NORTHWEST COMMUNITY EMERGENCY MEDICAL SERVICES SYSTEM

EST 1972

May CE 2020

SOPs: Environmental Emergencies – Cold, Hot and Submersion

Objectives:

After completing the class & reading the referenced documents, each participant will do the following with a degree of accuracy that meets or exceeds standards established for their scope of practice without critical error:

***Cognitive*:** Explain the elements needed to accurately and consistently document a cardiac arrest in the NWC EMSS.

Interpret rationale of the SOPs presented in this micro-learning format so they are applied appropriately to patient situations and documented accurately.

Psychomotor: Accurately document a cardiac arrest using the new electronic power tool found in Image Trend.

Affective: Appreciate the need for accuracy and consistency in documentation with CA management through utilization of the system's new approach through electronic reporting found in Image Trend.

Questions and comments are welcome. and should be directed to:

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May 2020 CE – Learning Mini-modules

A. Video: New power tool for ePCR documentation for cardiac arrest (CA).
   1. Watch Dr. Jordan and R. Glendenning CA power tool video
   2. Complete 1 mock scenario of a CA with TOR
   3. Complete 1 mock scenario of a CA with ROSC and transport

B. Environmental Emergencies (Scenario based):
   1. Heat Emergencies
   2. Cold Emergencies
   3. Submersion Emergencies
   4. Allergic Reactions
   5. COVID + patient and clean up care

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Environmental Emergencies

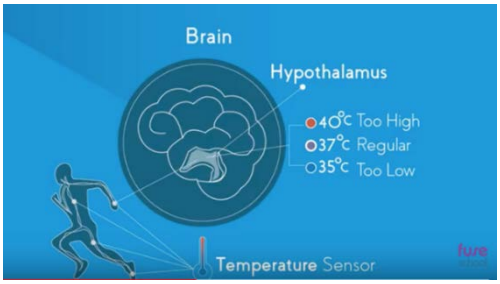
Heat and Cold Emergencies

A medical condition caused or exacerbated by environmental factors is known as an environmental emergency. The environment is defined as all of the surrounding factors that affect the development and functioning of a living organism. Examples: heat cramps, heat exhaustion, heat stroke, hypothermia, frostbite, drowning or near drowning, altitude illness, diving accidents, or barotraumas, and nuclear radiation exposure.

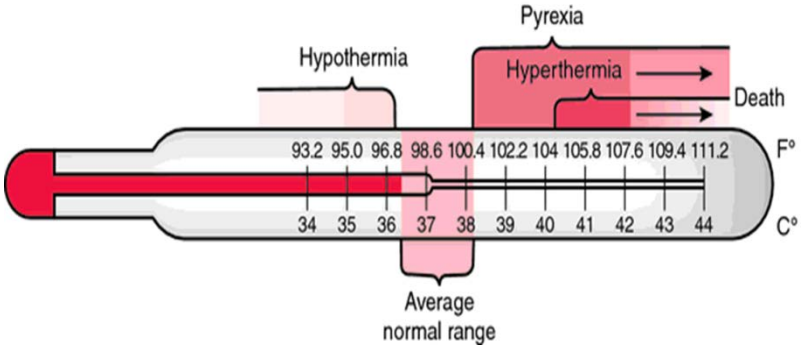
Who or what is responsible?

The primary area of control is found in the hypothalamus. The temperature in the deep tissues of the body is called the ***core temperature***. Normal core temperature in a healthy individual generally fluctuates slightly as a result of heat generated or gained and the heat lost, but usually remains stable at about 98.6° F (37° C) (*steady state metabolism*). It may vary 1 to 1.4° F in any one day. Higher temps usually occur in the evening.

Normal body temperature is regulated by the thermoregulatory center in the posterior hypothalamus, located at the top of the brain stem. The posterior hypothalamus receives information concerning body temperature from central



thermoreceptors in or near the anterior hypothalamus and deep receptors in the spinal cord, abdominal viscera and in and around the great veins. Peripheral thermoreceptors are located in the skin and some mucous membranes. Peripheral thermoreceptors are nerve endings that detect warm and cold, but



there are up to 10 times as many cold receptors as warm ones in many parts of the skin. Cold receptors are stimulated by a lower skin-surface temperature, and warm receptors are stimulated by higher temperatures (Sanders, 2001).

Central thermoreceptors are temperature-sensitive neurons that react directly to alterations in the temperature of the blood. They innervate skeletal muscle and affect vasomotor tone, sweating, and metabolic rate through sympathetic nerve output to skin arterioles, sweat glands and the adrenal medulla (Sanders, 2001).

Once feedback is received, the hypothalamus serves as the body's thermostat or radiator by establishing the thermal set point. Once the thermal set point is established, the body uses all available heat regulating mechanisms to maintain that temperature. If the environment is extremely hot or extremely cold, the body relies on its thermal compensatory mechanisms to keep the core temperature normal. The thermoregulatory system attempts to hold a core temp of approximately 98.6°F in face of continuous internally generated heat and widely variable temperatures.

If body temp rises, the hypothalamus responds by regulating heat loss (thermolysis) by ↑respirations, ↑ cardiac output, and ↑ sweating. If cold receptors are activated, the body sends appropriate output to increase heat production (thermogenesis).

Heat Production

The body can generate heat through mechanical, chemical, metabolic and endocrine activities. Several physiological and biochemical factors, such as the person's age, general health, and nutritional status, affect the direction and magnitude of these compensatory responses.

Internal Factors

Normal gains are controlled chemically through cellular **metabolism** (oxidation of energy sources at 75 Kcal/hr.) Diet-induced thermogenesis is caused by the processing of food and nutrients.

When a meal is eaten, digested, absorbed and metabolized, heat is produced.

All tissues participate in metabolism, but it is most pronounced in skeletal muscles, particularly when **shivering** occurs. The hypothalamus has an area called the primary motor center that controls shivering. It is excited by cold signals from the skin and spinal cord. *Shivering is the body's best defense against cold* and can increase heat production by as much as 400% (Sanders, 1080). *That is pretty cool! Or warm!*

Endocrine glands (*thermoregulatory thermogenesis*) regulate heat production through the release of hormones from the thyroid gland and adrenal medulla (epinephrine and norepinephrine) which immediately increase cellular metabolism.

Sympathetic excitation of heat preservation mechanisms. Norepinephrine and epinephrine are released following sympathetic stimulation (fight or flight mechanism) which causes an immediate increase in the rate of cellular metabolism and generates heat. The sympathetic response will also cause vasoconstriction to conserve as much heat as possible.

Physical activity (*work-induced thermogenesis*): Hard work or exercise can increase metabolic rates and heat production 12 fold to an ↑ temp of 102° F. It can also cause temp increases of 9° F/hour but only results in 2° increases if normal heat dissipating mechanisms are working.

External Factors

The body receives heat via the thermal gradient between the body and the environment. Several factors influence the thermal gradient.

<u>Ambient air temperature (heat waves)</u>: The body absorbs heat from a warm environment by radiation, convection, and conduction. As an example, the mortality rate is increased 3-5x the normal in a nursing home without air conditioning. Heat gradually overtaxes an elderly person's limited accommodative processes. Beware of heat absorption from the soles of the feet, i.e. (hot sand).

<u>Infrared radiation (sun: 150 Kcal/hr.)</u>

<u>Humidity</u>: reduces body's ability to evaporate heat, i.e., sauna baths, hot tubs.

Normal heat loss (thermolysis)

Heat is lost from the body to the external environment through the skin, lungs, and excretions. The skin is the most important factor in regulating heat loss.

Heat loss mechanisms: The first 3 need air temps cooler than 92° F. If higher, we gain heat. Ambient air temps must be less than skin surface temp for these to work.

