# Dive Medicine Aide-Memoire

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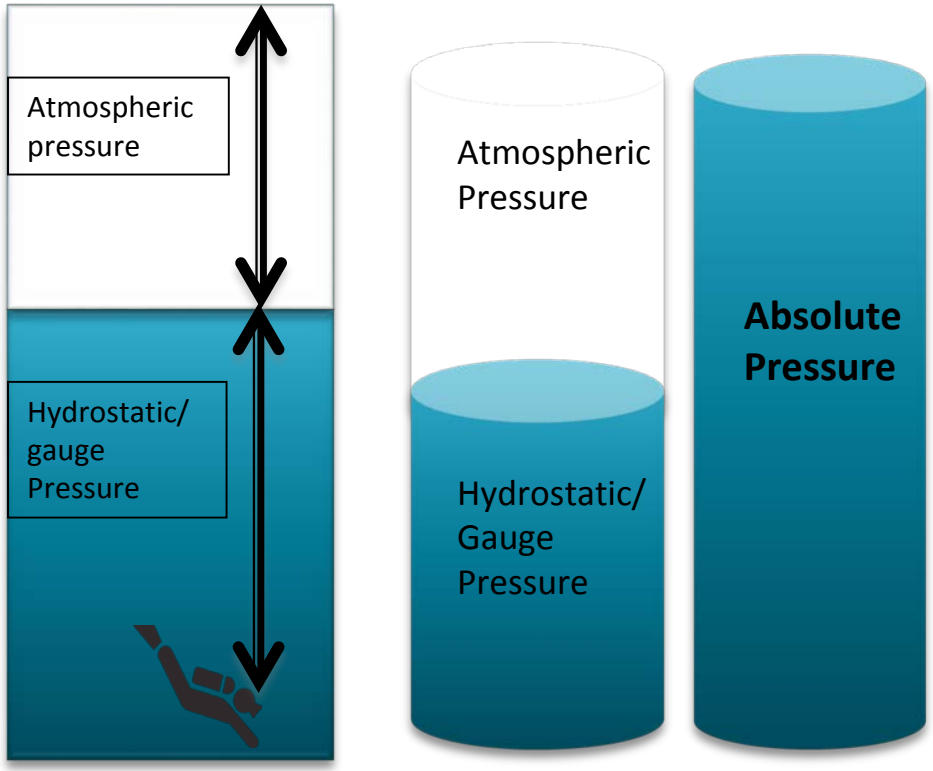
# Diving Physics

# Physics

- Air ~78% $N_2$, ~21% $O_2$, ~0.03% $CO_2$




| DEPTH | ABSOLUTE PRESSURE | GAUGE PRESSURE | AIR VOLUME | SURFACE VOLUME EQUIVALENT | EXAMPLE |
|---|---|---|---|---|---|
| 0 | 1 ATM | 0 ATM | 1 | 1 | 60 |
| 10 | 2 ATM | 1 ATM | 1/2 | 2 | 30 |
| 20 | 3 ATM | 2 ATM | 1/3 | 3 | 20 |
| 30 | 4 ATM | 3 ATM | 1/4 | 4 | 15 |
| 40 | 5 ATM | 4 ATM | 1/5 | 5 | 12 |

# Conversions

- Hydrostatic/ gauge pressure (P) = ~1 atm for every 10 msw/33fsw
  - Modification needed if diving at altitude
- Atmospheric P (1 atm at 0msw)
- Absolute P = gauge P + atmospheric P
- Water virtually incompressible – density remains ~same regardless depth/pressure
  - Density salt water 1027 $kg/m^3$
  - Density fresh water 1000$kg/m^3$
  - Calculate depth from gauge pressure you divide press by 0.1027 (salt water) or 0.10000 (fresh water)

- 1 bar = 101 KPa = 0.987 atm = ~14.5 psi
- 10 msw = 1 bar = 0.987 atm
- 33.07 fsw = 1 atm = 1.013 bar
- *Absolute P (ata)= gauge P* +1 atm
- °F = (9/5 x °C) +32
- °C= 5/9 (°F – 32)
- °R (rankine) = °F + 460 **absolute
- K (Kelvin) = °C + 273 **absolute

# Laws & Principles

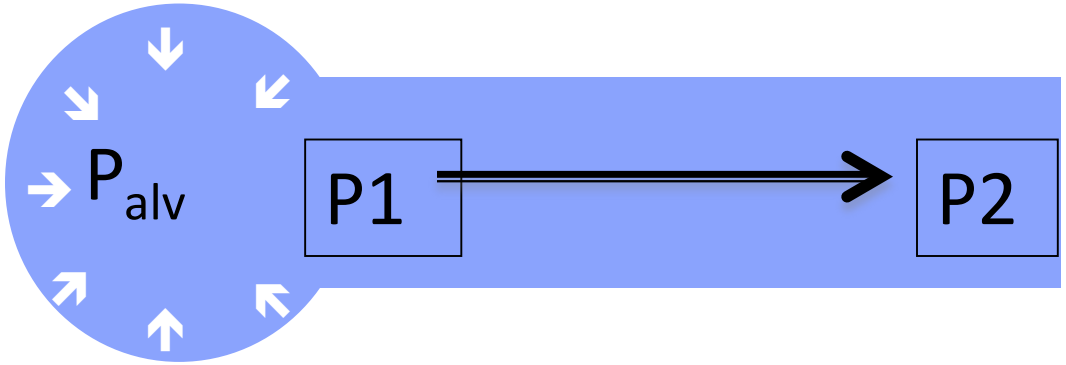
- All calculations require absolute units (K, °R, ATA)
- Charles' Law $V_1/T_1 = V_2/T_2$
- Guy-Lussac's Law $P_1/T_1 = P_2/T_2$
- Boyle's Law $P_1V_1 = P_2V_2$
- General Gas Law $(P_1V_1)/ T_1 = (P_2V_2)/ T_2$
- Archimedes' Principle
  - Any object immersed in liquid is buoyed up by a force equal to weight of the fluid displaced by the object
- Daltons' Law $P_{(total)} = P_1+P_2+\ldots+P_n$
  - The total pressure exerted by a mixture of gases is the sum of the pressures that would be exerted by each gas if it alone were present and occupied the total volume
- Henry's Law:
  - The amount of gas that will dissolve in a liquid is almost directly proportional to the partial press of that gas, & inversely proportional to absolute temp
- Partial Pressure (pp) – pressure contributed by a single gas in a mix
  - To determine the partial pressure of a gas at any depth, we multiply the press (ata) x %of that gas Henry's Law
  - Gas molecules enter liquid – add to gas tension (=partial press gas in liquid)
- Pressure gradient = Δ between gas tension in the liquid and gas partial press outside liquid
  - High gradient (low tension high PP) = high rate absorption of gas into liquid

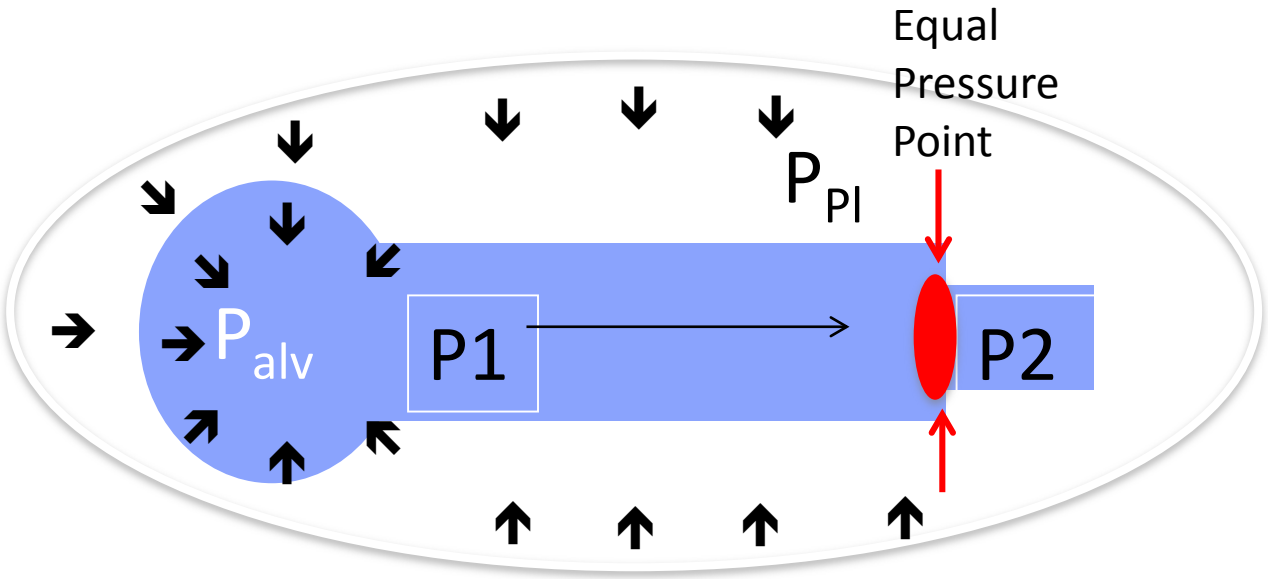
# Physiologic Implications Underwater Breathing Apparatus

# Physiologic Implications Underwater Breathing

- Increased $CO_2$ levels
  - $CO_2$ retention if breathing inadequate to eliminate $CO_2$ produced.
  - Common in all diving due to ↑WOB
    - Immersion effects
    - Gas density
    - Equipment effects
  - Exacerbated by exercise/work, panic, hyperventilation, dynamic airway compression
- Gas Density
  - increased depth = increased density (Gas law) = decreased flow (Poiseuille's law) = increased WOB
- Immersion Effects
  - P Δ feet to chest ~120cm $H_2O$, negative static lung load dev when lungs deeper than reg/counterlung
  - Fluid shift to central circulation
    - ↑cardiac afterload, ↑blood to lungs = ↓lung compliance, ↓ VC, ↑ airway resistance, diuresis
- Equipment Effects
  - All underwater breathing equipment increases breathing resistance
    - i.e. Demand valve, dead space in helmet/face masks/hoses
  - Movement of $CO_2$ from blood to lungs based on gradient/press Δ
    - Rebreathers – if inhaling $Co_2$ = smaller Δ = less $CO_2$ ventilated

# Dynamic Airway Compression (DAC)




- Normal Expiration – gas flows out along airway because P1 >P2
  - P1 -> P2 declines more quickly with dense gas
- At some point along the tube, airway P = intrapleural P ($P_{pl}$) = equal pressure point (EPP)
  - Beyond this point, P along airway is less than $P_{pl}$ = airway compression
- Catch 22
  - Harder you try to exhale,
    - higher the $P_{Pl}$ = shift EPP to the left
    - ↑ WOB which ↑ $CO_2$
- Distal movement of EPP
  - Increased a/w resistance (asthma, gas density), reduced lung elastic recoil, negative static lung load

# 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

# Oxygen Toxicity

- Toxic effects due to $PPO_2$ (not %)
- CNS main concern but pulm may become an issue with extended dive ops (serial bounce, sat)
- $PPO_2$ limits CF diving
  - 1.6 ATA routine
  - 1.8 ATA exceptional (requires CO sign-off)

# Ocular OxTox

- Decreased peripheral vision ~2.5-3.0 hrs at 3.0 ATA
  - Central vision unaffected, usually resolved within 30-45mins after treatment stops
- Progressive myopia
  - Generally 2-4 weeks of treatment and usually completely reversed after ~3-4 weeks (sometimes up to 1 year)
    - DM, elderly more susceptible
    - Unclear whether hood/monoplace increase risk vs. facemask

# CNS Ox Tox

- **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 $O_2$
  - 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

# 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)

# $CO_2$ 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, +/- $O_2$

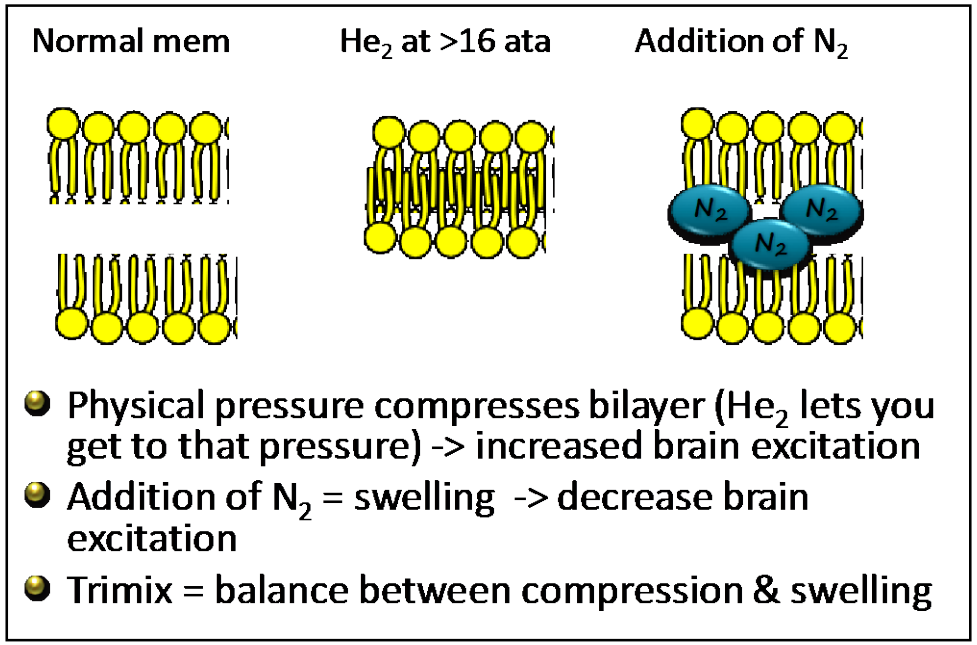
# Nitrogen Narcosis “Rapture of the deep”

- 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
- 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 tolerance
- RFs
  - Depth, gas mix, anxiety, task loading, cold, fatigue, exercise, EtOH, sedatives, ↑CO2
- S/Sx
  - ↓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

# 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

# Decompression Illness

# DCI

- Decompression illness (DCI) includes DCS and AGE
- ~1-10/10,000 dives
  - Higher in cold water, deep; lower in recreational warm water diving (1-4/10k)
- Traditional/Golding Classification
  - Type I (MSK, skin, lymph, fatigue)
  - Type II (neuro, cardio-resp, ENT, shock)
  - AGE
- Descriptive/Francis Smith Class
  - Evolution (spontaneous recovery, static, relapsing)
  - Progressive (increasing #, severity of s/sx)
  - Organ System
    - Neuro, cardio-pulm, MSK, skin, lymph, ENT
  - Time of onset (before or after surfacing)
  - Gas burden
    - Low (conservative within NoD), Med (D Dive), High (violation dive table)
  - Evidence of barotrauma
- Diagnosis – generally hx, estimation of likelihood
  - Sx depend on location of insult
  - <24 hrs possible, >24 hrs unlikely, >36 hrs very unlikely, >48 hrs almost impossible unless altitude change
  - There is no pathognomonic test for DCI
- Tx
  - 100% Surface O2
  - IVF
  - Evacuation considerations
    - Airway, foley, pressurized cabin or as low as possible
  - HBOT

# DCS Pathophysiology

- Henry's Law – amount of inert gas absorbed by blood/tissue increased at depth
- Boyle's Law $P_1V_1 = P_2V_2$
- Bubble effects
  - Intravascular - embolism, vasospasm, ischemia, transbolism, venous stasis, hemorrhage, blood-bubble interactions, mechanical stripping of endothelial cells
  - Extravascular -tissue disruption, tearing, hemorrhage, localized "compartment syndrome" – ischemia, stasis
  - Typically peri-alveolar capillary network 'traps' venous gas – but can be overcome (# bubbles, repet diving)
- Inflammatory & thrombogenic processes
  - Association with oxidative stress, microparticles
  - Bubbles biologically active – form plasma-protein coat activating WBC, plts, fibrin web
    - "Thick skin" stabilizes bubble, decreases diffusion of inert gas out of bubble
  - Recurrence of sx likely due to secondary rxn vice initial bubble

# Cutaneous, PNS

- Cutaneous angio-lymphangiologic DCI
  - Erythema, lymphedema
  - Cutis Marmorata –
    - Venous congestion, infl, WBC activation, and endothelial damage
    - Associated with pulmonary and neuro DCI, thus requires careful monitoring
- Cutaneous diffusion
  - ‘Diver’s Lice’: erythematous rash, typically with dry chamber dives or dry suits
    - ?inert gas enters skin directly, causing dermal bubbles and histamine release on decompression
    - Benign, no RCC required
    - Tx if dx in doubt
- PNS
  - All that tingles is not the bends!!!
  - Don’t forget about non-dysbaric neuropraxias: ulnar, median, inferior brachial plexus, lateral cutaneous femoral  and sciatic
  - Tight wet suit, weight belts, heavy equipment, BCD straps, sitting on side of boat etc.

