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Environmental Emergencies

Primary Care Paramedicine

Module:19
Section:01

Lecture Outline

- Introduction
- Pathophysiology of heat and cold disorders
- Heat disorders
- Cold disorders
- Near-drowning and drowning
- Diving emergencies
- Altitude Illness

Introduction

- Environment
  - All of the surrounding external factors that affect the development and functioning of a living organism
- Environmental factors create stress on the body
  - Becomes an emergency when a medical condition is created or exacerbated

Risk Factors

- Age
  - Very young and old
- Poor general health
- Fatigue
- Predisposing medical conditions
- Medications
  - Prescription and over the counter

Mechanisms of Heat Gain and Loss

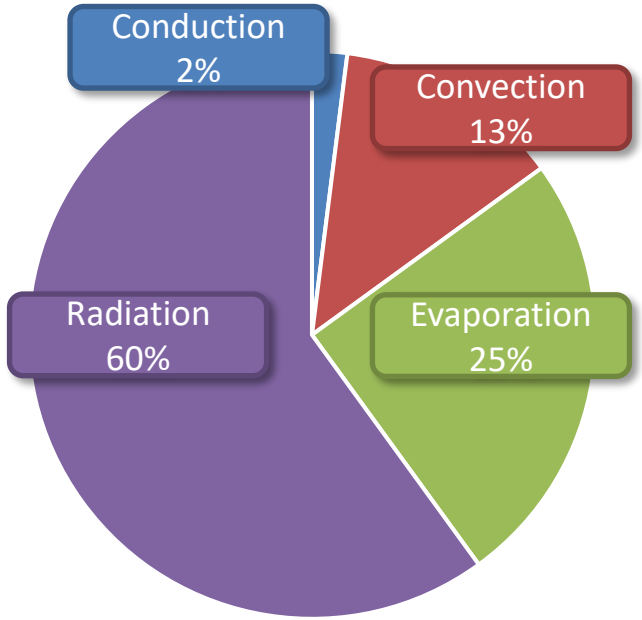
- Body gains and loses heat two ways
  - From within the body
  - Contact with the external environment
- Thermal gradient
  - Difference in temperature
  - Affects rate of heat loss/gain
- Also affect by wind and relative humidity

Thermogenesis

- Production of heat, especially from internal sources
  - Work-Induced
    - Exercise and shivering generate heat in muscles
  - Thermoregulatory
    - Metabolism, controlled by endocrine system
  - Diet-Induced
    - Processing of food and nutrients

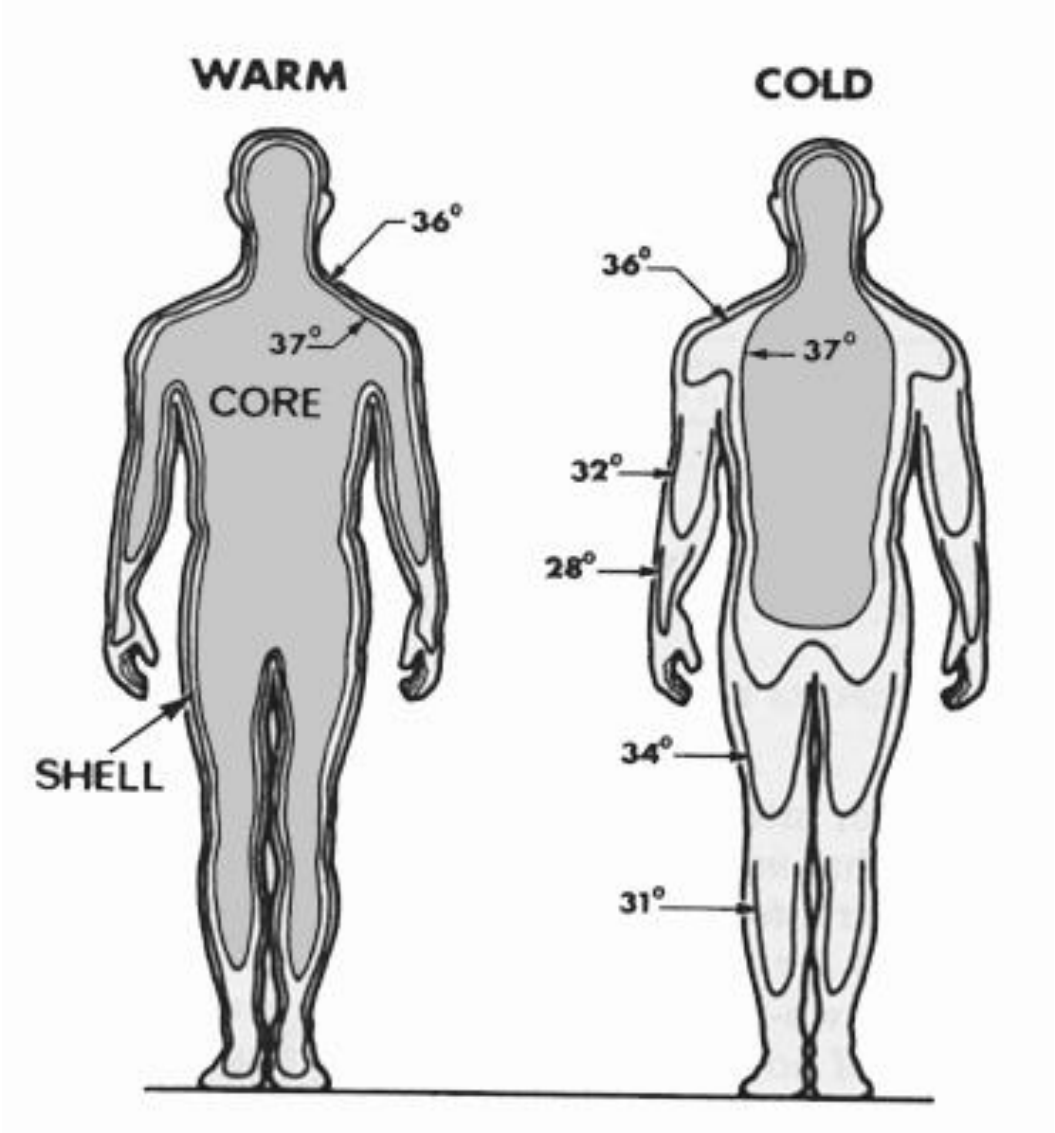
Normal Heat Loss

- At rest, humans produce 40-60 kilocalories (kcal) of heat per square meter of body surface area through generation by cellular metabolism.
- Heat production increases with movement
- Shivering increases the rate of heat production 2-5 times.
- Under dry conditions
  - Radiation 60%
  - Conduction and convection 15%
  - Respiration and evaporation 25%



Regulating Heat Production

- Heat is generated through:
  - Mechanical
    - Shivering
  - Chemically
    - Cellular metabolism
  - Endocrine
    - Hormone release



Heat Loss (Heat Transfer)

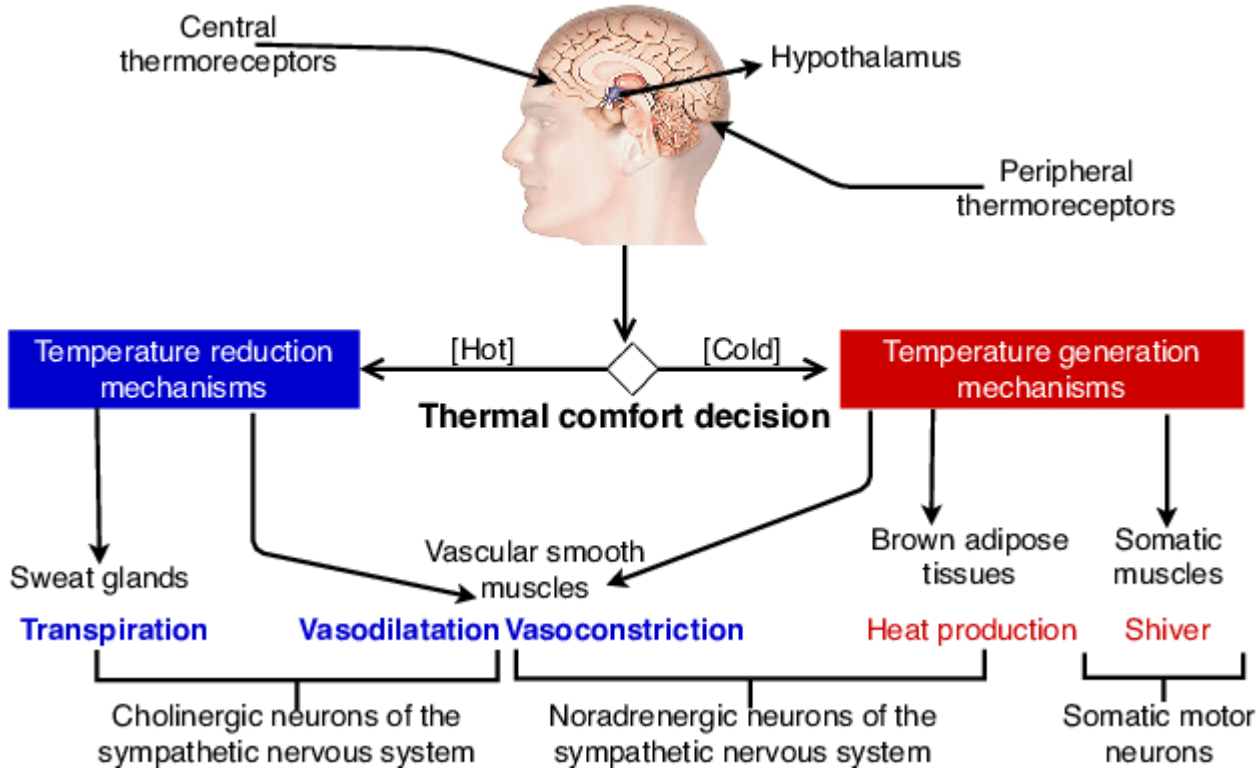
- Convection
- Conduction
- Evaporation
- Radiation
- Respiration

Regulating Heat Loss

- Heat is lost from the body to the external environment through the skin, lungs, and excretions
  - The skin is most important in regulating heat loss
  - Radiation, conduction, convection, and evaporation are the major sources of heat loss

Thermoregulation

- Hypothalamus
  - Responsible for thermoregulation
  - Acts as a thermostat
  - Negative feedback system



Thermoregulation

Heat Dissipation

- Sweating
- Vasodilation

Heat Conservation

- Shivering
- Vasoconstriction


TEMPERATURE REGULATION
BY THE HYPOTHALAMUS
44°
42°
40°
38°
37°
36°
34°
32°
30°
28°
26°
24°
°C
TEMPERATURE REGULATION
SERIOUSLY IMPAIRED
TEMPERATURE REGULATION
EFFICIENT
TEMPERATURE REGULATION
IMPAIRED
TEMPERATURE REGULATION
LOST
PMT

Thermoreceptors

Peripheral

- Skin and certain mucous membranes
- Cold receptors outnumber warm

Central

- Deep tissues
- Spinal cord, abdominal viscera, great veins
- Exposed to body core temperature
- Also respond mainly to cold

Basal Metabolic Rate

- Rate at which body consumes energy just to maintain stability
- Exertion
  - Increases metabolic rate
- Core temperature
  - Reflects internal energy production and consumption

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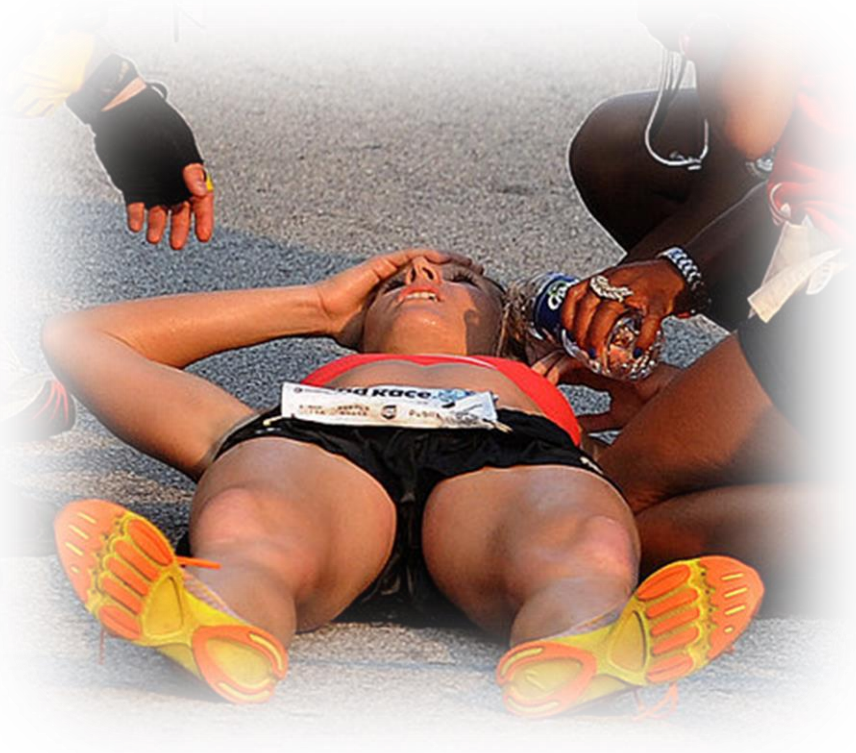
Environmental Emergencies

HEAT RELATED ILLNESSES

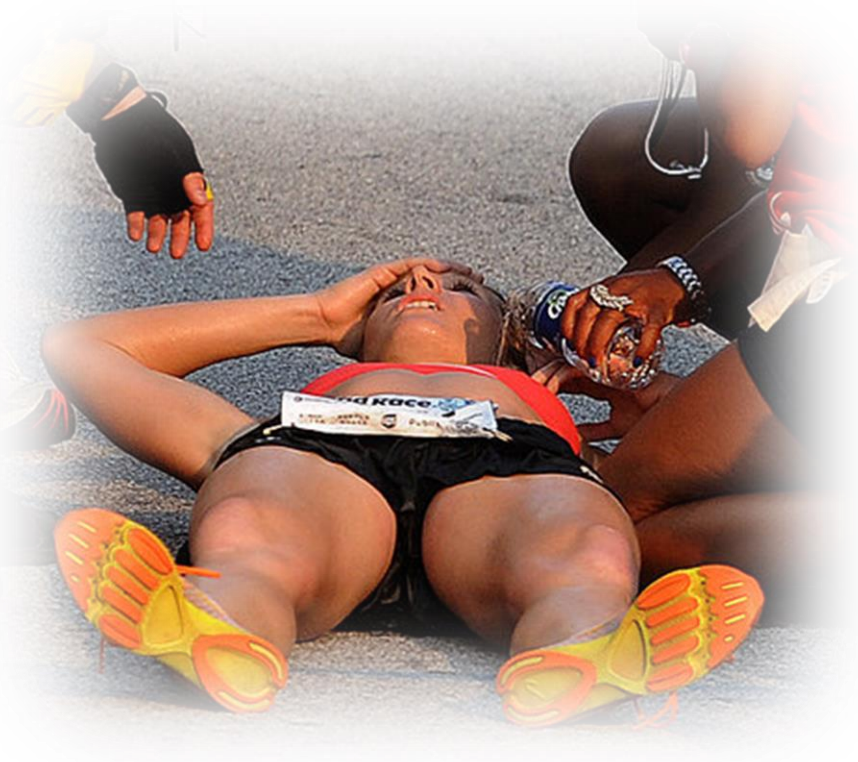
Case 01

- You respond for a 36 y/o F who is running in a full marathon on a hot humid summer day. 19 km into the run, the patient collapses with a brief loss of consciousness.
- Upon arrival the pt is conscious but confused and is complaining of dizziness and headache.
- No other priority symptoms.