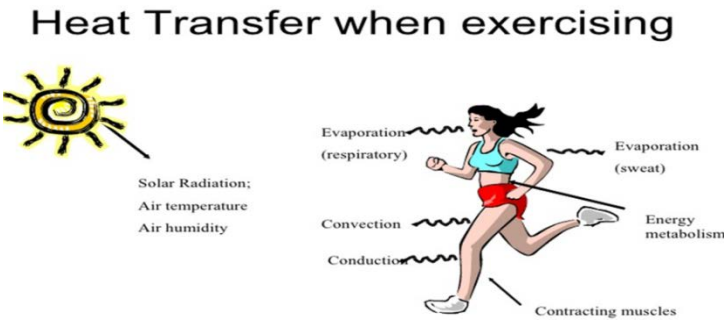
Radiation: Body heat is lost to nearby objects or environment even without touching them. The surface of the human body constantly emits heat in the form of infrared rays. The temperature of the radiating surface determines the rate of emissions.

If the surface of the body is warmer than the average of the various surfaces in the environment, net heat is lost because the rate depends directly on the temperature difference between the environment and the body (thermal gradient). An unclothed person will lose about 60% of total body heat by radiation at room temperature. All objects not at absolute zero temp will radiate heat.

Conduction: Direct transfer of heat (thermal energy) from higher temperature matter to lower temperature matter.

Heat moves down a concentration gradient from higher to lower temperature. Therefore, the body gains or loses heat by direct contact with cooler or warmer surfaces, including air. If ambient air temperature is lower than skin temperature, body heat is lost to the surrounding air by conduction.

Convection: Movement of air or water across the skin removes heated air near the skin surface. Convection can be greatly facilitated by external forces such as wind or fans and aids conductive heat loss by continuously maintaining a supply of cool air. Factors that contribute to convection: velocity of air currents and the temperature of the air are examples. A wind chill chart calculates the cooling effects of the ambient temperature based on thermometer readings and wind speed.



Evaporation: Evaporation is the change of a liquid to a vapor. Evaporation of any fluid absorbs heat from surrounding objects and the air. Ambient temperature and relative humidity greatly affect evaporative heat loss from moisture on the skin or lining membranes of the respiratory tract. Water evaporates via breathing and sweating at 600 mL/day.

Heat illness epidemiology and general causes

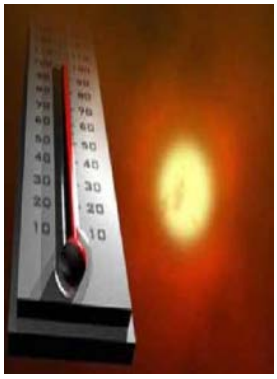
Heat illness defined: Heat illness encompasses the spectrum of pathologic and physiologic responses reflecting the body's attempt to maintain normal body temperature after a **large environmental or internal heat load** (thermal challenge) and/or **inadequate heat dissipation**.

Two situations may exist

1. Normal thermoregulatory mechanisms are overwhelmed by environmental conditions (heat stress, extreme exercise), or
2. Thermoregulatory (heat dissipating) mechanisms are inadequate/ defective.

Resetting the thermal set point won't correct the problem. The body must increase the net heat loss.

Hyperpyrexia vs. Hyperthermia



Temperature elevation accompanying an infection is classified as a ***fever*** or pyrexia. ***Hyperpyrexia*** is elevation of the body temperature above 106° F and is usually seen in the hospital.
Hyperthermia is a state of a higher than normal body core temperature. A rare form is *malignant hyperthermia* in susceptible patients after anesthesia (succinylcholine) administration.

Fevers are generally caused by ***pyrogens*** released by bacteria or viruses attacking the hypothalamus that elevate the set point in an effort to "cook the bugs". The patient usually has a history of being ill or having an infection. The fever will break when pyrogen production stops or when the pathogen attack is blocked. Treatment is directed at the underlying disease state. Example: ASA blocks pyrogen's action at the hypothalamus receptor site - most likely by blocking prostaglandin activity.

Heat illnesses cause an increase in body temperature without viral or bacterial disease, and result in **hyperthermia**. While heat stroke usually has a history of exertion or heat exposure, that is not always the case. Sometimes it is caused by a malfunctioning hypothalamus. If unsure if a very ill patient is suffering from hyperpyrexia or heat stroke, treat for heat stroke.

The NWC EMSS does not generally attempt to reduce low grade fevers. Up to 102° F, they are the body's way of attacking the illness and demonstrate an appropriate immune system response. We will attempt cooling in the presence of heat stroke and febrile seizures.

Predisposing factors for thermoregulatory emergencies

High-risk status is a function of age, overall health or activity level and environmental factors. Additional contributing factors:

- Dehydration
- Increased endogenous heat load
- Skin/sweat gland abnormalities
- Lack of acclimatization

Elderly: Unique susceptibility due to their inability to compensate; 40-80% mortality.

- Less able to sense and respond to temperature changes
- CV system unable to increase cardiac output
- Structural and functional changes occur in skin
- Decreased efficiency of sweating
- Decreased ability to acclimate
- Increased use of medications which generally interfere with heat dissipation

Infants and young children

- They are generally active and energetic, producing more metabolic heat.
- Limited reserves and inability to protect themselves.
- Difficulty in regulating body temps due to scanty layer of insulating fat & high ratio of skin surface to body mass (weight).
- Abundance of extracellular fluid that is rapidly turned over.
- Infants do not sweat; children have fewer sweat glands than adults.
- Frequently overdressed and may be left in hot, unventilated cars.

Chronic Diseases

- Cardiovascular diseases: atherosclerosis, HF, hypertension
- Circulatory insufficiency: dehydration, obesity, old age, CNS lesions, **diabetes**. The *autonomic neuropathy* associated with diabetes predisposes them to hyperthermia as it can interfere with vasodilation, perspiration and thermoregulatory input (Sanders, 1083).

- **Increased heat load**: Fever, seizures, exertion, hyperthyroidism, agitated psychiatric conditions. Status epilepticus: will generate huge amounts of heat.
- Parkinsonism
- Tumor of the adrenal gland (pheochromocytoma)
- Malnutrition
- Alcoholism, Alzheimer's disease and other mental status altering conditions lead to a failure to take sensible hot-weather precautions and place these patients at risk.
- Ill or debilitated

Drugs: Can impair accommodation or generate heat themselves

- Those that **decrease sweating capacity** and interfere with central thermoregulation: anticholinergics (Librax, Donnatol), antihistamines (Benadryl), tricyclic antidepressants, benzotropine (Cogentin); phenothiazines (Thorazine)
- Those that **increase heat production**: amphetamines, LSD, Cocaine, Phencyclidine; MAO inhibitors (↑ muscle activity)
- Those that cause **dehydration and electrolyte imbalances**: Lithium, Haldol, Navane, Prolixin; diuretics, alcohol
- **Beta blockers** (BB) (Corgard, Inderal, Lopressor, Tenormin), sympatholytic antihypertensives: Predispose a patient to dehydration, interfere with vasodilation, and reduce the capacity to increase heart rate in response to a volume loss (Sanders, 1083).

Amateur athletes (exertion) - largest number of cases in US.

- Marathon runners, high school football players. Runners need to drink 100-200 mL every 2-3 kilometers to prevent dehydration. Athletes may need to drink more than a gallon of liquid/day. Adequate intake is 1½ times that amount that quenches thirst.
- Second only to head and spine injuries as cause of death in young athletes in U.S. (800 die annually). 1000 cases/year of heat exhaustion.
- Body build may contribute: Stocky athletes with thick muscle mass (classic shot putter) generate more metabolic heat than those with thinner physiques, such as runners.

Farmers and outdoor workers (exertion)

Skin and sweat glands: impair heat dissipation

- Extensive prior burns; destroy sweat glands; ***grafts don't sweat***
- Prior heat stroke; necrotic sweat glands
- Cystic fibrosis
- Previous extensive scars
- Scleroderma
- Miliaria; prickly heat - damages sweat glands
- Spinal cord injury with poikilothermia: ↓ ANS tone = dysfunctional sweat glands; capillaries no longer dilate and constrict to regulate body temp by radiation.

