

# **Decompression Sickness**

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**Gregory Barefoot, PA-C**

PRIMARY HYPERBARIC TRAINING

# Decompression Sickness

Primary Training in Hyperbaric Medicine

Greg Barefoot, PA-C

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ON PROBABILITY  
*at the end..*  
*Whether a bubble*  
*Causes trouble*  
*Or dwindles in size*  
*Until its demise*  
*is ruled by chance*  
*- A happenstance*

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## 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

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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.

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## What is Decompression Sickness?

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

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## What is Decompression Sickness?

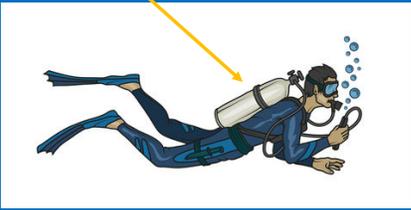
- Uncontrolled release of **gas** from tissues during or after surfacing with inadequate time for equilibration

# NITROGEN

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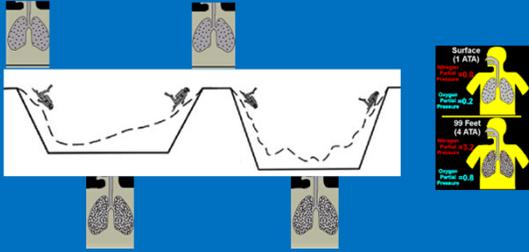
### Breathing Gas

- Compressed air
  - Oxygen - 21%
  - Nitrogen- 78%
  - Trace gases – 1%



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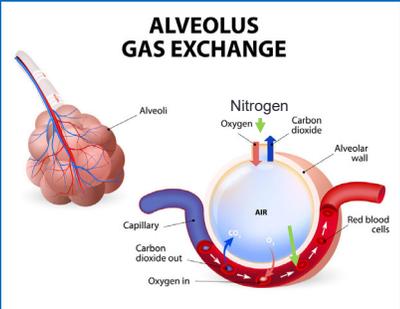
### Nitrogen Absorption



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### Nitrogen Uptake

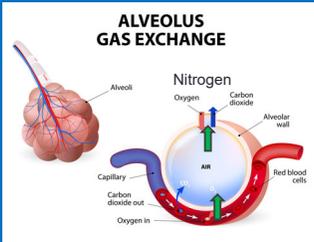
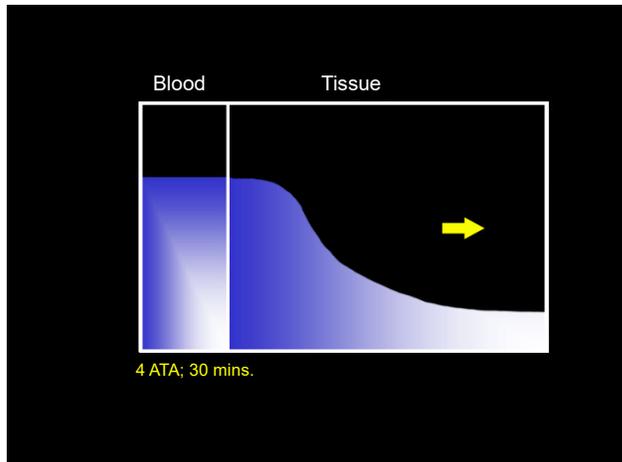
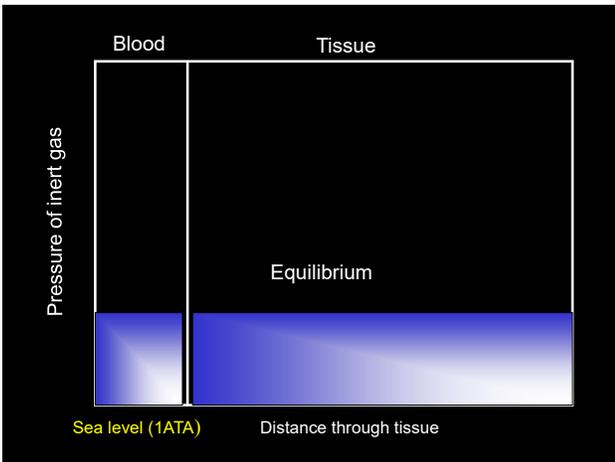
- AKA- "On gassing"
- Dependent upon
  - Time
  - Pressure
  - Diffusion
  - Perfusion
  - Solubility

