Hypobaric & Hyperbaric Conditions

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This Presentation is Approved for
1 CRCE Credit Hour

Learning Objectives

- Apply the gas laws to hyperbaric and hypobaric conditions
- Explain the effects of high altitude on human physiology
- Discriminate between the following high altitude illnesses
  - Acute mountain sickness (AMS)
  - High altitude cerebral edema (HACE)
  - High altitude pulmonary edema (HAPE)
  - Chronic mountain sickness (CMS)
- Explain the pathophysiology of high altitude illnesses
- Describe the signs and symptoms of high altitude illnesses

Learning Objectives

- Recommend preventative measures for high altitude illnesses
- Recommend management strategies for high altitude illnesses
- Explain the pathophysiology of decompression sickness (DCS) and arterial gas embolism (AGE)
- Describe the signs and symptoms of DCS and AGE
- Recommend preventative measures for DCS and AGE
- Recommend management strategies for DCS and AGE

Learning Objectives

- Explain the rationale and effects for hyperbaric oxygen therapy (HBOT)
- State the indications and complications for HBOT
- Describe general procedures and devices applied to HBOT
- Explain technical points pertaining to HBOT and respiratory therapeutics
- Describe the risks to caregivers associated with administration of HBOT

Gas Laws

Pertinent Gas Laws

- Boyle's law – relationship between volume and pressure
- Henry's law – solubility of gases in liquids
- Dalton's law – law of partial pressures

Boyle's Law

- For a given mass of gas at a constant temperature, the volume times the pressure equals a constant
  - PV = Constant
  - $P_1V_1 = P_2V_2 \rightarrow V_2 = P_1V_1/P_2$

Boyle's Law

- The volume is inversely proportional to the pressure →
  - Increasing pressure → decreases volume
  - Decreasing pressure → increases volume

FYI see link below for a demonstration of Boyle's law

Boyle's Law

- Pressure is measured in atmospheres (Atm) or absolute (ATA)
- 1 ATA = 760 mm Hg = 14.7 psi
- A balloon contains 1.0 L at 1.0 ATA
  - At 0.5 ATA, the V changes to 2.0 L
  - At 2.0 ATA, the V changes to 0.5 L

Depths, Altitude, & Ambient Pressure

- Each 33 ft underwater = 1.0 Atm → @ 33 ft = 2 ATA (absolute) = 1,520 mm Hg
- 19,000 ft = 0.5 ATA = 380 mm Hg
- Mt. Everest summit = 0.33 ATA = 250 mm Hg
- 100,000 ft approaches zero ATA (FO_2 remains 0.21)

FYI see link below for a chart with altitudes and pressure

Boyle's Law

- So?
  - 6 L lung volume compresses to 3 L at 33 ft depth
  - This would reverse on rapid ascent → diver holds breath during ascent and lungs burst from volutrauma
  - Gas bubble increase in volume → 'bends'

Boyle's Law

- Question: The limit to the length of a snorkel for underwater breathing is about 40 cm (16 in). What is the basis for this limit?

Boyle's Law

- Question: The limit to the length of a snorkel for underwater breathing is about 40 cm (16 in). What is the basis for this limit?
- Answer: The pressure surrounding the chest at greater depth makes the work of breathing unsustainable
  - e.g. at 1.0 m, the pressure is about 100 cm H2O
  - With SCUBA gear, the pressure equalizes

Henry's Law

- The amount of any given gas that will dissolve in a liquid at a given temperature is a function of the partial pressure of the gas that is in contact with the liquid and the solubility coefficient of the gas in the particular liquid.

Henry's Law

- Examples
  - PaO_2 x 0.003 = dissolved O_2 →
  - 100 mm Hg x 0.003 = 0.3 mL/dL
  - 2,183 mm Hg x 0.003 = 6.5 mL/dL

FYI see link below for more information on Henry's law

Henry's Law

- So what?
  - Hyperbaric oxygen increases dissolved O_2 available to tissues that are not perfused
  - Nitrogen dissolved under hyperbaric conditions produces bubbles during decompression

Dalton's Law

- The total pressure in a gas mixture equals the sum of the partial pressures of the gases
- Alveolar air equation (clinical) $PAO_2 = FO_2 (Pb - 47) - (PaCO_2 \times 1.25)$

FYI see link below for more on Dalton's law

Alveolar Air Equation

- At 1.0 ATA a person has a normal PAO_2 = 100 mm Hg (FiO_2 = 0.21)
  - At 6,900 ft Pb → 580 → PAO_2 = 0.21 (580 – 47) – (40*1.25) = 62 mm Hg
  - At 2 ATA Pb = 1,520 → PAO_2 = 0.21 (1520 – 47) – (40*1.25) = 259 mm Hg

