Environmental

Hypothermia

- Predispositions: extremes of age (kids due to ↑ body surface area (BSA), elderly due to comorbidities), altered mental status (psych/behavioral), intoxication (**EtOH is major cause**)

Non-Freezing Cold Injuries

Chilblains (aka Pernio)

- How: exposure to cold/damp → red/blue edematous plaques → itchy/burning pain
- Tx: **warm/dry**, topical steroid for itch, can try nifedipine but probably overkill

Immersion foot ("Trench foot")

- Who: homeless, psych
- How: prolonged immersion cold H2O → vasoconstrict → ischemia (gangrene if severe)
- Tx: **warm/dry, PREVENTION** (wear dry socks!)

Freezing Cold Injuries

- How: **Hunter's Response** = cycles of vasoconstriction/dilation (vasoconstrict to keep body warm which makes skin cold; when skin really cold → vasodilate)
- Balance core temp vs limb ischemia → ultimately core temp wins → limb ischemia

Frostnip

- NO ice formation, NO tissue loss
- Note: cannot initially distinguish frostnip/frostbite; if no tissue loss, it's frostnip
- Tx: rewarm

Frostbite

- YES ice formation, TISSUE LOSS
- Phase I (the freeze): ice formation extracellular/intracellular space → cellular dehydration → cell death
-

    - Like a crush injury (cells shredded by ice formation)
- Phase II (reperfusion injury): rewarming → arachidonic acid cascade 2/2 leak from damaged endothelium → prostaglandin (PG)/thromboxane (TXA) release
    - PG release → leaky capillaries → edema/blisters
    - TXA release → platelet agg + vasoconstriction → dry gangrene
- Graded by tissue depth (hard to predict initially)
    - $1^{st}/2^{nd}$ degree = superficial = dermis only = good prognosis
    - $3^{rd}/4^{th}$ degree = deep = SQ to bone = poor prognosis
- Tx: **rapid rewarming w/ warm H2O**
- **Tx Pearls**: ideal **H2O temp 107°F**, NO DRY HEAT (skin insensate so can cause thermal injury), refreezing after thaw very bad (↑ tissue destruction), rewarming painful, ok to debride clear blisters, leave hemorrhagic blisters alone, update tetanus, **delayed debridement** ("frostbite in January, amputate in July")

Generalized Hypothermia = <95°F (<35°C)

- Who: extremes of age (kids ↑BSA, elderly comorbidities)
- Causes: accidental, metabolic (adrenal/thyroid/hypoglycemia), sepsis, drugs (EtOH #1)

Hypothermia stages: mild, moderate, severe

- **Mild: 90-95°F**, excitation response (↑HR/BP/RR, **shivering**), mild confusion
- **Moderate: 86-90°F**, physiologic slowing, **NO shivering**, **AMS**/ataxia, **Osborn J-waves** (also seen in hyperCa and head injury)
- **Severe: <86°F**, **dysrhythmias** (brady>slow Afib>Vfib>asystole), irritable myocardium (no invasive heart procedures e.g. high central venous catheter), bronchorrhea 2/2 ↓ cilia action, fixed/dilated pupils, cold diuresis (diffuse vasoconstriction → functional bolus; cold impairs enzymes → can't concentrate urine)

Hypothermia Physiology

- Coagulopathy: **functional coagulopathy** (enzymes in clotting cascade don't work when cold) despite nl labs (lab heats blood up to test it)
- OxyHb curve left shift (↑ O2 affinity, ↓ O2 delivery)
- Hyperglycemia common (insulin ineffective <88°F); **don't give insulin until warm**

Hypothermia Treatment

- Avoid invasive cardiac procedures (irritable myocardium!)

- CPR/intubation per usual
- **Life threatening arrhythmias need heat** (electricity/drugs don't work on popsicles!)
- FOCUS on REWARMING; can try 1 shock for Vfib
- **Passive rewarming** (mild hypothermia) = insulating pt (blanket)
- **Active rewarming** (mod/severe hypothermia) = apply exogenous heat
    - **Noninvasive (must be hemodynamically stable)** = bair hugger, warm O2, warm IVFs (107°F)
    - **Invasive = (use if hemodynamically unstable)** = warm air on vent, lavage (bladder, stomach, peritoneal, chest tube), thoracotomy → dump hot H2O on heart (a county legend), **extracorporeal warming (most effective)**
- A word on lavages:
    - Peritoneal lavage: incision linea alba → place cordis → run warm H2O
    - Mediastinal lavage: place inlet (ant axillary) and outlet (post axillary)
    - Rectal lavage: don't recommend…gets messy
- *__Rewarming goal = 86°F in arrest:__ *if >86°F and still in arrest, likely not the cause*

High Altitude Emergencies

- Spectrum of disease with same pathophysiology
    - Acute Mountain Sickness = mil
    - High altitude pulmonary/cerebral edema = severe
- Who: rapid ascent, sleeping at high altitude, poor hypoxic ventilatory response, low vital capacity
- Neither young age nor fitness level protective (shrunken elderly brain may be protective!)

