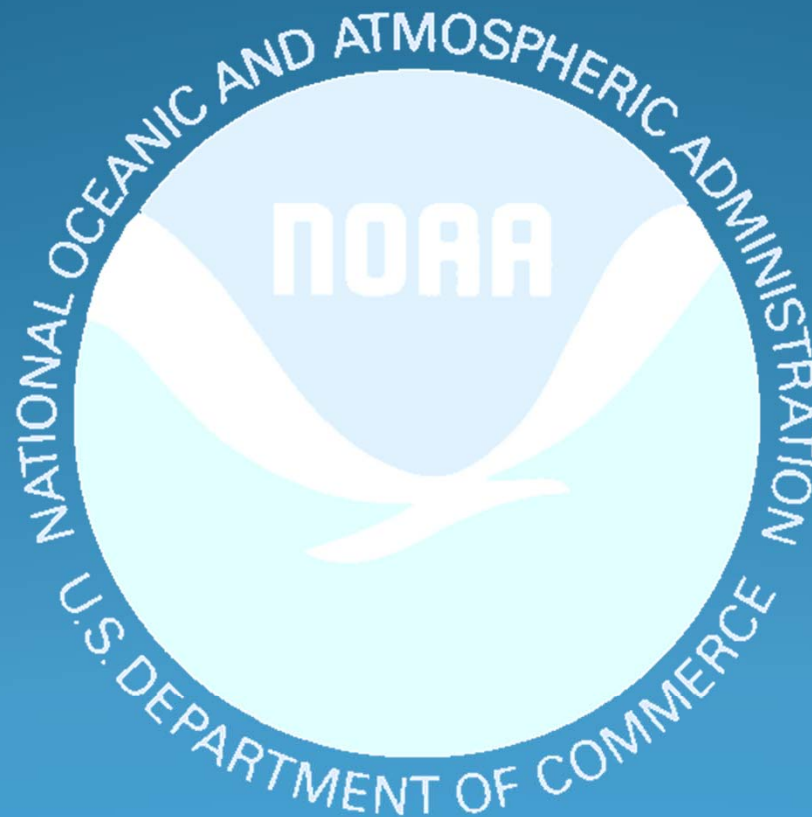


# Pathophysiology of Decompression



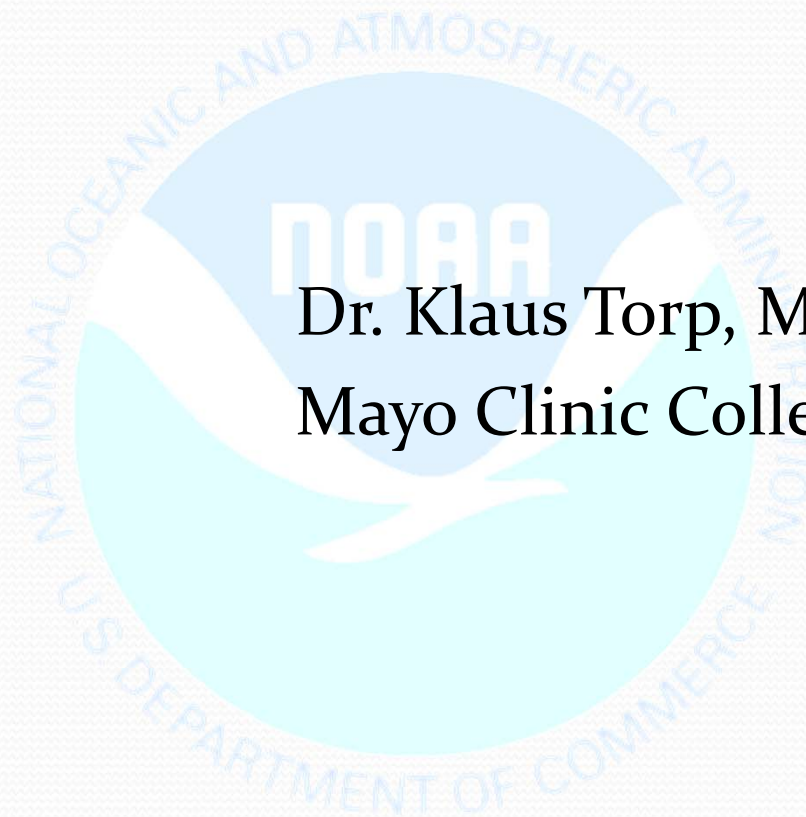
and

# Acute Dysbaric Disorders

# Acknowledgments

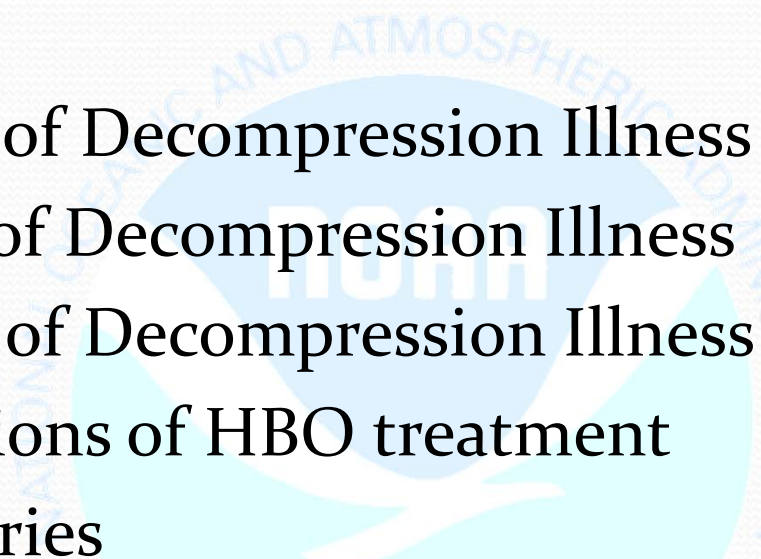
Dr. Klaus Torp, MD

Mayo Clinic College of Medicine





# Overview

- History
  - Definition of Decompression Illness
  - Diagnosis of Decompression Illness
  - Treatment of Decompression Illness
  - Complications of HBO treatment
  - Case Histories
- 

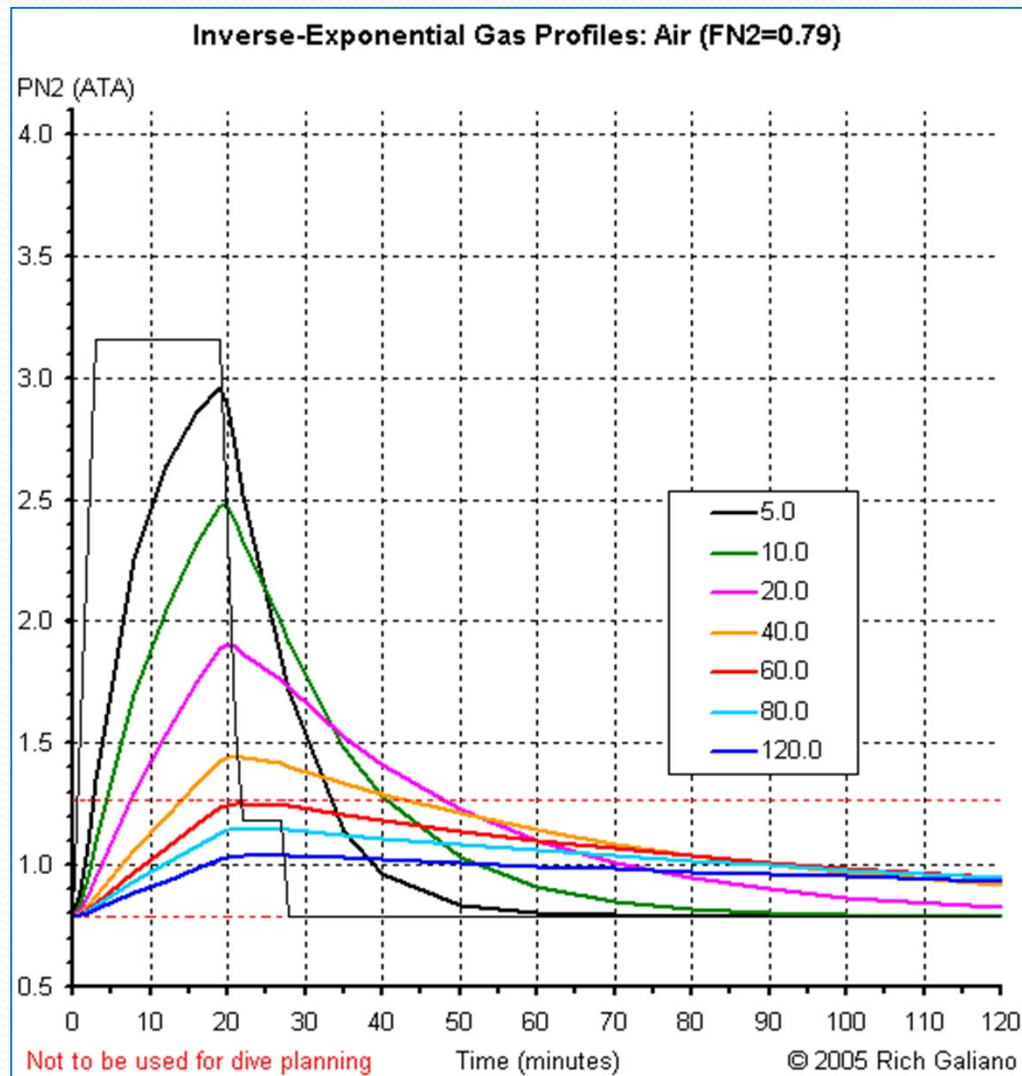


# Nomenclature

- AGE = Arterial gas embolism
  - CAGE=Cerebral arterial gas embolism
- DCS = Decompression sickness
  - Type 1=Bends pain, mild skin symptoms 1960
  - Type 2=Neurological symptoms Golding 1960 (*Caisson*)
  - Type 3=AGE and Type 2 DCS Neuman, Bove 1990
- DCI = Decompression Illness UHMS WS 1989
- Signs and Symptoms after diving
  - i.e. loss of balance after diving Ross Aberdeen 2003



# Compartments and Half-times



# Saturation Diving

- “Saturation” is when tissues hold all of the gas that they can hold at a given pressure
- Once saturated, divers can remain at “storage” pressure until their work is done and then surface one time at the end of the job
- Saturation diving poses special problems



# The Soda Pop Analogy

- Soda is saturated with gas ( $\text{CO}_2$ )
- Pressure (sealed bottle) keeps the gas in solution
- Rapidly reducing the pressure causes gas to rush out of solution and form bubbles
- A saturated diver ascending rapidly can experience a similar effect!
- Note: A bottle of soda with a slow leak does not fizz when opened slowly

Always ascend slowly to allow excess gas to escape!



# Bubble Formation

- Causes:
  - Ambient pressure reduction
  - Low pressure areas (joints)
  - Cavitation & turbulence
  - Microbubble nuclei
- Types:
  - “Silent” (asymptomatic)
  - DCS (symptomatic)

Silent bubbles are estimated to be as much as 5% of N<sub>2</sub> taken up

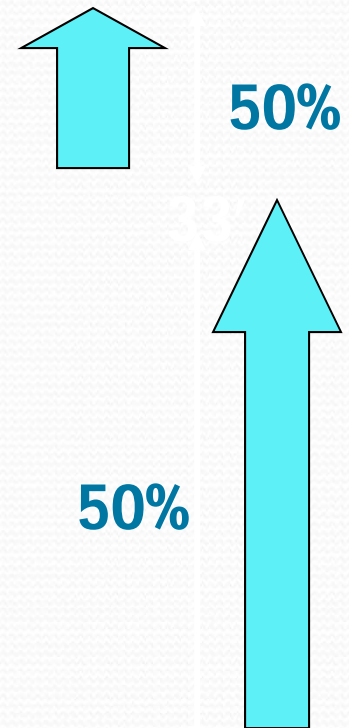
# Bubble Formation

- The type of dive has a significant bearing on where and when bubble formation takes place
  - Short deep dives (>100') tend to cause bubble formation in the fast tissues (only fast tissues on-gas enough  $N_2$  to form bubbles on ascent)
  - Long shallow dives tend to produce bubbles in the slow tissues (fast tissues eliminate their relatively modest  $N_2$  excess before a critical pressure differential develops)
  - Long deep dives cause bubble formation in all tissues



# The Haldanean Theory

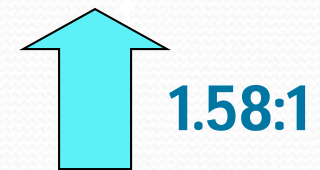
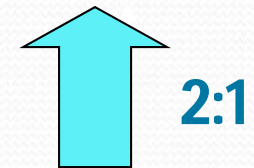
- J. S. Haldane, British physiologist in the early 1900's, theorized that tissues could withstand a 50% pressure reduction without bubble formation
- Haldane used 5 tissue compartment models: 5, 10, 20, 40, and 75 minutes
- The 2:1 Haldane ratio was too liberal and had to be modified





# U.S. Navy Modifications

- In 1937, the U. S. Navy modified Haldane's model and developed decompression tables:
  - Modified surfacing ratio from 2:1 to 1.58:1 (ignored oxygen component)
  - Modified compartments: 5, 10, 20, 40, 80, 120 minutes
  - Added a surface interval credit table
- The U. S. Navy Dive Tables have been tested and modified several times



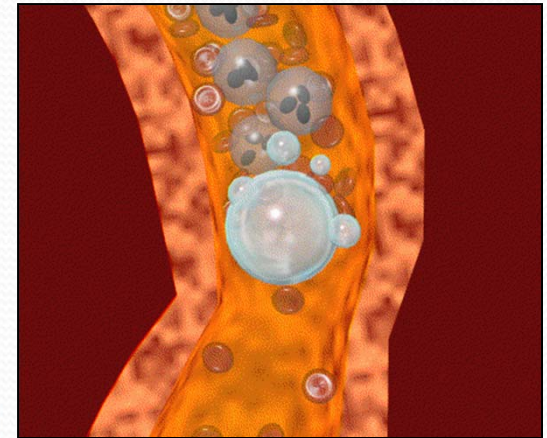
# Effects of Bubbles

- Tissue damage
  - Bubbles in blood obstruct blood vessels in vital organs
  - Bubbles in tissues may press upon blood vessels obstructing blood flow
- Complementary effects
  - Bubbles in blood can stimulate blood clotting which can obstruct blood flow
  - Bubbles are perceived as being a foreign body, thus triggering formation of chemical processes to surround and attack the bubble



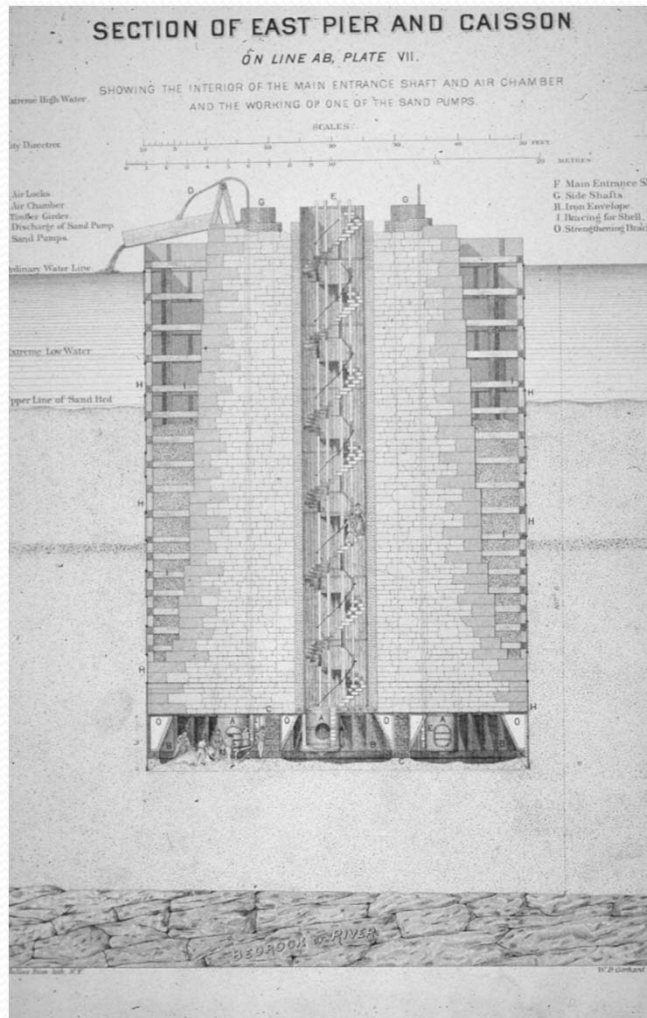
# Decompression Sickness

- Decompression sickness (DCS) is caused by the formation and growth of nitrogen bubbles in the body that result from too rapid a reduction of pressure and other factors
- The respiratory process can eliminate excess nitrogen from the body unless the nitrogen comes out of solution too rapidly and forms bubbles
- Bubbles become lodged in various parts of the body affecting normal functioning





# St. Louis Bridge, 1870



**119 cases of bends  
recorded: 2 crippled for  
life, 14 deaths.**

# Bends in a Caisson Worker

"...it would appear that...the man brought his fate upon himself. He had failed to bring his dinner, so went home to eat it, contrary to orders. Then, on the way back, he 'filled himself' with beer. Moreover, on coming up from his second watch, he left the works before his hour of rest was up. On reaching home in the afternoon, the man was taken sick with vomiting. In a few minutes general paralysis supervened."

