

# Lung in extreme environments

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# Introduction

- Lung - First barrier between the body and its surrounding atmosphere.
- Various activities expose humans to different environments in which the stresses are beyond our physiologic capabilities.
- *Extreme environments & the lung*
  - Underwater
  - High altitude
  - Space
  - Extreme cold

**History of Diving –  
Apnoea diving**

**SCUBA**

**Costeau & Gagnan  
1943  
French navy**



# Lung physiology in diving

- **Diving - Exposure to higher than normal ambient pressure.**
- **Compression, isobaric, and decompression phases.**
- **One atmosphere - 760 mm Hg or 101.3 kPa.**
- **One bar corresponds to a pressure of 750 mm Hg, 100 kPa, or 10 msw (Metres of sea water).**
- **Depth of 30 msw - pressure of 4 bars**
- **100 msw - pressure equivalent of 11 bars.**
  
- **4 bars / 30 msw, the fractional concentration of oxygen is still 0.21 but the partial pressure is 84 kPa.**

- **Hyperoxia** - > 50 kPa , reduction in VC dependent on pressure and exposure time.
- **Unit pulmonary toxic dose (UPTD)** - toxic effect equivalent to the exposure to oxygen at 101 kPa for 1 minute.
- **Symptoms of nonproductive coughing and a retrosternal burning sensation before a reduction in VC takes place.**
- **The recovery of VC reductions as large as 20%–30% is usually complete within 1 or 2 weeks.**
- **Toxic effects of oxygen are mediated by reactive oxygen species, and inflammatory changes in the lung parenchyma are induced.**

- **Gas density and respiratory mechanical loading**
- **Density increases proportionately with pressure.**
- **Airway resistance is proportional to density with turbulent flow characteristics**
- **Maximal expiratory flow rate is inversely proportional to the square root gas density.**
  
- **When breathing air at a pressure of 4 bars, gas density is 4 times normal and maximum voluntary volume (MVV) and FEV1 are reduced by 50% of normal.**
  
- **To reduce the internal resistance of breathing gas mixtures, helium is usually used instead of air when diving deeper**
  
- **The helium and oxygen mixture breathed in deep experimental dives to pressures (500–700 msw) has a fractional concentration of oxygen of less than 2%, with the rest being helium and, sometimes, some nitrogen.**

# Respiratory heat and water loss

- Gas mixtures used for diving are dry so as to prevent icing in the gas supply lines.
- Specific heat capacity for helium is five times greater than for nitrogen but density is only 0.18 g/L for helium and 1.25 g/L for nitrogen.
- Therefore, respiratory heat loss when diving with air remains higher compared with diving with helium-oxygen mixtures, because it is the product of density and specific heat capacity that determines heat loss.
- Respiratory heat loss when diving is always larger than at normal atmospheric pressure whatever the gas mixture is.

# Decompression stress and venous gas microembolism

- **Supersaturated fluids - Inherently unstable and there is a risk of free gas evolving in gas bubbles.**
- **Gas bubbles - Interstitially in the tissues / more commonly, intravascularly in venous blood.**
- **Large shower of venous gas microemboli (VGM) → large increase in pulmonary arterial pressure - spillover of gas bubbles to the pulmonary veins.**
- **Reductions in DICO and maximal oxygen uptake correlating with the cumulative load of VGM have been demonstrated.**



# **Pulmonary barotrauma (PBT)**

- **Exposure to abrupt pressure changes.**
- **Individuals at risk of PBT - Astronauts, aviators, compressed air workers, and divers.**
- **Diving related PBT – Second among all causes of SCUBA diving fatalities.**
- **PBT during descent of apnoea dive - Lung squeeze**
- **PBT during ascent**

# PBT during ascent

- Boyle's law – Relevance
- Rate of ascent vs rate of elimination.
- Overdistention – May lead to lung rupture
- Precipitating factors – Breath holding , airway obstruction.
- Transpulmonary pressure rather than absolute intratracheal pressure is the critical factor.

