ALTITUDE SICKNESS

Intermediate altitude.
5000-8000 ft [1520-2440 m]

Produces ↓ exercise performance & ↑ alveolar ventilation without impairment in arterial oxygen transport.
Acute mountain sickness (AMS) occurs at (or above) 7-8000ft.

High altitude.
8000-14000 ft [2440-4270 m]

- a hypoxic environment.
    - ~26% less O2 than sea level.
- At Everest summit ~ ⅓ barometric pressure of sea-level.

Associated w/ ↓ SaO2 & marked hypoxaemia during sleep & exercise.
Most cases of altitude-related medical problems occur at this elevation rage.

Very-high altitude.
14-18000 ft [4270-5490 m]

- Typically in South America & Himalayan tourists.
- Abrupt ascent can be dangerous.
- Acclimatisation is required to avoid illness.

Extreme altitude.
>18000 ft [>5490 m]

- Only experienced by experienced mountain climbers.

Accompanied by *severe hypoxaemia & hypocapnia.*
Sustained human habitation is impossible.

PHYSIOLOGY OF ALTITUDE ACCLIMATISATION.

Ventilation.

- Primary initial adaptation; maintenance of alveolar PO2 via ↑ ventilation.
    - *Hypoxic hyperventilation response*
- Initial hyperventilation attenuated by respiratory alkalosis.
    - Renal excretion of HCO3 compensates for this (pH normalises) [4-7 days].
    - Ventilation then continues to increase
    - = "*ventilatory acclimatisation*"
- Acetazolamide - forces bicarb diuresis; facilitates acclimatisation.

Blood.

- ↑ EPO levels; ↑ central blood volume.
    - Suppression of ADH/aldosterone → induces diuresis.
- ↓ plasma volume & hyperosmolality [290-300mOsmol/kg]
- Haemoconcentration.

Cardiovascular.

- SV ↓ initially. ↑ HR to maintain CO.
- Maximum exercising HR declines at altitude.
- Healthy cardiac muscle can withstand $PaO_2 < 30$ mmHg with ST-changes or ischaemic events.
- Pulmonary circulation constricts (hypoxic vasoconstriction) → a hinderance.
    - Predisposes to *high-altitude pulmonary oedema.*

Exercise capacity.

- ↓'s dramatically on ascent to altitude.
    - ↓ by 10% each 1000m of altitude gained.
- Likely secondary to lack of adequate O_2 supply to muscle.

Limitations to acclimatisation.

- Weight loss [fat & lean body mass]
- Lethargy & headache
- Poor quality sleep
- RV strain [from excessive pulmonary HTN]
- Polycythaemia & microvascular sludging.

Sleep at high altitude.

- ↓ stages 3 & 4 sleep at altitude.
    - Stage 1 ↑'d
- Frequent arousals are common.
- Period (Cheyne-Stokes) breathing is common, esp. > 2700m.
- Sleep seems improved w/ acetazolamide.

HIGH ALTITUDE SYNDROMES.

Summarised below...

Hypoxia-related	Non-hypoxia related
- Acute Mountain Sickness - Pulmonary oedema - Cerebral oedema - Retinopathy - Peripheral oedema - Sleep problems - 'neurological syndromes'	- Thromboembolic events (secondary to dehydration, polycythaemia, cold & immobility) - High-altitude pharyngitis - Bronchitis - UV-keratitis

Acute hypoxia.

- Occurs in setting of sudden & severe hypoxic insult, eg. accidental decompression of pressurised aircraft cabin (or failure of oxygen delivery at altitude).
    - Arterial desaturation & APO.
    - Unconsciousness at SaO_2 50-60% (unacclimatised)
- Requires administration of O_2, rapid descent & correction of underlying pathology.

Acute mountain sickness (AMS).
A syndrome characterised by headache, GI disturbance, dizziness & sleep disturbance.

- Occurs w/ more gradual & less severe hypoxic insult than in acute hypoxia syndrome.
- Varies by location, rate of ascent & sleeping altitude.
    - Assoc. w/ altitudes of 2200-2700m.
- Physiologic precipitants include *poor hypoxic-ventilatory response & low vital capacity.*

Pathophysiology.
- Due to hypobaric hypoxia.
    - Unclear mechanism. ?↑ cerebral blood flow & ↑ blood volume.
- Abnormal water handling
    - Cerebral, interstitial pulmonary & peripheral oedema, plus antidiuresis.
    - ?Renin-angiotensin, aldosterone, ADH contribution.

