

Northwest Community EMS System
Paramedic Education Program
HEAT EMERGENCIES
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Reading assignment:

Aehlert Vol. 2, pp. 543-547
SOP: Heat Emergencies

Knowledge objectives:

Upon reading the assigned text assignments and completion of the class and homework questions, each participant will independently do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Define environmental emergency.
2. Describe the incidence, morbidity and mortality associated with environmental emergencies.
3. Identify risk factors most predisposing to environmental emergencies.
4. Identify environmental factors that may cause illness or exacerbate a preexisting illness.
5. Identify environmental factors that may complicate treatment or transport decisions.
6. List the principal types of environmental illnesses.
7. Define "homeostasis" and relate the concept to environmental influences.
8. Identify normal, critically high and critically low body temperatures.
9. Describe several methods of temperature monitoring.
10. Identify the components of the body's thermoregulatory mechanism.
11. Describe the general process of thermal regulation, including substances used and wastes generated.
12. Describe the body's compensatory process for over heating.
13. Describe the body's compensatory process for excess heat loss.
14. List the common forms of heat and cold disorders.
15. List the common predisposing factors associated with heat and cold disorders.
16. List the common preventative measures associated with heat and cold disorders.
17. Integrate the pathophysiological principles and complicating factors common to environmental emergencies and discuss differentiating features between emergent and urgent presentations.
18. Define heat illness.
19. Describe the pathophysiology of heat illness.
20. Identify signs and symptoms of heat illness.
21. List the predisposing factors for heat illness.
22. List measures to prevent heat illness.
23. Discuss the symptomatic variations presented in progressive heat disorders.
24. Relate symptomatic findings to the commonly used terms: heat cramps heat exhaustion, and heat stroke.
25. Correlate the abnormal findings in assessment with their clinical significance in the patient with heat illness.
26. Describe the contribution of dehydration to the development of heat disorders.
27. Describe the differences between classical and exertional heat stroke.
28. Define fever and discuss its pathophysiologic mechanism.
29. Identify the fundamental thermoregulatory difference between fever and heat stroke.
30. Discuss how one may differentiate between fever and heat stroke.
31. Discuss the role of fluid therapy in the treatment of heat disorders.
32. Differentiate among the various treatments and interventions in the management of heat disorders.
33. Integrate the pathophysiological principles and the assessment findings to formulate a field impression and implement a treatment plan for the patient who has dehydration, heat exhaustion or heat stroke.

NWC EMSS Paramedic Education Program

HEAT EMERGENICES

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I. Introduction

The **environment** can be defined as all of the surrounding external factors that affect the development and functioning of a living organism. Factors such as temperature, weather, terrain, and atmospheric pressure can act on the body to produce stressors for which the body is unable to compensate. **A medical condition caused or exacerbated by environmental factors is known as an environmental emergency.** Examples: heat cramps, heat exhaustion, heat stroke, hypothermia, frostbite, drowning or near drowning, altitude illness, diving accidents, or barotraumas, and nuclear radiation exposure.

II. Thermoregulatory mechanisms

A. Primary control: hypothalamus (set point)

1. 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.
2. 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).
3. 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).
4. 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 approx 98.6° F in face of continuous internally generated heat and widely variable temperatures.
5. 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**).

B. Heat production/gain

1. 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.
2. **Internal**
 - a. 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.

- b. 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).
 - c. **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.
 - d. **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.
 - e. **Physical activity** (*work-induced thermogenesis*): Hard work or exercise can increase metabolic rates and heat production 12 fold. Can ↑ temp to 102° F. Can cause temp increases of 9° F/hour but only results in 2° increases if normal heat dissipating mechanisms are working.
3. **External environment:** The body receives heat via the **thermal gradient** between the body and the environment. Several factors influence the thermal gradient:
- a. **Ambient air temperature** (heat waves): The body absorbs heat from a warm environment by radiation, convection, and conduction. The mortality rate is increased 3-5 times normal in nursing homes without air conditioning. Gradually overtaxes an elderly person's limited accommodative processes. Beware of heat absorption from the soles of the feet, i.e. social/work situations (hot sand).
 - b. Infrared radiation (sun: 150 Kcal/hr.)
 - c. **Humidity:** reduces body's ability to evaporate heat: may be caused by mini or macro climates, i.e., sauna baths, hot tubs.