- Rupture → Peribronchial space → Pulmonary interstitial emphysema → Pneumomediastinum → Pneumothorax / Pneumoperitoneum
- Rupture into PA / Dissection into Pulmonary veins → AGE (Arterial gas embolism)
- Head position, buoyancy of the gas bubbles, and blood flow dynamics – Gas bubbles (Brain) – Critical injury
- Fatal cases of PBT - Obstruction of the heart (pneumocardium) and central circulation after massive AGE
- Misapprehension that diving accidents do not occur in shallow water – Max risk just below surface

- **Symptoms appear on or shortly after surfacing , 5 mins (90 % cases)**
- **Without AGE ,only pulmonary symptoms may be present.**
- **Hoarseness /strange voice with mild chest pain after scuba dives – Think of PBT**
- **Risk factors for PBT**
  - **Technique of ascent**
  - **Intrapulmonary air trapping – Bullae/cysts**
- **No convincing data indicating PFT's to be predictive for detecting those divers who are at risk of PBT.**

# Decompression illness

- AGE as a result of PBT
- DCS – Decompression sickness - Multiorgan system disorder - micro or macroscopic nitrogen bubble formation on surfacing.
- Pathogenesis – On ascent , dissolved inert gas in the tissue and blood forms a free gas phase to equalize the pressures
- Reaches lungs by venous return – Intravascular gas bubbles may be seen in absence of symptoms (Silent bubbles)

# Clinical features of DCS

- **Musculoskeletal/ Cutaneous – Type 1 DCS – “the bends”**
- **Cutis marmorata (Cutaneous manifestation)**
- **Most cases responsive to immediate recompression.**
- **More severe DCS- Type II- Neurologic, audiovestibular, or respiratory manifestations.**
- **Nitrogen saturation and elimination kinetics of neural tissues / Minimal ischemia tolerance.**
- **Marked interindividual variation – Sensory symptoms predominate – Risk of DCS negligible at 0 – 10 msw**

# Risk factors for DCS


- Exposure to cold – unclear mechanisms
- Pre-dive exercise – Beneficial effects
- Obesity may be contributory
  
- Exposure to high altitude post diving - a minimum interval of 24 hours is recommended before flying breathing at depth – Even longer
  
- Respiratory DCI – “the chokes”
- Rare
- Clinical symptoms tachypnoea, cough, cyanosis, and thoracic discomfort or pain.

# Management of DCI

- **Delay - inversely related to outcome.**
- **Postpone time-consuming diagnostic procedures.**
- **CXR – Pneumothorax – Treat before recompression.**
  
- **Fast elimination of the gas phase and the correction of tissue hypoxia.**
  
- **Hyperbaric oxygen therapy – Results in  $\text{PaO}_2 > 2000$  mm Hg**
- **Most commonly used Algorithm - US Navy Table 6**
- **Oxygen breathing at 18 msw for approximately 75 minutes and at 9 msw for approximately 3 hours, with air pauses in between to minimize adverse oxygen effects**



# Management of DCI

- If hyperbaric oxygen therapy not immediately available (eg, at remote locations)  early administration of 100% oxygen
- Elevated serum creatine kinase level has been shown to be related to the size and severity of AGE

Paradoxical gas embolism – PFO / Pulmonary capillaries / Intra or extrapulmonary right to left shunts

Studies – 2.6 fold increase in the risk of DCI with PFO , but absolute increased risk relatively small.

# Pulmonary edema

- Only recently been recognized as a diving-related clinical problem
- No evidence of a single common risk factor.
- Subjects affected once are at risk of further incidents.
  
- Dyspnea , cough, hemoptyses, hypoxemia, tachypnoea
- Radiographic findings usually normalize within 48 hours.
- Removal from water and with supportive treatment

Subjects should be advised not to dive again.

Nifedipine (5 mg) before the dive may prevent recurrence

# Apnoea diving

- *Hae Nyo*
- *Sponge divers*
- *Pearl divers*
- *Competitive freediving*

# Growing popularity of Apnoea diving

Constant weight diving

Free immersion

Variable weight diving

Static apnoea

Dynamic apnoea

***No limit freediving*** – Most dangerous

Not approved as sport

Exhibition sport for diving extremists

# Theoretical limits of diving depth

- Descent – Decrease in lung volume ( Boyle's law)
- Decrease below RV – Lung squeeze – harmful
- TLC/RV ratio – Determinant of diving depth
- Ex. TLC – 6L RV – 1.5 L so  $TLC/RV - 4 \text{ atm} = 30 \text{ msw}$  (Concept till 1970)

**Bob croft – Father of american freediving**

**73 msw - Had exceptionally high TLC**

- **Redistribution during apnoea dives – 1 to 1.5 L**
- **Therefore now TLC – 9.6 L (Pipin ferreiras WR ) RV– 2.2 L so TLC/RV = 4.4 Atm or 34 msw**  
**Redistribution 1.5 L – RV 0.7 L so TLC/RV = 127 msw**

**Side effects – may lead to Alv. Hemorrhage / hemoptysis**  
**Also may occur due to STRUGGLE PHASE**

# Lung packing / Buccal pumping – They Will do any thing to dive deeper !!!!

Can increase TLC by 30-50 %  
(Average 1.5-3 L)

Applying to Pipin Ferreiras

$TLC - 9.6 + 2 = 11.6$

$TLC / RV = 11.6 / 0.7 = 156$  msw

No reports of PBT associated  
with buccal pumping

# Change in partial pressures of breathing gases

Critical hypoxia - Extended diving duration at significant depth.

