Barometrics: Altitude and Exercise & Dive Medicine

The Sports Medicine Core Curriculum Lecture Series
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Altitude & Exercise

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What is high altitude?

High Altitude: 1500 - 3500 m (5000 - 11500 ft)
Very High Altitude: 3500 - 5500 m (11500 - 18000 ft)
Extreme Altitude: above 5500 m (Mt. Everest 8848m)
Altitude affects performance

1968 Olympics Mexico City (2300m)
1974 International Federation of Sports Medicine bans competition >3050m
Acute Physiologic Response at Altitude

- Partial pressure oxygen decreases
- Arterial SaO2 decreases
- Compensation:
  - hyperventilation
- Respiratory alkalosis

- Shifts oxy-Hemoglobin curve
Physiologic Response to Hypoxia

Hyperventilation
Hematopoiesis (epo)
Tachycardia
RAA axis: Na+ retention
ANP Na+ diuresis
ACTH, secretion cortisol
Insulin sensitivity

Pulmonary hypertension
(increases w/ exercise)
EKG: R axis deviation,
T inversion
Decreased intercapillary
distance
increased capillaries
loss muscle fiber size
Physiologic Response to Hypoxia

Psychomotor performance decline

No change:
- DLCO
- Blood pressure
- ADH
Acclimatization

>3000m, 6 weeks
Hematocrit increases
Red cell mass increases (4 wks)
Muscle-capillary density increases
Mitochondrial adaptation
Renal bicarbonate excretion normalizes pH
Diet and Altitude

High carbohydrate, low fat
Carbs use less oxygen to produce energy
Incr respiratory quotient, for a given PCO2
the PO2 is higher
Greater exercise endurance
Effect of lowering 2000 ft
Athletic Performance at Altitude

Reduction PpO2
Increased fatigue
VO2 max decreases
  10%/1000m
Max work rate decreases
Max exercise HR decreases
Endurance time decreases
Athletic Performance and Altitude

Aerobic: decreased performance
Anaerobic: unaffected
Sprinting: improved, decreased air density
“Live High, Train Low”

Prolonged altitude exposure:
  Increased erythropoietin, hematocrit, red cell mass, capillary density
Low altitude training:
  Rigorous intensity
“Live High, Train Low”

*Levine and Stray-Gundersen*

- live @ 2500m
- train @ 1250m
- randomized
- 39 competitive runners

**Results:**
- altitude increased red cell mass 9%
- altitude increased VO2max 5%
- only live high/train low improved 5K time 22.7s
Altitude CONS

Decreased VO2max
Decreased training intensity
Dehydration
Polycythemia
Risk Acute Mountain Sickness
Retinal hemorrhages (50% >5000m)
Thrombosis (PE, CVA)
Altitude PROS

- Increased hematocrit
- Increased red cell mass
- Increased ventilatory capacity
- Increased capillary density
Symptoms at Altitude

Dyspnea on exertion
Increased urination
Periodic breathing
Edema
Hyperventilation
Inadequate hypoxic ventilatory response
When $P_aO_2$ declines to 60mmHg →
stimulate chemoreceptors in carotid body →
immediate increase minute ventilation to
correct the PaO2 to sea level values
Unfortunate side effect: PCO$_2$ drops → resp alkalosis →
inhibit peripheral and central chemoreceptors →
decrease ventilatory drive
This leads to acid-base shifts in the CNS
Increase in cerebral blood flow and pulmonary arterial pressure
The increase in cerebral blood flow is offset by
the vaso-constrictive effect of hypocapnea.
Periodic breathing

Prominent during sleep
Apnea lasts 3-15 seconds
Not associated w/ altitude illness
12% at 2400m Colorado
67% at 3000m
Rx: Acetazolamide (Diamox) 125 mg po qhs
Insomnia

Secondary to cerebral hypoxia

Rx: Acetazolamide acts as respiratory stimulant
Edema at Altitude

Peripheral and facial
Not equivalent to Acute Mountain Sickness
No contraindication to ascend

