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)

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Altitude affects performance

1968 Olympics Mexico City (2300m)

1974 International Federation of Sports Medicine bans competition >3050m

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Acute Physiologic Response at Altitude

-Partial pressure oxygen

decreases

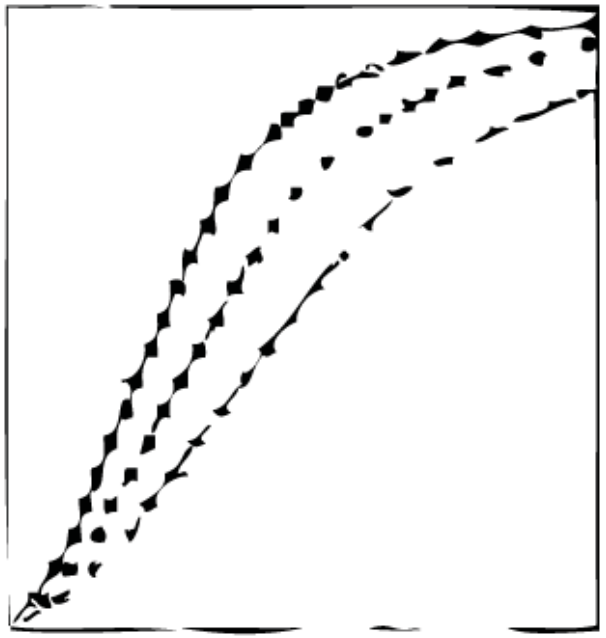
-Arterial SaO2

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

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Athletic Performance at Altitude

Reduction PpO2

Increased fatigue

VO2 max decreases

10%/1000m

Max work rate decreases

Max exercise HR decreases

Endurance time decreases

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Athletic Performance and Altitude

Aerobic: decreased performance

Anaerobic: unaffected

Sprinting: improved, decreased air density

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“Live High, Train Low”

Prolonged altitude exposure:

Increased erythropoietin, hematocrit, red cell mass, capillary density

Low altitude training:

Rigorous intensity

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“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

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Pathophysiology

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

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AMS-Incidence

2850m	9%
3050m	13%
3650m	34%
4550m	53%

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)

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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 Questionaire

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

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Lake Louis AMS Questionaire

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/semiconsciousness 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

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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?

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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

Acetazolamide

- rapid ascent >3000m
- gain >1000m sleeping elevation per day
- 125 mg bid, 24h in advance, discontinue 3rd night at maximal altitude or on descent

Dexamethasone

- 4 mg po/IM q 6 x 2
- delay ascent until asymptomatic and 18 hr after last dose

Ginko biloba

- 120 mg bid, 5d prior to ascent, continue
- 50% reduction symptoms/ milder S/S

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AMS Treatment

Descent

immediate

Rest at same elevation

24-48 h asymtomatic

Rest + acetazolamide

12-24 hr recovery

Rest + dexamethasone

2-6 hr recovery

Oxygen

4 L/m, 2 hr recovery, rebound

Hyperbaric oxygen/bag

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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 (absent 30%)
- central cyanosis
- tachypnea
- tachycardia

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”

Henry. The role of exercise in altitude pain. *Am J Physiol* 145:279, 1945.

Joint pain

Associated w/ exercise at altitude

Expansion of dissolved nitrogen

Embolic ischemia

Prevent: pretreat with Oxygen

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Dive Medicine

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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**

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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 → pneumomediastinum, pneumothorax, arterial gas embolism.

Gas embolism is due to

- Gas bubbles in the pulmonary veins → systemic circulation
- Venous gas emboli → overwhelm ability of lungs to reabsorb gas → systemic
- Venous gas emboli → functional right to left shunt → 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 → negative pressure in the middle ear → fills with serous fluid or blood → TM ruptures inward. This can be prevented by maneuvers that open the eustachian tube

Other Barotrauma

Sinus

Descent: mucosal engorgement → block the sinus ostia

- Usually affects the frontal sinus

Ascent: can lead to rupture of the sinus → 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

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