

Environmental Injuries

The successful prevention and control of cold, heat, and altitude injuries depend, first of all, upon vigorous command interest, the provision of adequate clothing, and a number of individual and group measures.

Cold Injuries

Trenchfoot and frostbite together have accounted for over 1million US casualties in WWI, WWII, and the Korean War.

Non-freezing Cold Injury

Chilblain

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

Pernio

- Continuum of events from chilblain.
- Exposure for >12 hours to cold/wet conditions.
- Tight-fitting footwear can shorten exposure time and increase severity of injury.
- Swelling is more severe; pain is more persistent.
- Thin, partial skin thickness, necrotic patches (from dorsum of the hands or feet).
- Plaques may slough without scarring, but may be particularly painful for months or years.

Trenchfoot

- **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 H. Shorter duration at or near 0° C result in same injury..
 - Can occur at higher temperatures from prolonged water immersion.
 - Blunt trauma of marching can produce more serious injury .
 - First symptoms are often feet becoming cold, mildly painful, and numb.
 - Tight boots increase risk of trenchfoot.
 - Common symptoms are “cold and numb” or “walking on wood”
 - **Foot may appear swollen, with the skin mildly blue, red or black.**
 - On warming limb is hot and often hyper-hydrotic
 - Upon rewarming pain is excruciating and may not respond to pain medication, including morphine.

- As time progresses, liquefaction necrosis occurs distally, but tissue proximal may also be compromised.
- No sharp line of demarcation of dead and 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 trenchfoot.
- Post-injury sequelae include pain, numbness, loss of proprioception, and cold feet.
- Hyperhydrosis with subsequent perinechial fungal infections are common.
- Life-long, life-changing injury.

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 débride
- Pain medication. The only effective approach is amitriptyline 50-150 mg at bedtime. Other analgesics are utilized but tend to be less effective.
- Any blisters should be left intact. Ruptured blisters require excellent antisepsis.
- Systematic antibiotics and tetanus prophylaxis are indicated when there are dead tissues, as within any other contaminated wound, or when there is evidence of infection.
- **Debridement of necrotic tissue may be required in trenchfoot.**
- Macerated or damaged skin requires topical antibacterial precautions.
- Avoid trauma.
- Early mobilization is vital to prevent long-term immobility.
- Return to duty is protracted; may require evacuation as it leads to weeks to months of pain and disability.
- Long-term sequelae are very common and include sensitivity to the cold (secondary Raynaud's), chronic pain, neurological impairment, and hyperhydrosis.

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.

- **Clinical Appearance.**
 - Skin initially becomes numb and feels stiff or woody.
 - Mottled, bluish, yellowish, “waxy”, or “frozen.”
 - Depth of involvement cannot be determined initially, nor does degree affect initial therapy.

Frostbite Grading.

- First degree: Erythema/edema at distal involved sites, no vesicles.
- Second degree: Clear fluid filled vesicles, extend to distal areas.
- Third degree: Deeper vesicles, purple/hemorrhagic.
- Fourth degree: Involvement of deeper structures, may be difficult to determine initially.
- A more clinically useful grading typically divides injuries into superficial (frostnip or firstdegree) or deep (Second or higher degree)
- After re-warming, the appearance and location of vesicles will allow a more accurate assessment of the severity of injury.

Superficial Frostbite.

- Involves only the skin with swelling, mild pain, and minor joint stiffness.
- No blisters form.
- Can be managed by nonmedical personnel simply by rewarming.

Deep Frostbite

- Involves deeper tissues to include bone.
- White-hard, anesthetic, blanched, and inflexible.
- Skin will not move over joints.
- Upon rewarming, there is great pain, and a blue-gray to burgundy color change.
- Blisters form and are clear, fluid-filled, or hemorrhagic (latter indicating a more severe, deeper injury). 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.
- Post-injury sequelae include: Raynaud's, pain, paraesthesias, hyperhidrosis, loss of proprioception, cold/discolored feet, and gait modification.

Field Treatment (first aid).

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.

Deep Frostbite.

- Prevent from further cooling. Apply dry, sterile bandage and elevate.
- Protect from refreezing during evacuation. Get definitive medical care

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

MTF Treatment.

