Chapter 29

Environmental Injuries

Introduction
The successful prevention and control of cold, heat, and altitude injuries depend on vigorous command interest, the provision of adequate clothing, and a number of individual and group measures. The medical officer must ensure that he or she understands how military duties impact the occurrence and severity of environmental conditions and advise the commander on preventive measures.

Cold Injuries
Trench foot and frostbite together have accounted for over 1 million US casualties in WW I, WW II, and the Korean War. Influencing factors include previous cold injury; fatigue; concomitant injury resulting in significant blood loss or shock; geographic origin; nutrition; tobacco use; activity; drugs and medication; alcohol; duration and exposure; dehydration; environment (temperature, humidity, precipitation, and wind); and clothing.

Non–Freezing Cold Injury
- Chilblain.
  - o Results from intermittent exposure to temperatures above freezing, usually accompanied by high humidity and moisture; 1 to 6 hours of exposure.
  - o Swelling, tingling pain, and numbness with pink-to-red flushing of skin (especially the fingers).
  - o Extremities will be pruritic as they warm up.
  - o Symptoms usually subside overnight; some superficial scaling may occur.
  - o Mild joint stiffness may occur acutely but subsides in a few hours.
  - o No permanent damage occurs.

- Pernio.
  - Continuum of events from chilblain.
  - Exposure for > 12 hours to cold and/or wet conditions.
  - Tight-fitting footwear can shorten exposure time and increase severity of injury.
  - Swelling is more severe; pain is more persistent than with chilblain.
  - Thin, partial-skin thickness, and necrotic patches (from dorsum of the hands or feet).
  - Plaques may slough without scarring but may be particularly painful for months or years.
- Trench foot.
  - Epidemiology/clinical appearance.
    - Occurs from prolonged exposure to cold, wet conditions or prolonged immersion of feet at temperatures as high as 17°C for > 12 hours. Shorter duration at or near 0°C results in the same injury.
    - Occurs in nonfreezing temperatures 0°C–12°C.
    - Can occur at higher temperatures from prolonged water immersion.
    - Blunt trauma of marching can produce more serious injury.
    - First symptom often is the feet becoming cold, mildly painful, and numb.
    - Tight footwear increases risk of trench foot.
    - Common symptoms are "cold and numb" or "walking on wood."
    - Foot may appear swollen, with the skin mildly blue, red, or black.
    - Limb is hot and often hyperhidrotic.
    - On rewarming, pain is excruciating and does not respond to pain medication, including morphine.
    - As time progresses, liquefaction necrosis occurs distally, but more proximal tissue may also be compromised.
    - No sharp line of demarcation of dead from viable tissue.
    - Nerve, muscle, and endothelial cells are most susceptible to this long-term cooling.
    - Microvascular vasospasm with tissue ischemia is the apparent etiology of trench foot.

- ♦ Postinjury sequelae include pain, numbness, loss of proprioception, and cold feet. Hyperhidrosis with subsequent paronychial fungal infections are common.
- ♦ Life-long, life-changing injury.

o **Treatment.**

- ♦ Prevent further cold exposure.
- ♦ Do not massage.
- ♦ Dry extremity, warm torso, and allow slow passive rewarming of feet. **Never immerse feet in warm or hot water.**
- ♦ Elevate feet.
- ♦ Rehydrate.
- ♦ If **vesicles** develop do not debride.
- ♦ Pain medication. The only effective approach is amitriptyline 50–150 mg at bedtime. Other analgesics are either completely ineffective, or (as with narcotics) do not actually relieve pain.
- ♦ Blisters should be left intact; ruptured blisters require meticulous antisepsis after unroofing.
- ♦ Systemic antibiotics and tetanus prophylaxis are indicated when there are nonviable tissues, as with any other contaminated wound, or when there is evidence of infection.
- ♦ Debridement of necrotic tissue may be required in trench foot.
- ♦ Macerated or damaged skin requires topical antibacterial precautions.
- ♦ Avoid trauma.
- ♦ Early mobilization is vital to prevent long-term immobility.
- ♦ Recovery is protracted and may require evacuation because trench foot may lead to weeks-to-months of pain and disability.
- ♦ Long-term sequelae are very common and include sensitivity to the cold (secondary Raynaud's phenomenon), chronic pain, neurological impairment, and hyperhidrosis.

- Frostnip.
  - Exposed skin appears red or minimally swollen.
  - Tissue is not actually damaged.
  - Not true frostbite; freezing is limited to skin surface only.
  - Signals imminent likelihood of frostbite developing.
  - Resolves quickly with warming.
- Frostbite.
  - Results from crystallization of water in the skin and adjacent tissues exposed to temperatures below freezing.
  - Depth and severity of injury is a function of temperature and duration—the lower the temperature, the shorter the time required to produce injury.
  - At low temperatures in the presence of wind, exposed skin can freeze within a few seconds—starts distally and progresses up the finger or toe.
  - Freeze-front (line where the ice is formed in the tissues) is where liquefaction and necrosis occur. Tissues immediately proximal to this line may also die, but therapeutic modalities are directed at improving their survival.
  - Clinical appearance.
    - Skin initially becomes numb and feels stiff or woody.
    - Mottled, bluish, yellowish, "waxy," or "frozen."
    - Depth of involvement may be difficult to determine until demarcation occurs, which may take an extended period.
  - Frostbite grading.
    - Classification into degrees is primarily a retrospective evaluation and has little treatment value.
    - A more clinically useful grading typically divides injuries into superficial or deep.
    - Superficial frostbite.
      - Involves only the skin with swelling, mild pain, and minor joint stiffness.
      - No blisters form.
      - Nonmedical personnel can manage simply by rewarming.
    - Deep frostbite.
      - Involves deeper tissues to include bone.
      - White-hard, anesthetic, blanched, and inflexible.

- ◊ Skin will not move over joints.
- ◊ On rewarming, there is great pain and a blue-gray-to-burgundy color change.
- ◊ Blisters form and are clear, fluid-filled, or hemorrhagic (the latter indicates a more severe, deeper injury). They should be left in place; will slough in 7–10 days without consequence.
- ◊ Failure to form vesicles in an obviously deep-frozen extremity is a grave sign.
- ◊ Postinjury sequelae include Raynaud's phenomenon; pain; paraesthesias; hyperhidrosis; loss of proprioception; cold, discolored feet; and gait modification.

