Introduction to (some) physiological aspects of apnoea diving (in humans)

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Conflicts of interest

• I am interested by the lunch and the workshop this afternoon!

Putative table of Contents

- Respiratory physiology
- Cardiovascular physiology
- Renal physiology
- ENT
- Thermal regulation
- Metabolism
- Training
- Duration of apnea
- Medical problems



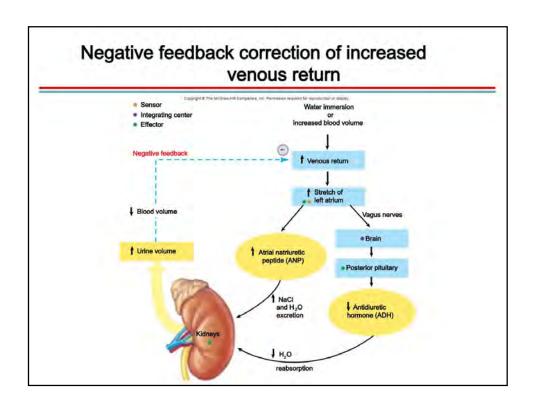
Let's dive! Immersion

- Hydrostatic pressure increases work of breathing when floating in near-vertical position
- Expiration is facilitated
- Blood shifts from the lower extremities into the chest
- Squeeze of abdomen shifts the diaphragm upwards → expiration
- Diuresis increases, t°

Diuresis 1



- Blood shift: increased central blood volume
- immersion blunts the thirst response



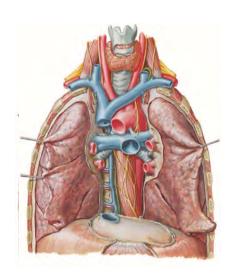
Blood volume

- Peripheral veins collapse → Blood shift
- Abdominal pressure increases →BS



Blood volume

- BS: increase heart and pulmonary vessels volume (+/- 700 ml)=
 - → work of breathing
 - — ↑ stroke volume and force of contraction (F-S law)
- Other mechanism: peripheral vasoconstriction



Blood volume

- Cardiac output ↑ 5-> 8 I/min
- HR ↓ 76->68 BPM

Vital capacity ↓+/- 10%

With tourniquets : VC \downarrow 2%



Facial immersion

Diving response: receptors Ophtwine Ophtwine

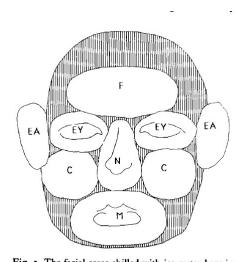
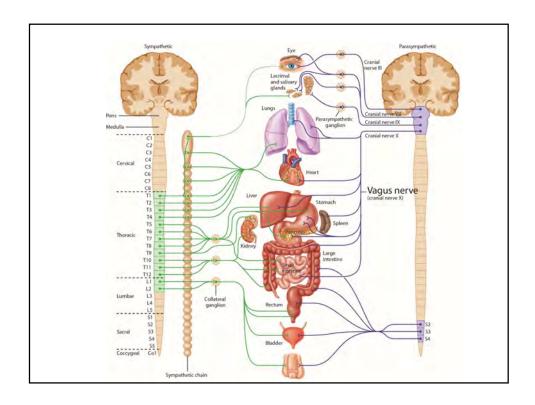
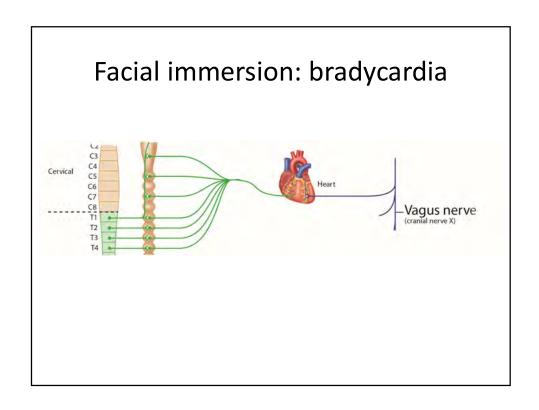