# MSK

- Most common presentation of DCS
- “Bends” typically only affects long bones of appendicular skeleton (not axial skeleton)
  - Adult long bones contain fatty marrow cavity ?reservoir for inert gas
- Pain mechanisms
  - Intra-articular
    - “Niggles”/ marginal DCS if mild sx that begin to resolve 10min after onset
  - Peri-articular - (within tendon, muscle); “niggles” if brief
  - Medullar/sinusoids - gas expansion within medullary cavity, fatty marrow and bone sinusoids
  - Referred pain - injury to nerve roots assoc with joint, generalized release of infl modulators

- MSK speculation
  - Unk if any long-term effects for #2-3 if no RCC (if this is DCS)

| | Pain | Potential Cause |
|---|---|---|
| 1. Localized sharp | Affected by movement | Tendon/muscle injury (this is what moves) |
| 2. Localized sharp | Unaffected by movement “diver rubs at it” | Local infl; (?DCS) |
| 3. Poorly localized, deep boring pain | Affected by movement | Intra-articular, joint capsule tension; (?DCS) |
| 4. Poorly localized, deep boring pain | Unaffected by movement | Bone medulla injury (DCS!) |

# Spinal Cord DCS Pathophys

- Spinal cord white matter in a C-shaped area around the spinal cord grey matter
  - Watershed zone between ant and post spinal cord circulation – susceptible to both inert gas accumulation and bubble-related ischemia
  - Cervical and lumbar enlargements particularly vulnerable
- Presentation is likely combo of interacting compressive/ ischemic mechanisms
  - Gas embolism
    - But blood flow favours embolization to the brain, and experimental spinal cord embolism generally produces grey matter pathology vs. white matter
  - Venous infarction – bubble accumulation in epidural venous plexus
    - Can't explain ultra-short-latency cases
    - Usually produces grey matter lesions, not white like DCI
- Autochthonous bubbles – spontaneous bubble formation in spinal cord white matter
  - Direct axon destruction with 2° effects (hemm, infl, stretch/compress)
  - Explains rapid onset, lesions in white matter
  - But how are these small isolated lesions able to produce such significant sx?
- Hemorrhage and inflammation
  - Observed in same areas where autochthonous bubble injuries were seen
  - Could explain why some cases of rapid onset spinal cord DCS resistant to recompression

# IE DCS Pathophys Theories

1. Counter diffusion
   - Conditions where inert gas in middle ear differs from gas in the breathing mixture (gas switch)
     - -> Diffusion through round/oval window could result in accumulation of inert gas with bubbling
   - Blood supply to inner ear isn't uniform (stria vascularis supplies endolymph directly, then diffuses to perilymph)
     - Endolymph could rapidly take up new inert gas before perilymph had time to eliminate former inert gas -> bubbles form in endolymph
2. Gas induced osmosis
   - Similar to #1, inert gas accumulation in endolymph induces osmotic fluid shift toward endolymph -> hydrops endolymphaticus (similar to Meniere's)
3. Explosive/hemorrhagic
   - Gas accumulation in temporal bone osteoclast pockets that explosively rupture into inner ear during decompression
   - Plausible for deep mixed diving, blow-up from saturation
4. Embolism
   - Inner ear blood supply is end-arterial, thus would be prone to embolic or vascular injury

# AGE

- Subset of DCI
  - ~1/5000 USN experimental dives
    - Low among mil working divers on standard tables, ~1/10k
  - 2nd most common cause diving fatalities
    - Australian dive fatalities 1972-2005, AGE cause in 25% of cases (2nd most common)
- Mechanisms
  - PBT
  - Intracardiac shunt (“safe dives make bubbles”)
  - Trans-pulmonary shunt
  - A-V or bronchopulmonary fistula (rare)
  - In-situ bubble formation (not likely)
  - 50% diving cases no identified cause, most neuropath info from iatrogenic CAGE
- DDx: Neuro DCS, CVA, carotid artery dissection, cardiac, other neuro process
- RFs – same as POS
- S/Sx
  - Rapid onset <10 mins, rapid progression
  - Neuro sx occurring immediately after surfacing (esp. shallow, short dive) should be considered AGE until proven otherwise
  - +/- POS Sx
  - Neuro deficits based on bubble location (LOC, confusion, paresis, sensory loss, apnea, aphasia, visual loss (field >acuity), vertigo, ataxia, seizure, isolated personality or cognitive change etc.)
    - +/- neuropsychiatric, EEG changes
  - 5% dead on the spot –cardiac airlock
  - Systemic hypertension and bradycardia
  - Liebermeister’s sign (sharply demarcated pallor on one half of tongue)
  - CK increased with peak 12hrs post onset
    - Correlates with severity, outcome (<1000 likely full resolution)
  - MR head: nothing -> focal or multifocal ischemia -> edema

# AGE

- Tx - ABCs
  - Supine, recovery position if LOC or airway not controlled
  - 100%  O2, euvolemia with IVF (RL)
  - +/- ETT (low press/volume)
  - Lidocaine (neuroprotective) 1mg/kg slow IV bolus, then 1-4mg/min (*controversial, may lower sz threshold)
  - IV Benzo for seizures, agitation
  - Chest tube if concurrent pneumo
  - HBOT - TT6 or Comex 30
    - ?Helium benefit after CVA (Comex 30 on 50/50)
    - Tx until plateau (h/a not indication for re-tx)
- Medevac ASAP – pressurized aircraft to 1 ATA, or fly as low as safely possible

- Complications
  - Relapse seen in ~30% as late as 68hrs
    - Edema, re-embolization, ischemia reperfusion, endothelial damage,
  - Concurrent DCS – can be resistant to Tx
  - Drowning
- Investigations
  - Carotid Doppler, contrast echo, CXR, CT chest, MRI brain (residual damage), PFTs
- Prognosis/RTD
  - Most have good outcome w/ resolution if prompt tx
  - R/O predisposing factors
    - ? Risk of recurrence if no RF identified
    - Unknown if 2nd occurrence means worse outcome than 1st
  - AUMB decision RTD
    - Any residual sx – unfit dive
    - Asx after HBOT – case-by case

# Pulmonary Barotrauma

# Pulmonary Overpressure Syndrome (POS)

- POS
  - Pneumomediastinum, subQ emphysema
  - Pneumothorax (rarely seen in diving)
  - AGE
- Overexpansion of lungs (breathing compressed gas) & can't properly ventilate expanding gas volume with ↓press
  - Boyle's law: Largest volume changes near surface, breath-hold ascent from 4fsw sufficient
  - Air tracks along bronchi to outside the lung, or into adjacent blood vessels
  - Extrinsic: breath-holding on ascent – panic, out of air, buddy breathe, laryngospasm, sub escape
  - Intrinsic – obstruction or restrictive lung disease -> local air trapping, Δlung tissue compliance
- Most pathological studies indicate shear at terminal bronchioles and marginal alveoli rather than rupture of alveoli
- ?Blebs and bullae
  - COPDers with B&B don't have ↑incidence of PBT during HBOT (BUT no immersion, slow ascent rates)
  - B&B found post PBT may be either cause or consequence
- ? Role of obstructive airways. Mixed evidence
  - FEV 1 has low correlation to PBT risk
  - MEF 25 has moderate correlation to PBT risk
  - FEV1/FVC does not correlate
  - Asthmatics don't bear a much greater risk for diving-related intrinsic PBT than non-asthmatics
  - 50% of PBT/AGE survivors, no abnormality of lung fx detected
- ? Role of compliance
  - Pathology more consistent with shear than rupture, implies regional differential compliance
  - FVC correlated with PBT risk
  - Chest binding (reducing relative regional difference in compliance) ↓risk for PBT
  - Higher incidence PBT while immersed vs RCC (pulm blood pooling with immersion reduces interstitial compliance)
  - Pathology - weak correlation between shear site and location of pre-existing scars/fibrosis

# POS

- S/Sx
  - Asx or if sx, usually on ascent or shortly after surface – cough, hemoptysis, CP, SOB, resp distress, pleuritic/substernal CP
  - +/- sx of AGE
  - Pneumomediastinum – decreased heart sounds, dysphonia (brassy, monotone), Hamman's sign, recurrent laryngeal paresis, pseudo-tamponade
  - SubQ emphysema – crepitations felt in soft tissues
- CXR
  - Free gas at margin of heart/vessels, pseudo-pneumopericardium, subQ gas
  - Pneumothorax
  - Pleural effusion
  - Intravascular gas with massive AGE
- Tx - ABCs
  - O2
  - Needle deco/CT for tension pneumo (rare)
  - RCC only if AGE
  - Supportive SC/mediastinal emphysema
  - ER/Thoracic referral – urgency based on sx
- Screening – controversial
  - CXR low predictive power
  - CT – many abnormalities (?clinical significance), expensive, ++radiation
  - Spiro – poor correlation to PBT risk
    - FEF 25-75% abnormalities – small airway Fx (ensure adequate curve before interpreting numbers)
  - MTC – not adequate sens/spec
  - Asthma Exercise Challenge
    - 80% HHR x 8 mins with FEV1 at 15, 30, 60 mins post
    - FEV1 decrease by 15% or more is positive
  - Eucapnic voluntary hypercapnea, hypertonic (4.5% saline) and mannitol
- Disposition (case by case)
  - CDSM/AUMB based on cause, investigations
  - PFT, HRCT (insp, expiration)
  - 'Deserved' with normal f/u invs – potential RTD
  - 'Undeserved' or persistent sx/pathology – likely unfit diving

# PBT of Descent (Lung Squeeze)

- Risk with deep breath hold diving
  - As descend, ambient pressure compresses lungs (Boyle's law, gas is compressible)
    - At surface, hold ~6L
    - At 6 ATA (50 msw) ~1L
    - Breath hold divers go much deeper than this! >150msw
  - Lungs begin to fill with fluid and blood, now more liquid and less compressible

# Non-pulmonary Barotrauma

# Non-pulmonary BT

- Sinus Squeeze
  - 12% divers experience
  - Air trapped in sinus (i.e. sinus passageway blocked) decreases in volume as you descend
  - Can “suck” soft tissues into gas space -> blood vessels engorge and leak blood
  - Blood will often drain during ascent
    - Air expands, open passageway
    - H/A commonly accompanies
- Sinus BT
  - Air in sinuses that expands on ascent
- S/Sx
  - Pain, press, bloody nasal discharge, odontalgia
- Treatment is symptomatic
  - Nasal, PO decongestants
  - Abx if dev infection
  - Refer if #
- RFs
  - URTI, allergic rhino-sinusitis, acute or chronic infectious sinusitis, nasal polyps, mucosal retention cysts, deviated nasal septum, congenital osteo-meatal dystrophy, Wegener’s, rhinitis medicamentosa
- Prevention
  - Don’t dive with URTI, allergies, nasal deformities (deviation, polyps)
  - Caution med use while diving – can wear off
  - ENT referral PRN, eg if recurrent

# Non-pulmonary BT

- Mask/Face Squeeze
  - Air in mask also follows Boyle's law
    - Need to add air to space during descent otherwise gas compressed and "sucks" facial soft tissues/eyes in
  - Tx largely symptomatic
  - Prevention - ensure to equalize your mask during descent
- Suit Squeeze
  - Air in drysuit compresses against skin on descent, skin forced into folds
  - Seal around neck can constrict
  - Tx – symptomatic (ensure DDx)
  - Prevention - light garment under suit, ensure adequate air layer
- Ear Squeeze/BT
  - See ENT section
- Dental BT (rare)
  - Air trapped within tooth expands on ascent or air pocket distorted during descent
    - Decay, dental surgery, improper/loose fillings
  - Tx: Dental assessment, analgesia
  - Prevention
    - Dental hygiene, dental fillings replaced if loose, ?appropriate time between dental surgery & diving
- GI BT (rare)
  - Air expands/contracts in GI tract
  - Perf rare – if occurs likely lesser gastric curvature with panic ascent
  - Prevention - avoid swallowing gas (anxious, nausea, head down ascent with Valsalva, excess reg press)
    - If cramps on ascent, stop and remove gas

# Non-Dysbaric Dive Injuries

# Underwater Blast Injuries

- EOD, explosives in salvage, UW cutting/welding, thunderflashes, combat explosives
- UW blast injuries
  - Primary due to shock wave = damage to gas containing organs (GI, lung, ENT, CNS)
  - Secondary due to displaced debris
  - Tertiary due to collision with stationary objects
  - Misc. - burns, radiation, etc.
  - Press wave travels 4x faster UW than in air
  - Typically no damage to non-gas containing organ unless impact/penetration wounds
- Tx
  - ABCs, ABCs, ABCs
  - $O_2$– lung damage may not be immediately apparent
  - HBOT only if signs of AGE, DCS
  - If require ventilation
    - Risk of AGE, pneumo
  - Judicious use of fluids
    - Avoid worsening pulmonary edema
    - Early use pressors vs IVF
    - NPO - May not absorb from GI
  - Specialist consult as PRN

# Heat-related Illnesses

- Heat cramps
  - Exertion, significant sweating, excess hypotonic fluid replacement -> salt depletion
  - Tx – PO salt solution (1/4 -1/2 tsp salt in quart of water), rest in cool env
- Heat Edema
  - Minimal edema, esp. feet & ankles
    - Diuretic not indicated
    - Tx – leg elevation +/- support stockings
- Heat Syncope
    - Generally elderly due to cutaneous vasodilation, pooling blood in lower limbs

- Heat Exhaustion (temp <40 °C)
  - Vague malaise, fatigue, headache, thirst, weakness, anxiety
  - Tachycardia, orthostatic hypotension, +/- dehydration
  - Tx
    - Cooling – ice packs (axillae, groins, neck), skin wetting/fans
    - Fluids, oral salt solution, +/- IVF
- Heat Stroke (temp > 40 °C)
  - Mild to severe CNS dysfx (delirium, LOC, seizure), skin hot/dry
  - Tx
    - Cooling – ice-water immersion, cold packs, skin wetting, fan, iced gastric lavage etc.
    - IVF (hypovolemic, rhambo -> ARF)
    - Lytes, glucose replacement PRN

# Cold Water Immersion

- Thermal conductivity water >> air
  - Body cools ~4-5x faster in water
  - EtOH impairs thermal perception
- Stage I – Initial immersion (0-3 mins)
  - Cold shock rxn= initial gasp, hypervent, intense vasoconstriction (due to rapid skin cooling)
- Stage II – Short-term response (3-30mins)
  - Superficial nerves & muscles cool
  - Immediate chilling hands/feet = inability to complete survival actions, swimming failure
- Stage III – Long-term immersion (>30mins) = hypothermia
  - Age, cold habituation delay onset shivering – faster fall body temp
  - Mild (35°C), mod (32-35°C), severe (25-32°C), profound (<25°C)
- Stage IV – post-immersion/ circumrescue collapse
  - ~15-20% immersion deaths during/immediately following rescue
  - Heart muscle cold, less efficient, increase viscosity blood, prone to arrhythmias
  - Rapid tissue warming may increase off-gassing before adequate periph blood flow restored, ?bubbles
  - During rescue – maintain pt horizontal
    - Collapse worse if rescued vertically, increased time of immersion
    - Cephalic redistribution of blood, BP maintained due to hydrostatic pressure

# Near-Drowning/Drowning

- Drowning ~100-150 scuba deaths per year
  - Entanglement, running out of air, cardiac events
  - Secondary to other injury, ?AGE/POS
- Major pathophys is hypoxemia
  - 10-15% do not aspirate water – laryngospasm
  - V/Q mismatch
    - Fluid-filled alveoli – perfused, not ventilated
    - Decreased ventilation -> increase $PaCo_2$, cardiovascular collapse
  - Dilute surfactant
  - Elevation diaphragm from gastric distention -> vomit & aspirate gastric contents
- Tx
  - Ventilation must be reestablished prior to developing end-organ injury from hypoxemia
  - ABCs, 100% $O_2$
    - +/- ETT, NG tube
  - Monitor serial Arterial Blood Gas
  - Antibiotics
    - Ocean water/pool generally not required unless dev fever, purulent excretions, new infiltrates on CXR
    - If aspirate known contaminated water, consider Abx
  - Ventilation – based on clinical presentation
    - BiPAP, PEEP etc.

# Immersion Effects

# Effects of Immersion

- CV Effects of Immersion
  - Increased central volume (700 ml) – hydrostatic P effect
  - Increased stroke volume
  - Dive reflex – bradycardia, peripheral vasoconstriction
  - Increased pulmonary artery pressure
  - Increased pulmonary capillary pressure

- Pulmonary Effects of Immersion
  - Reduced vital capacity (5%)
  - Reduced compliance
  - Increased closing volumes – functional gas trapping, increased FRC
  - Increased diffusion capacity
  - Relative V/Q mismatch apical vs basal
  - Active Na channel transport in alveolar epithelial cells impaired in cold -> reduced clearance alveolar fluid
  - Negative intra-alveolar pressures – dense gas, turbulent flow, regulator

# Immersion Pulmonary Edema

- Acute resp distress with pulm congestion, +/- hypoxia
  - No resolution on ascent (i.e. bad gas)
- S/Sx
  - Dyspnea, cough, +/- frothy or blood tinged sputum
  - Tachypnea, hypoxia
  - Rales ~25%, wheeze ~10%
  - Frequently become symptomatic during ascent or surface swim
- Diagnosis
  - CXR – no findings, interstitial edema, diffuse alveolar densities
    - ?CT more sensitive but ?significant
  - ABG: avg Sat 88%, PPO2 66mmHG, eucapnia
- Tx
  - Generally resolves within few hours ( 5 mins - 25hrs)
  - O2
  - Resistant cases
    - +/- β-agonist inhalation,
    - Rapid diuretic (IV lasix 20-40mg)
    - +/- BiPAP
- Usual CAF workup
  - BP, ECG, cardiac enzymes
  - Echo
  - EST
  - PFTs

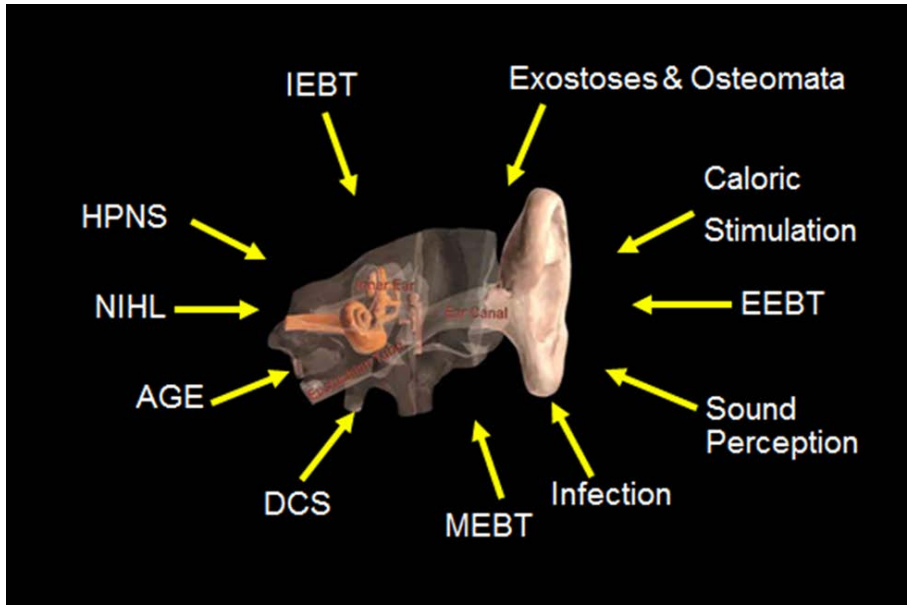
# Immersion Pulmonary Edema

- Prognosis
  - Recurrence 15-30%, unpredictable
  - CAF case by case RTD if all w/u negative
- Risk Factors
  - Incidence, mortality likely under-reported
  - Depth independent (2.9 msw), even surface only
  - Exertion during dive freq absent
  - Cold increases incidence but not necessary
  - Over hydration pre-dive may contribute (association)
  - ?tight wetsuit create neg intrathoracic press, ?NSAIDs – contrary or weak evidence
- Combination of immersion and:
  - Inspiratory restrictive load (high breathing resistance – faulty regulator)
  - Negative pulm pressure
  - Fluid overload
  - High intensity exercise
  - LV diastolic dysfunction
- Prevention
  - Equipment checks
  - Consider pre-dive hydration carefully
  - Sildenafil decr MPAP/CVP, incr venous capacitance/pulm vasodilation BUT possible risk DCS
  - Pre-immersion hyperbaric hyperoxia may protect in thermo-neutral water, not for cold water

