# Gas Issues in Diving

# Gas Issues

- TOO MUCH
  - $O_2$, $CO_2$, inert gas narcosis, High Pressure Neurological Syndrome
- TOO LITTLE
  - Hypoxia, hyperventilation
- WRONG GAS
  - CO poisoning, contaminants

# CNS Ox Tox "VENTID-C"

- **V**ision changes (↓acuity, dazzle, lat movement, constricted fields)
- **E**ars (tinnitus, auditory hallucinations, music, bells, knocking)
- **N**ausea/vomiting
- **T**witch (lips, cheek, eyelid, tremors…)
- **I**rritability, behaviour, mood changes (incl apprehension, apathy, euphoria)
- **D**izzy
- **C**onvulsions
  - Also pallor, sweaty, palpitations, brady, tachy, panting, grunting, unpleasant gustatory/ olfactory sensations, hiccups

- Risk Factors
  - Exercise
  - Hyper/hypothermia
  - Hypoventilation, hypercapnia
  - Immersion
  - Metabolic activity, blood flow to brain
  - Hypoglycemia (DM)
  - Seizure D?O, +/- meds that lower sx threshold
  - VitE deficiency
  - Pseudoephedrine, amphetamines, ASA, acetazolamide
  - Spherocytosis, hypercortisolism

# CNS OxTox

- No consistent pre-convulsion warning sx
  - Often not preceded by other sx
- $O_2$ convulsions **not** inherently harmful
  - No pathologic changes in human brain, no evidence of clinical sequelae
  - No apparent predisposition to future sz disorder
  - Harm based on context (seizure underwater = drowning)
- Very high intra & inter-individual variation in susceptibility
  - ?increased risk with drugs that lower sz threshold (not much evidence)

- Tx
  - Remove $O_2$
  - Stop travel
  - Protect from injuries if seizing
  - Check DDx (don't forget hypoglycemia)
  - Keep patient off $O_2$ for 15 mins *after all sx are gone*
  - Ignore treatment time lost and resume table where last interrupted
    - Don't forget to compensate for extra time for chamber attendants
- Preventive Measures
  - Air breaks
  - Clinical HBOT setting (rarely used)
    - Glutathione
    - Lithium
    - GABA agonists

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# Pulmonary Oxygen Toxicity

- Cumulative dose = fx of exposure time, ATA, and FiO2
- Acute Δ with FiO2 > 0.8 ATA
- Chronic Δ with FiO2 > 0.5 ATA
- Typically insidious mild substernal irritation, chest tightness -> ↑cough -> constant burning exacerbated by inspiration -> dyspnea (exertion or rest)
  - ~12-16 hrs @ 1 ATA, ~3-6 hrs @ 2.0 ATA
- CXR usually N, +/- patchy infiltrates
- Mechanical fx impaired earlier than gas exchange (CO diffusing capabilities)
  - No change FEV1
  - ↓FVC
    - 2%, asx, completely reversible over hrs
    - 10% = mild sx, reversible over several days
    - 20% = mod sx, probably reversible over weeks, acceptable for a TT
  - ↓Diffusion capacity, FEF 25-75, V/Q defect
- Acute Exudative (reversible)
  - Interstitial and alveolar edema, hemorrhage, destruction of pulm capillary endothelium, loss of type I alveolar cells (surfactant), inflam. cell infiltrates
- Acute Proliferative (non-reversible)
  - Type II alveolar cells replace damaged type I (blood-air barrier thickens), fibroblast infiltration, increased alveolar-capillary distance, ↓alveolar air vol, ↑collagen content
- Chronic
  - Progressive pulmonary fibrosis, similar to ARDS
- Preventions:
  - Air breaks
  - Unit Pulmonary Toxicity Dose (UPTD)
    - 1 UPT = pulm poisoning produced by 100% O2 x 1 min at 1 ATA
    - HBOT Max 1440 UPTD/24hrs (TT6 = 750 UPTDs)

# Nitrogen Narcosis "Rapture of the deep"

- Reversible depression of neuronal excitability due to inert gas
  - Potency Xe >Kr > Ar > N > H > Ne > He
  - ?Interfere with transmission of EP across synaptic gap
  - Meyer-Overton theory: ↑solubility = ↑narcotic effect
- Immediate onset at depth, stable after few mins at depth, rapid resolution upon ascent
  - Potentiated by ↑CO2 levels
  - **No true acclimatization**, divers may dev short term subjective tolerance, **NO** objective tolerance
- RFs
  - Depth, gas mix, anxiety, task loading, cold, fatigue, exercise, EtOH, sedatives, ↑CO2
- S/Sx
  - ++**Inter/intra-variability** (~60-120ft onset)
  - ↓performance mental/manual work (higher fx affected most)
  - Dizzy, euphoria, uncontrolled laughter
  - Overconfidence, overly talkative
  - Memory loss/post-dive amnesia
  - Perceptual narrowing (fixation)
  - Impaired sensory functioning
  - LOC >100msw
- Prevention
  - Depth <30msw, plan dive ahead & practice tasks
  - If affected – decrease depth
  - Heliox