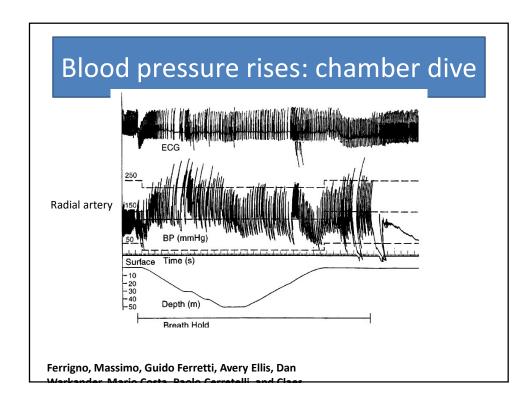
Fig. 1. The facial areas chilled with ice-water bags in six of the tests, the other two being only breath-holding (BH) and face immersion (FI) including all areas except the ears (EA).

Forehead and eye regions, produce slightly greater responses than the lower face

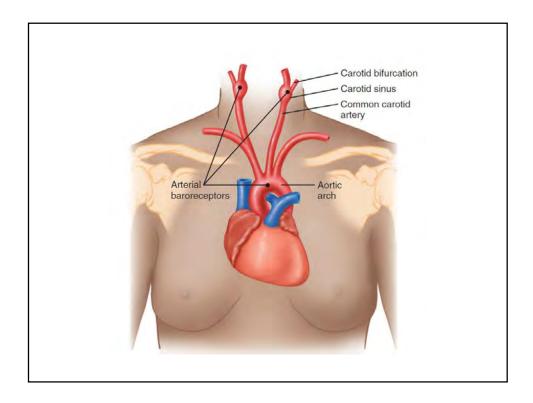
→ Removal of the facemask in cold water during apnea induces greater bradycardia





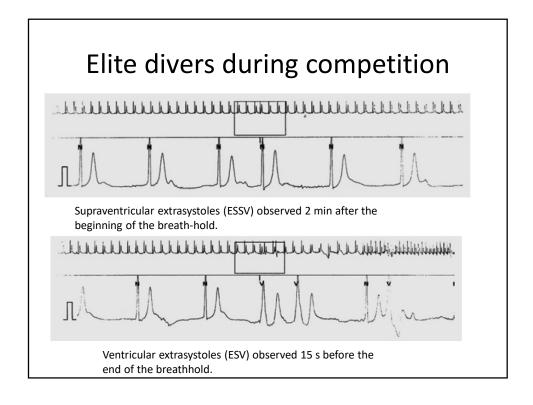


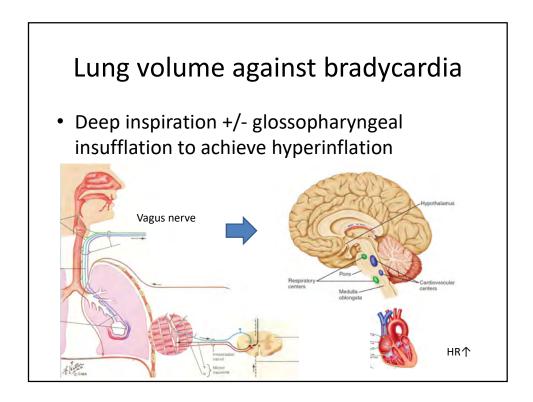
- A gradual and moderate rise in blood pressure is typical due to progressive hypoxia during surface apnea
- But depth creates a hyperoxic condition due to raised ambient pressure, so arterial hypoxia occurs only during the short late ascent phase near the surface.
- Physiological significance of this blood pressure surge ? CBF?
- The increase in arterial tension stimulates the circulatory baroreceptors and provokes bradycardia.