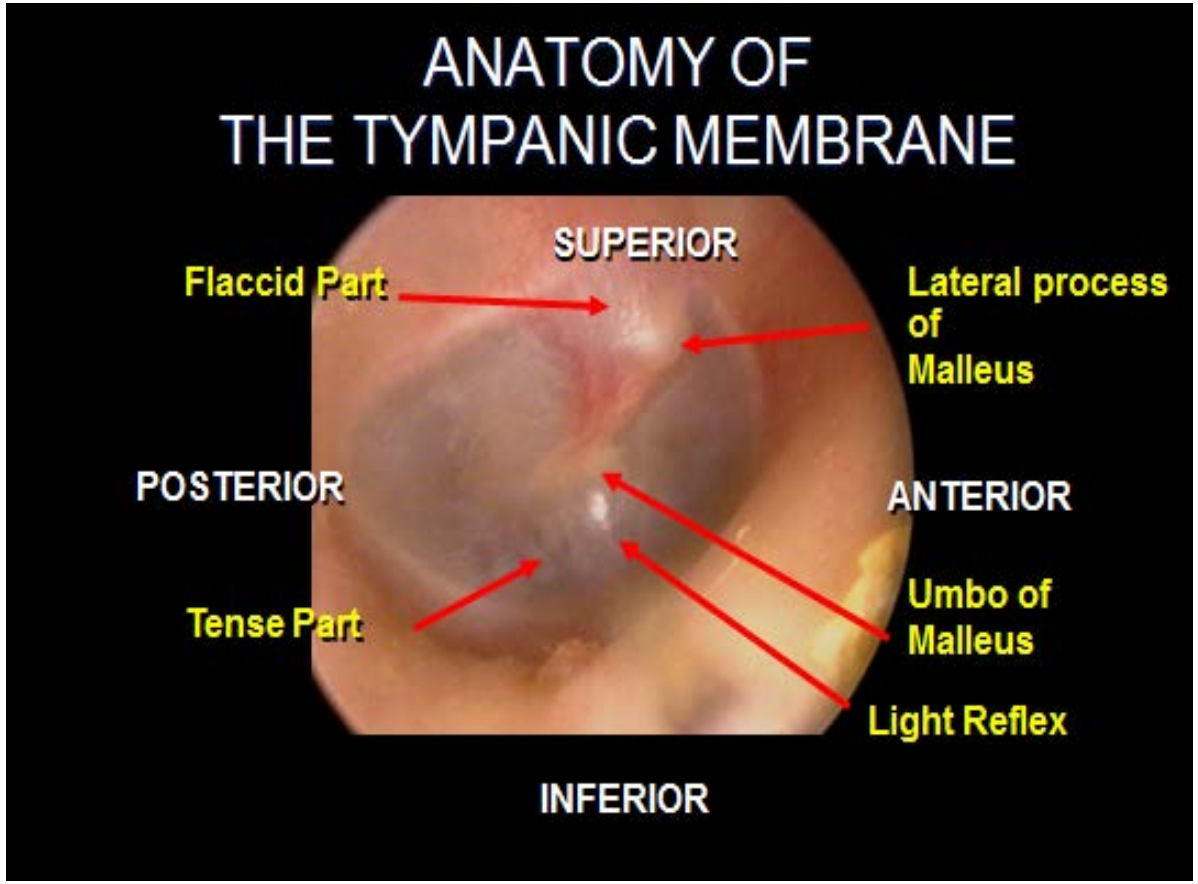
# Pulmonary Edema: Other Considerations

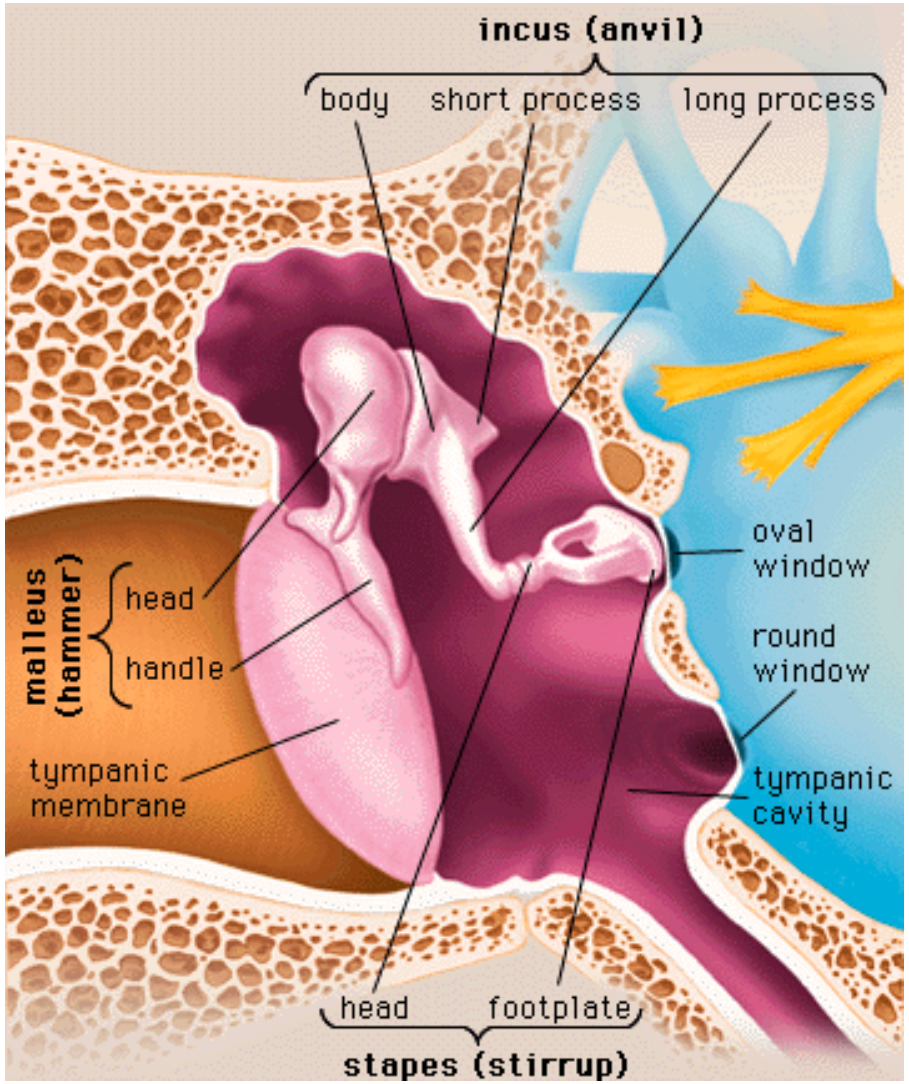
- Salt Water Aspiration Syndrome
  - Aspiration fine mist of seawater from leaking/flooded demand valve
  - May coexist with IPE
  - Tends to onset post-dive rather than on ascent
    - Often provoked by exercise, movement or cold exposure
  - Cough, fever, rigors, nausea, myalgia, SOB, mild leukocytosis
  - CXR – patchy consolidation v. interstitial edema
  - Tx – self-limiting, resolves without treatment within 2-24hrs
    - Rest, 100% O2
- Exercise on dry land
- Neurogenic Pulmonary Edema (elevated catecholamines -> peripheral vasoconstriction, increased pulm art P, pulm capillary leak)
- Hyperbaric hyperoxia in absence of immersion/exercise
- ~14% N. Americans have genetic β-2 adrenergic receptor polymorphisms predisposes to pulmonary edema with IVF

# Systems Focus: ENT



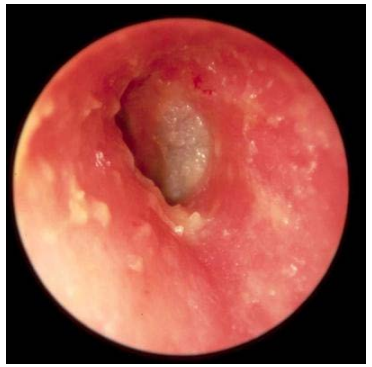
# Physiology


ANATOMY OF
THE TYMPANIC MEMBRANE
SUPERIOR
Flaccid Part
Lateral process of Malleus
POSTERIOR
ANTERIOR
Tense Part
Umbo of Malleus
Light Reflex
INFERIOR

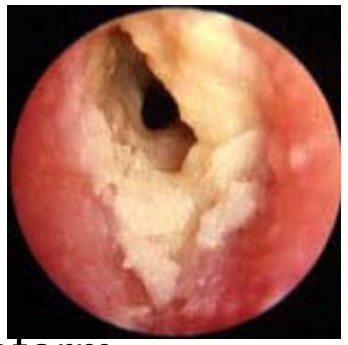

incus (anvil)
body
short process
long process
malleus (hammer)
head
handle
tympanic membrane
oval window
round window
tympanic cavity
head
footplate
stapes (stirrup)

# Otitis Externa

- Prevention
  - No Q-Tips
  - Olive oil/2% acetic acid/domeboro/ tea tree oil
- Tx
  - +/- gentle irrigation
  - VoSol HC
  - Cortisporin/Cipro HC/CiproDex/Garasone
  - Wick (betnovate 0.05%, gentamicin sulfate 0.1%, tolnaftate 1%)

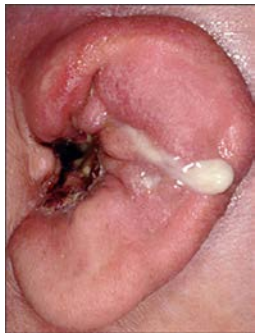


# Fungal Otitis Externa



- Aspergillus/Candida
- Can cause malignant OE
- Tx
  - $H_2O_2$ irrigation, wick
  - Clotrimazole 1%/Locacorten-Vioform ottic gtts
  - Lamisil/Sporanox PO

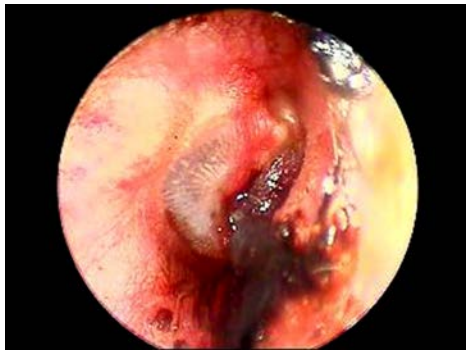
## Malignant (necrotizing)



- Pseudomonas
- Osteomyelitis/erosion skull base, CN paresis
- Cipro IV 400mg q8h or 750mg PO BID
  - Levoflox if increase resistance to Cipro

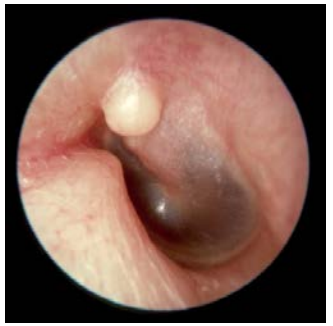
# Barotitis Externa

- Canal occluded
  - Hood, cerumen, oxostoses
- Doc's ProPlugs
  - Blocks water from entering ears in <20 fsw
  - Vented plug reduces abrupt press changes, ?easier equalization

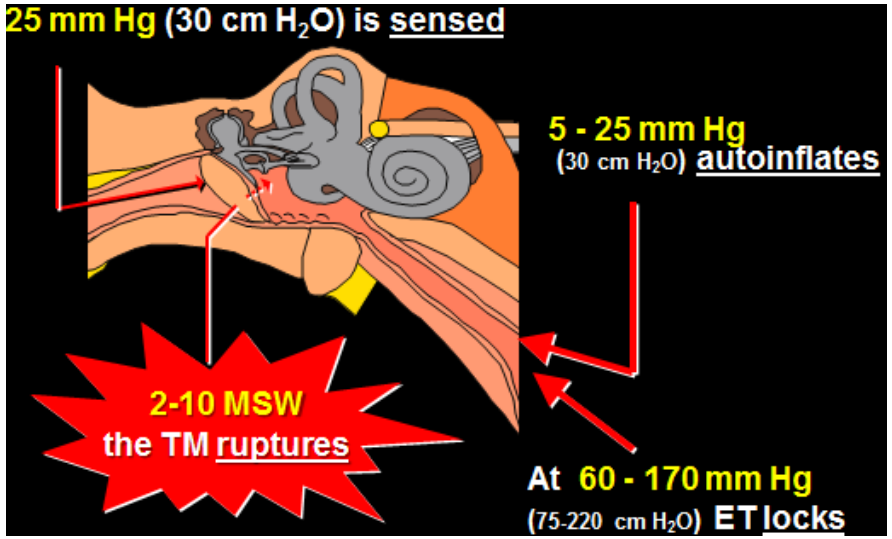


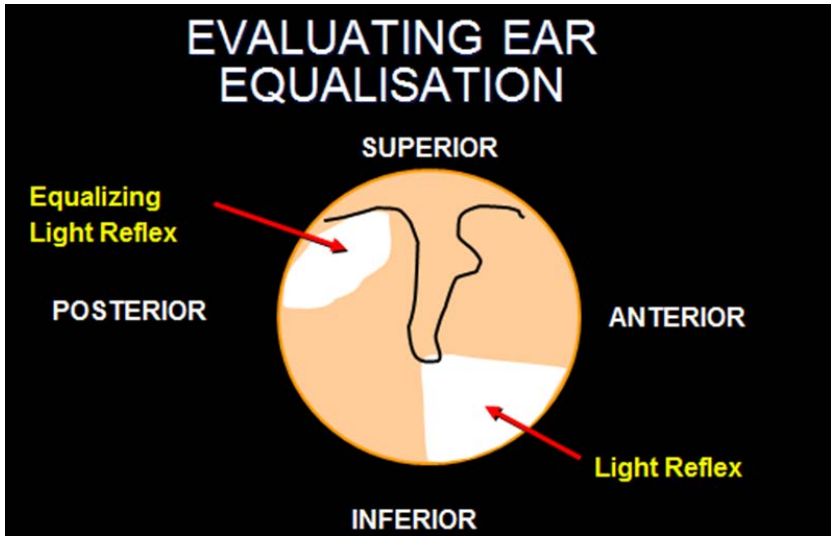
# Exostoses & Osteoma

- Periosteal rxn to cold water
  - "Surfer's ear"
  - Benign bony neoplasms
  - Typically single osteoma, multiple exostoses
- Occlusion -> hearing loss, infection, difficulties equalizing
- Surgical excision has high rate of associated HL & recurrence



# Equalization



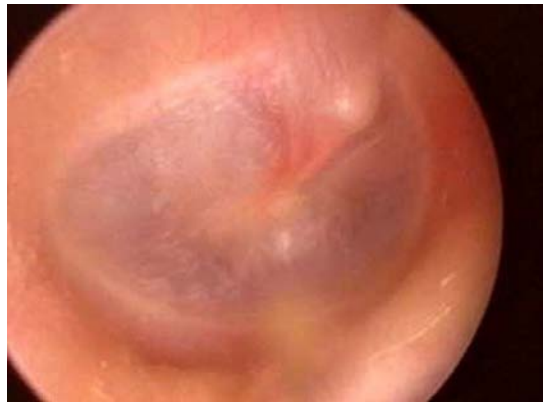

- Equalization
  - First equalization press felt at ~30cm
    - ET collapses if not equalized by 1.5msw
    - TM rupture if not equalized by ~10msw
- General Principles
  - Start early, blow gently
  - Don’t smoke, avoid agents causing vasomotor rhinitis (PDE -5)
  - Consider polyposis/deviated septum if persistent probs
  - Don’t dive when congested
    - Decongestants may allow descent and become ineffective for ascent
    - Don’t use if:
      - Unable to equalize without them
      - New diver
      - >4-5d continuously
      - CI present (anxiety, HTN…)
      - ↑ pO2 / ↑ pN2 (deep / mixed gas)

# Equalization Techniques

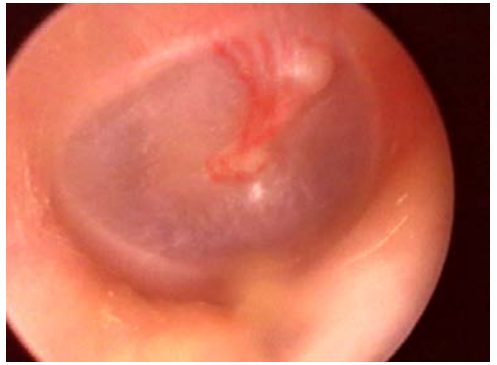
- Beance Tubaire Voluntaire (BTV)
  - Voluntarily open ET by “twitching” throat
  - Tensore veli palatini muscle
  - 30% pop can perform consistently
- Swallow/yawn
- Valsalva
  - Mod forceful attempted exhalation against closed airway
  - Never on ascent, never >5s
- Toynbee
  - Pinch nose & swallow
  - Small pressure diff, safe on ascent
- Frenzel
  - Closed glottis, move the tongue backwards quickly and forcefully against soft palate
  - Pinch nose for better effect
  - Gentle, safe for ascent
- Edmonds
  - Jut jaw forward
  - Combine with other techniques
- Lowry
  - Pinch nose, gentle blow against blocked nose & swallow
  - Difficult to perform
- Head tilt (bad ear up)
  - Combine with other techniques

# MEBT

- TEED 0
  - Sx of fullness/pressure with no otoscopic findings
  - Resolves in 2-24hrs
  - No Tx required
  - +/- decongestants