Woodward, 1881



# Pathophysiology

- Inert gas (Nitrogen, Helium)
- Stored in tissues
- Slow elimination by the lungs on ascent
  - Tissue half-times
- Bubble formation on ascent
  - In tissues
  - In venous system
  - In arterial system

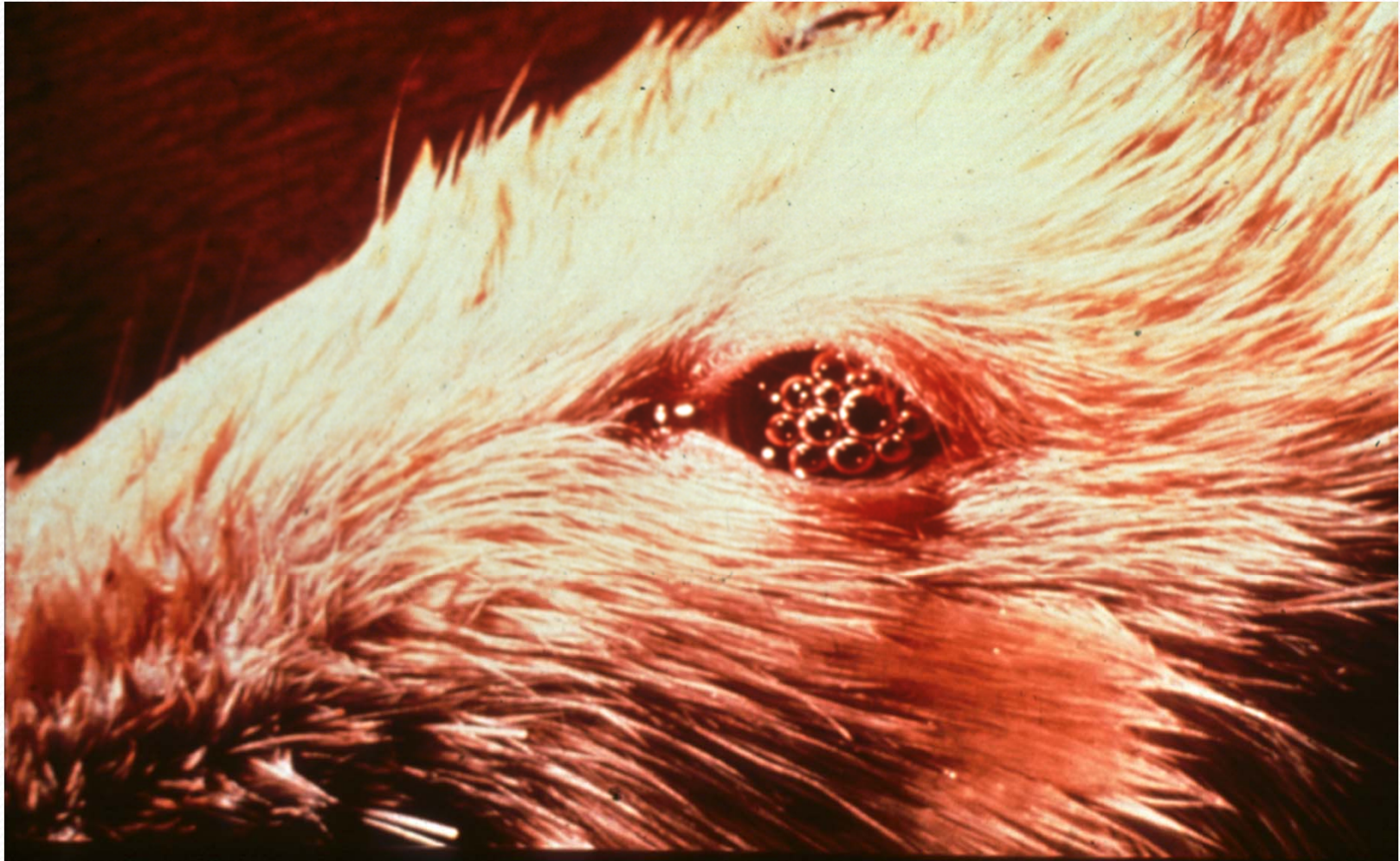
# Decompression Sickness

- Causes:
  - Ascent to sea level
  - Ascent to higher elevations
- Onset of symptoms:
  - 50% within the first hour
  - 90% will manifest within 6 hours
  - Delay in onset of 24 hours or greater has been described

In general, the earlier the symptoms, the more potentially serious the case

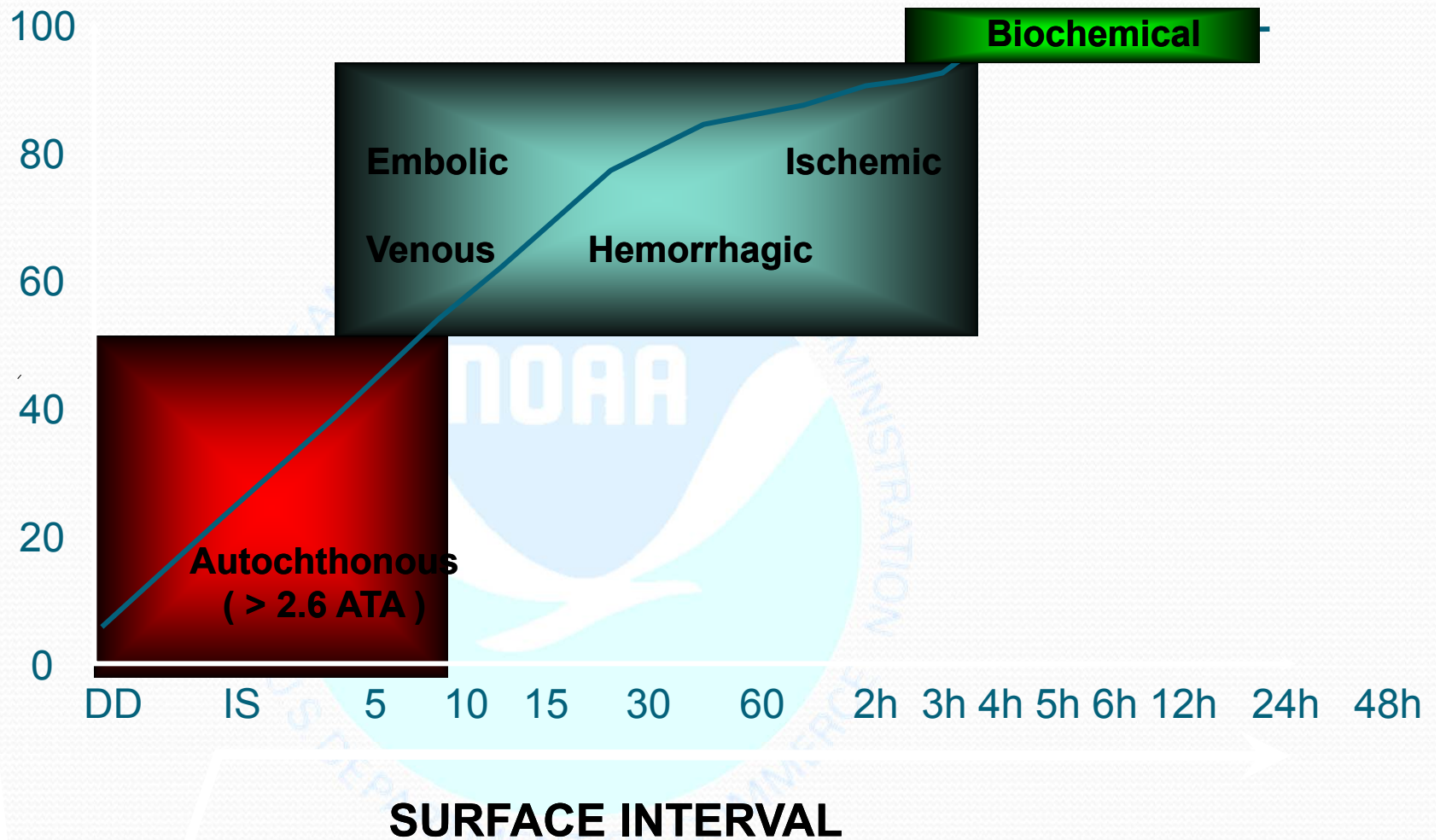


# Decompression Illness





# Latency vs. Mechanism



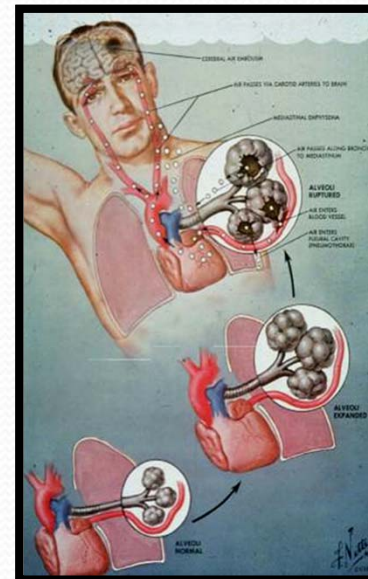
**DIVE**

Modified from: Francis et al. *UBR* 15: 403-417, 1988.



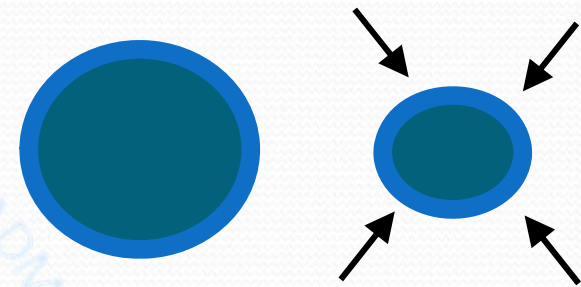
# Bubble Trouble

- **Decompression Sickness (DCS)**
  - SCUBA divers, pilots, and astronauts
  - Staff in multiplace chambers
- **Arterial Gas Embolism (AGE)**
  - SCUBA divers: breath holding upon ascent
  - Iatrogenic: with some medical procedures



# HBO<sub>2</sub> Effect on Gas Bubbles

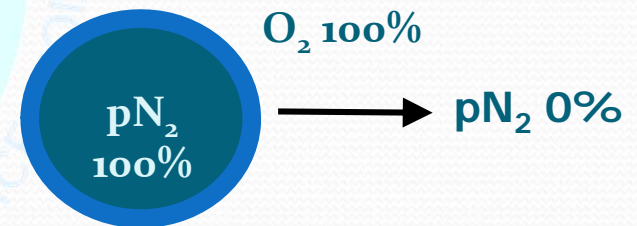
- **Pressure**
  - Shrink bubbles (Boyle's Law)
- **Gas Gradient**
  - Diffuse inert gas out of bubbles



$$P_1 \times V_1 = P_2 \times V_2$$

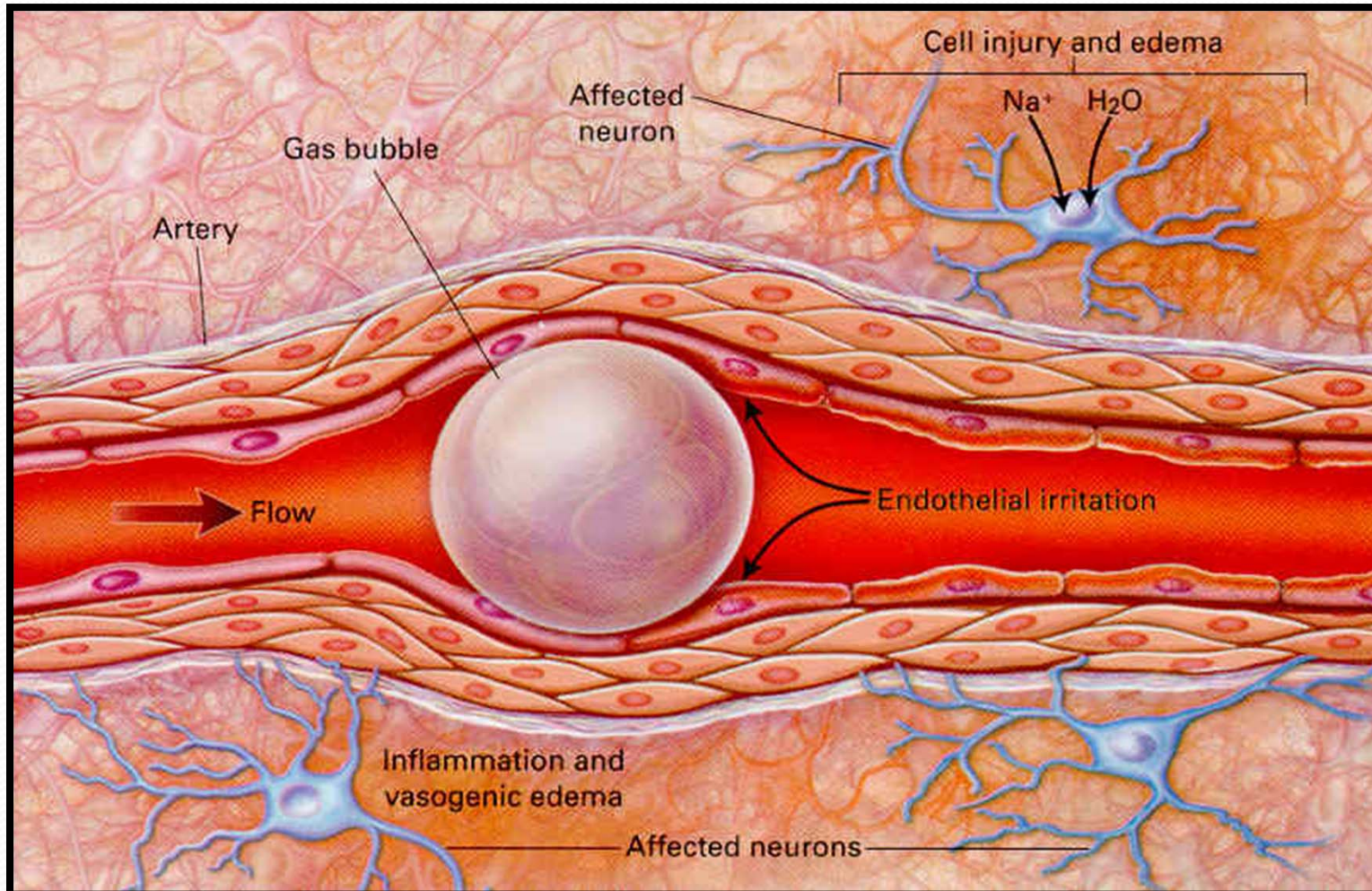
## Effective Intervention for:

- Arterial gas embolism
- Decompression illness





# Bubble Trouble

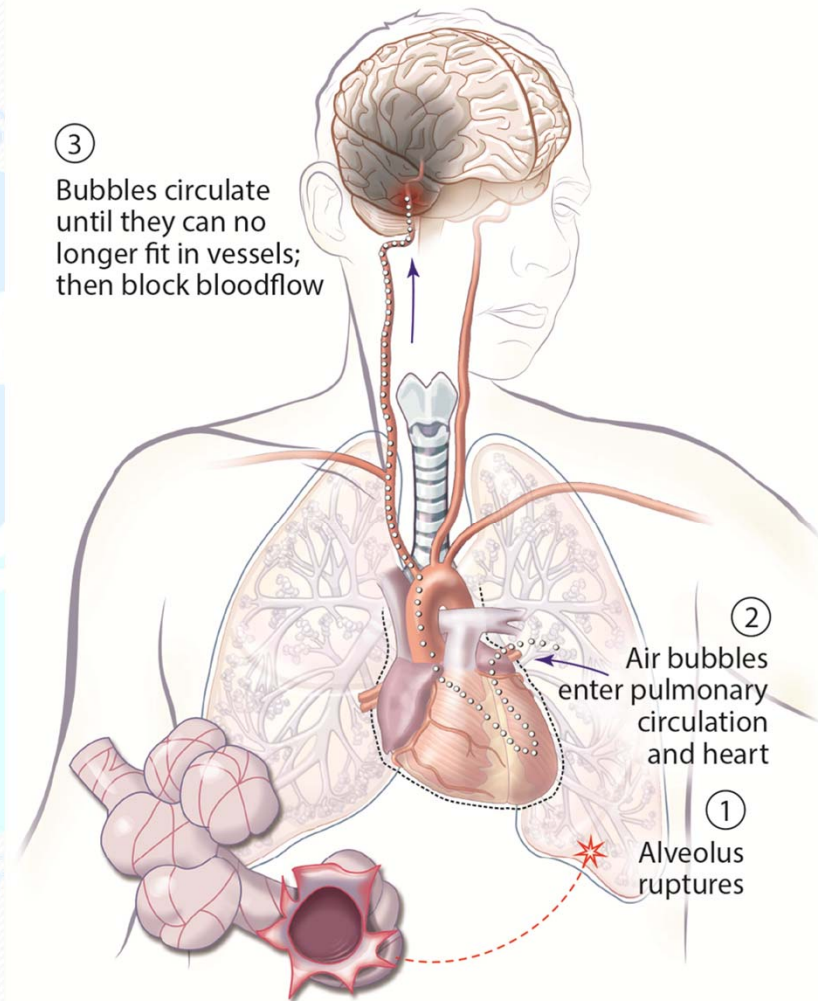


Muth CM. N Engl J Med 2000



# Arterial Gas Embolism

- Part of DCI
- Lung over-inflation
- Large or small
- Wide symptom range
  - Unconsciousness
  - Bloody frothy sputum
  - Mild bends pain

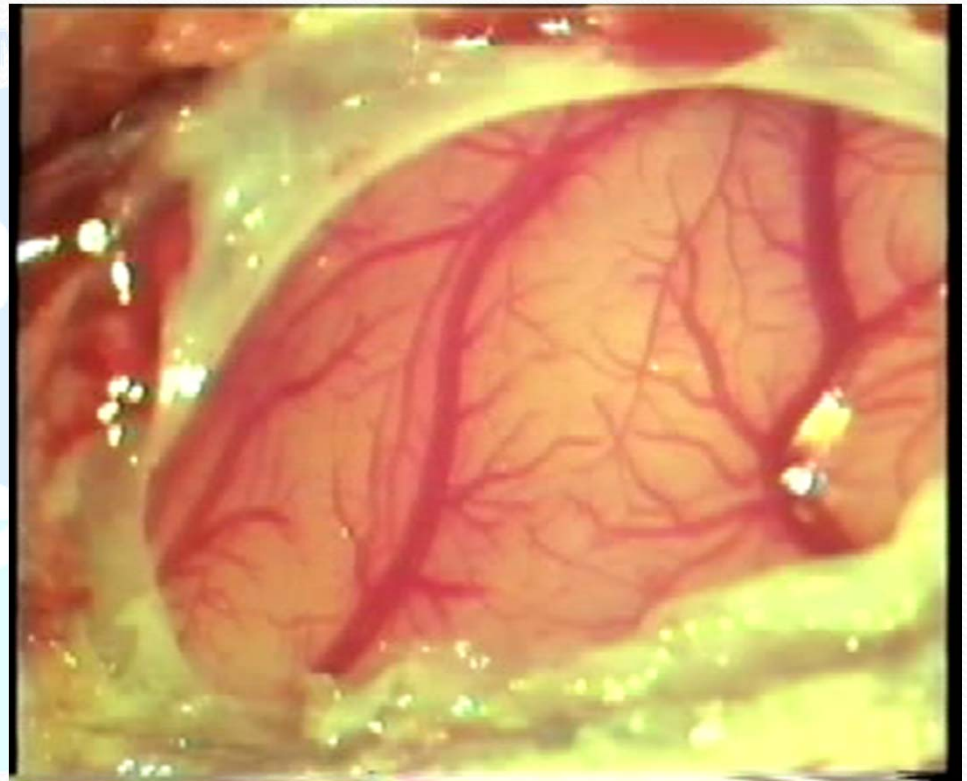




# Cerebral Arterial Gas Embolism

- Destruction of endothelial layer
- Bubbles on the venous side can send inflammatory cytokines (MP ?) to the arterial side
- Leucocyte adhesion
  - Activation by MP ?