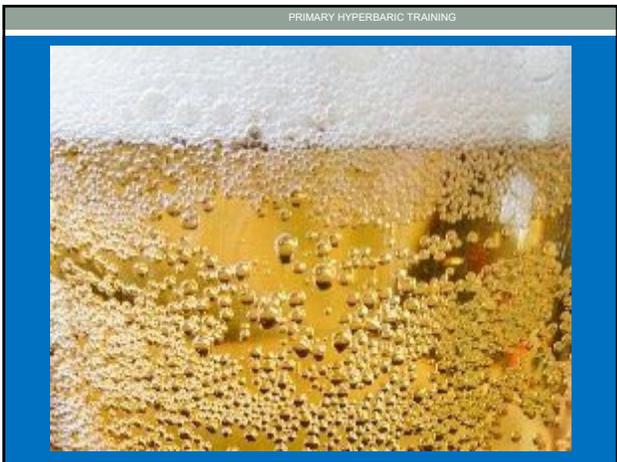
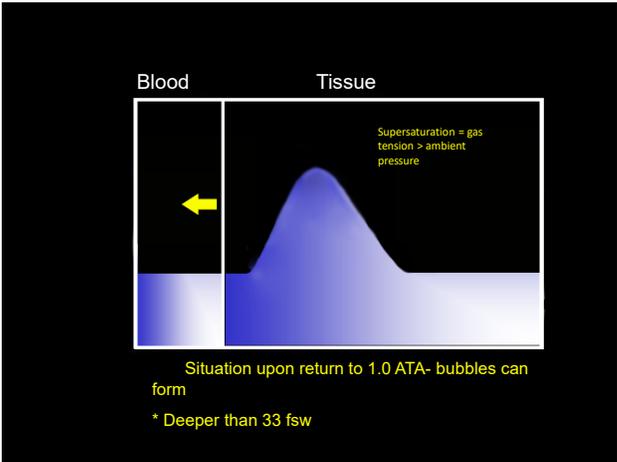
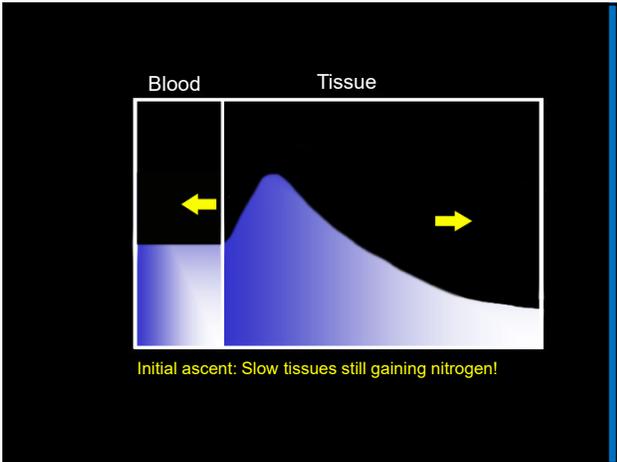
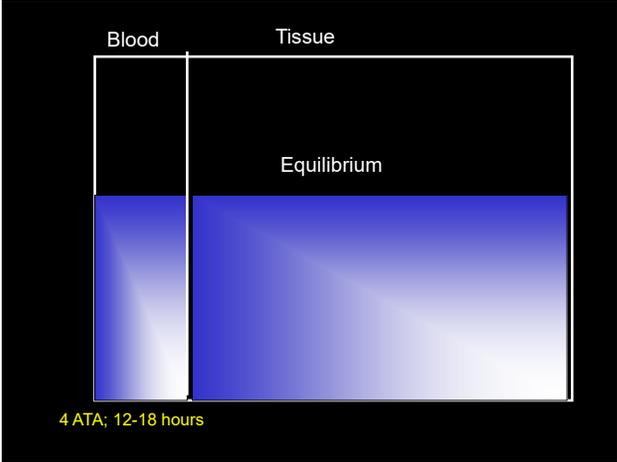
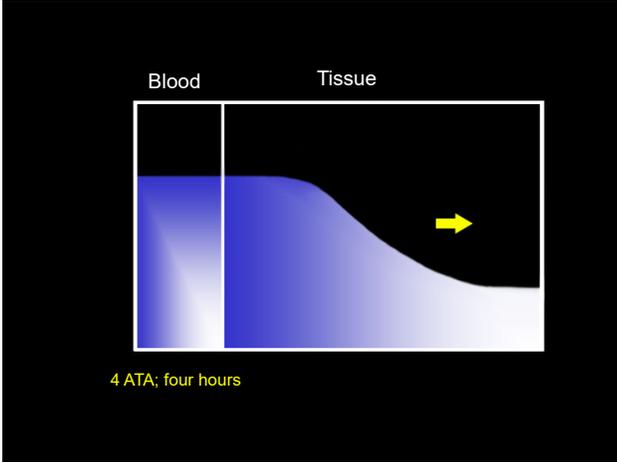