High Altitude Physiology & Acclimatization

High Altitude (HA)

- High altitude: 1,500 to 3,500 m (4,921-11,483 ft) – high-altitude illness common with abrupt ascent to above 2,500 m (8,202 ft)
- Very high altitude: 3,500 to 5,500 m (11,483-18,045 ft) – most common range for severe high-altitude illness

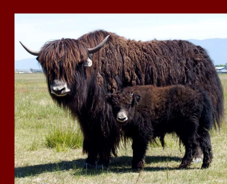
Note: 1 meter = 3.28 feet

High Altitude (HA)

- High altitude: 1,500 to 3,500 m (4,921-11,483 ft) – high altitude illness common with abrupt ascent to above 2,500 m (8,202)
- Very high altitude 3,500 to 5,500 m (11,483-18,045 ft) – most common range for severe high-altitude illness
- Extreme altitude: 5,500 to 8,850 m (18,045-29,035 ft) – progressive deterioration of physiologic function eventually overcomes acclimatization

High Altitude (HA)

- Highest permanent habitation – La Rinoconada, Peru (5,100 m)
- Lhasa, Tibet (3,650 m)



Living at High Altitude

- Acclimatization – changes within an individual to live at HA
- Adaptation – genetic changes in populations to live at HA (generations living at HA)
  - Indigenous Andeans – minimal, if any adaptation
  - Indigenous Tibetans – adaptations, e.g. normal pulmonary artery pressure

High Altitude Alterations

- Stimulus for all mechanisms is hypobaric hypoxia
- Hypoxic ventilatory response (HPV)
  - Peripheral chemoreceptors adjust ventilation for increased PaO_2
  - Occurs immediately
  - Capability to withstand extreme hypocapnea is one form of acclimatization

High Altitude Alterations

- HbO_2 dissociation curve
  - Hypocapnea shifts curve to left
    - Increases alveolar O_2 uptake
    - Inhibits release of O_2 to tissues
  - Increased production of 2,3 DPG shifts the curve rightward, increasing release of O_2 to tissues – partial compensation for hypocapnea

Pearl: One Everest climber developed a P50 = 19 mm Hg (normal = 27 mm Hg)

High Altitude Alterations

- Acid-base balance
  - Hypocapnea ➔ respiratory alkalemia
  - Chronic hypocapnea causes kidneys to excrete HCO_3- to balance Ph – e.g.: pH = 7.45; PCO_2 = 28; HCO_3 = 19; Base change = -5 mEq/L
  - Return to normal PCO_2 causes acidemia and hyperventiation until HCO_3- is retained

High Altitude Alterations

- Cardiovascular changes
  - Heart rate and cardiac output
    - Initial increase in heart rate and cardiac output
    - Resting heart rate returns toward normal over time
  - Pulmonary vasoconstriction ➔ pulmonary hypertension

High Altitude Alterations

- Cerebral circulation
  - Hypobaric hypoxia increases cerebral blood flow
  - Hypocapnea decreases cerebral blood flow
  - Cognitive impairment begins at 2,500 m

FYI see link below to view videos of high-altitude training for pilots

High Altitude Alterations

- Hematology – hypoxia stimulates erythropoietin release, which increases RBC production
  - Begins after 2 H at altitude
  - Increases oxygen content

Pearl: Theoretically, genetic variations that permit survival at high altitude also improve outcomes in critical illness

High Altitude Alterations

- Peripheral tissues – increased myoglobin
  - Increases diffusion of O_2 to muscles
  - Additional reservoir for oxygen

FYI see link below to download articles on high-altitude physiology

High Altitude Illness

Conditions

- Acute mountain sickness (AMS)
- High altitude cerebral edema (HACE)
- High altitude pulmonary edema (HAPE)
- Chronic mountain sickness (CMS)

FYI see link below to download an article on high-altitude illness

AMS/HACE

- Acute mountain sickness (AMS) and high altitude cerebral edema (HACE) – same pathophysiology, different levels of severity
- Etiology – abrupt ascent to altitude >2,500 m (8,200 ft)
- FYI – Vail, Colorado – 2,484 m (8,150 ft)

AMS/HACE

- Risk Factors
  - Rate of ascent
  - Altitude for sleep
  - Individual susceptibility
  - Preexisting cardiopulmonary disease
  - Physical exertion
  - Obesity

AMS/HACE

- No gender differences in susceptibility
- Neither youth nor physical fitness confer protection

AMS/HACE

- Pathophysiology – unclear, but elements include
  - Regional cerebral edema
  - Increased intracranial pressure
  - Cerebral vasoreactivity
  - Cerebral vascular leakage