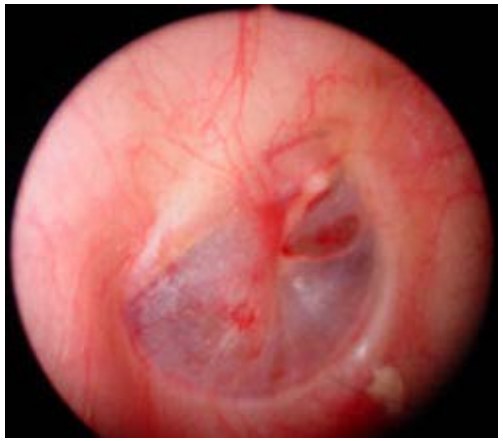


- Teed 1
  - Pressure, typically no pain
  - Erythema, retraction of TM
  - Resolves 24-48hrs
  - +/- decongestants

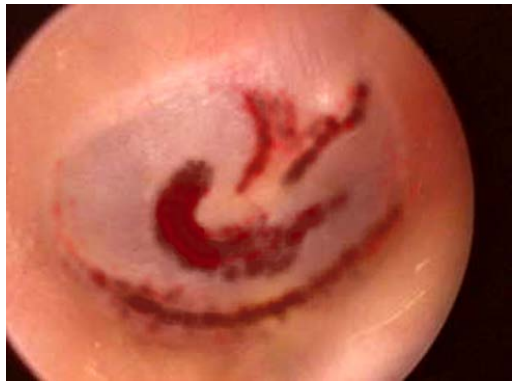


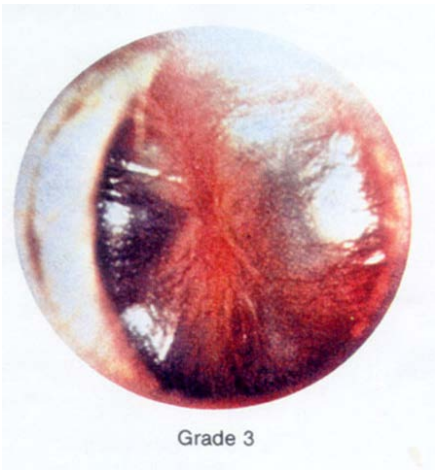
# MEBT

- Teed 2
  - Pressure > pain
  - Mild hemorrhage within TM
  - Erythema extending to umbo
  - Resolves 48-72hrs
  - Decongestants recommended



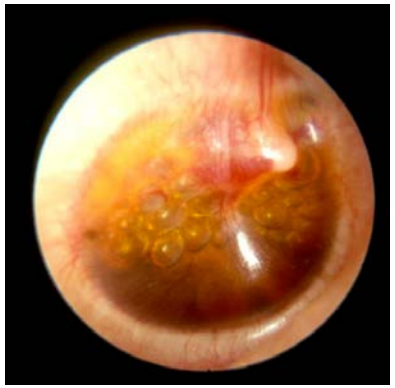
- Teed 3
  - Pain & pressure
  - More extensive hemorrhage within TM
  - Resolves 4-5 days
  - Decongestants recommended

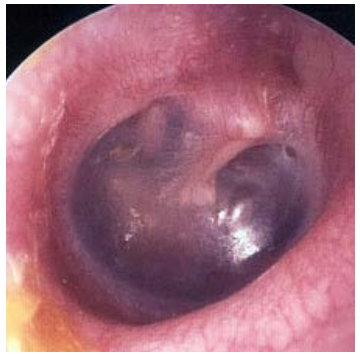




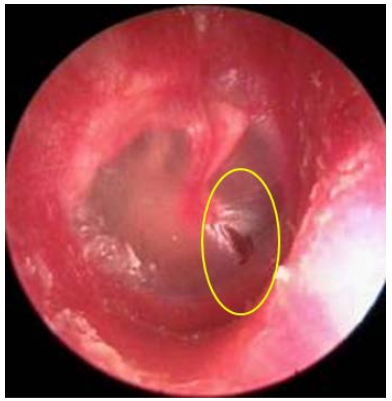
# MEBT

- Teed 4
  - Prominent pain
  - Blood behind TM, TM bulge
  - Resolves 5-14d
  - Decongestants recommended
  - +/- Abx if secondary infection
  - Consider myringotomy if not resolved at 7d



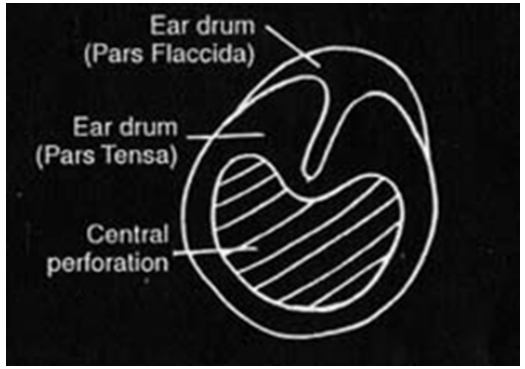


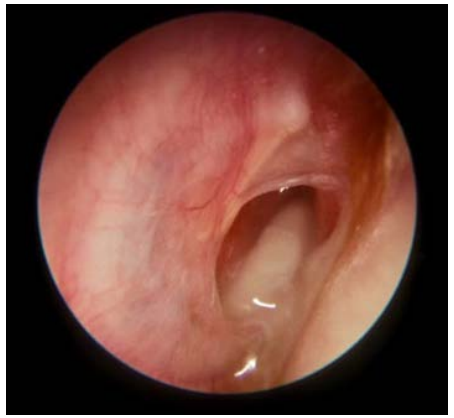
- Teed 5
  - TM rupture
  - Often initial relief of pain, resumes several hrs later
  - +/- acute dizzy/vertigo
  - Usually diminished hearing
  - Avoid diving until TM healed (2-6 weeks)
  - Abx drops (Cipro, Floxin) vs PO vs observation
  - ?serial audiograms
  - ENT referral if fails to heal
    - 90% heal within 90d



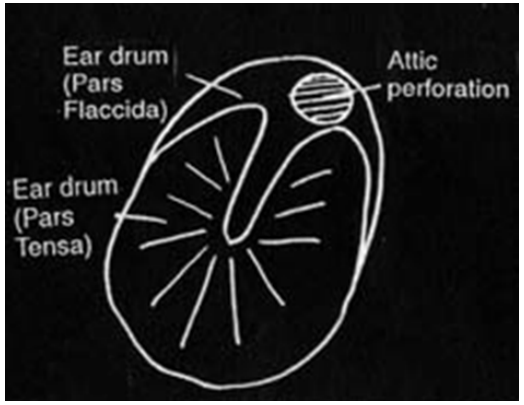
# TM Perforation

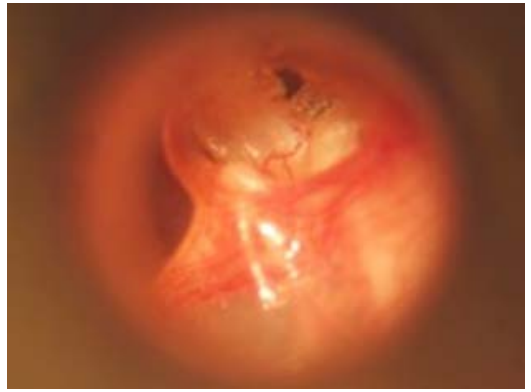
- Central perf =‘good’ perf

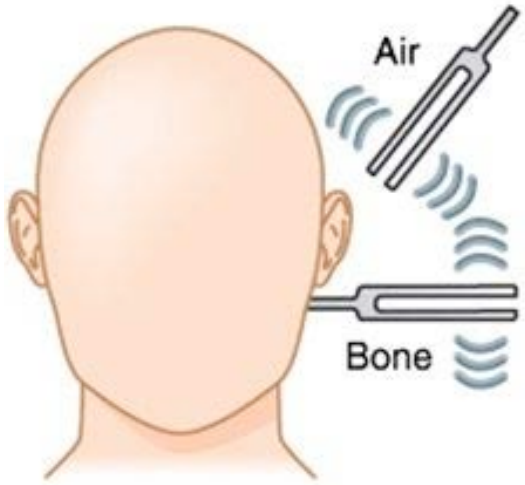


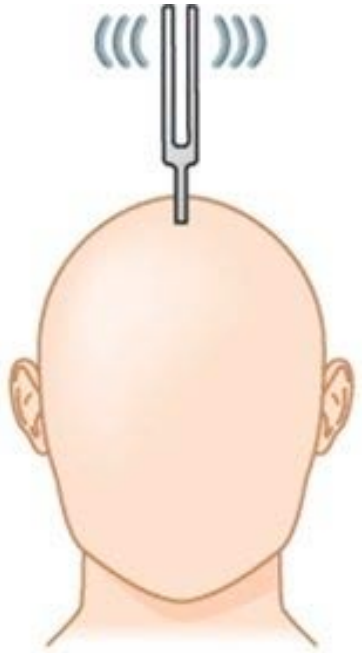
- Attic Perf = ‘bad’ perf





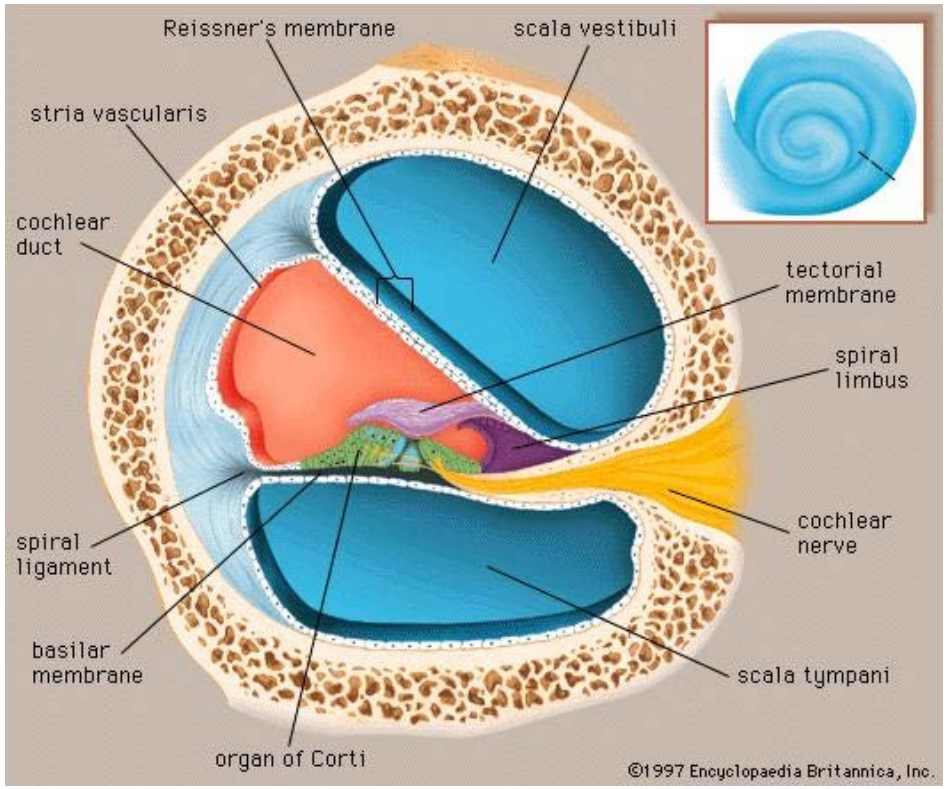

Rinne test



Weber test

| Hearing loss | Rinne test (Conduction) | Weber test (Localization) |
|---|---|---|
| None | Air > bone | Midline |
| Sensorineural | Air > bone | Normal ear |
| Conductive | Bone > air | Affected ear |

# IEBT




- May present
  - At time of forced equalization
  - At depth or immediately post dive with exertion (lift gear)
  - During otherwise normal ascent
  - Days later (usually hx of strain)
- Pathophys theories (likely combo)
  - Perilymphatic fistula (explosive or implosive force on windows 2° to press wave generated by TM or CSF)
  - Intralabyrinthine membrane tear
  - IE hemorrhage & gas
- RFs
  - URI or active allergy, hx of difficulty equalizing or poor equalizing technique (too late, too hard)
  - Forced Valsalva on ascent = sudden equal & implosion of stapes -> round window explosion
  - Wave trauma
  - Removing wetsuit hood
  - Heavy lifting (during, post dive)
  - Enlarged aqueducts (bigger wave)
  - Weakness of annular ligament of stapes

# IEBT

- S/Sx
  - Constant disequilibrium, loss of balance, ataxia, positional vertigo, nystagmus, N/V
  - Subjective ear fullness, high pitched tinnitus
  - Hearing loss of various degrees (progressive, fluctuating or positional)
  - Divers tend to have vertigo > SNHL compared to other causes IEBT
  - Acoustiphobia
- Initial Exam
  - Otoscopy: N, +/- MEBT
  - Neuro exam
  - Hennebert (cough, sneeze, Valsalva) & Tullio (noise)
  - Serial audiometry
    - Gen global (conductive) or HF SN loss
    - Positional – hearing gain of >10dB when supine with affected ear turned up
  - Special Investigations
    - VNG/VEMP – may show vestibular dysfx
    - ABR – differentiates central from peripheral
    - HRCT of temporal bones – consider for divers without other defined RFs
- Tx – involve ENT!
  - Bed rest with head elevated 30° until 7days post plateau of sx (~1-2 weeks)
  - Avoid cough, sneeze, Valsalva, strain at stool, sex, air travel, loud noises
  - Anti-nauseants, decongestants, sedative, laxatives – OK
  - Steroids – no evidence for IEBT, but make sure to consider SSNHL when HL is the only s/sx
  - NSAIDS CONTRAINDICATED
- Surgery – tympanostomy and window graft
  - Severe sx (repair within 48hrs), no improvement @ 7-10d, serial audiometry shows deterioration, co-existing TM rupture
  - ~90% successful vestibular sx, improve vertigo & tinnitus >HL (10% recurrence post surgery)
- RTD may be ok - CAF case-by-case
  - No non-compensated vestibular sx
  - HL stable, narrow, doesn't affect speech band
  - No anatomical risk factors, no issues equalizing
  - 6-12 weeks post injury (min no diving x 6 weeks post sx resolution or sx plateau)

# IE DCS

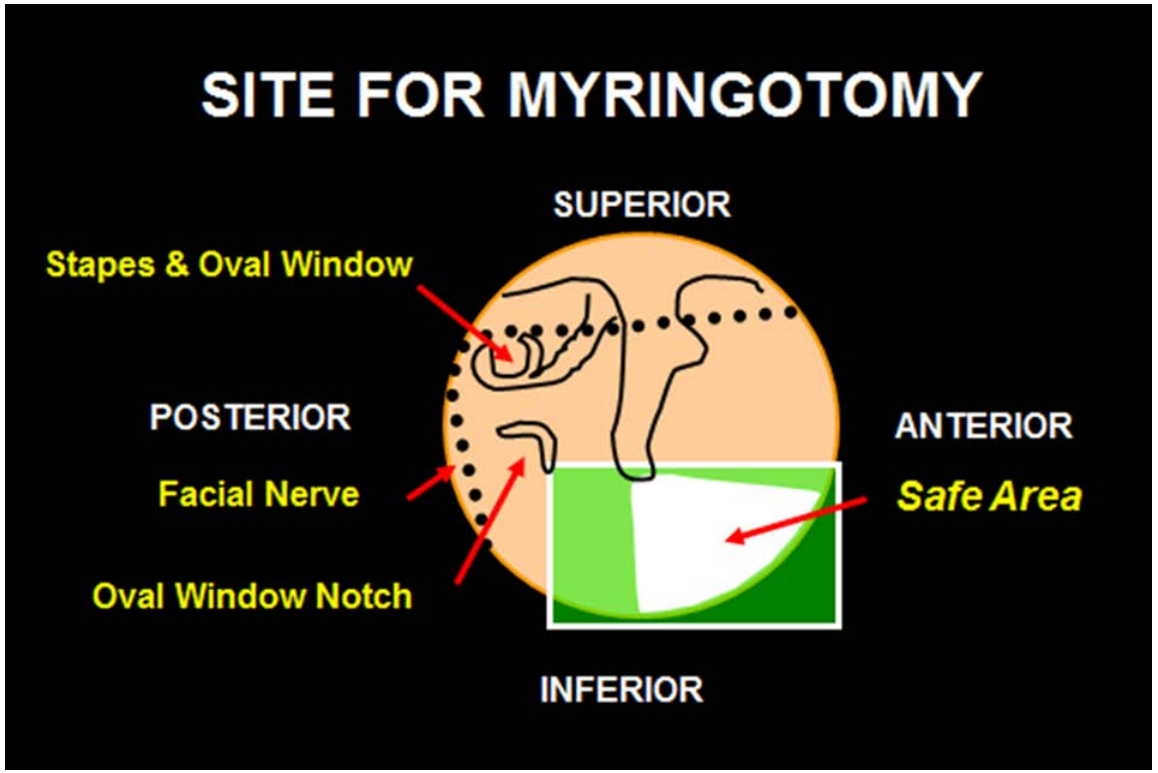
- Typically deep and deco dives, deco violation, mixed gas
- Pathophys theories:
  - Bubbles in osteoclasts – rupture bone lining otic spaces
  - Inert gas into perilymph from both blood and diffusion from ME via windows = supersaturation
  - Vascular emboli
  - Autochtonous bubbles arise in organs of inner ear
  - **Counterdiffusion -> total inert gas supersaturation
  - Gas switch from He to air – N2 diffuses in vascular space from blood, He from ME
- S/Sx
  - Usual onset near surface, on deco stop, or v. shortly after surfacing
  - Vertigo & SNHL > tinnitus
  - N/V, staggers, nystagmus
  - +/- presence of other DCS sx (<50%)
  - Usually MEBT absent
  - Neg Dix-Hall-Pike, Henneberts, Tullio
- Tx
  - Rxn reasonably well to early RCC (TT6)
  - High rate of residual sx if Tx delayed
- If unsure IE DCS vs. IEBT vs both:
  - Myringotomy then RCC, slow ascent
  - Some evidence ok to compress without myringotomy if no prob equalizing

# IEBT vs. IEDCS

| **IEBT** | **IE DCS** |
|---|---|
| More common | More rare |
| Typically during descent (+/- delay) | Typically ascent (+/- delay) |
| Hx of trouble equalizing, shallow dive, pain<br>+/- HL, tinnitus, N/V | Hx – painless, no issues equalizing, typically deep/mixed gas (Heliox) dive<br>+/- HL, tinnitus, N/V |
| Signs of MEBT, abnormal TM/perf<br>Hennebert/Tullio | No external signs |
| Tx<br>-No RCC<br>-Head elevated, avoid ↑CSF, steroids | Tx<br>-Urgent RCC<br>-?steroids |

