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/TT6A 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