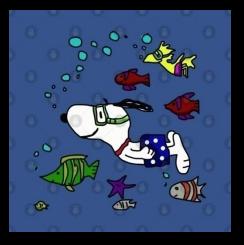




Dive Physiology









Your Instructor

U of MI Diving Safety Coordinator AAUS sanctioned Diving Safety Officer Internationally rated 3 - star instructor (CMAS)

National Master Scuba Instructor (President's Council)

- > 100 Diving Certifications
- > 200 Diving Publications

> 1,200,000 visitors to "Diving Myths & Realities" web site

Library: one of the best resources in North America

Scuba Diver since 1977 Scuba Instructor since 1980 DAN Instructor since 1991 EAN_x Instructor since 1992 Ph.D. Biochemistry



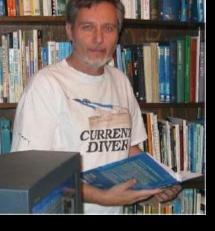










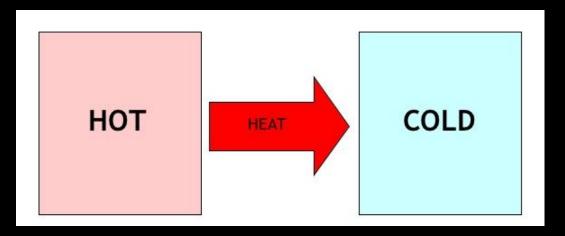




Heat



Heat is a Fluid

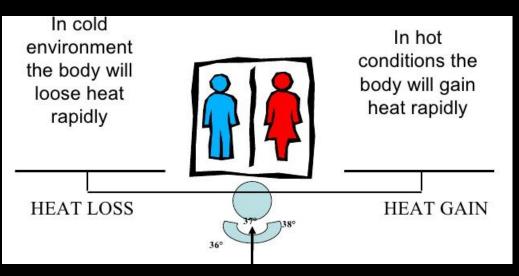


Flows from hotter to colder until temperatures are equal

Cannot stop movement Protection comes from slowing process

Thermal Balance

Heat Out: Environment Conduction Convection **Radiation** Cooling **Respiration Perspiration Excretion** Disease

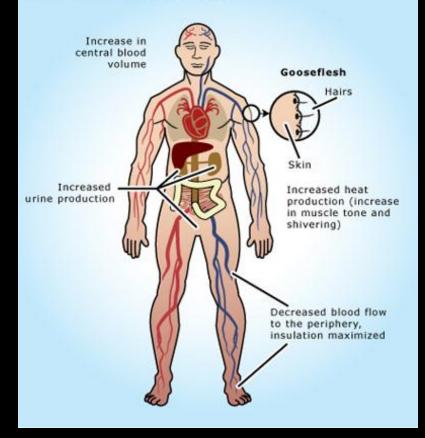




Heat In: Metabolism Muscle Movement Environment Conduction Radiation

Humans are Tropical Critters Better coping with heat than cold

How the body responds to cold



Hot

Vasodilation

Arterioles dilate (enlarge) so more blood enters skin capillaries and heat is lost.

Sweating

Sudorific glands secrete sweat which removes heat when water changes state.

Pilorelaxation

This means the hairs flatten.

Stretching Out

By opening up, the body was a larger surface area.

Cold

Vasoconstriction

Arterioles get smaller to reduce blood going to skin: keeping core warm.

Shivering

Rapid contraction and relaxing of skeletal muscles. Heat produced by respiration.

Piloerection Hairs on skin stand up.

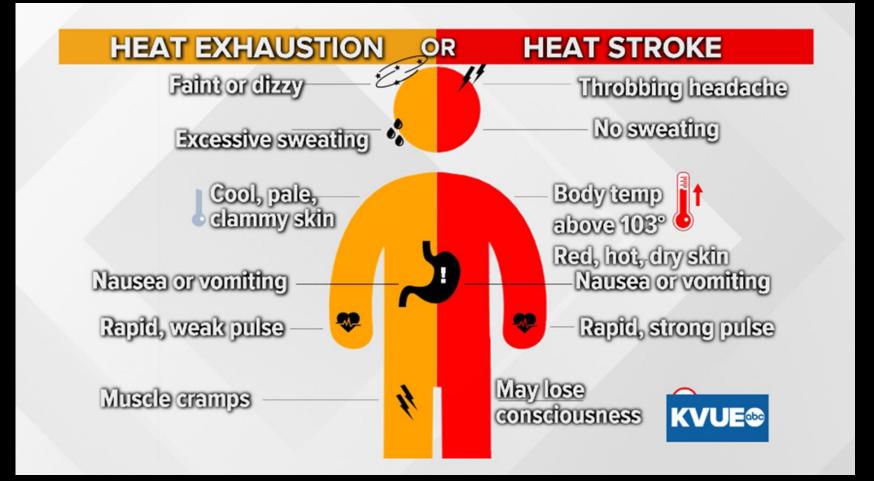
Curling Up Making yourself smaller so smaller surface area.



Hyperthermia



Hyperthermia Body temperature too high



Hyperthermia: Symptoms

40

30

20

10



nausea, vomiting, fatigue, weakness, headache, muscle cramps, aches, and dizziness.

Heat Stroke

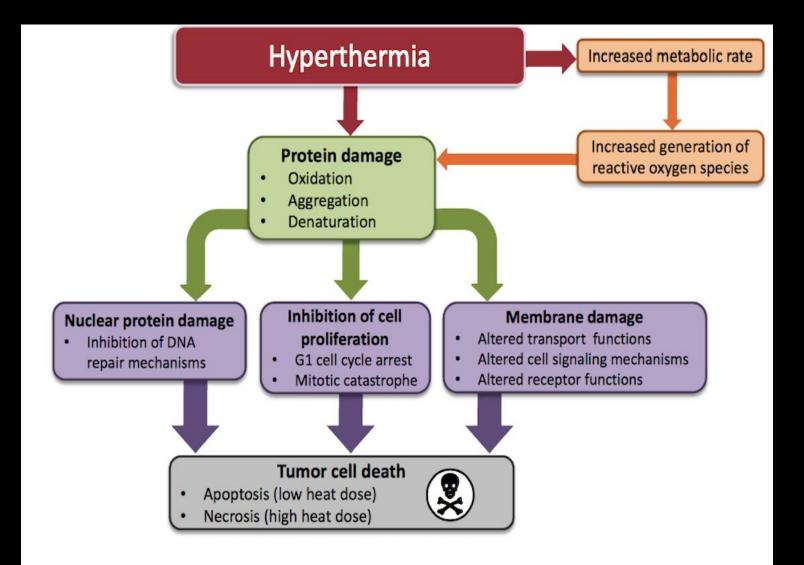
high body temp, absence of sweating, hot red or flushed dry skin, rapid pulse, difficulty breathing, strange behavior, hallucinations, confusion, agitation, disorientation, seizure, and/or coma.



lack of breathing, no heart beat, silence, rigor, and complete reduction in running pace.

anywhere5k.com

Hyperthermia: Biochemistry





Hypothermia



Must Understand Heat Loss to Protect From the Cold



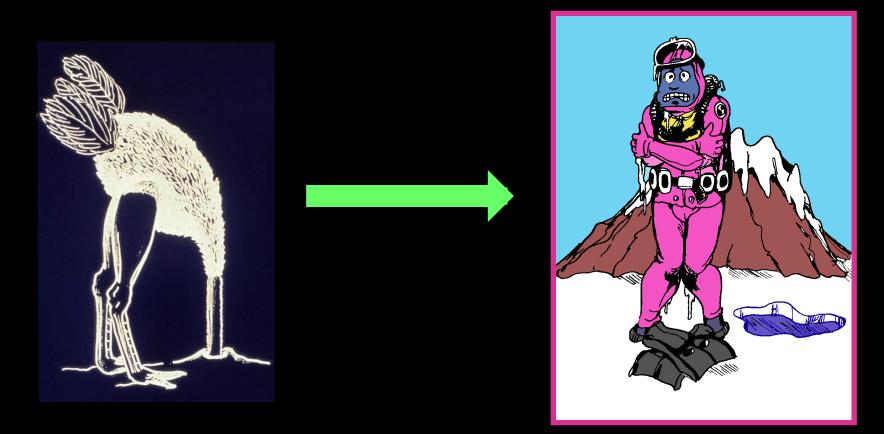






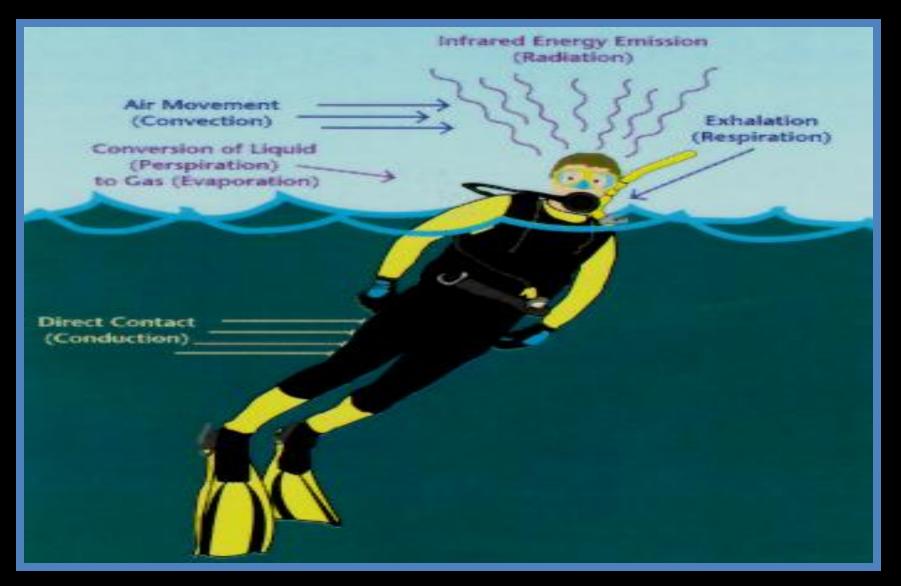


Ignoring (or Not Understanding) Heat Loss

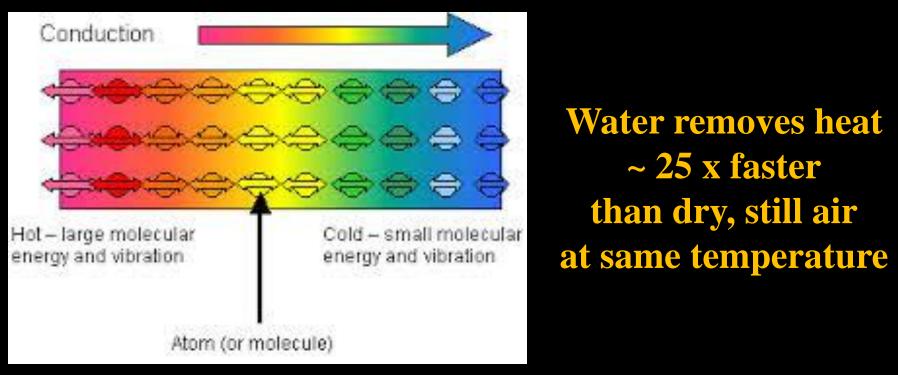


It is always the "not known" that poses the greatest risk

In-Water Heat Loss

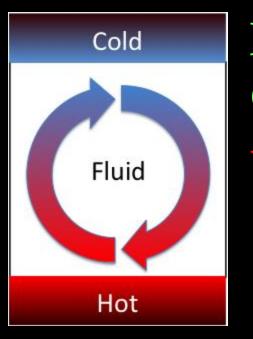


Thermal Loss: Conduction



Direct transfer of energy at the molecular level Major source of in-water heat loss Heat loss to water Heat loss warming breathing gas