- Delay situation = Valsalva after surfacing (ie. carrying tanks, lifting weights)
- When in doubt - myringotomy & recompress

# Myringotomy




- Myringotomy
  - Don’t do it for the first time on your own, with a diver requiring immediate RCC
  - Procedural otoscope, 22G spinal needle
  - Anesthesia
    - 10 drops of 8% tetracaine base in 70% isopropyl alcohol applied to TM for 15 mins or 1cc of 5% EMLA applied to TM for 60 mins
    - Then dab incision site with 20-25% phenol

# Alternobaric Vertigo

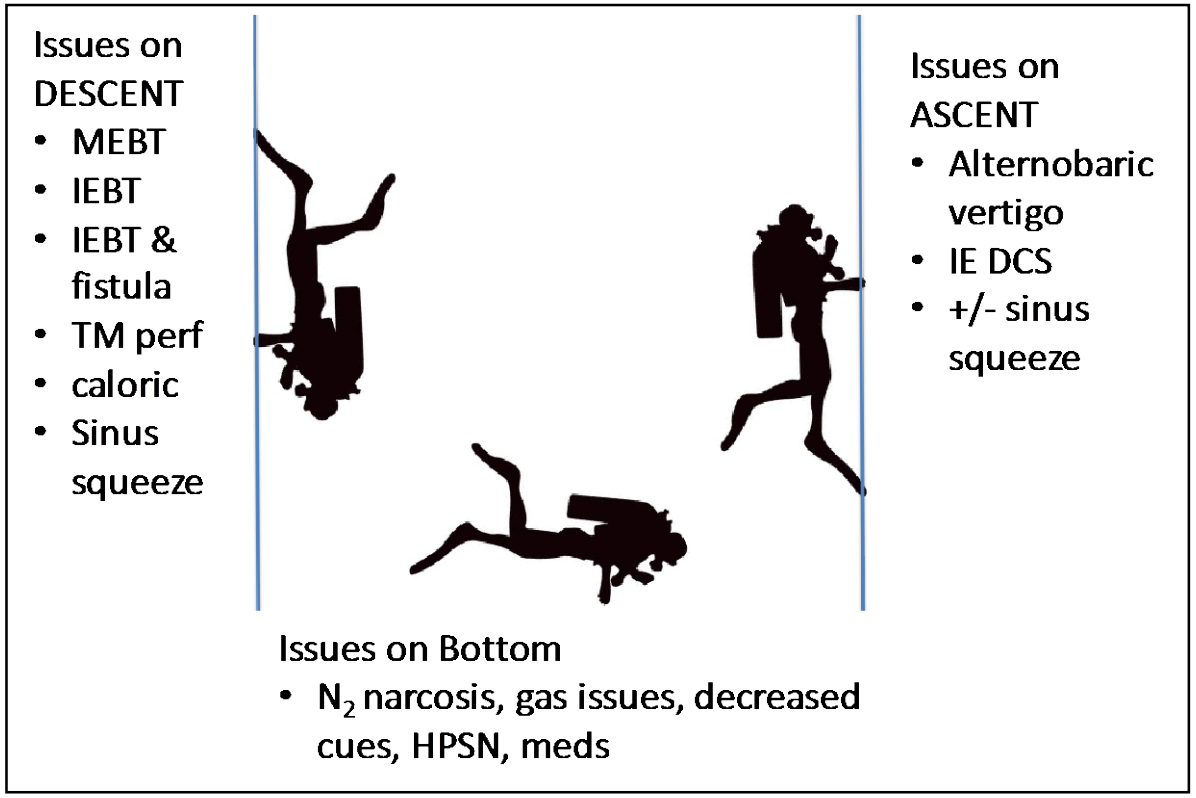
- Up to 25% divers, females 4x> males
- Due to press diff between two ME spaces
  - Δ0.6msw sufficient
- Rotational vertigo, nausea, nystagmus
  - Spinning toward side with related ETD dysfx
  - Resolves in mins(usually) to hours (v. unusual)
  - No HL or tinnitus
- Prevention:
  - Don’t dive with sticky ears
  - Descend little bit until sx resolve then ascend slowly
  - Toynbee or Frenzel, NOT Valsalva
- Recurrence may be indication to discontinue diving – case-based

# Transient Caloric Stim

- Unequal vestibular caloric stimulation
  - Cold water enters one ear
  - Esp. if horizontal canal is in vertical position – supine with head elevated 30° /prone head depressed 30°
  - Can also occur if TM perf with MEBT
- Common RFs
  - Obstruction one canal (cerumen, FB, exostoses, OE, ear plus, diving hoods)
  - TM perf

# ”Dizzy” Diver DDx

- Disorientation
  - Impaired vision/proprioception, N2 narcosis, hyper/hypocapnia, hyper/hypoxia, gas contaminants (CO), HPNS
- Diving Causes Vertigo
  - Caloric (poor fitting hood, unilat canal obstruction, TM perf)
  - Positional (prone with head down)
  - Pressure Δ (ABV)
  - MEBT, IEBT, IEDCS
  - HPNS
- Non-Diving Causes
  - BPV, Meniere's, vestibular neuronitis, labrynthitis
  - Acoustic neuroma, MS, migraine
  - AOM
  - Motion sickness
  - Meds
  - Factitious



# Facial Baroparesis

- Facial n. runs through facial canal along walls of middle ear cavities
  - Bony canal separates n. from middle ear space
- ~50% of people have “dehiscences” where nerve is covered by soft tissue/mucosa rather than bone
  - PΔ of only 0.8msw can cause relative ischemia of n.
  - Compression lasting >3.5 hrs can lead to permanent damage
- S/Sx
  - Setting of difficult equalization
  - LMB facial palsy, always unilateral
  - Onset shortly after surfacing
  - s/sx of MEBT usually evident
  - +/- coexist with IEBT
- Tx
  - 100% O2
  - Toynbee or Frenzel, no Valsalva
  - Decongestants
  - Dive (or RCC) to 1-2msw on $O_2$, slow ascent
    - Some authorities opine that HBOT at >10msw may be detrimental
  - Myringotomy – rarely if ever required
  - No evidence to support corticosteroid
  - Usually resolves within 1-2 hrs after equalization (unless compression >3,5hrs)
- DDx
  - DCS – provocative profile, no signs of MEBT, +/- other signs of DCS
    - Never reported as isolated DCS finding
  - CAGE/Stroke – UMN vs LMB
    - Able to frown and close eye normally = UMN

# Systems Focus: Cardiology

# CVS & Diving

- ↑cardiac demand
  - Heat generation, exercise, vascular redistribution due to immersion
- Cold/stress can induce coronary spasm
- CVS D/O in diving:
  - Sudden death – underwater usually due to CVD, arrhythmia
  - Acute MI
  - Stroke
  - Syncope
  - Arrhythmias
  - Pulmonary edema
  - Paroxysmal dyspnea
  - Vascular rupture/occlusion
  - Gas embolism

- CAF RF Screening
  - Hx – CVD, smoker, DM, lipids, FHx, HTN, exercise tolerance
  - Anthropomorphics
  - Labs – lipids, fasting gluc, A1C > FRS
  - ECG q4yrs to 40, then q2yrs >40 y.o.
  - +/- EST, stress echo, nuclear perfusion, CT Coronary Ca if indicated
- Why screen Asx?
  - Most ACS due to plaque rupture, often without flow limitation pre-ACS event
  - Up to 50% initial presentation CVD is sudden cardiac death

# CHD

- Septal defects
  - VSD most common upper septum
  - Small deficits gen no significant R to L shunt (unlikely to ↑risk AGE)
- Aortic stenosis
  - Exercise syncope, sudden death
- Patent ductus arteriosus
  - HF if severe
- Cardiomyopathy
  - Hypertrophic often asx, may hear murmur on exam if valve abN
  - Dilated – ↓LV fx may = exercise intolerance
  - * Any FHx of sudden death needs investigation to r/o cardiomyopathy!
  - CI's to diving
    - AbN LV Fx – EF <50% (risk arrhythmia)
    - Hx ICD, arrhythmias, syncope
    - Hx of HF (lose cardiac reserve to prevent syncope)
- PFO & Diving
  - Controversial- significant opinion (based on epi, animal models, path exam, pathophys reasoning) that shunt likely does not contribute to isolated spinal DCS
  - Relative risk DCS in significant shunt vs. no shunt ~5-25
    - Includes incidence of isolated spinal DCS, thus likely overestimate shunt-attributable risk
  - Consider underlying condition
  - CAF Screening – CDs, unexplained DCS/AGE hit
    - Echo with bubble contrast & Valsalva

# Valvular Disease

- Stenotic valves
  - ↓exercise tolerance (worse with immersion)
  - Lead to circulatory obstruction
- Regurgitant valves
  - Tolerated if mild
  - HF, pulm congestion if severe -> ↑dyspnea (exercise, immersion)
- Volume overload -> hypertrophy
  - ↑muscle mass demands requires ↑blood flow (risk underperfusion of endocardium)
- Bicuspid aorta (*requires cardiology W/U)
  - Assoc with abnormal coronary arteries, aortic root dilation, PDA, Turner syndrome, aortic stenosis, aortic insufficiency
- Aortic stenosis – exercise syncope, sudden death
  - Low risk if no LVH on ECG, HF, arrhythmias, syncope or angina
- Aortic insufficiency – HF if severe
- Mitral stenosis – exercise induced pulmonary edema
- Mitral insufficiency – HF if severe
- Mitral prolapse - +/- arrhythmia
  - Assoc with palpitations, tachy, extra beats, CP, regurg
- Pulmonic stenosis – reduced exercise tolerance if severe
- Tricuspid stenosis – reduced exercise tolerance if severe
- Prosthetic valves
  - What is fx status
  - ?anticoagulation required (increase hemorr risk in BT)
  - Likely low stress warm water diving only

# Arrhythmias

- Supraventricular
  - Episodic SVT, Afib can be N variant in young population
  - R/O Mitral sten, TSH, HTN, nicotine, stress, EtOH, caffeine, supplements etc.
    - Gen ok to dive if no organic heart disease, resolves with stim remove
    - CDSM review if requires BB/CCB
- Ventricular
  - PVC may be N variant – assess with exercise
  - No go diving if multifocal, R-on-T, freq coupling, ICD, LV dysfx (increase risk sudden death)
- Long-QT syndrome
  - QT > 440 msec
  - Assoc with sudden death/VF, syncope
    - Ppt by stress, exercise, lyte abN, drugs, meds etc.
    - Increase risk combo of exercise & immersion
  - Unfit dive
- Increased vagal tone
  - Bradycardia may be N variant in well conditioned applicants
  - Fit dive if asx
  - If palpitations (Afib), severe brady while diving – can increase risk syncope

# Conduction Abnormalities

- Often underlying cardiac disease
- 1° AV
  - May be due to excess vagal tone
  - Asx generally ok
- Fixed 2° AV block
  - Often lead to complete block = CI
- LBBB
  - Often due to cardiomyopathy or coronary dz
  - Requires workup
- RBBB
  - Incomplete
    - If stable, usually N variant, benign
  - Complete
    - May be N variant or congenital HD
    - Requires workup to r/o anatomic cardiac abnormality
- Pacemaker
  - If no other heart disease, pacemaker tested to pressure and good exercise tolerance
    - May be ok for sport diving
    - Unfit CF diving
  - ICD – unfit diving

# Conduction Abnormalities

- Brugada Syndrome
  - Mutation Na channel
  - Type 1 – ST elevation >2mm in >1 of V1-V3 followed by a negative T Wave
  - Type 2 - >2mm saddleback shaped ST elevation
  - Type 3 – morphology of Type1/2 but <2mm
  - Accompanied by documented VF/VT, FHx of sudden cardiac death <45, similar ECG in family mbrs, syncope, nocturnal agonal respiration, inducibility of VT with electrical stimulation
  - Tx = ICD, CI to diving
- Pre-excitation syndromes
  - Short PR not in itself CI
    - Asx, low risk arrhythmia may be ok sport diving
    - Recurrent paroxysmal or exercise-induced tachy requires w/u
  - WPW
    - Risk exercise induced tachy, palpitations, SOB, syncope, sudden cardiac death
    - If successful ablation – ok to dive
    - If sx – Unfit Diving

# Long-term Effects of Diving

# Noise-Induced Hearing Loss

- Risk Factors (RF)
  - Noise in environment – topside
    - Ship/boat noise, equipment/tools
    - Gas into chamber
  - Noise in environment – underwater
    - No hearing protection
    - Helmet, reg, ship/boat noise, tools, underwater explosions
  - Barotrauma
  - Inner ear DCS
- ↓Sound localization under water
  - ↑sound velocity in water vs air = reaches both ears at same time
- CAF screening
  - Audiogram qYear & on clinical indication
  - Occupational med (effect of diving on person) – audiogram
  - Fitness to dive considerations (whisper test, word discrimination, audiology)
- New studies show no difference between divers and those exposed to similar levels of surface noise

# DON

- Type of avascular necrosis due to hyperbaric exposures; in long bones with fatty marrow
  - Relatively ↓blood flow = prolonged wash-out
  - $N_2$ is 5x more soluble in marrow fat than in blood
- Location: prox humerus, prox/distal femur, prox tibia
  - "wet" (divers) – femur, shoulders, rarely hip
  - "dry" (caisson) – hip most common
- Juxta-articular (A) or shaft (B) lesions
  - A lesions often become sx – pain, ↓ROM
  - B lesions often remain asx
- RF
  - Depth exposures >30msw, or >4hrs at <30msw, hx of DCS (any depth), significant omitted deco
  - Submariner escape – DON risk after single provocative deco exposure even if no DCS
  - >40, obesity, EtOH, fatty liver, dyslipidemia
  - Hx of DON
- Dx based on imaging studies
  - Long-Bone Survey (LBS)
    - B/L upper humeri (including rotational view of shoulder), AP & lat knees (upper tibia & lower femur) & b/l hip
    - Xray changes may not be observed for months/years after lesions devo
  - MRI preferred – more sensitive & specific
    - Able to detect DON as early as 2 weeks post onset, and by 2 months virtually all lesions are demonstrable
  - Bone scan
    - High sens but low specificity, not diagnostic
- Prevention
  - Timely Tx of DCS with HBOT likely to reduce incidence of DON
  - Avoid RFs

# DON Classification

UK MRC

- Juxta-articular A Lesions:
  - A1 – Dense areas with intact articular cortex
  - A2 – Spherical opacities
  - A3 – Linear opacities
  - A4 – Structural failures: translucent cortical bands, collapse of articular cortex, sequestration of cortex
  - A5 – Secondary degenerative arthritis
- Shaft B Lesions:
  - B1 – Dense areas
  - B2 – Irregular calcified areas
  - B3 – Translucent and cystic areas

- Ficat 0
  - Ischemia, intravascular coag
  - Asx, no radiographic changes
- Ficat 1
  - Dead bone without repair, asx
  - Xray N, MR shows marrow edema by ~ 4weeks
- Ficat 2
  - Dead bone with repair, no collapse, asx until late
  - XR: sclerosis with irreg margins, spherical opaque areas (“snowcap”) and linear opacities
  - MRI: rings of low intensity surrounding necrotic center
- Ficat 3
  - Dead bone with repair & collapse
  - Sx – pain with joint motion or weight-bearing
  - Xray: subchondral # - radiolucent “crescent” line
  - MRI: necrotic centre, collapse of articular surface
- Ficat 4
  - Secondary degenerative arthropathy
  - May progress to complete destruction of joint

# DON

- Screening
  - ALL divers complete DON questionnaire @ PHA
    - LBS only if at risk
  - Student divers
    - DWD candidates LBS at end of course
    - SWD only if Q shows ↑risk = LBS within 1 month completing course
  - Divers with ongoing exposure to DON RFs (all DWD, SWD with increased risk) = LBS q5yrs
  - Any diver with DCS, significant violation deco or sub escape = LBS within one week of event, followed by MR (hips, knees, shoulders) @ 2 months post-event
  - All DWD, and any SWD who have required LBS = LBS at termination of diving career
- Management
  - All DCS, significant omitted-D, sub escape, or DON lesions require CDSM review
  - If ID DON – R/O lesions at other sites
  - Juxta-articular A lesion
    - Shall be further characterized by MRI, staged
    - MRI of b/l shoulders, hip and knees
    - Followed by XRq6 months x 2 years, then annually until no further interval change
    - Referred to ortho
    - Declared “unfit CF diving or hyperbaric env”
    - Strongly advised to d/c civi diving >30msw, avoid load-bearing activities on involved joint
  - Shaft lesion
    - MR of b/l shoulders, hip and knees
    - LBS q5years until end of diving career, exit LBS
    - F/U required if dev sx
    - May be able to continue diving as per their quals vs. <30msw – requires CDSM review

# Aquatic Hazards

# Hazard Categories

- Bites
- Barbs and Venoms
- Toxins
- Infectious agents
- Contamination
- Biological contaminants
- Chemical contaminants
- Radiological contaminants

# Coelenterates (Jellyfish, sea anemones, fire coral…)

- Nematocysts
  - Vinegar ↓nematocyst discharge
- Box jellyfish/sea wasps
  - Multiple interlacing whiplash lines in beaded/ladder pattern -> wheal -> necrosis/ulceration ~7-10d
  - Excruciating pain
  - Death can be within 10mins, likely survival after 1st hr
    - HTN, then hypo/hypertensive oscillations, VF, resp distress, apnea, edema, cyanosis, paralysis
  - Tx
    - Vinegar, remove tentacles asap
    - IV morphine, IV steroids, anxiolytics, antivenom
- Irukandji syndrome
  - Can follow negligible sting from variety of jellyfish
  - Stinging -> papules, dyshidrotic, severe abdo pain, muscle cramps, hypertonicity, ↑HR, ↑BP, resp distress
- Sea Bather's eruptions
  - Thimble jellyfish larvae "sea lice" when nematocysts trapped under bathing suit/wetsuit discharge
  - Painful, erythematous, pruritic rash under dive suit
  - If freq exposure, can dev allergies to toxin
  - Tx = topical steroids, avoid trigger