C. **Normal heat loss** (*thermolysis*)

1. 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.
2. **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.
 - a. **Radiation:** Body heat is lost to nearby objects or environment through 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.
 - b. **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.

- c. **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. A wind chill chart calculates the cooling effects of the ambient temperature based on thermometer readings and wind speed.
- d. **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.

3. **Body's physiological compensatory responses**

a. **Peripheral blood flow**

- (1) One of the ways the body regulates temperature is by sensing the warmth of blood flowing by cells in the hypothalamus. In response to \uparrow warmth, **blood is directed towards the skin surface** through capillary **vasodilation**. These responses are mediated by the endocrine glands and the autonomic nervous system.
- (2) This can increase flow 7-20 times beyond the normal rate and can shunt up to 4 L/min. through those vessels, thereby improving radiation and convection to dissipate heat.
- (3) Vasodilation with shunting necessitates an **increase in cardiac output** 2-4 X normal. The heart must be capable of keeping up with the increased demand. HR and SV both affect cardiac workload and oxygen consumption. If heat accumulates beyond the limits of a person to \uparrow their cardiac output, heat accumulates and the temperature rises.
- (4) Splanchnic beds contract and **sodium and water are retained** by the kidneys in response to the release of aldosterone and growth hormone.
- (5) Conversely, if the environment is too cold, *counter-current* heat exchange is used to vasoconstrict superficial vessels and shunt warm blood away from the body's outer shells near the skin back into the deeper veins near the core to keep vital organs warm.

b. **Sweating**

- (1) Sweating accounts for almost all heat loss until ambient air temperature rises to about 95° F or humidity reaches 75% when sweating becomes less effective. Diaphoresis can markedly increase evaporative heat loss provided that the relative humidity is low enough that the sweat can evaporate. At humidity levels above 75%, evaporation decreases. At levels approaching 90%, evaporation essentially ceases (Sanders, 1081).
- (2) For every 5° F increase in air temperature, less humidity is necessary to raise the risk. Example: At 70° F the "danger zone" would start at 80% humidity. Strenuous activity at 90° F would prove dangerous at just 40% humidity.

- (3) Heat loss = 1 Kcal of heat for each 1.7 mL of sweat vaporized. Can lose 1.5 L sweat/hr or 882 Kcal/hr. Under average summer humidity, the practical limit is 650 Kcal/hr due to dripping and pooling of liquid which does not dissipate heat.

Body tolerates volume losses of 5% without difficulty. Physiologic derangement if losses of 7% or greater without fluid replacement.

- c. With all these mechanisms available, about 97% of heat loss occurs at the skin/air interface with **blood acting as the coolant**.
- d. These mechanisms can create a difference between the core and peripheral temperature. Core temps taken from the rectum or tympanic membrane are the crucial measurement. Oral or axillary temps may be misleading in patients with an environmental illness.

III. Heat illness epidemiology and general causes

A. **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**.

1. **Two situations may exist**

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

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

B. **Hyperpyrexia vs. hyperthermia**

1. 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 an unusually high 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.

2. 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 patient is suffering from fever or heat stroke, treat for heat stroke.

3. The NWC EMSS does not treat regular fevers. Up to 102° F, they are the body's way of attacking the illness and demonstrate an appropriate immune system response. The only reason to give an antipyretic like acetaminophen (Tylenol) is to make the patient more comfortable. This can be accomplished at the hospital. Febrile seizures will be covered under seizure activity.