Case 01

- No PMHx, takes supplemental vitamins. Physical exam unremarkable except for hot dry skin.
- Vitals are:
  - HR 74
  - RR 20
  - BP 106/66
  - SaO_2 94% (RA)
  - BGL 6.4



Heat Loss

- Dominant forms of heat loss in a hot environment are radiation and evaporation
  - When air temperature exceeds 95°F (35°C) radiation of heat from the body ceases and evaporation becomes the only means of heat loss
- Evaporation is maximally efficient in a dry environment
  - If humidity reaches 100%, evaporation of sweat is no longer possible and the body loses its ability to dissipate heat

Heat Loss Continuum

- Initially, the body attempts to control the core temperature
  - Activates heat receptors in both the hypothalamus and the periphery
  - ↑ Shunting of blood to the periphery
  - ↑ Minute ventilation
  - Sweating maximize evaporative heat loss
- If these compensatory mechanisms fail
  - Central vasoconstriction and peripheral vasodilatation decrease
  - Resulting in less heat carried away from the core

Maintenance of Thermoregulation

- Hyperthermic compensation
  - Increased heat loss
    - Vasodilatation of skin vessels
    - Sweating
  - Decreased heat production
    - Decreased muscle tone and voluntary activity
    - Decreased hormone secretion
    - Decreased appetite

Heat Damage

- Cellular damage may occur anywhere from 45 minutes to 8 hours after exposure to core temperatures of 107.6°F (42°C)
- Nearly all cells respond to heat stressors by producing heat-shock proteins (HSP) to prolong cell survival at otherwise lethal temperatures
  - believed to act as molecular chaperones to prevent damage
- ↑ HSP is protective against not only heat but ischemia, hypoxia, endotoxin, and inflammatory cytotoxins
- Conditions associated with low levels of these protective proteins such as advanced age, lack of acclimatization, and genetic polymorphisms may place these patients at increased risk for thermal injury

Inflammatory Response

- Acute-phase response
- Inflammatory mediators released
  - Cytokines and interleukins
- Thought to be similar response to sepsis
- Organ hypoperfusion
  - May increase production of reactive oxygen and nitrogen species
  - These byproducts with inflammatory mediators may act to increase intestinal permeability resulting in endotoxemia
- These factors combine to further impair thermoregulation and the body's ability to prevent damage culminating in hypotension, hyperthermia and heatstroke

Mortality and Morbidity

- Risk of death is related directly to peak temperature, duration of exposure, and acclimatization period
- Estimates of fatalities caused by heat-related illness in the United States range from hundreds to several thousand per year
- The mortality rate in patients with heatstroke has been reported to be 10-70%
  - The highest number of deaths occurring when treatment is delayed for more than 2 hours
- Heat waves increase the mortality rate
  - The European summer heat wave of 2003 was exceptionally harsh in both duration and intensity
  - In France alone, the number of heat-related deaths reached 14,800 by August 20

Mortality and Morbidity

- Sex: Male-to-female ratio is 1:1
- Age:
  - Elderly persons
    - Underlying illness
    - Medication use
    - Declining adaptive thermoregulatory mechanisms
    - Poor access to air-conditioning
    - Cognitive obstacles to self-care
    - Limited social support networks
  - Neonates
    - Poorly developed thermoregulatory mechanisms
    - Inability to make behavioral adjustments

Heat Illness

- Major cause of preventable morbidity worldwide
  - Especially in regions characterized by high ambient temperatures
- Involve varying degrees of thermoregulatory failure that occur when individuals are exposed to elevated temperatures
- All heat illnesses exist along a continuum and share similar elements
- In all cases, the root cause is rate of heat gain exceeding the ability of the body to dissipate heat
  - If the responsible factors are not corrected, the individual will invariably develop heatstroke
  - Progression to heatstroke and end-organ dysfunction is related to the physiologic responses to hyperthermia, the direct toxicity of the heat, and the inflammatory response

Risk Factors

- More common in the summer
  - Can also occur in moderate conditions, depending on environmental factors
- Environmental factors
  - temperature, humidity, sun exposure, wind and clothing
- Body's thermoregulatory system is responsible for allowing the body to heat and cool itself as necessary
  - System can be overwhelmed during periods of heat stress if the sum of the environmental heat load and the metabolic heat load exceeds the body's capacity for heat dissipation

Risk Factors

- Dehydration of more than 3 percent of body weight
  - If lost fluids are not restored, the risk of heat-related illness is higher
  - Losses can be exacerbated by utilization of replacement fluids that are diuretic (i.e. beverages containing caffeine or alcohol)
- Thirst cannot be relied on as a measure of fluid loss
  - Athletes may not become thirsty until they are 5 % dehydrated
- Medical conditions
- Physiologic conditions
- Medications and abused substances

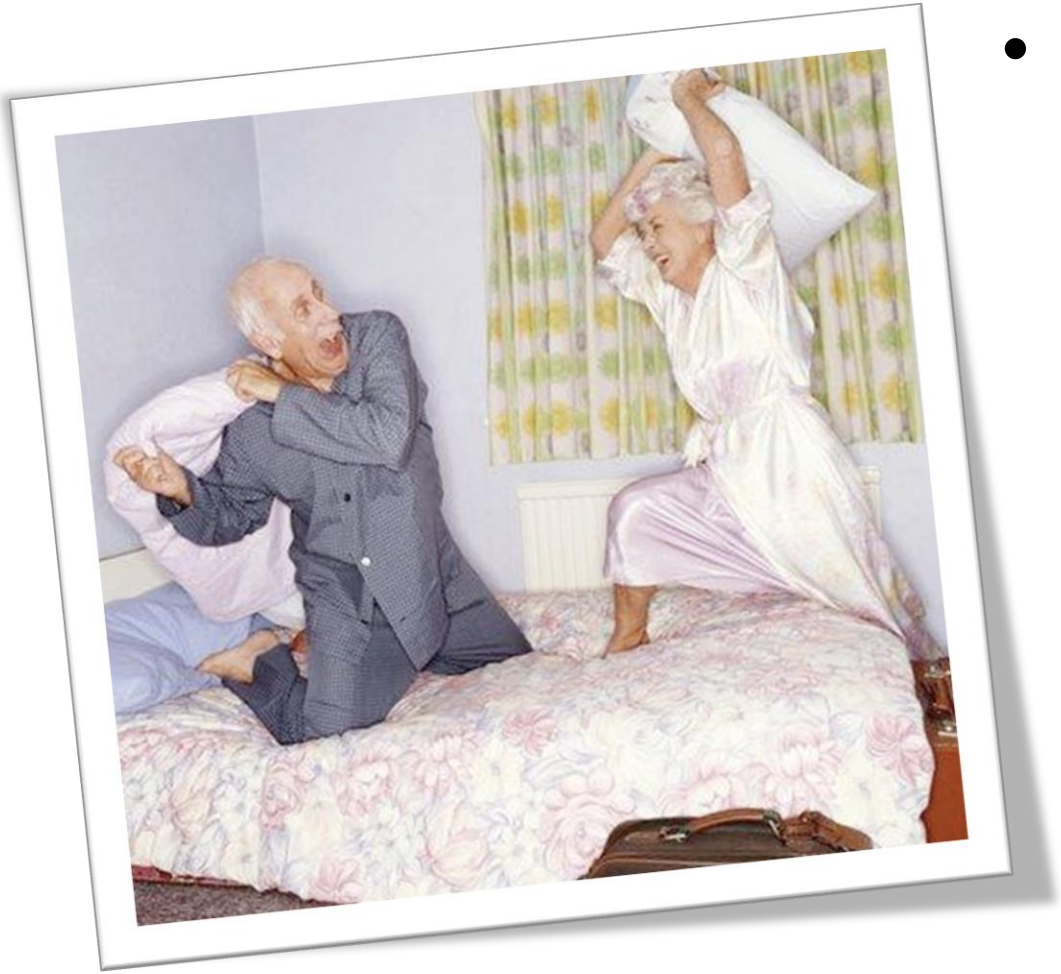
Contributing Factors

- Factors may include:
  - Physical Condition
  - Age
  - Body mass
  - Other additional factors

Physical Conditions

- Fever
- Dehydration
- Medications
- Prolonged exertion
- Chronic illnesses
- Cardiac conditions
- Cystic fibrosis
- Uncontrolled diabetes
- Uncontrolled hypertension
- Eating disorders
- Malignant hyperthermia
- Peripheral vascular disease
- Extensive skin disease or damage, or both
- Autonomic nervous system disorders
- Psychiatric conditions
- Hyperthyroidism

Age



- Elderly
  - Decreased vasodilatory response
  - Decreased maximum heart rate
  - Resulting in decreased maximum cardiac output
  - Decreased thirst response
  - Decreased fitness level
  - Decreased mobility resulting in increased difficulty of easily obtaining fluids

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Age

- Younger age
  - Decreased ability to sweat
  - Decreased cardiac output at a given metabolic rate
  - Greater core temperature required to initiate sweating
  - Slower acclimatization
  - More heat produced for the same level of activity

Increased Body Mass

- More heat generated for same level of activity
- Less efficient heat dissipation
- Fewer heat-activated sweat glands in skin overlying adipose tissue
- Decreased cardiac output per unit of body weight

Additional Factors

- Lack of access to air conditioning
- Residing in upper floors in tall buildings
- Sleep deprivation (decreases skin blood flow and rate of sweating)
- Previous heat stroke
- Use of equipment or heavy clothing (football player's pads, firefighter's protective gear etc.)
- Recent move from a temperate to a hot climate
- Urban setting

Hyperthermia

- Unusually high body core temperature
- Signs of thermolysis
  - Diaphoresis
  - Increased skin temperature
  - Flushing
- Signs of thermolytic inadequacy
  - Altered mentation
  - Altered level of consciousness

Predisposing Factors

- Age of the patient
- Health of the patient
- Medications
  - Diuretics, beta-blockers, psychotropics and antihistamines
- Level of acclimatization
- Length and intensity of exposure
- Environmental factors

Preventive Measures

- Maintain adequate fluid intake
- Allow for gradual acclimatization
- Limit exposure to hot environments

Heat Edema

- Mildest form
  - Occurs when swelling develops in dependent areas of unacclimatized persons during hot summer months
  - Caused by transient peripheral vasodilatation from the heat and orthostatic pooling during prolonged sitting or standing
  - Heat edema may improve with periodic exercise or elevation of the legs

Heat Cramps

- Painful spasms of skeletal muscles of the arms, legs or abdomen
- May be a warning sign of impending heat exhaustion
  - Believed to be caused primarily by a rapid loss of salt during profuse sweating
  - Cramps may worsen
    - If salts are not replenished
    - When Ca levels are low
    - Too much water is consumed by pt
      - Na / H2O ratio disruption

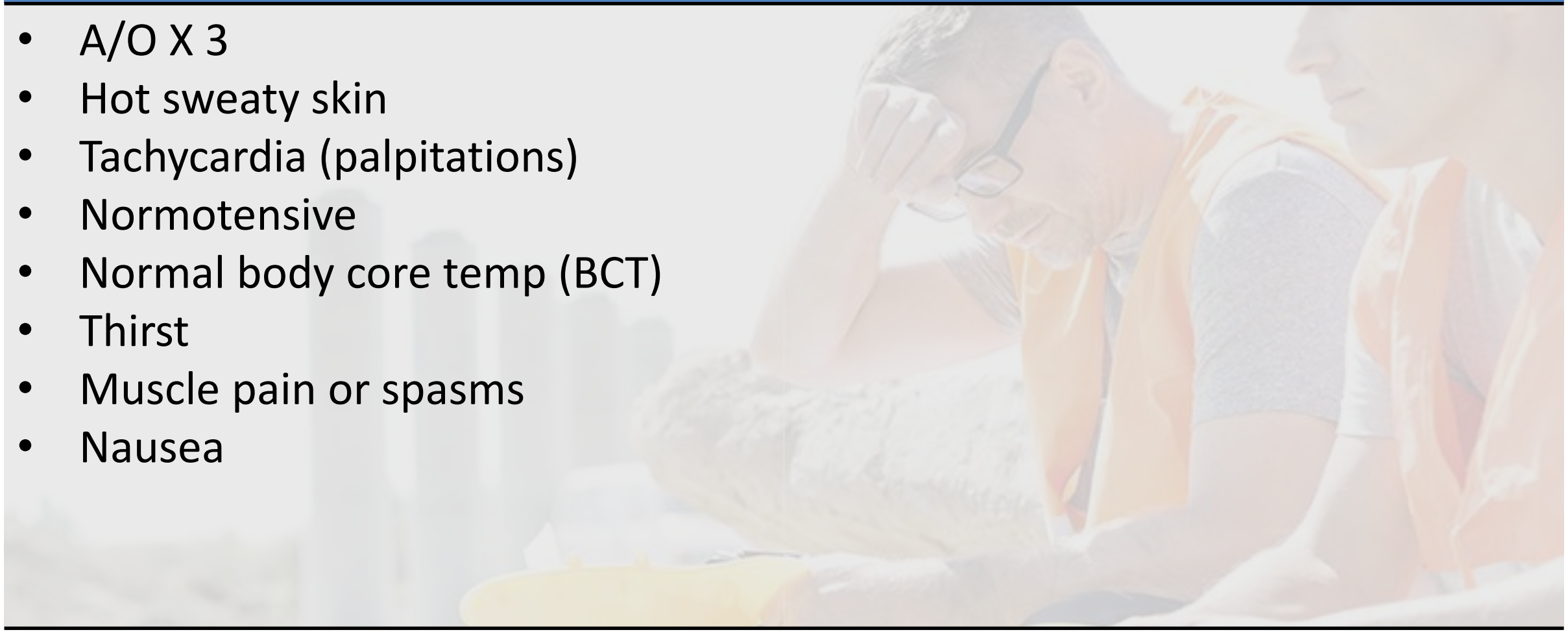
Heat Cramps

- Muscle cramps caused by overexertion and dehydration
  - Strenuous activity in a hot environment
- Thought to be related to local sodium losses related to sweating
- Painful but not considered to be an actual illness

Heat Cramps

Signs and Symptoms

- A/O X 3
- Hot sweaty skin
- Tachycardia (palpitations)
- Normotensive
- Normal body core temp (BCT)
- Thirst
- Muscle pain or spasms
- Nausea



Heat Cramps

Management

- Remove patient from environment
- Administer oral saline solution
- Opinions vary on whether to massage painful muscles

Heat Exhaustion

- A more severe form of heat illness
  - Mild-to-moderate core temperature rise above 38.0°C (100.4°F) but lower than 40.5°C (104.9°F)
- A relative state of shock
- Most commonly associated with:
  - Profuse sweating
  - Water and salt deficiencies cause electrolyte imbalance
  - Vasomotor response causes inadequate peripheral and cerebral perfusion from pooling

Heat Exhaustion

Signs and Symptoms

- LOC (irritable, poor judgment, dizziness, headache)
- Pale, cool, clammy skin
- Tachycardia
- Tachypnea
- Cramps, muscle weakness
- Nausea/vomiting
- Blurred vision or dilated pupils
- Profuse sweating
- Malaise
- Anorexia
- Hypotension

Heat Exhaustion

Management

- Remove form hot environment
- Place patient supine
- Saline solution
- Oral or intravenous
- Remove some of clothing
- Fan the patient
- Supportive care and treat for shock

Heat Syncope

- Orthostatic syncopal episode or dizziness
- Predisposing factors
  - Exercise without a cool-down period, dehydration and lack of acclimatization
  - Can result from inadequate cardiac output and postural hypotension
- Recovery is immediate once the patient falls to the ground
- Treatment consists of placing the patient in a supine position and replacing any water deficit

Heat Stroke

- Core body temperature of at least 40.5°C (104.9°F) and acute mental status changes
- This produces multi-system tissue damage and physiological collapse