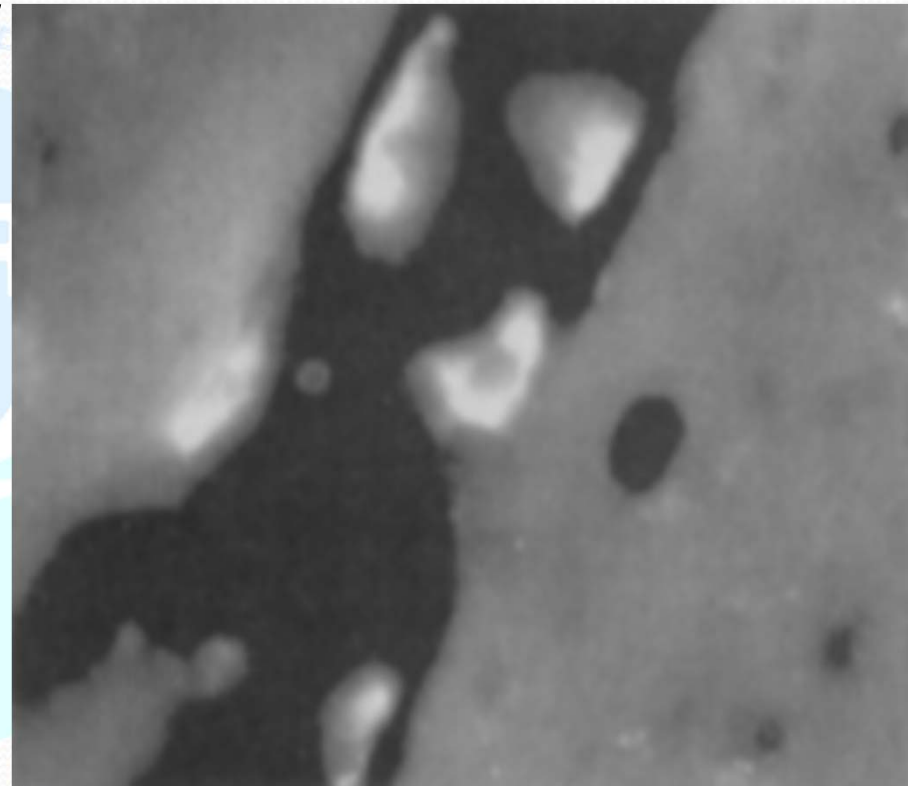
Thom UHM 2011



Helps SC, Gorman DF. Stroke 22:351 1991

# Leukocyte Adhesion in DCS/AGE

- Progressive obstruction of blood flow
- Absent in leukocyte depleted animals
- Inhibited by HBO &  $\beta_2$  Integrin antibodies
  - New human AB developed Eli Lilly



Martin JD, Thom SR. Aviat Space Environ Med 73:565 2002

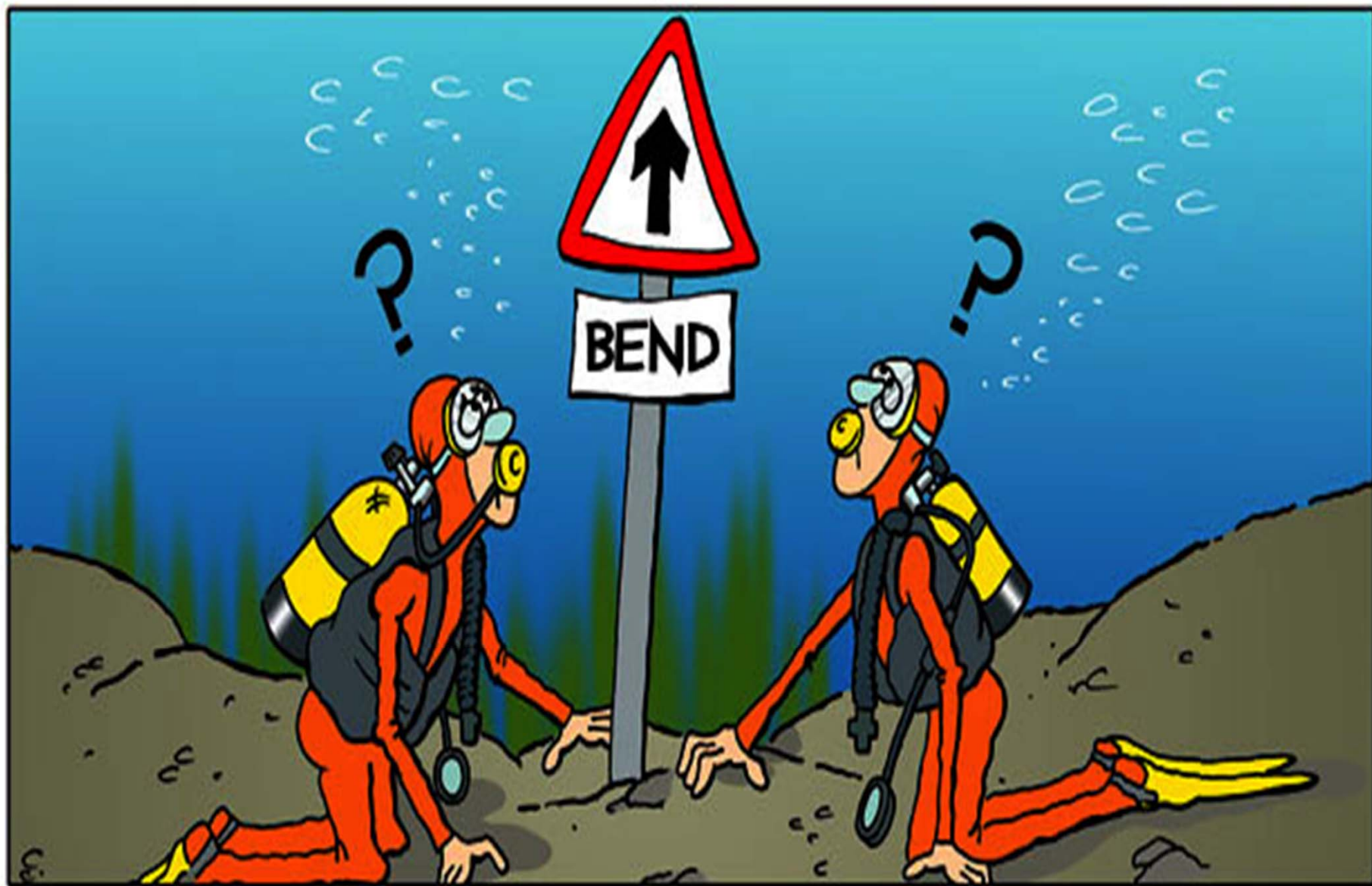
Helps SC, Gorman DF. Stroke 22:351 1991



# Who gets DCI ?

- Anybody
- Any dive profile at any depth can cause signs or symptoms after diving that may respond to recompression USN Dive Manual Rev 6
- Without mistakes (this leads to denial)
- It is a diving disease waiting to happen
- The only 2 ways not to get bent is
  - Never go in the water
  - Never come up

# BUSTER LUNG





# Incidence

- True incidence not known
  - Prevalence 1000 cases/year in US divers
- Difficult to obtain due to unknown number of dives
  - Reluctance of dive operators to report dives
  - Reluctance of diver to report DCI
  - Subsistence fishermen / pearl divers
- Small samples
  - Live-aboard, few dive centers
- Only few studies and data exist
  - Different dives, temperature etc difficult to compare

# Existing incidence data

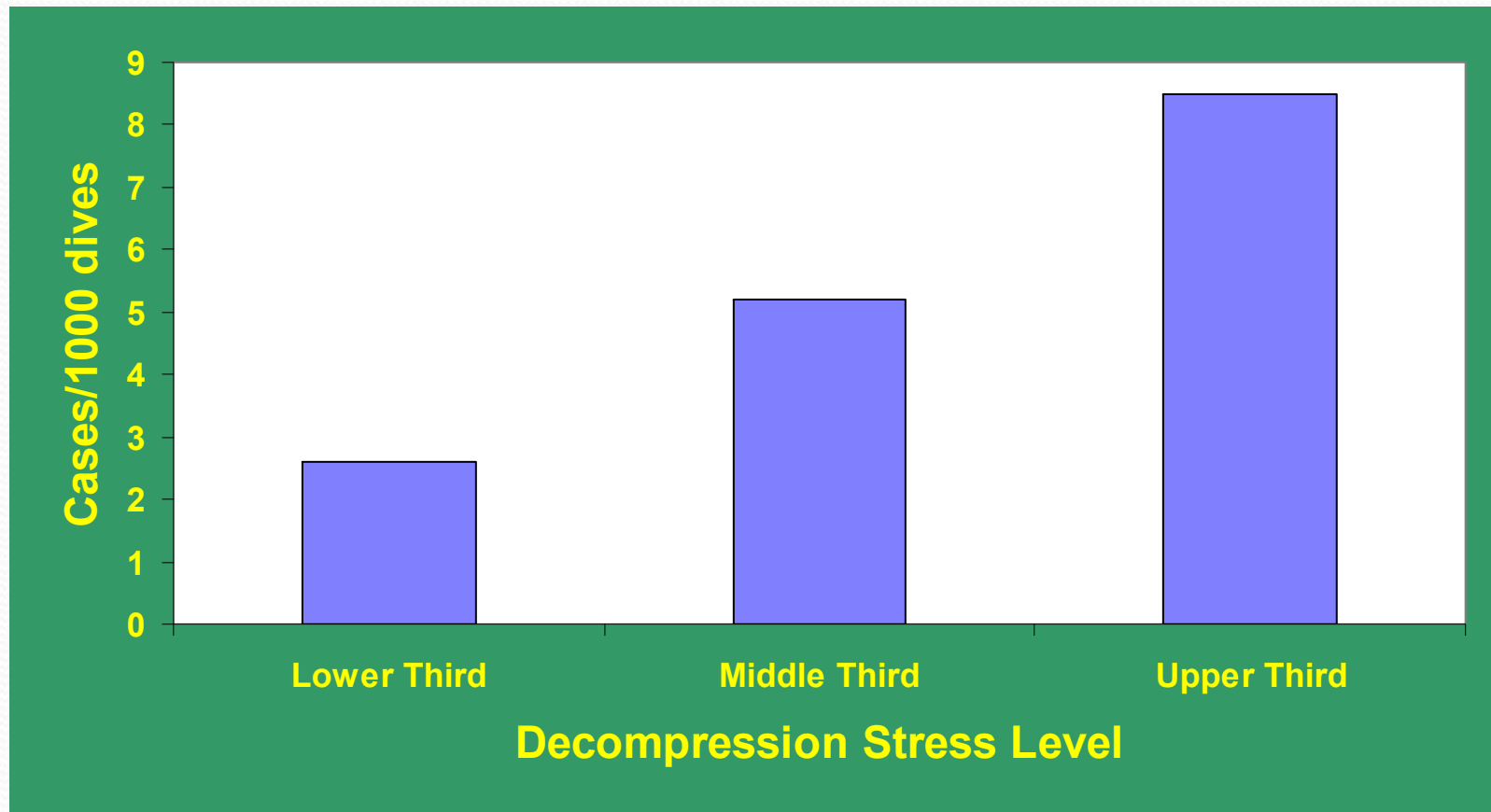
- “Warm Water” dives PDE DAN data
  - DCS incidence: 2 cases per 10,000 dives
- “Cold Water” dives PDE DAN data
  - DCS incidence: 28 cases/10,000 dives
- U.S. Navy Dive Trials Data
  - DCS incidence: 311 cases per 10,000 dives



# DCS Incidence with Standard Air

## Table 1971-96

(38,172 dives, 207 cases, 0.54%)



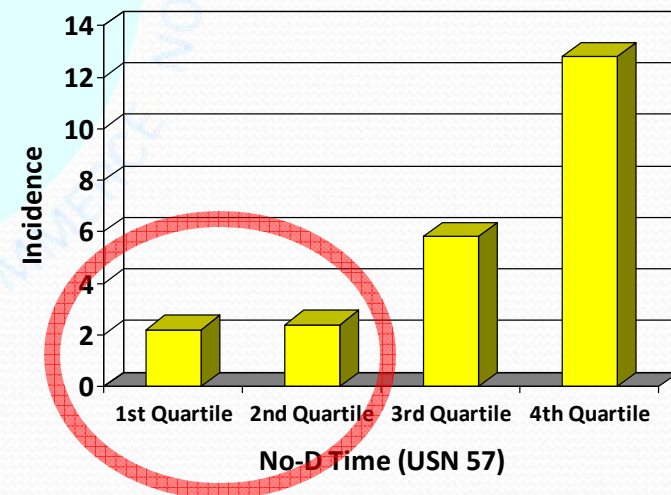
NAVSEA Flynn 2009

# Risk of DCS in no-D limits US Navy diving

- Naval Safety Center, 1990-1994
- 163,400 no-decompression dives, 21-55 fsw
- 48 DCS cases (all no-D time limit)
- Overall DCS incidence: 2.9 per 10,000 dives

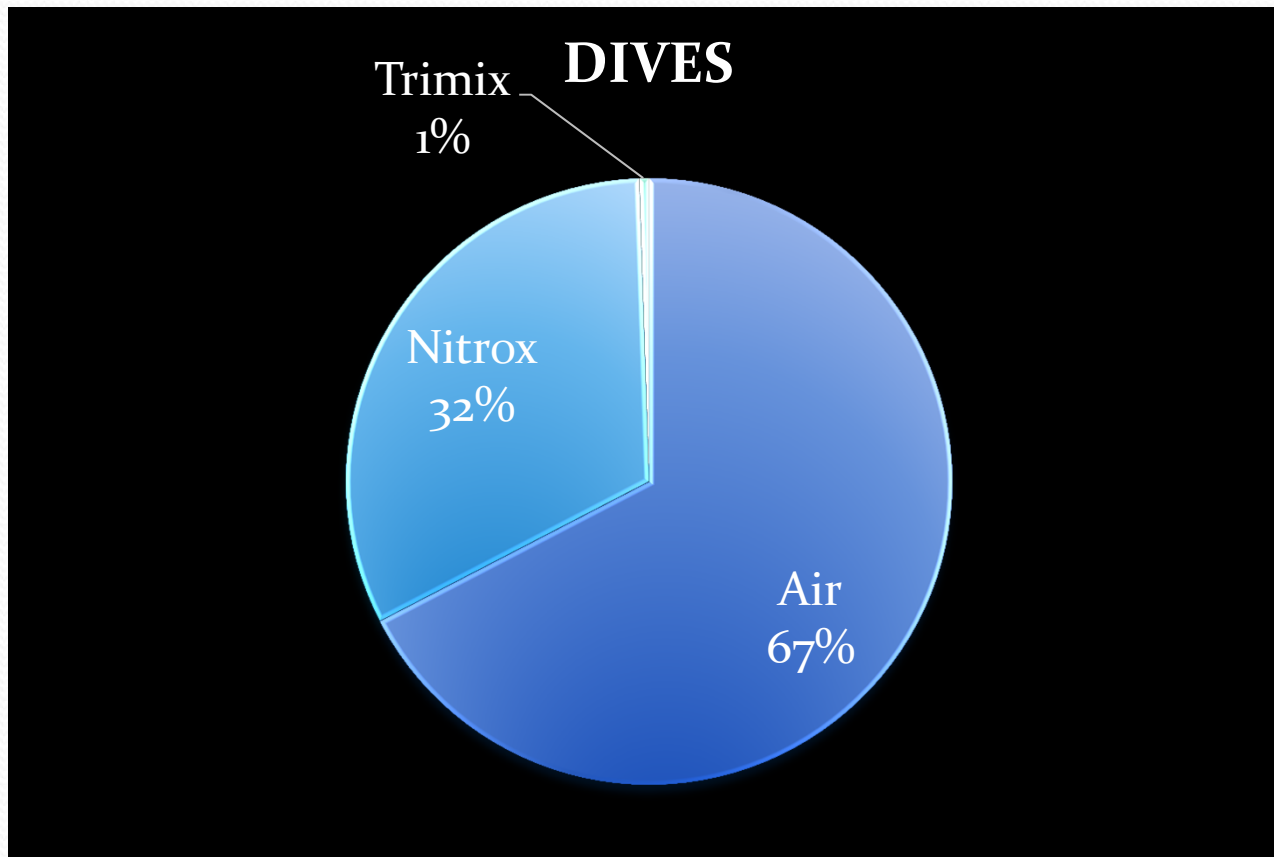
One can have clinical DCI with less than  $\frac{1}{2}$  of exposure time (M values)