C. **Predisposing factors for thermoregulatory emergencies**

1. High-risk status is a function of age, overall health or activity level and environmental factors. Additional contributing factors:
 - a. Dehydration
 - b. Increased endogenous heat load
 - c. Skin/sweat gland abnormalities
 - d. Lack of acclimatization
2. **Elderly:** Unique susceptibility due to their inability to compensate; 40-80% mortality.
 - a. Less able to sense and respond to temperature changes
 - b. C-V system unable to increase cardiac output
 - c. Structural and functional changes occur in skin
 - d. Decreased efficiency of sweating
 - e. Decreased ability to acclimate
 - f. Increased use of medications which generally interfere with heat dissipation
3. **Infants and young children**
 - a. They are generally active and energetic, producing more metabolic heat.
 - b. Limited reserves and inability to protect themselves.
 - c. Difficulty in regulating their body temps due to scanty layer of insulating fat and high ratio of skin surface to body mass (weight).
 - d. Abundance of extracellular fluid that is rapidly turned over.
 - e. Infants do not sweat; children have fewer sweat glands than adults.
 - f. Frequently overdressed and may be left in hot, unventilated cars.
4. **Chronic diseases**
 - a. Cardiovascular diseases: atherosclerosis, CHF, hypertension
 - b. 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).
 - c. **Increased heat load:** Fever, seizures, exertion, hyperthyroidism, agitated psychiatric conditions. Status epilepticus: will generate huge amounts of heat.
 - d. Parkinsonism
 - e. Tumor of the adrenal gland (pheochromocytoma)
 - f. Malnutrition
 - g. 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.
 - h. Ill or debilitated
5. **Drugs:** Can impair accommodation or generate heat themselves
 - a. Those that **decrease sweating capacity** and interfere with central thermoregulation: anticholinergics (Librax, Donnato), antihistamines (Benadryl), tricyclic antidepressants, benzotropine (Cogentin); phenothiazines (Thorazine)
 - b. Those that **increase heat production:** amphetamines, LSD, Cocaine, Phencyclidine; MAO inhibitors (↑ muscle activity)
 - c. Those that cause **dehydration and electrolyte imbalances:** Lithium, Haldol, Navane, Prolixin; diuretics, alcohol

- d. **Beta blockers** (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).
6. **Military personnel** (exertion)
 - a. Undergo rigorous training in hot, humid weather
 - b. Lack of conditioning, "macho" behavior taxes system
 - c. Excessive zeal, ↑ ambient temps, water deprivation, lack of warm-up exercises, occlusive clothing, and forced maximal participation contribute to hyperthermia.
7. **Amateur athletes** (exertion) - largest number of cases in US
 - a. 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.
 - b. 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.
 - c. 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.
8. **Farmers and outdoor workers** (exertion)
9. **Skin and sweat gland (sudomotor) dysfunction states** impair heat dissipation
 - a. Extensive prior burns; destroy sweat glands; grafts don't sweat
 - b. Prior heat stroke; necrotic sweat glands
 - c. Cystic fibrosis
 - d. Previous extensive scars
 - e. Scleroderma
 - f. Miliaria; prickly heat - damages sweat glands
 - g. Spinal cord injury with poikilothermia: ↓ ANS tone = dysfunctional sweat glands; capillaries no longer dilate and constrict to regulate body temp by radiation.
10. **Miscellaneous factors**
 - a. Fatigue
 - b. Lack of sleep
 - c. Unaccustomed physical tasks
11. **Level of acclimatization**
 - a. Most heat disorders stem from hypovolemia and electrolyte imbalances rather than hyperthermia, but they can have mixed causes. Lack of fitness and acclimatization leads to F&E disorders.
 - b. Acclimatization begins after prolonged exposure to high ambient temps of at least 2 weeks. In response to an environmental change, reversible body structure and function changes take place to help maintain homeostasis. Ex: Training periods should be scheduled for 5-6 weeks for hot-weather sports, building up gradually in the same heat conditions as the event. Athletes should be well hydrated before exertion. Thirst is an inadequate indicator of dehydration.
 - c. This produces an **increased ability to sweat**: up to 2.5-3 L/hr. plus a decrease in excreted Na = ↑ heat dissipation.
 - d. **ECF volume expansion**