High Altitude Pathophysiology

- **Hypobaric hypoxia**: ↑ altitude → ↓ barometric pressure → ↓ O2
    - Hypoxia → pulmonary vasoconstriction → PHTN → HAPE
    - Hypoxia → cerebral vasodilation → AMS/HACE

Acclimatization

- Hypoxic ventilatory response = hypoxia stimulates carotid bodies → hyperventilation
    - People who hyperventilate do better at altitude
    - Respiratory alkalosis → bicarb diuresis to compensate
- **Acetazolamide** (Diamox) = carbonic anhydrase inhibitor → bicarb diuresis → metabolic acidosis → **triggers compensatory hyperventilation → speeds acclimatization**
    - Best to start taking 24hr before ascent, but works anytime

Acute Mountain Sickness (AMS)

- **Very common**
- How: **"a little brain swelling"**
- When: >2000m (6500ft)
- Sx: headache, nausea/vomiting, anorexia, fatigue; onset in hours, lasts 2-3 days

High Altitude Pulmonary Edema (HAPE)

- **Most common killer**
- How: pulmonary hypertension → leaky capillaries → **noncardiogenic pulmonary edema**
- When: >3000m (9500ft)
- Sx: pneumonia-like (don't get faked out); **dyspnea at rest (classic finding),** cough, rales, hypoxia, fever

High Altitude Cerebral Edema (HACE)

- **Most severe, uncommon**
- How: **"a lot of brain swelling"**
- When: >4500m (14000ft)
- Sx: **ataxia (early finding),** lethargy, altered mental status, seizure

High Altitude Treatment

- **All 3 disorders 2/2 hypoxia so all include tx w/ O2 and DESCENT**
  - AMS: diamox, dexamethasone (↓ vasogenic edema), NSAIDs
  - HAPE: nifedipine/PDEIs (↓ pulmonary HTN), hyperbaric oxygen therapy (HBOT, e.g. Gamow bag)
  - HACE: diamox, dexamethasone, HBOT
- **Never ascend until sx resolve**
- **Small Δ altitude can make large Δ sx**
- Gamow bag = inflatable pressure bag that ↑ O2 environment
  - Same physics as HBOT or descent

Dysbarisms

- 2 classes: **barotrauma** (localized, pulmonary overpressurization syndrome [POPS], air gas embolism [AGE]), **dissolved gas problems** (decompression sickness [DCS], nitrogen narcosis, O2 toxicity)
- **Boyle's law (explains barotrauma):** volume = 1/pressure; ↑ depth → ↓ volume

    - **Note: Δ volume greatest near surface**; 50% Δ volume in first 10m (30ft); can have "squeeze" if ascend/descend improperly at only 30ft! (contrast going from 150-120ft = small Δ volume)
- **Henry's law (explains DCS)**: ↑ pressure → ↑ gas pushed into solution (tissues)

Barotrauma

Descent = Localized ("squeeze")

- **Barotitis Media** = most common
    - How: ↑ depth → ↑ pressure on tympanic membrane (TM); normally can relieve w/ Frenzel maneuver, if have URI and blocked eustachian tubes cannot equalize
    - Sx: pain, vertigo w/ rupture
    - Tx: antibiotics if TM ruptures; dry ear precautions
- **Barotitis Externa**
    - How: external auditory canal (EAC) blocked (e.g. cerumen, dive hood, ear plugs) → cannot equalize → ↑ EAC pressure w/ ↑ depth → EAC edematous/hemorrhagic
    - Tx: corticosporin; dry ear precautions
- **Barotitis Interna**
    - How: equalize EAC too much → good ear that isn't blocked overdistended → rupture/bleeding round window
    - Sx: ↓ hearing, vertigo, tinnitus, nystagmus
    - Tx: ENT evaluation
- **Sinus Squeeze** = sinus blocked → same pathophys as barotitis externa → epistaxis
- **Mask Squeeze** = if don't equalize pressure between face/mask → petechiae, hemorrhagic chemosis