*Flynn ET, Parker EC, Ball R., NMRI 1998*



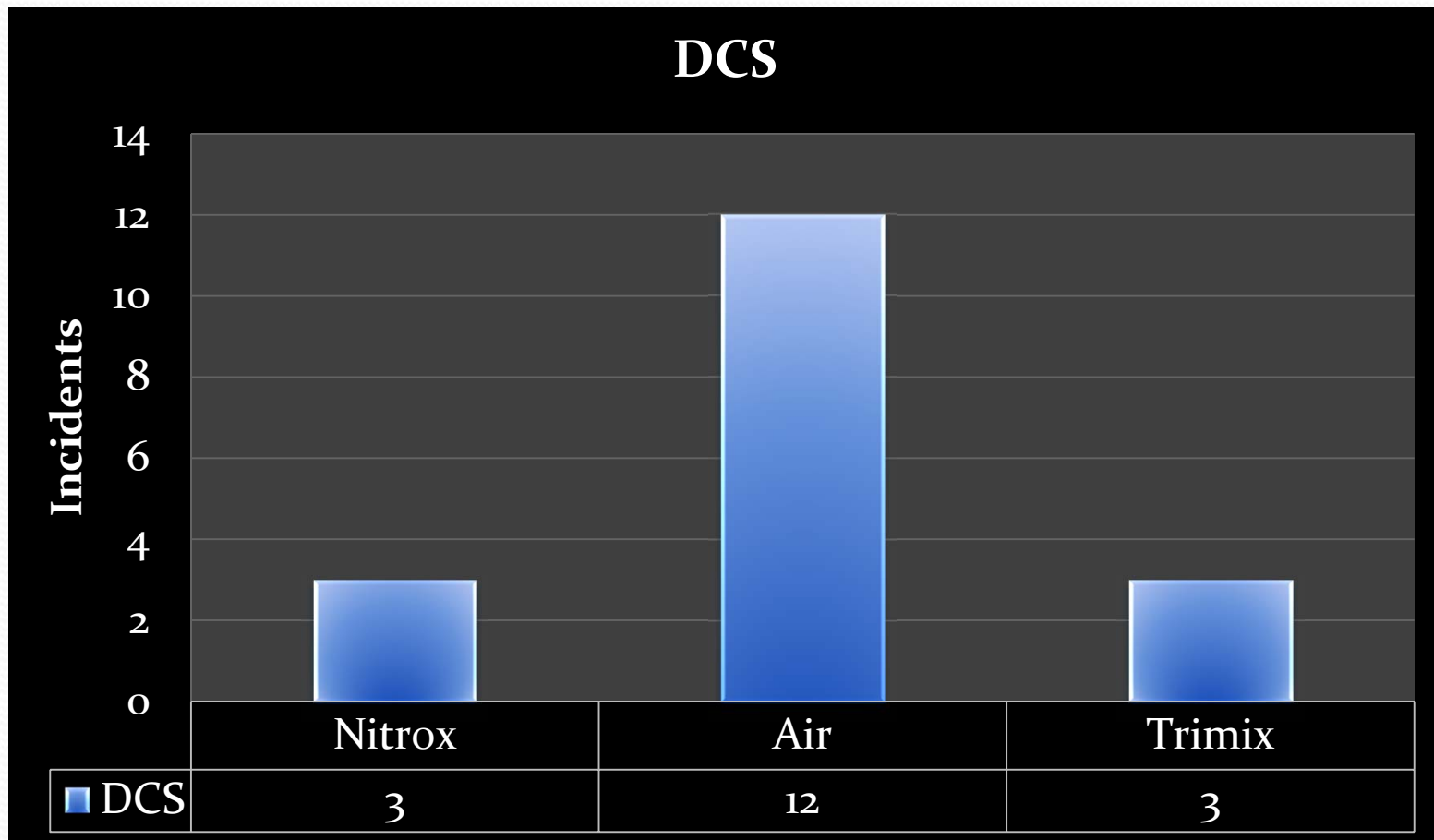


# NOAA Diving 2001 - 2011



2001-2011 NOAA data

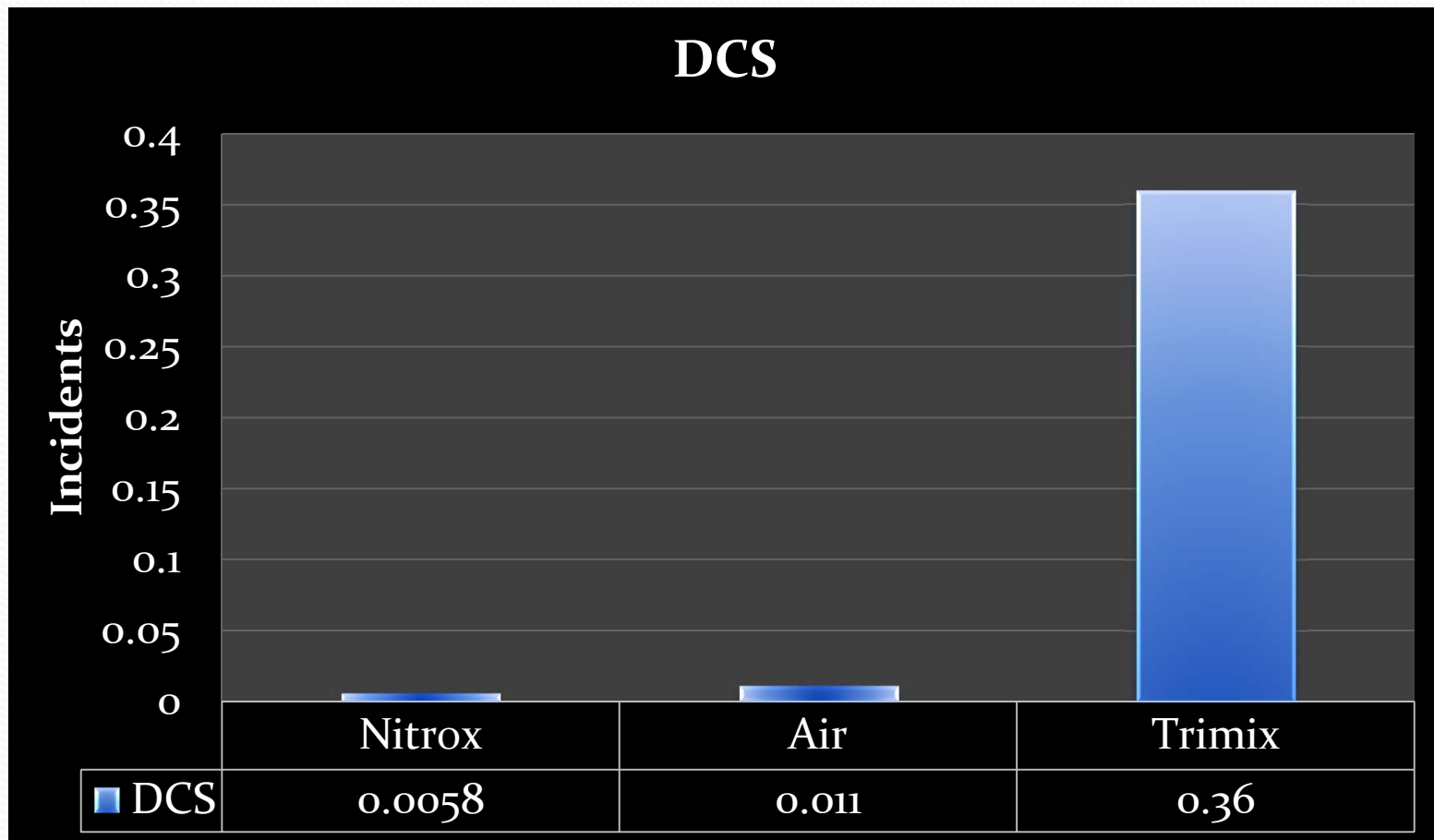
# NOAA Diving 2001 - 2011



2001-2011 NOAA data

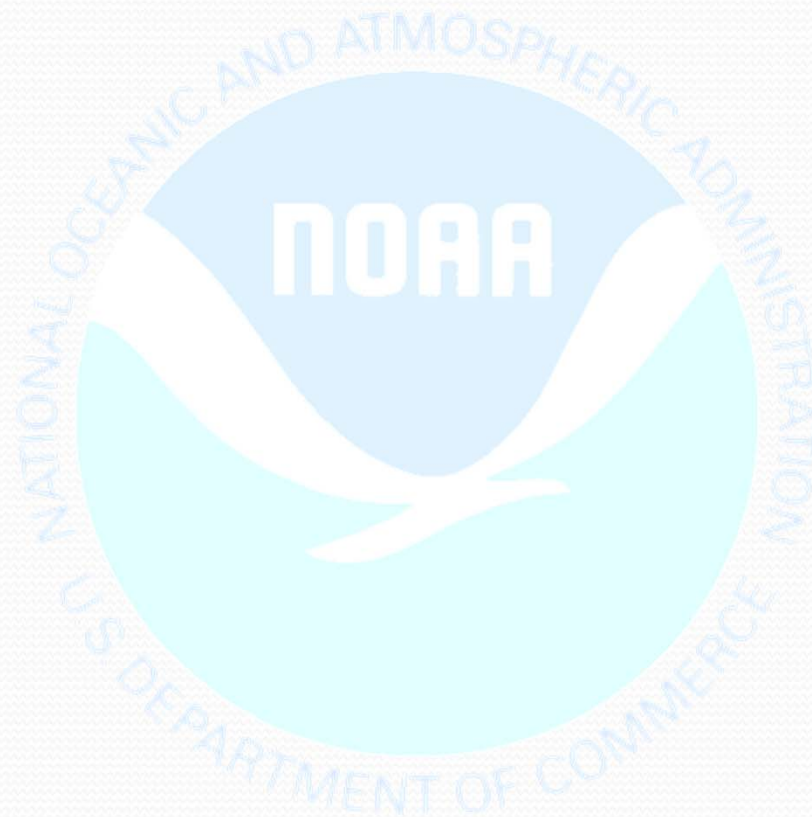


# NOAA Diving 2001 - 2011



2001-2011 NOAA data

Is it safe to dive?





# *Safety: “Freedom from Injury”*

- Oxford Dictionary

- Most activities are not injury-free
- Safety is determined by risk of injury
- Risk depends on probability & severity
  - Curable injury is more acceptable than permanent injury
- So acceptable risk is subjective
  - For an individual, it's personal
  - For an organization, it's political

# Risk factors

- Multiple dives per day ?
- Multiple diving days ?
- Multiple ascents
- Decompression dives
- Diving tables to the limits (USN 2-3%)



# Risk factors

- Dehydration
  - Alcohol

Fahlman et al. ASEM Feb 2006

- Fatigue

- Gender ?

Lee et al; ASEM Nov 2003

- PFO ?

Mutzbauer et al; UHM 2005

- Age

- Prone to more and more severe DCI

Freiberger et al. UHMS ASM June 2006

Conkin et al. ASEM 2003 (altitude)

# Multivariate Model for DCS

1250 DCS cases / 3800 non DCS cases

Denoble et al. UHMS ASM 2005

Variable	OR	LCL	UCL	p-value
Depth of last dive (per 50 fsw)	↑ <b>2.26</b>	2.01	2.53	<0.0001
Previous DCI	↑ <b>1.99</b>	1.35	2.94	0.0005
Days diving (per 5 days)	↑ <b>1.53</b>	1.14	2.06	0.005
Male	↑ <b>1.19</b>	1.01	1.40	0.044
Dives in past year (per 10 dives)	↓ <b>0.95</b>	0.94	0.96	<0.0001
Age (per 10 years)	↓ <b>0.92</b>	0.86	0.99	0.0247
Years diving (per 10 years)	↓ <b>0.89</b>	0.80	0.98	0.0145
Drysuit	↓ <b>0.72</b>	0.59	0.89	0.0018
Number of dives (per 5 dives)	↓ <b>0.56</b>	0.50	0.64	<0.0001
Nitrox	↓ <b>0.48</b>	0.39	0.61	<0.0001

↑  $OR > 1 = \text{risk factor}$

↓  $OR < 1 = \text{protective factor}$



# Effect of Exercise on DCS Risk



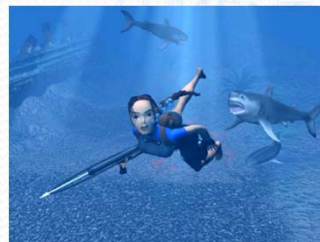
- ? Micronuclei depletion
- ? Micronuclei generation
- ? Nitric oxide effect



- ? Micronuclei generation
- ? ↑ Intrapulmonary shunt



- ↑ Inert gas washout
- ? ↑ Intrapulmonary shunt



- ↑ Inert gas uptake

# Micronuclei

- Gas bubbles grow from pre-existing gas nuclei ( $\sim < 1\mu\text{m}$ )
  - Need high super-saturation pressure for de-novo bubble formation
- Gas bubbles can exist in crevices on hydrophobic endothelial cells
- Exercise can produce “cavitations”, producing bubbles (tribonucleation)
- ? Tissue trauma effect on micro-nuclei



# Exercise before diving

- Vigorous exercise 24 hrs before the dive reduced bubble load (in man) however not at 12 hrs or 48 hrs or 30 min (in rats)

Dujik et al. J Physiol 2004, Berge et al. ASEM 2005; 76:326–8.

- Exercise 2 hr pre dive reduces VGE in man

Blatteau et al. ASEM 2005

- Timing?
- Intensity?
- Mechanism?

# Effect of Exercise on DCS Risk



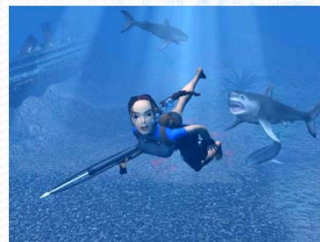
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- ? Micronuclei generation
- ? ↑ Intrapulmonary shunt



- ↑ Inert gas washout
- ? ↑ Intrapulmonary shunt



- ↑ Inert gas uptake



# Exercise on the bottom

- Increase CO<sub>2</sub> production
- Increase perfusion
- Increase ventilation
- Need longer off-gassing time
- In high PO<sub>2</sub> environment may decrease seizure threshold

# Exercise Effect During Bottom time on VGE in Wet Dives

RESEARCH ARTICLE

## Exercise Effects During Diving and Decompression on Postdive Venous Gas Emboli

LOUIS W. JANKOWSKI, PETER TIKUISIS, AND RONALD Y. NISHI

JANKOWSKI LW, TIKUISIS P, NISHI RY. *Exercise effects during diving and decompression on postdive venous gas emboli.* Aviat Space Environ Med 2004; 75:489-95.

**Background:** Exercise and diving have generally been associated with an increased risk of decompression sickness (DCS), thus accounting for the lack of studies involving exercise during decompression. However,

urated fluids produces gas nuclei (13) leading to bubble formation and a corresponding increase in the risk of DCS (11,27,29). Thus, strenuous exercise before, during, and/or after diving, for various reasons, has been associated with an increased risk of DCS (4).

- **Moderate intermittent exercise on the bottom:  
no effect on VGE**

Jankowksi et al. Aviat. Space Environ Med 2004



# Exercise Effect During Decompression on VGE in Wet Dives

RESEARCH ARTICLE

## Exercise Effects During Diving and Decompression on Postdive Venous Gas Emboli

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**Moderate intermittent exercise: reduced VGE**

**Mild continuous exercise: reduced VGE**

Jankowksi et al. Aviat. Space Environ Med 2004

Dujic et al. Med, Sci Sports Exerc. 2005

# Effect of Exercise on DCS Risk



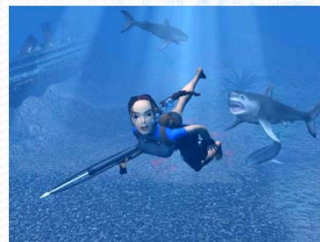
? Micronuclei depletion  
? Micronuclei generation



? Micronuclei generation  
? ↑ Intrapulmonary shunt



↑ Inert gas washout  
? ↑ Intrapulmonary shunt



↑ Inert gas uptake



# Exercise after diving

- Risk factor for DCI
  - Strenuous exercise
- May help off-gassing
  - Mild exercise
- Tough to avoid in dive instructors/masters
  - Need to stay fit
- Bouts of exercise can liberate bubbles
- Exercise can open intra-pulmonary shunts
  - 21/23 healthy volunteers on cycle ergometer

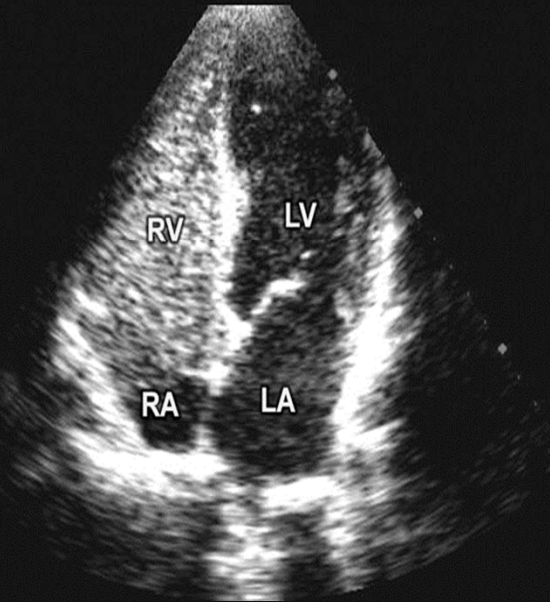
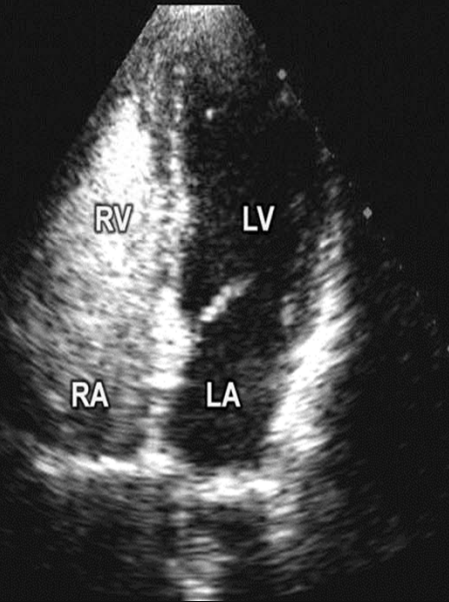
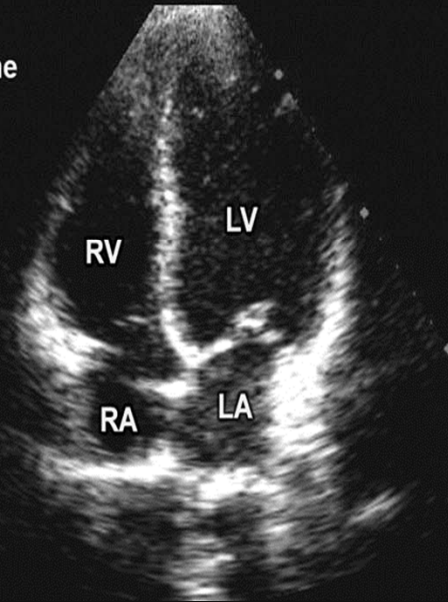
Eldridge JAP 97,2004

Before Injection

Contrast Injection

After Injection (>5 Cycles)

Baseline

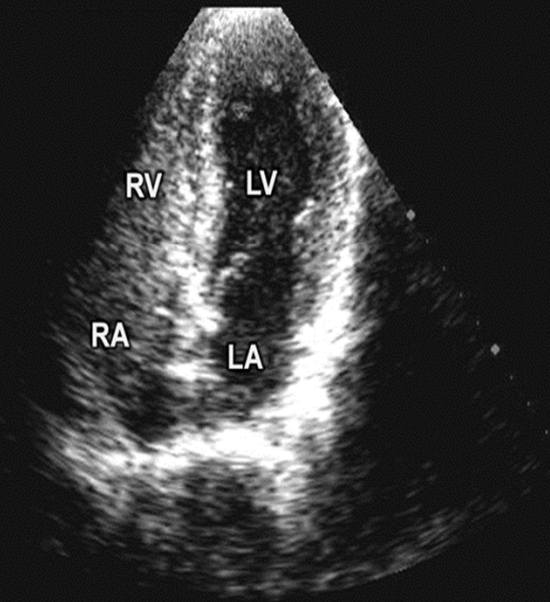
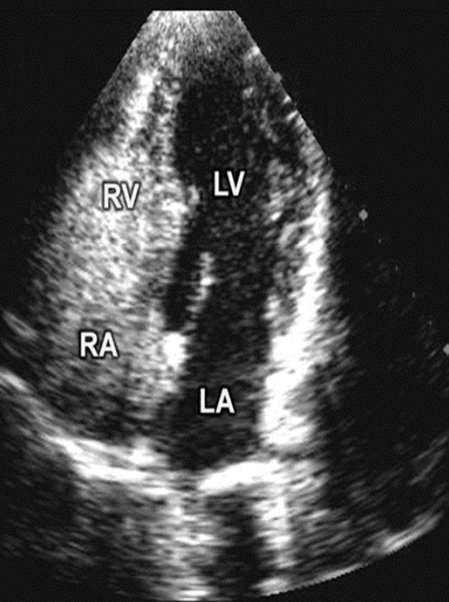
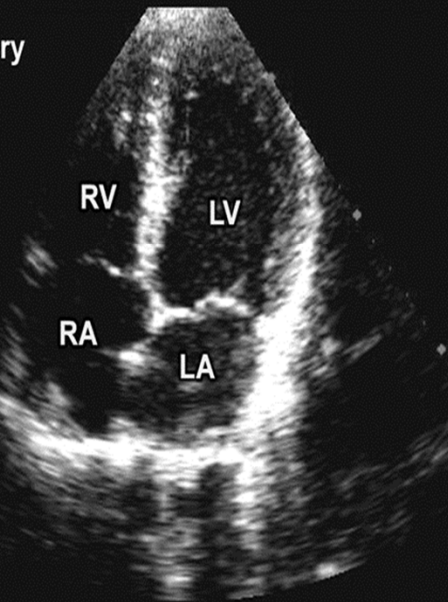


Before Injection

Contrast Injection

After Injection (>5 Cycles)