- e. ↑ **SV and aerobic efficiency** = cutaneous dilation
- f. ↑ **glycogen stores** in muscles = ↑ metabolic efficiency

- 12. Length of exposure
- 13. Intensity of exposure
- 14. Environmental factors such as humidity and wind

D. **General reactions to ↑ body temperature**

- 1. Early symptoms of heat injury: stumbling or clumsiness, headache, nausea, dizziness, sweating and subtle changes in mental status.
- 2. 102° F: Malaise, drowsiness, joint and muscle pains
- 3. > 104° F: Disorientation and ↓ awareness
- 4. 106° F: Seizures
- 5. 108° F: Irreversible brain damage: ***It doesn't take long to cook an egg or a neuron!***

IV. **Assessment**

A. **History** must include the following:

- 1. OPQRST of chief complaint
- 2. Patient's environment and activity level immediately before and at time of onset;
- 3. SAMPLE history
 - a. Current meds and dosing compliance
 - b. Past medical history to include chronic conditions, including skin diseases

B. **Secondary assessment**

- 1. Full VS: Baseline temp: Measure by touch or by taking an oral or axillary reading. Core temps can be measured using tympanic or rectal thermometers.
- 2. Review of systems

V. **Heat illnesses with thermoregulatory mechanisms intact**

A. **Heat edema**

- 1. **Pathophysiology:** Vasodilation and stasis cause fluid accumulation evidenced as mild swelling of the hands and feet which occurs in the first few days of exposure to a hot environment. Usually in the elderly with no underlying cardiac, hepatic, venous, or lymphatic disease. May reach pitting edema stage secondary to heat-induced hyperaldosteronism.
- 2. **Treatment:** Usually resolves after a few days of acclimatization. Elevate legs, reassure, place in cool environment.

B. **Prickly heat** (miliaria rubra)

- 1. **Pathophysiology:** Occurs in unacclimatized persons (especially children) subjected to hot temperatures with high humidity. Caused by blockage of the sweat glands, usually in an area where clothing binds or chafes.
- 2. **Signs and symptoms:** Characterized by tiny vesicles and papules with surrounding redness (looks like a rash). Patient experiences itching and a prickly sensation from sweat trapped beneath the skin. May be fatigued with a fever but sweating is usually minimal. Secondary staph infection sometimes develops.
- 3. **Treatment:** Patients should apply topical antiseptic cream, wear loose, light-weight clothing and keep cool and dry. Can be seen in bikers who wear the neoprene (rubber-like) bicycling shorts.

C. **Heat cramps**

1. **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.
2. **Signs and symptoms**
 - a. Painful spasms of the skeletal muscles: thigh, calf, foot, arms
 - b. Cramps of abdominal muscles usually follow rapid ingestion of large volumes of cold water or other liquid following intensive exercise
 - c. Temperature is generally normal
 - d. HR: Tachycardia
 - e. BP: Normal
 - f. Respirations normal to slightly increased
 - g. Skin: Warm w/ diaphoresis
 - h. Mental status intact; alert and oriented
 - i. Occasional nausea and gastritis
3. **Treatment**
 - a. Rest; remove from hot environment
 - b. Restore sodium and water. Slow rehydration with mildly cool but not cold Gatorade-type electrolyte drinks are effective. Sugary beverages that decrease gastric emptying and dehydrating alcoholic beverages should be avoided. IV fluids are usually not necessary.
 - c. Salt tablets sometimes delay gastric emptying causing stomach irritation, ulceration, and cramping or nausea and vomiting, so should be avoided.
 - d. Pain control; stretching the affected muscle groups useful but active massage is counterproductive; narcotics unnecessary.

D. **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.

E. **Heat exhaustion** (other terms: prostration, collapse or syncope)

1. **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.
2. **Two basic types**

- a. **Water depletion:** Develops in hours; can lose 1-3 L/hr. More serious of the two types. Fluid is sequestered in interstitial muscle and by dilation of skin (dermal) and muscular vascular beds during work and heat stress. Response to the sensation of thirst does not provide enough fluid. If cardiac output cannot meet demands, hypotensive collapse is imminent.
- b. **Salt depletion:** Develops over days due to failure of normal sweating mechanisms and polyuria.