- Field treatment (first aid).
  - o Superficial (blanched cheeks, nose, ears, fingertips).
    - ♦ Warm with palm of hand or warm, wet cloth; warm fingers in armpits.
    - ♦ Emollients may help prevent skin from drying or cracking.
    - ♦ Do not massage, rub with snow, or warm part by an open fire or high-heat source.
    - ♦ Meticulous skin care is required.
  - o Deep frostbite.
    - ♦ Prevent further cooling of body part as well as the patient as a whole.
    - ♦ Apply dry, sterile bandage and elevate involved extremity.
    - ♦ Protect from refreezing during evacuation.
    - ♦ Evacuate promptly to definitive medical care.

Avoid thawing and refreezing; this leads to the greatest damage to tissue and the poorest outcome.

- MTF treatment.
  - o **The outcome of a frozen extremity is not directly related to length of time frozen, but more importantly to the method of rewarming and any subsequent refreezing.**
    - ♦ If the soldier will again be at risk for refreezing, no attempt at rewarming should be initiated; the soldier

should ambulate on the frozen extremities until he reaches definitive care.
- ♦ For transport, the patient's extremity should be splinted, and padded with dry dressings and protected from heat sources that would slowly rewarm the extremity.

o **Rapid rewarming (without the possibility of refreezing) is the treatment of choice.**
- ♦ Immerse in gently circulating water (whirlpool bath) at 40°C (104°F) for at least 30 minutes longer than could be needed to defrost all affected tissues. If deep freezing of the leg or arm has taken place, thorough surgical fasciotomy is mandatory prior to rewarming, to prevent lethal increase in deep tissue pressures as ice melts. Extremities are rewarmed until pliable and erythematous at the most distal areas.
- ♦ Twice daily whirlpool baths at 40°C with topical antibacterial added to the water, together with oral ethanol. The alcohol reduces the need for analgesia and may improve outcome. Other drug regimens remain unproven.
- ♦ After rewarming, edema will appear within a few hours and vesicles form within the next 6–24 hours.
- ♦ Intensive mobilization is essential to avoid long-term immobility.

o Vesicles**.**
- ♦ Frostbite vesicles are typically left intact.
- ♦ Debridement is not recommended. Early surgery is only indicated in severely infected cases. Normally surgery should be delayed for at least 6 months.

o General considerations**.**
- ♦ Ibuprofen or ketorolac should be given as systemic thromboxane/prostaglandin inhibitors.
- ♦ Systemic antibiotics and tetanus prophylaxis are indicated when there are dead tissues, as with any other contaminated wound, or when there is evidence of infection.
- ♦ Dry, loose dressings may be applied.
- ♦ Cigarette smoking and/or nicotine use is contraindicated during treatment due to its effect on the microvasculature.

- ♦ Daily hydrotherapy is recommended. Pain control with NSAIDs and narcotics will be needed.
- ♦ Sequelae include contractures, cold sensitivity, chronic ulceration, arthritis, and hyperhidrosis.
- ♦ Frostbite cases will require prolonged hospital care (9 d on average); therefore, all but those with the most trivial injuries should be evacuated to more definitive care as soon as possible.
- ♦ Early surgery is indicated only in the most severe freeze-thaw-refreeze cases, where massive tissue destruction has taken place, and in some more severely infected cases. Normally, surgery should be delayed for at least 6 months ("Freeze in January, operate in July").

Due to the inability to reliably predict the outcome in the postthaw period, there is no role for debridement/amputation of necrotic or potentially necrotic tissue in the initial treatment of frostbite.

Hypothermia

Hypothermia is classically defined as whole-body cooling below 35°C. Degree of hypothermia is further defined according to the body's core temperature and the clinical effects seen in a given temperature range.

- Causative factors and prevention.
  - o Water immersion.
  - o Rain and wind.
  - o Prolonged exposure to severe weather without adequate clothing. The insulation effect of clothing is markedly decreased with wetness, which increases the conductive heat loss.
  - o Stay dry and avoid windy exposure.
  - o Shivering can provide five times the normal metabolic heat production. Exhaustion and glycogen depletion decrease the time of shivering. Compromise of shivering due to inadequate food intake (skipping meals), exhaustion, heavy exercise, alcohol, and drugs increases threat of hypothermia.

- Mild hypothermia > 33°C (> 91°F).
  - Shivering, hyperreflexia.
  - Amnesia, dysarthria, poor judgment, ataxia, apathy.
  - Cold diuresis.
- Moderate hypothermia 28°C–33°C (82°F–91°F).
  - Standard hospital thermometers, mercury as well as digital, cannot measure temperatures below 34°C (93°F).
  - Stupor, loss of shivering.
  - Onset of atrial fibrillation and other arrhythmias.
  - Progressive decrease in level of consciousness, respiration, and pupillary reaction, eventual pupil dilation.
- Severe hypothermia < 28°C (< 82°F).
  - Increased incidence of ventricular fibrillation, which often occurs spontaneously.
  - Loss of motion and reflexes, areflexic at approximately 23°C (72°F).
  - Marked hypotension/bradycardia.
- Profound hypothermia < 20°C (< 68°F).
  - Asystole.
  - Lowest known adult survival from accidental hypothermia is 13.7°C (56°F).

Treatment

- Prehospital (field) treatment.
  - Awake patients.
    - Remove wet clothing; dry and insulate the patient.
    - Give oral sugar solutions to hydrate.
    - Walk out or transport to MTF. (This should be attempted if it is the only alternative because it is likely to worsen the condition.)
    - Although walking may deepen hypothermia due to the return of peripheral colder blood to the core, adequate prehydration decreases the postexposure cooling.
  - Comatose patients.
    - Patient should remain horizontal and be handled gently to avoid inducing arrhythmias; do not massage.
    - IV fluids, warmed to 40°C–42°C, if possible.
    - Do not use lactated Ringer's solution because the cold liver cannot metabolize lactate; warm (40°C–42°C

[104°F–107.5°F]), D5NS is the fluid of choice.
- ♦ Remove wet clothes, dry, insulate, and add an outer vapor barrier. Wrap patient in multiple layers of insulation.
- ♦ Limit active rewarming principally to the body's center/core only.
  - ◊ Heated (40°C–45°C), humidified air/O_2 is the method of choice.
  - ◊ Norwegian personal heater pack (charcoal heater), with warming tube placed into insulation wrap.
  - ◊ Forced air (Bair Hugger) with rigid chest frame.
  - ◊ Hot water bottles in groin/axilla.
- ♦ Intubation and heated ventilation may be performed.
- ♦ If apneic and asystolic, consider CPR, because the brain may survive longer.

Remember: The patient is not dead until he is warm and dead. Continue resuscitation, if possible, until patient has been rewarmed.