Thermal Loss: Convection



Moving liquid removes heat Continual process Wet suits restrict convective flow

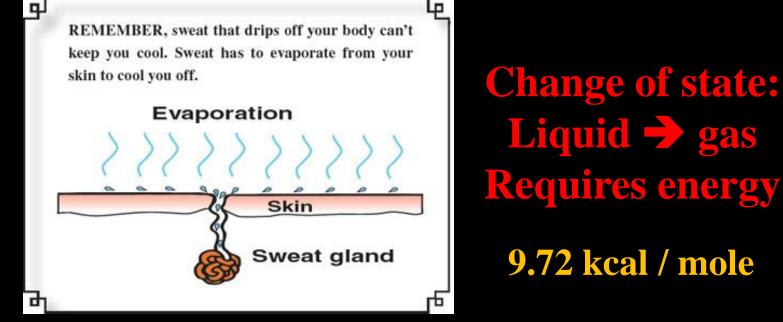


Thermal Loss: Radiation

Emission of infra red radiation

Minor problem in the water

Thermal Loss: Evaporation / Respiration



Pre-dive sweating Insensible perspiration Humidifying dry breathing gas

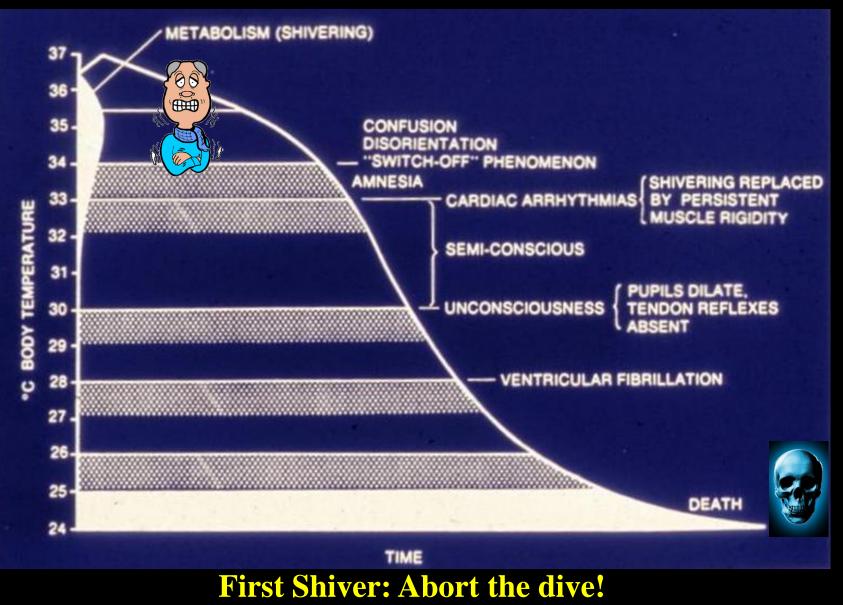
Simplistic View of a Biochemist: Heat = Life

Cold Robs Heat No Heat = No Life



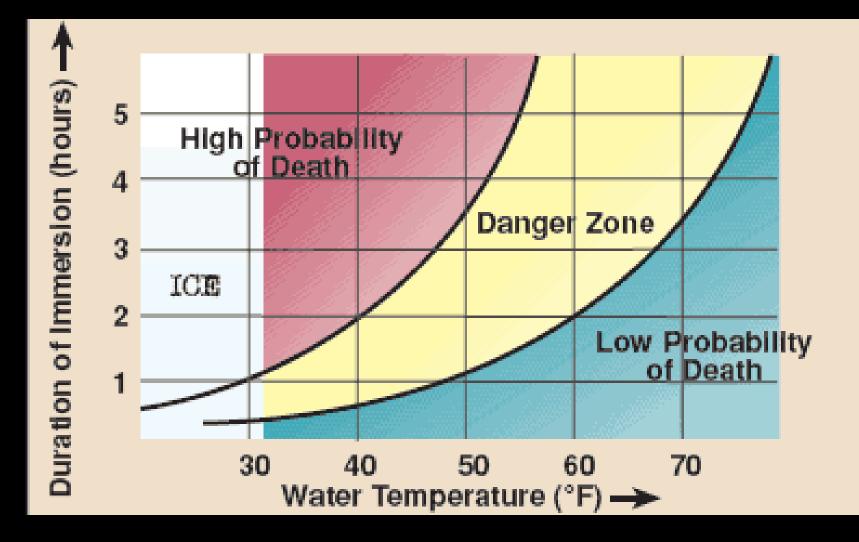


Body Response to Temperature Loss



LPT

Estimated Unprotected In-Water Survival Time



Wind Chill Can Lead to Substantial Heat Loss



Wind Chill Chart 🄇

Temperature (°F)																			
	Calm	40	35	30	25	20	15	10	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45
Wind (mph)	5	36	31	25	19	13	7	1	-5	-11	-16	-22	-28	-34	-40	-46	-52	-57	-63
	10	34	27	21	15	9	3	-4	-10	-16	-22	-28	-35	-41	-47	-53	-59	-66	-72
	15	32	25	19	13	6	0	-7	-13	-19	-26	-32	-39	-45	-51	-58	-64	-71	-77
	20	30	24	17	11	4	-2	-9	-15	-22	-29	-35	-42	-48	-55	-61	-68	-74	-81
	25	29	23	16	9	3	-4	-11	-17	-24	-31	-37	-44	-51	-58	-64	-71	-78	-84
	30	28	22	15	8	1	-5	-12	-19	-26	-33	-39	-46	-53	-60	-67	-73	-80	-87
	35	28	21	14	7	0	-7	-14	-21	-27	-34	-41	-48	-55	-62	-69	-76	-82	-89
	40	27	20	13	6	-1	-8	-15	-22	-29	-36	-43	-50	-57	-64	-71	-78	-84	-91
	45	26	19	12	5	-2	-9	-16	-23	-30	-37	-44	-51	-58	-65	-72	-79	-86	-93
	50	26	19	12	4	-3	-10	-17	-24	-31	-38	-45	-52	-60	-67	-74	-81	-88	-95
	55	25	18	11	4	-3	-11	-18	-25	-32	-39	-46	-54	-61	-68	-75	-82	-89	-97
	60	25	17	10	3	-4	-11	-19	-26	-33	-40	-48	-55	-62	-69	-76	-84	-91	-98
				I	Frostb	ite Tin	nes	30 minutes 📃 10 minutes 📃 5 minutes											
Wind Chill (°F) = 35.74 + 0.6215T - 35.75(V ^{0.16}) + 0.4275T(V ^{0.16}) Where, T= Air Temperature (°F) V= Wind Speed (mph) <i>Effective 11/01/01</i>																			

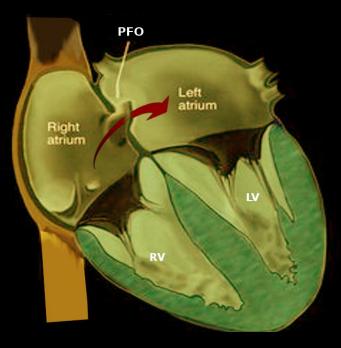




Cold: Major physiological stressor Major obstacle to diving participation Major obstacle to limited bottom time

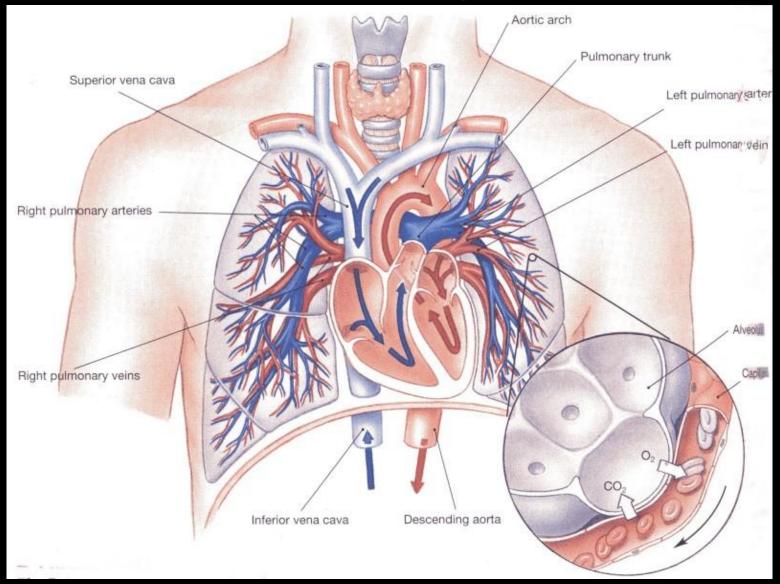
Thermal protection is part of life support

Patent Foramen Ovale

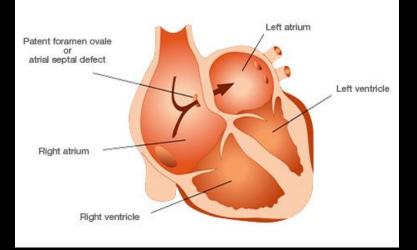




General Heart-Lung Circulation



Patent Foramen Ovale Opening in septum secundum



Patent: open Foramen: aperture in tissue or bone Ovale: oval shaped

Present in: Unborn (mom functions as lungs) ~25 – 30 % of population ~ 6% large opening 5% of serious DCS appear

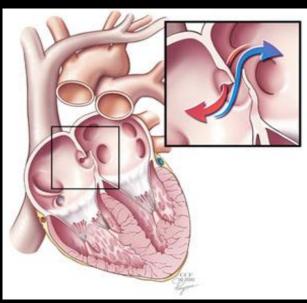
~ 5% of serious DCS cases

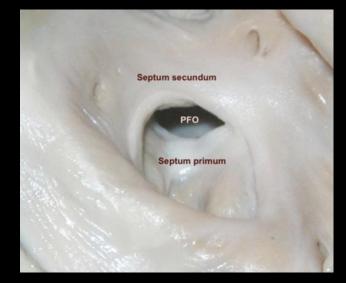
PFO:

Some blood flow bypasses the lungs (bubble filter) Bubbles in circulation: can pass into arterial circulation (Best to assume we bubble on every dive ascent) Possible source of CNS lesions seen in brain and spinal cord



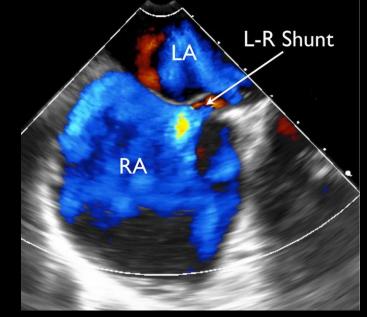
PFO: Allows Direct Path to Arterial Circulation Bubbles can move into arterial circulation