Loss of motor control and consciousness in most cases appears immediately before or shortly after surfacing.

Elite apnea divers are able to tolerate a lower  $P_{aO_2}$  and oxygen saturation than controls and are more tolerant to carbon dioxide



## **Excessive hyperventilation**

- **PaCO<sub>2</sub> decreased – Prolongs breaking point of approx. 55 to 60 mm Hg.**
- **Pure oxygen also prolongs the apnea time substantially.**
- **Prolonged oxygen uptake during apnea - decreased PaO<sub>2</sub>.**
- **While ascending, rapid decompression of the lungs - marked fall of PaO<sub>2</sub>. ( Sigmoid curve)**
- **More dangerous if divers engaged in strenuous exertion before diving**
- **SAMBA – Shallow water blackout (Fatal accidents)**

# **Nitrogen narcosis & Decompression sickness**

**“Rapture of the depth”  
“taravana” (Neurologic DCS)**

**Nitrogen elimination slower  
than uptake**

**Risk increases with repetitive  
dives**

# Cardiovascular effects of Diving

- **DIVING REFLEX** – Diving related bradycardia , usually associated with peripheral vasoconstriction
- **Breath-holding at elevated lung volume and stimulation of facial trunks of the trigeminal nerve by water**
- **The lower the water temperature, the more pronounced the bradycardia.**
- **Normally, the initial fall in heart rate - approx. 60% to 70% of the pre-dive level.**
- **Rates as low as 10 to 15 beats per minute, however, are reported.**
- **In contrast to diving animals, in humans, the diving reflex usually is not associated with a fall in cardiac output**

- **May lead to cardiac dysrhythmias with increasing incidence of supraventricular extrasystoles, particularly at the end of the dives – Sec. to cardiac dilatation**
- **Diving response is acknowledged as an oxygen-conserving reflex in diving species that maintains perfusion to the brain, reduces cardiac work and blood flow to viscera and muscle and, thus, limits overall oxygen consumption**
- **In humans – Less eff. Reduction in CO – Increased BP – Increased Metabolic rate**
- **Splenic contraction – Increased PCV – Prolongs apnoea times.**

# High altitude related pulmonary disease

**Marco polo 1272 – “ the headache mountains”**

**Spectrum of High altitude pulmonary illness**

- 1. AMS , HACE**
- 2. HAPE**
- 3. PH from chronic hypoxia**
- 4. CMS**

# Pulmonary physiology at height

- **Ventilatory adaptation - Hypoxic ventilatory response (HVR)**  
– Measurable
- **Minute ventilation is plotted against the PaO<sub>2</sub> or arterial oxygen saturation.**
- **Acute response in ventilation to hypoxia is blunted by repository alkalosis, but the carotid body continues to undergo adaptation.**
- **Net result - ↑ Ventilation at any PaO<sub>2</sub>.**
- **Related to climbers' success at climbing to extreme altitudes**

***Greater heights come for a  
greater price***

***Increased work of  
breathing for any given  
level of work , greater the  
ascent***

# Acute mountain sickness (AMS)

- Cause – Hypobaric hypoxia
- Incidence – 25 % of sojourners at 8000ft (2440 m)
  
- At 2400-3100 m , SpO<sub>2</sub> – 92-93 %.
- At 3660 m , SpO<sub>2</sub> – low 80's
  
- Sleeping at high altitudes – Nocturnal periodic breathing – 5-8 % decline in saturation
  
- Fact known to mountaineers – “ climb high sleep low”
  
- At himalayan trekking altitudes, (3000-4500m) Incidence of AMS approx. 50 %



# Features of AMS

- Onset within 24 hours , usually first few hours.
- Hallmark symptoms – Neurologic
- Lake Louise scoring system
- Headache and at least one of – GI complaints (anorexia ,nausea & vomitings) , lightheadedness, insomnia, and malaise.
- Pathogenesis – Cerebral edema appears to be the cardinal event
- HACE – Severest form of AMS
- Reascent AMS & Reascent HACE occur – Acclimatization is not permanent (Hypoxia induced gene expression)