Miscellaneous factors

Fatigue, lack of sleep, and unaccustomed physical tasks can play a role in heat exacerbations.

Heat Cramps

Pathophysiology: Acute painful spasms of the voluntary muscles fatigued following strenuous exercise in a hot environment without adequate fluid intake. The patient sweats profusely to dissipate heat. Sodium is transported to the skin and is lost with the sweat. This produces a rapid change in extracellular osmotic tension due to **sodium and water losses** with a build up of lactic acid in the tissues. (Losses may be up to 3-4 L/hr with intense physical activity. One liter of sweat has 20-50 mEq. of NaCl. The patient can experience a 50% net decrease in Na due to profuse sweating.)

Hyponatremia can interfere with skeletal muscle relaxation thus producing the cramps. Patients often replace sweat losses with water or non-electrolyte drinks that provide suboptimal salt intake and further dilute the serum sodium.

Signs and symptoms

- Painful spasms of the skeletal muscles: thigh, calf, foot, arms
- Cramps of abdominal muscles usually follow rapid ingestion of large volumes of cold water or other liquid following intensive exercise
- Temperature is generally normal
- HR: Tachycardia
- BP: Normal
- Respirations normal to slightly increased
- Skin: Warm w/ diaphoresis
- Mental status intact; alert and oriented
- Occasional nausea and gastritis

Treatment

- Rest; remove from hot environment

- Restore sodium and water. Slow rehydration with mildly cool but not cold Electrolyte-containing drinks are effective. Sugary beverages that decrease gastric emptying and dehydrating alcoholic beverages *should be avoided. IV fluids are usually not necessary.*
- Pain control; stretching the affected muscle groups useful but active massage is counterproductive; narcotics unnecessary.

Heat Tetany

Variant of heat cramps; occurs during exertion and develops secondary to the hyperventilation that universally occurs with a heat challenge producing hypocalcemia. It usually results in severe carpal-pedal spasms due to the rapid pH changes. Less common than heat cramps. Self-limiting by removal from the hot environment and reversal of the respiratory alkalosis.

Heat Exhaustion

Pathophysiology: More severe form of heat illness and reflects cardiovascular strain from an inability of a warmed person's circulatory system to handle heat demands. The body attempts to dissipate heat through **systemic vasodilation,** causing pooling of blood in the periphery which decreases circulation to the brain and other vital organs, producing signs and symptoms of shock. Diaphoresis results in **volume depletion and electrolyte losses**. Heat dissipation succeeds, but the body is unable to control the physiologic stress. May occur acutely or over several days or longer due to the cardiovascular system trying to maintain normothermia. If heat exhaustion is severe, it can lead to heat stroke. Treat aggressively.

Signs and Symptoms

Prodrome: Fatigue, weakness, thirst, muscle cramps, lightheadedness, (orthostatic dizziness may lead to syncope), irritability, headache, feeling of impending doom.

VS

- Increased heart rate to provide additional blood flow through the skin and muscles to enhance heat radiation; pulse is often weak and thready
- ↑Respiratory rate to dissipate heat through exhaled air.
- Decreased BP (due to vasodilation and/or dehydration) or orthostatic hypotension.
- Temperature may be below normal but may be as high as 101°-102° F (38.3-38.8° C) if mixed heat illness is present.

Skin: Diaphoretic to enhance evaporative heat loss; pale or flushed; can be dry if dehydrated

GI: Anorexia, nausea, vomiting suggesting flu-like illness

Neuro: *Irritability, anxiety and impaired judgment or mildly confused although **mental function remains intact.***

Important distinction.

Treatment

Move to a cool environment; remove as much clothing as possible to facilitate cooling if temp is increased.

Initial Medical Care; special considerations: position patient supine or on side if vomiting. The majority of hypotension relates to vascular dilation, not absolute fluid loss.

Rehydrate with IV NS. Four liters over 6-8 hours may be needed for young people throughout the entire treatment phase. Provide IVF challenges in the field. Calculate 20 mL/kg in peds. *Use caution to prevent fluid overload in the elderly or debilitated.*

Vomiting precautions; ondansetron prn; ready suction

Continuously monitor **ECG**

Monitor and record mental status. Can progress to heat stroke if left untreated.

Heat Stroke (HS)

Definition: Heat stroke is a state of **thermoregulatory failure** with hyperthermia after exposure to high environmental temperatures. If body's hypothalamic regulation is lost, uncompensated hyperthermia develops & the temp rises above **103°-105° F**. Heat loss mechanisms are overwhelmed or are deficient to meet environmental demands (usually after 3 or more days of heat wave). Rapidly increasing body temperature causes neurologic dysfunction and organ damage throughout the body. **TRUE MEDICAL EMERGENCY**.

Pathophysiology

- Gradual depletion and failure of body reserves in the face of **heat stress**.
- **Fluid & electrolyte depletion** due to copious uncompensated sweating = dehydration, hypovolemia & vasodilation.
- Anaerobic metabolism - cannot produce ATP (energy) - cells begin to die.
- This leads to **significant cardiac stress** with peripheral vascular shutdown, **sweat gland collapse**, and frank cardiac decompensation.
- Simultaneous **redistribution of volume** from the core to the vasodilated peripheral circulation **compounds hypotension**.
- As cooling occurs, significant fluid shifts back into the core circulation and blood pressure improves. Thus **overly aggressive fluid resuscitation is contraindicated to prevent overhydration and pulmonary edema**.

Signs and Symptoms

High temperature: >103° F (107°-108° not unusual). Rises rapidly when sweating ceases.

CNS disturbances diagnostic: Earliest to appear and last the longest. Increased temperatures markedly increase the metabolic demands of the brain. At 107° F proteins coagulate and neurons die rapidly. S&S are caused by cerebral edema, thermal damage, and uric acid neuropathy due to renal failure. Can look like a patient with a viral infection moving into meningeal encephalitis.

- Confusion, disorientation, irrational behavior, sudden loss of consciousness. (May appear psychotic)
- Lethargic to combative
- Fixed, dilated pupils
- Tremors, seizures, or abnormal flexor or extensor posturing
- Healthy people can get ill v. quickly. They become confused, aggressive, and euphoric - then collapse.

Respiratory: Hyperventilation universal to dissipate heat and compensate for metabolic lactic acidosis. May go into acute pulmonary edema from acute cardiac failure and local DIC from direct injury to tissues. Listen to lung sounds.

Cardiovascular - Early: tachycardia, hypertension from hyperdynamic state. Cardiac output can initially be 4 to 5 times greater than normal (CO = 20 L/min. or more). As temps continue to rise, stroke volume decreases and patients can demonstrate an elevated central venous pressure.

- High temp increases myocardial oxygen consumption, and, in the presence of hypertension or a volume overloaded myocardium caused by heart failure, arteriosclerotic vascular disease or CAD, may lead to a malignant dysrhythmia or AMI.
- Pump failure, pulmonary edema, and major dysrhythmias follow the hyperdynamic state resulting in hypotension (High output cardiac failure). Hypotension indicates a poor prognosis.
- Hypotension may be due to the following:

  Loss of Na and water

  Peripheral vasodilation with pooling (enhanced by lactic acidosis)

  Myocardial dysfunction causing dysrhythmias, infarction and failure due to local hemorrhage, cellular degeneration and spotty inflammation. May need a norepinephrine drip.

  Localized disseminated intravascular coagulation (DIC)

  Sweating may be present or absent depending on type of HS and whether or not the patient is dehydrated. Skin is hot, red and flushed due to vasodilation. About 25% of EHS patient have persistent sweating resulting from increased catecholamine release.