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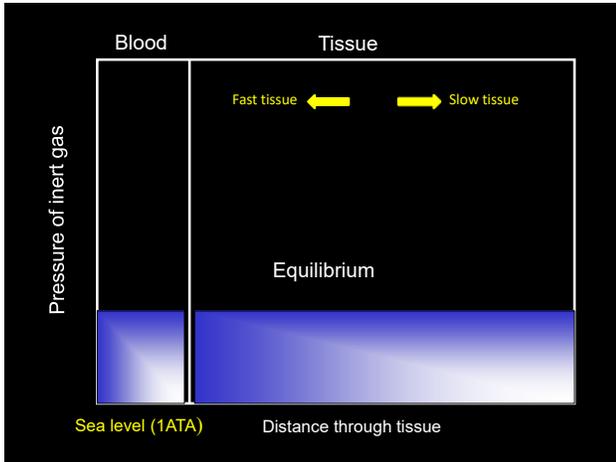
### Nitrogen Elimination

- AKA- "Off gassing"
- Ambient pressure reduction produces a gradient and the process is reversed



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- ### 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



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### Why Do Bubbles Form?

-volume of undissolved gas

- Gas nuclei – *de novo*

Liquid      Solid

Unsaturated      Gas nucleus      Super saturated      Bubble

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### Why Do Bubbles Form?

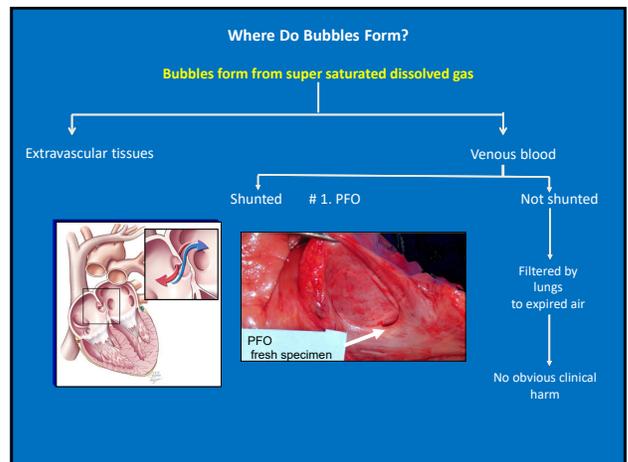
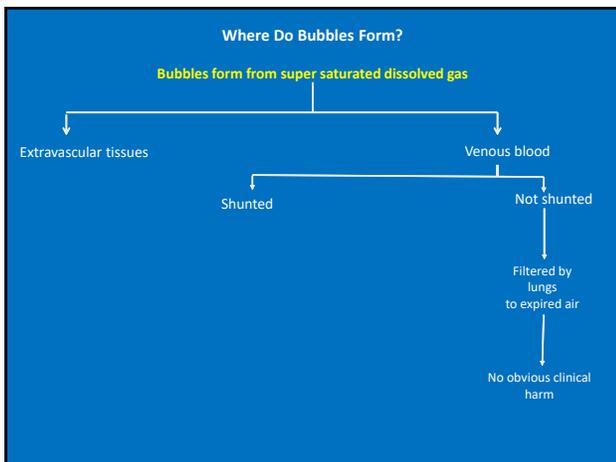
- Bernoulli Cavitation via tribonucleation