- **The outcome of a frozen extremity is not directly related to overall time frozen, but more importantly to the method of rewarming and any subsequent refreezing.**
 - If this requires walking on frozen feet then no attempt at rewarming should be initiated, and the patient should ambulate on the frozen extremities.
 - 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.
- **Rapid rewarming (without the possibility of re-freezing) is the treatment of choice.**
 - Immerse in gently circulating water (whirlpool bath) at 40° 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, then 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.
 - Oral alcohol has been recommended by European sources as an adjunct. The alcohol reduces the need for analgesia and may improve outcome.
 - Other drug regimes remain unproven.
 - After rewarming, edema will appear within a few hours and vesicles within the next 6-24 hours.
 - Intensive mobilization is essential to avoid long-term immobility.

Vesicles.

- Frostbite vesicles are typically left intact.
- Debridement is not recommended.

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 should be applied.
- Cigarette smoking/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 days on average); therefore, all but the most trivial should be evacuated to more definitive care as soon as possible
- Early surgery is only indicated in the most severe freeze-thaw-refreeze where massive tissue destruction has taken place, and in some more severely infected cases. Normally surgery should be delayed for at least six months.

Due to the inability to reliably predict the outcome in the post-thaw 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 a whole-body cooling below 35°C. Degree of hypothermia is further defined according to the bodies core temperature and the clinical effects seen in a given temperature range.

Mild Hypothermia >32°C.

- Shivering, hyperreflexia.
- Amnesia, dysarthria, poor judgment ataxia, apathy.
- Cold diuresis.

Moderate Hypothermia 28°C - 32°C.

- Stupor, loss of shivering.
- Onset of atrial fibrillation (a-fib) and arrhythmias.
- Progressive decrease in LOC, respiration and pupillary reaction, eventual pupil dilation.

Severe Hypothermia 20°C-28°C.

- Increased incidence of ventricular fibrillation (v-fib), often spontaneously.
- Loss of motion and reflexes, areflexic at approx 23°C.
- Marked hypotension/bradycardia.

Profound Hypothermia <20°C.

- Asystole.
- Lowest adult survival from accidental hypothermia 13.7°C.

Prehospital (Field) Treatment.

Awake Patients

- Remove wet clothing, dry and insulate

- Oral sugar solutions to hydrate.
- Walk out or transport to MTF. (This should be attempted only if it is the only alternative, as it is likely to worsen the condition).
- Although walking may deepen hypothermia due to the return of peripheral colder blood to the core, adequate pre-hydration decreases the post-exposure cooling.

Comatose Patients

- Patient should remain horizontal and be handled gently to avoid inducing arrhythmias; do not massage
- IV fluids, warmed to 40-42°C, if possible.
- Do not use lactated ringers, D5NS is fluid of choice; the cold liver cannot metabolize lactate.
- Remove wet clothes, dry, insulate and add an outer vapor barrier. Wrap patient in multiple layers of insulation.
- Limit active rewarming principally to body's center/core only.
 - Heated (40-45°C), humidified air/O₂ 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 ventilation may be performed.
- If apnic ventilate mouth-to-mouth for 5 minutes. Recheck for cardiac activity and spontaneous respiration. If none, then consider CPR if it will not compromise the rescue effort or the rescuer.
- When commencing CPR in hypothermic patients, frequency of thorax compression is to be reduced to 50-80 per minute. Vigorous compression activities must be avoided to vulnerable myocardial tissue.

Medical Treatment

- Ventilate
- Warmed intravenous fluids (lactate and potassium-free); glucose and insulin, if not shivering.
- Careful correction of acid/base balance.
- 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 re-warm past 33°C when potassium levels are high and pH is low.

Cardiopulmonary Resuscitation.

- Auscultate and palpate for at least 1-2 full minutes before initiating CPR.
- Initiate CPR on a clinically pulseless patient only if no cardiac monitor available.
- If cardiac monitor shows any electrical complexes DO NOT initiate CPR.
- CPR is appropriate if pt is in v-fib or asystole.