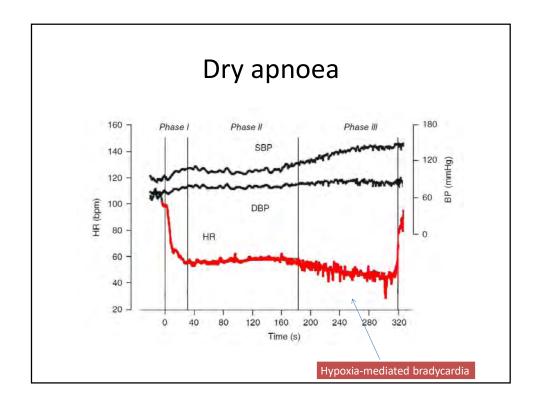


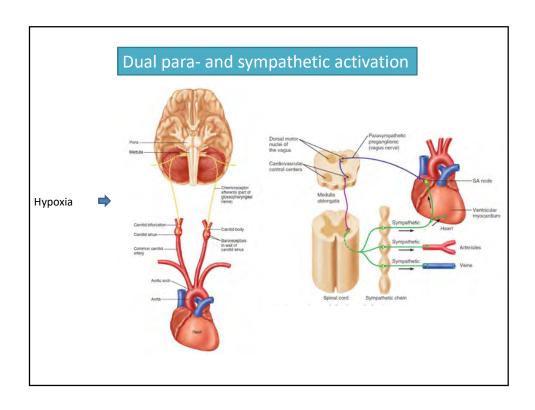
Few effect of depth on HR

- More rapid onset of bradycardia on dives to greater depths.
- A drop in water *temperature* with depth might contribute to greater bradycardia.
- The most extreme bradycardia reported during an ocean dive was 8 BPM during a dive to 107m by Pipin Ferreras
- Frequent: junctional rhythm and arrhythmias



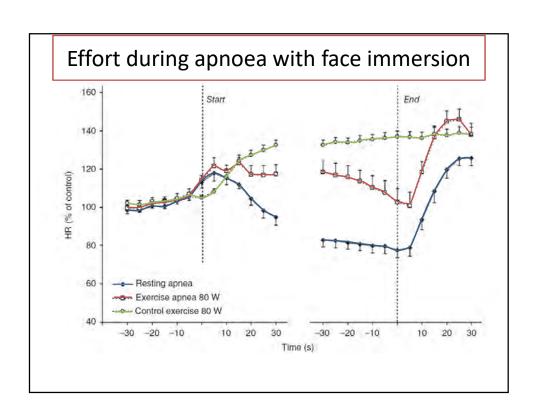






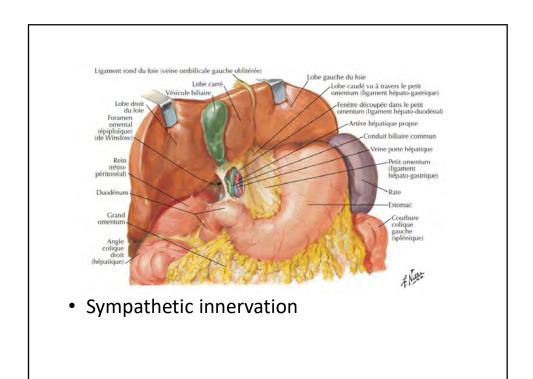
More on bradycardia

- Apnea (dry) usually induces bradycardia
- Facial immersion ↑ BC
- Hypoxia further enhances BC
- (not before the break point)
- Raise in BP also ↓ heart rate