# Stings/Spines/Bites

- General Tx: immerse in hot water
  - Up to 45°C, including N skin (as control)
- Stingray
  - Spine physical damage, envenomation
  - Tx
    - Supine, elevate limb, wash away venom
    - Gently remove spine
    - Hot water bath
    - Inject local anesthetic into wound
    - Monitor for 2° complications
- Sea Urchins
  - Spine causes physical damage, Ach-like venom
  - Tx
    - Remove spines vertically
    - Hot water bath
    - Local anaesthetic
- Stonefish
  - Myotoxin – blocks conduction skeletal/ cardiac/involuntary muscles, releases Ach
    - Immediate excruciating pain, edema, swelling, hot with numb centre & extremely tender surrounding
    - Cardiorespiratory collapse
  - Tx
    - Elevation, local anaesthetic, hot water bath
    - Antivenom if persistent pain
    - Debride necrotic tissue
- Sea Snake
  - 4 bite marks, venom blocks Ach
    - Euphoria, restless, thirst, dry throat, N/V, myalgias -> weakness -> paralysis (similar to Guillain-Barre), myoglobinuria
  - Tx
    - Pressure immobilization, antivenom (prophylactic epi/antihistamine), +/- ARF

# Medical Exam for Divers

# Diver PHA

- Purpose
  - Fit to dive, fit to work, med surveillance, assess impairment
- CAF SWD PHA (CFHS Instruction 4000-04)
  - Initial
  - Type I – P1, P2, 2452, 2552, DON screen, labs
  - Type II – P1, questionnaire review
  - P1 – anthropomorphics, vision, audio, 2452
  - P2 – full history and physical
- Initial Investigations
  - Blood – CBC, ‘lytes, cr, liver enzymes (AST, ALT, GGT, Alk Phos), lipids, FBS q2 years
  - Urine – dip and micro
  - CXR – PA insp/expiration, lat (3 views) q5 years
  - EKG – q4 yrs to 40yo, then q2 yrs
  - Spirometry screen
  - DON screening – annual Q’s; LBS if at-risk
  - CV risk assessment q4 to 40 then q2
- Physical Exam Highlights
  - HEENT – TM mobile with Valsalva, TM changes, TMJ ROM/crepitus
  - CVS
  - RESP
  - GI – scars, hernias, masses
  - MSK – document well any signs/symptoms, especially in the shoulders, elbows, hips and knees
  - Neuro – gait, cognition, CN, pronator drift, Romberg, sharpened Romberg, bulk/tone/power/sensory/DTR, peripheral vs.. dermatome nerve deficits, +/- SCAT

# Diver ROS

1. Asthma
   - Asthma after age 12, wheeze, SOB, ILD or COPD
   - Childhood Hx, ER presentations, hospitalizations
   - EIA, respiratory cold tolerance, bronchitis, pneumonia, cough (nocturnal, post URTI)
   - Puffer use
   - No go asthma = requires ongoing use of rescue inhalers, induced by cold/exercise/anxiety or exposure to airborne particulate/chemical/petroleum distillates found in the dive/sub atmosphere
2. Pneumothorax
   - Hx of spontaneous or traumatic, chest tube
3. Neuro
   - Seizure/epilepsy
   - Blackout/LOC
   - Migraines with deficits/impact on fx
4. MH conditions
   - Claustrophobia, anxiety, PTSD, phobias, substances (CAGE positive – context and consequences)
5. Significant Head trauma
   - TBI/concussion/LOC >2 mins, skull #, any ICH, post-traumatic amnesia >30 mins, focal neuro sx, post concussive epilepsy or post concussive sx lasting >24 hrs
   - For concussion/head injury
     - Total # of concussion or head injuries
     - Date last HI, interval between concussions/HIs
     - Change in threshold of trauma required to induce concussion/HI sx
   - Previous assessment by neurologist/specialist
   - Previous imaging results
6. ENT
   - Sinus problems, nasal polyps, deviated nasal septum, sinusitis
   - ETD problems clearing ears under pressure, flying
   - Chronic OM/OE, TM perforation
   - Vertigo, Meniere's
7. Hx of DI (DCS or AGE), DON, barotraumas

# Drugs & Diving

# Considerations

- Considerations:
  - Underlying disorder
  - Is drug inherently safe for diving?
  - What type of diving planned?
    - Will performance be impaired – unsuccessful mission
    - Proximity to medical care
  - Incapacitation – diver now a threat to themselves or safety to others
  - Change in drug's efficacy/safety profile due to hyperbaric env
- "Bad" Drugs
  - Affect alert/LOC/cognitive fx, exercise tolerance, change sz threshold
  - I.e. psychotropics, sedatives, BB etc.
- "Bad" Diseases
  - Affect alert/LOC
  - ↓exercise tolerance/general fx, ↓press equalization, ↑risk DCS
  - I.e. psychiatric, neuro, cardiac, resp, MSK, metabolic, ENT considerations
- "Bad" Dives
  - ↑exertion, exposure (hot/cold), elaborate equipment (rebreathers), dangerous env (overhead, combat, EOD, extreme depth/duration/deco), distance from med resources

# Initial Approach to Dive Injury

# 1st Principles

- DCI
  - Trapped gas -> POS, AGE
  - Evolved gas -> DCS
- Not all diving casualties are dysbaric
  - Thermal (hyper/hypothermia)
  - Near drowning, IPE
  - Environment (blast injuries, tools, mechanical dangers, marine life)
  - Previous med conditions (MI, Sz)
- Not all dysbaric casualties have DCS
  - POS, AGE
  - Other barotraumas
- Dead/VSA
  - At surface vs. depth
  - Witnessed/unwitnessed
  - AGE, MI, drowning due to blackout/medical condition
- Blackout
  - Gas issue (too much, not enough, wrong gas), seizure
- Vertigo and other ENT symptoms
  - Vestibular DCS, inner ear BT (pure or combined with vestib DCS), alternobaric vertigo, Meniere's/BPPV
- Chest pain/SOB
  - Chokes,  pneumothorax, immersion pulm edema, ACS/CHF
- Headache
  - CO2 toxicity, DCS (?type?), migraine, plain H/A

# Initial Approach

- Approach
  - Onset
  - Evolution
  - Presentation
  - Gas burden – dive profile
  - Evidence of barotrauma
- Mod – High risk = Manage as DCI
- Low Risk – consider other causes
  1) Non-DCI dysbaric (i.e. BT)
  2) Non-dysbaric diving-related (i.e. marine life)
  3) Non-diving-related CLASSICAL (ie. MI)

- Diver Hx
  - Dive Profile
    - Depth, time - estimate of N2 stress
    - ?repet dives – how many, when was last dive
  - Dive equipment & gas used
  - Mechanism of injury
  - When did problems start
    - Descent, at depth, ascent, at surface
  - What happened during dive
    - Uneventful; strong current; aborted; emergency ascent

# Initial Actions Dysbaric Casualty

- **ABCDE**
- Vitals
- 100% Oxygen
  - Accelerate inert gas elimination & improve $O_2$ delivery to hypoxic tissue
  - BMV, demand valve, rebreather
- IV Fluids – IL bolus, then titrate (~100-175ml/hr)
  - Avoid dextrose (can increase ICP)
  - PO fluids unlikely to benefit
  - Increase tissue perfusion = increase inert gas washout
- DDx
- Emergency (Red) – immediate, life-saving
  - Severe sx, rapid onset <1hr, progressive, neuro deficits, clearly ill
- Urgent (Yellow) – disability preventing
  - Pain only, stable or progressing, overall stable, without sx of severe DCS
- Timely (Green) – elective
  - Unclear/ambiguous sx, long delay from when sx 1$^{st}$ appeared (not when 1$^{st}$ reported)

# Time to Tx – DAN workshop 2004

- Consensus Statements
  1. Mild s/sx = limb pain, constitutional sx, some cutaneous sensory changes, rash; where these manifestations are static or remitting, and associated objective neuro dysfx has been excluded by medical exam
  2. Untreated mild s/sx due to DCI are unlikely to progress after 24hrs from end of diving (unless further deco or ascent to altitude)
  3. Level B epi evidence indicates that a delay prior to recompression for a patient with mild DCI is unlikely to be associated with any worsening of long-term outcome
  4. Some patients with mild s/sx after diving can be treated adequately without recompression. For those with DCI recovery may be slower in the absence of recompression.
  5. Some divers with mild s/sx after diving may be evacuated by commercial airliner to obtain treatment after a surface interval of at least 24hrs, and this is unlikely to be associated with worsening outcomes

- NB Mild s/sx – need monitoring

# General Approach RTD

- Impairment
  - Loss of use or derangement of body part/organ system or fx
- Disability
  - Inability to meet demand/need based on an impairment
- 5 key questions:
  - Diver or patient?
  - Disqualifying meds
    - Consider both med & indication
  - Ability to do job
    - Exercise tolerance, cognitive fx, roles (buddy, rescue diver, dive sup, boat driver, EOD disposal), stressors (pressure, gas toxicities, thermal, disorientation etc.)
    - Quantification of impairment
  - Immune function
  - Assessment of job - ?tailored diving

- Key Considerations:
  - Dive safety (individual & team)
  - Job/mission completion
  - Conserve trained resources
- CAF RTD after DCI
  - Type I DCS completely resolved
    - No fly 3d, no diving 7d
  - Type II DCS completely resolved after 1 TT
    - No flying 7d, no diving 30d
  - Type II DCS residual sx or repeated tx
    - No fly 10d, no diving until reviewed by CDSM
  - AGE completely resolved after 1 TT
    - No fly x 7d, no diving until reviewed by CDSM
  - AGE residual sx or repeated tx
    - No fly x 10d, no diving until reviewed by CDSM

# HBOT

# HBOT

- UHMS Indications
  1. Air or Gas Embolism
  2. CO poisoning
  3. Clostridial myositis and myonecrosis (Gas Gangrene)
  4. Crush injury, compartment syndrome, other acute traumatic ischemias
  5. DCS
  6. Arterial insufficiencies
  7. Severe anemia
  8. Intracranial abscess
  9. Necrotizing soft tissue infections
  10. Osteomyelitis (refractory)
  11. Delayed radiation injury (soft tissue, bony necrosis)
  12. Compromised grafts and flaps
  13. Acute thermal burn injury
  14. Idiopathic sudden sensorineural hearing loss (new Oct 2011)

- CAF Treatment Tables
  - Limits due to OxTox (decreased with air breaks)
  - If TT stopped due to CNS OxTox, allow 15 mins after sx resolved, then resume schedule at point of interruption
  - Tx repeated until resolution or plateau
  - Contraindications – almost all relative (depends on indication)
    - Largely same as diving
  - Other common treatment tables:
    - USN, Comex, Catalina, CO poisoning
    - Proprietary commercial tables

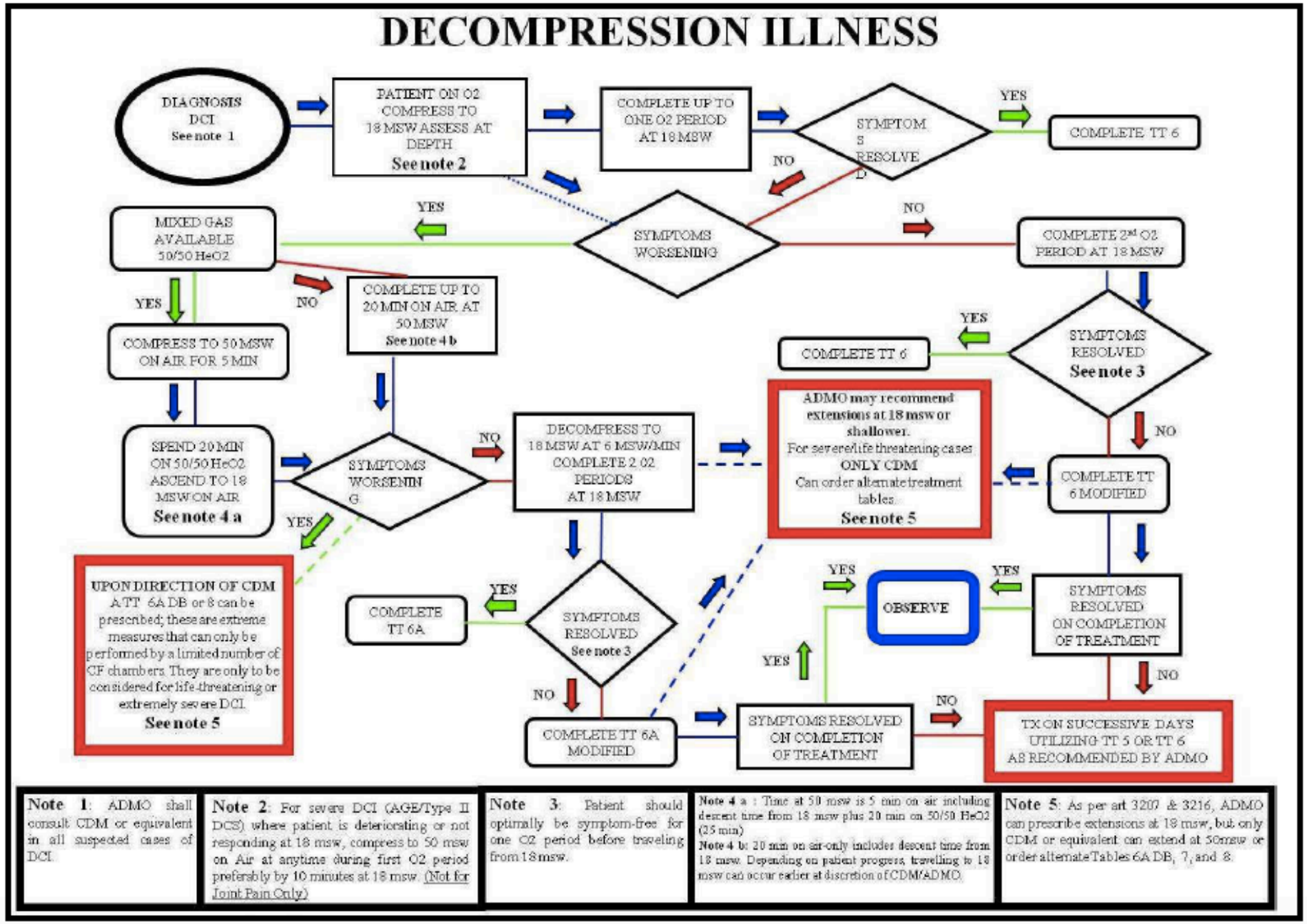
# HBOT Mechanisms

- Bubble Compression (Boyle's Law)
- Hyperoxygenation (Henry's Law)
  - 10-15x increase in plasma O2
  - 2-4 x increase in O2 diffusion capacity from capillaries
- Gas Gradient – support gas washout, prevent additional uptake of inert gas during HBOT
- Antimicrobial effect
  - Inhibit clostridial alpha toxin
  - Anaerobic bacteriostasis
  - enhance antibiotic activity,
  - improve PMN fx
- Blunt ischemia-reperfusion injury
  - Attenuates PMB-endothelial interaction
  - Prevent lipid peroxidation
- Decrease edema
  - Vasoconstriction (while tissues remain hyper-oxygenated)
- Angiogenesis/Wound healing
  - Stimulate vasculogenic stem cell mobilization
  - Increase growth factor synthesis
  - Stim fibroblast proliferation
  - Angiogenesis, reversal of tissue hypoxia

# Adjuncts

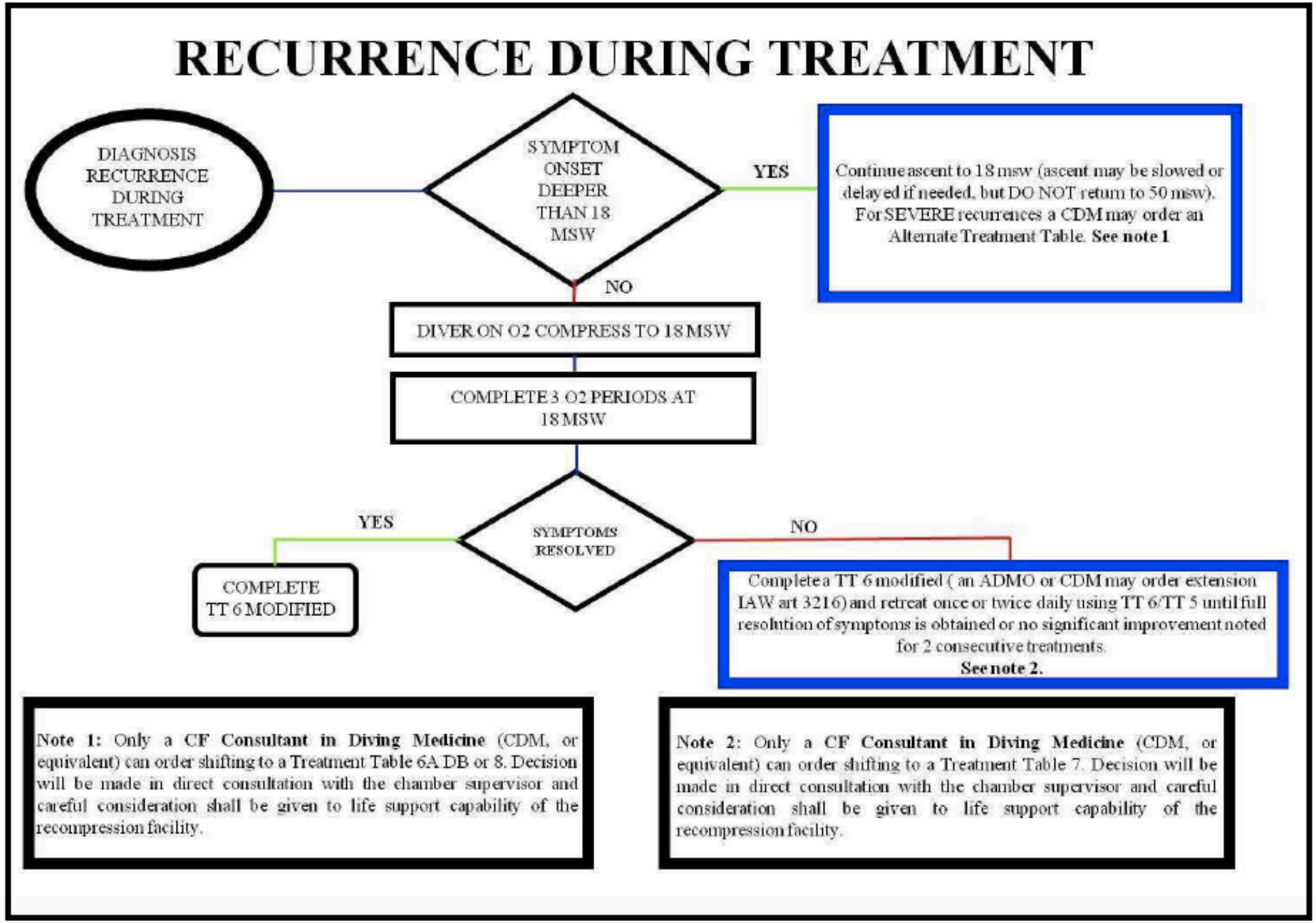
- NSAIDs
  - No diff in final outcome, but with tenoxicam divers needed fewer TT
  - NNT ~4-5
- Heliox
  - May decrease # of TT required
- Lidocaine may have neuroprotective effect
  - Insufficient evidence to support routine use
- Perfluorocarbons increased $O_2$ delivery, $N_2$ removal
  - Animal models only so far
- Not recommended: steroid, ASA, Heparin (unless DVT prophylaxis @24hrs)
- Other considerations
  - Hydration, food
  - Urinary catheter
  - DVT prophylaxis
  - Hospital admission
  - Specialists consults (i.e. neuro)