Can lead to: CNS lesions Severe neurological DCS Air embolism on descent

Diagnosed with Ultrasound

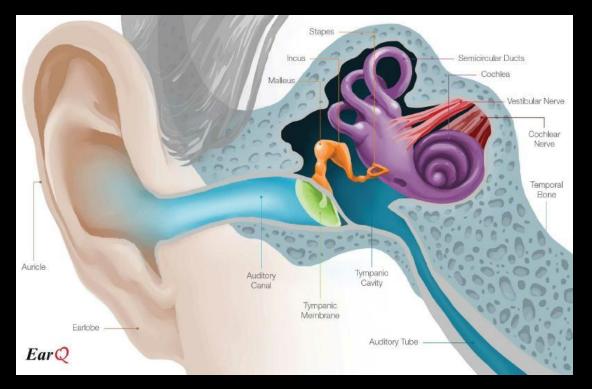




Ear Issues

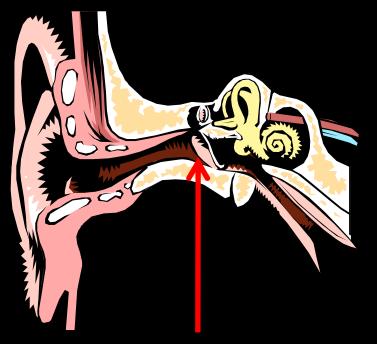


"Clearing" The Ears



> 80 % of basic students suffer ear barotrauma on first open water George Hapur (Canadian Hyperbaric Physician)

Equalizing ("Clearing") Middle Ear Pressure "Clearing" equalizes pressure across the tympanic membrane



On Descent: Outer Pressure > Middle Ear Pressure Tympanic Membrane Moves Inward

On Ascent: Middle Ear Pressure > Outer Pressure Tympanic Membrane Moves Outward

Tympanic Membrane Separates Outer and Middle Ear Transmits Vibrations to Middle Ear

> **Too much movement** (~ 8 fsw change) can rupture the ear drum Possible ear infection from water entering the middle ear

"Clearing" Techniques: (Most often a problem on descent)

Common Techniques: Valsalva: Pinch nostrils and blow Toynbee: Pinch nostrils and swallow

For all descents: Start prior to descent Slowly move feet first Look up If feeling pressure: Ascend a bit to relieve Extend jaw forward



Do NOT swallow Air in stomach can expand on ascent This can rupture the stomach

The Valsalva Maneuver



Pinch Nostrils and Gently Blow Most Taught Technique Vigorous Valsalva - Dangerous technique **Builds Internal Pressure Transmitted via CSF to Brain Possible Round Window Rupture Can drive bubbles thru PFO (if present) Possible air embolism on descent Can Constrict Eustachian Tubes**

Frenzel Technique

Developed During WWII For German Stuka Pilots

Rapid pressure increase during descent Pilots needed both hands on control stick Frenzel developed for hands free clearing



Frenzel Technique

Hands Free Equalization of Ear Pressures

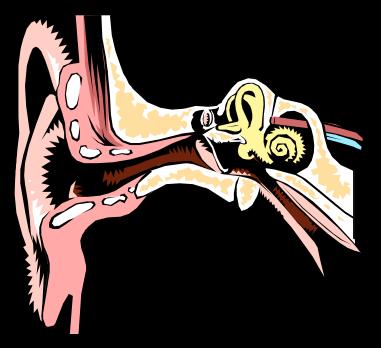
Place tongue on the roof of the mouth... as far forward as possible Hold tongue there

- **Imagine ('cause you can't physically do this):**
- Driving the tongue through the top of your head
- The "tongue flick" sends a gentle flow of air up the Eustachian tube

You should hear a "click" in each ear From the air flow hitting the back of the tympanic membrane

Avoids all the issues with Valsalva Safest method of equalizing ear pressures

"Clearing" While Ascending



Valsalva is opposite of need Need to decrease middle ear pressure Pinch nostrils and gently suck

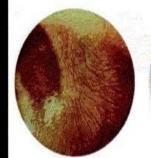
Middle Ear Barotrauma

Symptoms of mild ear barotrauma: pain in the ear difficulty hearing or mild hearing loss dizziness feeling of fullness and pressure in the ear

Symptoms of moderate to severe ear barotrauma: damage to the eardrum tearing allows water to enter middle ear *infections* bleeding from the ear increased pain in the ear constant feeling of pressure and fullness in the ear moderate to severe hearing loss Unequal response pressure different sensation of spinning termed alternobaric vertigo

Tympanic Membrane







Grade 2

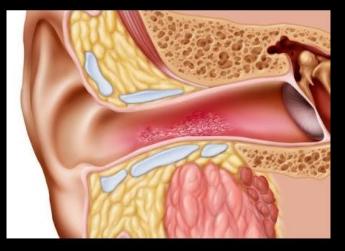
Grade 3





Grade 5

Swimmer's Ear (Otitis Externa)



Most freshwater contains microbes and fungi They survive well in warm, dark places They do not survive well in acidic environments

Prevention:

Rinse ears with vinegar after every diving day Avoid alcohol in ear: dissolves protective ear wax







Near Drowning





Near Drowning

Drowning: Death by submersion Near Drowning: Surviving unconscious submersion

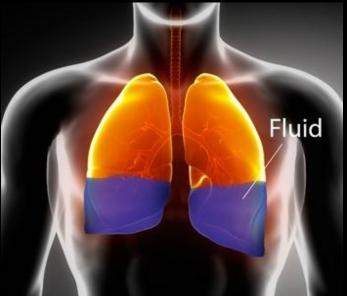
Drowning Sequence: Struggling Water enters airway Airway closes (laryngospasm) → Seizure, Hypoxia (Dry Drowning ~ 15 % of cases) Laryngospasm relaxes Water enters lung Lungs cannot function when filled with water Eventual death

Survival odds improved: Little struggling, very cold water, very young, good condition Aggressive first response

Near Drowning



Symptoms: **Difficulty breathing Chest pain Cyanosis (Bluish lip color) Abdominal distention** Confusion Coughing **Frothy sputum Irritability Unconsciousness**



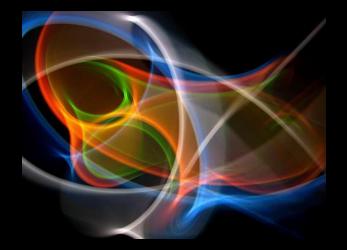




Gases







Gases in Air

Nitrogen 78%
Oxygen 20%
Carbon dioxide and other gases .03%
Inert gases (mainly argon) .97% Water vapor 1%

Oxygen: necessary for life We "burn fuel" $C_6H_{12}O_6 + 6 O_2 \rightarrow 6 CO_2 + 6 H_2O$ Too little oxygen (hypoxic) \rightarrow no life Too much oxygen (hyperoxic) \rightarrow toxic reactions

Nitrogen: considered physiologically inert Involved in nitrogen narcosis & DCS (DCI) Dilutes oxygen in air; limits combustion Others

Most not considered in this class ... assumed part of nitrogen component

Composition of Dry Air					
Gas		Concentration			
Name	Symbol	Volume %	ppmv		
Nitrogen	N ₂	78.084	780,840		
Oxygen	02	20.947	209,470		
Argon	Ar	0.934	9,340		
Carbon dioxide	CO2	0.033	330		
Neon	Ne	0.001820	18.20		
Helium	He	0.000520	5.20		
Methane	CH4	0.000200	2.00		
Krypton	Kr	0.000110	1.10		
Sulfur dioxide	SO2	0.000100	1.00		
Hydrogen	H ₂	0.000050	0.50		
Nitrous oxide	N ₂ O	0.000050	0.50		
Xenon	Xe	0.000009	0.09		
Ozone	O3	0.000007	0.07		
Nitrogen dioxide	NO ₂	0.000002	0.02		

Notes:

-- ppmv = Parts per million parts by volume

-- Water vapor varies up to maximum of 4 volume percent

 The total volume percent of the listed gases does not equal exactly 100 percent due to rounding numbers

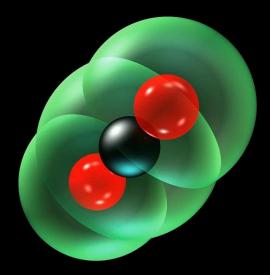
Air as a Breathing Mix

Air: Relatively inexpensive Commonly available Most common underwater breathing mix But, N₂ causes problems at deeper depths: Decompression Sickness Nitrogen Narcosis





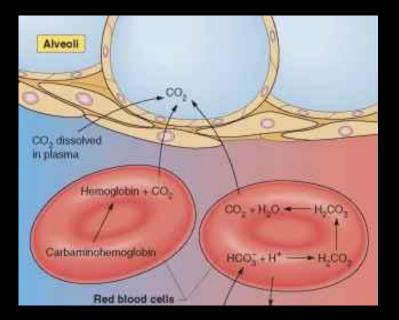




Carbon Dioxide (CO₂)



Carbon Dioxide (CO₂) Carbon Dioxide Metabolic waste product Potent vasodilator Helps maintains blood pH Breathing "Trigger" Excess levels in blood most undesirable CO₂ produced faster than eliminated

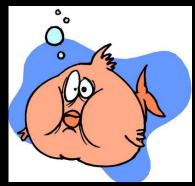


CO₂ Production: Resting: 300 mL/min Working: 2000 mL/min (unfit person has >> production) **Carbon Dioxide (CO₂): Major Problem in Diving Sources of Carbon Dioxide: Contaminated Gas (very rare)** Work load exceeding ventilation "Skip Breathing" **Poor ventilation (equipment dead space)** snorkel Sum poor regulator OCAL full face mask





Cardinal Rule of Diving "Never Hold You Breath"



But, you hold your breath every time you breathe with a regulator **Breathing On Surface:** Inhale ... exhale ... hold **Breathing With Regulator:** Inhale ... hold ... slow exhale ... hold **During the hold, you are:** holding breath (embolism risk) building up CO₂ **Don't consciously extend the "hold phase"** (called "skip breathing")

LPT

Hypercapnia (High CO₂)

A CO₂ "Hit" Slight CO₂ build-up Increased respiration (attempt to vent) Poor ventilation CO₂ continues to increase High CO₂ perceived as "regulator not working" Suspicion: many "out of air" emergencies are CO₂ hits



CO₂ Cascade

Carbon dioxide exacerbates most dive maladies

Increased Depth	Gas	Density gram/liter of gas	Mix	Density gram/liter of gas		
	Nitrogen	1.1009	Air at 1 ATA	1.138		
Increased Gas Density	rtial	0.1573	Air at 99 FSW	4.552		
Gas Density Pressure	Oxygen	1.2572	32% Nitrox at 99 FSW	4.605		
Decreased	Neon	0.7930				
Lung Ventilation Decrease Awareness		1.5696				
Increased CO ₂ Loss of Consciousn	Greater	Increased O₂ in EAN_x raises density Greater density increases work of breathing				
Increased Exertion		Israeli military studies: Breathing EAN _x increases CO ₂ retention				