Clinical features.
- Based on setting, symptoms & physical findings.
    - Rapid ascent in unacclimatised person.
    - Slightly lightheaded, breathless. (Develop w/in 1-6 hrs).
    - Bifrontal headache.
    - Anorexia, nausea. Lethargy.
- Progresses to severe headache, vomiting & oliguria.
    - Ataxia & altered consciousness → *high altitude cerebral oedema !!*
- SaO2 may be normal or ↓. Pulmonary crackles can be heard.
    - Fundoscopy = tortuous vessels w/ retinal haemorrhages.
    - Facial & peripheral oedema.

Treatment.
- Descent & oxygen.
    - Do *NOT* proceed to a higher sleeping altitude.

Treat immediately in presence of ↓ consciousness, ataxia or pulmonary oedema.

    - Descend.
- Oxygen therapy.
- Simple analgesia + antiemetics
- Acetazolamide 250mg bd
- Dexamethasone 4mg qid (moderate-severe disease)

High-altitude cerebral oedema.
Progressive neurological deterioration in a patient w/ AMS or high-altitude pulmonary oedema.

- Characterised by altered mental status, ataxia, stupor & progression to coma !!
- Focal neurological findings can arise (particularly 3rd & 6th CN palsies).

Treatment.
- Oxygen supplementation

- Descent (*highest priority*)
- Steroid therapy
    - Dexamethasone 8mg (stat), then 4mg qid.
- Acetazolamide may act as an adjunct.

Hyperbarics is an option for those who cannot descend.
- Evidence is also lacking for hypertonic saline, mannitol & diuretics.

High-altitude pulmonary oedema.

The *MOST LETHAL* of the altitude illnesses !!

Death arises from lack of early diagnosis, misdiagnosis & failure to descend.
- Easily reversed w/ descent & oxygen !!

Pulmonary hypertension of *any* cause greatly predisposes to HAPE.
- "Non-cardiogenic, hydrostatic oedema with normal LV function"
- High microvascular pressures.
    - Pulmonary HTN is essential.
    - Inflammation occurs later in disease process.

Clinically.
- Dry cough, ↓ exercise capacity, dyspnoea & localised crackles.
- SaO2 is lower than expected (for altitude)
- Tachycardia & tachypnoea are late signs.
    - Weakness, productive cough & cyanosis can occur.

A typical victim is normally well & may not portray strong signs of AMS prior to HAPE occurring (therefore delaying diagnosis).

Treatment.
- Immediate descent !!
    - Minimise exertion if possible.
- Supplemental oxygen (can completely resolve pathology).
    - Addition of PEEP may help w/ alveolar recruitment.
- Keep patient warm (cold-stress elevates pulmonary arterial pressures).
- Pharmacology (adjuncts only, does not replace O2 & descent):
    - Nifedipine 20mg tds → reduces pulmonary artery pressure (30-50%)
    - Nitric oxide (rarely available)
    - Sildenafil → blunts hypoxic pulmonary vasoconstriction.

Peripheral oedema.
- Face & extremities is common.
- Raises suspicion of altitude illness (prompts further assessment for cerebral or pulmonary oedema).
- Diuretics or conservative management.

High-altitude retinopathy.

- Includes retinal oedema, tortuosity & dilatation of retinal veins, retinal haemorrhages.
- Mostly asymptomatic (except when involving macular).
- Resolves w/in 10-14 days.

High-altitude pharyngitis & bronchitis.

- Dry hacking cough (can be purulent & painful).
- Not necessarily related to infection.
- Bronchospasm can result.

Chronic mountain polycythaemia / Chronic mountain sickness.

- More common in males.
- Headache, muddled thinking, difficulty sleeping, impaired circulation, drowsiness & chest congestion.
- Hb 200-220 !!
- Hypoxaemia at sea-level is *much worse* at altitude !
- Treatment involves phlebotomy, relocation & home-O2 use.

Ultraviolet keratitis (Snow blindness).

- Results from corneal burns.
- Symptom onset 6-12 hours post-exposure.
- Severe pain (FB or 'gritty' sensation).
    - Photophobia, tearing, marked conjunctival erythema, chemosis & swelling.
- Self-limiting (heals in 24 hours).
- Systemic analgesics + cold compresses.
- Sunglasses are excellent preventers.

Special Populations.

Chronic lung disease.

- ↑ dyspnoea, ↓ exercise tolerance.
- May require extra O2.

Arteriosclerotic heart disease.

- Less adaptive capabilities.
- CCF may worsen

Neurological syndromes.

- Most neurological events are attributed to HACE or AMS.
- Focal signs should be thoroughly evaluated & not just attributed to altitude illness.
    - Consider cerebral vasospasm, TIA/CVA, cerebral haemorrhage, cerebral venous sinus thrombosis.

Sickle-cell disease.

- Predisposes to vaso-occlusive crises.

Pregnant women.

- ↑ prevalence of HTN, low-birth weight infancy & neonatal hyperbilirubinaemia.
- Should avoid $SaO_2 < 85\%$ (eg. 3000m or higher)