AMS/HACE

- Symptoms (AMS) – occur 6 – 36 H after ascent
  - Headache
  - Dizziness
  - Disturbed sleep
  - Anorexia, nausea, vomiting
  - Fatigue
  - Shortness of breath
  - Malaise

FYI see link below to view Lake Louise altitude illness scoring

AMS/HACE

- Symptoms (HACE)
  - Change in mental status, e.g. confusion
  - Photophobia
  - Hallucinations
- Signs (HACE)
  - Ataxia (discoordination)
  - Coma
  - Can cause death from brain herniation

AMS/HACE & Pediatrics

- Pediatric Assessment
  - Infant fussiness
  - Appetite, vomiting
  - Playful activity
  - Afternoon nap

FYI see link below for an article on pediatric assessment for AMS/HACE

Cardiopulmonary Conditions & HA

- COPD
  - Altitude worsens hypoxemia
  - Altitude does NOT adversely affect lung mechanics
  - Baseline PaO_2 = 73 mm Hg required for 2,300 m (commercial airline cabins)
  - Patients with FEV_1 < 1.5 L may require supplemental O_2

Cardiopulmonary Conditions & HA

- Asthma
  - Decreased house mite load
  - Air quality can be worse, e.g. diesel exhaust and yak dung fire smoke
  - Hypoxemia can cause bronchospasm
  - Severe asthmatics ascend with caution

FYI see link below to download an article on high altitude and pre-existing lung disease

Cardiopulmonary Conditions & HA

- Pulmonary hypertension worsens
- Patent foramen ovale (PFO)
  - Predisposed to high-altitude pulmonary edema
  - Worsens hypoxemia, due to right-to-left shunt

Cardiopulmonary Conditions & HA

- Obesity hypoventilation – advice against high-altitude travel
- Obstructive sleep apnea – take CPAP
- Persons with migraine headaches may require slower ascent

AMS/HACE

- Prevention
  - Gradual ascent (>2,500 m or 8,000 ft)
    - ≤ 300 m/day
    - Rest day Q 2 – 3 D
    - No further ascent for symptomatic persons

AMS/HACE

- Prevention
  - Acetazolamide (Diamox) – carbonic anhydrase inhibitor
    - Ventilatory stimulant
    - Prevents sleep aplea
    - Mimics/hastens acclimatization
    - Makes carbonated beverages taste bad (including beer)

AMS/HACE

- Prevention
  - Acetazolamide (Diamox) – carbonic anhydrase inhibitor
    - Ventilatory stimulant
    - Prevents sleep aplea
    - Mimics/hastens acclimatization
    - Makes carbonated beverages taste bad (including beer)
  - Dexamethasone (Decadron) – believed to minimize vascular leakage
  - Ginkgo – is NOT effective

AMS/HACE

- Treatment
  - Descent
    - >500 m (1,600 ft)
    - Problematic for back-country trekkers
  - Acetazolamide (Diamox)
  - Dexamethasone (Decadron)
  - Theophylline (under study)

High Altitude Pulmonary Edema (HAPE)

- Occurrence
  - ≥ 3,000 m
  - 2 – 4 days after ascent
  - May be preceded or accompanied by AMS

HAPE

- Pathophysiology
  - Hypoxia – accentuated pulmonary vascular response → worsened pulmonary hypertension
  - Heterogeneous stress failure of pulmonary microvascular endothelium, causing fluid leak into alveoli

HAPE

- Manifestations – progressive
  - Initial nonproductive cough
  - Progressive dyspnea
  - Tachypnea
  - Tachycardia

HAPE

- Manifestations – progressive
  - Production of pink, frothy sputum
  - Crackles
  - Severe hypoxemia
  - Patchy infiltrates on chest X-ray
  - Lethargy
  - Coma
  - Death

See link below to view radiograph of HAPE

HAPE

- Susceptibility increased by
  - Male gender
  - History of HAPE
  - Patent foramen ovale
  - Pulmonary vascular disease

HAPE

- Prevention and treatment
  - Precautions, as for AMS/HACE, e.g. graded ascent
  - Pulse oximetry
    - Very high altitudes
    - Susceptible individuals
  - Immediate descent

HAPE

- Prevention and treatment
  - Precautions, as for AMS/HACE, e.g. graded ascent
  - Pulse oximetry
  - Diamox – reverses pulmonary hypertension
  - Ca++ channel blocker, e.g. nifedipine (Procardia) – reverses pulmonary hypertension
  - Phosphodiesterase inhibitor, e.g. tadalafil (Cialis) = reverses pulmonary hypertension