Ascent = Localized ("reverse squeeze"), POPS, AGE

- **Barodontalgia** = air in cavity/filling expands w/ ascent → pain, tooth can pop out
- **Alternobaric vertigo** = unequal mid ear pressure → vertigo
- **GI barotrauma** = excess gas in gut → burping/flatus
- **Pulmonary Over-Pressurization Syndrome (POPS)**
    - How: ascending w/o exhaling → alveoli rupture → pneumomediastinum (common), pneumothorax/tension PTX (rare)
    - Sx: may be delayed; hoarse, neck full, crepitus, chest pain; CXR continuous diaphragm sign
    - Tx: conservative, treat PTX
- **Air Gas Embolism (AGE) = severe POPS**
    - Second most common cause of diver death after drowning

- How: same pathophys as POPS → air bubbles forced into pulmonary veins
- Sx: arrest, MI, CVA, seizure, altered mental status
- Note: AGE quick onset w/in min after ascent, DCS gradual onset w/in hours; **if unconscious w/in 10min of surfacing, assume AGE**
- Tx: IVF (minimize vascular occlusion by air), O2 (minimize ischemia, ↑ nitrogen gradient to leave blood), HBOT

Dissolved Gas Problems

Nitrogen Narcosis = "rapture of the deep"

- How: usually >100ft (30m) nitrogen enters CNS/PNS, acts as general anesthetic
- Sx: drunk, "dumb" behavior
- Note: nitrogen narcosis won't kill you, but behavior will (e.g. drown because confused and run out of air)
- Tx: ascent

O2 toxicity

- How: ↑ depth → ↑ pO2 → O2 toxicity; can prevent by ↓ %O2 tank output
- Who: industrial divers diving very deep (300-400ft, 90-125m) or recreational divers using Nitrox (↑ %O2 in tank allows ↑ bottom time since ↓ nitrogen exposure)
- Sx: muscle twitch, nausea, Δ vision, SEIZURE AT DEPTH
- Tx: ascent or↓%O2

Decompression Sickness (DCS)

- How: nitrogen not H2O soluble but forced into solution (tissues) at high pressure → on rapid ascent nitrogen pops out of tissues before able to be absorbed by lungs
- Who: long/deep/multiple dives
- When: gradual onset, 95% w/in 3hr

DCS Type I

- **Musculoskeletal symptoms** = most common
- Sx: arthralgias (**"the Bends"**), cutis marmorata rash 2/2 lymphatic obstruction by gas bubbles

DCS Type II

- **Neurologic, Pulmonary symptoms** = more severe
- Sx: bubbles in spinal cord (usually thoracic/lumbosacral) → patchy cord xx (bladder dysfxn, paraplegia, paresthesias); bubbles in cerebellum → ataxia (**"the Staggers"**); bubbles in lungs (functionally a PE) → SOB/CP (**"the Chokes"**)

- Tx: same as AGE; IVF, O2, HBOT
- Note: **time to HBOT important**; if wait too long fibrin/platelet shell forms around bubble → Sx don't resolve w/ HBOT

Heat Illness

- The body uses two cooling mechanisms:
  - **Radiation** primarily occurs if the **environment** is **cooler** than the body
  - **Evaporation** occurs if the **environment** is **warmer** than the body
- Body sweats → creates water on surface of skin → heat from body causes water molecules to evaporate → carries the water molecules into space
- ***Humidity*** *causes evaporation to be* ***less efficient****.*

Heat cramps

- **Muscle spasms** secondary to fluid and **electrolyte depletion**
- Who: young athletes working out in the sun
- How: hard work in hot conditions. Excessive sweating èreplace lost salt (electrolytes) with plain water (hypotonic fluid) → electrolyte abnormalities →muscle cramping
- Tx: rest, cool, fluid replacement with PO or IVF, **salt replacement** (Gatorade, normal saline, salty beer)

Heat syncope

- Passing out while standing in heat for extended periods of time
- Who: Elderly (co-morbidities/medications), soldiers, band members
- How: **standing** out in heat → pooling of blood in peripheral vasculature secondary to **vasodilation** → decrease preload → environment becomes hotter → **decrease preload** → cardiac output falls → **collapse**
- Tx: cooling, **fluids**: PO or IVF