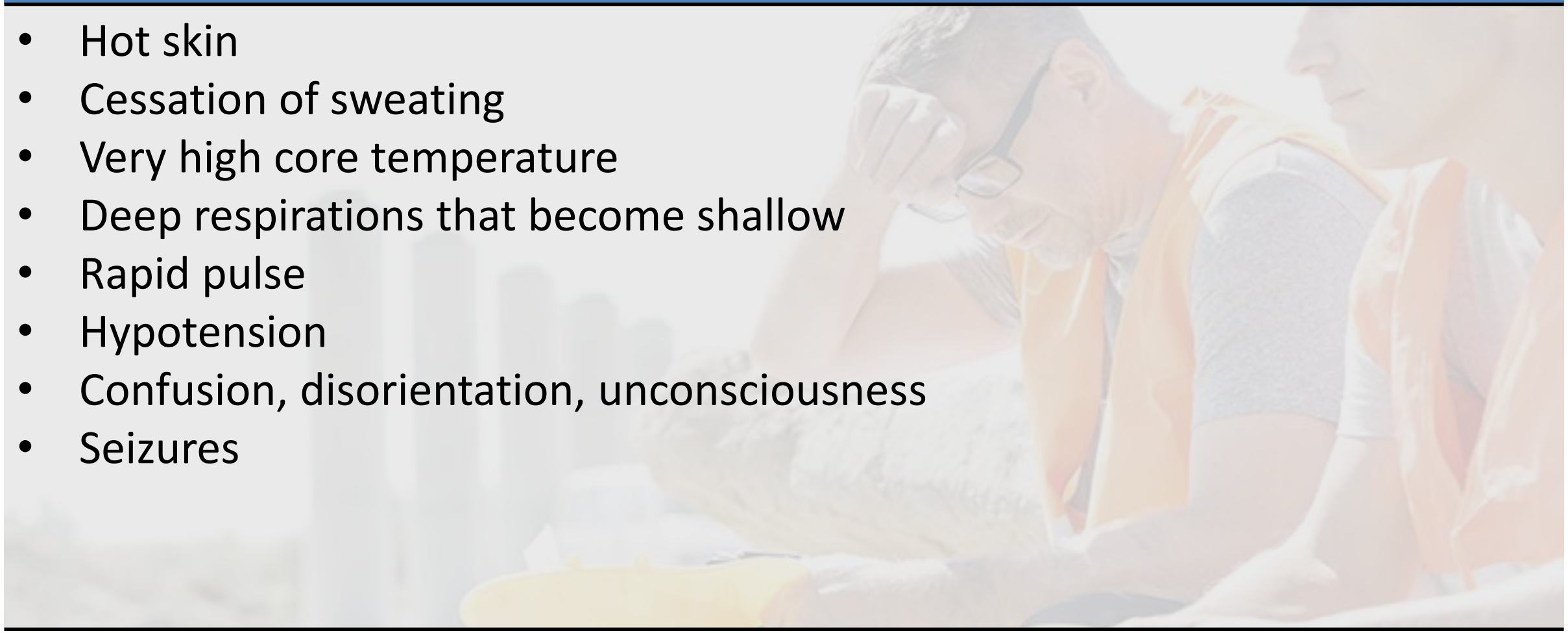
Heat Stroke

- Traditionally is divided into categories based on underlying etiology:
  - Classic heatstroke
  - Exertional

Heat Stroke

Signs and Symptoms

- Hot skin
- Cessation of sweating
- Very high core temperature
- Deep respirations that become shallow
- Rapid pulse
- Hypotension
- Confusion, disorientation, unconsciousness
- Seizures



Presentation

Classic heat stroke

- Patients with chronic illness
- Deficient temperature regulation
- Hot, dry skin

Exertional heat stroke

- Good general health
- Overwhelming heat stress
- High temperature and excessive exertion
- Sweating ceased but skin hot and moist from prior sweating

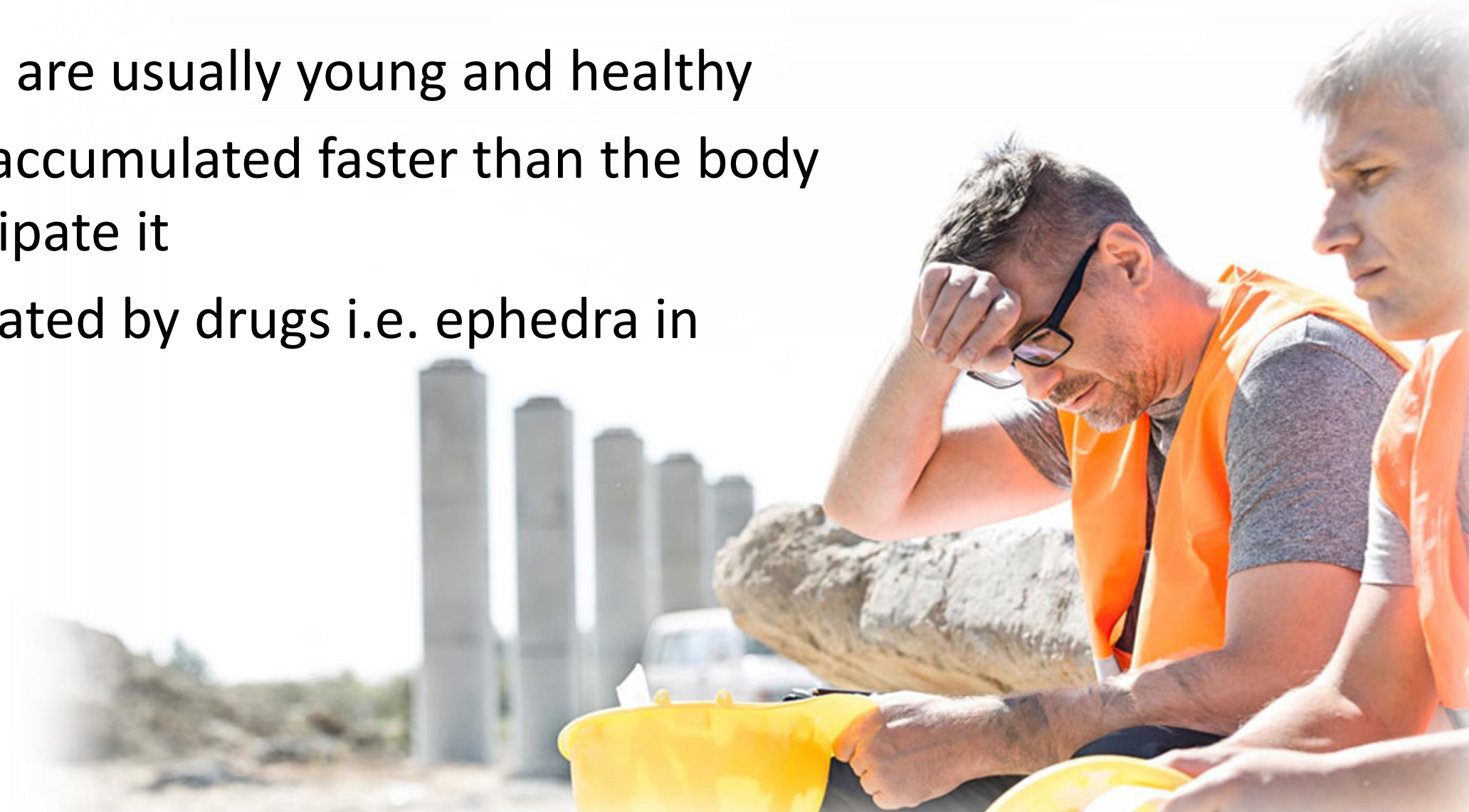
Classic Heat Stroke

- Occurs during periods of sustained high ambient temperatures and humidity
- Pts are unable to dissipate heat adequately

- Examples:
  - Children left in enclosed vehicle on hot afternoon
  - Elderly person confined to a hot room
- Predisposing factors:
  - Age
  - Chronic disease (DM, IHD, alcoholism and schizophrenia)
  - Medications

Exertional Heat Stroke

- Patients are usually young and healthy
- Heat is accumulated faster than the body can dissipate it
- Exacerbated by drugs i.e. ephedra in athletes



Heat Stroke

- In both cases
  - Thermoregulatory mechanisms fail
  - Results in accelerated hyperthermia
  - Increased expression of heat shock proteins
  - An exaggerated acute-phase response
  - End-organ dysfunction

Comparison

Characteristic	Classic Heat Stroke	Exertional Heat Stroke
General health	Predisposing health factors	Healthy person
Age	Older	Younger
Occurrence	Often occurs during high temperatures	Occurs sporadically
Sweating	Absent	Present
Activity	Sedentary	Strenuous
DIC	Uncommon	Common
Acute renal failure	Uncommon	Common
Lactic acidosis	Uncommon	Common
Hyperuricemia	Moderate	Severe
Hypokalemia	Rare	Common
Rhabdomyolysis	Rare	Common

Heat Stroke Assessment

- History
  - Signs and Symptoms of event
  - Preceding circumstances
  - A description of the neurologic changes
    - Premonitory symptoms such as weakness and dizziness often may go unrecognized, and patients frequently present acutely with collapse.
  - Medical history, medication history and history of illicit drug use
  - Treatment administered

Heat Stroke Assessment

- Physical Examination
  - Vitals (including core body temperature)
  - Neurologic examination
    - Seizures may or may not be present initially but may occur during cooling
    - Coma may be the most common presentation
    - Decorticate posturing may be present
  - Severe volume depletion and peripheral vasoconstriction will be present
  - The onset of coagulopathy may be signaled by ecchymosis, hematemesis, hematuria and epistaxis

Heat Stroke

Management

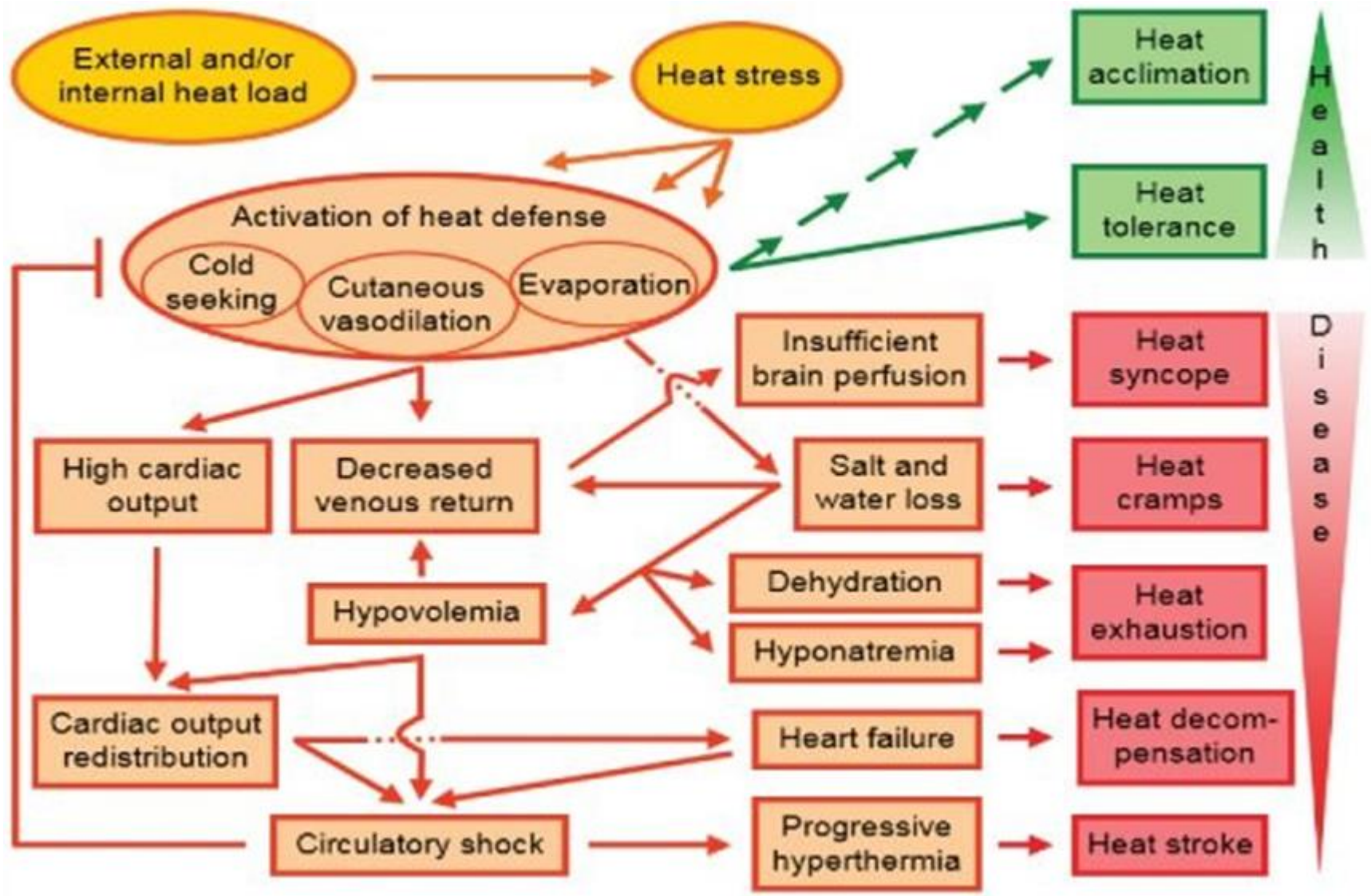
- ABC's (treat as required)
- Early recognition important
- Move pt to cool environment
- Remove excessive clothing
- Begin cooling
- Watch for rebound hypothermia
- IV access
- May require fluid challenge
- 20 ml/kg/hr to maintain BP > 90 mmHg systolic

Dehydration

- Closely associated with heat disorders
- Inhibits vasodilation and thermolysis
- Presentation
  - Orthostatic hypotension
  - Decreased urine output, poor skin turgor
  - Signs of hypovolemic shock

Fever (Pyrexia)

- Elevation of body temperature above normal
  - Pathogens enter body
  - Stimulate production of pyrogens
  - Reset hypothalamic thermostat
- Generally a compensatory mechanism
- Dangerous in very young and elderly


External and/or internal heat load
Heat stress
Heat acclimation
Heat tolerance
Health
Activation of heat defense
Cold seeking
Cutaneous vasodilation
Evaporation
Insufficient brain perfusion
Heat syncope
High cardiac output
Decreased venous return
Salt and water loss
Heat cramps
Hypovolemia
Dehydration
Hyponatremia
Heat exhaustion
Disease
Cardiac output redistribution
Heart failure
Heat decom-pensation
Circulatory shock
Progressive hyperthermia
Heat stroke

Environmental Emergencies

COLD RELATED ILLNESSES

Case 02

- You are responding for a 76 y/o F with Alzheimer's who wondered away from her long term care facility. She also has an extensive cardiac history. The patient was found by searchers three hours later in the nearby woods.
- Upon EMS arrival, the patient is unconscious and not responding. She has slow/shallow spontaneous breathing at 6 BPM and weak carotid at 44 BPM. She is only wearing a housecoat over a johnny shirt and it is soaked through. Her feet and hands are a waxy white appearance. She becomes apneic and pulseless while enroute to the Emergency Dept.

Hypothermia

- Is defined as a core temperature less than 35°C (95°F)
- Most commonly seen in cold climates, but can develop without exposure to extreme environmental conditions
- May result from:
  - A decrease in heat production
  - An increase in heat loss
  - A combination of these factors

Causes

- Accidental (increased heat loss)
- Primary (decreased heat production)
- Secondary (impaired thermoregulation)
- Miscellaneous

Predisposing Factors

- Age
- Peripheral neuropathies
- PVD
- Alcohol and/or tobacco use
- Inadequate protection
- Nutritional deficiencies
- Medication administration
- Injury/illness/fatigue

Hypothermia

- If left untreated, hypothermia can kill
- Nobody ever froze to death

Mechanisms of Heat Loss

- Standard mechanisms
  - Conduction, convection, radiation, evaporation, respiration
- Heat loss increased by:
  - Removal of clothing
  - Wet clothing
  - Air movement
  - Contact with cold or cold water immersion

Preventative Measures

- Dress warmly
- Ensure plenty of rest
  - Restore heat generating mechanisms
  - Replenish energy supplies
- Eat appropriately or at regular intervals
- Limit exposure to cold environments

Degrees of Hypothermia

- Mild
  - Core temp >32°C with signs and symptoms
- Moderate
  - Core temp 28°C -32°C with signs and symptoms
- Severe
  - Core temp <32°C with signs and symptoms
- Compensated
  - Normal core temperature with signs and symptoms

Degrees of Hypothermia

- Acute
  - Rapid onset of symptoms
  - E.g. person suddenly falls through ice
- Subacute
  - More gradual onset
  - E.g. mountain climber
- Chronic
  - Growing problem in inner cities
  - Homeless people endure frequent, prolonged cold stress