Before tribonucleation      After tribonucleation

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### Why Do Bubbles Form?

- 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



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

**Abstract** This study sought to evaluate the effect of catheter-based patent foramen ovale (PFO) closure on the occurrence of arterial bubbles after simulated dives.

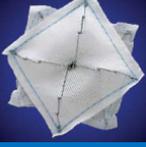
**Background** PFO is a risk factor of decompression sickness in divers due to potential embolization of bubbles. To date, the effectiveness of catheter-based PFO closure in the reduction of arterial bubbles has been debatable.

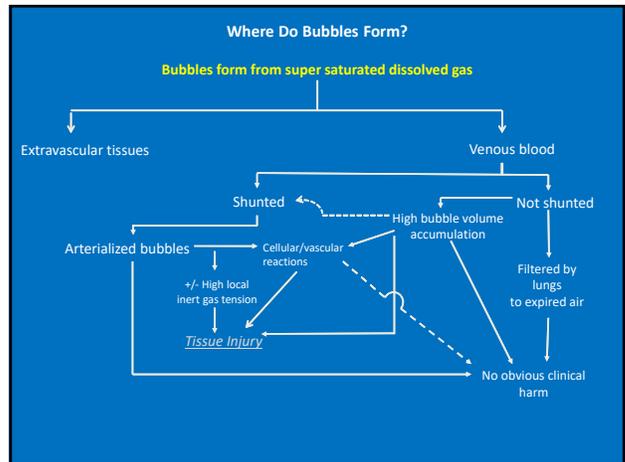
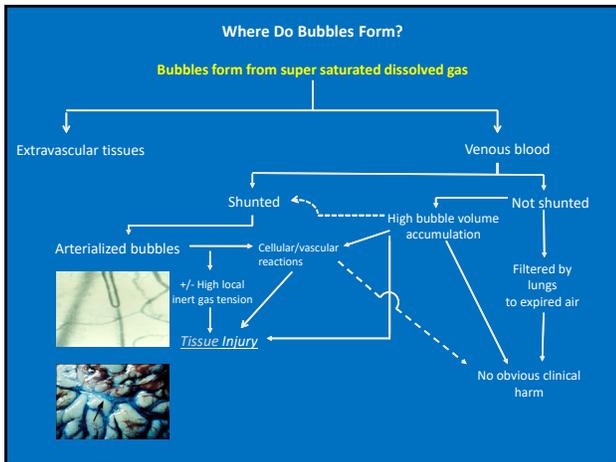
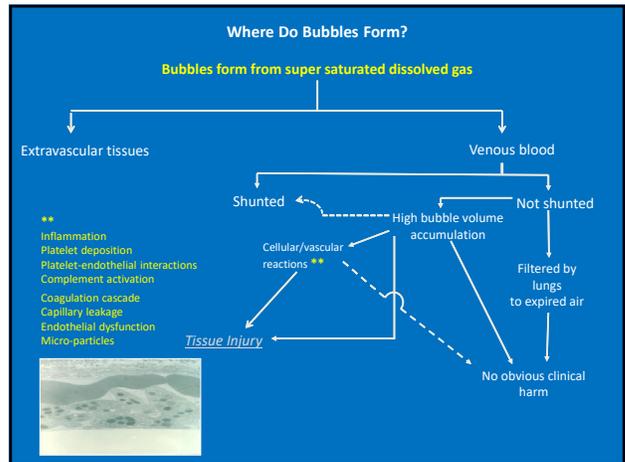
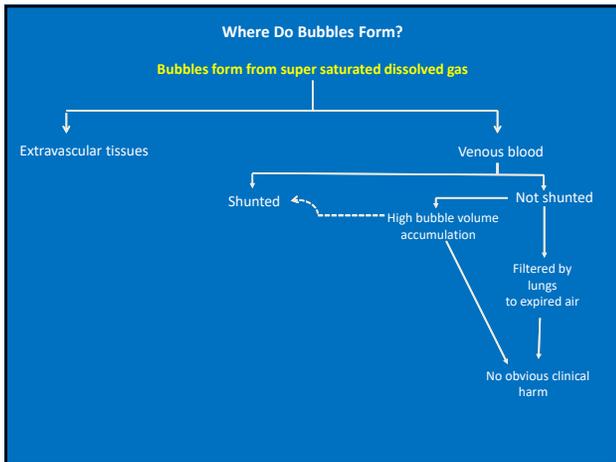
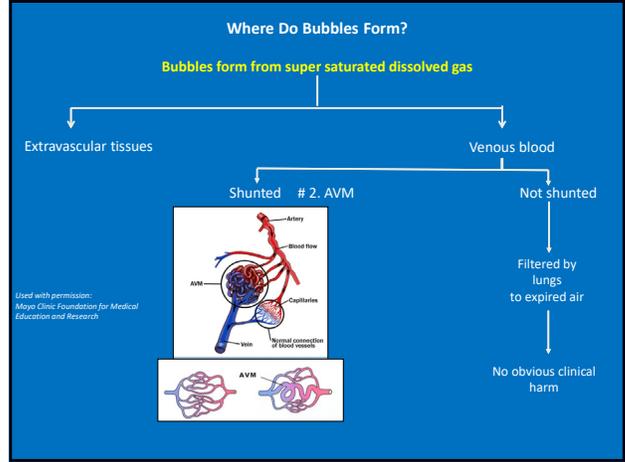
**Methods** A total of 47 divers (age 31.1 ± 8.6 years, 85% male) with a PFO (PFO group) or treated with a catheter-based PFO closure device (closed group) were enrolled in this non-randomized observational trial. All divers were equipped after a simulated dive in a hyperbaric chamber (4 bars, 20 min) in the PFO group. In the closed group, arterial bubbles were observed in 16 of 20 divers. In the PFO group, 14 of 20 divers had arterial bubbles. The presence of venous and arterial bubbles was assessed by echocardiography and transcranial color Doppler ultrasonography, respectively.

