



Decompression Illness



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Decompression Illness (DCI)

- What is it ?
- How to recognize it.
- What to do.

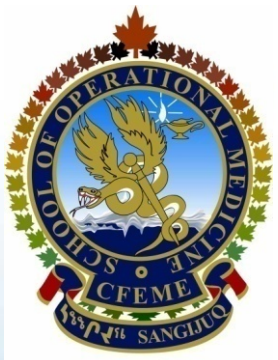




DCI – What is it ?

“Another suspicion we should have entertained concerning the death of our animals – namely, that upon the sudden removal of the wonted pressure of the ambient air, the warm blood of these animals was brought to an effervescence or ebullition; or, at least, so vehemently expanded as to disturb the circulation of the blood, **and so disorder the whole economy of the body.**”(Boyle, 1672)





DCI – What is it ?

- “**disease state** which develops as a result of tissue disturbances caused by the formation of inert gas bubbles in the tissues” (CF Dive Manual – 1980ish)





DCI or DCS ?

- DCI includes the spectrum of bubble related illnesses including decompression sickness (DCS) and arterial gas embolus (AGE).
- Separation of standard classification into Type 1,2,3 DCS and AGE often difficult to make clinically and
- While pathology leading to symptoms may differ, treatment is initially the same.





Bubbles: Source in AGE

- Complication of pulmonary barotrauma:
 - Tears allowing alveolar air to gain direct entry into pulmonary venules
 - Flow to left atrium and then to arterial circulation
 - Most often resulting in CAGE
- Iatrogenic:
 - Direct vascular access
 - e.g. large venous injections, cardiac surgery.





Bubbles: Source in DCS

- Henry's Law in action.
- Ability of inert gas to stay in solution is exceeded as ambient partial pressure of that gas decreases:

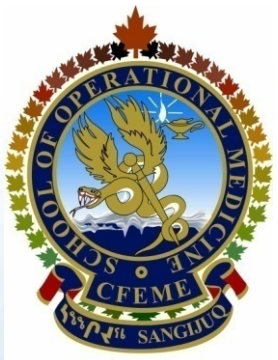




Its not that simple.

1. We aren't suddenly decompressed (usually) – partial pressure of N₂ is partially equalized by blowing it off.
2. Tissues can be supersaturated - can tolerate a partial pressure difference (Haldane said of 2 times) without bubbling.
3. Tissue uptake and release of gas is tissue dependent (Haldane again and many others) and depends on:
 - Movement of gas to and between tissues (blood flow, solubility, diffusability)
 - Individual characteristics (seasoned vs. naïve diver, predisposing factors)





Where do bubbles form ?

- Lymphatic system.
- Within tissue, peri-articular, nerve, inner ear, skin.
- Veins (VGE).
- Where bubbles form (and grow) depends on local tissue partial pressure differences and local area susceptibility, (scratches and eddies where bubble formation is more likely)





Trouble from Bubbles

- Not all bubbles cause trouble:
 - We can inject air IV
 - Doppler shows us that VGE in divers are common.
 - Bubbles cleared by the lung (to a limit).
 - Tissue bubbles may exist without symptoms.
- Bubbles, symptoms and decompression stress related but not perfectly:
 - Bubbles load from decompression stress varies within populations and individuals





Bubble Effects

1. Primary Effects – Mechanical

- vessel obstruction
- tissue distortion
- mechanical injury (endothelial) (Nossum et al, 1999)

2. Secondary effects - Immuno-inflammatory

- As a result of above mechanical effects and/or
- Direct activation of plasma and formed blood elements





Recognition: DCS Top 5

1. a neurological symptom as the primary presenting symptom;
2. onset time of symptoms
3. joint pain as a presenting symptom
4. any relief after recompression
5. the maximum depth of the last dive.

(Freiberger , 2004)





Recognition: AGE Top 5

1. onset time of symptoms
2. altered consciousness
3. any neurological symptoms as a presenting symptom
4. motor weakness
5. seizure as the primary presenting symptom





Recognition

- Based on history (symptoms, onset, dive)
- There is no test other than response to treatment.





Onset Time

- Many different studies on onset time but:
 - Large majority within 6 hours
 - Serious neurological starts early (10 min with 90% by 3 hours)
 - Median time for AGE, DCS onset 0, 1.8 hours respectively (DAN report, 2006)
- Recall bias at play, type of diving (recreational, military, commercial) can make a difference in reporting.