- Medical treatment.
  - o Ventilate; apply CPR if asystolic or in ventricular fibrillation.
  - o As the body cools, the peripheral vasculature constricts, causing pooling of cold acidotic blood.
  - o Rewarming the periphery of the body rather than the core causes an inrush of this cold acidotic blood into the core, further dropping the core temperature (afterdrop), and worsening cardiac instability.
  - o Core rewarming—peritoneal dialysis, thoracic lavage, heated and humidified oxygen, external warm blankets, and warm-water torso immersion.
  - o For ventricular fibrillation.
    - ♦ Bretylium tosylate, 10 mg/kg. Bretylium is the only known effective antidefibrillation drug for hypothermia. It remains functional in a cold heart. Other medications have not proven effective.
    - ♦ Warmed IV (lactate and potassium-free).

- Monitor core temperature via esophageal (preferred) or rectal probes.
- Careful correction of acid/base balance.
- Rewarm core to 32°C (90°F) and attempt cardioversion (360 J). Continue rewarming and repeat. Defibrillate after every 1°C rise in temperature.
- Monitor potassium, glucose, temperature, and pH.
- Major causes of failure to resuscitate include elevating central venous pressure too fast or too early; attempting defibrillation when core temperature is below 32°C, or continuing to rewarm past 33°C when potassium levels are high and pH is low. If serum potassium levels are high, consider the use of intravenous glucose and insulin.
- Avoid other antiarrhythmics and other medications.
- Patients with core temperature (rectal) above 30°C can generally be rewarmed externally in a variety of methods including warm blankets, warm-water torso immersion. Patients below 30°C rectal should be considered more fragile and will often require internal methods of rewarming (ie, warm gastric, colonic, and/or bladder lavage; warm peritoneal lavage dialysis; warm thoracic lavage; and arteriovenous (blood rewarming). Lavage fluids should be warmed to 40°C–42°C (104°F–107.5°F).
- Core temperature will continue to drop after the patient is removed from the cold exposure. Continued temperature drop can have grave prognostic implication and increases the likelihood of fibrillation. Post-rewarming collapse of an apparently functional heart often leads to a nonresuscitable heart and death.

- Cardiopulmonary resuscitation.
  - If cardiac monitor shows any electrical complexes, check carefully for apical and carotid pulses before initiating CPR. If any pulse—however thready—is present, **do NOT initiate CPR.**

Trauma patients should be considered to have hypothermia more profound than the core temperature indicates and be warmed more aggressively.

- Treatment of mild stable hypothermia.
  - o Insulation.
  - o Heat lamps.
  - o Warmed IV fluids.
  - o Warmed forced air (Bair Hugger). Hair dryers have been jury-rigged for this purpose.
  - o Consider arteriovenous anastomoses (AVA) warming.
    - ♦ Immerse hand, forearms, feet, calves in water heated to 44°C–45°C (111°F–113°F).
    - ♦ Opens AVAs in the digits causing increased flow of warmed venous blood to the heart and decreases afterdrop.
- **Treatment of severe hypothermia with hemodynamic instability**.
  - o Cardiopulmonary bypass with rewarming, when available, is the ideal technique in this circumstance because it provides core rewarming while ensuring circulatory stability.

Heat Injury

In the military setting, heat illness occurs in otherwise healthy individuals, and ranges from mild (heat cramps) to life threatening (heatstroke). Individuals typically present with exertional heat illness and are hot and sweaty, not hot and dry as seen in classic heatstroke.

Lack of sweating is not a criterion for heatstroke. Some military casualties of heatstroke have profuse sweating; especially with rapid onset of heatstroke.

Minor heat illnesses include heat cramps and heat exhaustion. Major heat injuries include exertional heat injury (EHI), exertional rhabdomyolysis, and heat stroke. The diagnostic categories of heat exhaustion, EHI, and heat stroke have overlapping features and should be thought of as different

regions on the continuum rather than discrete disorders, each with its own distinct pathogenesis.

- Heat injury prevention.
  - Easier to prevent than treat.
  - Occurs most commonly in unacclimatized individuals.
    - Acclimatization to heat requires 7–10 days.
    - Predeployment training in artificially warm environments does aid heat acclimatization.
    - One hour of progressively more difficult exercise sufficient to induce moderate sweating each day will maximize acclimatization. (Regular strenuous exercise sufficient to stimulate sweating and increase body temperature will result in a significant degree of heat acclimation.) Aerobic fitness provides cardiovascular reserve to maintain the extra cardiac output required to sustain thermoregulation, muscular work, and vital organs in the face of heat stress.
  - Utilize published work–rest cycle guides (eg, FM 21-10/MCRP 4-11.1D) or work–rest cycles tailored to the individual's physical capacity by direct medical oversight.
  - **Water restriction/discipline leads to increased heat injury and is contraindicated.**
    - Acclimatization does not reduce, and may actually increase, water requirements.
    - Service members will on average not feel thirsty until 1.5 L (1%–2%) dehydrated.
    - Fluid intake should be monitored to ensure urine appears dilute. Additionally, soldiers should be monitored for body weight changes and orthostatic blood pressure changes due to hydration.
    - The GI tract can absorb only 1–1.5 L/h.
    - Daily rehydration should not exceed 12 L/d orally. **Too much hydration can also be dangerous and lead to water intoxication!**
    - Leaders must reinforce hydration by planning for all aspects of adequate hydration—elimination as well as consumption. (Soldiers may not drink at night to avoid awakening and having to dress to urinate, or soldiers may not drink prior to a convoy because no rest stops are planned.)

- MOPP gear will increase fluid losses and the incidence of heat injuries.
- In the first few days of acclimatization, sweat–salt conservation will not be fully developed. Salt depletion is a risk if soldiers are exposed during this time to sufficient heat or work stress to induce high sweating rates (> several liters per day), particularly if ration consumption is reduced. Salt depletion can be avoided by providing a salt supplement in the form of salted water (0.05%–0.1%). Acclimation should eventually eliminate the need for salt supplementation
- Salt supplements are not routinely required and are only recommended in rare instances where adequate rations are not consumed.
- Coincidental illnesses increase heat casualty risk through fever and dehydration. Fever reduces thermoregulatory capacity leading to increased risk, even after clinical evidence of illness has disappeared. Requires increased command supervision and moderate work schedule.
- Sunburn and other skin diseases of hot environments reduce the ability of the skin to thermoregulate. Sunburn must be prevented by adequate clothing, shade, and sunscreen. Skin diseases are best prevented by adequate hygiene.
- Medications that effect thermoregulatory adaptations and increase risk of heat injury include anticholinergics, antihistamines, diuretics, tricyclic antidepressants, major tranquilizers, stimulants, and beta blockers.

Despite preventive measures, service members may suffer from heat illness. One case of heat illness is a warning sign that many others are imminent. The most life-threatening condition is heatstroke. Severity of heat illness depends on the maximum core temperature and duration.

- Heatstroke.