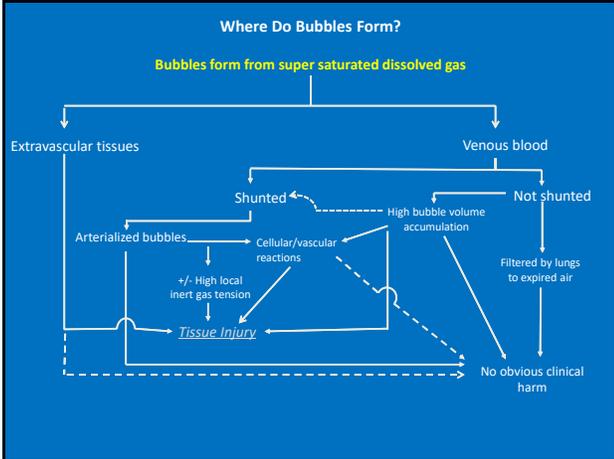
**Results** After the 20-min dive, venous bubbles were observed in 14% of divers in the PFO group versus 0% in the closed group ( $p < 0.05$ ), and arterial bubbles were observed in 10% versus 0% (significance  $p < 0.05$ ). The mean number of arterial bubbles was 1.66 versus 0.00, respectively ( $p < 0.05$ ), and arterial bubbles were observed in 20% versus 0% (significance  $p < 0.05$ ).