Presentation

Mild

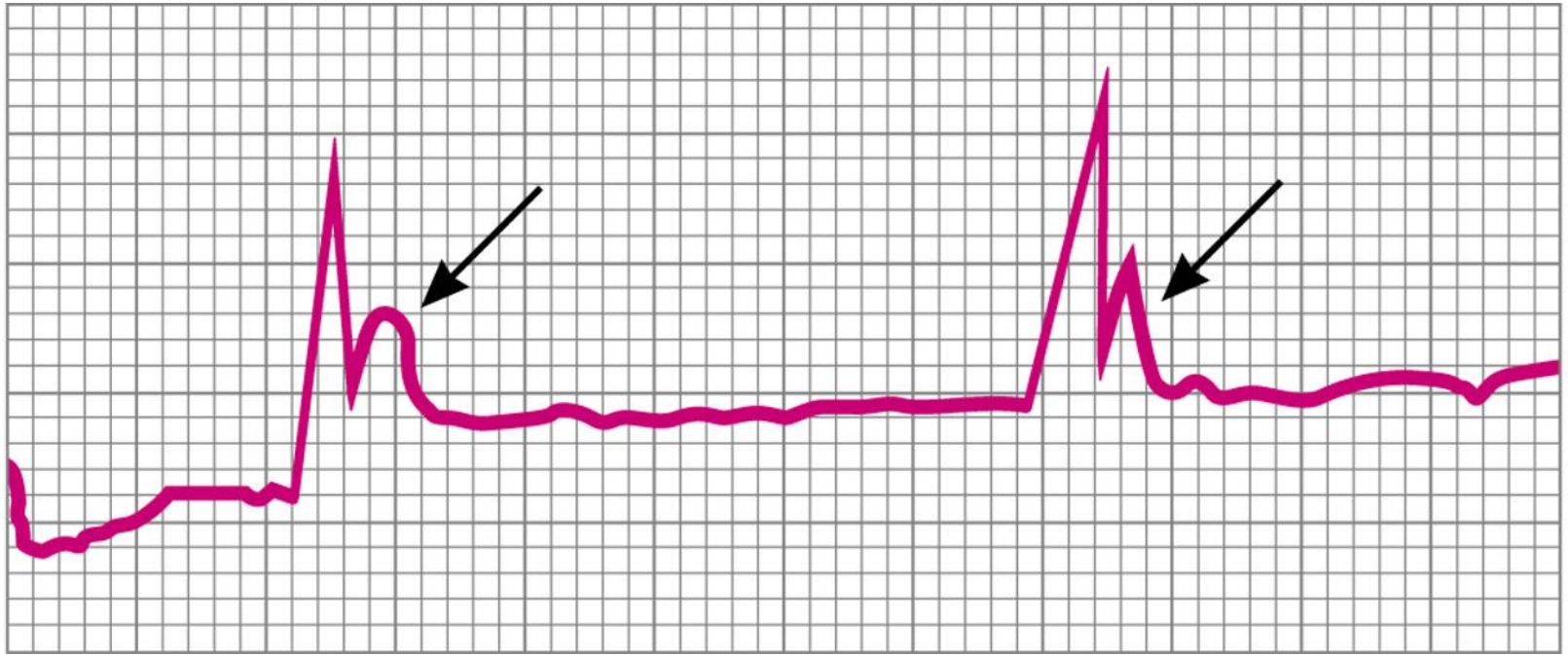
- Shivering
- Lethargic, somewhat dulled mentally
- Muscles stiff and uncoordinated

Severe

- Shivering usually stopped
- Disoriented to unconscious
- Muscles rigid and stiff
- Dysrhythmias especially bradycardia
- Increased risk of VF below 30°C

Osborne Wave

- ECG tracing showing J wave following the QRS complex as seen in hypothermia



Hypothermia

Management

- Remove wet garments
- Protect against further heat loss
- Horizontal position
- Avoid rough handling
- Monitor core temperature
- Monitor cardiac rhythm

Hypothermia

Management

- ABC's
- Secure airway if required (OPA, NPA, ETI)
- Ventilate as required

- Reduce Heat Loss
  - Additional layers of clothing
  - Dry clothing
  - Increased physical activity
  - Shelter

- Add heat
  - Fire or other external heat source
  - Body to body contact
  - Heat can be applied to transfer heat to major arteries (neck, armpits, groin and palms of the hands)
  - Hot water bottles, warm rocks, towels, compresses
  - For a severely hypothermic person, rescue breathing can increase oxygen and provide internal heat

Rewarming

Mild cases

- Warmed blankets
- Heat packs (insulated)
- IV fluids

Severe hypothermia

- Should be done only in hospital
- High risk of rewarming shock

Afterdrop (Rewarming Shock)

- Core temperature actually decreases during rewarming.
- Caused by peripheral vessels in the arms and legs dilating if they are rewarmed.
- Sends very cold, stagnate blood to the core further decreasing core temperature which can lead to death.
- This blood also is very acidic which may lead to cardiac arrhythmias and death.
- Afterdrop can best be avoided by not rewarming the periphery (rewarm the core only).
- Do not expose a severely hypothermic victim to extremes of heat.

Clinical Features

- Death from hypothermia
  - Breathing becomes erratic and very shallow
  - Semi-conscious
  - Cardiac arrhythmias develop, any sudden shock may set off ventricular fibrillation

Hypothermic Arrest

- When a person is in severe hypothermia they may demonstrate all the accepted clinical signs of death:
  - Cold
  - Blue skin
  - Fixed and dilated pupils
  - No discernable pulse
  - No discernable breathing
  - Comatose and unresponsive to any stimuli
  - Rigid muscles

"You're not dead until you are warm and dead"

Resuscitation

- Primary care
  - Longer pulse and respiration checks
  - Defibrillate one shock only until core temp ↑
- Advanced care
  - Intubation as necessary
  - Drug metabolism reduced
  - One round of defibrillation
- No further treatment until temp >30°C

Transport

- Gentle transportation
  - Due to myocardial irritability
- Patient level or head slightly down
- Consider availability of cardiac bypass rewarming

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Immersion Hypothermia

Immersion Hypothermia

- In relation to hypothermia, cold water has two specific threat characteristics:
  - Extreme thermal conductivity
  - The specific heat of water
- Worsened with saturation of clothing by water
- The body cannot maintain temperature if water is less than 30°C

Immersion Hypothermia

- Sudden immersion in cold water causes:
  - Peripheral vasoconstriction causing increased BP
  - Tachycardia due to anxiety
  - Lethal arrhythmias often occur, especially in patients with cardiovascular/cardioelectrical abnormalities

Immersion Hypothermia

- Immersion hyperventilation is the first risk
  - Immersion in cold water initially causes a breathing pattern of deep, involuntary gasps
  - Followed by a minute or more of deep, rapid breaths, with tidal volumes about five times normal
  - Drowning often occurs especially in conjunction with deep immersion or rough water

Immersion Hypothermia

- Hyperventilation causes alkalosis
- Alkalosis increases the blood's pH
- Physiologic responses to alkalosis causes cerebral hypoxia
- Syncope increases the risk of drowning

Immersion Hypothermia

- After as little as 5 minutes in icy water the patient is no longer able to assist in his or her rescue
  - In such cases water rescue is imperative
  - Hypothermia does not cause deaths early in cold water immersion emergencies
  - Death results from drowning or cardiac arrhythmias

Immersion Hypothermia

- After 10-15 minutes of immersion, shivering is constant and obvious
- Core temp cooling has not occurred
- Shivering may temporarily prevent heat loss in dry air, but not in cold water
- Core temp fall commonly occurs around 15-20 minutes in cold (50°F (10°C)) water

Environmental Emergencies

LOCAL COLD INJURIES

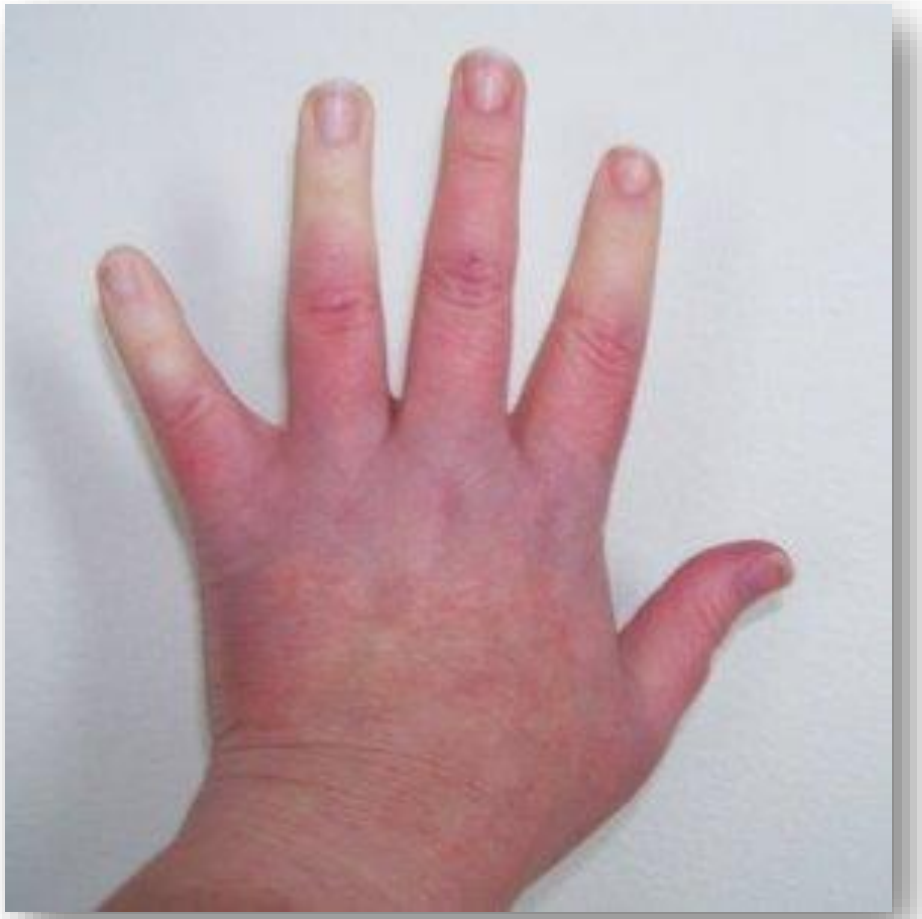
Local Cold Injuries

- Type of injury depends on
  - Degree of the cold to which the body is exposed
  - Duration of exposure

Classifications

- Practical purposes place them as:
  - Non-freezing
    - Frostnip
    - Chillblains
    - Immersion foot (trench foot)
  - Freezing
    - Superficial frostbite
      - There is at least some minimal tissue loss
    - Deep frostbite
      - There is significant tissue loss even with appropriate therapy

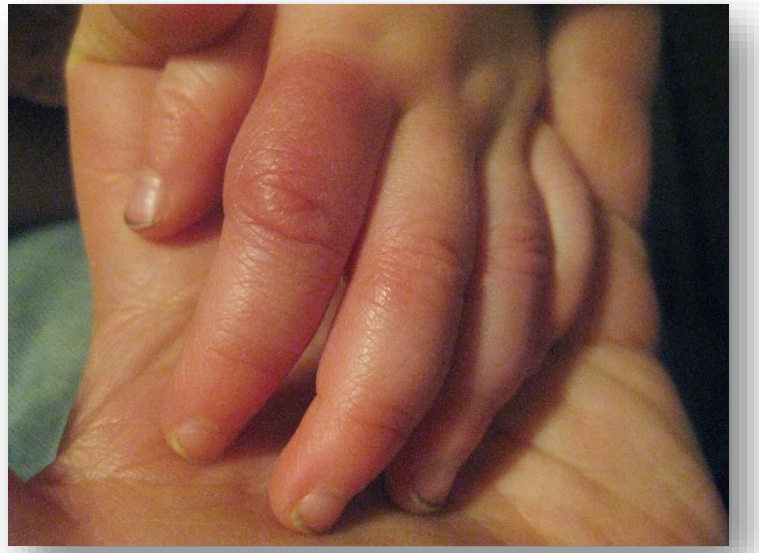
Frost Nip

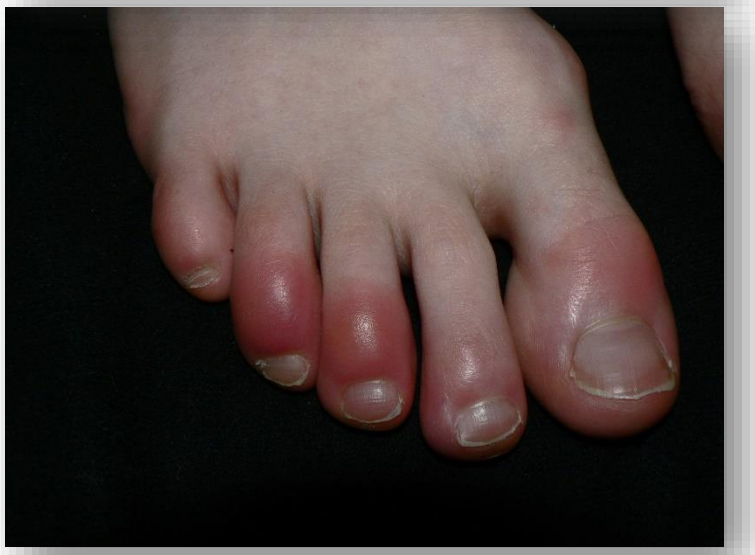


- Usually affects the tips of the ears, nose, cheeks, chin, tips of the fingers and toes usually in conditions of high wind, extreme cold or both
- It is manifested as a sudden blanching or whiteness of the skin characterized by numbness, coldness, and pain without swelling

Chilblains

- A more significant nonfreezing injury
- As tissue temperature drops below 15°C (59°F) tissue injury progresses
- Capillary walls damaged
- Tissue swelling occurs



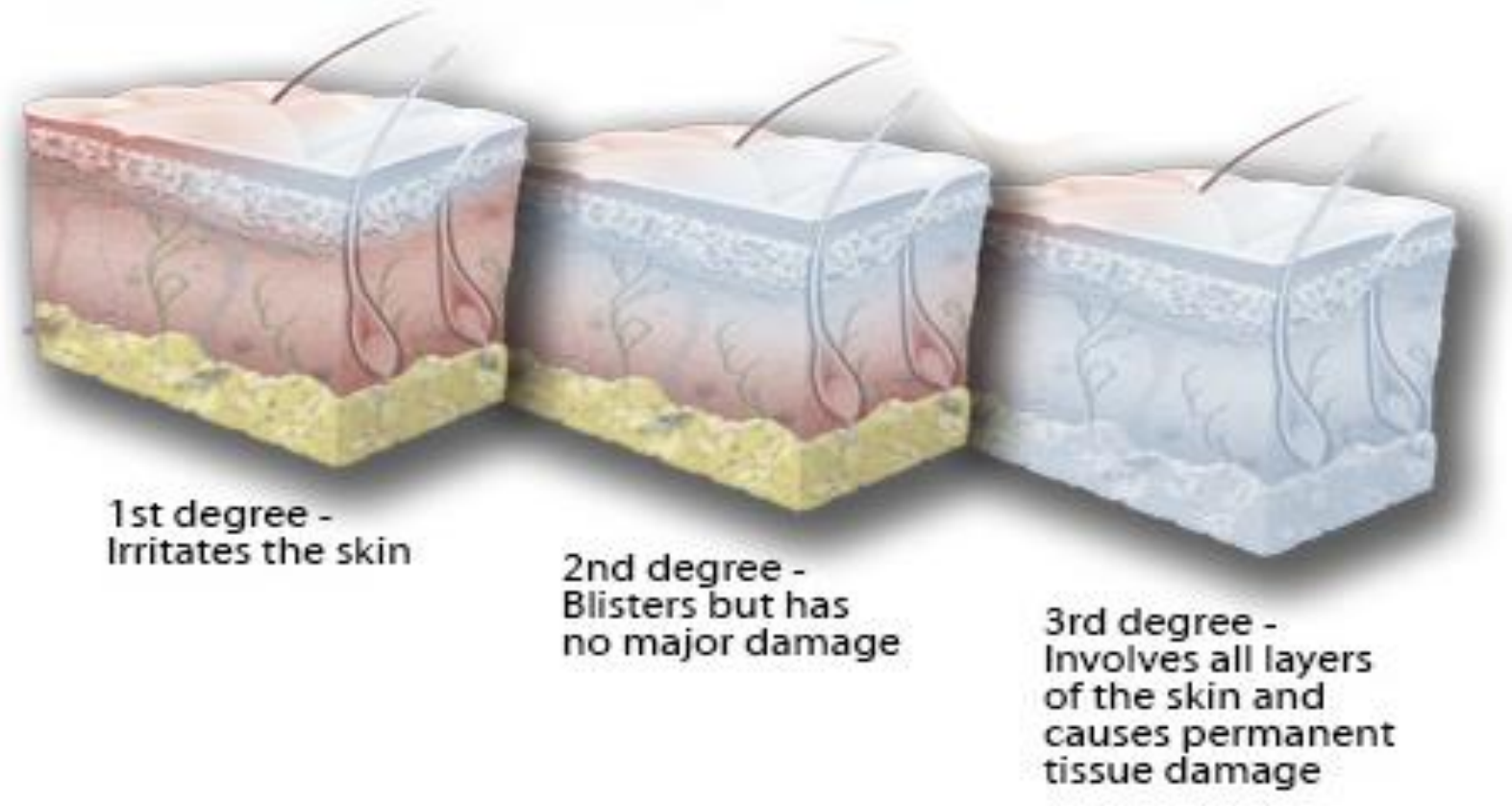


Immersion Foot (Trench Foot)