- CPR is contraindicated if patient has any signs of life, has obvious lethal injuries, or is frozen solid.
- CPR should be undertaken even if it can only be intermittent during transport.

Defibrillation.

- If patient is in v-fib defibrillate up to 3 times; if unsuccessful patient must be warmed prior to further attempts.
- Re-warm core to 32°C and attempt defibrillation (360 joules). Continue re-warming and repeat. Defibrillate after every one degree C rise in temperature
- Bretylium is the ACLS drug of choice; other drugs are ineffective or increase the risk of arrhythmias.
- Amiodarone may be effective on a theoretic basis, but has no studies to support its use, as well as 1 animal study showing a lack of efficacy
- IV magnesium (2gms) infusion may be beneficial.

Temperature measurement.

- Rectal temperature is most commonly used, but may lag true core temperature.
- Esophageal temperature is the most accurate if the temperature probe is 24cm below the larynx.

Afterdrop.

- As the body cools, the periphery vasoconstricts causing pooling of cold acidotic blood.
- Rewarming the periphery rather than the core causes an inrush of this blood into the core, further dropping the core temp and worsening cardiac instability.

Rewarming: Aggressiveness and type of rewarming is based on severity of patients hypothermia and need to prevent afterdrop.

Mild hypothermia (>30°C) requires only passive or noninvasive active warming techniques. As severity increases techniques become predominantly active and more invasive.

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

Mild Stable Hypothermia.

- **Insulation.**
- **Heat lamps.**
- **Warmed IV fluids.**
- **Forced air (Bair Hugger).**
- **Consider arteriovenous anastomoses warming (AVA).**
 - (AVA)
 - Immerse hand, forearms, feet, calves in water heated to 44-45°C.

- Opens AVAs in the digits causing increased flow of warmed venous blood to the heart and decreased afterdrop.

Moderate: Hemodynamically Stable Hypothermia.

- All of the above for mild stable hypothermia.
- Forced air into rigid frame over central body
- Heated humidified O₂.

Severe Hypothermia: Hemodynamically stable.

- All of the above for mild and stable hypothermia.
- Bladder lavage.
 - Of lesser value due to small surface area for heat exchange.
 - 200-300cc of fluid at 40-42°C is inserted via foley catheter; fluid removed and replaced every 20 mins as required.
- Gastric lavage is not recommended.
- Venovenous rewarming.
 - Hemodialysis catheter is placed.
 - Blood is passed through a hemofiltration system and pump with an attached countercurrent heat exchanger and returned to the venous system.
- Continuous Arteriovenous rewarming.
 - Femoral artery and venous 8.5 foley catheters are placed.
 - Patient requires a BP of 60mm or higher.
 - This sets up a functional arteriovenous (AV) fistula.
 - Blood flows through heparinized tubing through a heat exchanger (such as the Level 1 infuser.)
- Peritoneal Lavage.
 - A peritoneal catheter or equivalent is placed into the peritoneal cavity.
 - 2 L of fluid (NS/LR/ Or 1.5% dialysate) heated to 40-45°C is instilled for 20-30min.
 - Fluid is removed by aspiration and process repeated as required.
 - Process compatible with CPR.

Severe Hypothermia: Unstable or profound hypothermia or any degree with failure to warm by other techniques.

- Any of above (severe hypothermia: hemodynamically stable).
- Closed thoracic lavage.
 - A useful choice in patients in arrest.
 - Delivers heat directly to the mediastinum
 - Chest tubes are placed at the anterior axillary line 2nd intercostal space (ICS) and post axillary line 6th ICS.
 - Saline is infused at 40-42°C anteriorly and drains posteriorly.
 - Tubes can be placed bilaterally; avoid left side if patient is not already in asystole/v-fib.
 - CPR can continue as lavage is ongoing.
- Open thoracic lavage.
 - Useful in severely hypothermic patients in arrest.

- A standard left sided thoracotomy is performed.
- The pericardium does not need to be opened.
- The mediastinum is irrigated with fluid at 40°C.
- **Cardiopulmonary bypass.**
 - The definitive gold standard treatment for severe hypothermia.

Heat injury.

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

Heat Injury Prevention.