**Conclusions** No difference was observed in the occurrence of arterial bubbles between the PFO and closed groups. In the catheter-based PFO closure trial, no complete elimination of arterial bubbles was observed. This suggests that catheter-based PFO closure may not be an effective method for the prevention of arterial bubbles in scuba divers.









### Doppler Studies—Can We Measure Bubbles?

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

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### 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

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### 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

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### At Risk Population

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## 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

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## 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

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## Classification of Symptoms

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

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## Presentation-Type I Decompression Sickness

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

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## 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

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

Peter Geronzi<sup>1,2,3</sup>, Costantino Rubatta<sup>3,4</sup>, Georges Obied<sup>1,5</sup>, Dick Cares<sup>6</sup>

**ABSTRACT**

**INTRODUCTION**

**DISCUSSION**

**CONCLUSIONS**

**REFERENCES**

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Original article: Downloaded from <http://scs.sagepub.com> at Carleton U on October 27, 2015. Reprints: [jhs.sagepub.com](http://jhs.sagepub.com)

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**Systemic intravascular and extravascular air bubbles in type I decompression sickness**

**ABSTRACT**

**OBJECTIVE:** To determine the prevalence of systemic intravascular and extravascular air bubbles in type I decompression sickness (DCS) using contrast-enhanced CT.

**DESIGN:** Cross-sectional study.

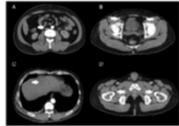
**SETTING:** A tertiary care hospital.

**PARTICIPANTS:** 100 patients with type I DCS.

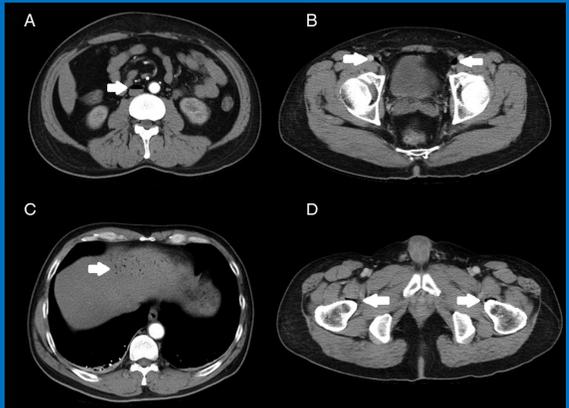
**MEASUREMENTS AND MAIN RESULTS:** Systemic intravascular air bubbles were detected in 100% of patients. Extravascular air bubbles were detected in 100% of patients.

**CONCLUSIONS:** Systemic intravascular and extravascular air bubbles are present in all patients with type I DCS.

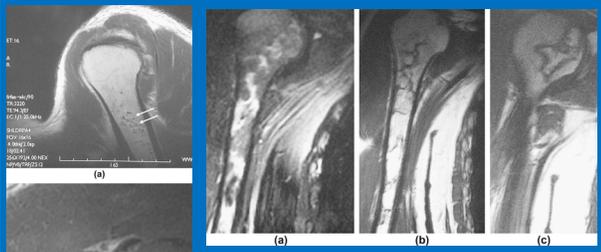
**KEY WORDS:** decompression sickness, air bubbles, CT, type I DCS.



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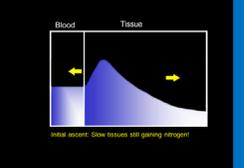
"The extent of these osteonecrotic lesions suggests that one should treat musculoskeletal DCS at once"

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

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**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.*

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**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  
45<sup>th</sup> UHMS Workshop, 1995

*such screening of heightened importance with monoplace chambers*

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**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  
45<sup>th</sup> UHMS Workshop, 1995

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**Treatment Table 5 Depth/Time Profile**

Depth (FSW) vs Time at Depth (minutes)

Descent Rate: 20 FSW/Min  
Ascent Rate: 1 FSW/Min

Total Elapsed Time: 125 Minutes  
2 Hours 15 Minutes (Not including Descent Time)