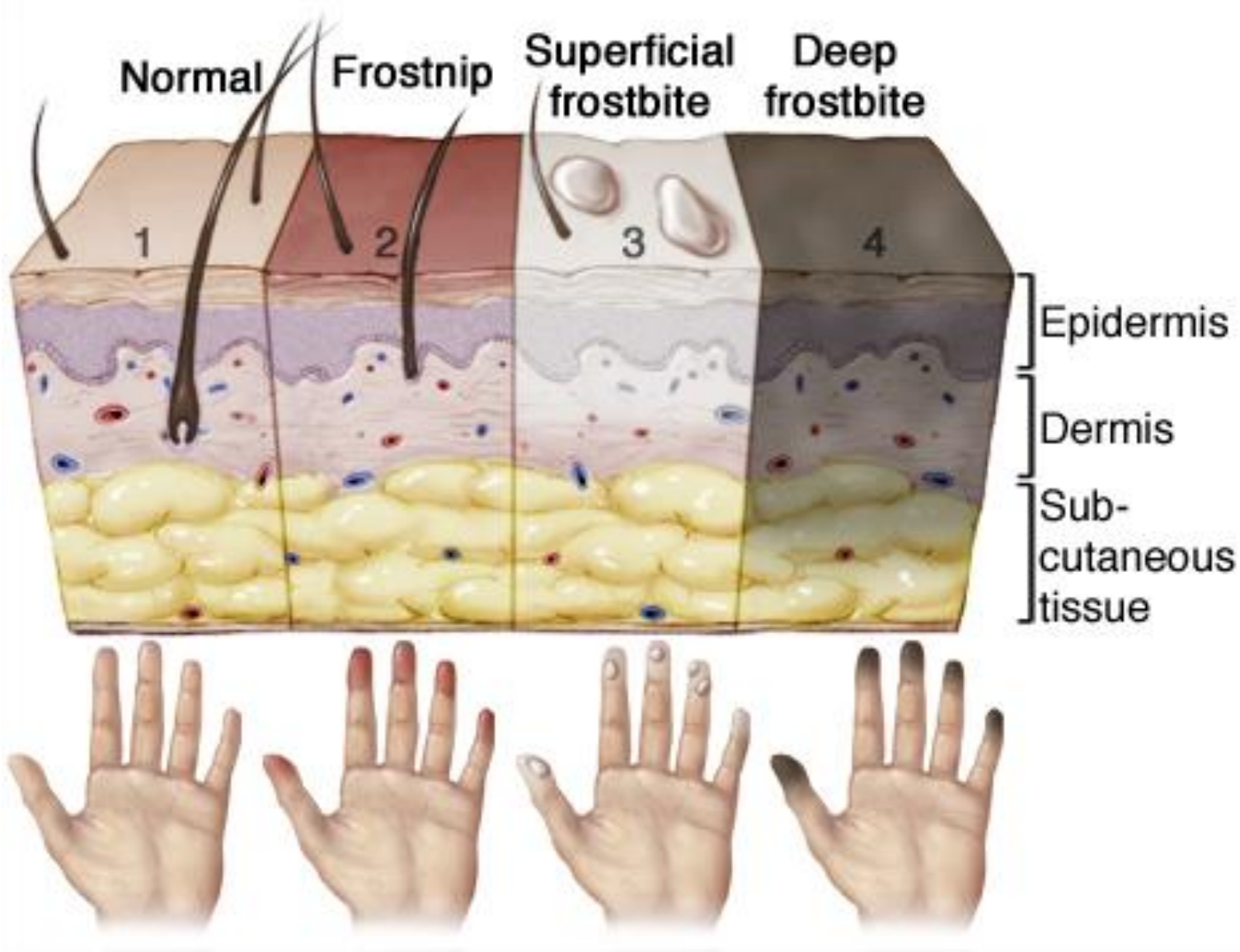
- Occurs when feet are cold and damp while wearing constricting footwear
  - Does not require freezing temperatures (can occur in temperatures up to 16°C)
  - Can occur with only twelve hours of exposure

Frostbite

- A localized injury that results from environmentally induced freezing of body tissues
  - Superficial frostbite
  - Deep frostbite



Frostbite

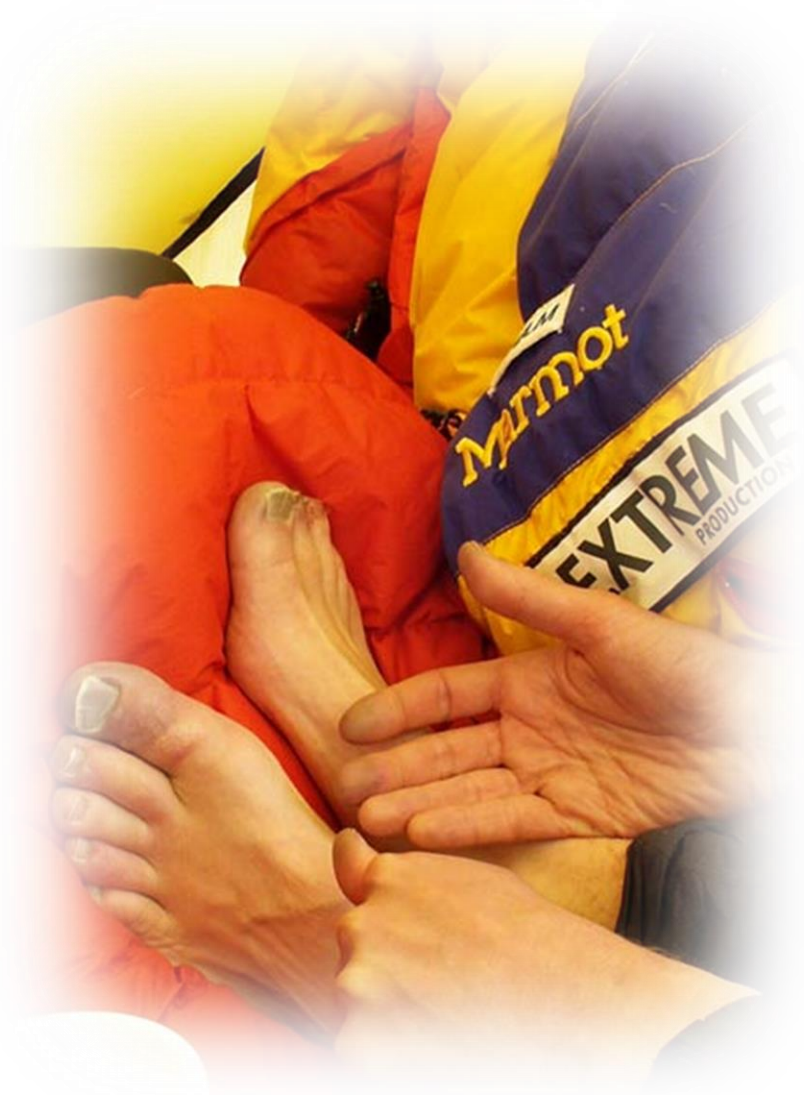

Normal
Frostnip
Superficial frostbite
Deep frostbite
1
2
3
4
Epidermis
Dermis
Sub-cutaneous tissue

Frostbite

- Environmentally induced freezing of body tissues
  - Ice crystals form
  - Water drawn out of cells
  - Crystals expand causing destruction of cells
  - Damage to blood vessels causes loss of vascular integrity
  - Tissue swelling and loss of nutritional flow

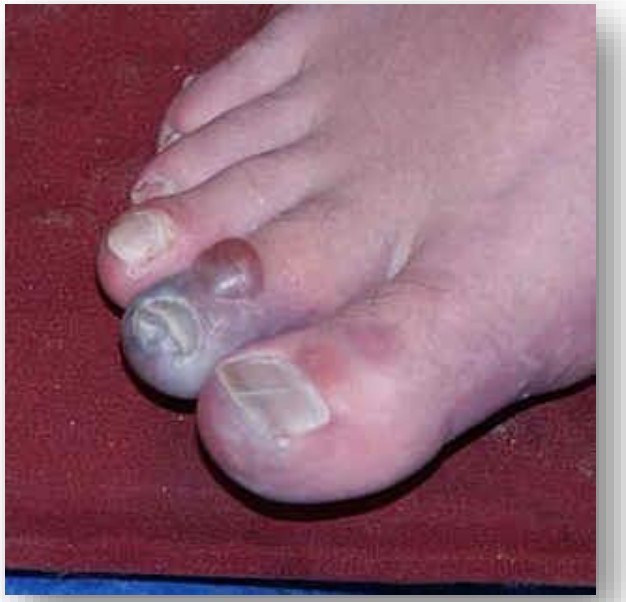
Superficial Frostbite

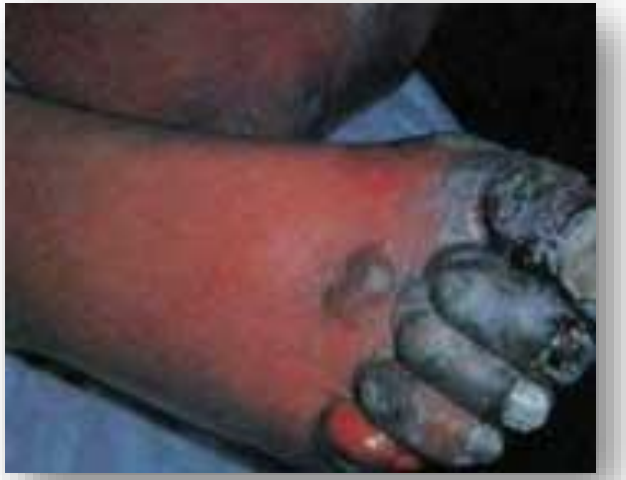
- Some freezing of dermal tissue
- Initial redness followed by blanching
- Diminished tactile sensation
- Pain



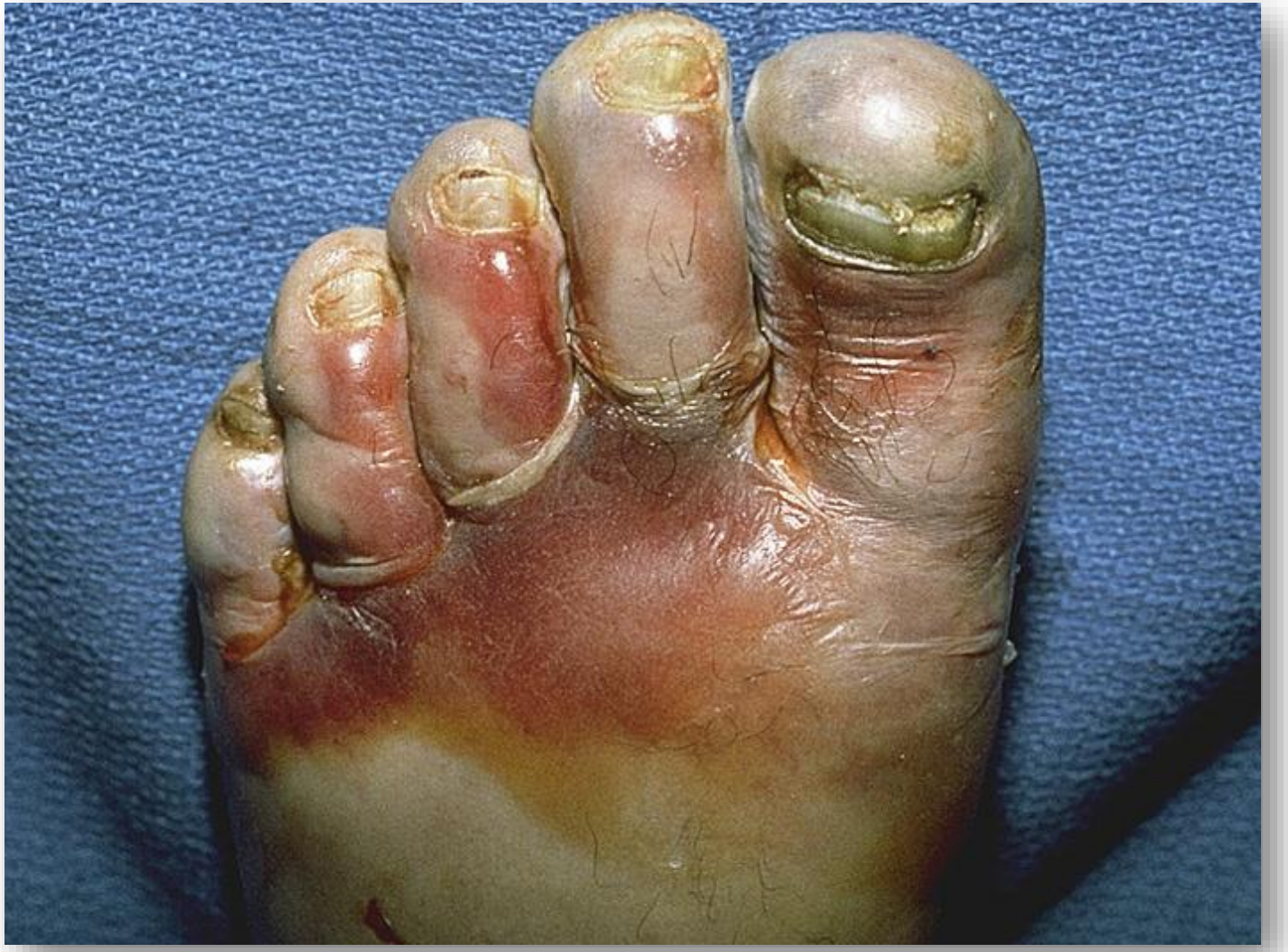
Deep Frostbite

- Freezing of dermal and subcutaneous layers
- White appearance
- Hard (frozen) to palpation
- Loss of sensation





Frostbite



Frostbite

Management

- Gentle handling
- Do not massage the affected area
- Do not puncture or drain blisters
- Do not rewarm feet if walking will be required
- Administer analgesia prior to thawing.
- Rewarm by immersion only if transport is lengthy or delayed
- Cover the thawed part with a loose, sterile dressing
- Elevate and immobilize the thawed part.

Environmental Emergencies

WATER RELATED EMERGENCIES

Case 03

- You respond to a lake for a report of 22 y/o F water skier who wiped out and was not found until five minutes later, face down in the water. A lifeguard on scene determined the patient to be apneic but did have a pulse. AR with pocket mask is begun immediately.
- Upon EMS arrival, the patient has begun to breathe on her own at 6-8/min. Other vitals included HR 106, BP 112/76, PEARL (sluggish), BGL 7.7mmol. Physical Exam unremarkable except A/E = with bilat crackles.

Drowning or Submersion

- Populations at high risk of death due to drowning
  - Toddlers
  - Youth aged 15–19 years
  - People with seizure disorders
  - Recreational fishermen
  - Aboriginal men aged 25–34 years
- Drowning remains one of the leading causes unintentional injury in Canada.
  - Leading cause of death for recreational and sporting activities
  - Numbers are declining.