- Easier to prevent than to treat.
- Occurs most commonly in unacclimatized individuals.
 - Acclimatization to heat requires 7-14 days.
 - Predeployment training in artificially warm environments does aid heat acclimatization.
 - 2 hours per day (may be split into 1 hour sessions) of progressively more difficult exercise sufficient to induce moderate sweating each day will maximize acclimatization
 - 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 (for example, 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 liters (1 to 2 percent) 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 liters/hr.
- Daily oral rehydration with hypotonic solutions should not exceed 12 liters/day or hyponatremia may develop.
- 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 awaking and having to dress to urinate or soldiers may not drink prior to a convoy as no rest stops are planned.)

- MOPP gear will increase fluid losses and the incidence of heat injuries.
- Consumption of the salt packet in MREs with the meal will provide adequate daily salt requirements in most cases
- Salt supplements not routinely required and are only recommended in rare instances where adequate rations are not consumed.
- In situations of extreme exercise intensity in hot environment “sports drinks” may be beneficial
- 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 which 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 or altered mental status. Degree of injury appears to relate to both the degree of temperature elevation and duration of exposure.

Clinical presentation.

- A true emergency. Involves Five organ systems: brain (encephalopathy), hemostatic, liver, kidneys and muscles.
- Encephalopathy ranges from syncope and confusion to seizures or coma with decerebrate rigidity.
- Prodromal symptoms include dizziness (lightheadedness), weakness, nausea, confusion, disorientation, drowsiness, irrational behavior, syncope, seizures, or coma..
- Collapse is a universal feature of heatstroke.
- An individual who collapses after exercise in a warm environment with a core temperature of $\geq 40.5^{\circ}\text{C}$ has heatstroke.
- Casualties who are **unconscious** and have a core temperature of $\geq 39^{\circ}\text{C}$ have heatstroke.
- Core temperature is often lower upon arrival at a treatment area.
- Differential diagnosis: infection (particularly meningococemia and P. falciparum malaria), pontine or hypothalamic hemorrhage, drug intoxication

- (cocaine, amphetamines, phencyclidine, theophylline, tricyclic antidepressants), alcohol or sedative withdrawal, severe hypertonic dehydration, and thyroid storm.
- Seizures occur frequently (>50% cases) with heatstroke
 - Hinder cooling efforts.
 - Treat with diazepam 5-10mg.

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

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 early evacuation to medical facilities with intensive care capabilities. Active cooling should be started immediately and continued during evacuation.

Treatment.

- Any effective means of cooling is acceptable.
- Cool water immersion (15-20°C) with skin massage is the classic technique. It provides rapid cooling. Closely monitor patient for and prevent shivering
- A variety of evaporative cooling techniques have been used, and, while evaporative cooling is less effective, the ice immersion method may prevent safe cardiac monitoring or rapid resuscitation
- **Cooling by spraying cool water over the body and vigorous fanning can be effective.**
- Cooling with cool water soaked blankets and vigorous fanning is effective.
- Do not use alcohol for cooling as toxicity can occur.
- Circulating cooling blankets (not available in the field) will also lower body temperature.

The goal of treatment is to effect a rapid lowering of the core temperature to 38.3°C, without inducing shivering.

- Rectal temperature should be closely monitored during cooling. Discontinue cooling efforts when core temperature reaches **38.3°C** to avoid hypothermia.
- **Aspirin and acetaminophen should not be given** to casualties of heatstroke.
- Aggressive fluid resuscitation is not required. Fluid requirements of 1 liter in the first 30 minutes, with an additional 2 or more liters in the next 2 hours. Since heat stroke patients are frequently hypoglycemic, the initial fluid should include dextrose. (chilled fluid is of limited benefit).
 - Base further hydration on fluid status/urinary output (Foley required.)
 - Over hydration can lead to congestive heart failure, cerebral edema, and pulmonary edema in the heat-stressed lung.

- If shivering develops, treat with diazepam (5-10mg IV) or chlorpromazine (50mg 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 their 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, directed at minimizing cerebral edema by avoiding fluid over replacement and assuring hemodynamic, thermal, and metabolic stability.

Complications.