Pure forms of either are rare

3. **Signs and symptoms**

- a. **Prodrome:** Fatigue, weakness, thirst, muscle cramps, lightheadedness, (orthostatic dizziness may lead to syncope), irritability, headache, feeling of impending doom
- b. **VS**
 - (1) Increased heart rate to provide additional blood flow through the skin and muscles to enhance heat radiation; pulse is often weak and thready.
 - (2) ↑ Respiratory rate to dissipate heat through exhaled air.
 - (3) Decreased BP (due to vasodilation and/or dehydration) or orthostatic hypotension.
 - (4) 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.
- c. **Skin:** Diaphoretic to enhance evaporative heat loss; pale or flushed; can be dry if dehydrated
- d. **GI:** Anorexia, nausea, vomiting suggesting flu-like illness
- e. **Neuro:** Irritability, anxiety and impaired judgment or mildly confused although **mental function remains intact. Important distinction.**

4. **Treatment**

- a. Move to a cool environment; remove as much clothing as possible to facilitate cooling.
- b. **Initial Medical Care;** special considerations:
 - (1) Position patient supine or on side if vomiting. The majority of hypotension relates to vascular dilation, not absolute fluid loss.
 - (2) Rehydrate with IV NS. Four liters over 6-8 hours may be needed for young people (fluid challenge in field - 20 mL/kg in peds). Use caution to prevent fluid overload in the elderly or debilitated.
 - (3) Vomiting precautions; ready suction
 - (4) Continuously monitor ECG
 - (5) Monitor and record mental status. Can progress to heat stroke if left untreated.

VI. **Heat illness with failed thermoregulatory mechanisms: Heat Stroke**

- A. **Definition:** Heat stroke is a state of **thermoregulatory failure** with **pyrexia** (↑ temperature) after exposure to high environmental temperatures. If the body's hypothalamic regulation is lost, uncompensated hyperthermia develops and the temp rises above 41° C (**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.**

B. Pathophysiology of heat stroke

1. Gradual depletion and failure of body reserves in the face of **heat stress**.
2. Fluid & electrolyte depletion due to copious uncompensated sweating = dehydration, hypovolemia and vasodilation.
3. Anaerobic metabolism uncouples oxidative phosphorylation - cannot produce ATP (energy) - cells begin to die.
4. This leads to **significant cardiac stress** with peripheral vascular shutdown, **sweat gland collapse**, and frank cardiac decompensation.
5. Simultaneous **redistribution of volume** from the core to the peripheral circulation **compounds hypotension**.
6. 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**.

C. Two types

1. **Classic (passive) heat stroke (CHS):** Most common in elderly, small children, the chronically ill and those who live in homes without air conditioning. Examples include children who are left in automobiles on a hot day and older persons confined to a hot room during a heat wave. These patients often suffer from chronic diseases such as diabetes, heart disease, alcoholism, or psychiatric disorders which predispose them to the syndrome.

Mortality is up to 70%-80% due to cell death and physiologic collapse, depending on the age of the patient (elderly, very young), availability of treatment, and underlying illnesses. Approximately 4000 deaths/year in U.S.

2. **Exertional heat stroke (EHS):** Occurs in fit, young persons who exercise in hot and humid conditions and usually when they are partially acclimatized. Heat accumulates more rapidly than it can be dissipated. Because it develops over hours, dehydration may not be present. Half of the victims will still be sweating profusely. (High output cardiac failure). Mortality about 20%.