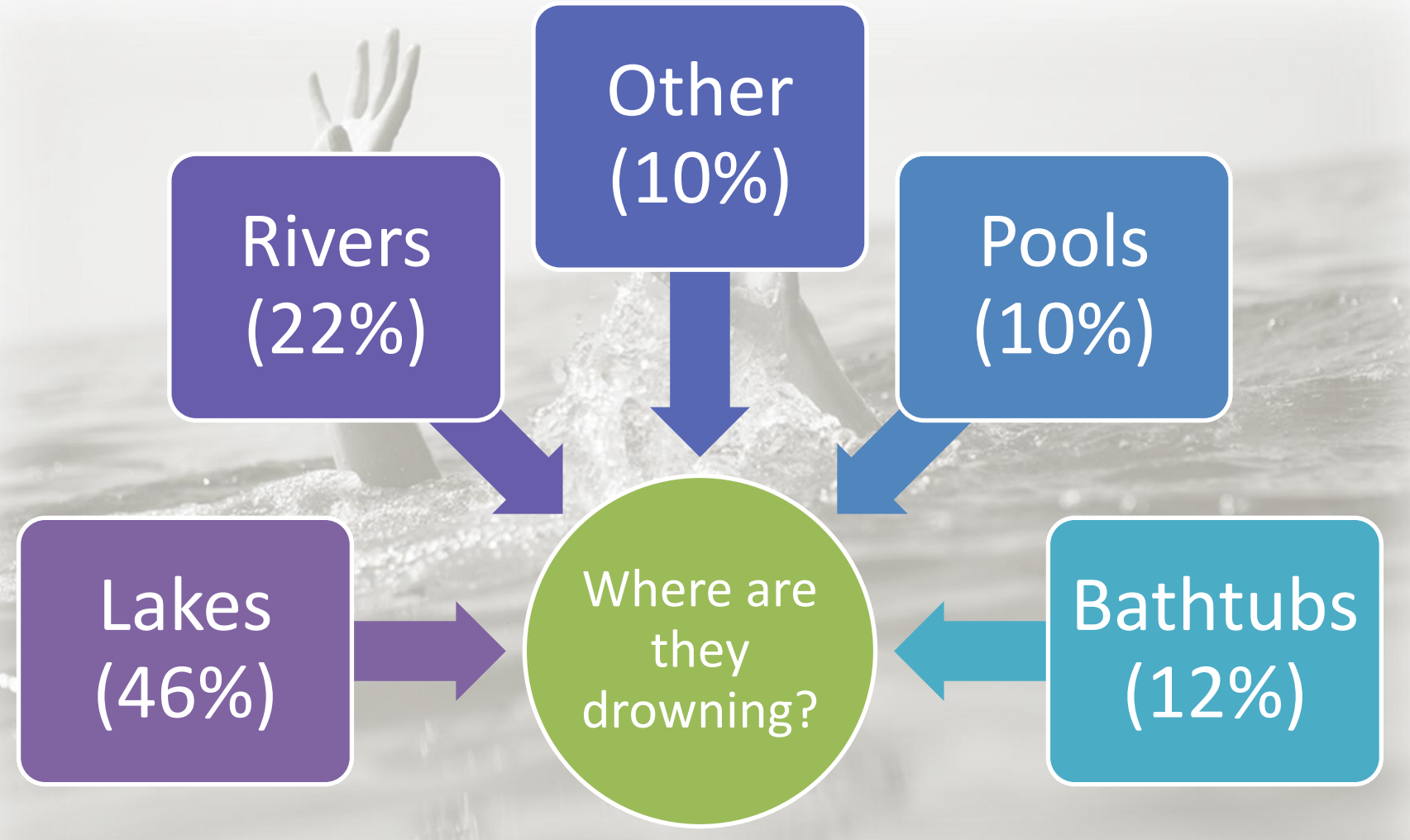
Drowning or Submersion

- At one, point 33 different definitions existed.
- Definition now in use: Process of experiencing respiratory impairment from submersion in liquid
  - Patient can be resuscitated at any point along the drowning continuum.

Table 38-8 Risk Factors for Drowning

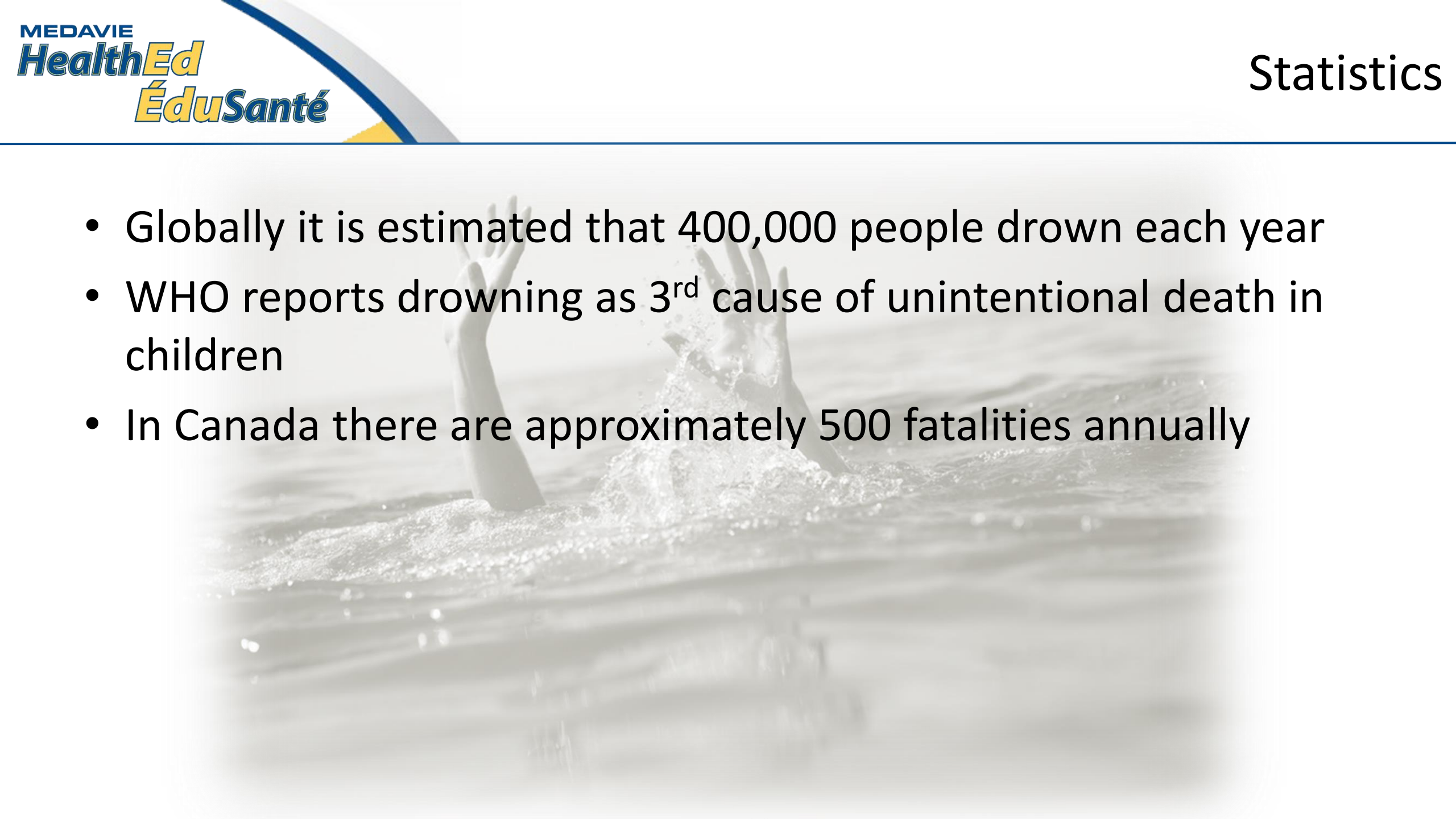
- Male gender
- Younger than 20 years (even higher for younger than 5 years)
- Preexisting conditions, such as seizure disorder, mental/physical handicaps
- Alcohol use
- Ineffective safety barriers (gates, locks, fences)
- Hyperventilation (may lead to shallow water blackout syndrome)

Statistics


Other (10%)
Rivers (22%)
Pools (10%)
Lakes (46%)
Where are they drowning?
Bathtubs (12%)

Statistics

- Globally it is estimated that 400,000 people drown each year
- WHO reports drowning as 3rd cause of unintentional death in children
- In Canada there are approximately 500 fatalities annually



Pathophysiology

Submersion

Apnea

- Involuntary reflex as victims strives to keep head above water
- Blood shunted to heart and brain

Hypoxia, hypercarbia

Drowning Pathophysiology

- Sequence of drowning:
  - After submersion and panic
  - Victim takes several deep breaths to conserve oxygen
  - Holds breath until reflex takes over
  - Water is aspirated causing laryngospasm (“Dry drowning”)
  - This results in hypoxia
  - Water begins to enter the lungs (“Wet drowning”)
  - Hypoxia leads to arrhythmias and CNS anoxia
  - Hypercapnia begins
  - Acidosis
  - Cardiac arrest

Pathophysiology

- Dry drowning
  - Water enters mouth and oropharynx
  - Laryngospasm and bronchospasm
- Wet drowning
  - Water enters lungs
- Secondary drowning
  - Hypoxia
  - Fluid causes lower airway irritation
  - Symptoms can begin hours after the drowning or “close call”

Pathophysiology

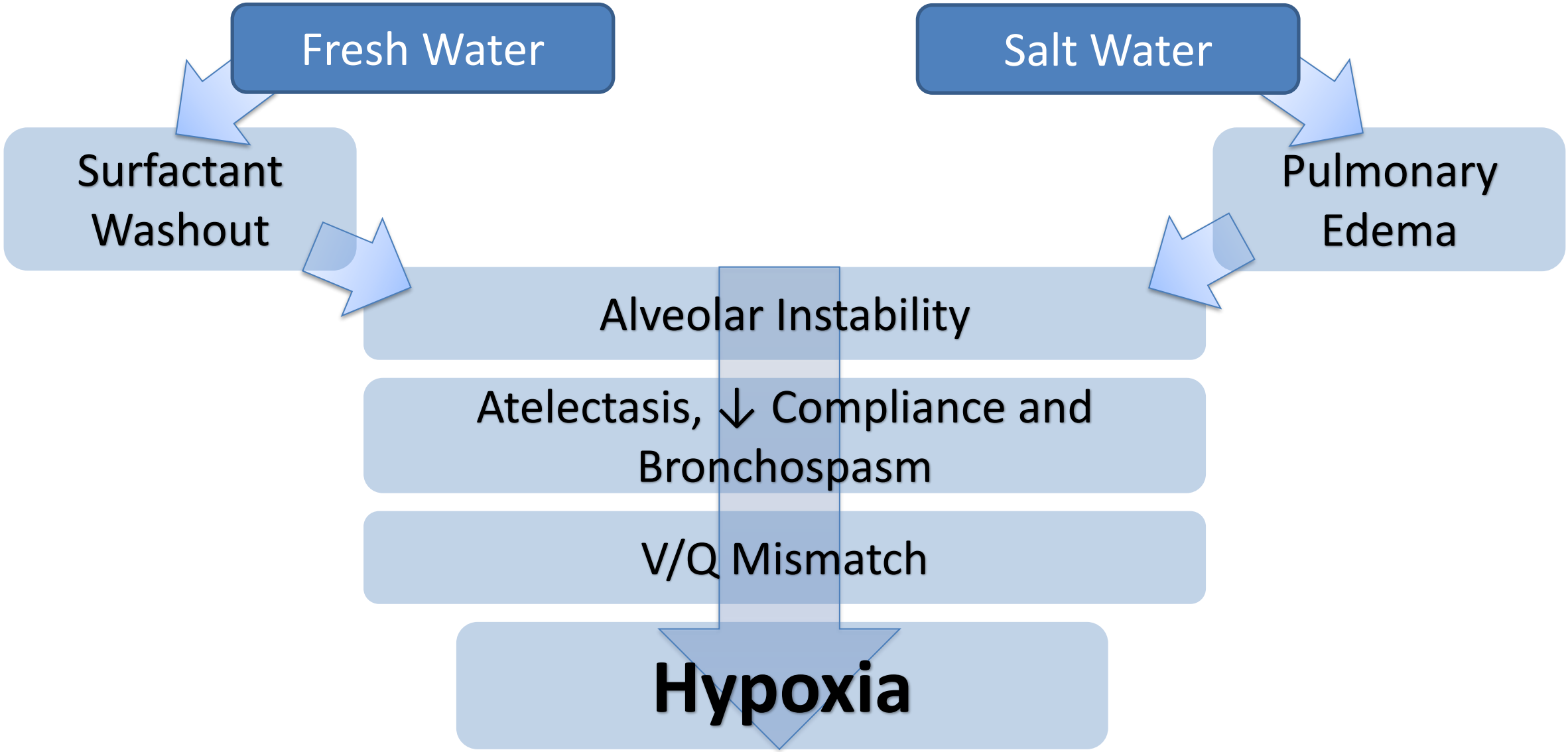
Fresh water drowning

- Hypotonic water diffuses across alveoli
- Hemodilution, expansion of blood volume
- Hemorrhagic pneumonitis, surfactant washout

Salt water drowning

- Hypertonic solution
- Draws water from blood into alveoli
- Profound shunting, hypoxemia
- Pulmonary Edema as a result

Pathophysiology



Factors Affecting Survival

- Cleanliness of water
- Length of time submerged
- Victim's age and general health
- Water temperature
  - Cold-water drowning.
  - Mammalian diving reflex.
  - The cold-water drowning patient is not dead until he is warm and dead.

Mammalian Diving Reflex

- Results from submersion of the face and nose in cold water, the result is constriction of blood flow everywhere except to the brain (the patient is not dead until they are WARM and dead)

Drowning or Submersion

Management

- Remove the patient from the water
- Initiate ventilation
- Suspect head and neck injuries
- Protect the patient from heat loss
- Anticipate airway problems
- Oxygen therapy
- Support ABCs
- Intravenous access

Acute Respiratory Distress Syndrome

- Post resuscitation complication
  - High mortality
- Inflammation of alveolar tissue
  - Parenchymal injury
  - Destruction of surfactant
  - Aspiration pneumonitis, pneumothorax
- Noncardiogenic pulmonary edema

- Symptoms may be subtle (slight cough, mild tachypnea) or patient may be asymptomatic.
- Patients may still die of drowning hours after the incident and after a period of apparent stability.

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Environmental Emergencies

DIVING EMERGENCIES

Case 04

- A call comes in from a local pier where a boat is arriving with a 50 y/o M diver who is having trouble breathing. The boat is docking when you arrive, and you see a diver on the deck wearing a blood-spattered oxygen mask. His wife, who was diving with him, says they were fairly deep and had begun ascending to end the dive when she noticed her husband had not ascended and was many feet below her, staring into the water. She banged on her tank to attract his attention and headed toward him, but, before she could reach him, he looked at his air gauge and headed “like a rocket” to the surface.

Case 04

- Immediately after surfacing he began complaining of pain in his chest and difficulty breathing. The crew placed him on oxygen and, within minutes, he began coughing up blood. The rapid ascent and quick onset of signs and symptoms indicates this may be a case of pulmonary barotrauma. His confused behavior during the dive indicates he may have been experiencing nitrogen narcosis, which ultimately leads to a rapid and dangerous ascent. He should be provided oxygen and transported immediately.

Diving Injuries

- Boyle's Law
  - The volume of a gas is inversely proportional to its pressure if the temperature is kept constant.
- Dalton's Law
  - The total pressure of a mixture of gases is equal to the sum of the partial pressures of the individual gases.
- Henry's Law
  - The amount of gas dissolved in a given volume of liquid is proportional to the pressure of the gas above it.

Pathophysiology

- Increased dissolution of gases during descent due to Henry's law.
- Boyle's law dictates that these gases have a smaller volume.
- In a controlled ascent
  - Gases escape through respiration.
- A rapid ascent
  - Gases to come out of solution quickly
  - Form gas bubbles in the blood, brain, spinal cord, skin, inner ear, muscles and joints.