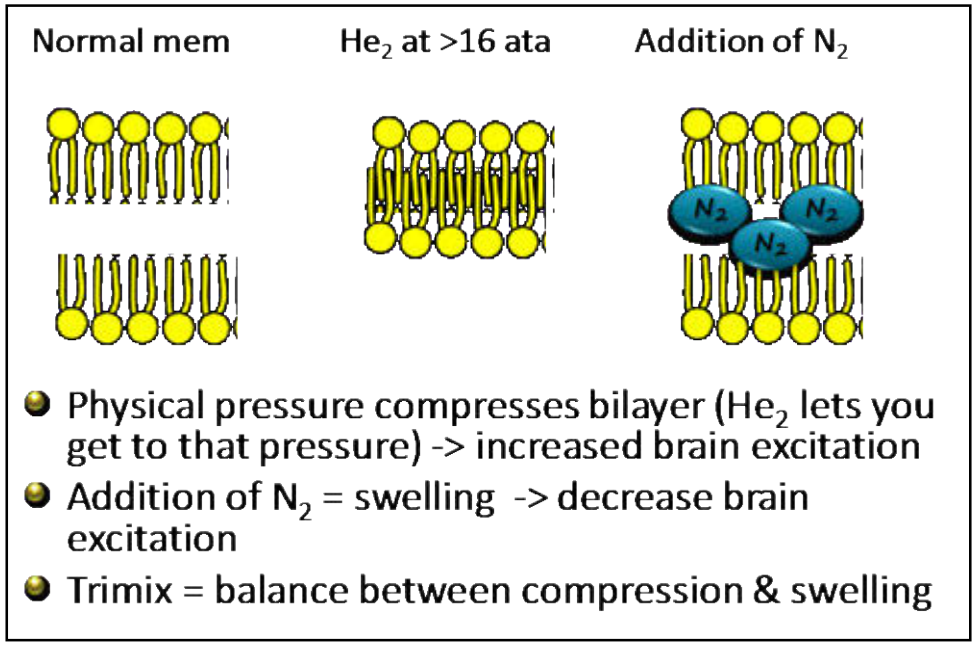
# $CO_2$ Toxicity

- Inadequate ventilation
  - Helmet diving, hyperbaric chamber
  - Alveolar hypoventilation
- Higher inspired CO2 = failure of $CO_2$ scrubbers in rebreather systems
- $CO_2$ retention (increased WOB underwater)
  - Increased $CO_2$ levels unpredictable, even in normal healthy divers
- Inadequate pulmonary ventilation
  - Increased density of gas
  - Deliberate hypoventilation or 'skip-breathing' – NEVER skip breath, esp. at high PP
- S/Sx
  - H/A, flushing, sweating
  - Dizzy
  - Dyspnea
  - Decreased cognition, disorientation
  - LOC/convulsions
  - Makes everything else worse (NN, OxTox)
- Tx
  - End dive
  - Fresh air, +/- $O_2$

# High Pressure Neurological Syndrome (HPNS)

- General excitation of brain
  - Opposite to narcosis
- Occurs in very deep diving >16 ATA
  - Usually Heliox mixtures at this depth
- Affected by rate of compression
  - Rapid rate = increased severity at shallower depth
- S/Sx
  - Marked tremor hands/arms/whole body, dizzy, anorexia, nausea, vomiting
  - Fatigue, somnolence
  - Can progress to myoclonic jerks -> clonic seizures

- Prevention
  - Diver selection
  - ↓compression rate, long stages/holds (allow adaptation)
  - Use of $N_2$ (or other narcotic) in trimix



# Hypoxia

- Same sx as on the surface
- Important to know onset for rebreathers
  - Open circuit
    - Hypoxia at depth – almost never $O_2$ issue, gen $CO_2$ issue
    - Hypoxia at surface – almost always $O_2$ issue
  - Closed circuit
    - Hypoxia – sensor failure
- Prevention
  - Maintain gear & checks
  - Don’t run out of breathing gas
- Shallow-Water Blackout
  - Breath-hold diving
  - Remember – $CO_2$ produces drive to breathe
  - Hyperventilation reduces $CO_2$ levels below normal levels
    - $O_2$ levels may fall to a level causing LOC before $CO_2$ increases to breakpoint trigger for breathing
  - LOC underwater is never a good thing…

# CO Toxicity

- Typically contaminated air from improperly directed compression engine exhaust
- Pathophys
  - CO relative affinity for Hb 250x greater than O2
    - ↓O2 carrying capacity, ↑unbound Hb = left shift = ↓tissue/intracellular O2
  - Disturbs mitochondrial e- transport, ↑NO radicals, lipid peroxidation in brain
  - Cerebral vessels dilate, ↑coronary blood flow with ↓central resp -> cerebral hypoxia & cardiac arrhythmias
  - Acute mortality often due to ventricular arrhythmias due to hypoxic stress, myocardial impairment
- S/Sx
  - Headache, N/V, dizzy, weakness, vision changes, disorientation, ↓LOC, auditory dysfunction, cardiac arrhythmias, skeletal muscle necrosis -> ARF, pulm edema
  - Concomitant smoke inhalation sx
  - Cherry red skin colour rare, very late
- CO best assessed by blood carboxyhemoglobin (COHb)
  - Mortality/morbidity not correlated with COHb level
  - Pulse oximetry overestimate arterial O2
- Tx
  - ABCs, preserve airway
  - Ventilation, oxygenation
  - HBOT hastens CO dissociation beyond rate achievable by surface 100% O2