HAPE

- Prevention and treatment
  - Decadron – stabilizes capillary endothelium
  - Inhaled beta agonists, e.g. salmeterol – high doses increase clearance of alveolar fluid
  - Oxygen
  - CPAP
  - Hyperbaric oxygen (portable chamber)

Chronic Mountain Sickness (CMS)

- AKA Monge's disease
- Occurs in high altitude natives or long-term residents (>2,500 m)
- Higher altitude ➔
  - Greater prevalence
  - Greater severity

Chronic Mountain Sickness (CMS)

- Categories
  - Primary CMS – acclimatized individuals who develop idiopathic CMS
  - Secondary CMS – individuals with conditions, e.g. obesity, neuromuscular disorders, chronic lung disease

Chronic Mountain Sickness (CMS)

- Pathophysiologic Components
  - Excessive erythrocytosis (Hct >58%)
  - Relative hypoventilation
  - Exaggerated hypoxemia
  - Pulmonary hypertension, leading to cor pulmonale

Chronic Mountain Sickness (CMS)

- Symptoms
  - Dyspnea
  - Reduced exercise tolerance
  - Headache
  - Anorexia
  - Burning palms, plantar surfaces
  - Muscle & joint pain
  - Inability to concentrate
  - Memory loss

Chronic Mountain Sickness (CMS)

- Signs
  - Excessive erythrocytosis
    - Hb females >19 g/dL
    - Hb males >21 g/dl
  - Severe hypoxemia, cyanosis
  - Pulmonary hypertension, which may result in cor pulmonale

Chronic Mountain Sickness (CMS)

- Management
  - Descent – permanent
  - Supplemental oxygen
  - Acetazolamide (Diamox)
  - Phlebotomy (by Vampires?)

Chronic Mountain Sickness (CMS)

- Management
  - Antihypertensives (studies needed)
    - Ca++ channel blockers (nefedipine)
    - Phosphodiesterase inhibitors (Cialis)
    - Endothelin antagonists (bosentan)
    - Prostacyclins (Flolan, Ventavis)
    - Nitric oxide

FYI see link below to download an article on medial advice for commercial air travelers

Decompression Sickness & Arterial Gas Embolism

Decompression Sickness (DCS)

- Rapidly decreased ambient pressure allows dissolved N_2 to leave solution and form enlarged bubbles in circulation
  - Henry's law – N_2 leaves solution
  - Boyle's law – bubble enlargement

Decompression Sickness (DCS)

- Contexts:
  - Underwater diving – bends
  - Underground construction – caisson disease
  - Aircraft at altitude – loses cabin pressure – altitude DCS
  - Hyperbaric chambers

FYI see link below for a joke about mine workers

Decompression Sickness (DCS)

- Physical factors
  - Depth (determines pressure)
  - Time at depth
  - Time for decompression
  - Altitude, e.g. mountain lakes, caves

Decompression Sickness (DCS)

- Pathophysiology – N_2 bubbles cause physical and biochemical damage to tissues
  - Accumulate in join capsules & muscles
  - Obstruct blood flow to spinal cord
  - Endothelial damage activates leukocytes and platelets →
    - Inflammation
    - Coagulopathy

Decompression Sickness (DCS)

- Predisposing factors
  - Fatigue
  - Obesity
  - Dehydration
  - Hypothermia
  - Female gender

Decompression Sickness (DCS)

- Predisposing factors
  - Increased age
  - History of DCS
  - Recent alcohol use
  - Flying within 24 H after diving (altitude DCS)
  - Cardiovascular shunt, e.g. PFO

Decompression Sickness (DCS)

- Manifestations
  - Bends – pain in large joints
  - Chokes – cough, substernal pain
  - Skinny bends – cutaneous, itchy rash
  - Lymphedema

Decompression Sickness (DCS)

- Manifestations – spinal cord DCS
  - Ascending paresthesia (tingling)
  - Ascending paralysis
  - Loss of bowel and bladder control

Arterial Gas Embolism (AGE)

- Pathophysiology
  - Rapid decompression
  - Alveolar gas expands and ruptures pulmonary vessels
  - and/or passes through PFO, then
  - Gas bubbles enter systemic circulation

See link below for an illustration of AGE

Arterial Gas Embolism (AGE)

- Pathophysiology
  - Blockage of arteries → distal ischemia
  - Bubbles cause cellular damage → leukocyte activation →
    - Edema
    - Coagulopathy → focal hemorrhages
    - Increased permeability of blood-brain barrier

Arterial Gas Embolism (AGE)