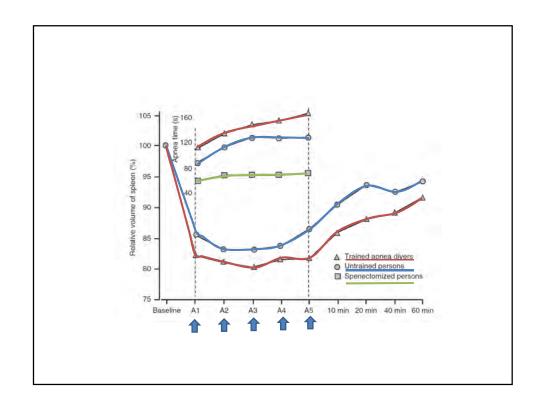
The spleen of the skin diver

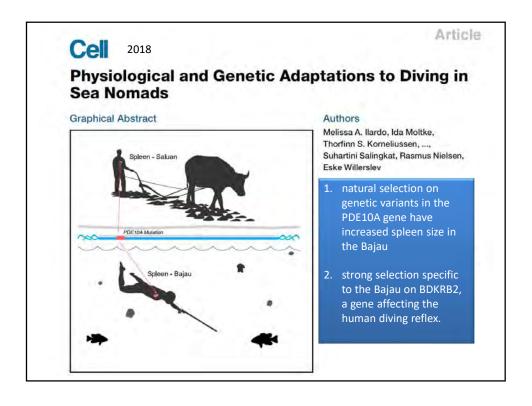
- A component of the diving response
- But also of stress, including loud noise, exercise, hypoxia, and hemorrhage
- Korean Ama: Mean spleen volume decreased from 206 to 165 mL, hemoglobin concentration increased by about 10%.



Spleen contraction: training

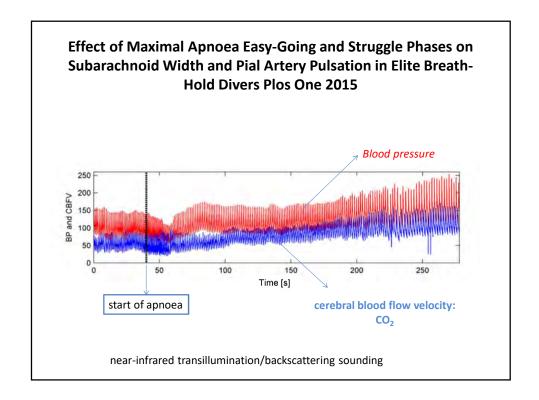
- Decrease in spleen size is higher in trained divers
- Apnea times progressively increase with repeated breath-holds spaced a few minutes apart → associated with progressive reduction in spleen volume
- Spleen contraction of 200 mL → CaO₂ ↑ 80 mL , +/- 20 seconds of apnoea ...

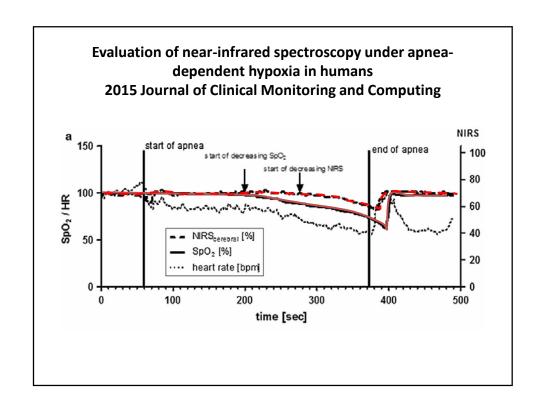




Brain blood flow

- enhanced by cold face immersion
- Peripheral vasoconstriction // increase CBF
- Hypoxia/hypercapnia
- Training effect?



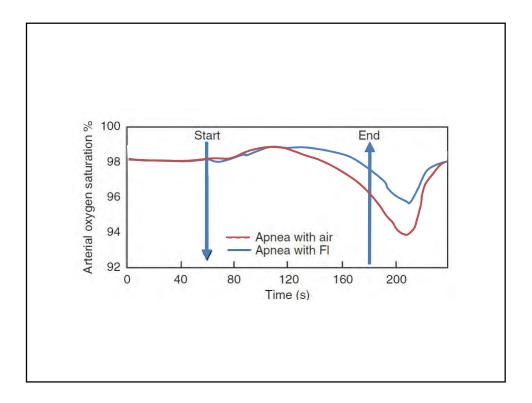


Diving: integrated response

- HR reduction
- Selective peripheral vasoconstriction
- Spleen contraction
- Increased CBF