Heat exhaustion

- Due to volume/electrolyte loss during heat stress
- Who: People working out in a hot environment
- How**:** Hydration helps regulate temperature. With dehydration body starts to **increase sodium pumping** especially in the sweat glands and kidneys → **increases basal metabolic rate** and makes core temperature increase
- Sx: Flu-like symptoms, headache, dizzy, weak, nauseas, but there are **NO CNS changes which is the major difference from heat stroke**, core temperature is generally **< 104 F**
- Tx: cool, rest, fluids, electrolytes as needed

**Fun Fact: With voluntary dehydration, when the thirst mechanism kicks in, the body is already dehydrated! The abnormal plasma osmolality triggers the hypothalamus to activate the thirst mechanism. So stay on top of your fluids!*

Heat stroke

- Extreme version of heat exhaustion with a complete failure of thermoregulatory mechanisms
- **Mortality is 30-80%**
- How: cannot get rid of heat fast enough. **Thermoregulatory mechanisms fail**
- Sx: **Neurological dysfunction**: AMS (combative/bizarre), seizures, ataxia; temperature generally **> 104 F**; can overlap with heat exhaustion
- **Complications**: **transaminitis** (may take 8 hours to develop), coagulopathy, **DIC** (due to thermal damage to endothelial cells, can be delayed 24 hours), rhabdomyolysis, ATN, pulmonary edema from high output failure due to severe dilation (fluid back up into lungs because heart has trouble moving fluid forward)
- Two types: classic and exertional
    - **Classic heat stroke**: nonexertional (e.g. heat wave in July in Chicago, old lady without AC vasodilates → heart rate increases and cannot keep up with vasodilation → dies)
        - Fewer complications (less rhabdo/ATN), likely due to shorter course of illness
        - **Higher mortality** than exertional heat stroke
        - Sx: not always dehydrated, often with dry skin, AMS
        - Tx: cool +/- fluids
    - **Exertional heat stroke**: exertional (e.g. young athlete, very sweaty); **profound dehydration** (4-5 liters down), **hypoglycemia** is common. More morbidity (liver failure, renal failure, DIC)
- **Treatment**: Rate of treatment correlates with mortality
    - **Rapid evaporative cooling** is the best in a cool ED. Spray warm water onto naked body and have fans evaporate directly off skin (cold water can be worse and cause inappropriate shivering). **Stop cooling around core temperature of 102 F**. Extremities may be very cold (e.g. 68 degrees), the temperature gradient will allow them to continue to move heat from core to extremities. **Avoid hypothermia.**
    - **Ice packs** to neck/groin/axilla
    - Liberal intubation (aspiration common)
    - **Cold water GI lavage** (intubated patients)
    - Treat blood pressure with **IVF**, but volume can be deceptive. BP often improves with cooling secondary to vasoconstriction. Average patient needs 1.5 L, some need 4-5 liters (exertional)
        - **Avoid pressors**: worsens ischemia and decreases heat exchange

- Treat **inappropriate thermogenesis**: shivering is a cutaneous reflex that slows down the cooling process
  - Tx: **low dose of benzodiazepines** or **thorazine**, monitor blood pressure closely

**Fun Fact: Antipyretics are NOT effective in heat stroke, this is NOT a hypothalamus problem! Drugs like Aspirin/Tylenol can make problems like DIC and liver failure worse!*

Thermal Burns

General principles

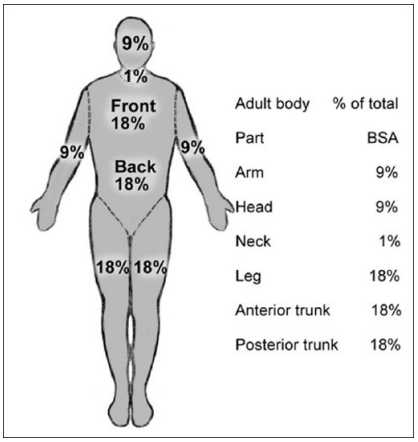
- Most burns in young adults, most deaths at extremes of age
- Early death: smoke inhalation
- Late death: sepsis
- Age + %body surface area (BSA) = mortality (Baux formula)
- Location of burn important
  - Specialized areas such as hands, feet, perineum, or circumferential burns → burn center
- **Early clinical assessment is difficult until wound declares itself in 1-2 days**