- Rhabdomyolysis and secondary renal failure
 - Elevated Creatine Phosphokinase (CPK) (in the thousands).
 - Administer IV fluid and possibly furosemide to maintain urinary output > 50cc/h (Assurance of adequate renal perfusion and urine flow will moderate the nephrotoxic effects of myoglobin and uric acid)
 - Alkalinize urine with Na bicarbonate IV (2 amps NaHCO₃/liter D₅W)
- Coagulopathy due to hepatic injury.
 - Hepatic injury is common. 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 PT time at 48-72 hours post injury.
 - Thrombocytopenia and disseminated intravascular coagulation (DIC) peak at 18-36 hours post injury.
 - Beware of coagulopathy timeframe when planning evacuation
- 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 DIC 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. Immunoincompetence and infection are late complications particularly in patients with severe renal failure.

- Hyperkalemia is the most life-threatening early clinical problem. Measurement of plasma (potassium) is an early priority.

Heat Cramps.

Clinical Presentation.

- Brief, intermittent, recurring, and often excruciating tonic muscle contractions
Preceded by palpable or visible fasciculation and lasts 2-3 minutes.
- 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.

Treatment.

- Mild cases can be treated with oral 0.1-0.2% salt solutions. Salt tablets should not be 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 grams, 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 versus Heat Stroke.

The distinction between heat exhaustion and heat stroke, is often difficult. Individuals who do not respond dramatically to rest and fluid-electrolyte repletion should be observed for 24 hours with laboratory surveillance for the delayed complications of heat stroke. Encephalopathy, coagulopathy, or persistent elevations of body temperature suggest probable severe heat stroke. Since renal and hepatic complications of heat stroke can be delayed 48-72 hours, any evidence of renal or hepatic injury during the initial 24 hours of observation should lead to the presumptive diagnosis of heat stroke.

Heat Exhaustion

Clinical Presentation.

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

- Oliguria, clinical dehydration, ataxia, tachycardia and tachypnea with symptomatic hyperventilation with acroparesthesia and carpopedal spasm.
- Syncope may occur.
- Core temperature is < 39°C, even at time of collapse.

Treatment.

- Oral rehydration (if patient is not vomiting).
- Parental fluids produce more rapid recovery: No more than 1000ml NS bolus without laboratory surveillance; After 2.5 liters of plain saline, add dextrose as a source of energy (2.5% glucose/0.45% NaCl); subsequent fluid replacement should be D5/0.5 NS or D5/0.2 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, since 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

Miliaria Rubra, Miliaria Profunda, and Anhidrotic Heat Exhaustion

- Subacute (miliaria rubra) pruritic inflamed papulovesicular skin eruption which appears in actively sweating skin exposed to high humidity. Becomes generalized and prolonged (miliaria profunda); lesions are truncal, non-inflamed 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 increases the risk of heat illness 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 have 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. Individual is more at risk for heat exhaustion and high risk of heat stroke in conditions tolerated by others.

- Evacuation to a cooler environment until restoration of normal eccrine gland function.

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.)
- 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.

Heat Edema

- Seen early in heat exposure.
- Plasma volume expanding to compensated 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.

Sunburn

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

Heat Tetany

- Rare; occurs in individuals acutely exposed to overwhelming heat stress.
- Extremely severe heat stress induces hyperventilation; principal path physiologic etiology.\
- Manifestations include: respiratory alkalosis, carpopedal spasm, and syncope.
- Treatment: remove from heat source and control of hyperventilation (recreating 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 Cerebral Edema (HACE) and High Altitude Pulmonary Edema (HAPE).

Altitude Basics.

The occurrence of altitude illness is based on altitude and rapidity of ascent.

- Physiologic changes due to altitude begin to occur at just over 1,500m.
- These changes are the body's attempt to acclimatize to altitude.
- Symptoms occurring below 2,250m 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 14,500ft in 36 hours can result 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 percent of soldiers who ascend rapidly (<24 hours) to altitudes between 1,829 meters to 2,446 meters (6,000 to 8,000 feet) experience some mild symptoms
 - Rapid ascent to elevations of 3,670-4,300 m (12,000 to 14,000) feet result in moderate symptoms in over 50 percent of the soldiers and 12-18 percent may have severe symptoms.
 - Rapid ascent to 5,333 m (17,500 feet) causes severe, incapacitating symptoms in almost all individuals.