D. Clinical presentation: onset acute or subacute based on type

1. **CHS:** Prodromal S&S similar to heat exhaustion; though they may become more severe until all temperature control is lost. Generally brought to hospital after coma, fever, and hypotension present.
2. **EHS:** Prodromal S&S of chills, throbbing pressure in the head, nausea, unsteadiness and gooseflesh on chest and upper arms. Usually seek medical attention because of bizarre behavior or collapse.
3. **Signs and symptoms**
 - a. **High temperature:** greater than 105° or 106° F (107°-108° not unusual). Rises rapidly when sweating ceases. High temp increases myocardial oxygen consumption, and, in the presence of hypertension or a volume overloaded myocardium caused by congestive failure, arteriosclerotic vascular disease or CAD, may lead to a malignant dysrhythmia or AMI.
 - b. **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.

- (1) Confusion, disorientation, irrational behavior, sudden loss of consciousness. (May appear psychotic)
 - (2) Lethargic to combative
 - (3) Fixed, dilated pupils
 - (4) Tremors, seizures, or abnormal flexor or extensor posturing
 - (5) Healthy people can get ill v. quickly. They become confused, aggressive, and euphoric - then collapse.
- c. **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.
- d. **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.
- (1) Pump failure, pulmonary edema, and major dysrhythmias follow the hyperdynamic state resulting in hypotension (High output cardiac failure). Hypotension indicates a poor prognosis.
 - (2) Hypotension may be due to the following:
 - (a) Loss of Na and water
 - (b) Peripheral vasodilation with pooling (enhanced by lactic acidosis)
 - (c) Myocardial dysfunction causing dysrhythmias, infarction and failure due to local hemorrhage, cellular degeneration and spotty inflammation. May need a Dopamine drip.
 - (d) Localized disseminated intravascular coagulation (DIC)
- e. **Sweating** may be present or absent depending on type of HS and whether or not the patient is dehydrated. Skin 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.
- f. **Liver involvement:** ↓ production of clotting factors, hepatocellular necrosis can be severe. Assess for GI bleeding, ileus, liver failure, clotting disorders and electrolyte imbalances.
- g. **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.
- h. **Fluid & electrolyte (↓ K) imbalances** play a crucial role. The patient may suddenly collapse with no prodrome (advance warning).
- i. **Kidney involvement:** Commonly affected due to hypovolemia and hypoperfusion. Ten percent develop acute tubular necrosis (ATN) or myoglobin tubular block. Rhabdomyolysis (muscle breakdown) results from direct injury or ischemia. Manifested by muscle pain, myoglobinuria (EHS). (Dipstick for hemoglobin is heme positive in absence of microscopic hematuria = ↑ risk for kidney damage but should be reversible).
- E. **Treatment:** High mortality rate if untreated. Need rapid assessment with aggressive interventions. Patient in hypermetabolic state.

1. Move patient to a cool environment, remove all clothing and check for injuries. Obtain a baseline temperature reading.
 2. **Rapid cooling**
 - a. Apply cold packs to axillae, groin, sides of neck and chest; spray 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).
 - b. Cool at a rate of 0.2° C/minute. 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.
 - c. Gently massage skin to counter peripheral vasoconstriction, accelerate heat loss, and promote venous return.
 3. If in pulmonary edema, may need to intubate and provide ventilatory assist
 4. 15 L oxygen (C-PAP may be very useful)
 5. Monitor ECG: anticipate ST-T segment changes and dysrhythmias
 6. Monitor VS and BS carefully. With pulmonary edema, BP will elevate rapidly - develops hours after collapse.
 7. **Careful monitoring of fluid volume status.** If hypovolemia is present, give a fluid challenge of 500 mL over 15 minutes. 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.
 8. Anticipate seizures and prepare midazolam.
 9. Anticipate hypoglycemia: obtain capillary blood glucose reading
 10. Observe for development of cerebral edema and ↑ ICP
 11. **ASA ineffective;** may contribute to coagulopathies and hepatic damage
- F. **If coma persists after cooling, physicians** will work patient up for the following:
1. Meningitis
 2. Encephalitis
 3. Septic shock
 4. Malaria
 5. Typhus
 6. Typhoid fever
 7. Hypothalamic lesion (unilateral anhidrosis, diabetes insipidus)
 8. Midbrain hemorrhage
 9. Thyroid storm
 10. Drug intoxication withdrawal: metabolic overdrive, DT's
 11. Drug induced heat illness (cocaine, anticholinergics, PCP, salicylates)
 12. Malignant hyperthermia
- G. **Prognosis:** Depends on prior state of health, length of time under heat stress, and state of oxygenation.