Figure 3-2-2 Decompression Illness



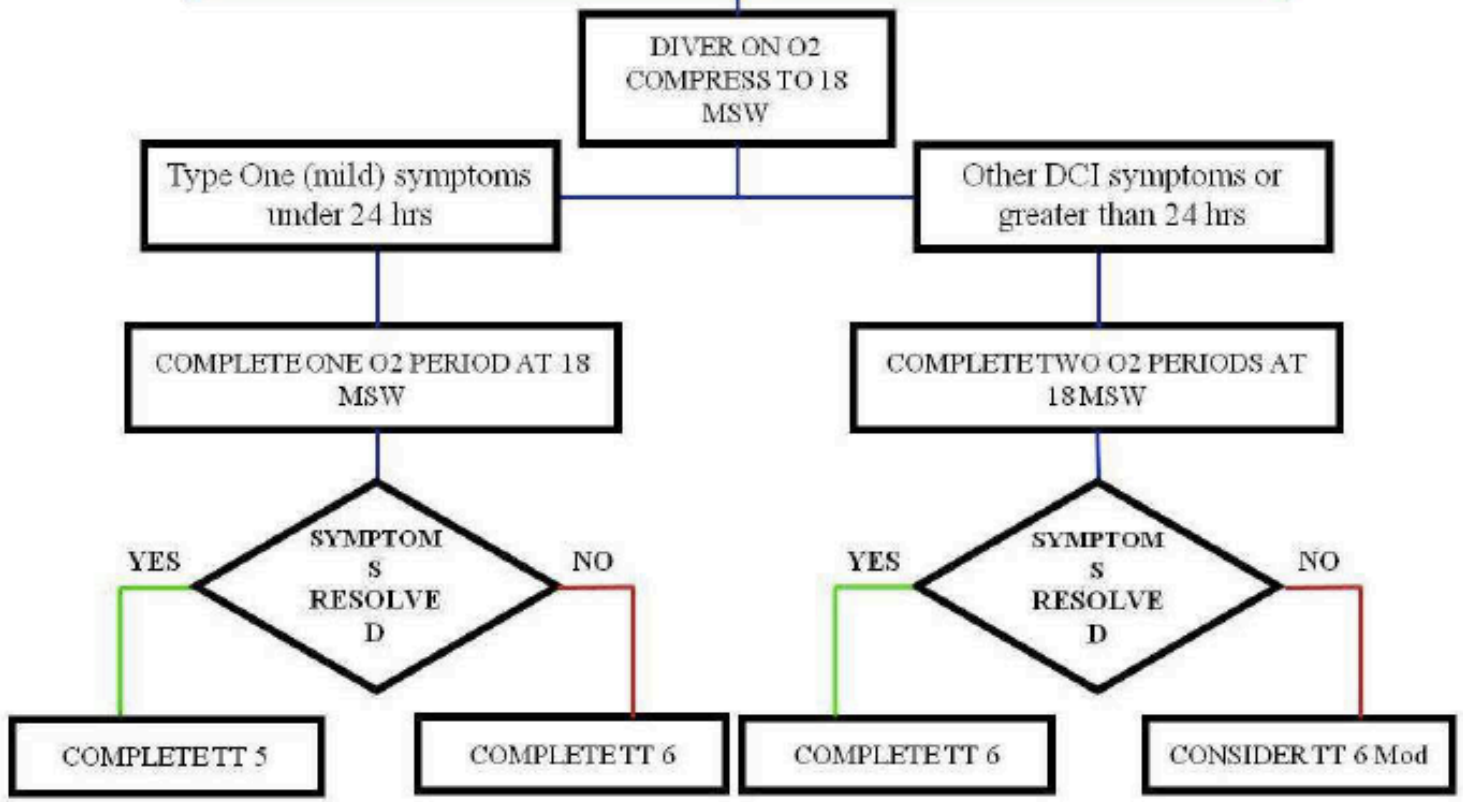

B-GG-380-000/FP-005

Figure 3-2-3 Recurrence During Treatment Summary



# RECURRENCE AFTER TREATMENT

**Supervisor/ADMO can initiate re-treatment.**
Any symptom that occurs within 7 days after a treatment for DCI must be regarded as a possible recurrence.
In all cases a CDM shall be consulted and decision not to re-treat shall only be made by a CDM or equivalent.



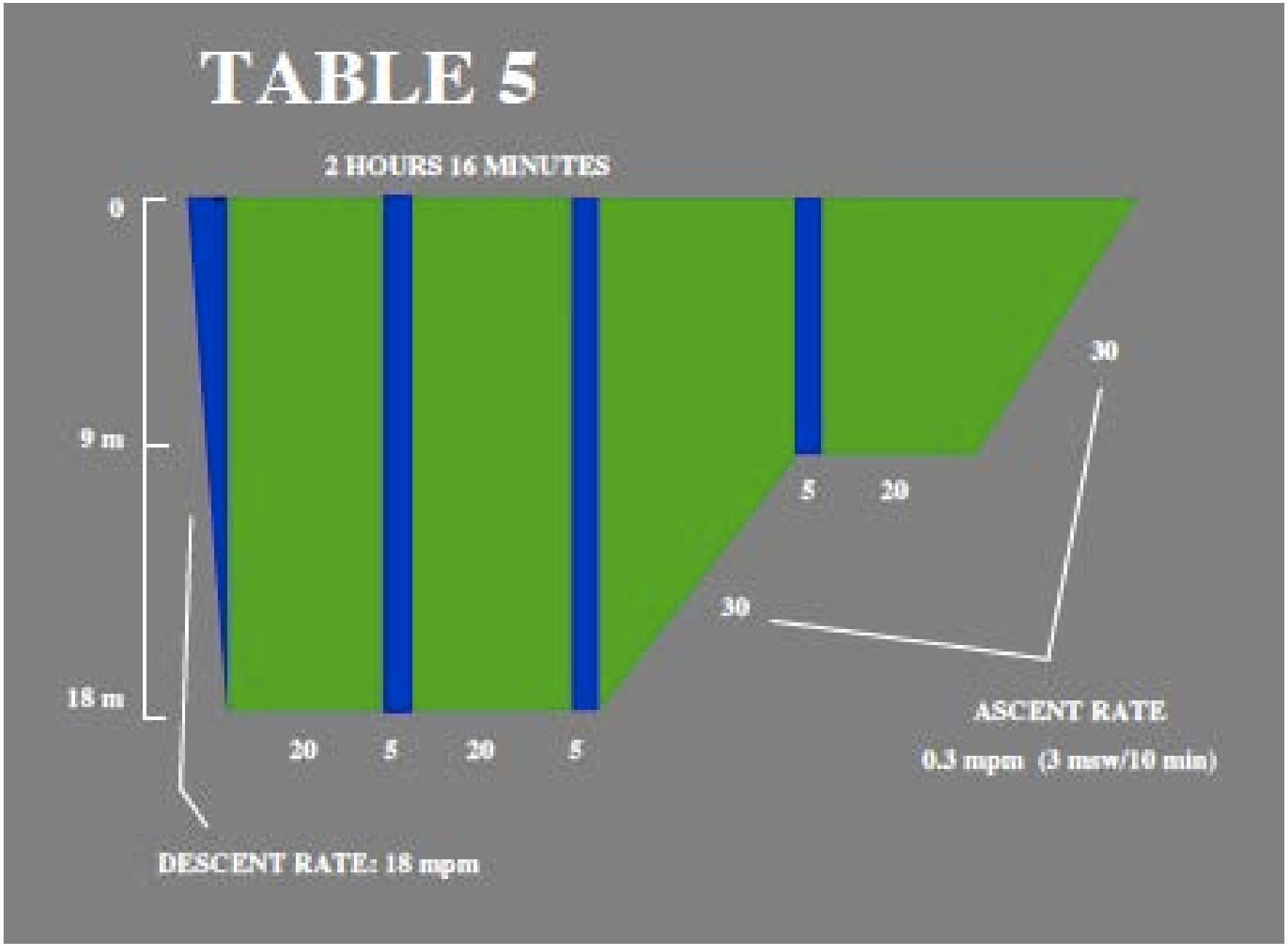

NOTE: Retreat once or twice daily using TT 6/TT 5 until full resolution of symptoms is obtained or no significant improvement is noted for 2 consecutive treatments.

Figure 3-2-4 Recurrence After Treatment

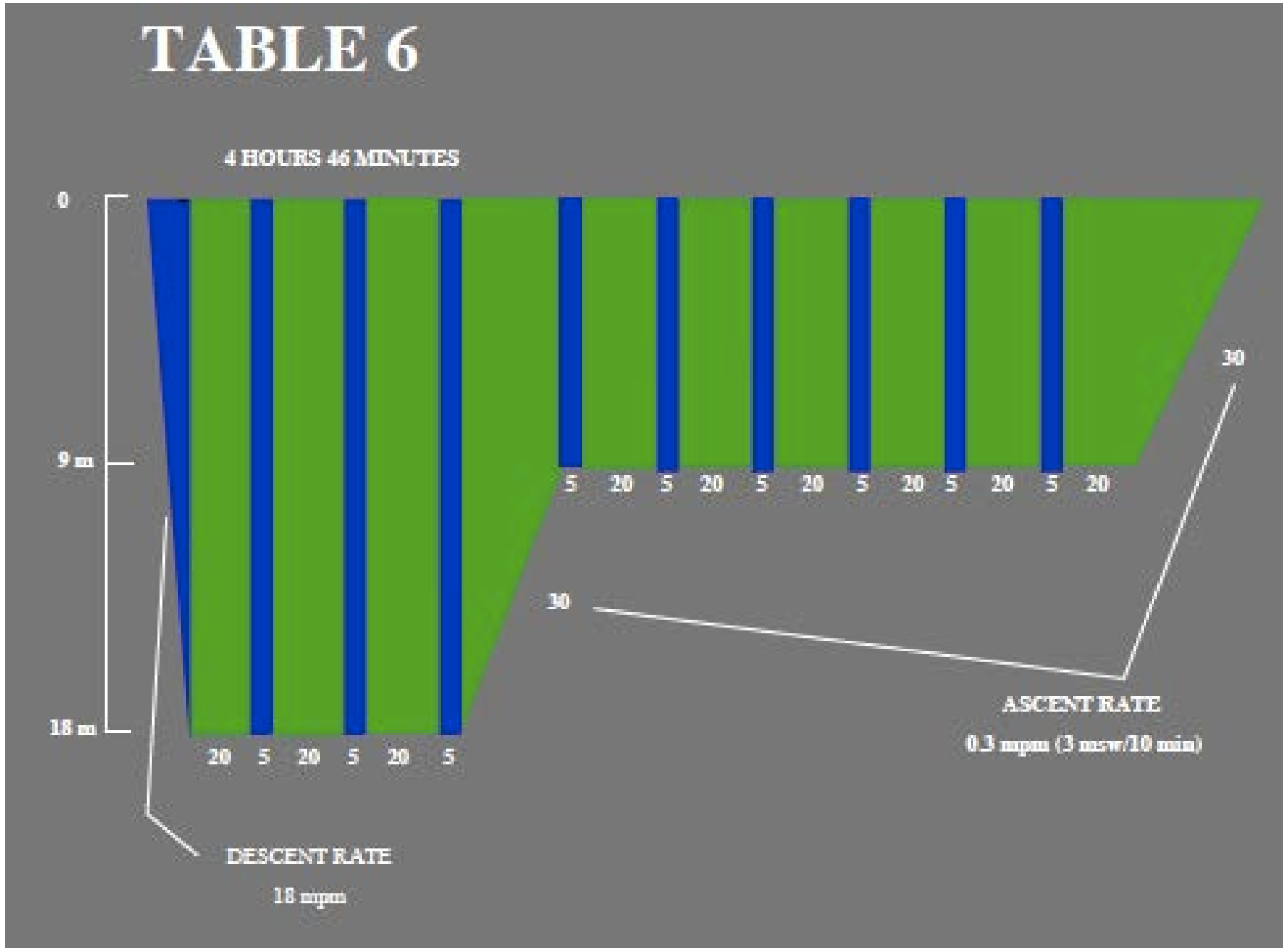
# Treatment Table Summary

| Table | Indications |
|---|---|
| 5 | Type 1 DCS only if sx completely resolved during transport to RCC<br>Recurrence Type 1 Sx (complete resolved after 1x $O_2$ period at 18msw)<br>Omitted-D, uncontrolled ascent/blow-up – for Asx if omitted D ≤30 mins) |
| 6 | DCS, AGE responding to initial 18msw RCC<br>Omitted D, uncontrolled ascent/blow-up: Asx individual with ≥ 30 mins |
| 6 Mod | Extension of TT6, if patient remains sx by end of $2^{nd}$ $O_2$ period at 18msw |
| 6A | Severe AGE/DCS deteriorating or not responding at 18msw, resolving @50msw |
| 6A Mod | Extension of TT6A, if patient remains sx by end of $2^{nd}$ $O_2$ period @18msw |
| 7 | Heroic measure, used in extreme cases |
| 8 | Deteriorating severe AGE/DCS sx @50msw, recurrence of severe AGE/DCS during deco from 50msw-18msw etc. |

# TABLE 5


2 HOURS 16 MINUTES
0
9 m
18 m
20
5
20
5
5
20
30
30
ASCENT RATE
0.3 mpm (3 msw/10 min)
DESCENT RATE: 18 mpm

# TABLE 6


4 HOURS 46 MINUTES
0
9 m
18 m
20 5 20 5 20 5
5 20 5 20 5 20 5 20 5 20 5 20
30
30
DESCENT RATE
18 mpm
ASCENT RATE
0.3 mpm (3 msw/10 min)

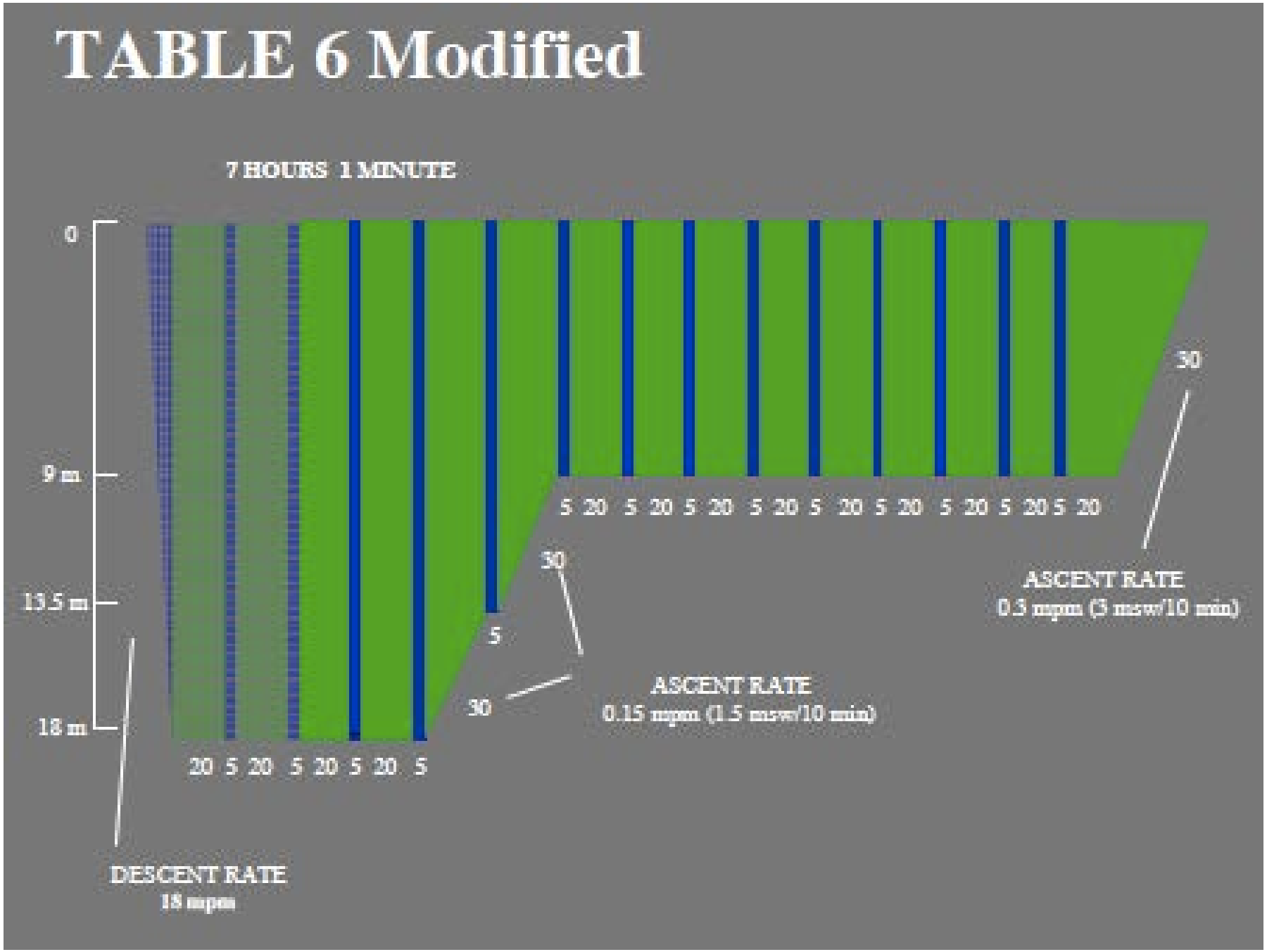


TABLE 6 Modified
7 HOURS 1 MINUTE
0
9 m
13.5 m
18 m
20 5 20 5 20 5 20 5
30
5
30
5 20 5 20 5 20 5 20 5 20 5 20 5 20 5 20 5 20
30
DESCENT RATE
18 mpm
ASCENT RATE
0.15 mpm (1.5 msw/10 min)
ASCENT RATE
0.3 mpm (3 msw/10 min)