Heat stroke is distinguished from heat exhaustion by the presence of clinically significant tissue injury and/or altered mental status. Degree of injury appears to relate to both the degree of temperature elevation and duration of exposure.

- Clinical presentation.
  - Heat stroke is a true emergency. Involves five organ systems: brain, hemostatic, liver, kidneys, and muscles.
  - Encephalopathy ranges from syncope and confusion to seizures or coma with decerebrate rigidity. Profound neuropsychiatric impairments present early and universally in casualties of advanced exertional heat stroke.
  - Coagulopathy: thermal damage to endothelium, rhabdomyolysis, and direct thermal platelet activation causes intravascular microthrombi. Fibrinolysis is secondarily activated. Hepatic dysfunction and thermal injury to megakaryocytes slow the repletion of clotting factors. Hepatic injury is common. Transaminase enzyme elevation (values 100 or more times the upper normal limit), clotting factor deficiencies, and jaundice (within 24–36 h of onset). Transaminase levels may be transient and reversible, but if they persist 48 hours, it is indicative of more severe injury. Hypoglycemia is a frequent complication of exertional heat stroke.
  - Renal failure: myoglobinuria from rhabdomyolysis in exertional heat stroke, acute tubular necrosis due to hypoperfusion, glomerulopathy due to disseminated intravascular coagulation (DIC), direct thermal injury, and hyperuricemia.
  - Muscles are often rigid and contracted: Rhabdomyolysis is a frequent acute complication of exertional heat stroke. Acute muscular necrosis releases large quantities of potassium, myoglobin, phosphate, uric acid, and creatine, and sequesters calcium in exposed contractile proteins.

If heat stroke is suspected and temperature is elevated, cooling should not be delayed to accomplish a diagnostic evaluation. Cooling and evaluation should proceed simultaneously.

The patient with heat stroke requires immediate evacuation to medical facilities with intensive care capabilities. Active cooling should be started immediately and continued during evacuation.

- ♦ Prodromal symptoms include headache, dizziness (lightheadedness), restlessness, weakness, ataxia, confusion, disorientation, drowsiness, irrational or aggressive behavior, syncope, seizures, or coma.
- ♦ Collapse is a universal feature of heat stroke.
- ♦ An individual with a core temperature of ≥ 40ºC (104°F) and CNS dysfunction that results in delirium, convulsions, or coma has heat stroke.

o Casualties who are **unconscious** and have a core temperature of ≥ 39°C (102.2°F) have heatstroke.

- ♦ Core temperature is often lower on arrival at a treatment area.
- ♦ Seizures.
  - ◊ Occur frequently (> 50% of cases) with heatstroke.
  - ◊ Hinder cooling efforts.
  - ◊ Treat with diazepam 5–10 mg.

o Treatment.

- ♦ Rapid cooling can reduce heat stroke mortality anywhere from 50% down to 5%. Cooling by spraying cool water over the body and vigorous fanning can be effective though not as effective as ice water immersion. Any effective means of cooling is acceptable.
- ♦ A variety of techniques have been used, and, while evaporative cooling is less effective, the ice immersion method may prevent safe cardiac monitoring or rapid resuscitation.
- ♦ Cool water immersion (20°C) with skin massage is the classic technique. It provides rapid cooling. Closely monitor patient for, and prevent, shivering.
- ♦ Cooling with cool-water–soaked sheets or ice chips and vigorous fanning is highly effective.
- ♦ Do not use alcohol in the cooling solution because freezing of the skin can occur.

The goal of treatment is to effect a rapid lowering of the core temperature to 38°C (101°F), without inducing shivering.

- Rectal temperature should be closely monitored during cooling. Discontinue cooling efforts when core temperature reaches 38.3°C (101°F) to avoid hypothermia.
- Aspirin and acetaminophen should **NOT** be given to casualties of heatstroke.
- Aggressive fluid resuscitation is not required. Fluid requirements of 1 L in the first 30 minutes, with an additional 2 L or more in the next 2 hours may be sufficient. Because heat stroke patients are frequently hypoglycemic, the initial fluid should include dextrose (chilled IV fluid is of limited benefit).
  - Base further hydration on fluid status/urinary output (Foley required).
  - Overhydration can lead to congestive heart failure, cerebral edema, and pulmonary edema in the heat-stressed lung.
- If shivering develops, treat with diazepam (5–10 mg IV) or chlorpromazine (50 mg IV).
- Patients are frequently agitated, combative, or seizing. Diazepam is effective for control and can be administered IV, endotracheally, or rectally, but should be used with caution.
- Airway control is essential. Vomiting is common and endotracheal intubation should be used in any patient with a reduced level of consciousness, or otherwise unable to protect the airway. Supplemental oxygen should be provided when available.
- Hypotensive patients who do not respond to saline should receive inotropic support. Careful titrated use of dopamine or dobutamine is reasonable and has the potential added advantage of improving renal perfusion.
- Pulmonary artery wedge pressure monitoring should be used in patients with persistent hemodynamic instability.
- Management of encephalopathy is supportive in nature and is directed at minimizing cerebral edema by avoiding fluid overreplacement and by assuring hemodynamic, thermal, and metabolic stability. IV mannitol has been used to treat life-threatening cerebral

edema, but is questionable unless renal function is adequate and the patient is fully hydrated. The efficacy of dexamethasone for treating heat-stroke–induced cerebral edema is not known.

- Complications.
  - Rhabdomyolysis and secondary renal failure due to myoglobinuria and hyperuricemia; hyperkalemia; hypocalcemia; and compartment syndromes due to muscle swelling.
    - Elevated creatine phosphokinase (CPK) (in the thousands).
    - Administer IV fluid and possibly furosemide to maintain urinary output > 50 cc/h. (Assurance of adequate renal perfusion and urine flow will moderate the nephrotoxic effects of myoglobin and uric acid.)
    - Hyperkalemia can be managed by K/Na ion exchange resin (Kayexalate) given orally or rectally as an enema. If available, dialysis may occasionally be indicated.
    - Hypocalcemia does not usually require treatment.
    - Increasing tenderness or tension in a muscle compartment may represent increasing intracompartmental pressures. Direct measurement of intramuscular pressure or fasciotomy should be considered. Pain and paresthesia from a compartment syndrome may not be present until after permanent damage has occurred.
  - Alkalinize urine with sodium bicarbonate IV (2 amps $NaHCO_3$/L D5W). Management of acute renal failure requires exquisite attention to fluid and electrolyte balance. Uremic metabolic acidosis and hyperkalemia require dialysis for control.
  - Coagulopathy due to hepatic injury.
    - Hepatic injury is common, resulting in transaminase enzyme elevation, clotting factor deficiencies, and jaundice. Transaminase levels may be transient and reversible, but if they persist 48 hours, then it is indicative of more severe injury.