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

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**Treatment Table 6 Depth/Time Profile**

Depth (FSW) vs Time at Depth (minutes)

Descent Rate: 20 FSW/Min  
Ascent Rate: 1 FSW/Min

Total Elapsed Time: 285 Minutes  
4 Hours 45 Minutes (Not including Descent Time)

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

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**Treatment Table 9**

- Descent rate - 20 fsw/min
- Ascent rate - 20 fsw/min (Rate may be slowed) 15 fsw/min depending on the patient's medical condition
- Time at 60 fsw depth on arrival at 60 fsw
- Flange breathing must be interrupted because (1)DCS flange breathing may appear to be asymptomatic but to be included 10 minutes after all symptoms have subsided (see section on depth of immersion (see paragraph 20.7.11.1.1))
- Severe cramping 100 percent O<sub>2</sub> during last 15 minutes of 60 fsw and during ascent to the surface regardless of ascent rate used
- Flange may transfer air or oxygen during ascent
- Flange cannot transfer oxygen at 45 fsw; this rate can be modified to allow a treatment depth of 50 fsw. The appropriate time can be extended to a maximum of 15 hours

**Treatment Table 9 Depth/Time Profile**

Depth (FSW) vs Time at Depth (minutes)

Descent Rate: 20 FSW/Min  
Ascent Rate: 1 FSW/Min

Total Elapsed Time: 107 Minutes  
2 Hours including Descent Time

Figure 20-10. Treatment Table 9

Indication: Incomplete resolution Type 2 DCS

U.S. Navy Diving Manual  
Revision 7 December 2016

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096-142

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

L. K. WEAVER

Head of Hyperbaric Medicine, Chief Medical Officer, Department of Surgery, US Naval Hospital, San Diego, CA

Since 1984, monoplace hyperbaric chamber use of U.S. Navy Table 6, a 20-year experience, Veterans Hospital San Diego, 3523-34th St. The report is derived from a 2004 report, San Diego, CA, using the U.S. Navy Treatment Table 6 (TT6) as an oxygen field in monoplace hyperbaric chamber (198-2004). Air breathing was provided via a closed-circuit rebreather (CCR) in compliance with the report under a 2004 protocol. The report was submitted to the Navy's Hyperbaric Medicine Committee with a summary report published as follows: (1) The report was submitted to the Navy's Hyperbaric Medicine Committee. They received a total of 108 TT6 (71% of intended patients). Ninety-four percent of the TT6 schedule were completed and completed. The remainder were not completed because of medical reasons. (2) The TT6 schedule was. Tables to complete the TT6 schedule to complete (2) and (3) and (4) and (5). (3) The U.S. Navy TT6 was well tolerated by patients with DCS or AGE treated in monoplace hyperbaric chambers. No fatalities were reported. (4) No patients were treated in the monoplace chamber.

**INTRODUCTION**

Decompression and hyperbaric oxygen therapy are recommended for decompression sickness (DCS) and arterial gas embolism (AGE) in 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100. Further concerns were expressed that patients might not tolerate TT6 in the monoplace chamber because of the treatment duration, confinement anxiety, and management of side effects, such as nausea.

As the only hyperbaric oxygen service from 1984 to 2004 in a several hundred-mile radius, we received and treated a number of patients with DCS and AGE. We began to use the U.S. Navy TT6 in 1985. Here we report a 20-year experience treating patients in the monoplace hyperbaric chamber with U.S. Navy TT6.

**METHODS**

We reviewed all charts of patients with DCS and AGE from 1983 - 2004 at LDRS in monoplace chamber due to lack of direct access to the patient (See comments from the

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## Therapeutic Endpoints

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

**USN:** Until no sustained improvement after two consecutive treatments

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## 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*

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## 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

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## 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

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## 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)*

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## 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)

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+1.919.684.9111  
*emergencies*

+1.919.684.2948  
*general medical requests*

DAN

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## 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

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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.

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### 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

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### 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