Classification of Diving Emergencies

- Injuries on the surface
- Injuries during descent
  - Barotrauma
- Injuries on the bottom
  - Nitrogen narcosis
- Injuries during ascent
  - Decompression illness
  - Pulmonary overpressure

General Assessment

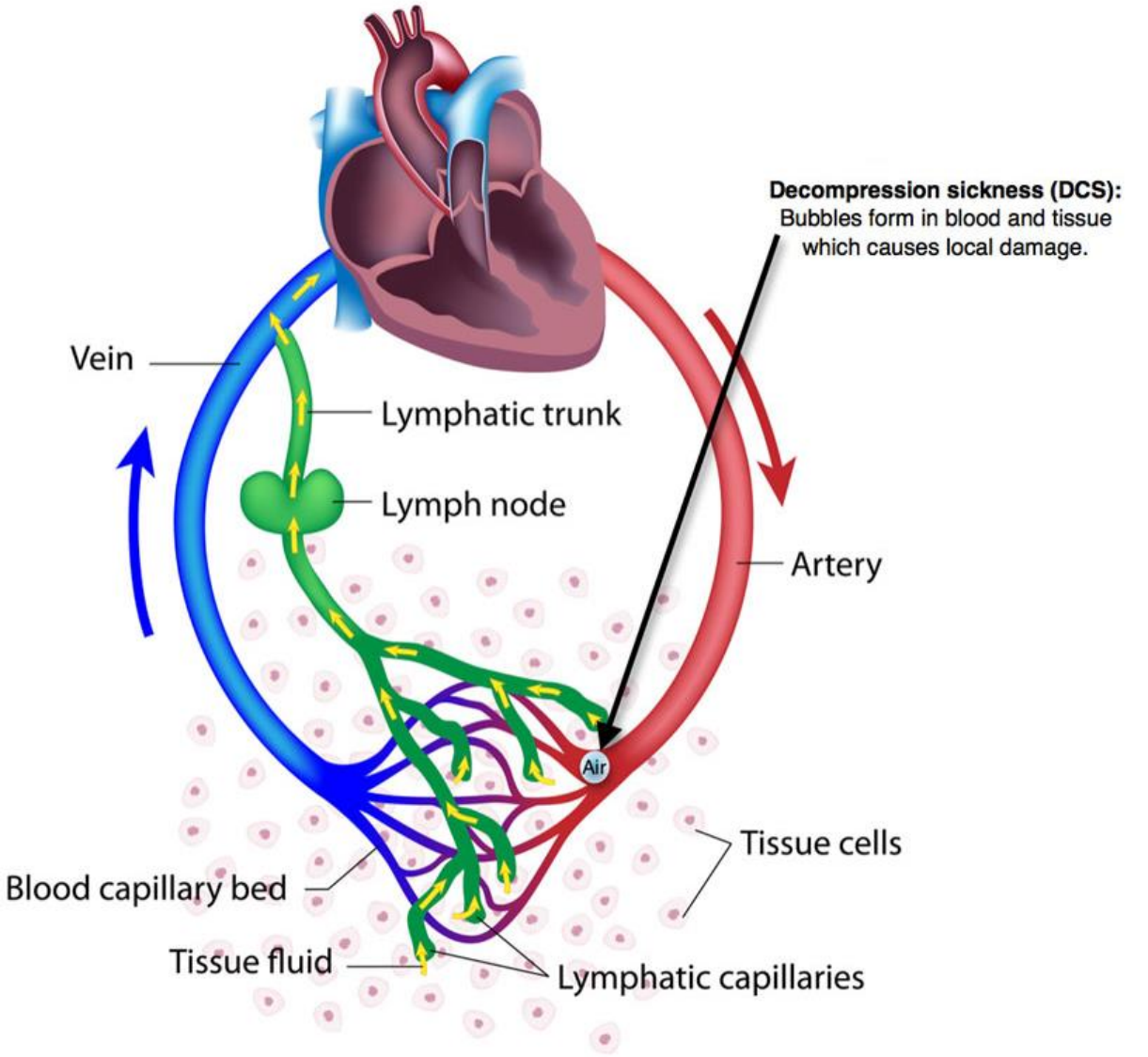
- Time at which signs and symptoms appeared
- Type of breathing apparatus used
- Type of hypothermia-protective garment worn
- Parameters of the dive
  - Number of dives, depth and duration
- Aircraft travel following a dive

General Assessment

- Rate of ascent
  - Associated with panic forcing a rapid ascent
- Inexperience of the diver
- Improper functioning of depth gauge
- Previous medical diseases
- Old injuries
- Previous episodes of decompression illness
- Use of alcohol or medications

Decompression Illness

- Exposure to depth > 10 meters
  - Long enough to allow tissues to be saturated with nitrogen
- Rapid ascent
  - Nitrogen bubbles out of solution
- Increases pressure in body structures
  - Occludes circulation in joints, tendons, CNS, inner ear



Presentation

- Symptoms occur within 36 hours
- Joint/abdominal pain
- Fatigue, paraesthesias and CNS disturbances
- Obstruction of blood flow
  - Local ischemia
  - Anoxic stress

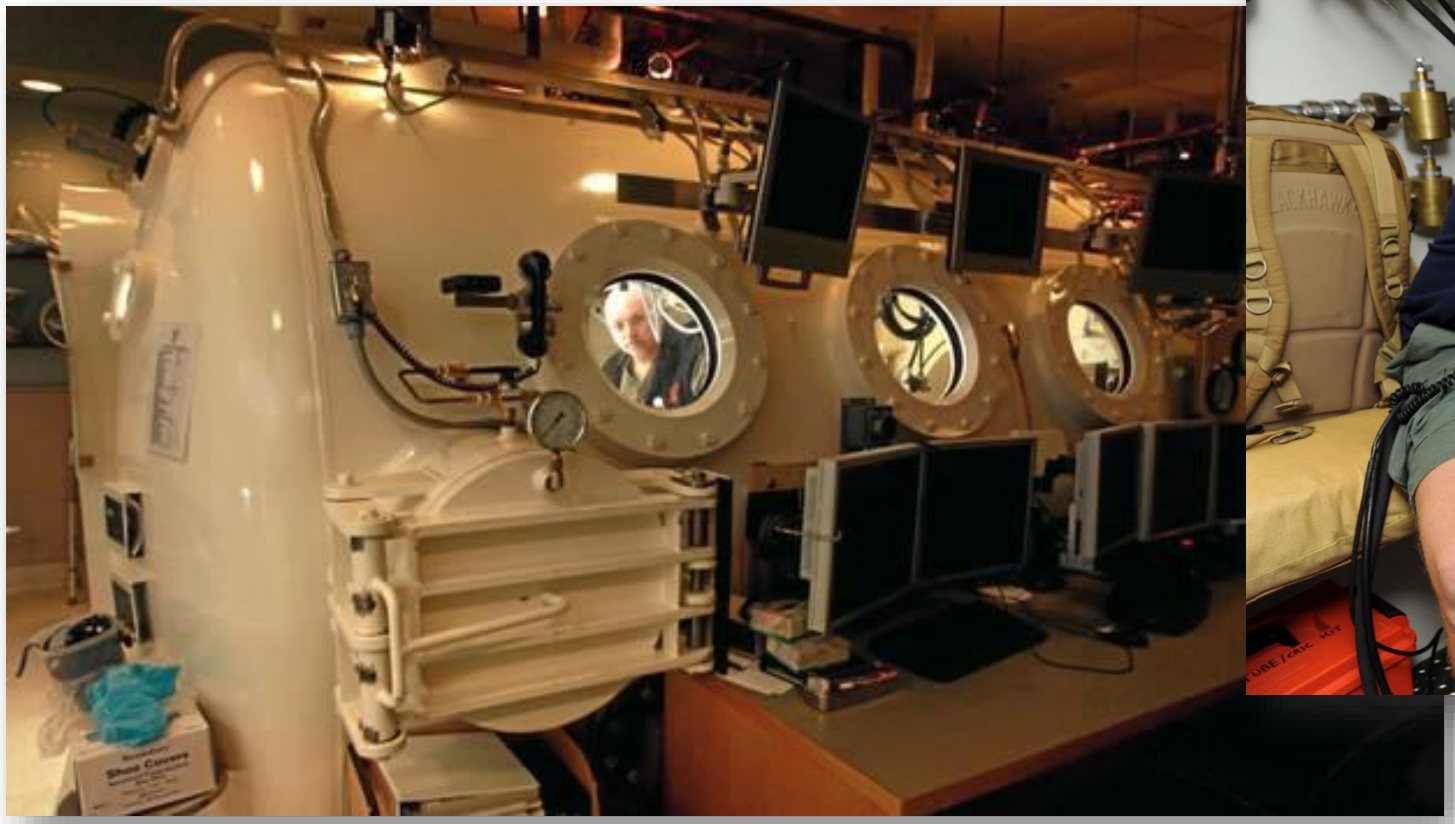
Decompression Illness

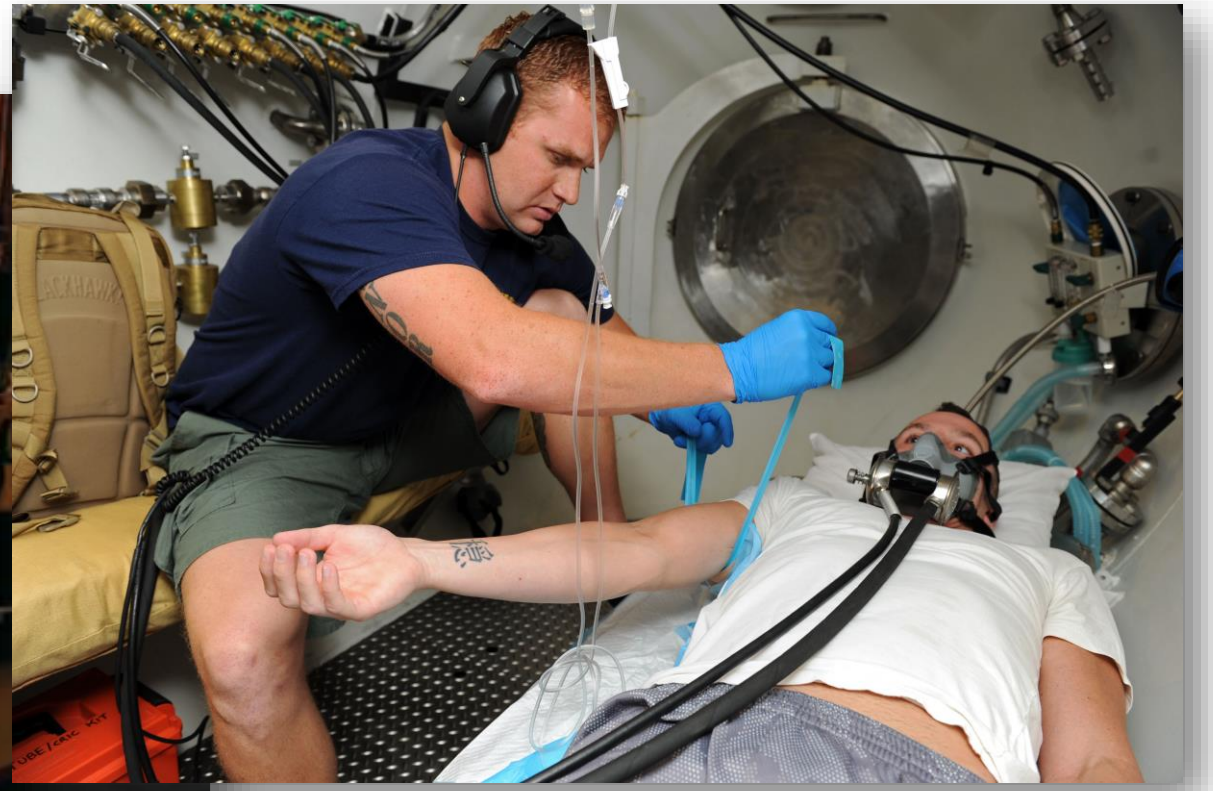
Management

- Supportive care
- Oxygen therapy, airway management
- Keep patient supine
- Fluid administration
- Rapid transport
- Appropriate ER
- Hyperbaric facility

Hyperbaric Chamber

- Hyperbaric oxygen chamber used in treatment of decompression illness



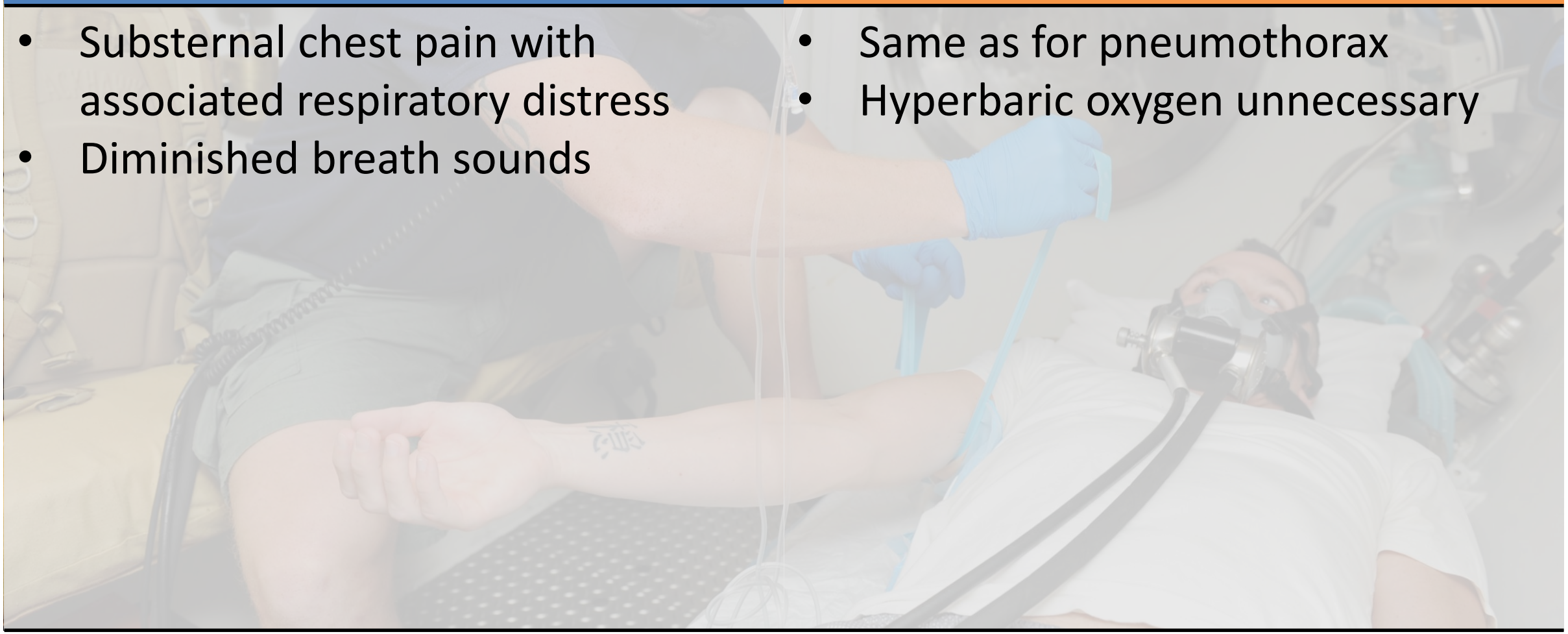


Pulmonary Overpressure Accidents

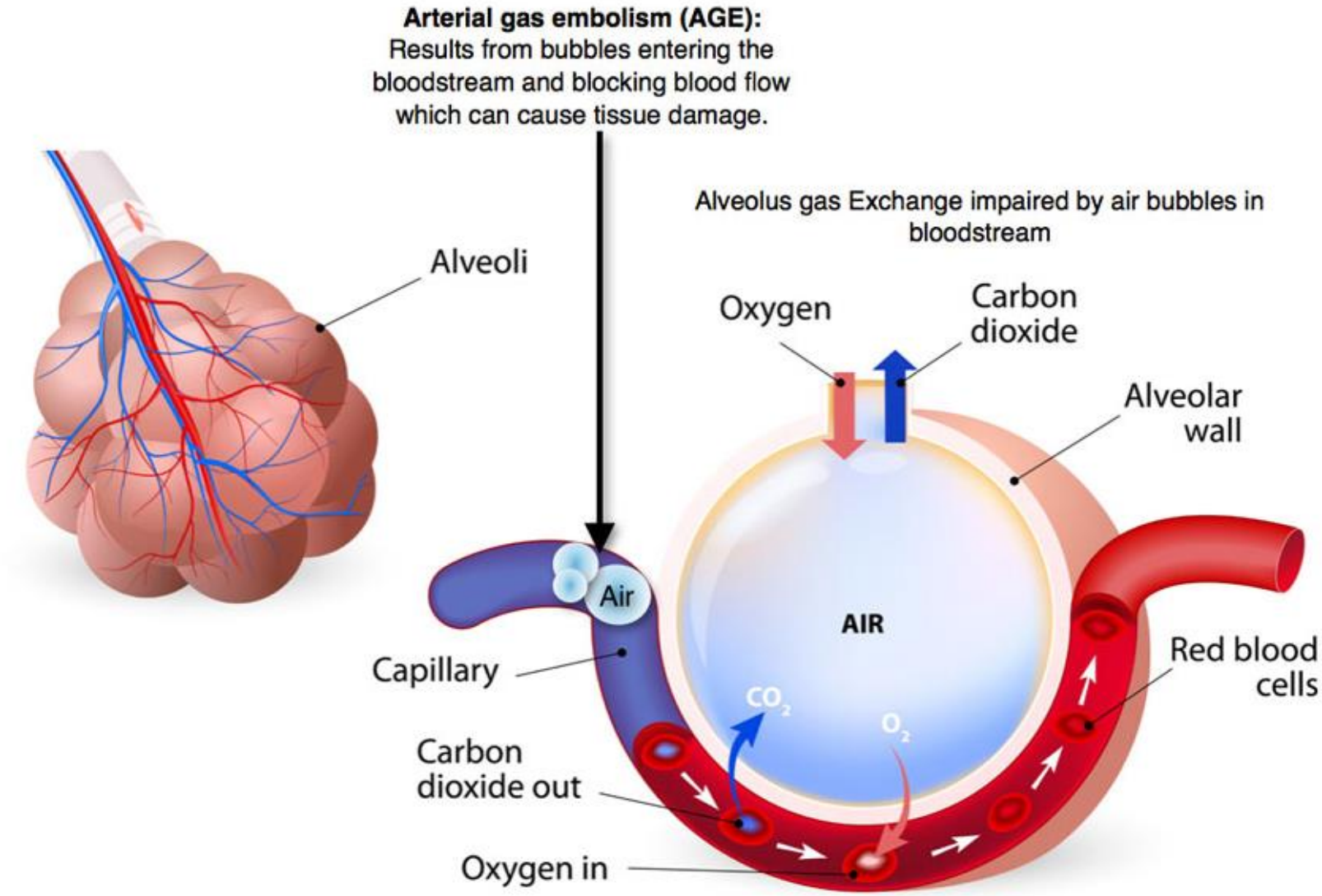
- Can occur at depths < 2 meters
- Breath holding
  - Air becomes trapped in lungs
  - Allowed to expand
  - Ruptures alveolar membranes
  - Hemorrhage, reduced gas transport
- Air may escape into nearby tissues
  - Pneumothorax, subcutaneous emphysema