Depth of injury

- **Important determinant of outcome**
- First degree: epidermis only
  - Red, blanching, no blisters
  - e.g. sunburn
  - Tx: NSAIDs, H2 blockers
  - *not counted in total %BSA
- Second degree: dermis involved
  - Blistering, sensation intact
  - Tx: NSAIDs, topical antibiotics
- Third degree: full thickness
  - Anesthetic, leathery/waxy
  - Tx: requires grafting
- Fourth degree: deep tissue involvement
  - Involvement of muscle/tendon/bone

Rule of Nines

- Calculates total BSA in adults (doesn't work for kids)
- Only include 2nd-4th degree burns
- Adult palm (not fingers) = 1% BSA
- Child hand (including fingers) = 1% BSA



Adult body Part	% of total BSA
Arm	9%
Head	9%
Neck	1%
Leg	18%
Anterior trunk	18%
Posterior trunk	18%

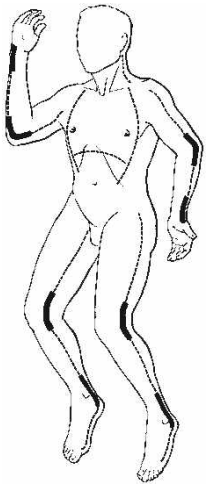
Inhalation injury

- Due to toxins that cause edema and loss of surfactant
- Clinical presentation is often delayed
- Not caused by direct thermal injury, which is usually above the cords
- Key hx: **Enclosed space fire**
- Key exam findings:
  - **Carbonaceous sputum: reliable sign of inhalation injury**
  - Cough, hoarseness, stridor, tachypnea, hypoxia, drooling, edema/blistering of upper airway
- ***Can't rule out inhalation injury with absence of findings initially due to delayed injury**
- If worried, intubate early
- **Consider additional injuries**
  - Toxic exposures to carbon monoxide, cyanide
  - Trauma
  - Rhabdomyolysis
    - Most common with electrical burns and 4th degree thermal burns (to muscle)
  - DIC

Thermal burn treatment

- Intubate PRN
- **IV fluids: Parkland Formula**
  - 4cc/kg/%BSA over 24 hours
  - 50% over first 8 hours
  - 50% over remaining 16 hours
  - **Time starts from burn, not arrival**
  - Use Lactated Ringers: Normal Saline will cause a hyperchloremic metabolic acidosis (loss of bicarb)
  - **Don't decrease amount of fluid even if lung injury present**
  - Urine output goal >1cc/kg/hr
- Check carboxyhemoglobin (08:42)
- Tetanus vaccination
- Avoid hypothermia (loss of skin barrier)
- +/- escharotomy
- No prophylactic antibiotics

Escharotomy



Done to prevent limb ischemia and restricted chest movement when eschar constricts in circumferential burns

Procedure:

- Use electrocautery if possible
- Cut into subcutaneous tissue until fat is exposed to release pressure
- Avoid neurovascular bundles near joints (bold areas in diagram)
- Start 1cm before eschar and continue 1cm beyond
- Stay posteriorly on neck

Tar burns

- Usually not full thickness; thermal injury will stop once tar is cooled
- Tx: Cool tar with cold water, then use an emulsifier to remove
    - Can do as an outpatient → put large volume of emulsifier under a dressing, then have pt return to rinse off in 24 hours
    - Don't peel tar off - it will pull skin off as well!

Burn unit referrals – when?

- 2nd degree burn >10%BSA; or any 3rd degree burns
- All inhalation injury
- Burns to specialized areas (face, hands, feet, genitalia, perineum, major joints
- Electrical and chemical burns
- Circumferential burns
- Patients with significant comorbidities

Electrical Injuries

General principles

- Epidemiology: Toddlers, teens, and industrial workers (trimodal injury peak)
- Household circuits = 110V
- High voltage = 1000V
- Significantly increased mortality >600 V
- High voltage is more dangerous, but more injuries are 2/2 low voltage (more accessible)
- Low voltage accounts for 50% of deaths

- Voltage = Current x Resistance (V= I x R) (01:02)
    - Thus, electricity travels on the path of lease resistance (nerves, blood vessels, wet skin)

    - There is a lot of damage **inside** tissue
    - Most damage is due to the heat produced by resistance
- **Clinical effects depend on current:** amount, duration, type (alternating current vs direct current), path (across chest/head)

Alternating Current (AC) vs Direct Current (DC)

- DC: tends to throw patient 2/2 muscular contraction (think trauma)
    - Acts like a defibrillator
    - Asystole more common, intact pacemaker may then restart heart
- AC: causes tetany → pt cannot let go of object
    - More likely to cause fibrillation → death
    - ***AC 3x more lethal than DC**