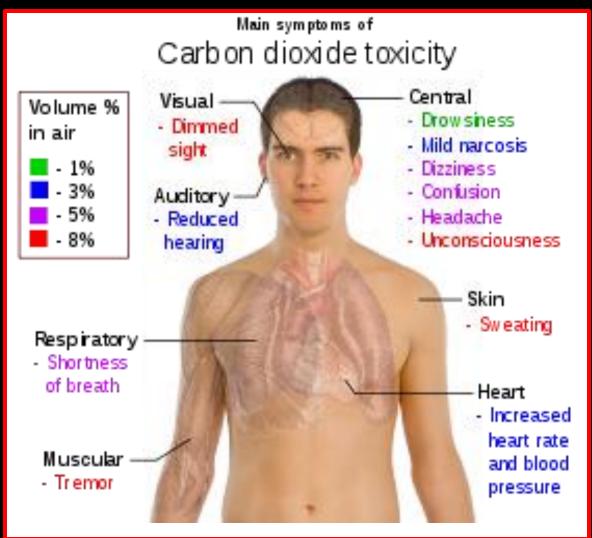
Hypercapnia (High CO₂)

Studies show oxygen-enriched air promotes CO₂ retention Higher the O₂ concentration, the greater the effect Greater density at depth requires more work to breathe

> Important to monitor breathing Suspect CO₂ build-up → Stop Breathe slowly (Imagine STOP sign) Until breathing returns to normal



Hypercapnia (High CO₂)



Main Diving Issues: Respiratory Starvation Headache Center of forehead







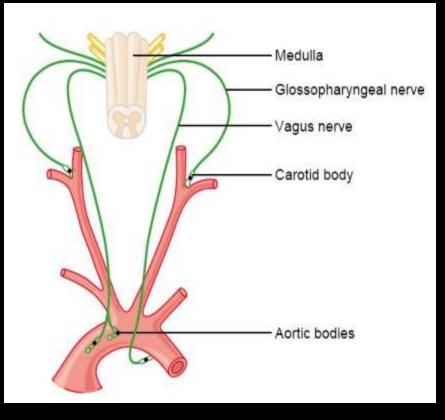




Problem During Ascent: Skin Diving Free Diving Only Using Mask & Snorkel



Associated with pre-dive hyperventilation Ventilation exceeds metabolic demands



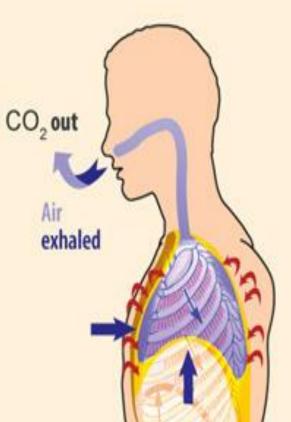


Carotid Body: Chemoreceptor Senses CO₂ & O₂

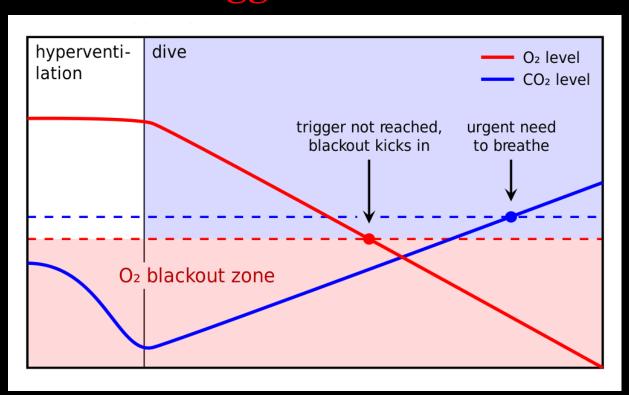
High CO₂ communicates to respiratory center "Need to breathe NOW to ventilate xs CO₂"

To avoid "Breathe now" from high CO₂ To extend bottom time

Divers will hyperventilate Rapid deep exhalations Drops blood CO2 levels



Shallow Water Blackout **During Free Dive:** Metabolism consumes blood oxygen Reach "blackout" (not enough O₂) Before CO2 triggers "Need to breathe"



A DANGER

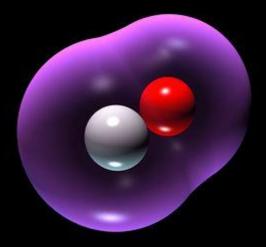


NO prolonged breath holding or underwater swimming.

Competitive and repetitive breath holding can be deadly.

Doing so tells your body not to ask for oxygen, which can cause you to pass out and drown.

AUSTRALIAN SAFETY SIGNS by StikyStuf 1800 33 03 63 www.australiansafetysigns.net.au



Carbon Monoxide (CO)



CO Binds To Heme In Hemoglobin ~250 X Stronger than O2 **Prevents Heme from carrying O_2** BOUND CARBON MONOXIDE BOUND OXYGEN

Too Much CO \rightarrow No O₂ \rightarrow Lethal

Symptoms of Carbon monoxide poisoning

- Dizziness
- Headache
- Disorientation
- Impairment of the cerebral function
- Coma
- Visual disturbances
- Disease of the heart and respiratory

- · Muscle weakness.
- Muscle cramps
- Seizures
- · Nausea
- Aggravation of preexisting diseases

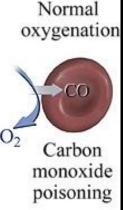


Carbon Monoxide (CO)

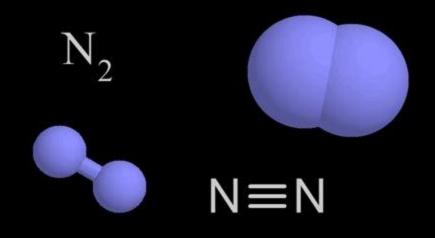
From incomplete combustion: Compressor oil Engine exhaust Cigarette smoke Kerosene heaters







Humans metabolism releases CO Minor amount: factor in closed environments (habitats, subs & space capsules) 1 cigarette: more CO than USN allows in their breathing gas



Nitrogen





Nitrogen Narcosis



Nitrogen Narcosis

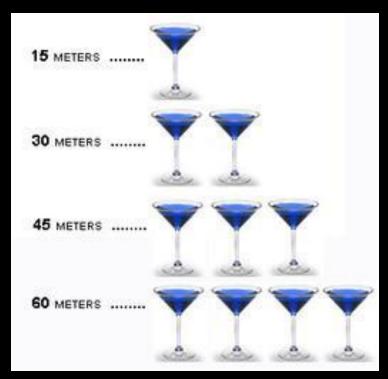
Narcosis:

Pronounced "anesthetic effect"

Observed when breathing N₂ containing mixes at depth Deeper the depth, more intense the effect

So-called Martini's Law: (Not considered valid) Every ~50 fsw of depth = 1 dry martini on an empty stomach





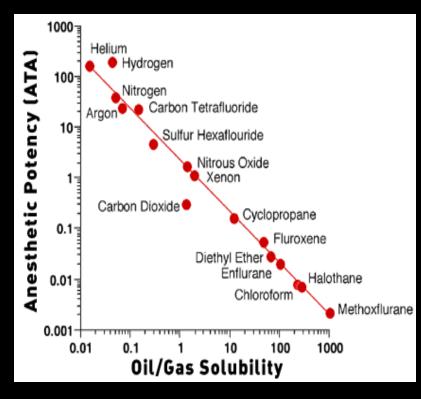


Many Gases Have a Narcotic Potency

Gas	Relative narcotic
	potency
Ne	0.3
H_2	0.6
N_2	1
O ₂	1.7
Ar	2.3
Kr	2.5
$\rm CO_2$	20
Xe	25.6

Meyer-Overton Theory of Anesthesia Gases dissolve in nerve tissue myelin (lipid layer) Altered electrical conduction of nerves

Oxygen metabolized, does not build up Diminishes at-depth narcotic potency



Lipids NOT total picture GABA receptors involved

Complex Problem Not all understood

Nitrogen Narcosis

Signs & Symptoms Warm, clear water: euphoria ("Laughing Gas" as model) **Tendency to giggle Tunnel vision (syncope) Idea fixation (repetitive behaviors) Shortened attention span Declining neuro-muscular coordination** Numb lips **Inability to concentrate** Cold, limited visibility water: dread Sense of being stalked ("It" is out there ... somewhere) Loss of confidence (sense of helplessness) **Intense anxiety**

Nitrogen Narcosis

Symptoms exacerbated by: cold work load (CO₂) anxiety fatigue drugs alcohol menses (?)



Symptoms:

Typically noticeable ~ 100 fsw, but onset as shallow as ~ 60 fsw Sense of well-being: masks CNS impairment May be not be noticed by affected diver Individually variable CNS impairment increases with time / depth Ascent relieves problems; typically, no residuals

Underwater "Narcosis Test"

"OK" signal is "automatic reflex" Often NOT reliable indicator



Narcosis Test (for cognitive processing) Show 1 to 4 fingers to diver Diver adds 1 to # fingers Shows added count



Nitrogen Narcosis: Classic Myth Narcosis is reason for 130 fsw sport diving depth limit

Turns out, 130 fsw is US Navy limit to use vintage scuba on a salvage dive 'cause At the time diving rules were established, Double hose regulators would not support hard working below 130 fsw













"Bubble Trouble": Decompression Sickness

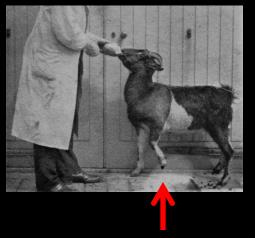


Origin of "The Bends"



Building of Brooklyn Bridge (1870's) Caisson workers experienced pain on surfacing Assumed postures similar to women dancing "Grecian Bend" Wanted to return to work to lessen the pain Being "Bent" was an insult

Established: Caisson's disease and Sponge Diver's disease Same malady

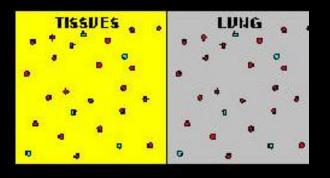




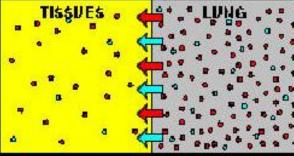


Haldane used goats to develop dive tables (1930's) Goats forelimbs would bend on too rapid ascents So, they were "bent" Developed ascent tables that would not bend goats

Gases at Depth

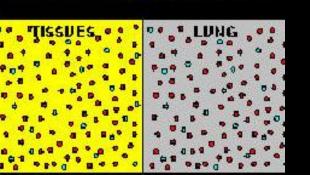


On the surface: Gases diffuse across cell membranes Concentration reaches equilibrium Each gas acts independently



On descent: Gases diffuse across cell membranes Movement based on gas pressures

Each gas acts independently



At depth: Gases diffuse across cell membranes Concentration reaches equilibrium For all components in breathing gas

Gases at Depth

Gases eventually equilibrate tissue gas pressure with environment Increased depth increases amount of dissolved gas Nitrogen: accumulates ... not used by metabolism

Different tissues (solubility compartments) build up gas at different rates Compartment nitrogen level is mathematically approximated Basis of decompression tables

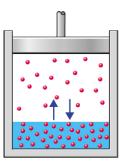
No correlation between a biological tissue and a mathematical compartment

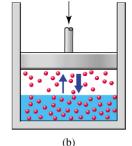
Different models will use

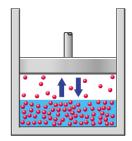
Different number of tissue compartments

Different mathematical expressions to approximate gas concentration

Gas Solubility – Effect of Pressure







(c)

Nitrogen Partial Pressures: Surface: $0.79 \ge 1$ ata = 0.79 ata 99 fsw: $0.79 \ge 4$ ata = 3.16 ata