Descent Basics.

- Almost everything improves with descent if it is not delayed.
- For illness requiring descent one should try to descend at least 1,000m if not more.
- A Gamow® bag (US) (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 also linger for several days.
- Victims of HACE and HAPE should not reascend until 72 hrs after symptoms abate and then must ascend much slower than previously.
- Have a victim descend at the earliest sign of HACE or HAPE, before they become moribund and incapable of aiding 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.

- If one became ill previously at a given altitude they 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.
- If a rapid ascent to altitude must be made, use prophylaxis against AMS.

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 feet) and limiting the sleeping altitude to no more than 305 m (1,000 feet) per day above previous night's sleep altitude.
 - Combination of both staged and graded ascent is the safest and effective prevention method.
- Diet: High carbohydrate diet (<70 per cent of total energy intake as carbohydrate) (stimulation of ventilation through increased carbon monoxide produced from metabolism of carbohydrates).
- Acetazolamide 125 mg -250 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 percent of soldiers and reduces symptoms in most others.
- **Contraindicated in sulfa allergy.**
- If required, Dexamethasone 4mg PO QID 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 4000M), short duration (<48 hrs) profile (i.e. raids, rescues).
- Ginkgo Biloba a herbal supplement at 120mg PO BID has been shown to be effective for AMS prophylaxis but as a herbal supplement has no FDA status.
- It started 5 days prior to ascent and is not as effective as acetazolamide, though its side effects are minimal.

High Altitude Pharyngitis and Bronchitis

- Common condition occurring after 2-3 weeks at altitude.
- Common at altitudes over 5,486 m (18,000 feet).
- 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 to 5.4 kg (6-12 lbs) over several days; most evident upon awakening.
- Diagnosis based upon 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 and the acetazolamide regimen used to prevent AMS is often successful for peripheral edema.
- Treatment with diuretics (one 20-40 mg dose of furosemide or 250 mg of acetazolamide every eight hours for three 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 blood-pressure "surges" within the distended vessels.
- Usually asymptomatic; normally do not adversely affect military operations, however can affect an individual soldier's vision.
- Hemorrhages are self-limiting and resolve in one to two 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 include 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 feet). At very high and extreme altitudes (>4,200 m) these events are not uncommon, and thrombophlebitis appears to be relatively common.
- Clinical manifestations are similar to manifestations at low altitude, except for the occurrence in young and otherwise healthy personnel.
- Prevention relies on reducing the risk factors by maintain adequate hydration and warmth and by avoiding by avoiding conditions that might cause venous stasis.
- Evacuation to lower altitude is required. Treatment follows standard treatment guidelines, including appropriate anticoagulation. In field setting, fractionated heparin (one dose of 250 IE/day) can be used prior to and during evacuation.

Subacute Mountain Sickness

- Prolonged deployment (weeks to months) to elevations above 3,658 m (12,000 feet).
 - 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 acetazolamide.
 - Evacuate to lower altitude as soon as practical.
- **Immune Suppression and Poor Wound Healing**
 - Some degree of immune suppression 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.

Acute Mountain Sickness (AMS).

- This 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 lightheadiness.
- 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).

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 feet) to high altitude or high altitude to higher altitude in the previous 24 to 48 hours.
- Differential diagnosis includes viral gastroenteritis, hangover, exhaustion, dehydration, carbon monoxide poisoning, and HACE
- Presence of neurological symptoms such as incoordination, ataxia and excessive lethargy or cognitive dysfunction is indicative of progression to HACE, which requires immediate therapeutic intervention.

Treatment.