Clinical effects of electrical injury

- Cardiac arrest – usually an arrhythmia
    - Main cause of death
    - Both delayed arrest and acute MI uncommon
- Asystole
    - DC current
    - High voltage AC or DC
- Ventricular fibrillation
    - Low voltage AC
- Cardiac arrhythmias (PVC/Afib/SVT/Blocks)
    - High voltages
- Burns: most injury is deep
- Vascular spasm vs thrombosis (coagulation necrosis): can see pulseless extremities
- Fractures/dislocations: **think posterior shoulder dislocation (light bulb sign)**
- Neuro findings: usually high voltage
    - Altered mental state, seizure
    - Intracranial hemorrhage
- Peripheral neuropathies (delayed)

Management

- Low voltage injuries
    - If asymptomatic, can go home without any testing
    - If mild symptoms (small burn, etc.) → EKG + urine dip (checking for rhabdo)
    - If normal → home
- High voltage injuries
    - Monitor 12-24 hours regardless of symptoms

    - Urine dip/CK
    - Basic labs
    - Head CT if altered
    - Tetanus vaccination for burns
- Pediatric commissure burns of the mouth
    - Home if: no LOC, normal EKG, no other injury, tolerating orals, reliable parents
    - No urine testing necessary
    - **Instruct parents regarding delayed bleeding of labial artery (day 5)** → hold pressure
    - F/u with plastics/oral maxillofacial
    - Wound check

Lightning injury

- Mechanism: Huge "DC" voltage
    - Survivable because very brief + flashover phenomenon (cascades over body)
- Main effects
    - Cardiovascular: asystole
        - SA node will often restart on its own
    - Respiratory:
        - Brainstem shock → apnea
        - Will last longer than asystole and if not corrected → arrest

**** Exception to the mass casualty rule – if apneic/pulseless → still viable with rescue breathing**

- Keraunoparalysis (60% occurrence)
    - Current goes up one leg and down the other → vasospasm/neuroparalysis → cold, insensate, pulseless leg
    - Usually spontaneously resolves in ~6h
- Trauma common
    - 2/2 being thrown
    - TM rupture classic (caused by superheating of air → vacuum)
- Lichtenberg figure: pathognomonic for lightning injury, resolves in 6h
- Serious burns uncommon, but can get steam burns under clothing if skin is wet/sweaty
- Transient amnesia common

Submersion Injury

General

- Bimodal age distribution-:Toddlers (pools/tubs); Teenagers (lakes/rivers)
- #3 cause of accidental death

Submersion Pathology

- Breath holding: involuntary gasp, aspiration/laryngospasm/LOC, active aspiration of fluid leading to death
- Wet drowning is 90% and Dry drowning 10%
- Aspiration of water causes: loss of surfactant, atelectasis, V/Q mismatch, alveolar membrane injury (ARDS)
- #1 Metabolic Derangement is **metabolic acidosis** secondary to hypoxia
- Airway obstruction with particulate matter
- Delayed pneumonia secondary to aspiration
- **Mammalian diving reflex** (children>adults): sudden cold water (<70F; 21C) leading to bradycardia, blood shunting to CNS, slowed metabolism

Clinical Presentation

- Respiratory distress: cough/dyspnea, progresses over hours
- Altered mental status: correlates to prognosis
- Shock uncommon: rule out trauma
- Cardiac Arrest: bad prognosis in warm water (cold core in “warm” water is not good)
- Consider C-spine trauma: most guidelines do not recommend for every case
- Hypothermia common

Management

- BiPaP: very effective bridge, patient must be alert/cooperative
- Airway: intubate for usual reasons, remember to add PEEP
- ECMO: for poor oxygenation on ventilator
- Albuterol: can help with reactive bronchospasm
- No prophylactic antibiotics and warm up as needed

Disposition

- Asymptomatic patient (with minor event) and normal pulse ox: Home after brief observation (obs) about 2-3 hours
- Asymptomatic/Mildly symptomatic patients with normal pulse ox and CXR: Home after 4 hours obs
- Mild-Moderate symptoms and hypoxia: Admit for obs
- Severely symptomatic patients go to ICU

Radiation Exposure

Definitions

- The amount of energy deposited in tissues is measured in rads
- International unit is gray (Gy); 1 gy = 100 rads
- Alpha and Beta particles have poor tissue penetration, danger with ingestion or skin
- Gamma rays have deep tissue penetration and lead to whole body exposure