# Management of AMS

- **Most patients - Mild to moderate symptoms that abate in 2–3 days without specific treatment.**
- **Mild to moderate AMS - Several therapies are efficacious in hastening recovery (acetazolamide, dexamethasone, oxygen, and descent)**
- **Severe symptoms/HACE - Transport to lower altitudes.**
- **Those who develop AMS - susceptible and usually have a recurrence with future ascents unless they take preventative measures.**
- **AMS rarely results in death, unless evacuation of rare patients who have HACE to lower altitude is not possible.**

# HAPE (High altitude pulmonary edema)

- **Symptoms at least 2 of 4: Dyspnea at rest, cough, weakness or decreased exercise performance, and chest congestion or tightness.**
- **Diagnosis of HAPE - At least 2 of 4 criteria: (1) crackles or wheezing, (2) central cyanosis, (3) tachypnea, and (4) tachycardia.**
- **The incidence of HAPE (0.1 – 1 %) is lower than that of AMS.**
- **HAPE usually occurs within the first 5 days (Not on the first day)**

# Pathogenesis

- **Most compelling theory - PAH with uneven hypoxic vasoconstriction leading to focal areas of pressure and flow-related vascular shear and overperfusion.**
- **Endothelial failure, capillary leak, and eventual noncardiogenic pulmonary edema .**
- **Acute PAH is a central feature of HAPE and resolves with oxygen and descent.**
- **HAPE easily can result in death if descent and adequate oxygen cannot be provided.**
- **Effective preventive treatments available – Oral Nifedipine & Inhaled Salmeterol**

# Chronic mountain sickness

- **CMS has a more important human impact worldwide than AMS and HAPE.**
- **Sequelae of polycythemia, fatigue, and right heart failure from pulmonary hypertension in adulthood after decades of life in a hypoxic environment.**
- **High-altitude dwellers sustain distinct illnesses related to chronic hypoxia, such as having low birthweight children.**
- **After a period at low altitude, some high altitude residents also experience more severe PH, even the development of HAPE, on returning to their usual high-altitude residence.**
- **Acclimatization is not permanent.**

# Lung in Space

# Making measurements in Microgravity

*Parabolic flight in aircraft*

*Adv. – Accesible / Inexpensive*

*Disadv. – Short periods of  
microgravity interspersed with  
hypergravity*

*Motion sickness*

# Space shuttle & ISS

**US led ISS – Research friendly environment**

**Spacelab / Spacehab**

**Problems –  
Small study population**

**Limited time for experiments**

**Absence of experimenter**

**High maintenance requirements**

**Studies require design so viable results with  $n=4$  or  $3$**

**Subjects serve as their own controls**

Analogs of microgravity – HDT , Thermoneutral water immersion  
Do not adequately simulate microgravity



# Lung volumes & Chest wall mechanics in space – Vital Capacity

**Changes  
likely due to  
changes in  
intrathoracic  
blood volume**

- **FRC**

- **$\mu\text{g}$  reduces FRC by approx. 10 %.**

- **Mainly due to cranial shift of diaphragm & abdominal contents when gravity removed.**

- **RV**

- **In sustained  $\mu\text{g}$  , RV shown to ↓ by app. 18 %.**

- **Plausible cause - Large apicobasal gradients in regional lung volume present in 1 G, are abolished in  $\mu\text{g}$ .**

- **Reduction in MEFV & PEFV 's**
- **Lack of a firm platform to push against during the maneuver , recovery later - may be the result of improved subject performance as they adapted to  $\mu g$**
  
- **Shape & movement of the chest wall**
- **Inward displacement of the abdominal wall seen in  $\mu g$  causing a reduction in lung volume**
- **Increase in abdominal wall compliance increase in the abdominal contribution to TV from 33% in 1 G to 51% in  $\mu g$ .**
  
- **Diffusing capacity**
- **Substantial  $\uparrow$  by 28% on exposure , remained elevated over the course of 9-day flight.**
- **Due to parallel increase in pulmonary capillary blood volume ( $V_c$ ) & membrane-diffusing capacity ( $D_m$ ).**
- **Attributed to transition of the lung from its zone 1, 2, 3 configuration to an entirely zone 2 or zone 3 – more uniform filling.**

# Pulmonary perfusion

- **No direct measurements of the distribution of pulmonary blood flow during space flight.**
- **Radioactively labeled microaggregated albumen- some increase in apical blood flow compared with the upright position in 1 G.**
- **Carbon dioxide expirogram - Cardiogenic oscillations & terminal fall in CO<sub>2</sub> – Indicators of inhomogeneity of perfusion**