Symptoms

- DAN prevalence data (1992-1998):
 - Neurologic symptoms 40% (sensory 23%, motor 8%)
 - Pain 22%
 - Audiovestibular 12.6%
 - Cutaneous 3.5%
 - Cardiopulmonary 2 %
 - Constitutional 14.7%

(Brukbakk and Neuman, 2003)





What to do with DCI ?

- Stop diving
- Surface O₂
- Fluids
- Hyperbaric O₂, repeated until plateau





Surface O2

- Why it should work:
 - Increases bubble/tissue N2 gradient.
 - Improves O2 delivery to hypoxic tissue.
- Does it ?
 - Persistent complete relief 14%
 - Improvement 51%
 - Decreased requirement for multiple recompression treatments. (Longphre et al, 2007):





Rehydration

- Why it should work:
 - Divers (in particular with DCI) are dehydrated.
 - Fluids increase volume; increases delivery of O₂ and removal of inert gas.
- Does it ?
 - Dehydrated saturated pigs suffer worse DCS (death, cardiopulmonary) when surfaced (Fahlman,2006).
 - Increase in diver's Hct after a single dive (Williams 2007) and Hct >48 associated with worse neuro DCS in women (Newton 2008)





Hyperbaric O2

- Bert in 19th century noted benefit from surface O2
- Benefit of air tables exploited in the early 1900's
- Not until the 1950-60's was O2 combined with pressure





HBO – Why it should work.

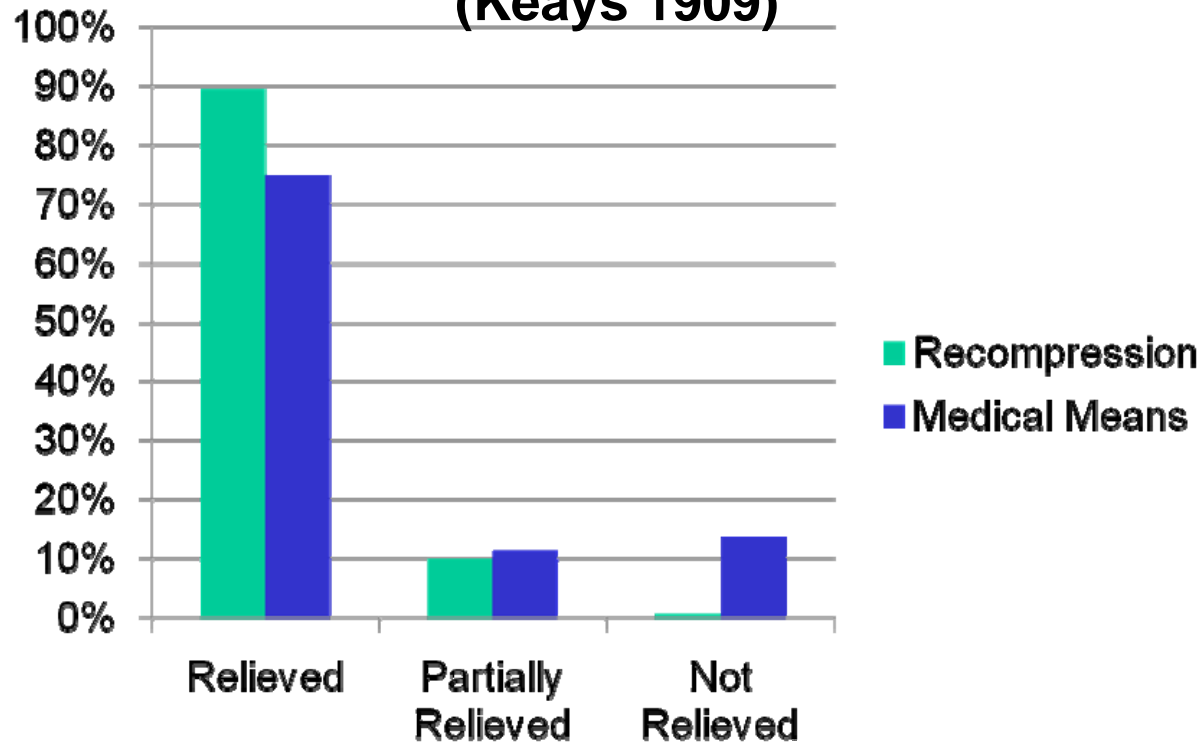
- Why it should work:
 - Decrease bubble size (Boyle).
 - Increase bubble/tissue N₂ gradient (3 X that of surface O₂ at 2.8ATA).
 - Increased delivery of O₂ to hypoxic tissues.
 - Immune modulation leading to decreased tissue inflammation and re-perfusion injury.
- Does it ?
 - Sure