Rx:
   Acetazolamide
   Lasix
   Descent
Acute Mountain Sickness

Spectrum of disease
Exercise exacerbates AMS
Self limiting 24-48 hr
AMS-Incidence

<table>
<thead>
<tr>
<th>Altitude (m)</th>
<th>Incidence (%)</th>
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<tbody>
<tr>
<td>2850</td>
<td>9</td>
</tr>
<tr>
<td>3050</td>
<td>13</td>
</tr>
<tr>
<td>3650</td>
<td>34</td>
</tr>
<tr>
<td>4550</td>
<td>53</td>
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Higher if abrupt exposure (flying in) v. walking up
AMS Definition

1991 International Hypoxia Symposium Lake Louise, Alberta

Setting: Gain in altitude >2400m

Lake Louise scoring
  mild
  moderate (>4)
  severe (>8)
AMS

S/S: Headache + (1 or more):
  GI upset (loss of appetite, nausea, vomiting)
  fatigue/weakness
  dizziness/light-headedness
  insomnia (more than just the usual frequent waking)
  anorexia
## Lake Louis AMS Questionnaire

1. **Headache:**
   - No headache 0
   - Mild headache 1
   - Moderate headache 2
   - Severe, incapacitating 3

2. **GI:**
   - No GI symptoms 0
   - Poor appetite or nausea 1
   - Moderate nausea or vomiting 2
   - Severe N&V, incapacitating 3

3. **Fatigue/weak:**
   - Not tired or weak 0
   - Mild fatigue/weakness 1
   - Moderate fatigue/weakness 2
   - Severe F/W, incapacitating 3

4. **Dizzy/lightheaded:**
   - Not dizzy 0
   - Mild dizziness 1
   - Moderate dizziness 2
   - Severe, incapacitating 3
Lake Louis AMS Questionnaire

5. Difficulty sleeping:
   - Slept well as usual 0
   - Did not sleep as well as usual 1
   - Woke many times, poor night's sleep 2
   - Could not sleep at all 3

6. Change in mental status:
   - No change 0
   - Lethargy/lassitude 1
   - Disoriented/confused 2
   - Stupor/semicorconsciousness 3

7. Ataxia (heel to toe walking):
   - No ataxia 0
   - Maneuvers to maintain balance 1
   - Steps off line 2
   - Falls down 3
   - Can't stand 4

8. Peripheral edema:
   - No edema 0
   - One location 1
   - Two or more locations 2
AMS

Dehydration is a common cause of non-AMS headaches

Diagnostic/therapeutic trial:

liter of fluid and take a mild pain-reliever

Symptom-free = acclimatized,

continued ascent OK
AMS

Exceed the "standard" 300 m (1000 ft) sleeping elevation gain per night

Has this patient ascended so rapidly that you should be expecting deterioration?
AMS Medications

**Acetazolamide**
- accelerates acclimatization
- respiratory stimulant
- improves oxygenation

**Dexamethasone**
- suppress AMS
- no improvement acclimatization
- rebound AMS off med
- equivalent to Gamow bag

**Ginko biloba**
# AMS Prophylaxis

<table>
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<tr>
<th>Acetazolamide</th>
<th>Dexamethasone</th>
<th>Ginko biloba</th>
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<tbody>
<tr>
<td>rapid ascent &gt;3000m</td>
<td>4 mg po/IM q 6 x 2</td>
<td>120 mg bid, 5d prior to ascent, continue</td>
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<tr>
<td>gain &gt;1000m sleeping elevation per day</td>
<td>delay ascent until asymptomatic and 18 hr after last dose</td>
<td>50% reduction symptoms/milder S/S</td>
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<tr>
<td>125 mg bid, 24h in advance, discontinue 3rd night at maximal altitude or on descent</td>
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AMS Treatment

Descent
  immediate
Rest at same elevation
  24-48 h asymptomatic
Rest + acetazolamide
  12-24 hr recovery
Rest + dexamethasone
  2-6 hr recovery
Oxygen
  4 L/min, 2 hr recovery, rebound
Hyperbaric oxygen/bag
HACE-High Altitude Cerebral Edema

