
- Although administration of surface oxygen often resolves symptoms, they frequently recur after cessation of oxygen breathing. Most participants agreed that for diving related DCI surface oxygen breathing is not a substitute for hyperbaric treatment.

OPTIMUM P_{O_2}: Animal data suggest that the optimal inspired P_{O_2} is between 2 and 3 atmospheres absolute (atm abs) (33–35). One report of human DCI based upon a retrospective review of recurrence rates after follow-up treatments suggests that 100% O_2 administration at 60 fsw (18 msw, P_{O_2} = 2.8 atm abs) is superior to 46 fsw (14 msw, P_{O_2} = 2.4 atm abs) (36). Although some participants in this workshop suggested that the optimal inspired P_{O_2} may be lower than 2.8 atm abs, no new data have been published in the literature or were presented at the workshop.

EXTENDED INITIAL TREATMENT: Patients who fail to respond adequately to the initial recompression, or to exhibit significant neurological deterioration during decompression, can be managed in several ways (11). In

Table 1: Treatment regimens that are currently being used for the initial treatment and medical management of DCI

- Ground level (1 atm abs) oxygen for treatment of altitude-induced DCO (e.g., USAF treatment guidelines)
- Pressurization to 40 fsw (12 msw) using oxygen (e.g., COMEX Treatment Table 12)
- Pressurization to 45 fsw (14 msw) using oxygen.
- Pressurization to 60 fsw (18 msw) using oxygen (e.g., USN Treatment Tables 5, 6)
- Pressurization to 100 fsw (30 msw) using heliox or nitrox (e.g., COMEX Treatment Table 30)
- Presserization to 100 fsw (30 msw) using nitrox saturation [e.g., as described by Miller et al. (61)]
- Pressurization to 165 fsw (50 msw) using either air or nitrox then decompressing according to USN Treatment Table 4, 6A, or 8 (44), RN Treatment Table 71 (43), ECOSystems Treatment Table 7A (39), or a modification of USN Treatment Table 6A (41)
- Pressurization to 200–225 fsw (60–68 msw) using either air or nitrox (e.g., Hawaiian tables), air, nitrox, or heliox [USN Treatment Table 8 (44), RN Treatment Table 71 (43), ECOSystems Treatment Table 7A (39)]
- Pressurization to depth of relief using heliox (e.g., one of the tables listed above or saturation decompression)

patients with severe neurological injury, lack of improvement or deterioration at 60 fsw (18 msw) should prompt strong consideration of further compression to 100 fsw (30 msw), 165 fsw (50 msw) or deeper, depending upon the patient's response and hardware, life support and logistic consideration.

For non-life-threatening situations, when the first recompression has been immediate, there is also a case for a more aggressive treatment than a full USN Treatment Table 6 with extensions. Not all such residua will respond well to surfacing, then administration of repetitive daily hyperbaric oxygen treatments. For a working diver any residuum could mean the end of his or her career and a more aggressive initial therapy may be appropriate.

The following options are available.

- Administer additional oxygen-breathing cycles at the initial treatment pressure and decompress according to a modification of the standard treatment table [e.g., extended USN Treatment Table 6, Catalina Treatment Table (37,38)].
- Initiate saturation treatment at the initial treatment pressure. This is usually reserved for patients with DCI that is life threatening or causing severe neurological impairment. A widely disseminated table with a saturation depth of 60 fsw (18 msw) and air as the chamber atmosphere is USN Treatment Table 7.
- Compress to greater pressure [e.g., 100 fsw (30 msw), 165 fsw (50 msw)] and initiate a different treatment table [e.g., ECOSystems Treatment Table 7A (39), USN Treatment Table 4, RN Table 71] or consider saturation treatment. Both saturation treatment and prolonged subsaturation treatments such as ECOSystems Treatment Table 7A require sophisticated chamber monitoring and life support capabilities, and should only be considered if the chamber facility and personnel have the necessary equipment and experience to deal with the additional complexities of this mode of treatment
- Increase treatment depth and change the breathing gas (e.g., compress to 100 fsw, 30 msw, and switch to 50-50 helium–oxygen or nitrogen–oxygen and decompress according to COMEX Treatment Table 30).