- ◊ Worst prothrombin time occurs at 48–72 hours postinjury.
- ◊ Thrombocytopenia and disseminated intravascular coagulation (DIC) peak at 18–36 hours postinjury.
- ◊ Beware of the coagulopathy timeframe when planning evacuation.
- ◊ Subclinical coagulopathy does not require active management. Clinically significant bleeding is an ominous sign. Treatment is directed at reducing the rate of coagulation and replacement of depleted clotting factors. Intravascular coagulation can be slowed by cautious heparin infusion (5–7 U/kg/h), followed in 2–3 hours by FFP and platelets. Successful management leads to a decline in indices of fibrinolysis (eg, fibrin split products). Heparin is tapered gradually over 2–3 days as directed by laboratory evidence of control.
- ◊ Monitor for hypo- or hyperglycemia.

♦ Prognosis is worse in patients with more severe degrees of encephalopathy. Permanent neurologic sequelae can develop after heat stroke, including cerebellar ataxia, paresis, seizure disorder, and cognitive dysfunction.

♦ Neurologic deterioration after initial recovery may represent intracranial hemorrhage related to diffuse intravascular coagulation or hematoma related to trauma unrecognized at the time of initial presentation.

♦ Other complications include gastrointestinal bleeding, jaundice, aspiration pneumonia, noncardiogenic pulmonary edema, and myocardial infarction. Immune incompetence and infection are late complications, particularly in patients with severe renal failure.

♦ Hyperkalemia is the most life-threatening early clinical problem. Measurement of serum potassium is an early priority.

- Heat cramps.
  - o Clinical presentation.
    - ♦ Brief, intermittent, recurring, and often excruciating tonic muscle contractions that last 2–3 minutes. Preceded by palpable or visible fasciculations.

- Typically involve muscles of the abdomen, legs, and arms (voluntary muscles of the trunk and extremities). Smooth muscle, cardiac muscle, the diaphragm, and bulbar muscles are not involved.
- Occur often with heat exhaustion. (Despite the salt depletion associated with heat cramps, frank signs and symptoms of heat exhaustion are unusual.)
- There are no systemic manifestations except those attributable to pain.
- Occur in healthy individuals who exercise for prolonged periods in warm environments.
- Occur in salt-depleted patients, generally during a period of recovery after a period of work in the heat.
- Differential diagnosis: tetany due to alkalosis (hyperventilation, severe gastroenteritis, cholera), hypocalcemia, strychnine poisoning, black widow spider envenomation, and abdominal colic.

o Treatment.

- Mild cases can be treated with oral 0.1%–0.2% salt solutions. Salt tablets should not used as an oral salt source.
- Most "sports drinks" (diluted 1:1 with water) effective for mild cases.
- IV NS provides rapid relief in more severe cases
- Patients with heat cramps usually have substantial salt deficits (15–30 g over 2–3 days, usual dietary intake). These individuals should be allowed 2–3 days to replenish salt and water deficits before returning to work in the heat.

● Heat exhaustion.

o Clinical presentation.

- Thirst, headache, dyspnea, lightheadedness (orthostatic dizziness), profound physical fatigue, anorexia, confusion, anxiety, agitation, mood change, chills, piloerection, nausea, and vomiting. There is no combination of presenting symptoms and signs that is pathognomonic.
- Often accompanied by heat cramps.
- Oliguria, clinical dehydration, ataxia, tachycardia, and

tachypnea resulting in symptomatic hyperventilation with acroparesthesia and carpopedal spasm.
- ♦ Syncope may occur.
- ♦ Core temperature is < 39°C (102.2°F), even at time of collapse.

o Treatment.
- ♦ Oral rehydration (if patient is not vomiting).
- ♦ Parenteral fluids produce more rapid recovery: no more than 250 mL NS bolus without laboratory surveillance; after 2.5 L of plain saline, add dextrose as a source of energy (D2.5$^1/_2$NaCl); subsequent fluid replacement should be D5$^1/_2$NS or D5$^1/_4$NS. Individuals with significant salt depletion have coincident potassium depletion, often amounting to 300–400 mEq of KCl. To begin restoration of potassium deficit, inclusion of potassium in parenteral fluids after volume resuscitation is appropriate if there is no evidence of renal insufficiency or rhabdomyolysis.
- ♦ Does not require active cooling; however, because symptoms are difficult to distinguish from heat stroke, the **safest course** is to provide active cooling for all casualties who are at risk for heat stroke.
- ♦ Removal from hot environment.
- ♦ Stop exercising, move out of the sun.

- Minor heat illnesses.
  - o Miliaria rubra, miliaria profunda, and anhidrotic heat exhaustion.
    - ♦ Subacute (miliaria rubra) pruritic inflamed papulovesicular skin eruption that appears in actively sweating skin exposed to high humidity. Becomes generalized and prolonged (miliaria profunda); lesions are truncal, noninflamed papular, with less evidence of vesiculation than the lesions of miliaria rubra.
    - ♦ Each miliarial papulovesicle represents an eccrine sweat gland whose duct is occluded at the level of the epidermal stratum granulosum by inspissated organic debris.
    - ♦ Eccrine secretions accumulate in the glandular portion of the gland and infiltrate into the surrounding dermis.

- ♦ Pruritus is increased with increased sweating.
- ♦ Miliarial skin cannot fully participate in thermoregulatory sweating, therefore the risk of heat illness increases in proportion to the amount of skin surface involved. Sweat does not appear on the surface of affected skin.
- ♦ Sleeplessness due to pruritus and secondary infection of occluded glands has systemic effects that further degrade optimal thermoregulation.
- ♦ Miliaria is treated by cooling and drying affected skin, avoiding conditions that induce sweating, controlling infection, and relieving pruritus. Eccrine gland function recovers with desquamation of the affected epidermis, which takes 7–10 days.
- ♦ Miliaria profunda causes an uncommon but disabling disorder: anhidrotic heat exhaustion (or tropical anhidrotic asthenia). Miliaria profunda causes a marked inhibition of thermoregulatory sweating and heat intolerance similar to that of ectodermal dysplasia. That individual is more at risk for heat exhaustion and at high risk of heat stroke in conditions tolerated by others.
- ♦ Evacuation to a cooler environment until restoration of normal eccrine gland function.

o Heat-induced syncope.