Recovery



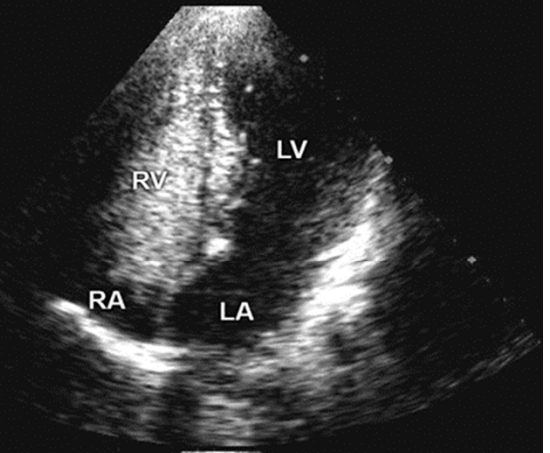
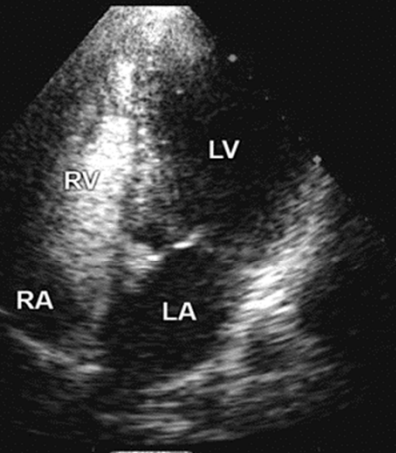
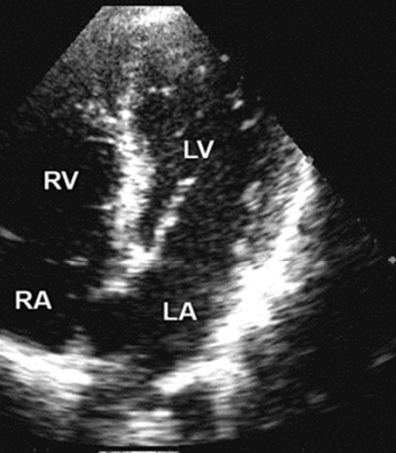


Before Injection

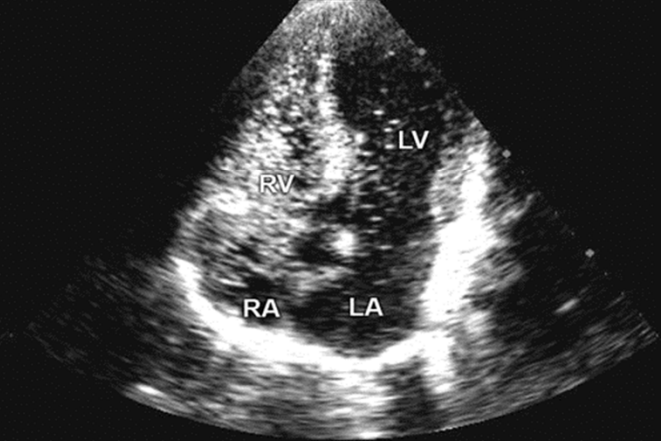
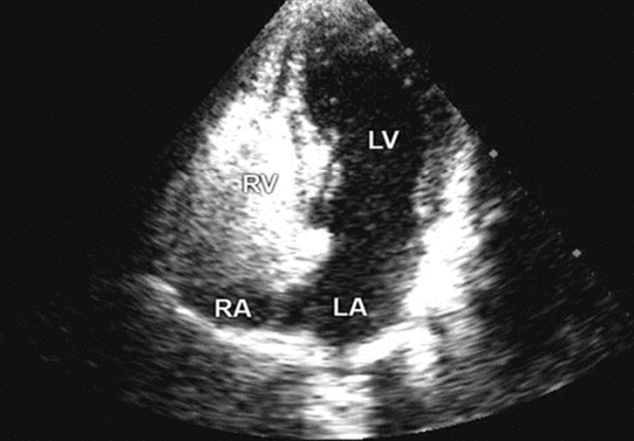
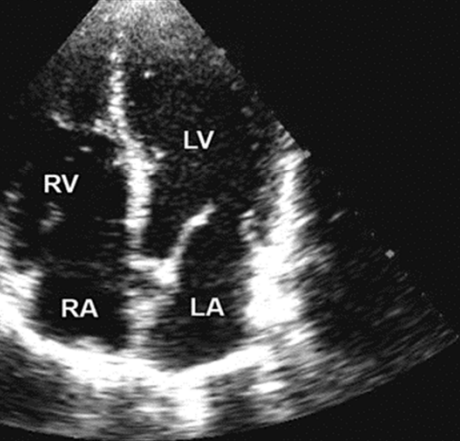
Contrast Injection

After Injection (>5 Cycles)

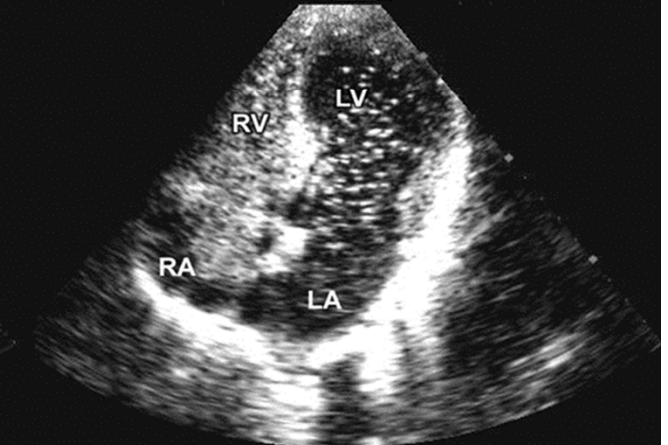
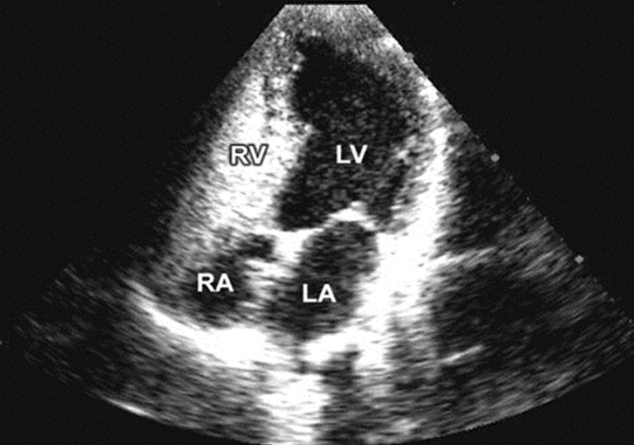
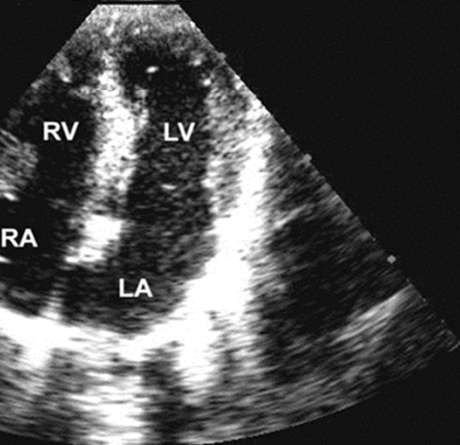
100 watts



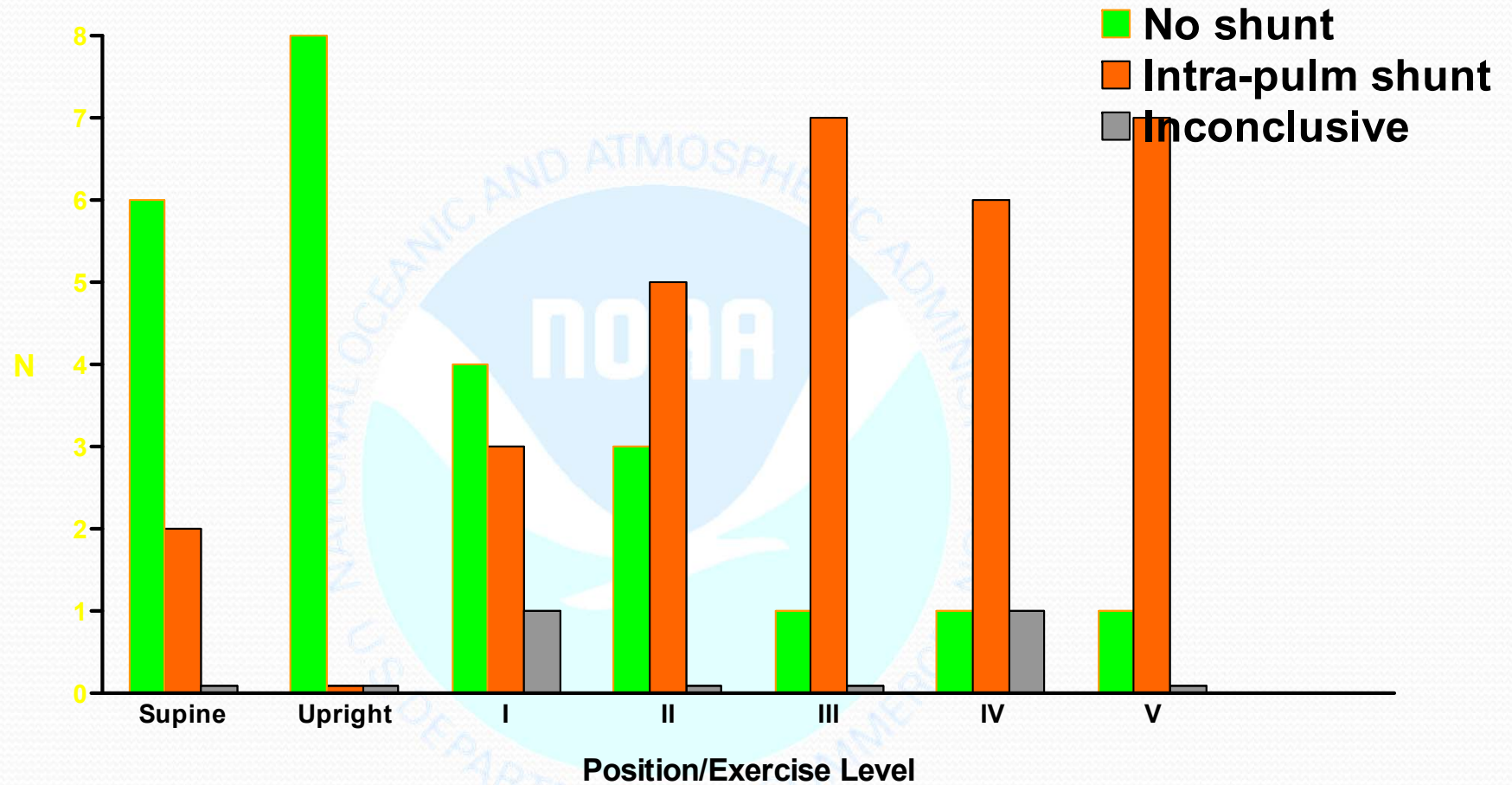
230 watts



260 watts



# Transpulmonary Shunt during Exercise



Stickland MK, et al *J Physiol* 561:321, 2004



# Exercise after diving

- One study failed to show right to left shunt after diving followed by exercise (85% max  $\dot{V}O_2$ )
  - Bubble score was quite a bit lower than the previous contrast studies (10/11 divers)
  - 1 diver had no bubbles

Dujic et al. J Appl Physiol 2005

# Symptoms of DCI

- Paresthesias
  - most common symptoms in recreational divers
  - can be intermittent and migrating
  - 67% of 200 consecutive divers

Newton, Padilla; UHM 34(5); 2007



# Symptoms of DCI

- Dull aching limb pain
  - Mostly in joints, without change during movement
  - May respond to BP cuff around the joint
- Often upper extremities in bounce diving
- Often lower extremities in saturation diving



# Symptoms of DCI

- Skin symptoms
  - rash, peau d'orange, itching, tenderness
  - 16-24% out of 8424 cases
  - 21% -> 50% had neurologic symptoms


Bird, N; UHMS ASM 2010 ;  
Newton, Padilla; UHM 34(5); 2007
- Lymphedema
  - Often on the trunk



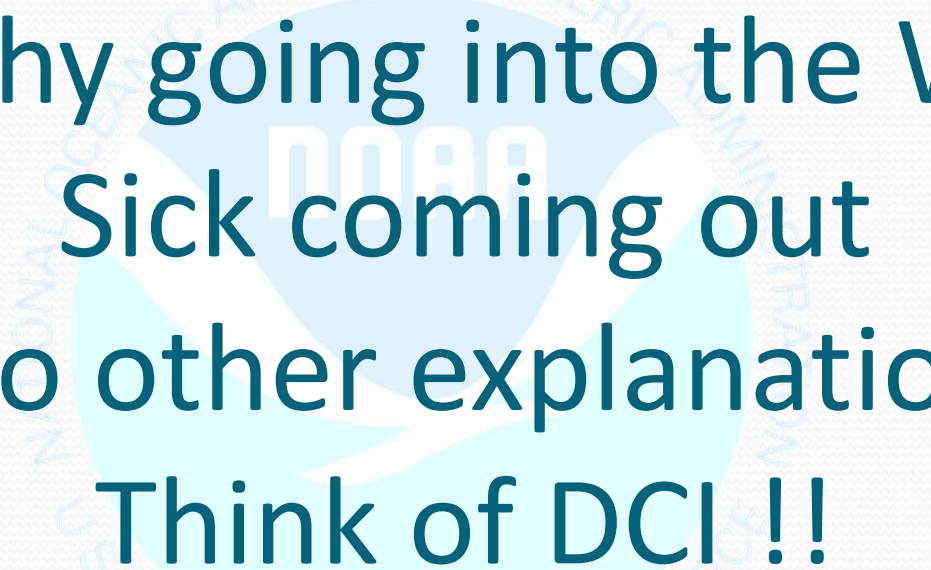
# Symptoms of DCI

- Headache
- Extreme fatigue
- Nausea / Vomiting
- Abdominal cramps
- Personality change/Mental aberration 44.5%

Cianci, Slade ASEM 2006



Healthy going into the Water  
Sick coming out  
No other explanation  
Think of DCI !!



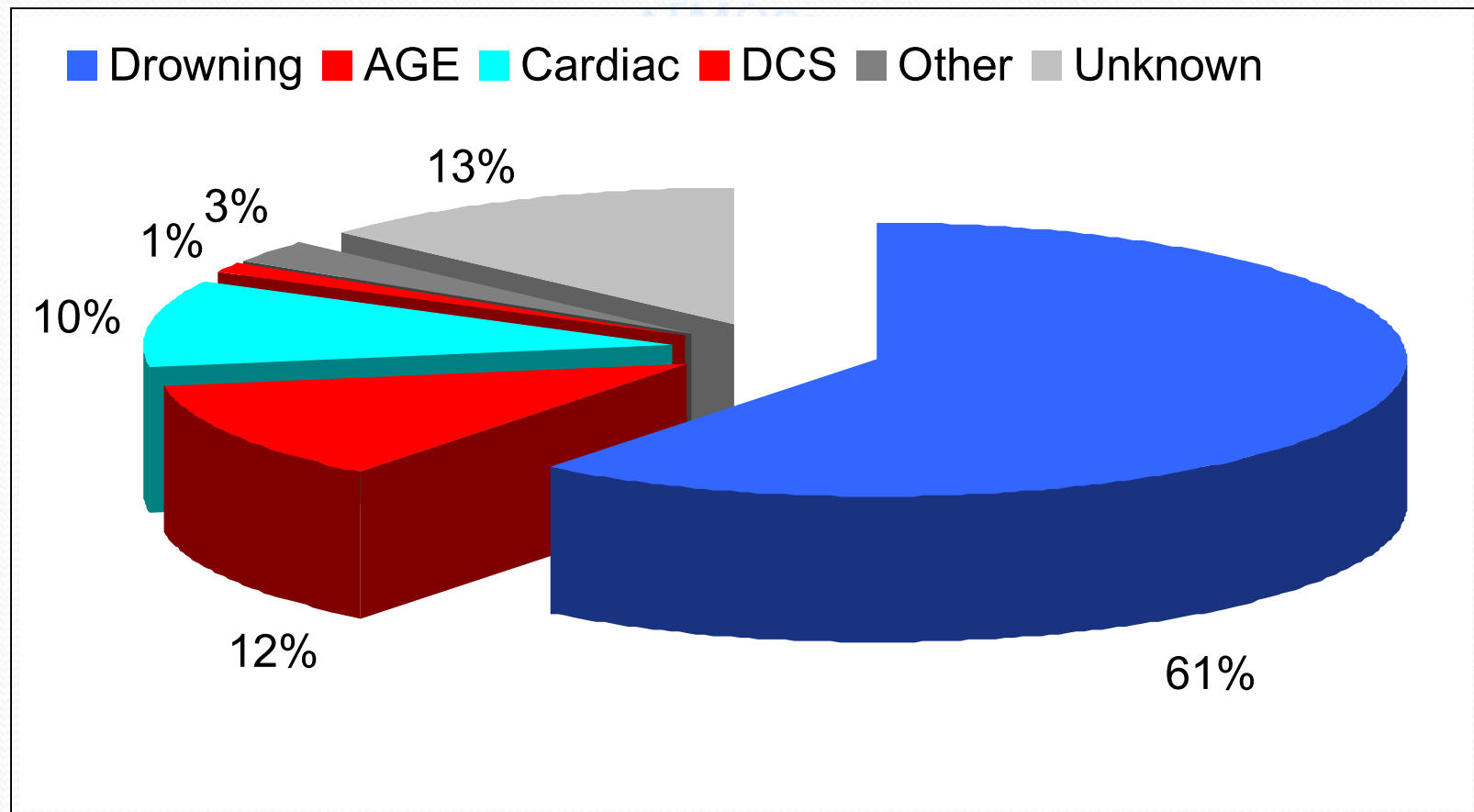


# Symptoms of DCI

- Blurry vision
- Hearing problems
- Balance disturbance (“Staggers”)
  - Inner ear or cerebellar symptoms
- Muscle weakness
- Breathing problems (“Chokes”)
  - May also be immersion pulmonary edema
- Paralysis
- Coma

# Symptoms of DCI

- Death (85 – 90 cases/year in US divers)



Caruso et al, UHMS ASM 2010



# Diagnosis

- Dive History
  - Important details of dive profile ?
  - Onset of symptoms (most within 12-24 hrs)
    - Time, relation to the dive
    - Treatment and evolution of symptoms
  - Interview spouse or buddy
- General Medical history
  - Medications
    - Anti-malaria medications (i.e. Mefloquin)
      - Not a Contraindication to diving

# Drugs and Diving

- Survey 442 British divers age 16-59
  - 65% male, 35% female
- 21% Prescription drugs
- 60% OTC drugs 4-6 hrs before diving
- 22% Recreational drugs since certification
- 20 divers used schedule 1 or 2 drugs within 5 min to 24 hrs prior to diving
  - Cannabis, cocaine, ecstasy 5 min - 6 hrs pre-dive

St. Leger-Dowse et al. DDRC, Plymouth UHMS 2009



# Diagnosis

- History
- Physical examination
  - Look at the entire body
- Detailed neurological examination
  - Findings may not be apparent to the diver
  - Positive findings can be followed
  - No positive findings leaves only subjective symptoms to follow