Mental status changes
Ataxia
Clinical Testing:
walk heel-toe along a straight line
HACE does **not** affect finger-nose tests
Papilledema
Focal neurologic-uncommon
HACE-Treatment

Immediate descent

If unable to descend:

  Dexamethasone
  Portable hyperbaric chamber 4-6 h
  Oxygen

Preventable

  Occurs only if ascent w/ AMS
HACE-Course

Incidence: at 4200m ~1.8%

Untreated: death in hours

Treated: full recovery, no neurologic deficits
HACE Prevention

Slow ascent – the only preventative measure!

Dexamethasone is used, but has not been studied.
High Altitude Pulmonary Edema (HAPE)

Most common fatal manifestation of altitude illness

Occurs among individuals who rapidly ascended above 12,000 feet
It is possible below 8000 feet

Non-cardiogenic pulmonary edema
HAPE

Symptoms (>1):
- dyspnea at rest
- cough
- weakness or decreased exercise performance
- chest tightness or congestion

Signs: (>1)
- crackles or wheezing
- central cyanosis
- tachypnea
- tachycardia (absent 30%)
HAPE

Pathophysiology:
- patchy hypoxic vasoconstriction,
- shunt,
- high pressure vascular leak

SaO2 low
SaO2 may be normal at rest
Provoke w/ exercise: walk 100m
Oxygenation at altitude

Example at 4200m:
- Normal SaO2: 80-86%
- HAPE: 50-60%

>6500m
- increase ventilation
- respiratory alkalosis
- shift hemoglobin dissociation curve
- oxygenation stabilizes
HAPE

Fever is common
Do not misdiagnose as pneumonia
*Exercise intolerance
Cold worsens
Exercise worsens
HAPE can result in HACE
(functional higher altitude w/ lower PaO2)
HAPE

Incidence:

high-altitude ski areas in Colorado:
1/10,000 skier-days

at 4200m ~2.5%
HAPE-Treatment

Descent

Nifedipine: 10 mg po q 4 h; pulmonary vasodilation

Hyperbaric: 2-4 h treatment

Oxygen

Bedrest and oxygen for mild HAPE

Diuretics

Nitric oxide – experimental
Hyperbaric Treatment

HAPE: 2-4 hr

HACE: 4-6 hr

Resolution of symptoms

Improve enough that patient can walk down
HAPE-Prophylaxis

Slow ascent
Nifedipine slow release 20mg po q 8h
Acetazolamide (respiratory stimulant)
Nifedipine
   - Lowers coronary artery systolic pressure and
   - Lowers alveolar/arterial oxygen gradient
Dexamethasone or Tadalafil
   - Lowers pulmonary artery systolic pressure
Salmeterol – B-agonist
   - Increase in fluid transport out of alveoli through Na+ channels
Garlic (experimental)
   - Upregulating Nitric Oxide synthesis
HAPE-Course

Untreated:
   Death in hours

Treated:
   Resolve 24-48 h after descent
   Death 10-15%
Prevention

At altitudes above 3000 meters (10,000 feet):
Sleeping elevation should not increase more than 300 meters (1000 feet) per night.
Every 1000 meters (3000 feet) you should spend a second night at the same elevation.
THE GOLDEN RULES

Any illness at altitude is altitude illness until proven otherwise
Never ascend w/ AMS
If you are getting worse, HACE, or HAPE: 

descend

“Aviator Bends”


Joint pain
Associated w/ exercise at altitude
Expansion of dissolved nitrogen
Embolic ischemia
Prevent: pretreat with Oxygen
References

www.high-altitude-medicine.com/AMS-medical.html


Dive Medicine

Neha Raukar, MD MS
The behavior of gases under pressure

**Boyle’s Law**
At a constant temperature, the volume of a gas is inversely related to the pressure to which it is subjected.

Explains **Barotrauma** and **Air Embolism**

**Henry’s Law**
At a constant temperature, the amount of a gas that is dissolved in a liquid is directly proportional to the partial pressure of that gas.