  In classic HS, sweating is usually absent due to dehydration, drugs that impair sweating, direct thermal injury to sweat glands, or sweat gland fatigue. The cessation of sweating is not the cause of heat stroke.

Liver involvement: ↓ production of clotting factors, hepatocellular necrosis can be severe. Assess for GI bleeding, ileus, liver failure, clotting disorders and electrolyte imbalances.

Coagulation disturbances: DIC, ↑ capillary permeability and platelet disruption due to heat. Look for petechiae or purpura, bleeding into the GI tract, lungs, brain or other organs.

Fluid & electrolyte (↓ K) **imbalances** play a crucial role. The patient may suddenly collapse with no advance warning.

Kidney involvement: Commonly affected due to hypovolemia and hypoperfusion. Ten percent develop acute tubular necrosis (ATN) or myoglobin tubular block. Rhabdomyolysis (port-wine colored urine caused by muscle breakdown) results from direct injury or ischemia. Manifested by muscle pain, myoglobinuria (EHS). Hospital dipstick for hemoglobin is heme positive signaling an ↑ risk for kidney damage.

Treatment

High mortality rate if untreated. Need rapid assessment with aggressive interventions.

Move patient to a cool environment, remove all clothing and check for injuries. Obtain full baseline vital signs including temperature reading.

Rapid cooling

Initiate rapid cooling, but ***take caution to avoid shivering*** (creating more internal heat.) Apply cold packs to cheeks, palms, soles of feet. Avoiding large muscles will limit shivering risk. If additional cold packs available, apply to neck, lateral chest, groin, axillae, temples, and/or behind knees. Sponge or mist skin with tepid (warm) water and fan to promote evaporation.

Cool or cold water may induce shivering which increases temperature. The thermal conductivity of water is 32 times faster than that of air. The amount of heat required to evaporate one gram of water is seven times that to melt one gram of ice. The rate of cooling by air rapidly increases with increasing wind velocity (the wind chill factor).

Take and record the temperature at least every 5 minutes during the cooling process to ensure adequate rates of cooling and to avoid rebound hypothermia. Rebound hypothermia can best be avoided by stopping the cooling process when the core temp reaches about 102° F (39° C). The body will continue to cool on its own.

Gently massage extremities to counter peripheral vasoconstriction, accelerate heat loss, and promote venous return.

If in pulmonary edema with ventilatory failure, will need to provide ventilatory assist

If hypoxic: 15 L O_2 (C-PAP may be very useful if awake with good ventilatory effort)

Monitor ECG: anticipate ST-T segment changes and dysrhythmias

Monitor VS and breath sounds carefully. With pulmonary edema, BP will elevate rapidly - develops hours after collapse.

Careful monitoring of fluid volume status. If hypovolemia is present, give a fluid challenge of 200 mL to maintain SBP ≥ 90 (MAP ≥ 65) and inform hospital of the total amount of IVF infused. They may need 1-1.5 L in first 4 hours. In most patients, the BP rises to a normal level during the cooling process as large volumes of blood shift back from the cutaneous vessels into the central circulation.

Fluid overload is a definite hazard for those with underlying disease, high output failure, vasomotor paralysis, and renal failure and reduces the margin for error/ safety. Carefully monitor and record intake and output. Fluid replacement can cause pulmonary edema and heart failure especially in older adults.

Anticipate seizures and prepare midazolam.

Anticipate hypoglycemia: obtain capillary blood glucose reading and give dextrose 10% to achieve normal glucose readings

Observe for development of cerebral edema and ↑ ICP

ASA ineffective and contraindicated; may contribute to coagulopathies and hepatic damage

Immediate complications

- ✓ Violent shivering from cooling: prevent shivering so more heat is not generated.
- ✓ Seizures; Rx with midazolam per SOP
- ✓ Hypotension in absence of dehydration due to shunt through dilated skin vessels. Monitor fluid status and VS carefully as fluid overload can occur when vasoconstriction returns large volumes to the central circulation resulting in pulmonary edema.
- ✓ Electrolyte imbalances (hyponatremia, hypokalemia)
- ✓ Acute renal failure; rhabdomyolysis
- ✓ Liver failure; clotting abnormalities; DIC
- ✓ Acute lung injury (ALI)

References

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Cold Injury

HYPOTHERMIA is defined as low body temperature usually resulting from deficient thermo-regulation with a core temp < 95° F (35° C); heat losses exceed heat generating capacity that can happen either by accidental means such as inadvertent over exposure to low temperatures or by intentional means such as therapeutic hypothermia after ROSC.

When heat exchange becomes vastly unbalanced and excessive heat loss cannot be countered by increased heat production, hypothermia occurs. Hypothermia isn't necessarily associated with extreme weather conditions.

Primary vs. Secondary

Primary: Heat production in an otherwise healthy person is overcome by the stress of excessive cold especially when body energy stores are depleted.

Secondary: Can occur in ill persons with a wide variety of medical conditions, even in a warm environment due to impaired regulation of internal body heat (sepsis). Death in these patients is often caused by the underlying condition rather than by hypothermia.

Those at risk

Elderly and Children: same as heat

Trauma

*Hypothermia occurs in **29%** of all trauma pts and has potentially lethal complications.* Multiple studies have demonstrated increased morbidity and mortality when hypothermia is present. **Scene exposure is a significant factor, especially if extrication is prolonged.**

Open cavities, large abrasions, large head wounds contribute to hypothermia as does the use of unwarmed irrigation/lavage fluids. *MOST* hypothermia in trauma patients occurs during the resuscitation phase when they are inside but physically exposed and receiving large volumes of "room temperature" fluids intravenously. The incidence of hypothermia can be high on EMS arrival at the scene. Body temperature measurement and immediate thermal protection should be routine, and special attention should be given to patients who are wet. (https://doi.org/10.1186/s13049-017-0349-1). Neuromuscular blockade, anesthesia, and infusion of crystalloids or blood products at rates > 200 mL/hr is sufficient to lower core temperature to 30° C (86° F).

Effects on metabolism

1. A decrease in core temp reduces metabolic rate and decreases oxygen demand. All metabolic processes slow by 7% for every 1° C temperature drop. This produces a decreased RR & CO.
2. At 82.4° F (28 ° C) the metabolic rate falls to 50% normal.
3. ATP synthesis is reduced - leads to cellular death (this is particularly troublesome in trauma patients where concomitant shock also reduces ATP) production.
4. Blood flow through liver decreases; all meds dependent on the liver for detoxification will have a prolonged effect.
5. Lactic acid builds up in tissues – big problem during rewarming.

Effects on oxygenation

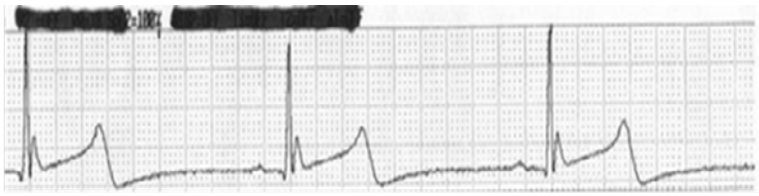
Decreased temperature shifts oxyhemoglobin dissociation curve to the left (O_2 is bound more tightly and not released to tissues). Each 1° C drop causes a 6% increase in affinity (binding).

SpO_2 may be inaccurate secondary to peripheral vasoconstriction.

Shivering will ↑ O_2 consumption by as much as 500%! Good rule of thumb: provide high flow O_2 to shivering patients, regardless of SpO_2.

Effects on cardiovascular system

1. MAP and cardiac output decrease secondary to myocardial depression/ arrhythmias. Cellular O_2 consumption increases and will cause myocardial depression at 34° C.
2. The heart becomes increasingly susceptible to arrhythmias as the temperature falls below about 86° F (30° C). ECG changes most often seen in hypothermia:
    - Sinus bradycardia: decreases depolarization of cardiac PM cells causes bradycardia. It is not vagally mediated, so will not respond to atropine.