UHMS ASM 2006

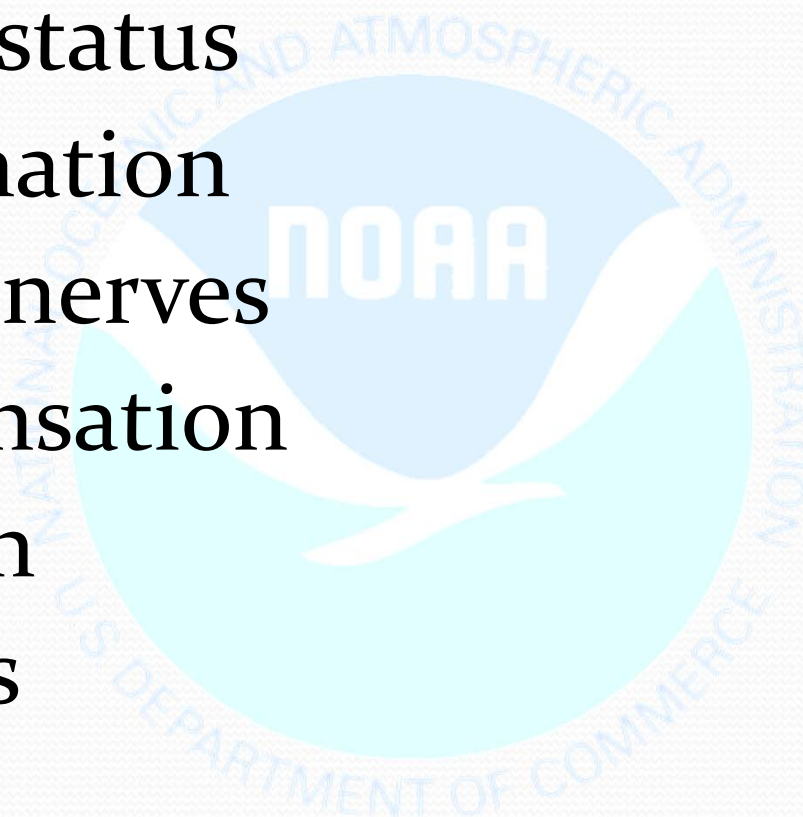
# General Examination

- Vital signs
- Head & Neck
  - Tympanic membranes
- Chest
  - Thorax expansion, auscultation, tracheal deviation
- Heart
- Abdomen
  - Soft, tender, normal peristalsis
- Skin
  - Warm, dry, rash, erythema





# Neurologic Examination

- Mental status
  - Coordination
  - Cranial nerves
  - Skin Sensation
  - Strength
  - Reflexes
- 

# Lab Tests

- Hematocrit (keep < 48%)

>48% persistent neuro sequelae @ 1 month

Boussuges et al. Int. J. of Sports Med 1999

>48% correlated with severe neuro symptoms in females

Newton, Padilla et al; UHMS 35, 2008

- Glucose (especially in unconscious patient)
- EKG if clinical suspicion
- Chest X-Ray
  - If suspicion of pneumothorax
  - Best test for pneumothorax/mediastinum is CT



# Treatment Tables

- Derived by educated guess
- **Field tested !!!!** and fine tuned
- Developed by USN, RN, COMEX etc.
  - For their divers !!!
  - Failure rates may be higher in other populations

Lee JHM 6(1), 1991

- Provide pressure / time / gas profile
- They treat the patient's illness
- Provide tender decompression algorithm

# Air Treatment Tables

- Air as chamber gas and breathing gas
- Take long time
- High failure rate
  - (up to 47% in recreational divers USN TT4)
  - Only 6% when originally used for USN divers
- Used for saturation treatments

Cianci, Slade ASEM 2006

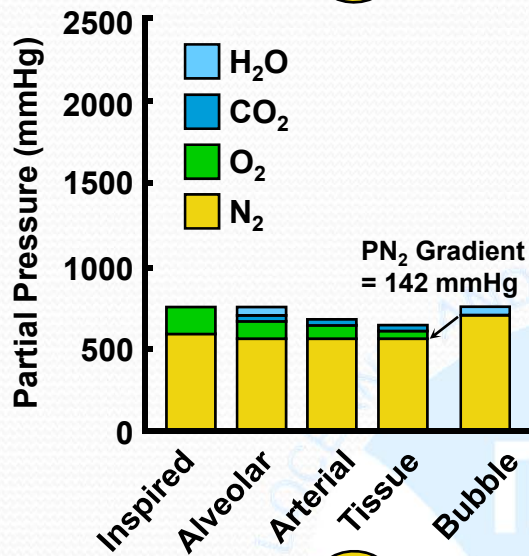


# Oxygen Treatment Tables

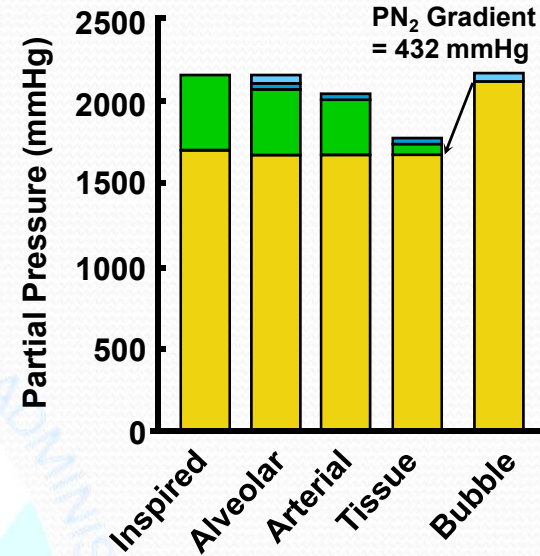
## Most widely used

- Low relapse rate, good track record
- O<sub>2</sub> as breathing gas or chamber gas (mono)
- No on-gassing of inert gas
- HBO inhibits white cell adhesion
  - Hyperbaric air or normobaric O<sub>2</sub> does not
  - For life of leucocyte (24 hrs) ⇒ serial treatments
- Oxygen toxicity risk ⇒ air breaks
- Provides large inert gas gradient

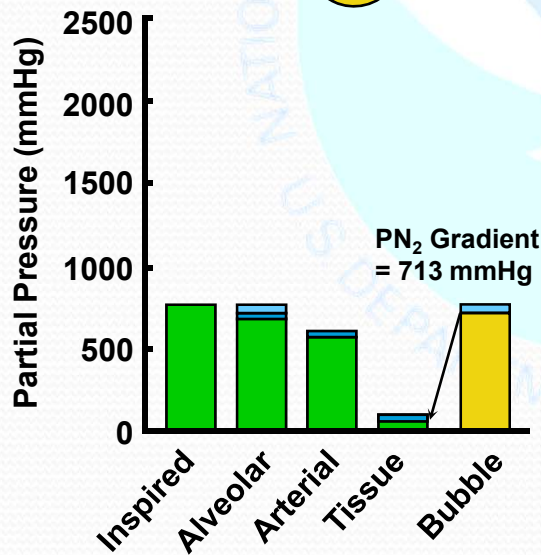
1 ATA  
Air



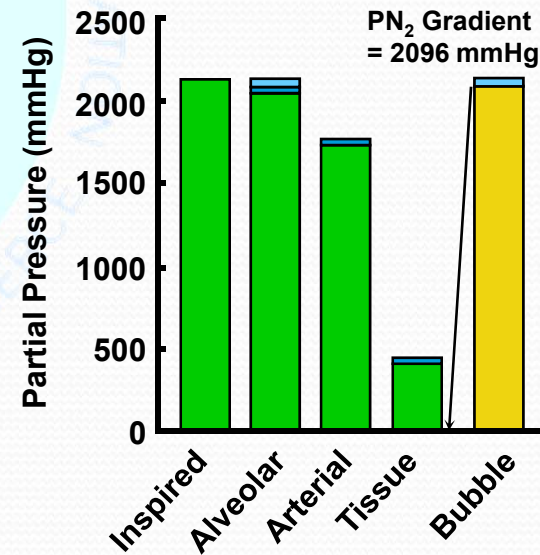
2.8 ATA  
Air



1 ATA  
100% O<sub>2</sub>



2.8 ATA  
100% O<sub>2</sub>





# Mixed Gas Treatment Tables

- Used for “deeper” treatments to dilute O<sub>2</sub>
- Use Heliox or Nitrox as breathing and/or chamber gas in various mixtures
- Incurs an inert gas load
- Complex dynamics and kinetics
  - Counter diffusion superficial or deep
- Gas switching may have benefits ?
- ex: COMEX 30 (with it’s many variables)

# Recompression Treatment

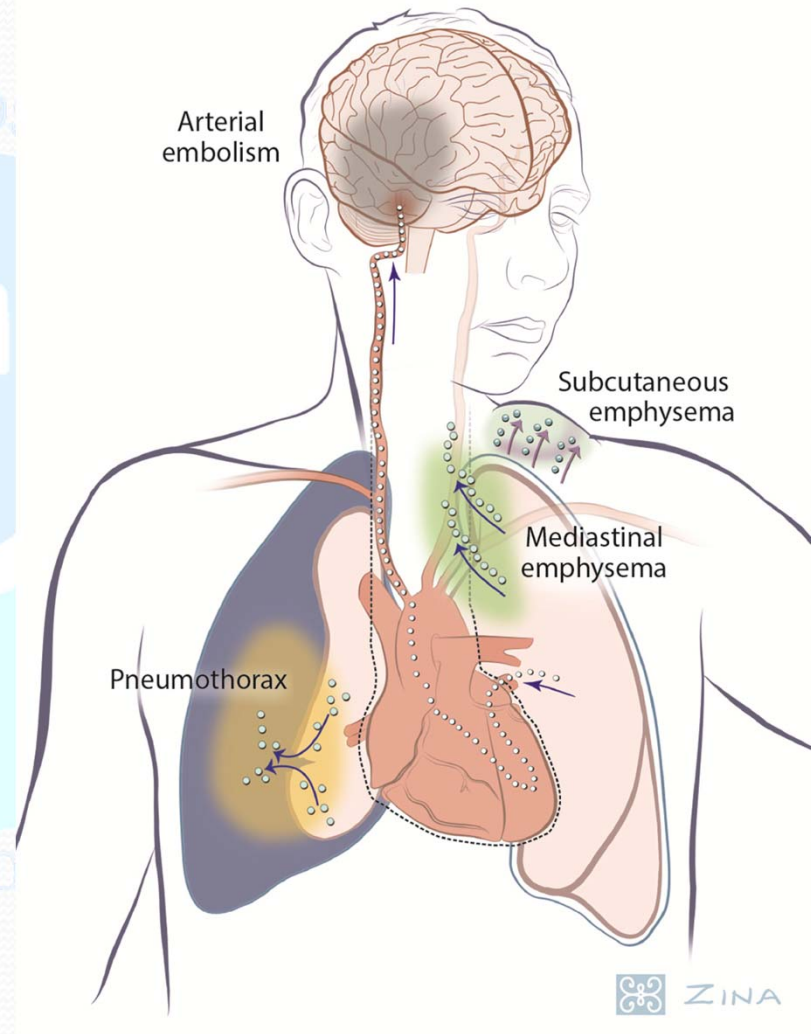
- How late after onset of symptoms do we treat ?
  - Nobody really knows
  - Clinical response to treatment days after onset of symptoms  
Vann, Lancet 337, 2011
  - Up to 2 weeks out, significant improvement
  - Longer intervals ??
  - Short Rx delay more important in mild DCI Severe cases should get transferred to best facility  
Ross UHMS ASM 2007



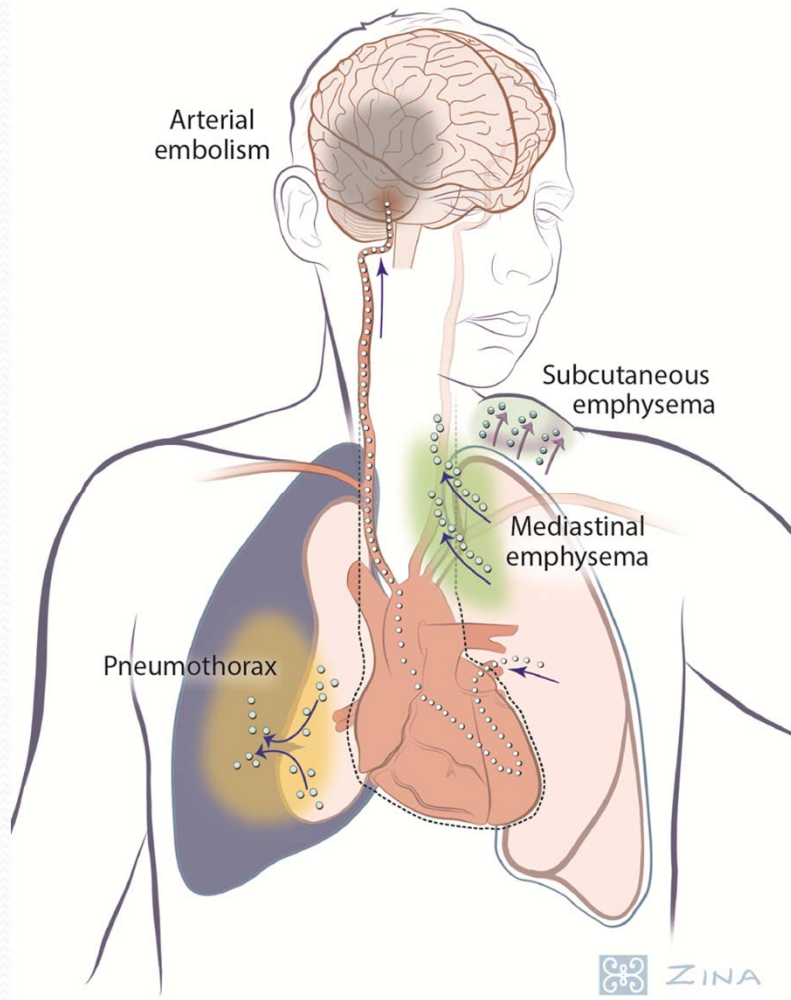
# Pulmonary Barotrauma

- Causes:
  - Holding breath while ascending with compressed air in lungs
  - Air trapped in part of lung from mucus plug or lung defect

Lung injuries are urgent medical emergencies



# Pulmonary Barotrauma



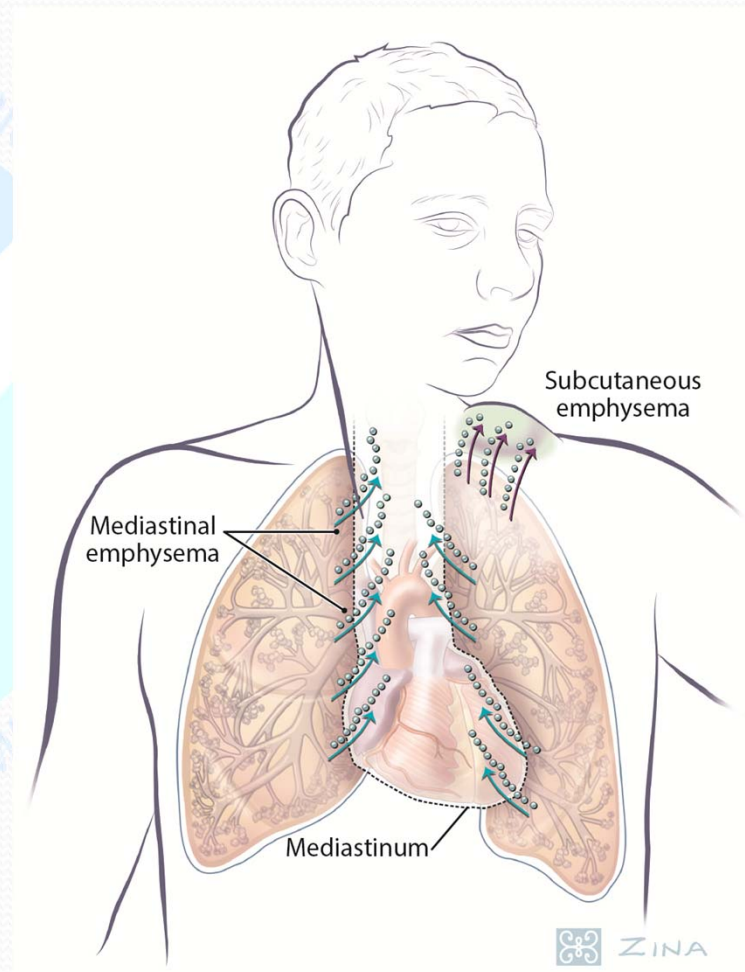
## • Types:

- Mediastinal emphysema: Air in middle of chest
- Subcutaneous emphysema: Air under skin at base of neck
- Pneumothorax: Air in chest cavity - simple or tension
- Arterial gas embolism: Air bubble in artery blocking circulation



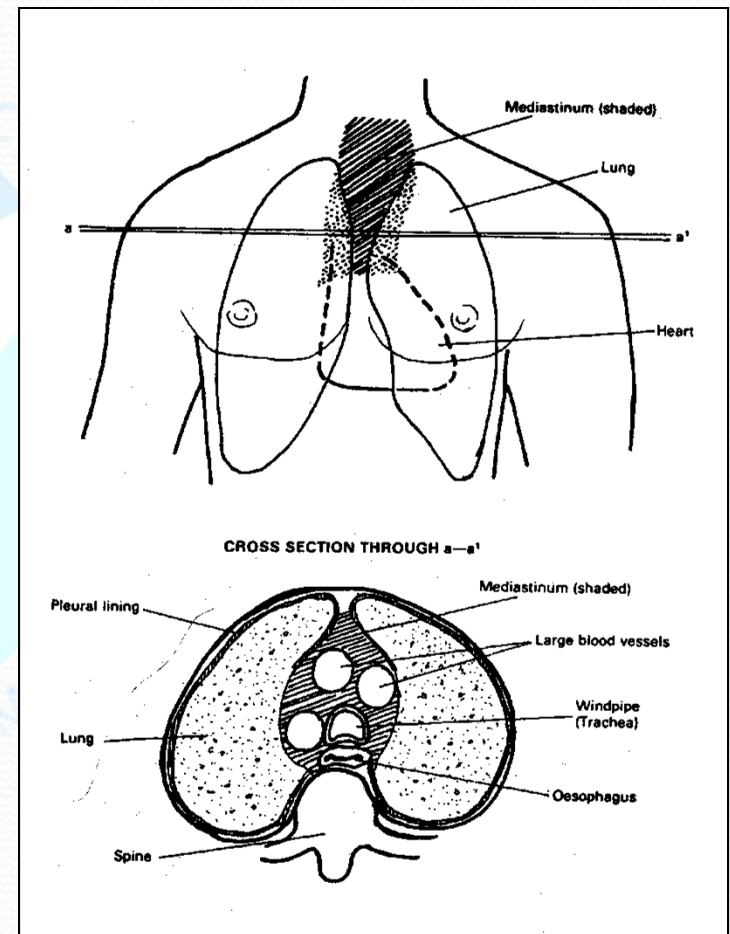
# Mediastinal Emphysema

- Cause:
  - Over-pressurization of lungs resulting in a tearing of the alveoli allowing gas to escape into the tissues surrounding the lung. Gas tracks along the lung tissues to the area under the breastbone