Factors in radiation injury

- Time, Distance (inverse square law, get farther away!), shielding, radioactive half-life
- Nature of cells (**rapidly diving cells most radiosensitive**: GI lining, bone marrow)

Radiation Exposure Types

Localized

- Epilation (hair burned off)
- Skin burns
- These develop several days **after** exposure

Whole Body Exposure Syndromes

- **Hematopoietic Syndrome**
  - >2 Gy dose; onset at <2 days
  - Sx: pancytopenia, infection
- **GI syndrome**
  - >6 Gy dose; onset over hours
  - Sx: nausea/vomiting, dehydration, GI bleed
- **CV/CNS Syndrome**
  - >10 Gy dose; onset over minutes
  - Sx: circulatory collapse and CNS damage

Radiation Exposure Internal (Inhaled/Ingested)

- Example: Radioactive Iodine
- Source: power plants, nuclear weapon
- Tx: Potassium iodide taken before or shortly after can prevent thyroid cancer/hypothyroidism

Radiation Exposure External Contamination

- Removal of clothing eliminates 90% of contamination
- Low risk to health care works using universal precautions
- Wash with soap/water
- **Don't abrade skin** as it is unnecessary and increases exposure to patient.

HIGH YIELD

- **Lymphopenia FIRST** to develop
- Absolute lymphocyte count (ALC) >1000 at 48 hours (good prognosis)
- ALC <300 at 48 hours (LETHAL)

Bites and Stings

Mammalian bites

- Human bites: the most important bug is *Eikenella corrodens*
- Dogs and cats: remember *Pasteurella multocida*
- Dogs crush, cats puncture, but not true that cat bites are more prone to infection
    - Pts just don't go to the hospital unless infected; deceptive depth of cat bites is important around joints/tendons
- Treatment (Tx) for all of the above: PO amoxicillin/clavulanate or IV ampicillin/sulbactam
    - Penicillin allergy: levofloxacin and metronidazole

Arthropod bites and stings

- Most morbidity due to Hymenoptera: bees, wasps, hornets, and ants
- Risk of so-called "killer bees" is simply that they attack aggressively and in groups
- Venom contains histamine (redness, edema) and other proteins (cause anaphylaxis); **the latter can commonly cross-react with other species' venom**

4 classes of sting reactions

Local

- E.g. Standard bee sting
- Swelling, redness, pain

Toxic reaction

- Many stings, usually >10, so usually fire ants or killer bees
- Can clinically resemble anaphylaxis (and if unsure, should be treated as such)
- Syncope, headache, nausea/vomiting

- **No generalized hives/edema and no bronchospasm**

Anaphylaxis

- Can be from single sting
- Onset within minutes
- Bronchospasm, hypotension, rash (hives)

Delayed reaction

- Immunologic response, like serum sickness
- Arthralgias, fevers, malaise 1-2 wks after sting

Treatment

- **The same for all hymenoptera**
- Remove stinger if present: using tweezers is fine as all the venom is already in.
- Local wound care: wash, ice
- Antihistamines, NSAIDs
- **Infection is very uncommon, though the sting may initially appear infected;** if several days out and worsening, though, may be superinfected
- Anaphylaxis Tx: **epinephrine 0.3-0.5mg 1:1000 IM** is the mainstay
- Systemic symptoms (toxic reaction or anaphylaxis) require epinephrine auto-injector Rx and allergist referral
- Fire ants crawl all over pt then all bite at once, causing widespread **papules and pustules that are sterile and do not require antibiotics**

Brown recluse spider (*Loxosceles*)

- Violin pattern on the back, live in warm, dry places (e.g., woodpile), shy
- Located only in Midwest
- **Painless** bite with erythema on day 1, classic (though rare) necrotic ulcer on day 2 caused by **cytotoxic** toxin
- Systemic symptoms rare
- Supportive care, tetanus (Td) vaccine if ulcer; delayed debridement
- **No dapsone**.