- 30% of patients at 30° C will develop an **Osborn / J wave** that is a short, broad positive wave appended to the S wave of the QRS in leads II and V6. J waves can be a nl variant & may be seen with sepsis & myocardial ischemia (Li & Decker, 2005).
- As hypothermia worsens, there is slowed conduction with a prolonged PR interval, then a widened QRS, then most characteristically a prolonged QTc interval, inconsistent inverted T-waves and ST-segment abnormalities, atrial fibrillation or flutter, AV block and/or nodal rhythm or PVCs, ventricular fibrillation, asystole.

Mild, Moderate and Severe Hypothermia

Mild: Core temp 32-35°C (90.6 - 95° F); physiologic effects typically mild; may be influenced by comorbid conditions such as traumatic injury, intoxication, or diabetes, the body maintains ability to compensate and produce heat via shivering and vasoconstriction

Signs and Symptoms

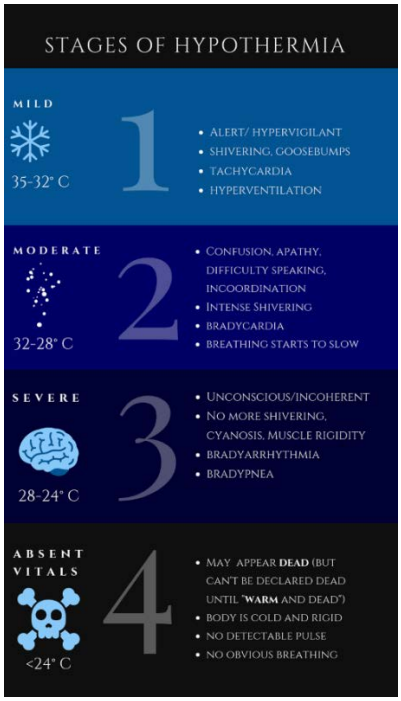
Conscious, alert to confusion, sensation of cold, hyperventilating shivering; impaired motor ability with noted ↑ HR; ↑ RR; ↑ CO

Moderate: Core temp 82.4-90.6° F (28-32° C); shivering ceases <31°C (87.8°F); heat production falls, replaced by muscle rigidity, slowed reflexes; metabolic processes slow; ECG changes begin.

Signs and Symptoms

Alerted mental status, O_2 consumption decreases and CNS depression worsens; slowed decision making; may be unaware of their deteriorating condition and may deny that they have a problem; slowed/slurred speech and ataxia may mimic a stroke. Slow RR, CO2 retention; bradycardia, ↓ CO

Severe: Core temp <28°C (82.4° F); Muscle rigidity; at risk for cardiac dysrhythmias: marked bradycardia, absent pulse, hypotension, VF (may present in cardiac arrest); slowed RR to apnea, pupils fixed & dilated.



Allergic Reactions and Anaphylaxis

The immune system is the principal body system involved in allergic reactions. Others include the cutaneous, cardiovascular, respiratory, nervous, and gastrointestinal systems. It defends against foreign substances, including antigens (foreign proteins) and combats infection.

An immune response is a series of events that occur following activation by an invading antigen (foreign protein) or pathogen (disease-producing agent or invading substance). The goal is to destroy or inactivate the pathogens, abnormal cells, or foreign molecules, such as toxins (poisonous chemical secreted by bacteria or released following destruction of the bacteria).

If a person has not encountered a particular antigen, the body will go through a sensitization phase which is different than if it has been previously exposed. The initial exposure to an antigen is called **sensitization** or the primary response. Following first time exposures to an antigen, several days are required before both the cellular and humoral components of the immune system respond.

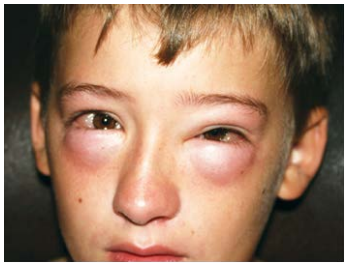
The secondary response occurs when the body is exposed to that antigen again, it will respond much more quickly with a much stronger secondary response.

Types of immunity

1. **Natural** (innate) immunity is genetically predetermined. It is present at birth and has no relation to previous exposures to a particular antigen. Everyone is born with some innate immunity.
2. **Acquired**: Develops over time and results from exposure to antigens and production of specific antibodies. See process explained above. Subsequent exposures to that antigen will have an immune response.
   A. **Naturally acquired:** Begins to develop after birth and is continually enhanced by exposure to new pathogens and antigens throughout life. Example: life-long immunity to chicken pox after having the disease.
   B. **Induced active immunity** (***Artificially acquired*** *immunity)*: Designed to provide protection from exposure to an antigen at some time in the future. Achieved through **vaccination** where an antigen is injected into the body to generate an immune response. This results in the creation of antibodies specific for that antigen and provides protection against future infection. Ex: tetanus, diphtheria, pertussis, Hepatitis B, measles, polio vaccines. Re-emerging: smallpox vaccine.
   C. **Active:** Occurs following exposure to an antigen and results in the production of antibodies specific for the antigen. It takes some time for these antibodies to develop and provide protection. (see above)
   D. **Passive:** Administration of the antibodies to provide protection until active immunity can take place.

Epidemiology

Type 1 or immediate hypersensitivity is the most familiar; allergic or atopic reaction (predisposition to allergic responses) caused by contact with an antigen (allergen) against which the host has a large number of existing IgE antibodies. These patients have elevated IgE levels (10-100 times normal). 20%-30% of population has a type I hypersensitivity to common environmental substances. There is a rapid, immediate reaction as well as a late phase component with the initiation time: 2-30 minutes.



Histamine: Most important chemical released in an allergic reaction.

3 histamine receptors

H_1: Responsible for coronary artery vasoconstriction, **bronchoconstriction**, **vasodilation** (flushing**)**, **increased vascular**

permeability resulting in fluid shifts (hypotension) **and tissue edema**, and increased secretion of airway and nasal mucus, intestinal smooth muscle contraction (diarrhea, abdominal pain and cramping) and prostaglandin production. It decreases AV conduction time, so can lead to dysrhythmias. It is also responsible for the pruritus (**itching**), pain and burning seen in many hypersensitivity reactions as well as airway Vagal nerve stimulation. Positively, stimulation of H_1 receptors helps to modulate the reaction. Further histamine release from H1 receptors is **blocked by diphenhydramine**.

H_2: Stimulates ventricular and atrial contraction (+ inotropic effect), increases HR (+ chronotropic effect), coronary and peripheral vasodilation, **gastric acid secretion**, lower airway mucus production, inhibits basophil and neutrophil activity, suppresses T-cell stimulation, and ↑ cyclic AMP in the cells. Causes nausea, vomiting. (Reversed w/ H_2 blockers)

H_3: Inhibits central and peripheral NS neurotransmitter release and may inhibit histamine formation.

Histamine takes only 2½ minutes to appear in the blood, reaches peak levels in 5 minutes, and **returns to baseline in 15-30 minutes**.

The goal of histamine release is to limit the body's exposure to the allergen. Bronchoconstriction decreases the amount that can be inhaled through the respiratory tract. Increased gastric acid helps destroy an ingested antigen. Increased intestinal motility moves it through the GI tract faster minimizing absorption. Vasodilation and capillary permeability help to remove allergens from the circulation where they have the most potential for doing harm.

Among other chemicals that are released, would be **bradykinin** that causes ↑ vascular permeability, vasodilation, cough and mucosal edema. As well, **leukotrienes** that cause mucosal edema, mucus secretion, airway inflammation, bronchospasm (coughing, sneezing, wheezing) not inhibited by antihistamines, vasodilation, vascular permeability, and urticaria (hives).