# Mediastinal Emphysema

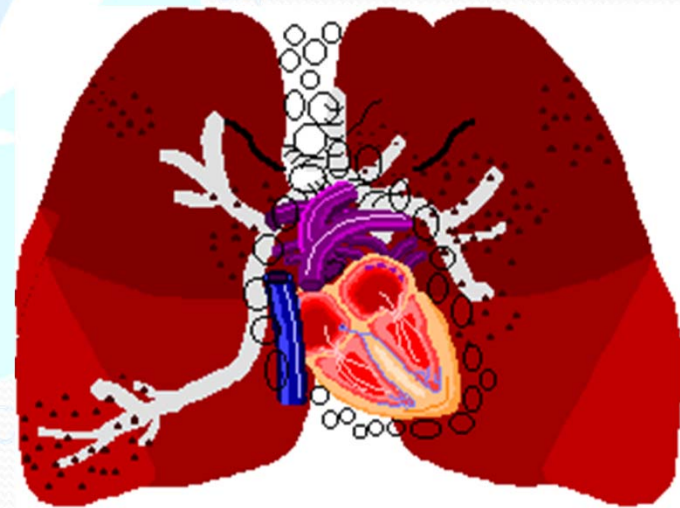
- Effect:
  - Air expanding in middle of chest may affect circulation and breathing



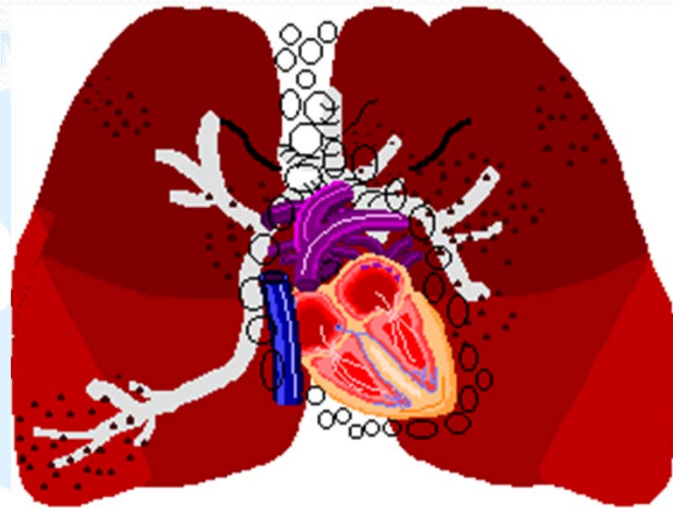


# Mediastinal Emphysema

- Signs & symptoms:
  - Sudden, severe pain in chest
  - Shortness of breath
  - Possible fainting
  - Difficulty breathing



# Mediastinal Emphysema

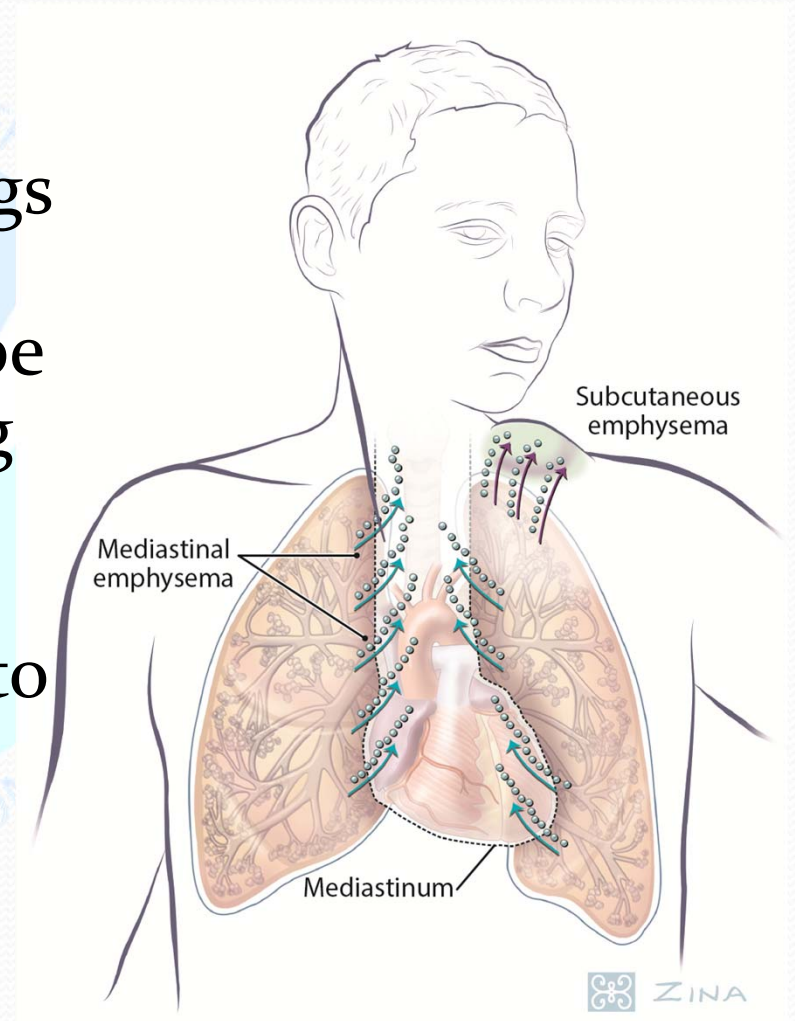


- Treatment:
  - CAB
  - Administer oxygen and monitor for shock
  - Examine diver for other signs of pulmonary barotrauma
  - Mediastinal emphysema causing respiratory or circulatory impairment may require recompression



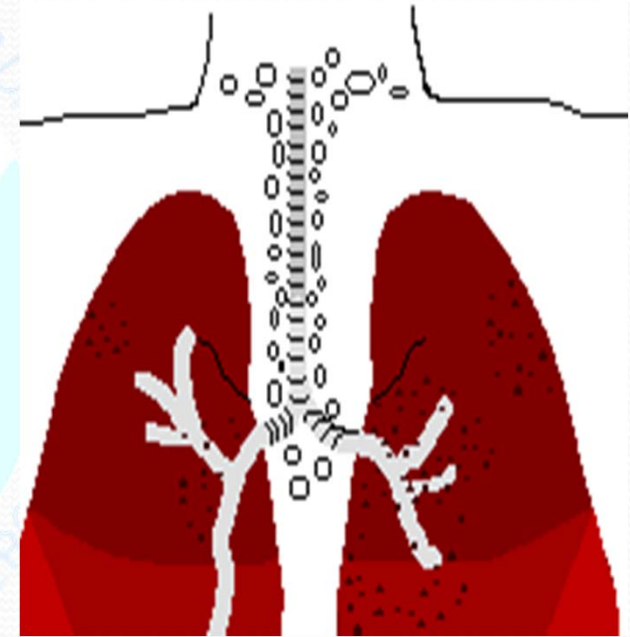
# Subcutaneous Emphysema

- Cause:
  - Over-pressurization of lungs resulting in a tearing of the alveoli allowing gas to escape into the tissues surrounding the lung. Gas tracks along the lung tissues to the area under the breastbone then to the neck region



# Subcutaneous Emphysema

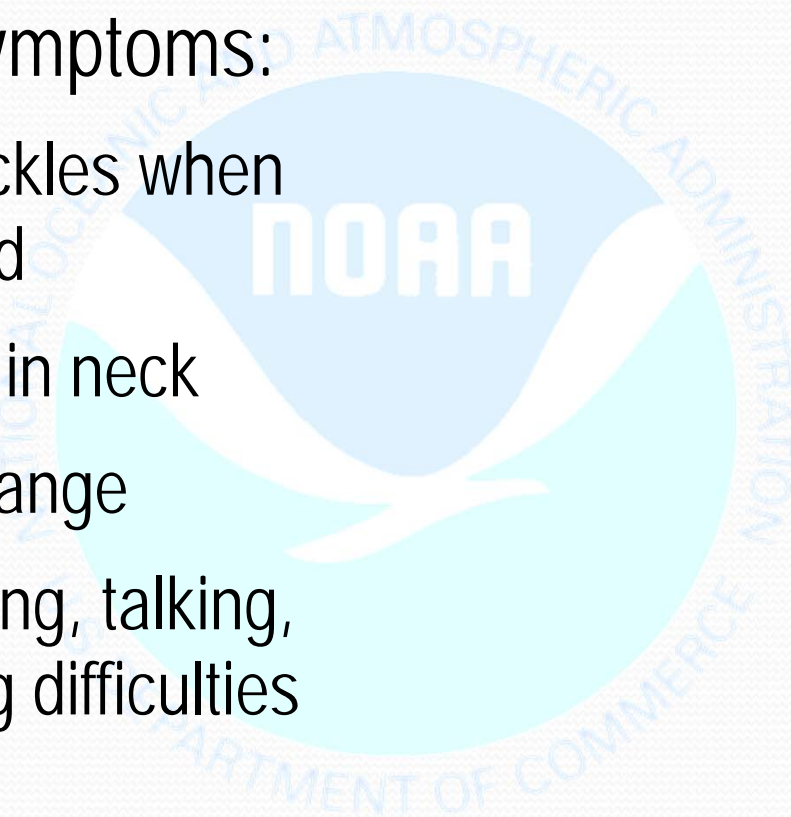
- Effect:
  - Air expanding under skin at base of neck may affect swallowing, talking, and breathing





# Subcutaneous Emphysema

- Signs & symptoms:
  - Skin crackles when squeezed
  - Fullness in neck
  - Voice change
  - Swallowing, talking, breathing difficulties



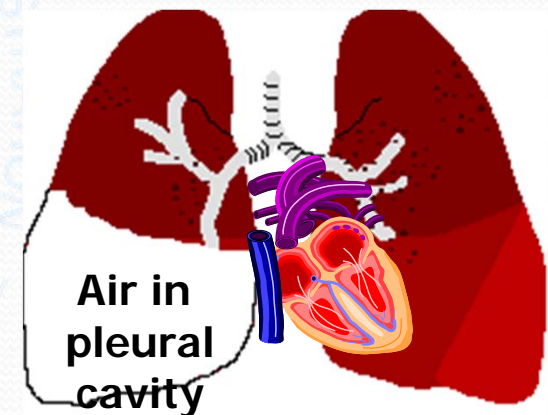
# Subcutaneous Emphysema

- Treatment:
  - CAB
  - Administer oxygen and monitor for shock
  - Examine diver for other signs of pulmonary barotrauma
  - Recompression not normally required



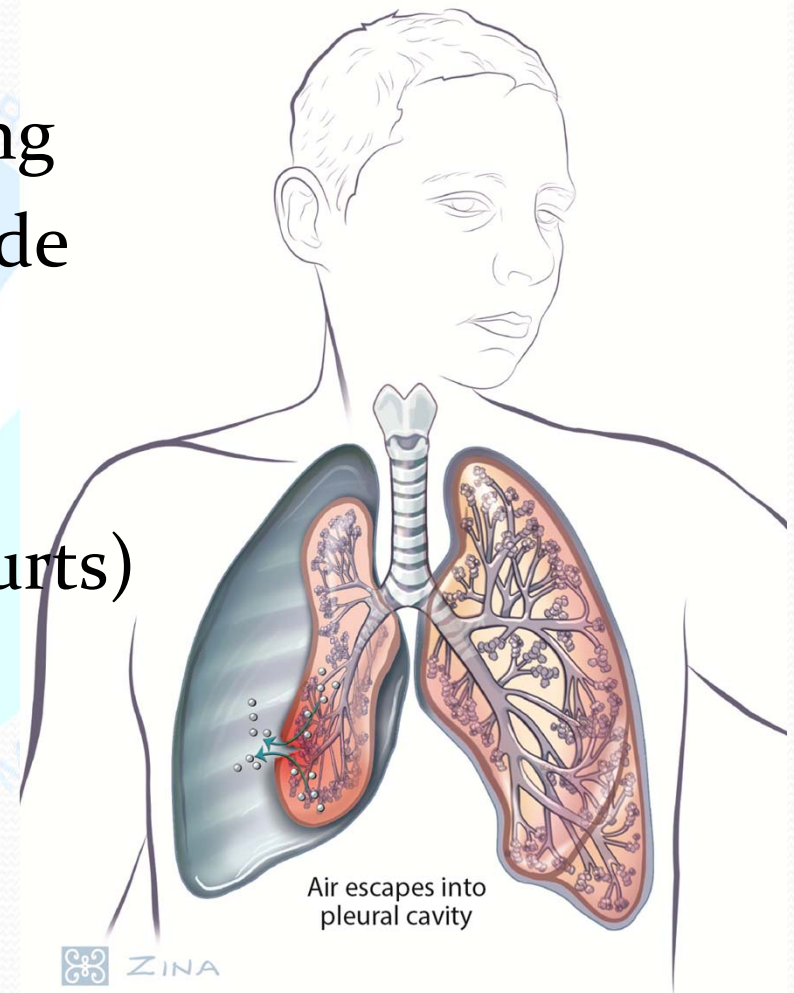
# Pneumothorax

- Simple Pneumothorax:
  - Cause: Lung over-pressurization resulting in a one-time leakage of air into the pleura space between the lungs and chest wall
  - Effect: Lung partially collapses
- Tension Pneumothorax:
  - Cause: Air continues to enter but not exit the chest cavity with each successive breath thus progressively enlarging the air pocket
  - Effect: Lung totally collapses - expanding air exerts pressure on heart, trachea, esophagus, etc.



# Pneumothorax

- Signs and symptoms:
  - Difficulty or rapid breathing
  - Leaning toward affected side
  - Hypotension
  - Cyanosis & shock
  - Chest pain (deep breath hurts)
  - Shortness of breath
  - Decreased or absent lung sounds on affected side
  - Death





# Pneumothorax

- Treatment:
  - Simple Pneumothorax:
    - Normally improves with time as air is reabsorbed
    - Monitor for signs of tension pneumothorax
    - Monitor ABC's and administer oxygen
    - Transport to nearest medical facility
  - Tension Pneumothorax:
    - Position patient on injured side
    - ABC's
    - Treat for shock & administer 100% oxygen
    - Transport immediately to nearest medical facility (air must be vented from chest cavity)

Pulmonary Barotrauma

Alveolar rupture

Alveolar hemorrhage

Extravascular air

Air into periaveolar capillaries

Hemoptysis

Air into left heart

Ventilation/perfusion abnormalities

Perivascular air tracking

Air into aorta

Pleura

Mediastinum

Coronary emboli  
Cerebral emboli  
Systemic emboli

Pneumothorax

Subcutaneous emphysema

Mediastinal emphysema

Pneumopericardium  
Pneumoperitonium



Pulmonary Barotrauma

Alveolar rupture

Alveolar hemorrhage

Extravascular air

Air into periaveolar capillaries

Hemoptysis

Air into left heart

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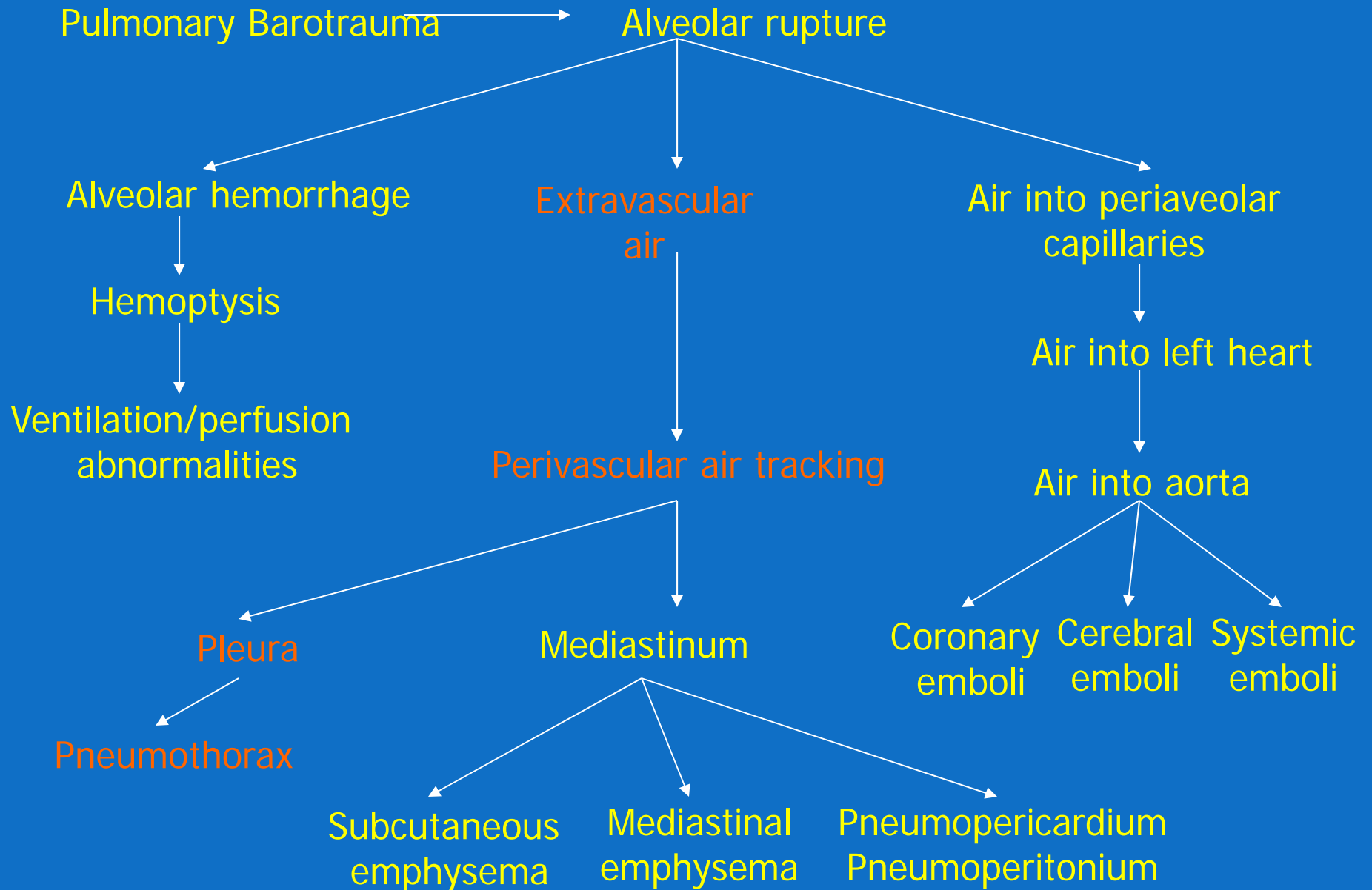
Coronary emboli  
Cerebral emboli  
Systemic emboli