- ♦ Due to a reduced effective blood volume. (Thermal stress increases risk of classic neurally mediated [vasovagal] syncope by aggravating peripheral pooling of blood in dilated cutaneous vessels.)
- ♦ Symptoms range from light-headedness to loss of consciousness.
- ♦ Typically someone standing in a hot environment.
- ♦ Greatest risk on first day of heat exposure, subsequent risk decreases daily.
- ♦ Risk almost zero after 1 week of heat exposure; however, syncope occurring during or after work in the heat, or after more than 5 days of heat exposure, should be considered evidence of heat exhaustion.
- ♦ Core temperature is not elevated or only very minimally so.
- ♦ Patient regains consciousness immediately after syncope.

- ♦ Clinical evaluation and management should be directed toward the syncopal episode, not potential heat illness. Treatment is oral hydration and continued acclimatization.

o Heat edema.
- ♦ Seen early in heat exposure.
- ♦ Plasma volume expanding to compensate for the increased need for thermoregulatory blood flow.
- ♦ In absence of other disease, condition is of no clinical significance.
- ♦ Will resolve spontaneously.
- ♦ Diuretic therapy is not appropriate and may increase risk of heat illness.

o Sunburn.
- ♦ Reduces thermoregulatory capacity of skin.
- ♦ Systemic effect: hyperthermia.
- ♦ Preventable.
- ♦ Affected soldiers should be kept from significant heat strain until the burn has healed.

o Heat tetany.
- ♦ Rare; occurs in individuals acutely exposed to overwhelming heat stress.
- ♦ Extremely severe heat stress induces hyperventilation.
- ♦ Manifestations include respiratory alkalosis, carpopedal spasm, and syncope.
- ♦ Treatment: removal from heat source and control of hyperventilation (rebreathing into paper bag to reverse respiratory alkalosis).
- ♦ Dehydration and salt depletion are not prominent features.

Altitude Illness

Exposure of troops to the hypobaric hypoxia of altitude results in a decrement of performance, as well as the possible development of altitude illness. Altitude illness spans a spectrum from high-altitude bronchitis, to acute mountain sickness (AMS), to death from high-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE).

- Altitude basics.

The occurrence of altitude illness is based on altitude and rapidity of ascent. Contributory factors include level of exertion, physiologic susceptibility, age, and coexisting medical conditions.

  - Physiologic changes due to altitude begin to occur at just over 1,500 m (4,900 ft).
  - These changes are the body's attempt to acclimatize to altitude.
  - Symptoms occurring below 2,250 m (7,400 ft) are rarely due to altitude illness.
    - Rapid ascent to high altitudes results in a high incidence of altitude illness.
    - Climbing Mt. Rainier brings one from sea level to 14,500 ft (4,400 m) in 36 hours and results in a 70% incidence of altitude illness. An ascent to a similar height over the course of 5 days would only result in a 5% incidence of altitude illness.
    - 10%–20% of soldiers who ascend rapidly (< 24 h) to altitudes between 1,800 to 2,500 m (6,000–8,000 ft) experience some mild symptoms
    - Rapid ascent to elevations of 3,600 to 4,300 m (12,000–14,000 ft) results in moderate symptoms in over 50% of the soldiers, and 12%–18% may have severe symptoms.
    - Rapid ascent to 5,300 m (17,500 ft) causes severe, incapacitating symptoms in almost all individuals.

- Descent basics.
  - Almost everything improves with prompt descent.
  - For illness requiring descent, one should try to descend at least 1,000 m (3,300 ft) if not more.
  - A Gamow bag (USA) (portable fabric hyperbaric chamber) or Certec SA (Europe) can temporize a patient if evacuation / descent is not possible.
  - Symptoms typically resolve quickly with descent, but may linger for several days.
  - Victims of HACE and HAPE should not reascend until 72 hours after symptoms abate, and then must ascend much slower than previously.

- Victims of HACE or HAPE should descend at the earliest sign, before they become moribund and incapable of aiding in their own descent.
- **There are no reliable predictors of susceptibility to AMS except prior experience at altitude.**

Incidence and severity of symptoms vary with initial altitude, rate of ascent, level of exertion, and individual susceptibility.

- Vigorous physical activity during ascent or within 24 hours after ascent will increase both the incidence and severity of symptoms.
  - If a soldier became ill previously at a given altitude he or she will likely become ill at the same altitude unless the ascent is slower to allow for better acclimatization.
  - Physical fitness level has **no effect** on susceptibility to altitude illness.
  - Oral sildenafil (Viagra) 50 mg qd increases exercise tolerance in healthy volunteers at altitude (5,200 m [17,000 ft]), although it has not been approved for this purpose. The role of this drug in the treatment and/or prophylaxis of AMS and HAPE has not been established.
  - If a rapid ascent to altitude must be made, use prophylaxis against AMS.

- Acute mountain sickness.
  - AMS is the most common form of altitude illness.
  - Onset is shortly after arrival at high altitude. Onset occurs 3–24 hours after ascent. Symptoms reach peak severity in 24–72 hours and usually subside over the course of 3–7 days.
  - Further ascent without an acclimation period usually exacerbates symptoms and can result in increased incidence of HAPE and HACE. The majority of AMS cases do not progress to more serious altitude illness without continued ascent.
  - Symptoms.
    - Headache: Symmetric, global in location, and throbbing in character. Most intense during night and shortly after

arising in the morning, attributed to increased hypoxemia caused by altitude-induced sleep apnea.
- ♦ Anorexia.
- ♦ Nausea.
- ♦ Fatigue (weakness).
- ♦ General malaise.
- ♦ Decreased coordination.
- ♦ Dizziness or light-headedness.
- ♦ Oliguria.
- ♦ Emesis (vomiting).
- ♦ Lassitude.
- ♦ Insomnia: Sleep disturbances with periodic breathing with recurrent apneic periods during sleep are usually present, but are not necessarily a component of AMS.

o Diagnosis.
- ♦ Occurrence of a headache and at least one other sign/symptom in an individual who ascended from low (1,524 m or < 5,000 ft) to high altitude, or high altitude to higher altitude in the previous 24–48 hours.
- ♦ Differential diagnosis includes viral gastroenteritis, hangover, exhaustion, dehydration, carbon monoxide poisoning, and HACE.
- ♦ Presence of neurologic symptoms such as incoordination, ataxia, and excessive lethargy or cognitive dysfunction is indicative of progression to HACE, which requires immediate therapeutic intervention.

o Prophylaxis for AMS.
- ♦ Gradual acclimation.
  - ◊ Staged ascent: Soldiers ascend to intermediate altitudes and remain there for 3 or more days before ascending further.
  - ◊ Graded ascent: Limits daily altitude gain to allow partial acclimation. Sleep altitude is most important. Have soldiers spend 2 nights at 2,743 m (9,000 ft) and limit the sleeping altitude to no more than 305 m (1,000 ft) per day above previous night's sleep altitude.
  - ◊ Combination of both staged and graded ascent is the safest and most effective prevention method.