(a) Initial equilibrium

Increased pressure

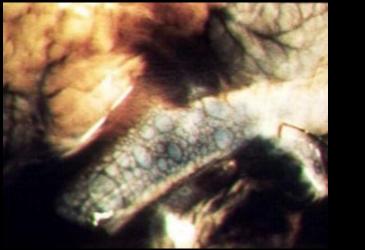
New equilibrium

LPT

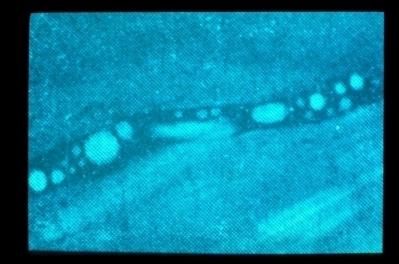
Decompression Sickness

On ascent, gas pressure in tissues greater than ambient Gas bubbles out of tissues

Bubbles may form on dive (diver / profile dependent) Some dives /divers may not show significant bubbles) Too many bubbles: Decompression Sickness (DCS) Symptoms observed depend on where bubbles form







Bubbles in Tissue

Bubbles in Veins

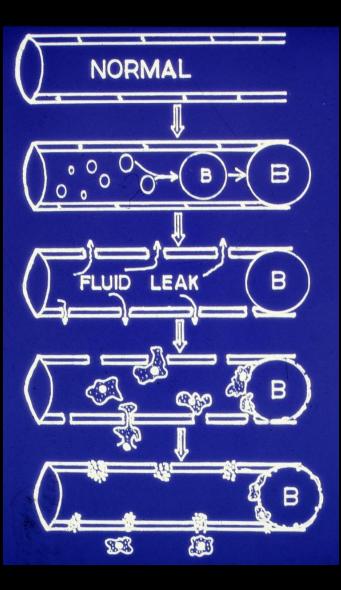
Bubble Trouble

Most bubbles safely eliminated via venous circulation and lungs Too many bubbles overwhelm physiology Proteins of coagulation cascade react to gas bubbles in tissues coat bubble and initiate clotting at site of bubble



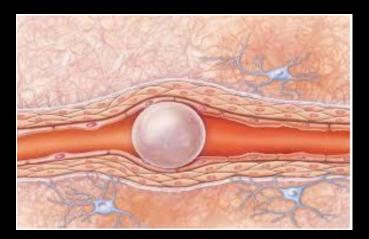
Electron micrograph of protein coated bubble Arrow points to platelet adhering to coagulation protein coat

Bubble Trouble



Bubbles in capillaries block flow Pressure builds up Vessel walls split Fluid leakage Activation of inflammatory response

Very complex biochemical complications Much still not understood



Symptoms Depend on # Bubbles and their Location Frequency and Onset of Symptoms

Symptoms	Frequency
local joint pain	89%
arm symptoms	70%
leg symptoms	30%
dizziness	5.3%
paralysis	2.3%
shortness of breath	1.6%
extreme fatigue	1.3%
collapse/unconsciousness	0.5%



Time to onset	Percentage of cases
within 1 hour	42%
within 3 hours	60%
within 8 hours	83%
within 24 hours	98%
within 48 hours	100%



Bubble Trouble

Bubble trouble assumed to be primarily a "too much N₂" malady N₂ builds up

abundance of N₂ in breathing mix

increased time / depth drives N_2 into tissues

N₂ not used in metabolism

Over abundant N₂ escapes tissues on ascent

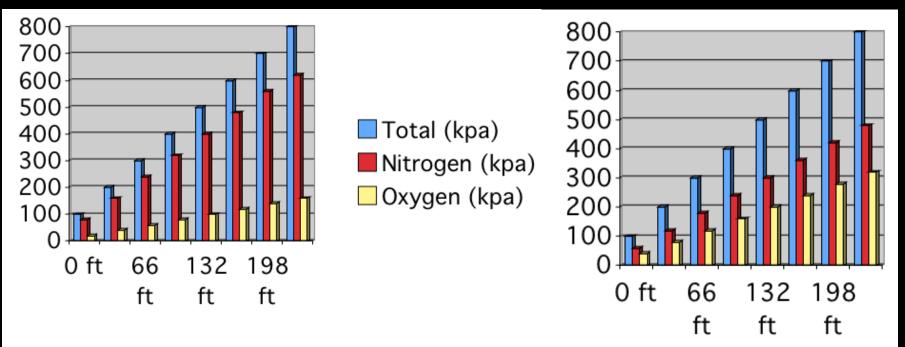
Basis of oxygen-enriched air

Obvious remedy: decrease amount of N₂ in breathing mix Use gas involved in metabolism (oxygen)





Oxygen Enriched Air Reduces Nitrogen Tissue Loads Breathing Air Breathing EAN₃₂



Primary advantage of oxygen enriched air Decompression obligation depends on N₂ tissue load Decompression obligation reduced by replacing N₂ with O₂

Too Many Bubbles: Decompression Sickness

Chest and body: skin rash

Fingers and feet: tingling, pins and needles vertigo, poor balance, confusion, nausea, fatigue, unconsciousness

Spine

abdominal pain, loss of bladder function, paralysis

Knees, elbow, shoulders: joint pain

DAN: > 60% of DCS involve depths > 80 fsw

Type I (Pain Only) Musculoskeletal Insult limb or joint pain Itching **Skin rash** Localized swelling **Type II (CNS Involved) Spinal Involvement** numbness / tingling **bi-lateral paralysis** no bladder function loss of sexual response **Inner ear (staggers)** Lungs (chokes) **Cardiac arrest** Type I on ascent



Skin Bends Signs & Symptoms: Skin discoloration Purplish and flat Compared to a rash: More reddish and "textured" Itching



Most often:

Chamber dives Females Hot shower post-dive





~ 20% show neurological involvement

DCS Risk Factors

The following conditions are considered to increase DCS risk: dive (deeper depth/longer time) profile

older age obesity (poor physical condition) dehydration poor circulation (tight clothing) **illness** scar tissue alcohol (12 hours pre or post dive) fatigue strenuous exercise during dive cold repetitive dives multiple ascents / descents on same dive multi day diving history of DCS being female (?) misuse of dive tables / dive computers





Serious DCS Cases Involve Spinal Cord



Bi-lateral dysfunction/numbness May increase with time May result in permanent dysfunction Affects Ability to: walk excrete have sex



Every dive is gambling with spinal cord function: Your body chemistry on the day you dive Best tactic: Love your spinal cord: dive conservatively





Lowering Bubble Formation

Minimize risk by:

Not "pushing" tables Slow ascents especially "shallow" Safety Stops Staying hydrated

Agonizingly slow: Monitor with gauges



Ascent Rate: A Compromise



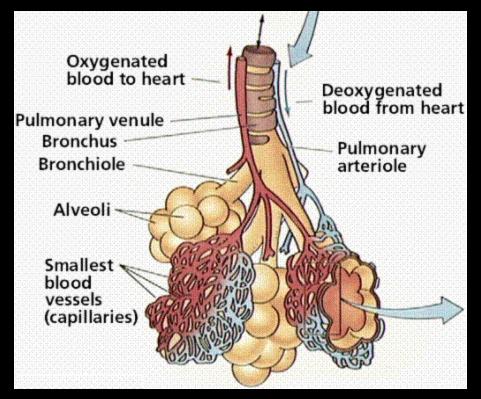
US Navy Combat Swimmers: ~120 fsw / min US Navy Salvage Divers: ~25 fsw/min US Navy Compromise: 60 fsw / min No correlation to physiology

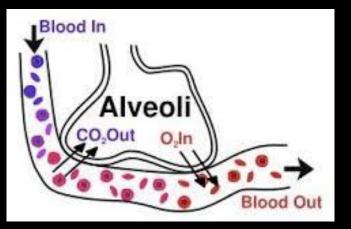


Arterial Gas Embolism (AGE)



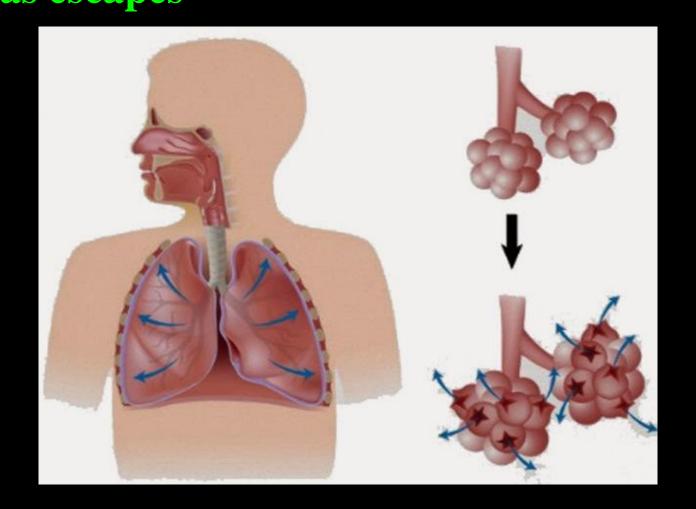
Alveoli: Site of Gas Exchange





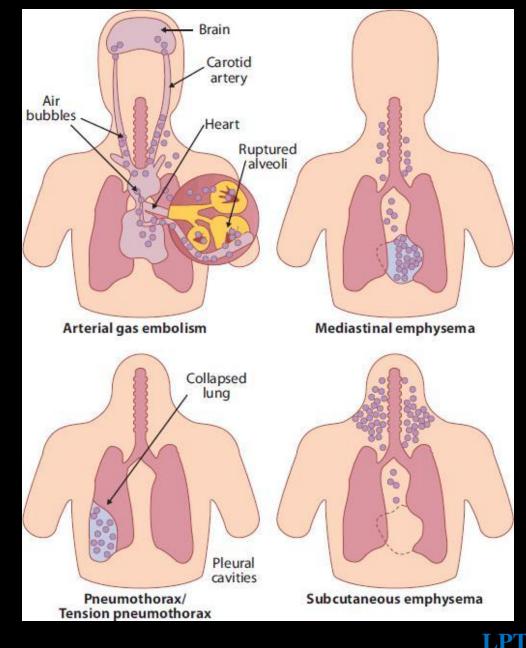


Alveoli: One cell thick (allows gas exchange) Rupture: D P ~ 1.5 psi Pulmonary Barotrauma of Ascent Expanding gas in alveoli exceeds capacity Alveoli tissue tears Gas escapes

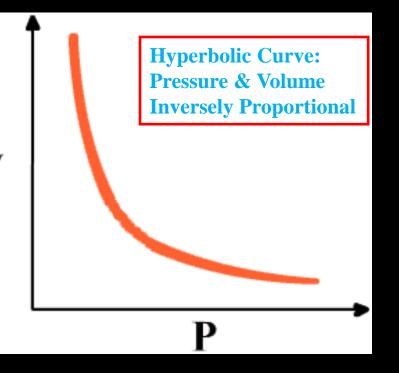


Pulmonary Barotrauma of Ascent

Ruptured Alveoli: If Gas Goes: Under Skin: Subcutaneous Emphysema Chest Cavity: Mediastinal Emphysema Against Lung: Pneumothorax Into Arterial Circulation: Air Embolism



Remember Boyles's Law



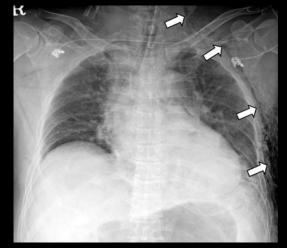


Greatest volume change: where pressure is less Means greatest risk to tissue: shallow water