Pneumothorax

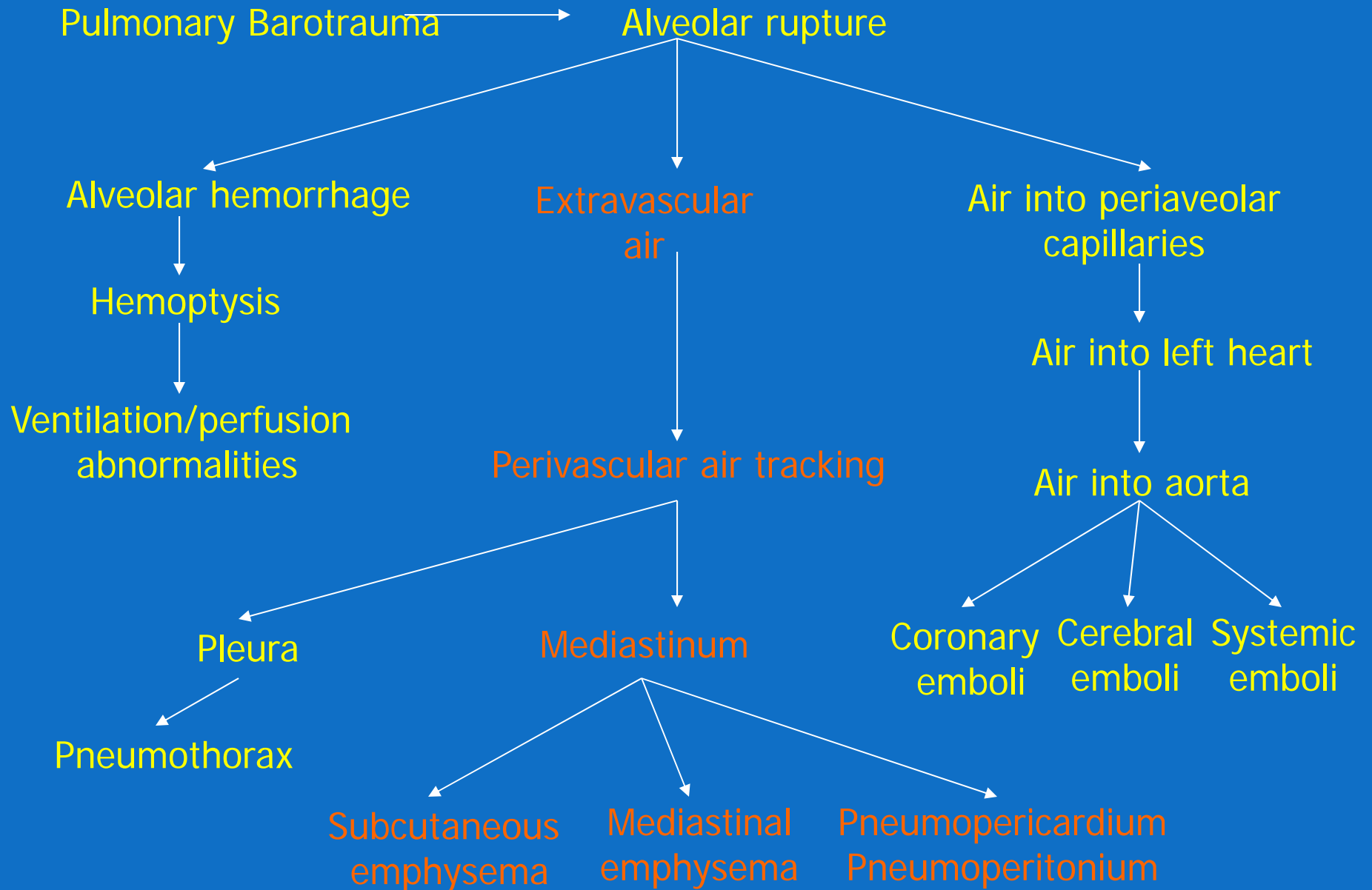
Subcutaneous emphysema

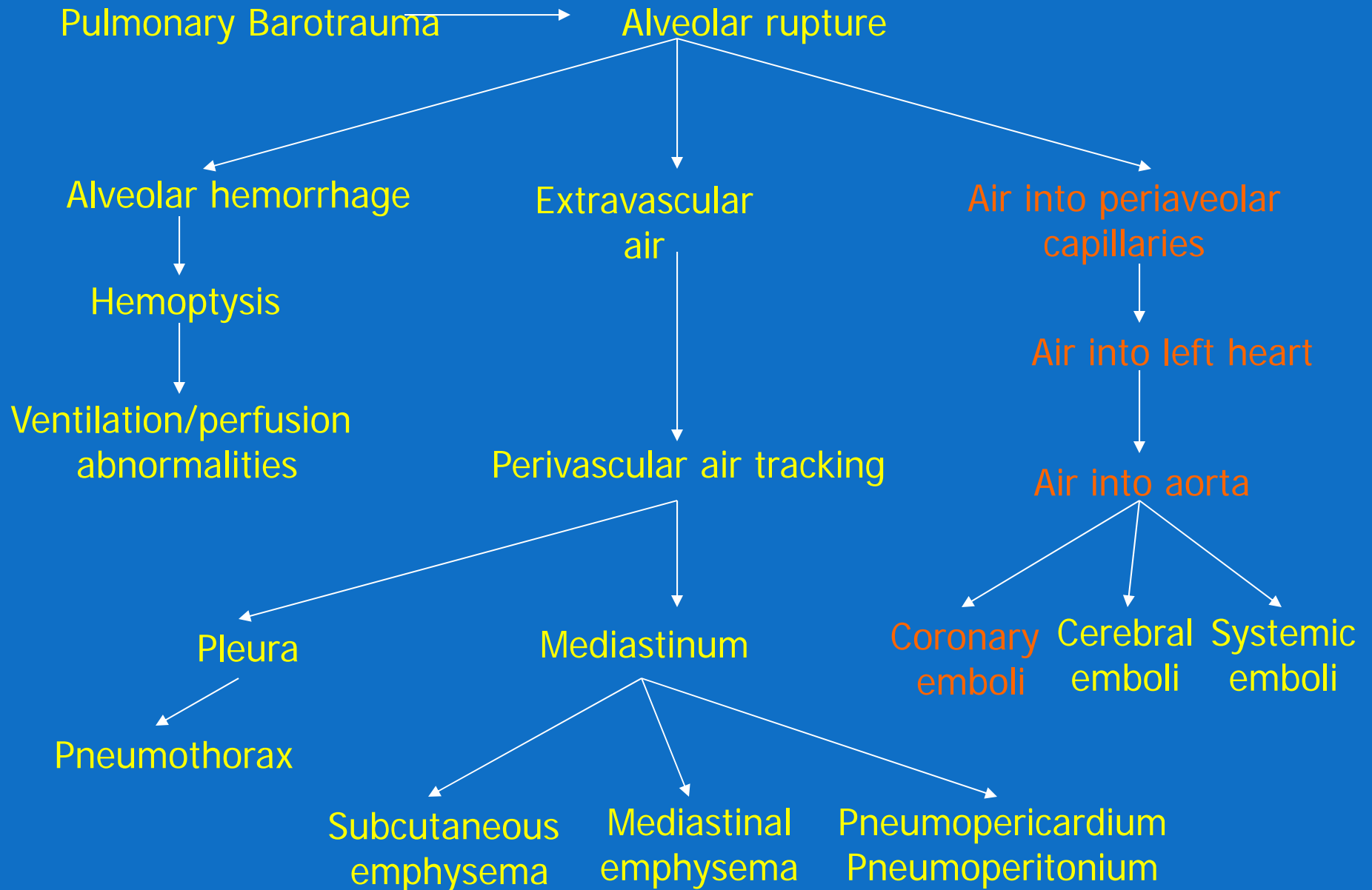
Mediastinal emphysema

Pneumopericardium  
Pneumoperitonium

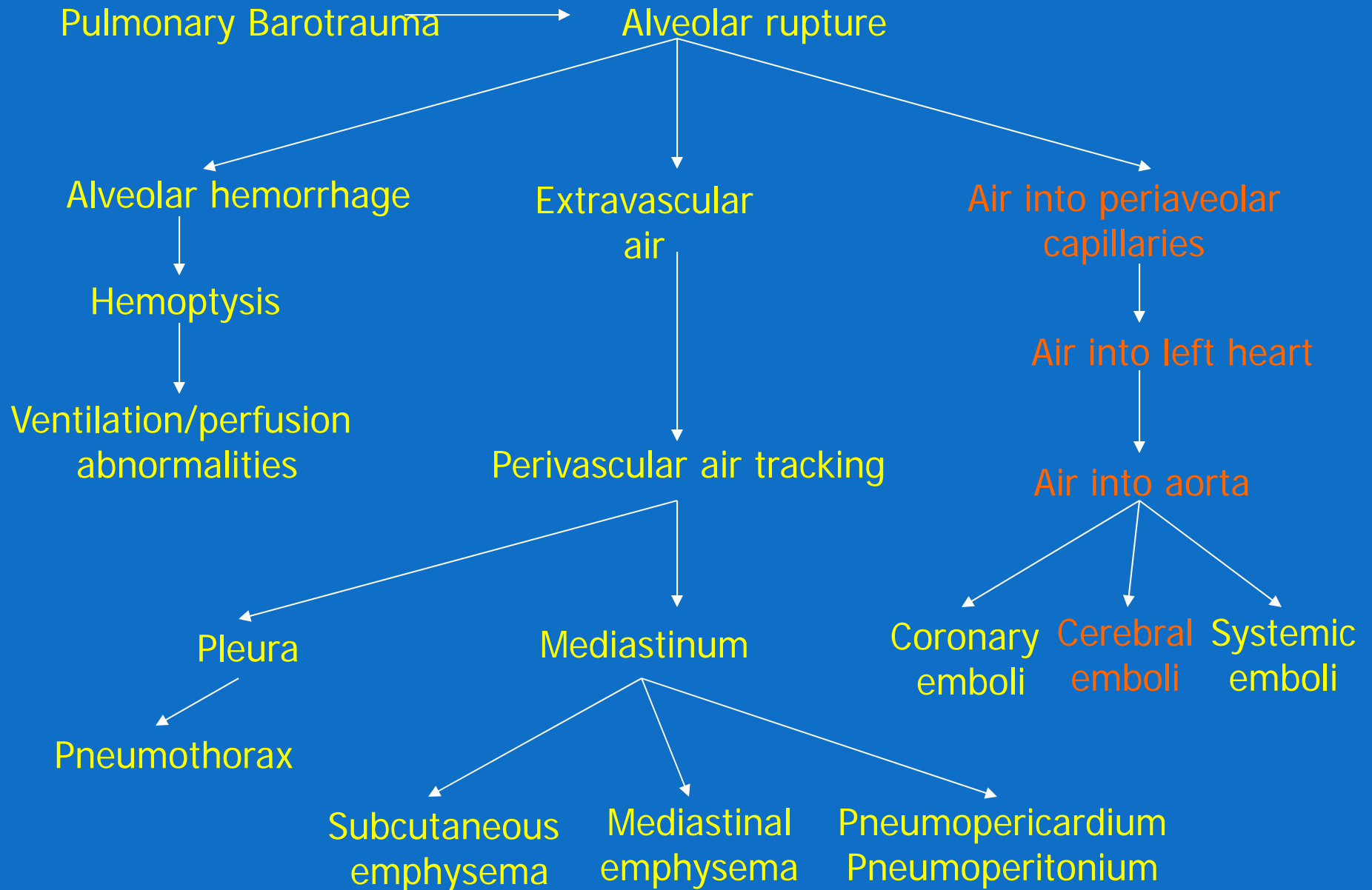


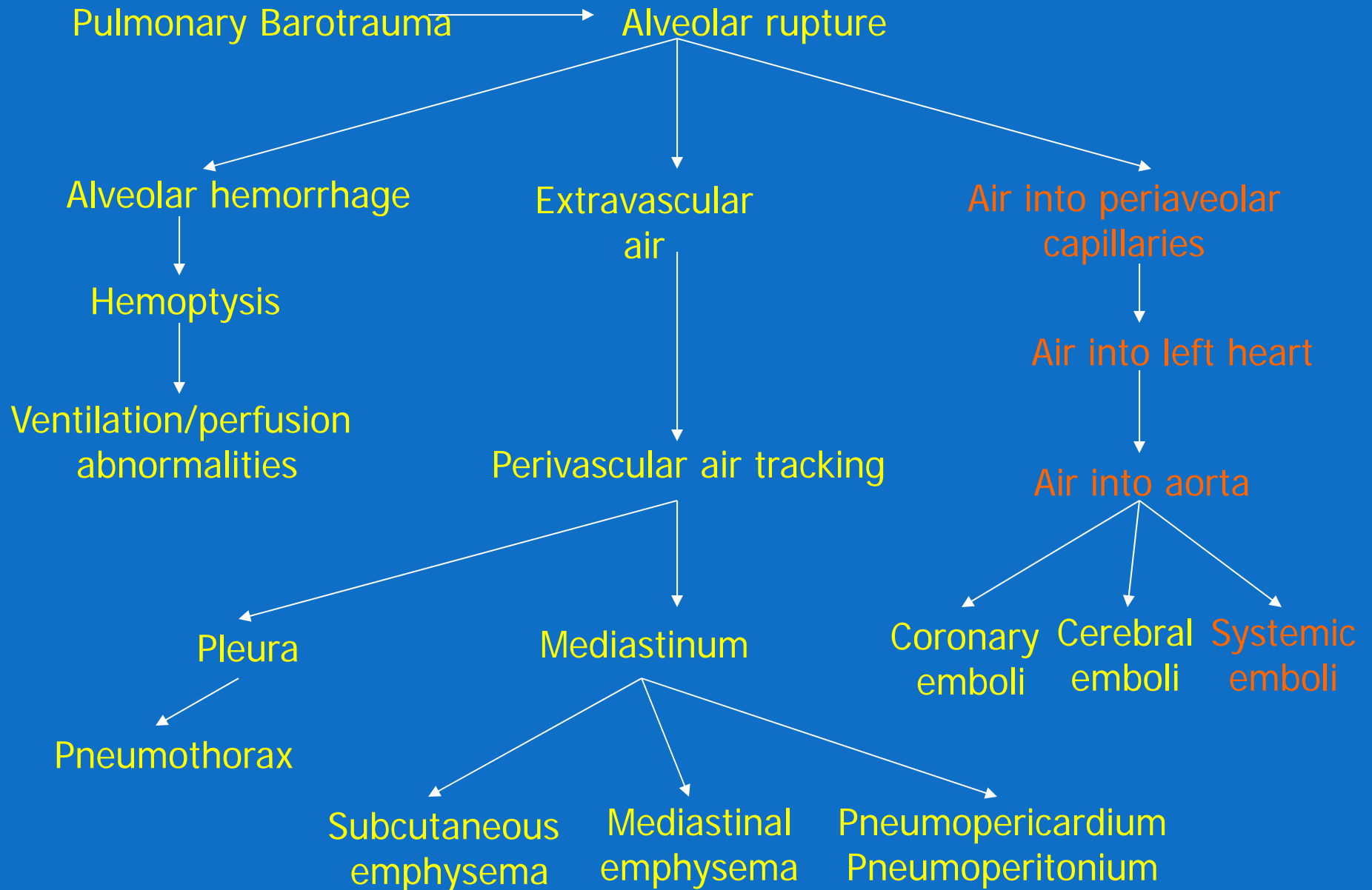










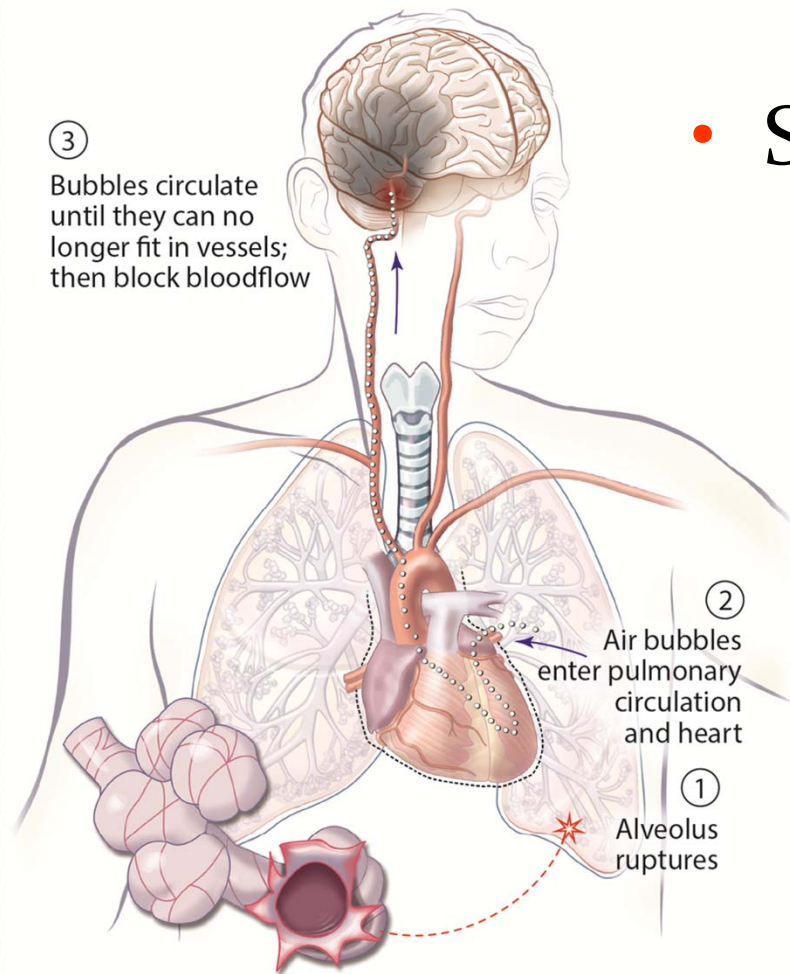




# Arterial Gas Embolism

- Cause:
  - Over-pressurization of lungs resulting in the tearing of alveoli allowing gas to enter the blood circulation. Bubbles are conducted to the left side of the heart then to other parts of the body through the arterial circulatory system
- Effect:
  - Air bubbles block arteries--usually in brain

# Arterial Gas Embolism



- **Signs & symptoms:**
  - Disturbances of the brain function
    - Sensation (i.e. numbness or tingling)
    - Movement (i.e. paralysis or weakness)
    - Vision
    - Speech
    - Balance or coordination
  - Chest pain
  - Shortness of breath
  - Bright red frothy sputum



# Gas Embolism

- The entry of gas into vascular structures
- Common iatrogenic problem
- Can occur from procedures in almost all specialties
- Potential for serious morbidity/mortality
- Usually gas embolism = air embolism
  - Occasional carbon dioxide, nitrogen, helium
- Venous
- Arterial

# Mechanisms of Gas Entry

- Extracorporeal-bypass circuit operations
  - Air entering circuit
  - Incomplete removal of air from heart after cardioplegic arrest
  - Carbon dioxide-assisted harvesting of peripheral veins
- Pulmonary barotrauma
  - SCUBA
  - Positive pressure mechanical ventilation
- Rupture of intaortic balloon



# Arterial Gas Embolism

- Treatment – Hyperbaric Oxygen
  - The patient breaths 100% oxygen at a pressure above that of the atmosphere at sea level
  - Reduces the size of the bubble by raising the ambient pressure
  - Reduces the size of the bubble by increasing the oxygen window
    - Enormous diffusion gradient for oxygen into the bubble and for nitrogen out of the bubble
  - Arterial partial pressure of oxygen of greater than 2000 mm Hg is common
    - Increases the extent of oxygen diffusion into the tissues

# Arterial Gas Embolism

- Treatment – Hyperbaric Oxygen
  - Proposed benefits
    - Prevent cerebral edema by reducing the permeability of blood vessels while supporting the integrity of the blood-brain barrier
    - Diminish the adherence of leukocytes to damaged endothelium (Thom 1997)
  - Considered to be first-line treatment of choice for arterial gas embolism as soon as cardiopulmonary stabilization has been achieved



# Questions?