Explains **Decompression Sickness** and **Nitrogen Narcosis**
Barotrauma

A closed, air filled, space fails to equilibrate with the environment when there is a change in ambient pressure.

Descent → decreasing air volume in the space which contains tissue → mucosal edema of the tissue with vascular engorgement and hemorrhage

Ascent → increasing gas volume in a confined space → tissue disruption and rupture
Pulmonary Barotrauma

Descent: pulmonary edema and hemorrhage.

Ascent: alveolar rupture $\rightarrow$ pneumomediastinum, pneumothorax, arterial gas embolism.

Gas embolism is due to
- Gas bubbles in the pulmonary veins $\rightarrow$ systemic circulation
- Venous gas emboli $\rightarrow$ overwhelm ability of lungs to reabsorb gas $\rightarrow$ systemic
- Venous gas emboli $\rightarrow$ functional right to left shunt $\rightarrow$ systemic circulation

These gas emboli result in distal ischemia of the heart, brain, kidney, and can enter the mucocutaneous circulation.
**Ear Barotrauma**

Usually involves the middle ear which equilibrates with the environment via the eustachian tube.

Eustachian tube can be narrowed by edema due to URI, anatomic variations.

Descent $\rightarrow$ negative pressure in the middle ear $\rightarrow$ fills with serous fluid or blood $\rightarrow$ TM ruptures inward. This can be prevented by maneuvers that open the eustachian tube.
Other Barotrauma

Sinus
Descent: mucosal engorgement \(\rightarrow\) block the sinus ostia
- Usually affects the frontal sinus
Ascent: can lead to rupture of the sinus \(\rightarrow\) pneumocephalus

Dental
Occurs during both ascent or descent
Can lead to toothache or breakdown of the tooth
Decompression Sickness

Air consists of approximately 79% nitrogen
Descent → increased pressure → tissues become loaded with increased oxygen and nitrogen (Henry’s Law)

Ascend → liberate free gas from the tissue as bubbles → ischemia

Most of these nitrogen bubbles are harmlessly filtered out by the lungs but if the nitrogen bubbles overwhelm the filtering capacity of the lungs, the diver will develop “the bends”

The nitrogen bubbles activate the inflammatory pathway leading to cytokine release, complement activation, platelet aggregation and thrombosis
Decompression Sickness

Type 1
  Mild – affects the musculoskeletal system, the cutaneous system, and the lymphatic system

Type 2
  Can lead to permanent injury/death – affects the neurologic and pulmonary systems
Nitrogen Narcosis

Caused by the increase in partial pressure of nitrogen in the CNS

Looks like alcohol or benzodiazepine intoxication

Leads to impaired judgment of the diver
Treatment “Crush the bubble”

Preventing Decompression Sickness
  Slow, controlled ascents
    • No greater than 30 feet per minute
  However, this is not totally preventative and divers can get DCS even when being careful.

Treatment
  Make the diagnosis!
  Time is tissue
  The patient is placed in the recompression chamber
Pulmonary Overinflation Syndrome

Upon ascent the pressure decreases with a concomitant increase in volume

When a diver breaths compressed air at depth the gas expands on ascent and may result in rupture of the lungs.

Lung alveoli burst due to increased pressure.

Releases air directly into vasculature or surrounding structures.

Symptoms determined by location of leak.
Pulmonary Overinflation Syndrome

Clinical sequelae
- Arterial gas embolism
- Pneumothorax
- Mediastinal emphysema
- Subcutaneous emphysema

Treatment
- Make the diagnosis!
  - High index of suspicion
- Pneumothorax treated in conventional fashion
- All other POIS sequelae can benefit from recompression therapy
Take Home Points

If you develop signs or symptoms of altitude illness, DESCEND
Perform controlled ascents in dives to avoid decompression illnesses
Contact the Diver’s Alert Network if you have questions on hyperbaric treatment
if you need to locate a hyperbaric chamber
References


References