- AMS alone does NOT mandate descent.
- Remain at the same elevation (do not ascend) until symptoms abate.
- Acetazolamide 125 mg q.i.d to 500 mg, t.i.d, p.o -- do not use in patients with sulfa allergies. (If already receiving a preventive dose of acetazolamide (1,000 mg/day) and still symptomatic, then 500 mg can be added with caution.
- Dexamethasone in doses of 2 to 4 mg every 6 hrs (has the same potentially serious side effects as when used as prophylaxis). Symptoms may recur when medication stopped.
- O₂ by nasal cannula 2-6L/min (severe headache).
- Do NOT advance sleeping altitude.
- Symptomatic treatment with ASA, acetaminophen, ibuprofen for headache, prochlorperazine for nausea/vomiting
- Minimalize utilization of sleeping agents at altitude, they can worsen illness. Acetazolamide for sleep disorders, 250 mg, q.i.d or t.i.d, p.o

High Altitude Pulmonary Edema (HAPE).

- Potentially fatal, noncardiogenic pulmonary edema.
- Occurs in < 10% ascending above 3,700m.
- Onset 2-4 days after rapid ascent to altitudes greater than 2,438 m (11,500 feet).
- Repeated ascents and descents above 3,700m 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.
- **Dyspnea at rest.**
- 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.

Progressing Pulmonary Edema

- Productive cough of frothy and sometimes pink or bloodstained sputum.
- Rales more numerous and widespread.
- Wheezing may develop.
- Lung sounds progress to an audible (without stethoscope), especially when individual is supine.
- Orthopnea may occur (<20%)
- Progressive hypoxemia causes dyspnea and cyanosis.
- Marked hypoxia by oximetry
- Mental status deteriorates with progressive confusion and sometimes vivid hallucinations.
- Obtundation, coma, and death occur without treatment.
- Subfebrile temperature <38° (100.5°) and a mild increase in white blood cell count may be present

Dyspnea at rest and cough should be considered to be the onset of HAPE.

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

Treatment.

- Depends on severity.

- 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 can not be accomplished rapidly.
- O₂ by mask or cannula. 2-6 L/min (mild) or 4-6 L/min by mask (moderate and severe) DO NOT DELAY DESCENT
- Portable fabric hyperbaric chamber may be lifesaving.
- Nifedipine 10 mg tid sublingually or 20 mg orally. A second 10 mg sublingual dose can be administered in 15-20min if no improvement in symptoms is apparent. Followed by 30 mg q.i.d.

Nifedipine should not be used in lieu of descent; supplemental oxygen, or treatment in a hyperbaric bag is appropriately used in conjunction with each of the other therapies.

- Move to lower elevation; if resolved, wait at least 72 hrs 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 directed toward ensuring adequate oxygenation and reducing pulmonary artery pressure: 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.

HAPE Prophylaxis.

- Individuals who are susceptible to HAPE who cannot ascend slower than the rate which previously induced HAPE should utilize prophylaxis.
- Nifedipine 20 mg t.i.d, p.o, 24 hrs before ascent, continuing 72 hrs after ascent.
- Salmeterol 125 ug BID the day before ascent and the first few days at altitude has been shown to reduce HAPE in susceptible individuals but is not FDA approved for this use.

High Altitude Cerebral Edema (HACE).

- 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 to 13 days.
- Incidence lower than AMS or HAPE (<1 percent of individuals making rapid ascent).
- Potentially fatal, uncommon (< 2% above 3,700m) Can occur as low as 2,430 m (8,000 feet) but vast majority of cases above 3,600 m (12,000 feet).. Untreated HACE can progress to death over 1 to 3 days or become more fulminant with death occurring in <12 hours.
- Exacerbation of unresolved, severe AMS.

- **Most often occurs in people who have AMS symptoms and continue to ascend.**

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.
- **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.
 - Soldiers appear withdrawn and behavior is mistakenly attributed to fatigue or anxiety.
 - Cyanosis and general pallor are common.
 - Symptoms of HAPE.
- **Untreated HACE.**
 - Variety of focal and generalized neurologic abnormalities may develop
 - Papilledema may be present in up to 50 percent of the soldiers, but is *not* universal.
- Coma.

Ataxia at altitude is HACE.

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 feet) may be required for clinical improvement, and descents to altitudes of less than 2,500 m (8,000 feet) 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.
- O₂ 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 q.i.d, p.o, i.v., or i.m. 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.
- Hospital management consists of supplemental oxygen (if needed to maintain arterial oxygen levels), dexamethasone, 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.