Primary assessment

1. General level of consciousness
2. Airway; ability to speak: if impaired go immediately to Rx
3. Breathing; gas exchange: if impaired go immediately to Rx
   - General RR (tachypnea), effort (labored, use of accessory muscles), prolonged expiration
   - Skin color temperature, moisture – obvious evidence of hives, swelling
   - SpO_2: Use to follow general trends. Patient may be or become hypoxic.
   - $ETCO_2$: As anaphylaxis progresses CO_2 levels rise due to respiratory and metabolic acidosis and poor ventilation.

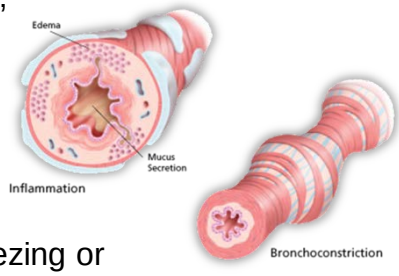
Signs of airway/ventilatory impairment

Upper airway:

Angioneurotic edema, also known as **angioedema,** involves the deep dermis, sub-q or submucosal tissues and can be either hereditary or acquired, well-demarcated, non-pitting, non-pruritic edema commonly found in the periorbital, oral, lingual (tongue), and pharyngeal areas, although the hands, feet, penis and scrotum may also be involved. It does not itch and may persist for days.

Lower airways:

Dyspnea; sensation of tightness in chest and throat; coughing; retractions; **wheezing** due to intense bronchial swelling and spasm. May be evident without a stethoscope. No wheezing or diminished breath sounds may mean no air movement! Bronchospasm and laryngeal edema may induce swift respiratory arrest; tachypnea.



Signs of cardiovascular decompensation

Circulatory status; ECG: if impaired go immediately to Rx

Assess: General HR (fast or slow), quality, rhythmicity; ECG, chest pain, tachycardia, dysrhythmias: ST, AF, AV and IV conduction delays, transient left bundle branch block (LBBB), ↓ BP due to massive peripheral vasodilation and 3rd space losses due to ↑ permeability of capillaries **resulting in marked loss of plasma from the circulation**. This **leads to cardiovascular collapse, hypotension** (**SBP ≤ 90; MAP <65**), cardiac dysrhythmias, shock & coma.

Secondary assessment

Full set of vital signs

- BP: Important to determine stability. Will fall w/ significant vasodilation & capillary leak.
- P: rate, rhythmicity, quality, location (anticipate reflex tachycardia in most patients). HR will fall late in anaphylaxis. *Ominous sign.*
- RR, pattern, depth, & effort. Will initially ↑, but severe obstruction can ↓ rate.

History important if time permits

OPQRST of chief complaint

Allergies: Hx of allergies to food, meds, plants insect stings environmental triggers, bites or others?

Medications

- *Epi-pen* prescribed?
- Have they taken anything or applied anything already to relieve their symptoms?
- Have they taken any NEW medications?
- On ace-inhibitors, BB, or allergy meds?

Past history

- Ever had an allergic reaction in the past? If so, when was their last reaction?
- What was their response to therapy? Risk of recurrent systemic reactions about 28%.
- How severe was last reaction? Ever needed to be intubated or admitted to ICU?
- Do they have any significant illnesses? History of asthma, CVD

Last oral intake: Food or drink. What did they consume? How much?

Event specifics

What was the patient doing prior to the onset of their symptoms?

What was the patient exposed to that could have caused a reaction? Any new soaps, foods, household products, etc.

What was the route of exposure if known - injection, ingestion, inhalation, or contact?

Physical exam

Neurological - level of consciousness

- Anxiety, apprehension, restlessness
- AMS; confusion, disorientation, decreased level of consciousness
- Dizziness, headache, hypotension or dysrhythmias may manifest by c/o lightheadedness or syncope
- Perioral tingling Seizures, syncope, coma

Cutaneous (skin - 90%); Warmth, redness (flushing), tingling

Pruritus (itching): Itching of lips, hands, soles of feet, or back of throat; an early sign

Urticaria: Histamine release will cause fluid to diffuse from leaky capillaries resulting in a wheal and flare reaction characterized by red, raised bumps that may appear and disappear across the body. Also called hives.

Edema, diaphoresis, cyanosis if hypoxic

Mucus membranes

Edema, burning, ↑ secretions (drooling), rhinorrhea, ocular itching and ↑ tearing

Gastrointestinal

- Bloating, abdominal pain, cramping
- Hyperactive bowel sounds
- Nausea, vomiting, diarrhea
- Difficulty swallowing
- Loss of bowel control

Renal: Urgency, incontinence or ↓ urinary output

Submersion and Water-Related Emergencies

Epidemiology:. Drowning is considered 3rd most common accidental cause of death in children. About 90% of all submersion incidents occur w/in 10 yards of safety. Incidence ↑ dramatically during warm weather months due to water-related activities. The elderly drown as a result of submersion in bathtubs.

Factors contributing to drowning (risk factors)

- Inability to swim
- Diving incident causing c-spine/head injury
- Alcohol/drug ingestion or intoxication: Be alert for SUD in victim or supervising adult
- Hypothermia
- Exhaustion
- Seizures
- Suicide
- Hyperventilation prior to underwater swimming
- Battery/abuse: Have a high index of suspicion for child abuse or neglect
- Lack of parental supervision complicated by improper barriers that fail to limit access to pools, lakes, bathrooms, or buckets of water

Sequence of events

1. Something goes wrong - unexpected submersion: surprise/panic; dysrhythmic breathing or voluntary apnea for up to three minutes. May be complicated or precipitated by altered mentation (seizure/ trauma/ drugs/alcohol).

2. Victim attempts to keep the head elevated above the water with deep inhalation antecedent to submersion and the instinctive downward movement of the arms. During this time, blood is shunted to brain and heart (mammalian diving reflex).

3. Submersion w/ struggling. Victim holds breath while struggling to raise up. Apnea produces ↑CO_2/O_2 deprivation. Stimulant effects of the hypoxia ultimately overrides sedative effects of hypercarbia resulting in CNS stimulation: tachycardia, hypertension, hypoxia, hypercarbia, acidosis.

4. Until unconsciousness, victim experiences air hunger & panic. Breakpoint is reached resulting in involuntary inspiratory & swallowing efforts. Water enters mouth, pharynx and stomach, stimulating laryngospasm & bronchospasm. Laryngospasm often prevents water from entering lungs (*dry drowning*). If laryngospasm does not occur & water does enter lungs, it's referred to as *wet drowning*.

5. Arterial hypoxemia, tachycardia, tissue hypoxia, and tissue acidosis worsens. In the lungs, ↑ peripheral airway resistance, reflex pulmonary vessel constriction, ↓ lung compliance, ↓ surfactant and fluid shifts across alveolar membranes.

6. Agitation stops; Pt loses consciousness.

7. **Decompensation:** CNS stimulation with reflex gasping and inspiratory efforts results in further water aspiration and/or swallowed resulting in gastric distention & ↑risk of vomiting. Pt experiences 2° emesis & 2° apnea.
   - 85% - 90%: Laryngospasm aborted. Aspiration of water triggers vicious cycle of inflammatory events.
   - 10% - 15%: Laryngospasm recurs resulting in anoxia, seizures & death without aspiration.

8. Extinction of reflexes

9. Neuronal dysfunction: EEG becomes flat, blood/brain barrier breaks down

10. Cardiac dysfunction: bradycardia, dysrhythmias, asystole

11. Terminal phase
    A. Somatic death within 1-60 minutes after submersion (median 3-10 minutes), depending on age, water temp, and degree of tissue hypoxia
    B. Brain death

Pulmonary system: **Hypoxia**, bronchospasm and pulmonary vasospasm, hypercapnia, and acidemia. Fluid aspiration produces alveolar/arterial O_2 difference (V_A/Q mismatch).