- **The ventilatory response to exercise largely is unaffected by  $\mu\text{G}$ .**
- **Cardiac output increase with increasing  $\text{VO}_2$  is substantially lower than that measured upright or supine on the ground preflight.**
- **Ventilatory control - Substantial changes in the ventilatory response to hypoxia (Nearly halving of hypoxic drive)**
- **Ventilatory response to carbon dioxide essentially unchanged**
- **Given the magnitude of the reduction in hypoxic response in mG and supine, it is interesting to speculate on the possible role this may have in some hypoventilation syndromes and their postural components.**

- **Increased BP at the level of the carotid bodies.**
- **In the upright position in 1 G, BP at the carotid bodies is lower than at heart level because of the hydrostatic pressure difference.**
- **Difference is abolished in the supine position and in  $\mu$ G.**
- **In dogs, it is established that stimulation of the carotic baroreceptors results in an inhibition of the carotid chemoreceptor output**

# **Sleep in space**

**Significant ↓ in AHI and time spent snoring**

**Demonstration of gravitational component of OSA**

**Marked reduction in Respiratory related arousals**

**Conclusion – Poor sleep in space flight not related to respiratory system**

# Extravehicular activity (EVA)

- **Space Shuttle and the ISS - 760 mm Hg breathing air environment.**
- **Need for mobility - Space suits operate at a much lower pressure with a 100% oxygen environment .**
- **Denitrogenation procedure must be performed before an EVA (220 mm Hg United States space suit).**
- **A direct transition from cabin pressure to suit pressure would result in decompression sickness (DCS).**
- **To date, no astronaut formally has reported DCS during EVA**
- **Presence of VGM - More than 50% of the participants had detectable bubbles, as indicated by Doppler ultrasound.**



# Lung physiology in Antarctica

## CHANGES IN RESPIRATION DURING ACCLIMATIZATION IN THE INTERIOR OF ANTARCTICA

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The climate of the interior of Antarctica combines the characteristic features of a high-mountain climate with the specific polar conditions. The effect of this combination of man has not been discussed in the literature, although fairly detailed studies have been made of the action of both high-mountain and polar conditions separately. An increase in the rate and depth of respiration has been found at high altitudes [1, 9, 14, 16]. Most investigators have observed hyperventilation within a few minutes of exposure to hypoxia. Z. I. Barbashova [4] and N. K. Vereshchagin and V. B. Boldyrev [6] consider, however, that the increase in pulmonary ventilation develops very gradually during acclimatization to high altitudes. Hyperventilation usually does not develop before a certain threshold altitude has been reached: a height of 3000 m is usually mentioned [3, 8, 11, 13]. N. I. Averin [2] found no significant changes in pulmonary ventilation in persons living at an altitude of 2000 m. Investigation of the alveolar air revealed a considerable decrease in the CO<sub>2</sub> and oxygen pressure, as a result of which the blood oxygen saturation was lowered [7, 8, 15, 19]. Harvath and co-workers [17] found an increase in pulmonary ventilation during exposure to cold. During the polar night, Lindhard [18] recorded a decrease in pulmonary ventilation of persons wintering in the Arctic, associated with a fall in the respiration rate and an increase in the partial pressure of carbon dioxide in the alveolar air.

### EXPERIMENTAL METHOD

- **Observations made at the Soviet antarctic station, Vostok.**
- **Tachypnea and considerable hyperventitation during the first days after arrival.**
- **With acclimatization, the frequency of respiration normal, depth of resp. increased.**
- **Pulmonary ventilation decreased, lowest level during the polar night period (Still 1 1/2 times greater than the normal value).**
- **Every individual had Cheyne-Stokes respiration, especially during sleep.**
- **The blood oxygenation during quiet respiration was 80–87%, increasing with voluntary hyperventilation to 85–94%.**
- **Hypoxemia rose considerably with voluntary breath holding and physical strain accomp. with dyspnoea**

## **Respiratory changes due to extreme cold in the Arctic environment**

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- **Healthy tropical Indian men (n= 10).**
- **Baseline recordings at Delhi.**
- **Initial decrease in VC, FVC, FEV1, PEFV and MVV on acute exposure to cold stress**
- **Gradual recovery during acclimatisation for 4 weeks and a further significant improvement after 9 weeks of stay at the arctic region.**
- **On return to India all the parameters reached near baseline values except for MVV which remained slightly elevated.**
- **Respiratory responses during acute cold exposure are similar to those of initial altitude responses.**

# Summary

- **Humans are exposed to a number of extreme environmental stresses and lung is a major organ affected.**
- **Understanding of physiological response of the respiratory system to these stresses is important.**
- **It can help in prevention and timely treatment of illnesses resulting due to exposure to extreme environmental stresses.**