Do NOT need "panic ascent" to develop overpressure ~ 20-25 % of injuries "undeserved" Its Your body chemistry on the day you dive: internal air trapping, asthma, cysts, fibrosis, inflammation tumors, blebs, wave height, diseases, inadequate exhalation

Pulmonary Barotrauma of Ascent The Emphysemas: Signs & Symptoms

Under Skin: Subcutaneous Chest Cavity: Mediastinal May be asymptomatic **Crepitus** (Noisy gas) **Voice change** (if gas moves into throat) **Cardiovascular Issues: Tachycardia** Cyanosis **Hypotension** X-rays may show trapped air

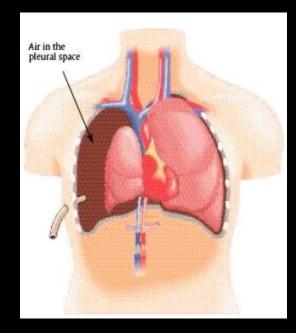


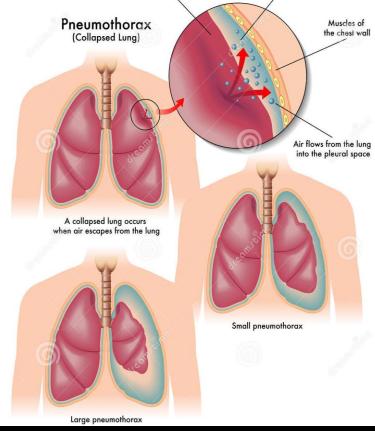
Subcutaneous



Pulmonary Barotrauma of Ascent Pneumothorax: Mechanism of Injury

Visceral pleura (tissue surrounding the lungs) ruptures Gas enters into pleural space Expanding gas compresses lung Lung function diminishes





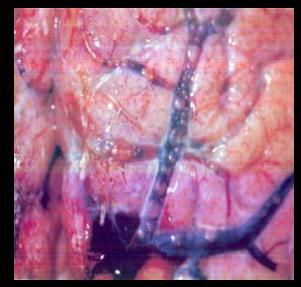
Pulmonary Barotrauma of Ascent Pneumothorax: Signs & Symptoms Sudden, intense, unilateral chest pain **Difficulty breathing Rapid breathing Chest rises less on affected side Diminished Breath sounds** X-ray may show air Collapsed

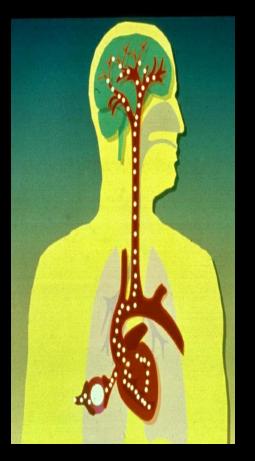
Medical staff will check for this malady prior to recompression

Lung

Pulmonary Barotrauma of Ascent Arterial Gas Embolism (AGE)

Gas enters pulmonary vein Gas moves into heart Gas enters arterial circulation Gas follows carotid artery to brain Worst case scenario



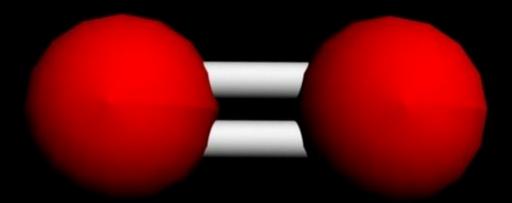


Autopsy bubbles in brain

Pulmonary Barotrauma of Ascent: Signs & Symptoms

Bubbles in Brain ("stroke"): Damage depends on where bubbles lodge Major clue: CNS involvement; may see

Unconsciousness **Cognitive Impairment** Aphasia (can't talk) **Auditory dysfunction** Visual Disturbances (tunnel vision; unequal pupil sizes) **Partial Paralysis (usually unilateral) Parathesis (unilateral numbness)** Vertigo **Rapid Onset: Before surfacing** Convulsions < 10 minutes after dive **Chest pain Skin marbling Personality change** Libermeister's Sign (white triangle on tongue) Gas bubbles visible in retina of eye **Abnormal EKG Red froth at the mouth**



OXYGEN

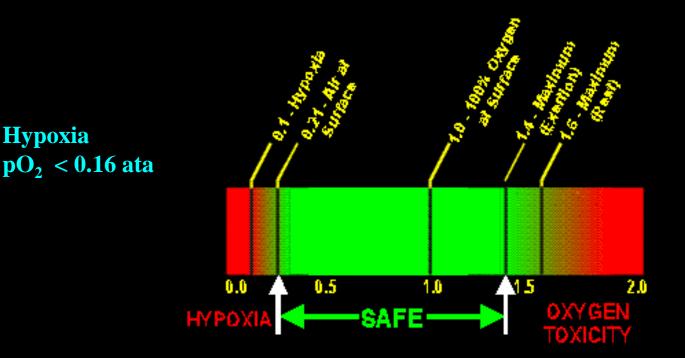




Oxygen Toxicity



Oxygen Necessary For Life Metabolism: narrow oxygen partial pressure window **Too little oxygen (hypoxic) → no life** $C_6H_{12}O_6 + 6 O_2 \rightarrow 6 CO_2 + 6 H_2O$ **Too much oxygen (hyperoxic) → toxic reaction** Cellular components $+ O_2 \rightarrow$ "Bad stuff" (ROS)

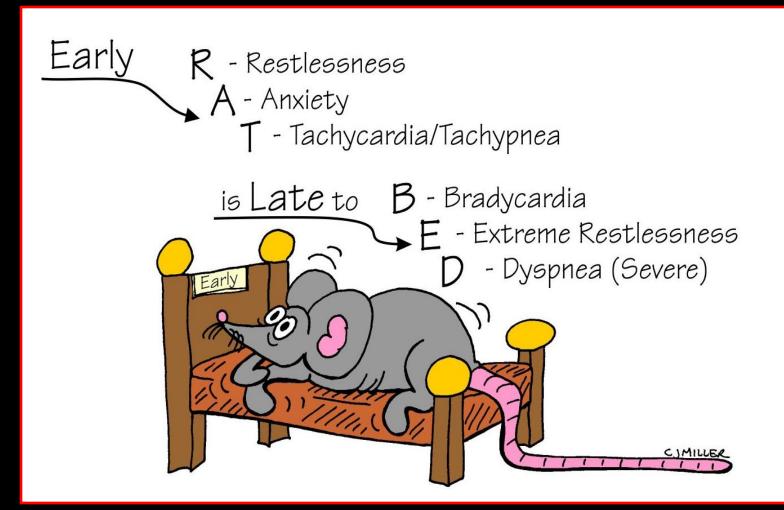


Hypoxia

Hyperoxia $pO_2 > 1.6$ ata

Hypoxia (too little oxygen) **Simplistic View of Body Chemistry:** Fuel (sugar, fat, protein) + $O_2 \rightarrow Energy$ Brain can only use sugar supplied by blood flow Any trauma (shock) reduces blood flow / sugar / O₂ Brain among first tissue to be compromised Brain uses ~ 20% of total metabolic O₂ consumed Low O₂ reduces / stops above energy reaction: **Normal cell function compromised Blood vessels swell to increase blood supply Skull impairs brain tissue expansion**

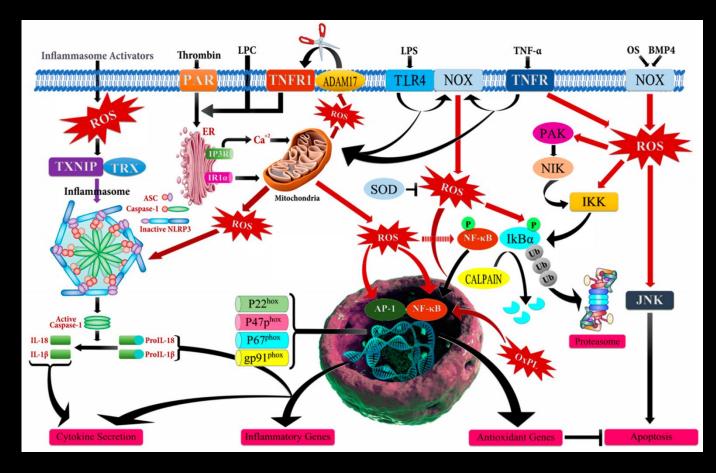
Hypoxia Symptoms



Ultimately: No oxygen no life

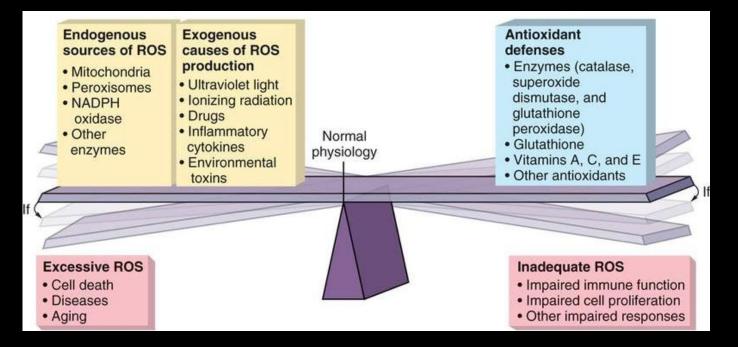
Hyperxia (Too Much Oxygen)

Oxygen is a highly reactive element Cellular components + O₂ → "Bad stuff" (ROS) ROS: Reactive Oxygen Species (Cellular Saboteur)



Hyperoxia

Reactive Oxygen Species (ROS) Constantly Produced Direct result of oxygen molecule's chemical reactivity



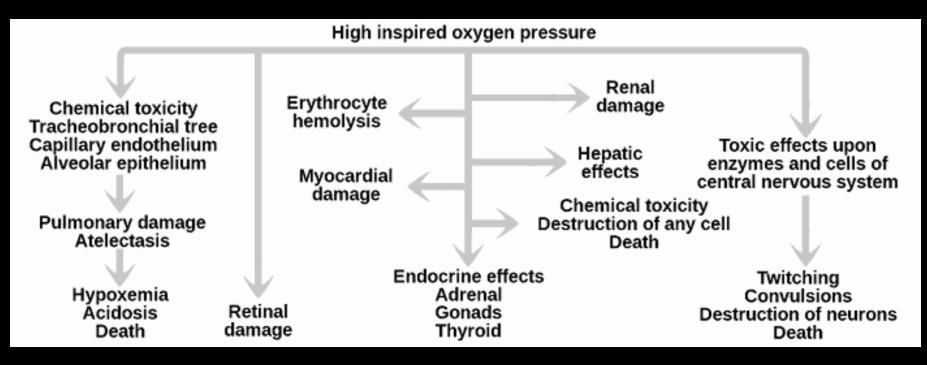
ROS are biologically very destructive Numerous biological defenses against ROS SOD Superoxide Dismutase GTP Glutathione Peroxidase Lots of anti-oxidant molecules