- ♦ Diet: High carbohydrate diet (< 70% of total energy intake as carbohydrates) (stimulation of ventilation through increased carbon dioxide produced from metabolism of carbohydrates).
- ♦ Acetazolamide, 250 mg qid or 500 mg bid po, starting 48 hours before ascent, continuing for 48 hours after ascent. Side effects include peripheral paresthesias, fatigue, increased urination (polyuria), and altered taste imparted to carbonated beverages. It prevents AMS in 50%–75% of soldiers and reduces symptoms in most others. Short-term use when changing altitude significantly (400 m). **Contraindicated in sulfa allergy**.
- ♦ Dexamethasone, 4 mg qid po is the prophylaxis of choice in sulfa-allergic individuals. Dexamethasone does not aid acclimatization and effects are gone when it is stopped. Dexamethasone +/– acetazolamide is also prophylaxis of choice for missions of a rapid, high (over 4,000 m [13,000 ft]), short-duration profile (raids, rescues).
- ♦ Cyanosis: Oxygen 2–6 L/min. Do not delay descent.

o **Treatment.**

- ♦ AMS alone does NOT mandate descent.
- ♦ Remain at the same elevation; do **not** ascend until symptoms abate.
- ♦ Acetazolamide, 125 mg qid to 500 mg, tid, po—do not use in patients with sulfa allergies. (If already receiving a preventive dose of acetazolamide (1,000 mg/d) and still symptomatic, 500 mg can be added with caution.
- ♦ Dexamethasone in doses of 2–4 mg q6h (has the same potentially serious side effects as when used as a prophylaxis). Symptoms may recur when medication stopped.
- ♦ Oxygen by nasal cannula 2–6 L/min (severe headache).
- ♦ Do NOT advance sleeping altitude.
- ♦ Symptomatic treatment with ASA, acetaminophen, prochlorperazine for nausea and vomiting 5–10 mg tid–qid, po or IM, or 25 mg bid prn also stimulates respiration; ibuprofen for headache.
- ♦ Minimize utilization of sleeping agents at altitude; they

can worsen illness. Acetazolamide for sleep disorders, 250 mg qid or tid po. Temazepam for insomnia 30 mg qhs po; triazolam for insomnia 0.125–0.25 mg qhs po. Short-term use only. Possible short-term memory loss.

- High-altitude pharyngitis and bronchitis.
  - Common condition occurring after 2–3 weeks at altitude.
  - Common at altitudes over 5,486 m (18,000 ft).
  - Sore throat, chronic cough, and severe cough spasms (severe enough to cause rib fractures).
  - Environmental, from breathing cold dry air.
  - Altitude-induced tachypnea aggravates the problem.
  - Cold-induced vasomotor rhinitis, especially at night, stimulates mouth breathing and also aggravates problem.
  - Usually not caused by infection, although infection can occur.
  - Patient will **not** have dyspnea at rest.
  - Symptomatic treatment with lozenges, mild cough suppressant, and decongestant nasal sprays. Personnel can use a mask or a porous, breathable silk balaclava as a mouth covering to reduce respiratory heat and moisture loss.
  - Maintain hydration.
- High-altitude peripheral edema.
  - Altitude-related edema of hands and face.
  - Hypoxia-induced retention of sodium and water.
  - Not considered related to AMS/HACE edema-spectrum or HAPE.
  - Decreased urine output and weight gain of 2.7–5.4 kg (6–12 lb) over several days; most evident upon awakening.
  - Diagnosis based on association of characteristic peripheral edema with ascent to high altitude; recurs consistently with repeat ascents; more common in females.
  - Differential diagnosis includes cardiogenic edema, allergic reactions, and edema of the upper extremities caused by pack straps or binding by tight clothes.
  - Prophylaxis includes salt restriction. The acetazolamide regimen used to prevent AMS is often successful in preventing peripheral edema.

- Treatment with diuretics (one 20–40 mg dose of furosemide, or 250 mg of acetazolamide every 8 h for 3 doses) and salt restriction.

- High-altitude retinal hemorrhage (HARH).
  - Bleeding from retinal vessels during altitude exposure. One of the manifestations of hypoxia-induced retinopathy.
  - Caused by BP "surges" within the distended vessels.
  - Usually asymptomatic; normally does not adversely affect military operations; however, can affect an individual soldier's vision.
  - Hemorrhages are self-limiting and resolve in 1–2 weeks after descent.
- Thromboembolic events.
  - Increased possibility of thromboembolic event with ascent to high altitude: thrombophlebitis, deep venous thrombosis, pulmonary embolus, transient ischemic attacks (TIAs), and stroke.
  - Probably result from hypoxia-induced polycythemia and clotting abnormalities but also may result from environmental and mission factors such as dehydration, cold, and venous stasis caused by prolonged periods of inactivity during inclement weather or by constriction of tight-fitting clothing and equipment.
  - Unusual below 4,267 m (14,000 ft). At very high and extreme altitudes (> 4,200 m [13,700 ft]) these events are not uncommon, and thrombophlebitis appears to be relatively common.
  - Clinical manifestations are similar to manifestations of thromboembolic events at low altitude, except for their occurrence in young and otherwise healthy personnel.
  - Prevention relies on reducing the risk factors by maintaining adequate hydration and warmth and by avoiding conditions that might cause venous stasis.
  - Evacuation to lower altitude is required. Treatment follows standard treatment guidelines, including appropriate anticoagulation. In the field setting, fractionated heparin (one dose of 250 IU/d) can be used prior to and during evacuation.
- Subacute mountain sickness.

- Prolonged deployment (weeks to months) to elevations above 3,658 m (12,000 ft).
- Common manifestations include sleep disturbances, anorexia, weight loss, fatigue, daytime somnolence, and subnormal mentation.
- Caused by failure to acclimatize adequately.
- Some relief of symptoms obtained from low-flow oxygen and from acetazolamide.
- Evacuate to lower altitude as soon as practical.
- Some degree of immune suppression and poor wound healing occurs in personnel at very high and extreme altitudes. Injuries resulting from burns, ballistics, and physical trauma should be considered more clinically significant at high altitude.

- High-altitude pulmonary edema.
  - Potentially fatal, **non**cardiogenic pulmonary edema.
  - Occurs in < 10% of personnel ascending above 3,700 m (12,000 ft).
  - Onset 2–4 days after rapid ascent to altitudes greater than 2,438 m (8,000 ft).
  - Repeated ascents and descents above 3,700 m (12,000 ft) increase susceptibility.
  - Risk factors.
    - Moderate to severe exertion.
    - Cold exposure.
    - Anxiety.
    - Young age.
    - Male sex.
    - Obesity (possibly).
  - Early symptoms (pulmonary edema).
    - Nonproductive cough.
    - Rales (few).
    - Dyspnea on exertion.
    - Fatigue.
    - Weakness with decreased tolerance for physical activity and increased time for recovery after physical exertion.
    - Resting tachycardia and tachypnea greater than induced by altitude alone.