Pulmonary Overpressure Accidents

Signs and Symptoms	Management
• Substernal chest pain with associated respiratory distress • Diminished breath sounds	• Same as for pneumothorax • Hyperbaric oxygen unnecessary



Arterial Gas Embolism (AGE)

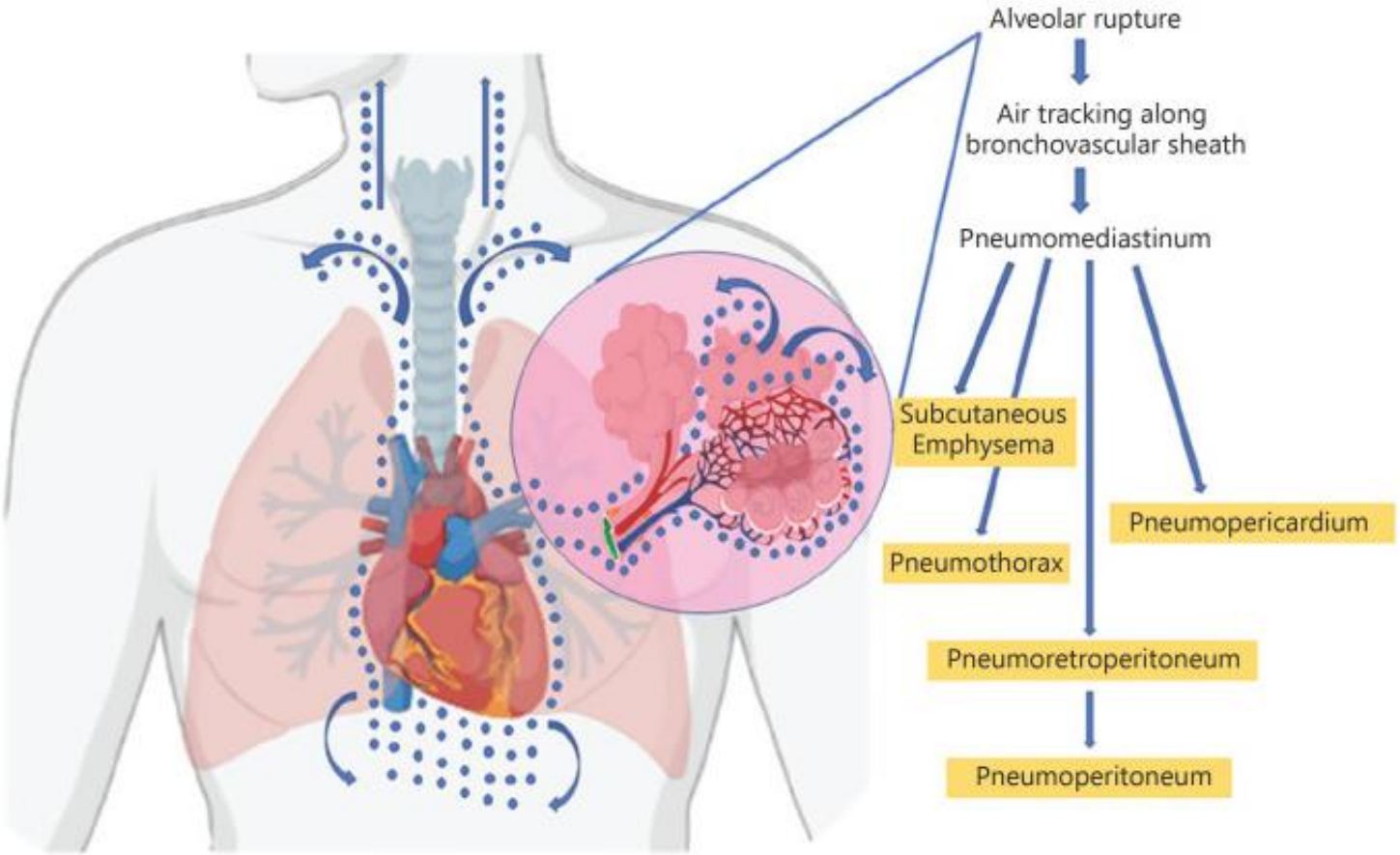


Arterial Gas Embolism (AGE)

Signs and Symptoms	Management
• Rapid and dramatic onset after ascent • Sharp, tearing pain • Often mimics a stroke • Suspect in patients presenting with neurological deficits immediately after ascent	• Support ABCs • Oxygen therapy • Maintain a supine position • Monitor vital signs frequently • Establish IV access • Rapid transport to a recompression chamber

Pneumomediastinum

- Release of air through visceral pleura into mediastinum and pericardial sac

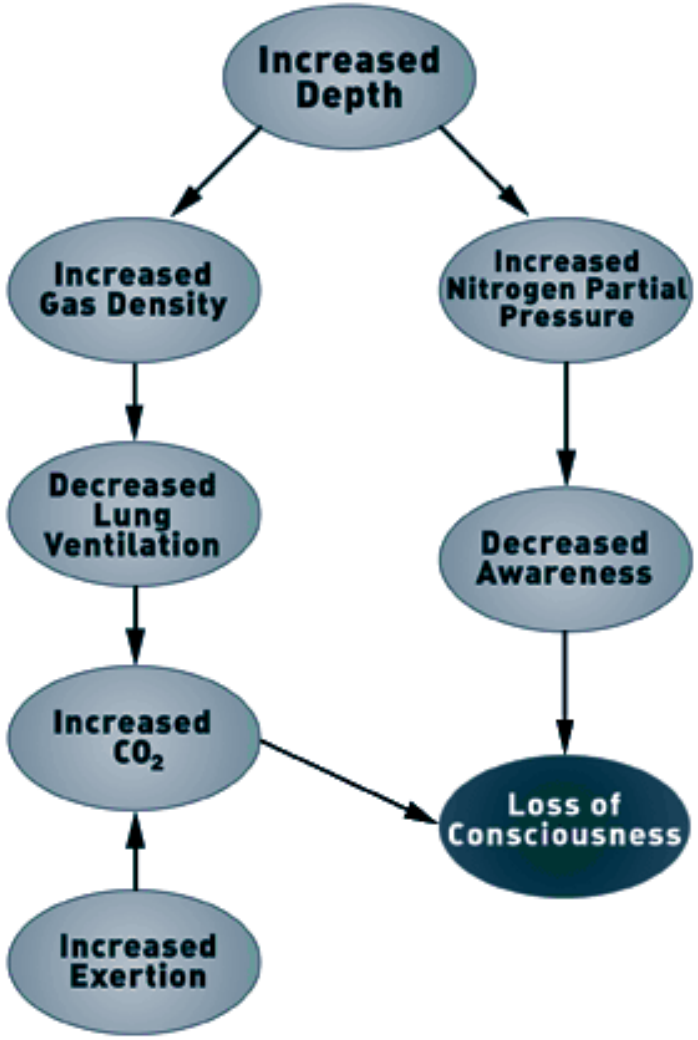


Pneumomediastinum

Signs and Symptoms	Management
• Substernal chest pain • Irregular pulse, abnormal heart sounds • Hypotension • Narrow pulse pressure	• Support ABCs • Oxygen therapy • IV access • Rapid transport • Treatment ranges from observation to decompression

Nitrogen Narcosis

- Occurs during deeper dives
- More nitrogen dissolves into blood
- Intoxication, altered LOC
- Management
  - Return to shallow depth
  - Use oxygen/helium mixture in deeper dives



Diving Related Issues

- Oxygen toxicity
  - Prolonged exposure to high partial pressures
  - May cause lung collapse
- Hyperventilation
  - Due to excitement or panic
- Hypercapnea
  - Inadequate breathing or faulty equipment
  - Increased CO_2
  - May lead to unconsciousness

Environmental Emergencies

ALTITUDE ILLNESS

Altitude Illness

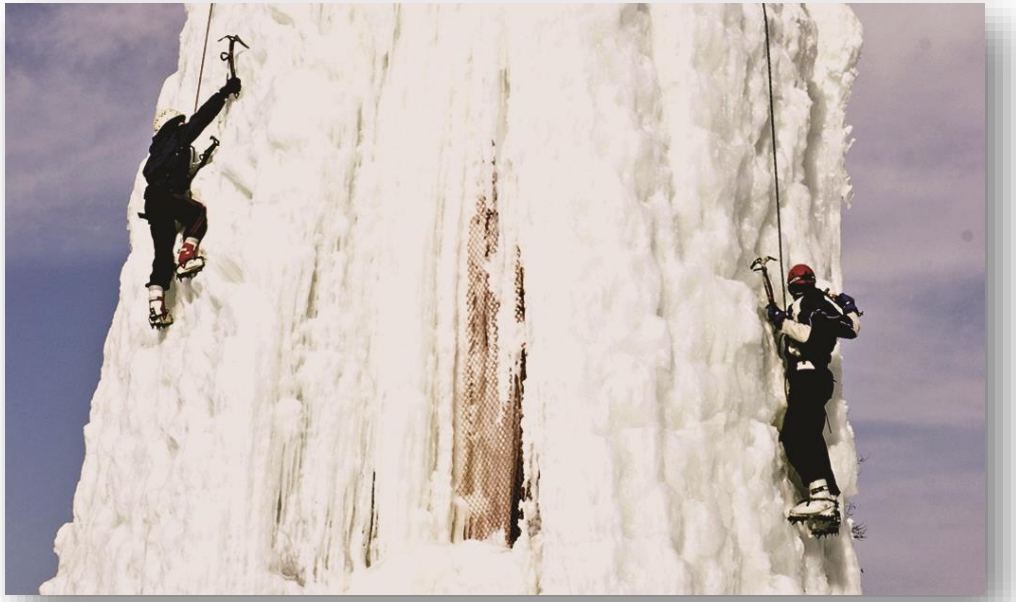
- Cause
  - Effects of hypobaric hypoxia on the CNS and pulmonary system
  - Unacclimatized people ascending to altitude
- Types
  - Acute mountain sickness (AMS)
  - High-altitude cerebral edema (HACE)
  - High-altitude pulmonary edema (HAPE)

Altitude Illness

- Likely to affect
  - People who rapidly ascend to heights above 2,500 m
  - Can occur at altitudes as low as 2,000 m
- Hypoxia
  - Low atmospheric pressures
  - Partial pressure of oxygen in the atmosphere decreases with increasing altitude.
  - Barometric pressure varies according to how far north you are located.
  - Typically lower in winter

Risk Factors for Altitude Illness

- History of AMS
- Normal residence below 914 m (3,000 ft)
- Physical exertion
- Presence of COPD
- Sleeping above 2,438 m (8,000 ft)
- Physical fitness is not a factor.



The Clinical Picture of Altitude Illness

- Acute mountain sickness (AMS)
  - All three of the following criteria must be present:
    - Recent gain in altitude
    - At least several hours at the new altitude
    - The presence of headache combined with any one of these symptoms: fatigue or weakness, gastrointestinal symptoms (nausea, vomiting, or loss of appetite), dizziness or lightheadedness, or difficulty sleeping
    - The headache is often described as throbbing that is worse over the temporal or occipital areas and is exacerbated by the Valsalva maneuver.

The Clinical Picture of Altitude Illness

- High-altitude pulmonary edema (HAPE)
  - At least two of the following symptoms:
    - Dyspnea at rest
    - Cough
    - Weakness or decreased exercise performance
    - Chest tightness or congestion
  - Also, at least two of the following signs: central cyanosis, audible rales or wheezing in at least one lung field, tachypnea, or tachycardia

The Clinical Picture of Altitude Illness

- High-altitude cerebral edema (HACE)
  - Requires the presence of a change in mental status and/or ataxia in a person with AMS
  - Presence of mental status changes and ataxia in a person without AMS
  - Can progress to a coma

Pathophysiology of Altitude Illness

- Hypoxia
  - Main culprit behind the pathophysiologic responses observed in altitude illness
  - Exact mechanism remains poorly understood.
    - Believed to initiate a complex series of reactions that result in overperfusion to the brain and lungs, with resultant increases in capillary pressures, leakage, and then cerebral and pulmonary edema

Management and Prevention of Altitude Illness

- Mainstays of management
  - Oxygen, descent, and evacuation
- Prevention
  - Best accomplished via acclimatization; slower ascents; and occasionally, the use of acetazolamide

Management and Prevention of Altitude Illness

Table 38-13 Management and Prevention of Altitude Illnesses

Clinical Condition	Signs and Symptoms	Management	Prevention
Mild AMS	Headache with nausea, dizziness, and fatigue during first 12 h after rapid ascent to high altitude (>2,438 m [8,000 ft]); "hung over"	Descend ≥500 m (1,600 ft); or rest and acclimatize; or speed acclimatization with acetazolamide (125–250 mg twice daily); or treat symptoms with analgesics and antiemetics; or use a combination of above	Slower ascents; spend a night at an intermediate altitude; avoid overexertion; avoid direct transport to ≥ 2,758 m (9,000 ft); consider taking acetazolamide (125–250 mg twice daily) beginning 1 day before ascent and continuing for 2 days at high altitude
Moderate AMS	Worsening headache with marked nausea, dizziness, poor sleep, fluid retention at high altitude >12 h	Descend ≥ 500 m (1,600 ft); if unable, use a portable hyperbaric chamber or low-flow supplemental oxygen (1–2 L/min); if descent is not possible and oxygen is not available, give acetazolamide (250 mg twice daily), dexamethasone (4 mg orally or intramuscularly every 6 h), or both until symptoms resolve; treat symptoms; or use a combination of above	Same as above, but treat and monitor AMS early

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Management and Prevention of Altitude Illness

Clinical Condition	Signs and Symptoms	Management	Prevention
High-altitude cerebral edema	AMS for ≥24 h, ataxia, severe lassitude, mental confusion	Initiate immediate descent or evacuation; if not possible, use a portable hyperbaric chamber; give supplemental oxygen (2–4 L/min); give dexamethasone (8 mg orally, intramuscularly, or intravenously initially, and then 4 mg every 6 h); administer acetazolamide if descent is delayed	Avoid direct transport ≥2,758 m (9,000 ft); slower ascents; avoid overexertion; consider taking acetazolamide (125–250 mg twice daily) beginning 1 day before ascent and continuing for 2 days at high altitude; treat and monitor AMS early
High-altitude pulmonary edema	Dyspnea at rest and cough, severe weakness, drowsiness; later may see cyanosis, tachycardia, tachypnea, rales	Give supplemental oxygen (4–6 L/min until condition improves, and then ↓ to conserve supplies); descend as soon as possible, with minimal exertion, or use a portable hyperbaric chamber; if descent is not possible or no oxygen, give nifedipine (10 mg orally then 30 mg of extended release orally every 12–24 h); add dexamethasone if neurologic deterioration occurs. No evidence for furosemide or morphine	Slower ascents; avoid overexertion; consider using nifedipine (20–30 mg of extended release every 12 h) in persons with repeated episodes; long-acting beta-2 agonists

Modified from Hacket, P.H., and Roach, R. C.: High-altitude illness. *N Engl J Med* 2001; 345:107–114.

Conclusion

- Pathophysiology of heat and cold disorders
- Heat disorders
- Cold disorders
- Near-drowning and drowning
- Diving emergencies
- Altitude Illness