Hyperoxia Effects

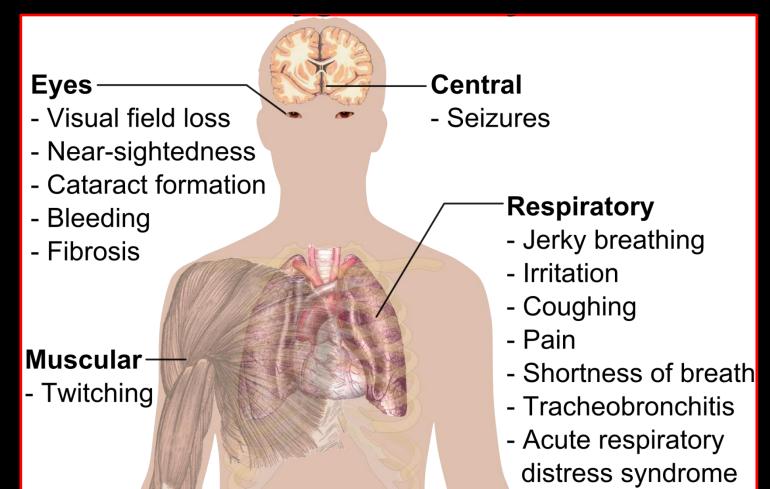
Higher pO₂ increases ROS concentrations

Le Châtelier's Principle

Increase partial pressure: drive reaction to the right Cellular components + O₂ → "Bad stuff" (ROS)



Hyperoxia Effects



Symptoms depend on pO₂ and exposure time

VENTID – C

- V Vision
- **E Ears**
- N Nausea
- T Twitching
- I Irritability
- **D Dizziness**
- **C Convulsions**

ConVENTID

Hyperoxia Effects on CNS

- Not a progression ... maybe no warning May start with convulsions
- Twitching usually starts at lower lip

Common causes: Exceeding the oxygen exposure limits Using an incorrect mix for the depth Using wrong deco gas at depth

Recognition of ANY Symptom immediately ascend



(reduce pO₂)



Hyperoxia Effects on CNS

Oxygen toxicity effects may be enhanced by: Heavy exercise Breathing dense gas Breathing against resistance Increased CO₂ buildup Chilling or hypothermia Water immersion (as opposed to "chamber diving")

Individual tolerance to oxygen toxicity varies over time Tolerance varies from individual to individual

Oxygen tolerance tests no longer considered valid



Hyperoxia Effects on CNS Seizure in sport diving equipment is usually fatal Spit out regulator (reflex inhale) and breathe water Panic and "escape to surface" (embolize) Diving EAN_x requires monitoring oxygen exposure Surviving convulsions: reason to use full face mask







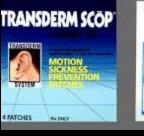


Hyperoxia Effects on CNS Anecdotal suggestion that Sudafed increases seizure risk (seizures are a side effect in children) **Other concerns:** anti-motion drugs (especially transderm (scopolamine)) aspirin, caffeine, viagra, nitro heart medication **Never rigorously studied Best to avoid diving with any drugs**















Biological Defenses Occasionally Sold to Divers No evidence that ingestion of unprotected SOD has any physiological effects Ingested SOD is broken down into amino acids before being absorbed SOD bound to wheat proteins MIGHT improve its ROS protection

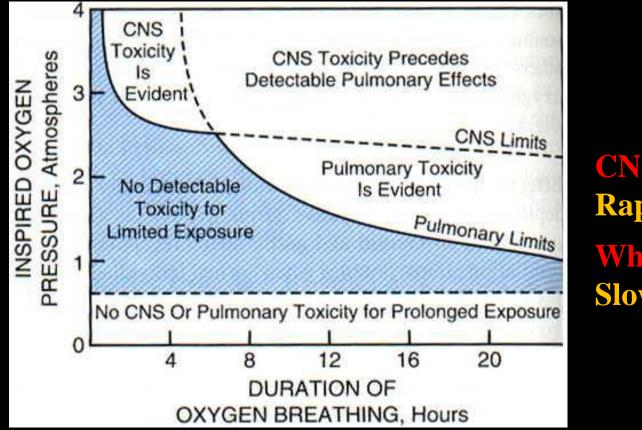
Nitrox Therapy is a power workout Nitric Oxide promoter An absolute contraindication for diving (Nitric oxide implicated in oxygen toxicity convulsions)

Recent Findings suggest eating dark chocolate bar ~ 30 minutes pre-dive offers some protection from oxidative cell damage





Whole Body Oxygen Toxicity Formerly Pulmonary Toxicity (Lorrain Smith Effect) Contrasted to CNS Toxicity (Paul Bert Effect)



CNS: Rapid Onset Whole Body Slow Onset

Whole Body Oxygen Toxicity No-deco stop diving concerned primarily with CNS toxicity

Whole Body a concern for: Extended range Deco diving Intensive, multiple dive operations Mixes with high O₂ concentration



Onset: breathing high pO_2 (> 0.5 ata) for hours **Relief: breathing** $pO_2 < 0.5$ ata

Primarily effects the lungs Typically, not a concern in standard range diving

Whole Body Oxygen Toxicity Symptoms

Pulmonary Chest pain or discomfort Coughing Chest tightness Fluid in the lungs Reduction in vital capacity

Non-pulmonary Skin numbness and itching Headache Dizziness Nausea Visual disturbances Diminished aerobic capacity Body optimized for 21% O₂ High pO₂ alters tissue structure Lung tissue Thickens Becomes less pliable Reduces vital capacity



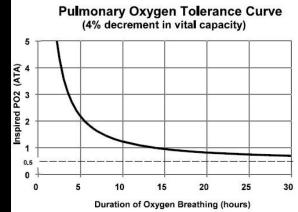
Oxygen Toxicity Units (OTU)

Based on decreased lung vital capacity while breathing 100 % O_2 1 OTU = Breathing 100% O_2 for 1 minute Pulmonary Oxygen Tolerance Curve (4% decrement in vital capacity)

At constant depth:

OTU = t [$(pO_2 - 0.5) / 0.5$]^{-0.83}

Ascending and descending:



OTU = 0.27 t [{(pO_{2f} - 0.5) / 0.5)}^{1.83} - {(pO_{2i} - 0.5) / 0.5 }^{1.83}] pO_{2f} - pO_{2i}

time (t) in minutes
pO₂ at constant depth in absolute atmospheres
pO_{2f} at final condition in absolute atmospheres
pO_{2I} at initial condition in absolute atmospheres
Solving involves integration of pressure over time → best done by computer

Oxygen Toxicity Units (OTU)

EAN_x diving below OTU threshold, so typically not tracked

OTU Daily (24 hours) Limits Allowed Daily Exposure: 1440 Typical DCS Treatments: Table 5: 297 Table 6: 607 Table 6A: 820 EAN_X Diving: ~40 - 300 Extended Range Diving: ~850 Typical Technical: ~300 - 400





Divers Track OTU's By Computer Planning Software In-water Dive Computers OTU Tables OTU Spreadsheets



NOAA Oxygen Exposure Limits

Example: EAN_{32} mix at 130 fsw

Determine pO₂ at depth % O₂ Depth to Pressure $pO_2 = 1.58$ ata

NOAA Oxygen Exposure Limits										
PO ₂ (atm)	Maximum Single Exposure (minutes)	Maximum per 24 hr (minutes)								
1.60	45	150								
1.55	83	165								

Single Dive limit of 45 minutes

Example: EAN_{40} mix at 130 fsw

Determine pO₂ at depth % O₂ Depth to Pressure $pO_2 = 0.32[(130 \text{ fsw}/33 \text{ fsw/atm}) + 1 \text{ atm}] pO_2 = 0.40[(130 \text{ fsw}/33 \text{ fsw/atm}) + 1 \text{ atm}]$ $pO_2 = 1.98$ ata

pO₂ exceeds oxygen exposure limits

pO₂ too high for 130 fsw Unacceptable oxygen toxicity risk

NOAA Oxygen Exposure Limits

Example: Using EAN₃₂ Reduce allowed pO₂ to 1.40 ata



1.45	135	180
→ 1.40	150	180
1.35	165	195

Decreased allowed pO_2 Lowers maximum depth (MOD) $MOD = [(1.4 \text{ ata}) - 1 \text{ atm}] \frac{33 \text{ fsw}}{33 \text{ fsw}}$ 0.32 atm

Single Dive limit of 150 minutes MOD = 111 fsw

Need to determine time and max depth for all EAN_x dives

Percent CNS Oxygen Exposure % Daily O₂ Allowance = [Dive Time / 24 hour Allowed] x 100

NOAA Summary for Common Dives

Max, PO2	Dive		ann an a'				:ninitun;						
Exposure	Time												
(atm)	(minutes)	:5	:10	:15	:20	:25	:30	:35	:40	:45	:50	:55	:60
	210											P10001000	entrantica -
1.2	Max.	2%	5%	7%	10%	12%	14%	17%	19%	21%	24%	26%	29%
	195					(jener)							
1.25	Max.	3%	5%	8%	10%	13%	15%	18%	21%	23%	26%	28%	31%
	180												
1.3	Max.	3%	6%	7%	11%	14%	17%	19%	22%	25%	28%	31%	33%
	165												
1.35	Max.	3%	6%	9%	12%	15%	18%	21%	24%	27%	30%	33%	36%
	150						ang	all tone of the				See St	alla anna anna anna anna anna anna anna
1.4	Max.	3%	7%	10%	13%	17%	20%	23%	27%	30%	33%	37%	40%
	135										Listoniani,		
1.45	Max.	4%	7%	11%	15%	19%	22%	26%	30%	33%	37%	41%	44%
	120				E								
1.5	Max.	4%	8%	13%	17%	21%	25%	29%	33%	38%	42%	46%	50%
	82												
1.55	Max.	6%	12%	18%	24%	30%	36%	42%	48%	55%	61%	67%	73%
	45												
1.6	Max.	11%	22%	33%	44%	56%	67%	78%	89%	100%			

For repetitive Dives: Treat Residual O₂ Like Residual N₂ Use Surface Credit Table (Next Slide)

CNS% Oxygen Exposure Surface Interval Credit Table

				Surface	Interval			
Start	30 MINS	60 MINS	90 MINS	120 MINS	180 MINS	240 MINS	300 MINS	360 MINS
100%	83%	66%	49%	41%	24%	16%	11%	7%
95%	79%	63%	46%	38%	22%	15%	10%	7%
90%	75%	59%	44%	37%	22%	15%	10%	7%
85%	71%	56%	42%	35%	21%	14%	9%	6%
80%	66%	53%	39%	32%	19%	13%	9%	6%
75%	62%	49%	37%	31%	18%	12%	8%	5%
70%	58%	46%	34%	28%	17%	11%	7%	5%
65%	54%	43%	32%	27%	16%	11%	7%	5%
60%	50%	40%	29%	24%	14%	9%	6%	4%
55%	46%	36%	27%	22%	13%	9%	6%	4%
50%	41%	33%	24%	20%	12%	8%	5%	3%
45%	37%	30%	22%	18%	11%	7%	5%	3%
40%	33%	26%	20%	17%	10%	7%	5%	3%
35%	29%	23%	17%	14%	8%	5%	3%	2%
30%	25%	20%	15%	12%	7%	5%	3%	2%
25%	21%	16%	12%	10%	6%	4%	3%	2%
20%	17%	13%	10%	8%	5%	3%	2%	1%
15%	12%	10%	7%	6%	3%	2%	1%	1%
10%	8%	7%	5%	4%	2%	1%	1%	1%
Start	30 MINS	60 MINS	90 MINS	120 MINS	180 MINS	240 MINS	300 MINS	360 MINS