- ♦ Once symptoms appear, HAPE can progress very rapidly (< 12 hours) to coma and death.
- ♦ Nail beds and lips may be more cyanotic than other unit members.

o Progressing pulmonary edema.

- ♦ Productive cough of frothy and sometimes pink or bloodstained sputum.
- ♦ Rales more numerous and widespread.
- ♦ Wheezing may develop.
- ♦ Lung sounds become audible even without stethoscope, especially when individual is supine.
- ♦ Orthopnea may occur (< 20%).
- ♦ Progressive hypoxemia causes dyspnea and cyanosis.
- ♦ Arterial blood gas (if available) documents hypoxemia, hypocapnia, and slight increase in pH.
- ♦ Mental status deteriorates with progressive confusion and sometimes vivid hallucinations.
- ♦ Obtundation, coma, and death occur without treatment.
- ♦ Subfebrile temperature < 38°C (100.5°F) and a mild increase in white blood cell count may be present.
- ♦ Dyspnea at rest.
- ♦ Marked hypoxia by oximetry.
- ♦ **Dyspnea <u>at rest</u> and cough should be considered to be the onset of HAPE.**

DELAY IN TREATMENT OF PROGRESSIVE PULMONARY EDEMA AT ALTITUDE USUALLY RESULTS IN DEATH.

o Treatment.

- ♦ Depends on severity.
- ♦ Immediate descent is mandatory! Descent of even a few hundred meters (300–1,000 m) can be helpful or even lifesaving in severe cases.
- ♦ Mortality can approach 50% if descent cannot be accomplished rapidly.
- ♦ Oxygen by cannula 2–6 L/min (mild), or by mask 4–6 L/min (moderate and severe). DO NOT DELAY DESCENT!

- ♦ Portable fabric hyperbaric chamber may be lifesaving—Gamow bag/Certec SA.
- ♦ Nifedipine, 10 mg tid sublingually, or 20 mg po. A second 10-mg, sublingual dose can be administered in 15–20 minutes if no improvement in symptoms is apparent, followed by 30 mg qid.

Nifedipine should not be used in lieu of descent, supplemental oxygen, or treatment in a hyperbaric bag. It may be used in conjunction with other therapies.

- ♦ Immediate descent to lower elevation; if symptoms resolve, wait at least 72 hours before attempted return to previous elevation.

Neither furosemide nor morphine sulfate should be used in the treatment of HAPE unless other more effective treatment options are not available.

- ♦ Treatment after descent, at an MTF, is directed toward ensuring adequate oxygenation and reducing pulmonary artery pressure; includes bed rest, supplemental oxygen, and nifedipine.
- ♦ Invasive diagnostic procedures such as bronchoscopy or pulmonary artery catheterization are **NOT** indicated unless clinical course deteriorates and the diagnosis is in doubt. Endotracheal intubation is seldom necessary.

o HAPE Prophylaxis.

- ♦ Nifedipine, 20 mg tid, po, 24 hours before ascent, continuing 72 hours after ascent.

- High-altitude cerebral edema.
  - o Onset following ascent is highly variable and occurs later than either AMS or HAPE. Mean duration of onset 5 days with a range of 1–13 days.
  - o Incidence lower than AMS or HAPE (< 1% of individuals making rapid ascent).
  - o Potentially fatal, uncommon (< 2% above 3,700 m). Can occur as low as 2,430 m (8,000 ft) but vast majority of cases

above 3,600 m (12,000 ft). Untreated HACE can progress to death over 1–3 days or become more fulminant with death occurring in < 12 hours.

- o Exacerbation of unresolved, severe AMS.
- o **Most often occurs in people who have AMS symptoms and continue to ascend.**
- o Signs and symptoms.
  - ♦ Most signs and symptoms are a manifestation of progressive cerebral edema.
  - ♦ **Early signs resemble AMS (these symptoms are not invariably present).**
  - ♦ Severe headache
  - ♦ Nausea
  - ♦ Vomiting.
  - ♦ Extreme lassitude.
- o Progressing **signs.**
  - ♦ Mental status changes: Confusion, disorientation, drowsiness, and impaired mentation.
  - ♦ Truncal ataxia (swaying of upper body, especially when walking). As the edema progresses, soldier may also exhibit an ataxic gait in addition to the truncal ataxia.
  - ♦ Soldier appears withdrawn, and behavior is mistakenly attributed to fatigue or anxiety.
  - ♦ Cyanosis and general pallor are common.
  - ♦ Symptoms of HAPE.
- o Untreated HACE.
  - ♦ Variety of focal and generalized neurologic abnormalities may develop: visual changes, anesthesias, paresthesias, clonus, pathological reflexes, hyperreflexia, bladder and bowel dysfunction, hallucinations, and seizures.
  - ♦ Papilledema may be present in up to 50% of the soldiers, but is **NOT** universal.
- o Coma.

Ataxia at altitude is HACE.

- o Prophylaxis.

No definitive evidence; however, due to similarity with AMS, prophylactic measures for HACE include use of staged or graded ascent, high carbohydrate diet, and use of acetazolamide.

- Treatment.
  - Immediate descent is mandatory. Definitive treatment of HACE is immediate descent. In general, the greater the descent the better the outcome. Descent of more than 300 m (1,000 ft) may be required for clinical improvement, and descents to altitudes of less than 2,500 m (8,000 ft) is optimal.
  - If descent is delayed, treatment with a portable cloth hyperbaric chamber may be lifesaving. May require at least 6 hours of pressurization in chamber.
  - Oxygen by mask or cannula 2–6 L/m; should not be used as a substitute for descent.
  - Dexamethasone, 4–8 mg initially and then 4 mg qid, po, IV, or IM. DO NOT DELAY DESCENT! Few side effects if used only 3–4 days.
  - Loop diuretics and osmotic diuretic agents, such as mannitol, urea, and glycerol, have been suggested, but there is little experience with them in this role. Careful attention is required before diuretics are used. Individual may have altitude-induced decrease in intravascular volume concomitant with cerebral edema.
  - Hospital management consists of supplemental oxygen (if needed to maintain arterial oxygen levels), supportive care, and possibly diuretics. Comatose patients may require intubation and bladder catheterization.

HACE and HAPE often coexist. Individuals with HACE will often have HAPE; however, most individuals with HAPE do not have concomitant HACE.