Consider the tonicity of fluid, contaminants, & amount of fluid aspirated.

"In addition, extrapolations from early animal models led to the belief that large amts of water are aspirated into lungs; however, these models were based on controlled massive aspirations, and the true volume experienced during a typical drowning, while unknown, is likely much smaller" (Schmidt & Sempsrott, 2015).

"Only small amounts of water are needed to disrupt the surfactant that lines the cells in the lung responsible for exchanging oxygen and other gases. The problem in drowning, especially in cases of mild drowning that worsens, is surfactant disruption, not a measurable level of fluid in the lungs that fills up like a cup and prevents breathing. After a mild or moderate drowning, inflammation and infections in the lungs can cause the initial symptoms to get worse. Parents should seek additional care whenever a child has an excessive cough, isn't breathing normally, or isn't acting right immediately after being pulled from the water." (Hawkins, Sempsrott & Schmidt, 2017).

Cardiovascular system: CV complications due to myocardial ischemia resulting from hypoxia, acidosis, hypothermia, & electrolyte disturbance. Clinical findings: ↑ HR, ventricular dysrhythmias: VF, ↓ HR, or asystole, varying degrees of heart block, CP, ↓ BP related to ↓ CO due to ↓ contractility, fever, unless hypothermic, cyanosis or pallor.

Central nervous system

Five minutes of cerebral anoxia is sufficient to produce irreversible brain damage in warm water. Patient may present with AMS including irritability, restlessness, confusion and lethargy. Seizures, coma, or hyporeflexia may develop.

Hypoxia & hypercarbia coupled with ventricular dysrhythmias and/or asystole promotes arterial ↓BP = may cause an acute drop in cerebral perfusion pressure (CPP).

> A sudden drop in CPP leads to brain tissue hypoperfusion, cerebral anoxia, tissue acidosis and impaired neuronal metabolism.

Ensuing neuronal cell damage is associated with loss of cell membrane integrity and extracellular leakage of fluid, and diffuse cerebral edema.

Severe cerebral edema will produce ↑ ICP. Not common early; indicates a poor prognosis.

Hypercapnia (high CO_2 levels) and vascular endothelial (inner lining) damage resulting from hypoxia disrupt intracerebral vasomotor auto-regulatory mechanisms and uncontrolled intracranial hypertension may develop contributing to further cerebral ischemia.

Ultimate insult: brain herniation. Brain tissue is non-compressible. Based on Monroe-Kellie hypothesis, swollen brain tissue will encroach on vessels or subarachnoid space, further obstructing blood flow to tissues. Final insult = brain shift (sub-falx or transtentorial herniation).

Instructional Set

Now that you have reviewed the basic background material for environmental emergencies that include heat and cold, allergic reactions and submersion emergencies, the next portion of the class allows you to put that knowledge into practical application through scenarios.

While it is possible to elect to complete this packet in a group format, each question should have **independent** thought put into the written answers on the page.

Upon successful completion, the packet should be submitted to your provider coordinator. You will receive written confirmation from your HEMSC if the submission is accepted. If the packet is not satisfactory, it will be returned for completion or partial credit will be assigned.

Northwest Community EMS System	May 2020 CE: Environmental Emergencies
PM Name:	Date submitted:
EMS Agency/hospital:	Credit awarded (date):
EMSC/Educator reviewer:	Returned for revisions:
	Revisions received:

Scenarios

#1: You find a 70 y/o unconscious female in her apartment two days after a record-breaking heat wave began. She has no fans or air conditioning and the heat in her room is suffocating. Her PMH is unknown.

Physical exam:

HEENT: Unresponsive to verbal but responds purposefully to painful stimuli.
Lungs: Clear
CV: ECG: ST with PVCs
GI: Soft, no guarding, masses
Skin: Hot, flushed and dry
VS: BP: 84/50; P: 112; R: 32; SpO2 91% ; T: 106° F

What is the paramedic impression?	
What assessment findings indicate the condition noted above?	
What neurological complications should be anticipated?	
How should pts airway be secured?	
How should oxygen be delivered to this patient?	
What type of fluid therapy appears indicated?	
How should you attempt to cool this patient? *What should be avoided?*	
If the above neurological conditions occur, what measures should be taken to protect the pt?	

#2: A 16 y/o conscious male is found at football practice propped up by the fence. Bystanders state that he had been complaining of fatigue, lightheadedness, headache and nausea after the first 45 minutes. Outdoor temperature is 92°F. The patient has a history of diabetes and asthma.

Physical exam:

HEENT:	Mental status slightly agitated. Able to appropriately answer questions. PERRL
Lungs:	Breath sounds clear bilaterally
CV:	ECG: SR; no ectopics; no murmurs
GI:	Soft, non-tender, no guarding or masses
Ext.	Unremarkable
Skin:	Pale, diaphoretic
VS:	BP: 98/60; P: 108 weak and thready; R: 24; T: 99° F; BG 70

What is your paramedic impression?	
What assessment findings indicate the condition noted above?	
What is the pathophysiology that led to his current condition?	
What interventions should be initiated per SOP?	
Identify other signs and symptoms that would support the impression listed above?	
What indicators would make this patient at high risk for developing a heat disorder?	
What is the known acuity level of this patient?	
What 4 ways does the body release heat and give an example of each	1. 2. 3. 4.

#3: A 48 y/o conscious male is found in a garage where he and a friend have been working to clean out the family's collection for donation. Outdoor temperature is 94 F. The patient is alert and oriented and is complaining of severe cramping pain in his abdomen and legs that began 30 minutes after they began working. He is also c/o slight nausea. He has no significant past medical history; takes no meds and denies any allergies.

Physical Exam:

HEENT:	WNL; PERRL
Lungs:	Clear bilaterally
CV:	ECG – ST
GI:	Abdomen tender with voluntary guarding; BS active
Ext:	No evidence of trauma; bilateral leg cramps. SMV intact
Skin:	Warm, flushed, diaphoretic
VS:	108/66; P: 90; R: 20; T: 99 °F

What is your paramedic impression?	
What is the pathophysiology which led to his presenting physical signs? What electrolyte has been lost?	
What level of acuity is this diagnosis?	
What treatment should you initiate per SOP? .	
Does this pt need IVF replacement?	
What other chronic conditions can elevate the thermoregulation of a person's body temperature?	

#4: Paramedics called to a local office building for pt c/o difficult breathing. 36/M is complaining of fullness in his throat and difficulty breathing, began ~20 minutes after taking an Aleve (given to him by a co-worker) for a headache. wt 210 lbs

Physical Exam:

HEENT: WNL; PERRL
Lungs: Expiratory wheezing
CV: ECG – ST
GI: Abdomen non-tender; BS active
Ext: No evidence of trauma; bilateral leg cramps. SMV intact
Skin: Warm, flushed, diaphoretic, urticaria noted on chest
VS: BP 124/72, P 120, ECG ST, R 24, O2 sat 95% RA
PMH-Meds: Pt. denies any PMH or known allergies

What is your initial impression/diagnosis?	
How would you rate the severity – mild, moderate, or severe? What sign/symptoms support that level of severity?	
What is the 1st, 2nd, and, 3rd med after O2, that EMS should give this pt? *Dose? Route? Site?* *Why is that site for the first drug recommended?* *What is the desired action for these medications?* *What are side effects of this medication?* *Can this medication be repeated? If so, when?*	
How does the action of the 1st differ from the 2nd and 3rd medication?	
What is the 4th med that should be given to this patient? Dose? Route? Action?	
If a person has not encountered a particular antigen, the body will go through a phase which is different than if it has been previously exposed. What is the name of that phase and why is that initial phase different? What occurs in the secondary response to an antigen?	

What chemical is released in the body to stimulate an allergic response and identify 2 responses for each receptor? How long does the chemical response take to appear in the blood, peak and return to baseline? What is the goal of the chemical release?	