- Manifestations (sudden onset)
  - Bloody froth from mouth, nose
  - Marbling of skin
  - Headache
  - Confusion
  - Sensory deficits
  - Motor deficits
  - Convulsions (worst case)
  - Coma (worst case)
  - Death (worst case)

DCS & AGE Prevention

- Predive medical clearance
- Slow ascent – one-half the rate of the smallest bubbles
- Breathing evenly during ascent – avoid breath holding
- No flying for 12 – 24 H after dives

FYI see link below for an article on scuba diving health

DCS & AGE Management

- Basic life support
- Transport – low-flying craft
- Oxygen
- Recompression (hyperbaric chamber)
- Staged decompression

Hyperbaric Oxygen Therapy (HBOT)

HBOT Definition

- The patient intermittently breathes 100% O_2 in a chamber pressurized to greater than 1.0 ATA

Actions (Rationale)

- Increased ambient pressure
  - Dissolves N_2 bubbles in tissues
  - Shrinks gas bubbles
  - Increases PO_2 in all tissues, e.g. at FiO_2 = 1.0 and 3 ATA, the PaO_2 = 2,100 mm Hg → dissolved O_2 = 6.3 mL/dL

Effects

- Promotes genesis of new blood vessels (speeds wound healing)
- Kills some anaerobes
- Prevents growth of species, e.g. pseudomonas
- Prevents production of clostridial alpha toxin (gangrene)

Effects

- Increases bacteriocidal effectiveness of WBCs
- Reduces WBC adhesion in reperfusion injury, preventing release of proteases and free radicals

Indications

- Strong evidence – main treatment
  - Decompression sickness
  - Arterial gas embolism
  - Severe CO poisoning

Indications

- Strong evidence as adjunctive treatment
  - Prevention and treatment of radionecrosis
  - Improved skin graft and flap healing
  - Clostridial tissue infections

Indications

- Some evidence
  - Refractory osteomyelitis
  - Acute traumatic ischemic injury
  - Prolonged failure of wound healing
    - Diabetic ulcers
    - Thermal burns
    - Crash injury
    - Skin grafts
    - Sternal wound infections

Indications

- Some evidence
  - Severe anemia
  - Autism
  - Cirrhosis
  - Stroke
  - Intracranial abcess
  - Invasive fungal infections, e.g. aspergillus
  - Cerebral palsy

Complications

- Fire hazard
- Claustrophobia
- Near-sightedness (reversible)
- Barotrauma – ear damage
- Oxygen toxicity – brain and lung
- Pulmonary edema
- DCS, AGE

FYI see link below for an article on HBOT

HBO Chambers

- Monoplace – one patient
  - Greater claustrophobia
  - Portable
- Multiplace – more than one patient
  - Chamber compressed with air – less fire hazard
  - O_2 administered via mask, ventilators

See links below to view monoplace HBO chamber and a virtual tour of a multiplace HBO chamber

Procedures

- Parameters
  - Pressure – ATAs
  - Duration of sessions
  - Number of sessions
- Parameters vary by condition treated
  - AGE – up to 6 ATA
  - DCI – 2-4 H @ 2.5 – 3.0 ATA
  - Wound healing – 1.5 H @ 2-3 ATA for multiple treatments

Technical Points

- O_2 toxicity decreased by intermittent changes to room air breathing
- Tube cuffs inflated with fluid
- IV infusion pumps lose accuracy in chambers
- Ventilator volume delivery is affected by pressure in chamber

Risk for Personnel

- Cerebral oxygen toxicity – seizures
- DCS
- Preventative measures
  - Assessment for fitness to dive
  - Adhering to decompression schedule
  - Breathing O_2 during decompression
  - Diving chamber time among attendants
  - Avoid flying after HBO

Summary & Review

- Gas Laws
  - Boyle's law – volume and pressure
  - Henry's law – pressure and dissolved gas contents
  - Dalton's law – pressure and partial pressure

Summary & Review

- Physiologic responses – driven by hypobaric hypoxia – diminishing pressure with altitude
  - Hypoxic ventilatory response
  - HbO_2 curve shifts with hypocapnea and 2,3 DPG
  - Acid-base balance – compensated respiratory alkalemia
  - Pulmonary hypertension
  - Erythropoietin – increased RBCs

Summary & Review

- High altitude illnesses
  - Acute mountain sickness
  - High altitude cerebral edema
  - High altitude pulmonary edema
  - Chronic mountain sickness

Summary & Review

- Decompression sickness
- Arterial gas embolism

Summary & Review

- Hyperbaric oxygen therapy
  - Actions – gas laws
  - Effects
  - Indications
  - Complications
  - Technical aspects – chambers, etc.
  - Risks to personnel

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