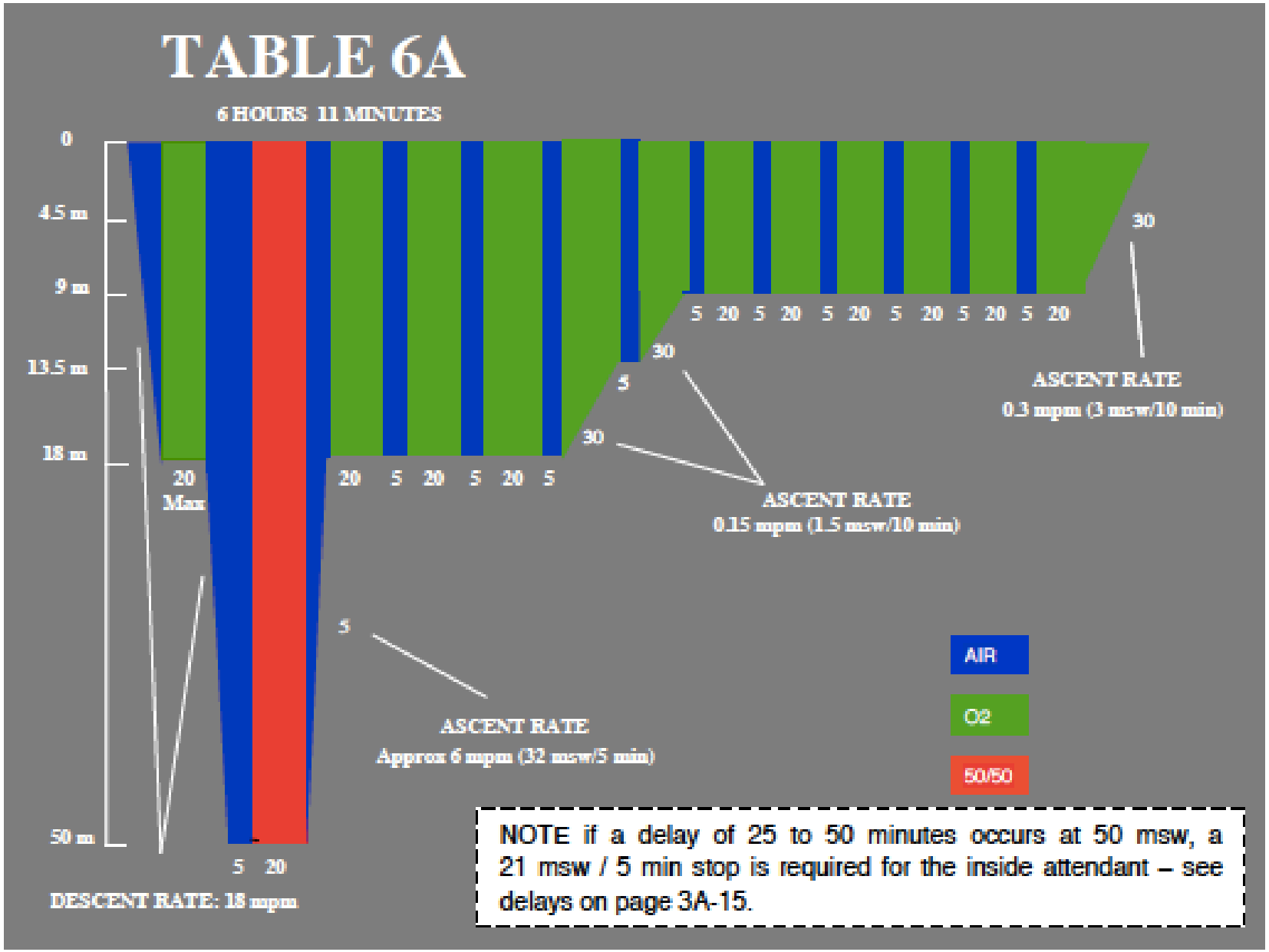


TABLE 6A
6 HOURS 11 MINUTES
0
4.5 m
9 m
13.5 m
18 m
50 m
20
Max
20 5 20 5 20 5
30
5
30
5 20 5 20 5 20 5 20 5 20 5 20
30
ASCENT RATE
0.3 mpm (3 msw/10 min)
ASCENT RATE
0.15 mpm (1.5 msw/10 min)
5
ASCENT RATE
Approx 6 mpm (32 msw/5 min)
5 20
DESCENT RATE: 18 mpm
AIR
O2
50/50
NOTE if a delay of 25 to 50 minutes occurs at 50 msw, a 21 msw / 5 min stop is required for the inside attendant – see delays on page 3A-15.

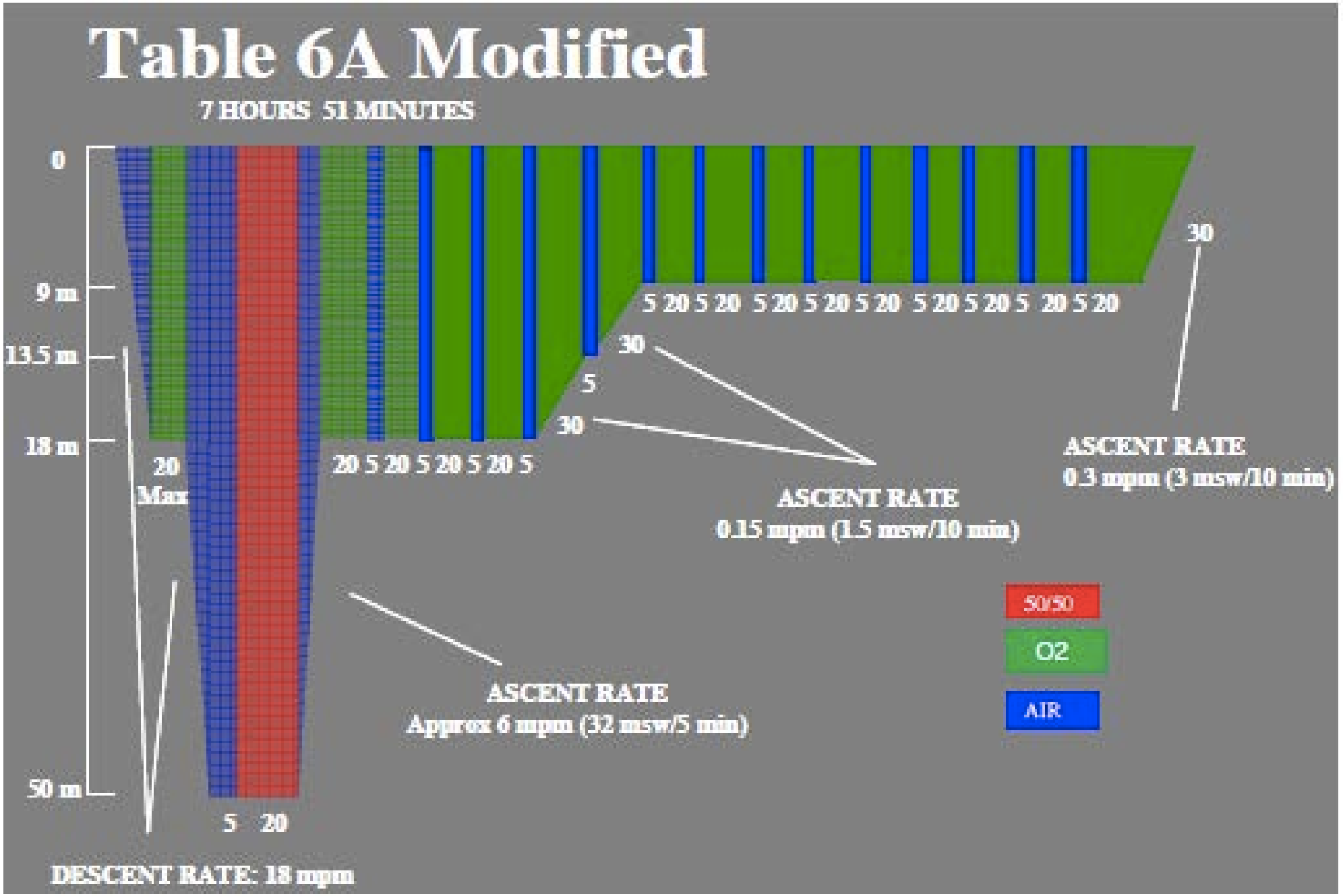


Table 6A Modified
7 HOURS 51 MINUTES
0
9 m
13.5 m
18 m
50 m
20
Max
20 5 20 5 20 5 20 5
5 20 5 20 5 20 5 20 5 20 5 20 5 20 5 20 5 20
30
5
30
30
5 20
DESCENT RATE: 18 mpm
ASCENT RATE
Approx 6 mpm (32 msw/5 min)
ASCENT RATE
0.15 mpm (1.5 msw/10 min)
ASCENT RATE
0.3 mpm (3 msw/10 min)
50/50
O2
AIR

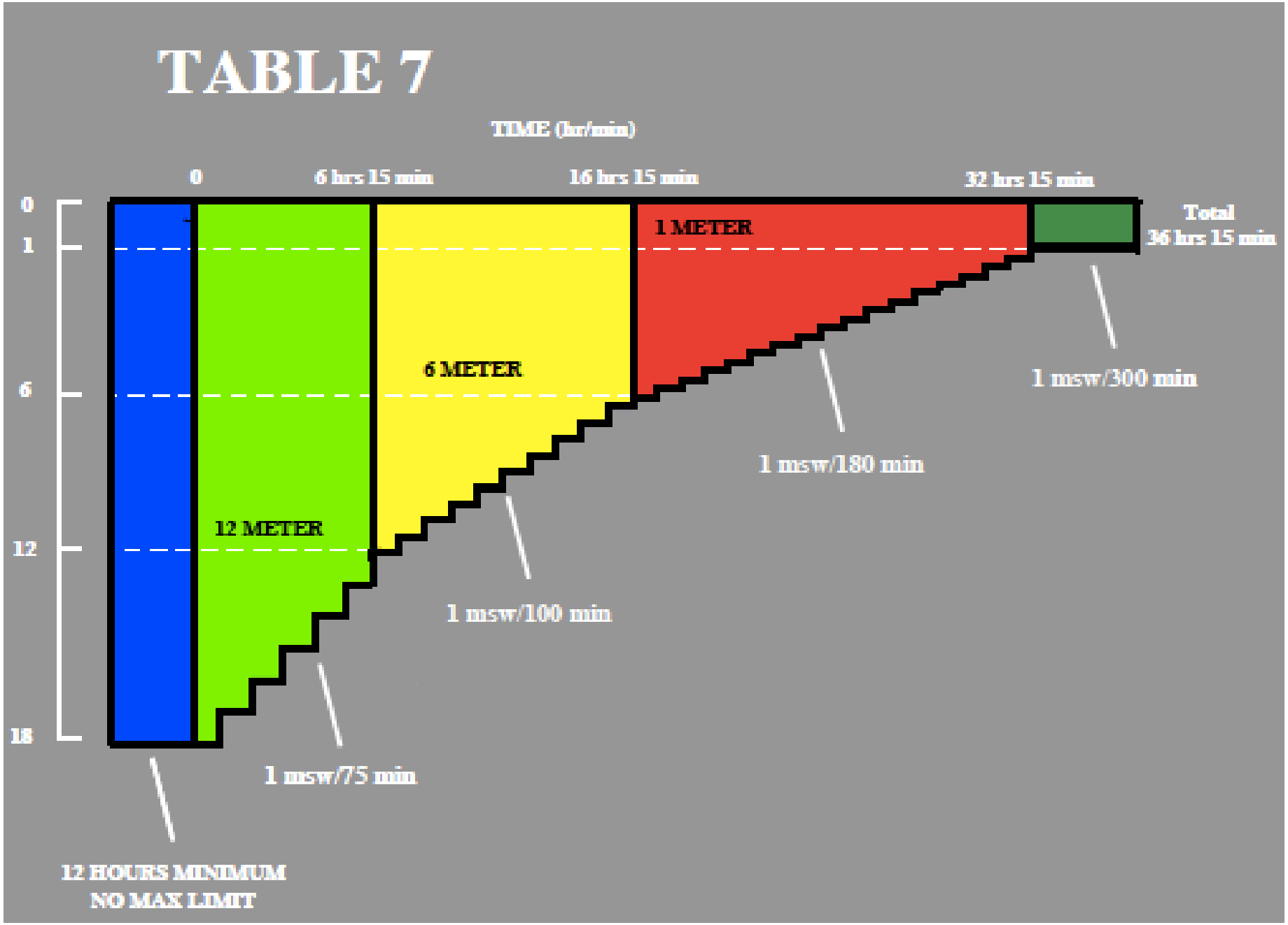


TABLE 7
TIME (hr/min)
0
6 hrs 15 min
16 hrs 15 min
32 hrs 15 min
0
1
6
12
18
Total
36 hrs 15 min
1 METER
6 METER
12 METER
1 msw/300 min
1 msw/180 min
1 msw/100 min
1 msw/75 min
12 HOURS MINIMUM
NO MAX LIMIT

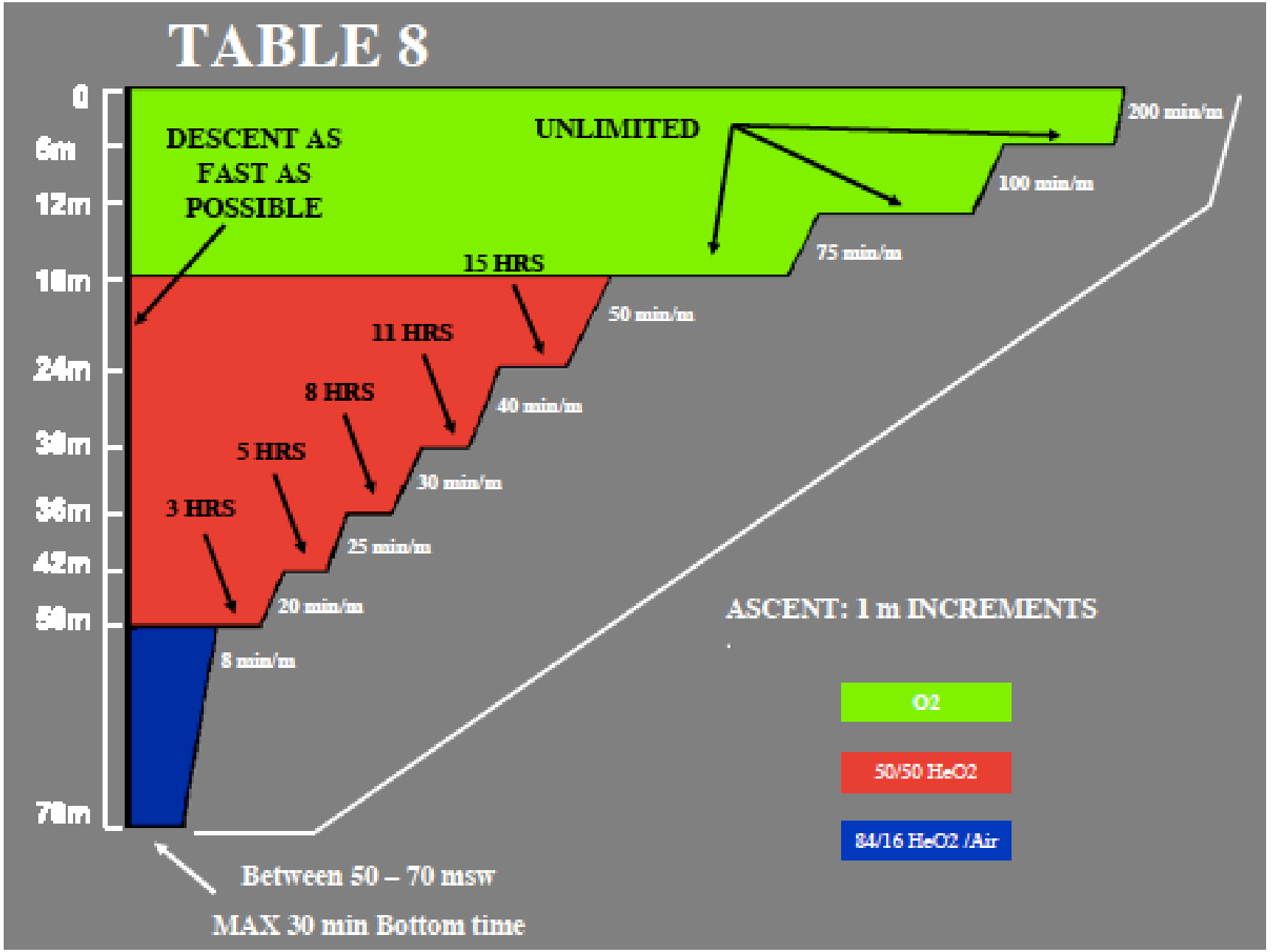


TABLE 8
0
6m
12m
18m
24m
30m
36m
42m
48m
70m
DESCENT AS FAST AS POSSIBLE
UNLIMITED
200 min/m
100 min/m
75 min/m
15 HRS
50 min/m
11 HRS
40 min/m
8 HRS
30 min/m
5 HRS
25 min/m
3 HRS
20 min/m
8 min/m
ASCENT: 1 m INCREMENTS
O2
50/50 HeO2
84/16 HeO2 /Air
Between 50 – 70 msw
MAX 30 min Bottom time