5: Paramedics called to a picnic area for a woman with a bee sting.

CC/HPI: Friend stated pt was drinking a pop and a bee must have flown inside the can; she went unconscious right before medics arrived; very allergic to bees; did not have her EpiPen with her
PMH-Meds:
Lungs: audible stridor noted
CV: ECG – ST
Skin: Facial edema w/ skin warm, flushed, diaphoretic, urticaria noted on chest
VS: BP 78/50, P 142, ECG ST, R 30, shallow, lungs ↓ BS bilat, O2 sat 77% RA

No other PMH, meds or allergies known; wt ~150 lbs

What is your initial impression/diagnosis?	
Based on what criteria did you base your impression?	
How or what defines the “2+ Rule” as mentioned in SOP?	
What questions should be asked that may help to determine the severity of response for this current reaction?	
Based on this initial impression, what is the first course of action that should be taken for this pt?	
If cardiac arrest occurs in the above pt, how should the delivery of epinephrine change to accommodate this type of arrest?	

#6: A 23 year-old male started drinking after work on Friday. He left the bar at closing. At 7:00 A.M. you are called for a man down in a bar parking lot by the A.M. cleaning crew. The temperature dropped to 20° F during the night. On your arrival, the patient is unresponsive to verbal stimuli. The patient is stiff, and no shivering is noted.

Physical Exam:

Lungs: Minimal air movement

CV: ECG – bradycardia with a rate of 30 with curious waves at the junction of the QRS & ST segment

Skin: His skin is cold and pale with a temp of 90° F

VS: BP and P not able to be obtained, R is very slow at 4/min

PMH-Meds: Unknown

What is your initial impression of this patient's problem?	
How should you approach this patient's airway and ventilatory management?	
What findings are likely when ventilating this patient with a BVM?	
Should you hyperventilate this patient? Why?	
Since the patient has such a profound bradycardia, is atropine indicated at this time?	
If the patient goes into asystole what interventions are indicated?	

#7: An adult was rescued from a lake after being submerged for about 5 min after falling off an inflatable raft. After 2 min of CPR the pt has ROSC, wakes up, has good respiratory effort, and is refusing transport.

Physical Exam:

HEENT: WNL; PERRL

Lungs: chest wall movement symmetrical; BS congested

CV: ECG – sinus rhythm without ectopy

Skin: His skin is cool and wet; temp of 97° F

VS: BP 110/70; P 60; R 16; SpO2 92%

PMH-Meds: none

Should this pt refuse care and transport? Why or why not?	
What level of severity would you classify this pt in accordance with SOP?	
What treatment should be given to this pt after this incident? What purpose does this treatment serve?	
If this pt was still in need of resuscitation upon EMS arrival, what difference is noted for CA management?	
Does this pt require SMR? Why or why not? Any other evaluation needed based on MOI?	

#8: It is April 2020 and EMS is called to a single-family home for the elderly pt who lost their balance and fell onto the carpeted floor. There is a language barrier, but family is present to give history. They state there was no LOC, but the pt has been “sick” for the past 2 weeks with fever, SOB, and a loss of smell and taste after returning from his “homeland.”

Lungs: cough noted with + congestion

CV: ECG – A-fib

Skin: Skin is hot to touch and moist; temp of 101.9° F

VS: BP 102/50; P 60; R 16; SpO2 90%

PMH: AFib, diabetes, HTN

System memos 389, 390, 391, 392 as a reference for these answers

Dispatched for an elderly person who fell. How and who enters the residence to evaluate the situation?	
After assessment and treatment has been rendered, what necessary information should be relayed to OLMC?	
After transporting the pt to the ED for evaluation, what special criteria is needed for cleaning of the ambulance?	
What expectation should EMS have of the hospital if the pt should receive a diagnosis of COVID?	
Misc Questions: EMS is at the FH for shift and at 2000, your temperature is noted to be 100.8 °F and you feel extreme fatigue. What process should be followed immediately upon identifying a fever?	
Three days later, you continue to have a fever and are not able to return to work. What notification steps should now be taken by the ill individual?	

The following two pages are an update from the IPC Center discussing the risk of using chemical products for cleaning. As much as we have encouraged you to protect yourself from transmission of diseases such as COVID-19, it is also important for you to maintain awareness of the situation and not contaminate yourself in any way.

Please keep yourself up to date on the latest information sent by the EMS system and on the website.

Stay safe and a HUGE THANK YOU for all you are doing for our communities!

POISON Help 1-800-222-1222 | ILLINOIS POISON CENTER

April 13, 2020

Contact: Danny Chun: 630-276-5558
dchun@team-iha.org

IPC Cleaning Product Exposures Up 30%

COVID-19 Pandemic Causes Surge in Exposures reported to Nation's Oldest Poison Center

The Illinois Poison Center (IPC) urges people to use caution with cleaning products, especially during the COVID-19 pandemic. According to IPC data, exposures to cleaning products are up 30%, compared to last year, related to novel coronavirus transmission precautions and concerns.

Examples of exposures the IPC is managing include:

- People using non-traditional chemicals to wash their hands (e.g. bleach, hydrogen peroxide, wipes, etc.) instead of regular hand soap resulting in rash/irritation and cracked skin.
- People using chemicals (e.g. bleach, wipes, cleaning powders) to wash their groceries, including produce and are then concerned about toxicity upon ingestion;
- Mixing cleaning chemicals together and inadvertently producing toxic gas; and
- Pediatric exposures to cleaning products left open/unattended.

"It is critical that consumers read all cleaning product labels carefully before use and heed any warnings," says IPC Assistant Vice President Carol DesLauriers, Pharm.D. "While good hand hygiene and household disinfecting efforts are important in the fight against COVID-19, people must remember to use cleaning products for their intended use. If there is any doubt about the safe use of cleaning products, call the IPC for guidance."

While many people are using the stay-at-home order to tackle spring cleaning, in conjunction with disinfecting to prevent the spread of COVID-19, the IPC offers the following tips for poison-proofing your home:

- Whenever using cleaning products, always read the product label first and use the product according to the label directions;
- Keep all cleaning products in their original containers with original labels;
- Store cleaning products out of sight, in locked cabinets;

- Keep all household cleaning products and other potentially harmful products separated from food products;
- Never leave a cleaning product open and unattended;
- When using cleaning products, work in well-ventilated areas; and
- Dispose of cleaning products according to the instructions on the label or at your community chemical waste drop-off site.

The good news is that approximately 90% of poison exposures can be treated safely and effectively at home, by calling the IPC at 800-222-1222 and providing the name of the poisonous substance and the approximate amount involved in the exposure.

In case of a poisoning exposure, follow these first-aid steps, then call the IPC:

- **Swallowed:** Give a few sips of water to drink. If the patient is unconscious, call 911 or take them to the nearest hospital emergency department. Never try to chemically neutralize any poison.
- **Skin:** Remove contaminated clothing and wash skin gently with soap and cool water.
- **Eyes:** Rinse eyes with lukewarm water for 15 minutes.
- **Fumes:** Remove patient to fresh air, taking care not to become exposed yourself. If the patient is not breathing, call 911 and start artificial respiration and continue until medical help arrives.

IPC experts are available to provide information and treatment advice 24 hours per day, 365 days per year, including holidays. If you suspect that you or someone you know has been exposed to a potentially harmful substance, please call the IPC at 800-222-1222. The call is free and confidential. For more information, visit the IPC's website: https://www.illinoispoisoncenter.org/

###

The Illinois Poison Center is a nonprofit health service that provides the people of Illinois with comprehensive and trusted information and treatment advice on potentially harmful substances via a free, confidential 24-hour helpline staffed by specially trained physicians, nurses and pharmacists.

https://www.illinoispoisoncenter.org/PR-Increase-in-Cleaning-Product-Exposures