Black widow spider (*Lactrodectus*)

- Red hourglass pattern on back, live in warm dry places, aggressive
- Located throughout USA
- **Neurotoxic** venom causes acetylcholine (ACh) and norepinephrine release
- **Immediately painful** bite, no necrosis, local or generalized muscle cramping (very painful, can even mimic peritonitis), localized sweating (ACh release)
- Tx: analgesics, benzodiazepines

- For severe pain, antivenin relieves pain in about 20 min, but risk is anaphylaxis (horse serum)
- No calcium gluconate

Snake bites

- 2 types in North America: pit vipers (Crotalids) and coral snakes (Elapidae)

Crotalids

- Rattlesnakes, copperheads, cottonmouths
- Wide distribution in United States
- **3 toxin classes**
  - Cytotoxic: swelling, edema, cell lysis
  - Hemorrhagic: coagulopathy, disseminated intravascular coagulation (DIC; predigests prey)
  - Neuromuscular (less common)
- 25% bites "dry" (no venom)
- Symptoms
  - Locally, venom causes redness, swelling, pain within 1 hr
  - Ecchymosis/bullae, rarely compartment syndrome
  - Systemic: n/v, metallic taste, fasciculations. Care is supportive, monitor for DIC (fibrinogen, platelets, INR)
- First aid: get away from snake! Immobilize extremity and minimize movement; **NO tourniquet** (this is for neurotoxic venom)
- Emergency department Tx: wound care, Td, supportive care, labs q2 hr (CBC, coags, fibrinogen), **antivenin**
  - Antivenin = CroFab; covers **only N. American vipers**, unlike old equine-derived antivenom, but less anaphylaxis
  - Same dose for adults and children (amount of venom, not pt's wt)
    - Slow infusion x10 min, if no allergic reaction, then give the rest over 1 hr
    - Titrate to arrest of symptoms, marking skin and getting labs with each dose (4-6 vials)
    - May rebound
- Crotalid compartment syndrome is rare, only with bite into deep compartment causing capillary leak
  - Classic findings (5 P's), but **not a surgical disease**
  - Proper Tx is antivenin; surgery only if persists despite aggressive antivenin

Coral snakes (Elapidae)

- 3 US species
- **"Red on yellow, kill a fellow; red on black, venom lack"**
  - Not true outside United States
- Passive: must hang on to inject venom; **bite marks are uncommon**
- Venom is **neurotoxic**, so few local effects
- Delayed neuro symptoms (up to 12 hrs), so require admission
- Most commonly cranial nerve abnormalities, n/v, but can develop respiratory depression and paralysis lasting days
- Antivenin no longer available, so intubate PRN and give them time

Marine envenomation

- Most important are Cnidaria: jellyfish, fire coral
- Cells called **nematocysts** fire tiny harpoon
  - Poison causes local pain, erythema, pruritus
  - Rarely life-threatening
- Treatment: deactivate (5% acetic acid, isopropyl alcohol)
  - If none available: **saltwater rinse, not fresh** (isotonic, makes nematocysts fire)
- Box jellyfish does have an antivenin

Marine vertebrates

- Most injuries due to stingrays, usually stepped on by pt walking in the surf
- Spines can break off
  - Cartilaginous (like sharks), so **may not show up on X-ray**
- Symptoms mostly local (pain, edema)
- Tx: wound care, **antibiotics** to cover *Staphylococcus*, *Streptococcus*, *Vibrio* (e.g. cephalexin/doxycycline)
- Of note: **toxin is heat labile**, denatures at >113˚F (45˚C), so **hot water immersion** relieves pain
- Leave wound open due to infection risk

- Sea snakes live mostly in warm water, related to Elapidae (i.e. neurotoxic, very passive, need to "chew" to cause significant envenomation); pts usually fishermen
  - Again, few local effects, delayed neurotoxicity: focal deficits, respiratory depression
  - Polyvalent antivenin does exist

Marine microorganisms

- *Erysipelothrix*: Gram-positive rod that lives in salt water and is often on fish
    - Seen in fishmongers, fishermen (enters through scrape/cut)
    - "Erysipeloid" cellulitis: beefy, red, sharp demarcation
    - Tx: fluoroquinolone or beta-lactam
- *Mycobacterium marinum*: "fish tank granuloma"
    - Acid-fast bacillus, salt water
    - Classically hard, red skin nodules at the site several weeks after a cut/scrape while cleaning salt-water fish tank
    - Tx similar to tuberculosis
- *Vibrio vulnificus*: Gram-negative rod, salt water
    - **Two presentations**:
        - Necrotizing fasciitis (scrape while in the water followed in about 8 hrs by bulla, cellulitis, nec fasc)
        - Primary septicemia (classically in known cirrhotic who eats raw shellfish, develops sepsis in 12 hrs)
        - 75% have noncontiguous bullae
    - Tx: 3rd-generation cephalosporin plus doxycycline