The particular option chosen should also reflect the clinical condition and the magnitude of any deterioration, for example, a significant increase in motor weakness *vs.* minor sensory impairment.

If the chamber facility permits, deeper compression was suggested by several participants. While a published review (40) provides little support for it, one retrospective comparison (41) and many workshop participants reported successful outcomes with deeper recompression. This may particularly be the case after deeper dives (42), perhaps because of higher tissue inert gas loads, allowing ongoing evolution of bubbles at shallow recompression depths. There is less experience with the use of higher pressures to treat recreational divers, in whom inert gas burdens are usually less, and for whom treatment delays are considerable (10). The other available options are equally supportable.

Specific recommendations for treatment of commercial and military divers have been published elsewhere (39, 42–45).

Inert Breathing Gas

For surface supplied air and scuba divers, the principal treatment gases are oxygen and air. For treatment depths greater than 60 fsw (18 msw), enriched nitrogen–oxygen (nitrox) or helium–oxygen (heliox) may also be used.

There is general agreement that helium is the preferred inert gas for all treatments deeper than 200–220 fsw (60–67 msw). Data on the possible advantages of using heliox to for treatment of DCI in air divers are inconclusive and controversial. Animal data have demonstrated that heliox breathing may initially accelerate bubble shrinkage when administered at the surface, and may be superior to 100% oxygen under these conditions (46–49). However, data

presented at this workshop showed that when combined with recompression 100% oxygen breathing shrank bubbles more rapidly than a breathing mix of 50% helium–50% oxygen. In a guinea pig model of severe DCI no benefit of heliox administration was been observed (50).

Anecdotal evidence in the literature (51–53) and from participants in the workshop, has shown that heliox may be at least as effective as nitrox in treating human DCI. There are no reports of human DCI in which heliox administration caused deterioration or other adverse effects. Heliox has an advantage over nitrox for deep recompression, in that nitrogen narcosis is avoided. There is evidence that switching breathing gases from air to heliox while at depth can produce bubbles due to isobaric gas supersaturation (54,55) However, there is no evidence that heliox breathing after an air dive causes bubble formation or growth if it is associated with recompression treatment.

The majority of DCI experienced by heliox divers appears to respond well to recompression while breathing air–nitrox–oxygen (56). A problem can occur if there is no oxygen breathing period between helium breathing and air recompression. There is anecdotal evidence that for some divers who have breathed heliox all the way to the surface, heliox is the preferred treatment gas (57). Otherwise, as yet there is no evidence that heliox is superior to oxygen or nitrox mixes in treating DCI.

The effectiveness of using air and oxygen for the treatment of DCI in air divers has been well established by years of clinical experience and published series. Clinical trials are underway to see if helium–oxygen mixes are superior to standard air, oxygen, and nitrox mixes in the treatment of air DCI. Until these trials are completed and evaluated, and clear procedures for the use of heliox established, except for facilities in which these are in place and there is the necessary experience and expertise, or in the setting of an experimental trial, nitrogen–oxygen and 100% oxygen are recommended as the breathing gases.

In-Water Recompression

In-water recompression with oxygen breathing has been used in remote areas of Australia. There is evidence that, when used promptly under carefully controlled conditions (maximum depth of 30 fsw (9 m), oxygen administered via tethered line and full face mask, thermal protection suit, buddy present, and ascent at 12 min/m), it can be both safe and effective. However, its effectiveness in comparison with standard recompression techniques has not been assessed. In-water recompression is not recommended in areas where adequate hyperbaric chambers are available.

The risk of hypothermia in cold water may be ameliorated by adequate thermal insulation of the diver, however without appropriate thermal protection equipment it must be emphasized that there is a substantial risk of hypothermia using this technique. In general, in-water recompression is best used in warm tropical waters.

