Respiratory Adaptations of Mammalian Divers

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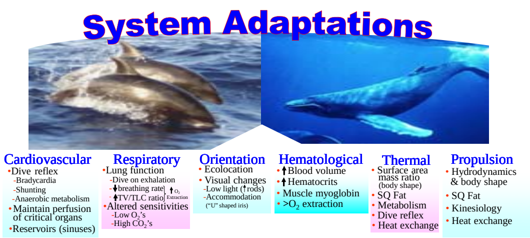
Introduction

At the 2006 UHMS we presented an overview, in a poster format, of mammalian adaptations to diving and their counterparts in human divers. The adaptations were divided into six systems as follows:



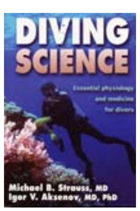
This presentation is a sequel to the 2006 overview that describes the respiratory adaptations of diving mammals

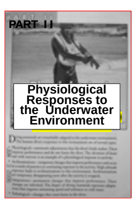
In addition, our presentation compares & contrasts the respiratory adaptations of diving mammals with those found in human divers

Finally, it shows how the human diver is able to make adjustments (temporary changes) in the respiratory system as well as use equipment to improve diving performance

Materials & Methods

In researching material for Part II *(Physiological Responses to the Underwater Environment)* for our ***Diving Science—Essential physiology and medicine for divers*** book, we discovered a wealth of information about mammalian adaptations to diving

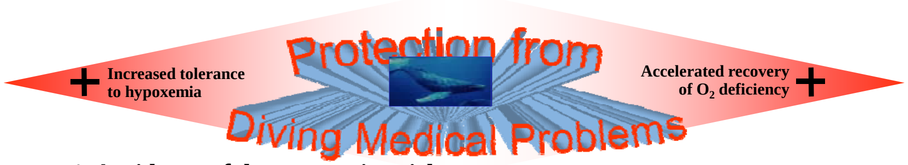




The primary role of the respiratory adaptations of diving mammals is to provide protection from medical problems associated with breath-hold diving

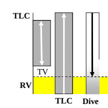
Like the other mammalian adaptations to diving, the respiratory adaptations have secondary roles which contribute to the aquatic performances of these animals

Features of the Respiratory Adaptations

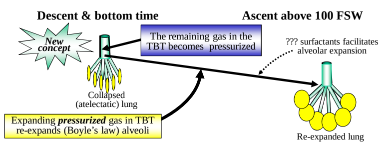


1. Avoidance of decompression sickness

- **Dive after exhalation**
  - Residual volume (RV)…the only air remaining in the lungs when starting the dive
  - This automatically reduces the amount of air in the alveoli—for on-gassing to the blood—by 80-90%

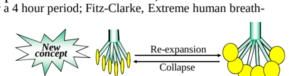


- **By the time a depth of 100 FSW is reached** (Irvine L. ***Science*** 1935; 822:560-561)
  - The residual volume of air in the lungs decreased to ~ zero volume i.e. atelectasis
  - The remaining air in the respiratory system remains in the tracheal-bronchial tree (TBT) so off-gassing to tissues does not occur and the air becomes pressurized in the TBT
- **Re-expansion of alveoli occurs with ascent**



Note: Human breath-hold divers have developed decompression sickness: Cross, Tuamotu divers--Taravana syndrome; Paulev with repeated dives to 100 FSW over a 4 hour period; Fitz-Clarke, Extreme human breath-hold diving (UHM, 36(1) Mar/Apr 2009)

2. Eluding thoracic squeeze



- **Intrinsic elastic properties of the alveoli**
  The pressure of the compressed RV gas as well as the air in the TBT (dead space) that occurs with breath-holding while descending provides the positive pressure (~PEEP) to re-expand the lungs with ascent
- **Reduction of the alveolar space from vascular engorgement** To "obliterate" an alveolar space (0.004 mm^3) would require ~ 1/5000th as much engorgement as to achieve the same effect in the middle ear space (19.6 mm^3)
- **Symmetrical compression of the chest wall with descent** This decreases the volume of the chest cavity and proportionally decreases the lung capacities and volumes while pressurizing them—The effect is equilibration of pressure of the air in the lung with the ambient pressure as a SCUBA regulator does
- **Blood shifts from the extremities to the core** Shunting as a component of the dive reflex shifts blood from the extremities to the core to complement vascular engorgement of the alveoli and the visceral organs
- **Shifting of abdominal contents into the thoracic cavity** Elevation of diaphragm and upward movement of visceral organs into thoracic space in conjunction with compression/pressurization of the air-filled bowel

Note: Human breath-hold divers also use the techniques of buccal pumping and increasing their thoracic squeeze threshold (an acclimatization that increases total lung capacity with relative decrease of RV

3. Prevention of breath-holding blackouts

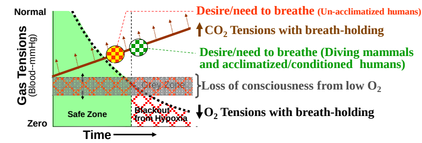
- **Alteration of the signals to breathe** Increased tolerance to rising CO_2 tensions and increased tolerance to low O_2 tensions before consciousness is lost. Conditioned human divers develop these as acclimatizations with practice
- **Averting diffusional blackout** After the 100 FSW depth, gas is neither compressed—for increased O_2 extraction at depth—or diluted during ascent because of collapse of the alveoli and cessation of gas exchange

4. Circumvention of nitrogen narcosis, O_2 toxicity, air embolism & CO poisoning as well as DCS by not breathing compressed gas at increased ambient pressures

↑ Tolerance to Hypoxemia

- Aquatic mammals are less sensitive to low alveolar O_2 tensions and elevated CO_2 tensions than non-aquatic mammals

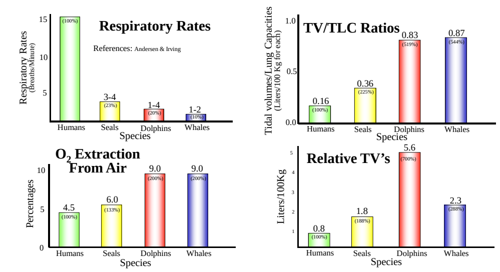
Seals: Resting alveolar CO_2 tensions are higher & O_2 tensions are lower than in humans
Decreased ventilatory responses to ↑CO_2 & ↓O_2



- Human acclimatizations (Temporary improvements with practice)
  - Decreased ventilatory responses to 10.5% CO_2
  - Improved O_2 utilization
  - Tolerance of larger O_2 deficiency (debts)
  - Increased tolerance to elevated tissue CO_2 levels
  (Schaefer)
  - Markedly elevated end-tidal CO_2 volumes observed in trained underwater swimmers including the Ama (diving women) of Japan & Korea (Craig, Elsner, Koff)

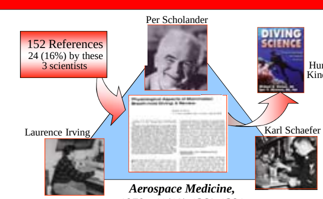
Accelerated Recovery

- Accelerated recovery of O_2 depletion secondary to improved ventilatory mechanics; Secondary benefits include reduction of respiratory heat loss and moisture + conservation of energy



- Improved diffusion capacity with immersion–16% increments with immersion to the neck level (Guyatt) secondary to pulmonary vascular engorgement from the pressure gradient; this compliments dive reflex shunting response & compensates for lung volume decreases secondary to immersion

References



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