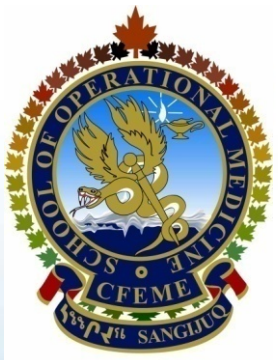
What if you do nothing (bends defined by pain) ?

Air Recompression vs. Medical Treatment For Caisson Illness- Pain
***(Keays 1909)**



*Chi Square = 249.9,df 3 p<0.0001





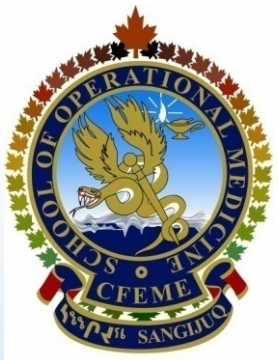
What if you do nothing (bends defined as serious DCS) ?

- RN experience of 186 not “pain only” bends (Green and Leitch, 1987)
 - 12 % - Spontaneous improvement
 - 3% - Spontaneous recovery
 - 25 % - Relapse rate in those not treated due to spontaneous improvement

With treatment (air or O2 tables):

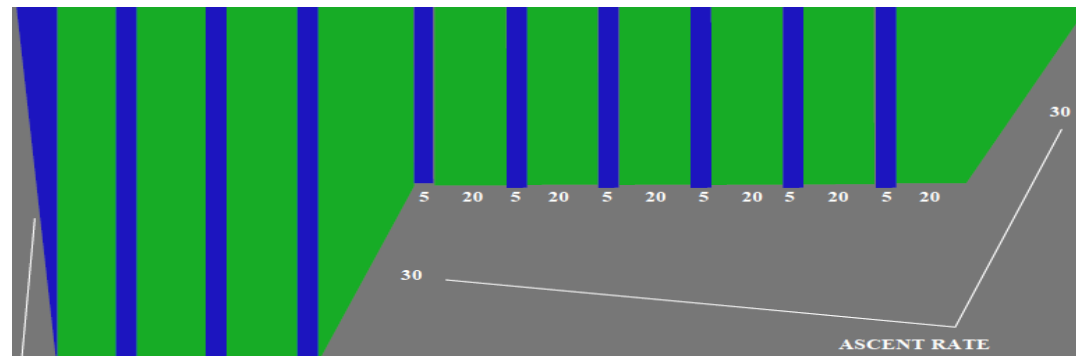
- 42% cure
- 27% mild residual
- 31% more severe residual
- 5.6% Relapse





What tables ?

- Trend away from deep air tables (6A).
 - Initially designed for AGE – difficult to diagnose vs DCS 2
 - Uptake of inert gas on air at depth a problem (patient and tender).
 - At best, ambiguous evidence
 - Role may be more important in acutely treated deep blow up.
- Reliance on Table 6 (in various forms).
- Table 6 or 5 for follow-on treatments.





Treatment delay – when to call the plane.

- Delay of treatment in mild DCI unlikely to affect long term outcome (DAN workshop, 2004).
- Greatest benefit of early treatment seems to be in more severe DCI (Ball, 1993. Zeindler,2010)
- Benefit of treatment in prolonged delays (mean 5,median 2 days) in even severe DCI (Barrat ,2004; Cianci, 2006)





Adjuncts

- NSAIDs and Heliox may decrease number of required recompressions (Bennett, 2007)
- Lidocaine may have neuroprotective effect and may be protective in AGE (Mitchell, 2001)
- Perflourocarbons increase delivery of O₂ and speed N₂ removal. Works very well in animal models (Dromsky, 2004).





Summary

- Despite a 300 year history mechanisms, diagnosis and treatment of DCI are still evolving.
- Mechanical, biochemical and immune factors are at play – both in the development and treatment of DCI.
- DCI is a systemic illness.





Questions ?

- There are still a few left to answer.





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