Follow-up ("Tailing") Hyperbaric Oxygen Treatments

Patients with residual signs or symptoms after the initial hyperbaric treatment table have obtained further clinical improvement through repetitive hyperbaric oxygen treatments. It is therefore recommended that follow-up hyperbaric oxygen treatments be continued, based upon clearly documented symptoms and especially physical findings, until no further sustained stepwise improvement occurs. Usually no significant improvement on two consecutive treatments will establish this. It is not recommended that repetitive treatment be based upon changes in electrophysiological or imaging studies. Pain and paresthesias which wax and wane usually resolve spontaneously and do not require prolonged follow up treatments. 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 in recreational divers in the DAN database (58) supports the efficacy of no more than 5–10 repetitive treatments for most individuals. Many diving physicians stated that three or four treatments are sufficient for most cases.

RETURN TO DIVING AFTER DCI: This issue was not specifically discussed at the workshop. Guidelines have been published elsewhere (43,44,59).

FUTURE DIRECTIONS

Although there is great variation and strong opinions on the optimal treatment method, there are no published randomized trials comparing one treatment protocol with another. The need for such trials of both recompression protocols and adjuvant treatments was strongly supported by the workshop participants.

Better education of the diving community is required in order to promote early recognition of DCI and to implement administration of 100% oxygen at the dive site and during transport, and early definitive treatment.

Careful postmortem examination of spinal cord and brain in diving fatalities is recommended.

Experience with transportable chambers has been published, demonstrating that evacuation and transportation of an injured diver under pressure to a multiplace facility from a remote dive site is both feasible and effective (60). With the recent advances in transportable

chambers it would be appropriate to investigate the feasibility of making transportable chambers available on a wider scale, along with the necessary operator training, for dive sites where chamber coverage is inadequate.

Because of the circumstances under which a transportable chamber might be used, there may be a greater probability that the treatment table would have to interrupted and decompression instituted emergently. At 60 fsw (18 msw) equivalent depth this may engender some risk of inducing decompression illness for an inside tender. A treatment depth shallower than 60 fsw (18 msw) may permit direct decompression to the surface without incurring a requirement for decompression stops.

The new, lighter transportable chamber could be used to administer an immediate 100% oxygen treatment at the dive site and would provide a solution for those diving hours or days away from a definitive hyperbaric center. If necessary, the chamber could also transport a patient while either under pressure or, to avoid the effects of altitude, maintaining ground level pressure.

Unanswered Questions

1. Determination of the factors which lead to incomplete resolution of symptoms after the initial hyperbaric treatment.
2. Elucidation of the role of intramedullary hemorrhage in spinal cord DCI.
3. Agreement on a uniform database for collection of epidemiological data.
4. Development of a clinical classification system which could be used to determine optimum treatment protocol and prognosis, and entry criteria into therapeutic trials.
5. Investigation of adjunctive pharmacotherapy.
6. Determination of the circumstances (if any) under which oxygen at ambient pressure provides sufficient treatment of diving related DCI.
7. Determination of the optimum hydrostatic pressure, oxygen partial pressure and the best diluent breathing gas (when treatment depth exceeds 60 fsw, 18 msw) for resolution of bubbles and clinical signs of DCI: a) for the first treatment; b) for repetitive subsequent treatments.
8. Determination of the relationship between treatment delay and ultimate outcome.
9. Development of a method of coordinating multicenter trials to obtain enough data to make sound judgments regarding the efficacy of various treatment regimens.
10. Development of an animal model of DCI in which long-term functional outcome can be assessed.
11. Selection and validation of a battery of neuropsychological tests which can be conveniently administered in a chamber in order to assess cortical dysfunction due to DCI.
12. Determination of the feasibility of deploying and using civilian transportable chambers at remote dive sites, and the necessary procedures for administering recompression treatment without on-site medical supervision.
13. Promotion of the collection of brain and spinal cord pathology in divers who die.
14. Determination of the relationship between intravascular gas bubbles and tissue damage.
15. Determination of the mechanisms of tissue damage.
16. Investigation of brain imaging technology to determine whether there is a role for such techniques in the assessment of both acute and chronic residual injury due to DCI.

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