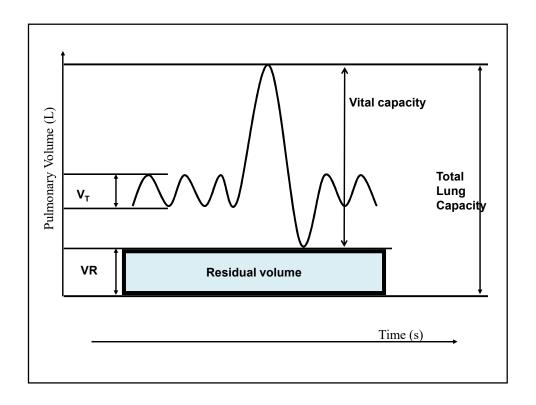
DR and training effect: apnoea duration

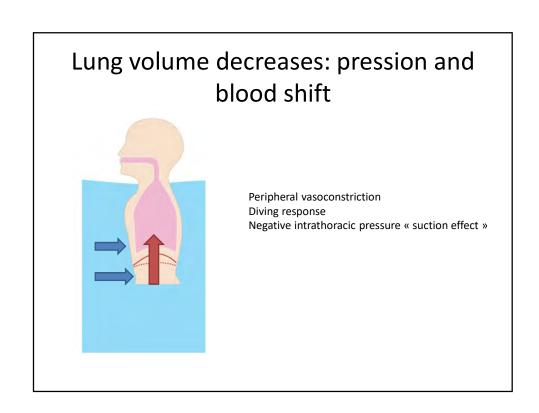
- Face immersion ↑ breath-hold time in <u>trained</u> apnea divers, but had the opposite effect in untrained individuals
- Negative correlation between maximum apnea time and lowest heart rate reached: activation of the diving response does prolong apnea time.
- Diving response is <u>oxygen conserving</u>, at least in trained apnea divers.

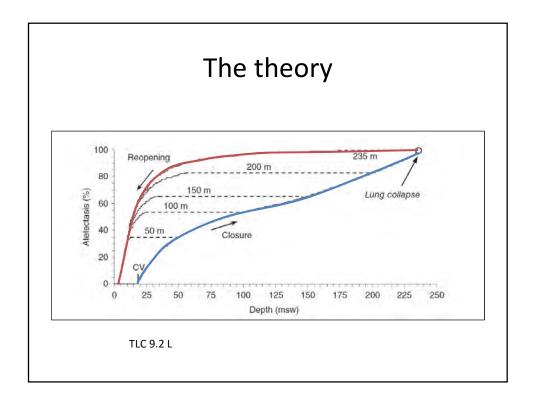


Deep, deeper

- Prior to the 1960s, it was incorrectly assumed that lung RV represented the lowest tolerable limit of chest wall compression.
- It was believed that ribs might crack or lungs would bleed from negative pressure injury





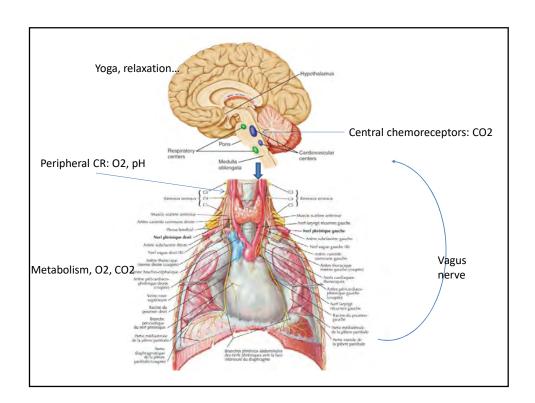


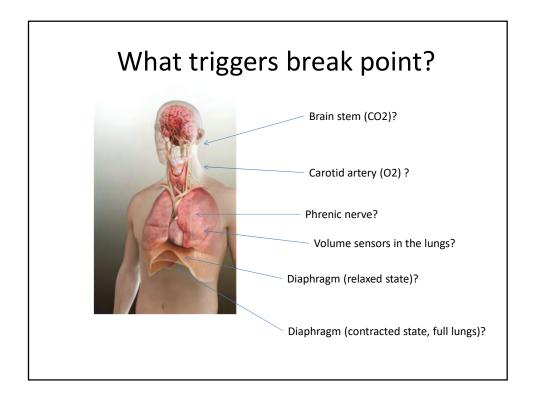
Surfacing to life?