Cumulative % CNS Oxygen Exposure

Example: First Dive: 40 minutes at pO_2 of 1.60 ata % CNS Oxygen Exposure: (40 min / 45 min x 100) = 89% Surface Interval: 120 minutes New (Residual) % CNS Oxygen Exposure: 37 % Second Dive: 30 minutes at pO_2 of 1.2 ata Dive % CNS Oxygen Exposure: (30 min / 210 min x 100 = 14 % Total CNS Exposure = (14 + 37) % = 51 %



Per Minute % CNS Oxygen Exposure

PO 2	CNS%/min	PO2	CNS%/min	PO2	CNS%/min
0.6	0.14	1.02	0.35	1.42	0.68
0.62	0.14	1.04	0.36	1.44	0.71
0.64	0.15	1.06	0.38	1.46	0.74
0.66	0.16	1.08	0.4	1.48	0.78
0.68	0.17	1.1	0.42	1.5	0.83
0.7	0.18	1.12	0.43	1.52	0.93
0.72	0.18	1.14	0.43	1.54	1.04
0.74	0.9	1.16	0.44	1.56	1.19
0.76	0.2	1.18	0.46	1.58	1.47
0.78	0.21	1.2	0.47	1.6	2.22
0.8	0.22	1.22	0.48	1.62	5
0.82	0.23	1.24	0.51	1.65	6.25
0.84	0.24	1.26	0.52	1.67	7.69
0.86	0.25	1.28	0.54	1.7	10
0.88	0.26	1.3	0.56	1.72	12.5
0.9	0.28	1.32	0.57	1.74	20
0.92	0.29	1.34	0.6	1.77	25
0.94	0.3	1.36	0.62	1.78	31.25
0.96	0.31	1.38	0.63	1.8	50
0.98	0.32	1.4	0.65	1.82	100
1	0.33				

Maximum Operating Depth (MOD)

MOD – the maximum depth that should be dived with a given EAN_x mixture

$$MOD = \left(\frac{(PO_2 \ limit, \ ata)}{(FO_2 \ mix)} - 1 \ atm\right) 33 \ fsw \ / \ atm$$

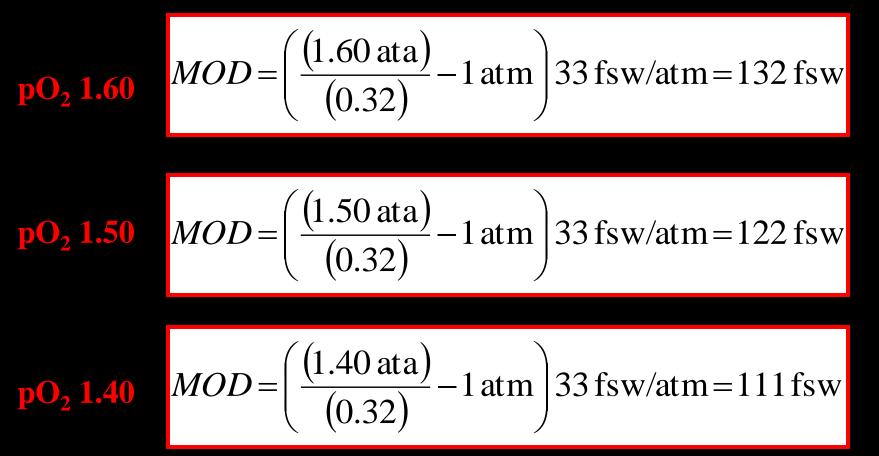
Example: Determine MOD for a 36% mix with a pO₂ 1.60 ata:



$$MOD = \left(\frac{(1.60 \text{ ata})}{(0.36)} - 1 \text{ atm}\right) 33 \text{ fsw} / \text{ atm} = 114 \text{ fsw}$$



Maximum Operating Depth (MOD) For NOAA I (32% O₂)





Maximum Operating Depth (MOD) For NOAA II (36% O₂)

pO₂ 1.50

pO₂ 1.40



pO₂ 1.60
$$MOD = \left(\frac{(1.60 \text{ ata})}{(0.36)} - 1 \text{ atm}\right) 33 \text{ fsw/atm} = 114 \text{ fsw}$$

$$MOD = \left(\frac{(1.50 \text{ ata})}{(0.36)} - 1 \text{ atm}\right) 33 \text{ fsw/atm} = 105 \text{ fsw}$$

$$MOD = \left(\frac{(1.40 \text{ ata})}{(0.36)} - 1 \text{ atm}\right) 33 \text{ fsw/atm} = 95 \text{ fsw}$$

EAN_x has shallower onset of CNS toxicity than air (Has more O₂)

For 1.4 ata limitAir187 fswNOAA I111 fswNOAA II95 fsw

For 1.6 ata limitAir218 fswNOAA I132 fswNOAA II114 fsw

Higher the pO₂ Shallower the MOD





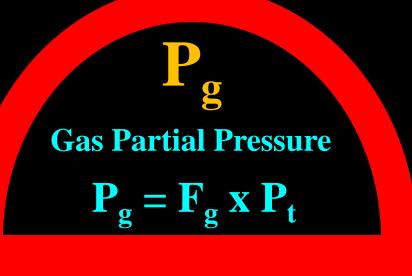
Using Dalton's Law (Determine Partial Pressures) Dalton's law (based on fraction of component gas) $P_g = F_g \times P_t$ P_{g} = partial pressure of the component gas $\mathbf{F}_{g} = \mathbf{fraction} \ \mathbf{of} \ \mathbf{the} \ \mathbf{component} \ \mathbf{gas}$ $\mathbf{P}_{t} = \mathbf{total \ pressure \ of \ gas \ mixture}$ (determined from depth) For air (21 % O₂) being breathed at 90 fsw: $P_g = F_g \times P$ where P = [(D fsw /33 fsw/atm) + 1 atm] $pO_2 = 0.21$ [(90 fsw / 33 fsw/atm) + 1 atm)

pO₂ = **0.78** ata



Classic Recreational Diving Dalton's "Pie" Hide wanted segment: Result Solves for hidden segment

Fraction Of a Gas $\mathbf{F}_{g} = \frac{\mathbf{P}_{g}}{\mathbf{P}_{t}}$



F_g P_t

Also called: "T" Diamond

Total Pressure

 $\mathbf{P}_{t} = \frac{\mathbf{P}_{g}}{\mathbf{F}_{g}}$

NOAA pO₂ for Depth vs. Fraction of Oxygen in the Breathing Mix

De	pth	atm	Ś.	9		2	2		2	3)			S		· · · · · ·
(fsw)	(msw)	abs	21%	28%	30%	31%	32%	33%	34%	35%	36%	37%	38%	39%	40%
0	0	1.00	0.21	0.28	0.30	0.31	0.32	0.33	0.34	0.35	0.36	0.37	0.38	0.39	0.40
35	11	2.05	0.43	0.57	0.62	0.66	0.66	0.68	0.70	0.72	0.74	0.76	0.78	0.80	0.82
40	12	2.21	0.46	0.62	0.66	0.69	0.71	0.73	0.75	0.77	0.80	0.82	0.84	0.86	0.88
50	15	2.52	0.53	0.71	0.76	0.78	0.81	0.83	0.86	0.88	0.91	0.93	0.96	0.98	1.01
60	18	2.82	0.59	0.79	0.85	0.87	0.90	0.93	0.96	0.99	1.02	1.04	1.07	1.10	1.13
70	22	3.12	0.66	0.87	0.94	0.97	1.00	1.03	1.06	1.09	1.12	1.15	1.19	1.22	1.25
80	25	3.42	0.72	0.96	1.03	1.06	1.09	1.13	1.16	1.20	1.23	1.27	1.30	1.33	1.37
90	28	4.73	0.78	1.04	1.12	1.16	1.19	1.23	1.27	1.31	1.34	1.38	1.42	1.45	1.49
100	31	4.03	0.85	1.13	1.21	1.25	1.29	1.33	1.37	1.41	1.45	1.49	1.53	1.57	1.61
110	34	4.33	0.91	1.21	1.30	1.34	1.39	1.43	1.47	1.52	1.56	1.60	1.65	1.69	1.78
120	37	4.64	0.97	1.30	1.39	1.44	1.48	1.43	1.58	1.62	1.67	1.72	1.76	1.81	1.85
130	40	4.94	1.04	1.38	1.48	1.53	1.58	1.63	1.68	1.73	1.78	1.83	1.88	1.93	1.98
140	43	5.24	1.10	1.47	1.57	1.62	1.68	1.73	1,78	1.85	1.89	1.94	1.99		
150	46	5.55	1.17	1.55	1.6"	1.72	1.78	1.83	1.89	1.94	2.00				
160	49	5.85	1.23	1.64	1.76	1.81	1.87	1.93	1 99						
170	52	6.15	1.29	1.72:	1.85	1.91	1.97								

Using the NOAA pO₂ Chart Determine pO₂ of a 32% mix being breathed at 110 fsw

Depth		atm	2	25 - C		2	₹		· · · ·	2) - A					
(fsw)	(msw)	abs	21%	28%	30%	31%	32%	33%	34%	35%	36%	37%	38%	39%	40%
0	0	1.00	0.21	0.28	0.30	0.31	0.32	0.33	0.34	0.35	0.36	0.37	0.38	0.39	0.40
35	11	2.05	0.43	0.57	0.62	0.66	0.66	0.68	0.70	0.72	0.74	0.76	0.78	0.80	0.82
40	12	2.21	0.46	0.62	0.66	0.69	0.71	0.73	0.75	0.77	0.80	0.82	0.84	0.86	0.88
50	15	2.52	0.53	0.71	0.76	0.78	0.81	0.83	0.86	0.88	0.91	0.93	0.96	0.98	1.01
60	18	2.82	0.59	0.79	0.85	0.87	0.90	0.93	0.96	0.99	1.02	1.04	1.07	1.10	1.13
70	22	3.12	0.66	0.87	0.94	0.97	1.00	1.03	1.06	1.09	1.12	1.15	1.19	1.22	1.25
80	25	3.42	0.72	0.96	1.03	1.06	1.09	1.13	1.16	1.20	1.23	1.27	1.30	1.33	1.37
90	28	4.73	0.78	1.04	1.12	1.16	1.19	1.23	1.27	1.31	1.34	1.38	1.42	1.45	1.49
100	31	4.03	0.85	1.13	1.21	1.25	1.29	1.33	1.37	1.41	1.45	1.49	1.53	1 .57	1.61
110	34	4.33	0.91	1.21	1.30	1.3	1.39	1.48	1.47	1.52	1.56	1.60	1.65	1.69	1 73
120	37	4.64	0.97	1.30	1.39	1.44	1.48	1.43	1.58	1.62	1.67	1.72	1.76	1.81	1.86
130	40	4.94	1.04	1.38	1.48	1.53	1.58	1.63	1.68	1.73	1.78	1.83	1.38	1.93	1.98
140	43	5.24	1.10	1.47	1.57	1.52	1.68	1.73	1.78	1.83	1.89	1.94	1.99		
150	46	5.55	1.17	1.55	1.6"	1.72	1.78	1.83	1.89	1.94	2.00				
160	49	5.85	1.23	1.64	1.76	1.81	1.87	1.93	1.99						- <u> </u>
170	52	6.15	1.29	1:72:	1.85	1.91	1.97			10 J			· · · ·		A

pO₂ 1.39 ata