- Hypoxic blackout
- Pulmonary capillary injury (lung squeeze)
- Alternobaric vertigo
- Narcosis
- Decompression sickness

Apnoea duration

- Size matters!
- Relaxation and fasting: slowing metabolism
- Hyperventilate but not too much
- training
- Stéphane Mifsud 11 min 35 s, 2009
- Inhale pure oxygen: Alexi Segura Vendrell, 24 min 03 s, 2016





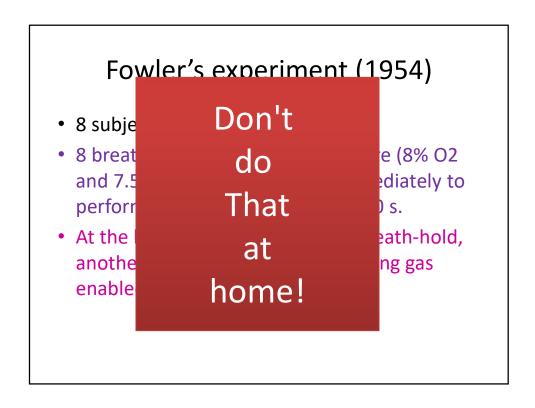
O2 level? CO2? Both...or none!

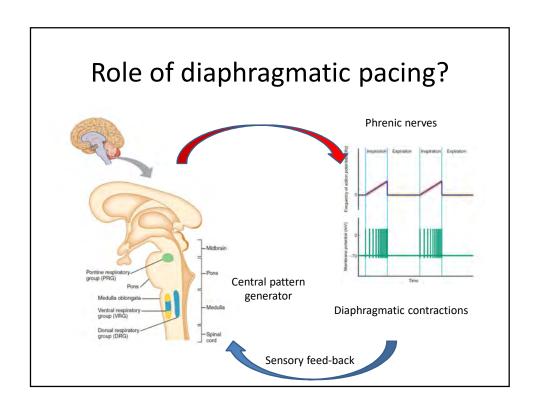
- At breakpoint from maximum inflation in air, the PetO2 is typically 62±4 mmHg and the PetCO2 is typically 54±2 mmHg
- loss of consciousness: PaO2 <~27 mmHg and PaCO2 between 90 and 120 mmHg
- But breakpoint levels close to these have been reported

- Breath-hold duration is almost doubled by breath holding with hyperoxic gas mixtures
- BP is also delayed by hyperventilation: PCO2
- But 1st apnoea is usually short and ABG normal
- Nor is the breakpoint at some unique combination of low PetO₂ and high PetCO₂
- Even after the longest possible breath-holds from hypocapnia with preoxygenation, blood gas levels at breakpoint are remarkably benign.

Fowler's experiment (1954)

- 8 subjects, dry apnoea → BP
- 8 breaths of an asphyxiating mixture (8% O2 and 7.5% CO2) enabled them immediately to perform another breath-hold for 20 s.
- At the breakpoint of the second breath-hold, another 8 breaths of the asphyxiating gas enabled a further 20 s breath-hold





Two healthy, conscious volunteers

Campbell's experiment (1966, 1967, 1969)

skeletal muscles temporarily paralyzed with intravenous curare—except for one forearm, with which they could signal their wishes.

The subjects were kept alive with a mechanical ventilator

breath holding was simulated by switching it off, and the subjects indicated their break point by signaling when they wanted the ventilator restarted.

- Both volunteers were happy to leave the ventilator switched off for at least four minutes, at which point the supervising anesthetist intervened when PetCO₂>70 mmHg
- After the effects of the curare had worn off, both subjects reported feeling no distressing symptoms of suffocation or discomfort
- mean 'breath-hold' durations were prolonged
 3x

This slide to let you take a deep breath again

To conclude

- A series of mechanisms are associated with diving: integrated response
- So far, human data are
 - Scarce
 - Limited to some elite divers
 - Limited by water
 - Incomplete (dry vs. wet apnoeas, rest, effort...)
 - Variable
 - But...