1. If coma is present longer than 10 hours; a fatal outcome is likely
2. Rectal temp exceeding 108° F (42.2° C) indicates a poor prognosis

H. **Immediate complications**

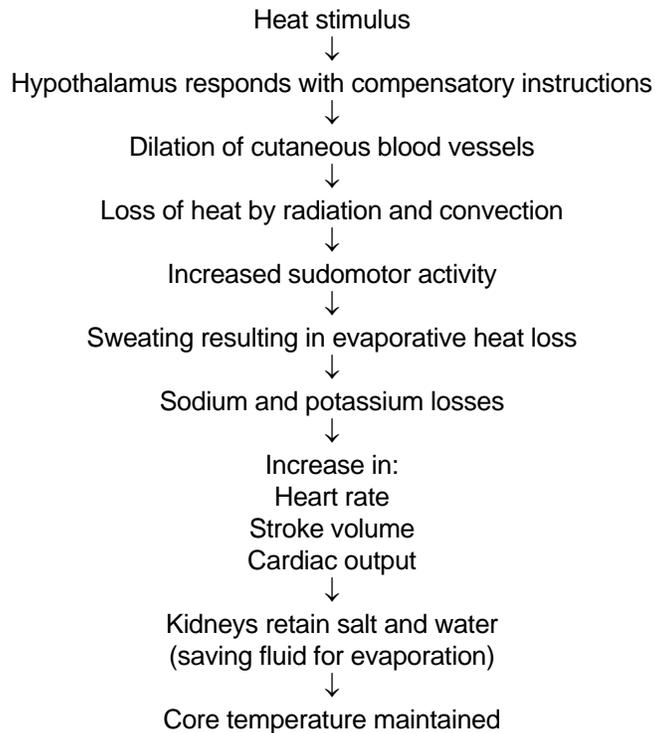
1. Violent shivering: prevent shivering so more heat is not generated.
2. Seizures; Rx with midazolam per SOP
3. Hypotension in absence of dehydration due to shunt through dilated skin vessels. Monitor fluid status and VS carefully as fluid overload can occur when vasoconstriction returns large volumes to the central circulation resulting in pulmonary edema.
4. Electrolyte imbalances (hyponatremia, hypokalemia)
5. Acute renal failure
6. Rhabdomyolysis
7. Liver failure
8. Clotting abnormalities; DIC
9. ARDS

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Clinical Presentation	Heat Cramps	Heat Exhaustion	Heat Stroke
Muscle cramps	+	+ / -	-
Temperature	Normal	Normal→low/may be higher with mixed illness	Very high
Respirations	Normal to tachypnea	Tachypnea w/ hyperventilation	Hyperventilation→ventilator y failure
Pulse	Normal to tachycardia	↑ or ↓; weak and thready	Full, rapid
BP	Normal	Decreased or orthostatic hypotension.	May be ↑ or ↓
Skin temp/color	Normal	Cool/pale or flushed	Hot, red, flushed
Skin moisture	Diaphoretic	Diaphoretic	Diaphoretic or dry
Mental status	Intact	Intact; may have syncope	Always altered; confused → Coma
Degree of severity/urgency	Low	Moderate	High/severe

THERMOREGULATION IN RESPONSE TO LIMITED HEAT STRESS



Homework Questions

1. A medical condition caused or exacerbated by the weather, terrain, atmospheric pressure, or other local factors is called a/n _____ emergency.
 - A. Homeostatic
 - B. External
 - C. Environmental
 - D. Pyrexia
2. Which DOES NOT predispose certain individuals to developing environmental illnesses?
 - A. Age
 - B. Complexion
 - C. Fatigue
 - D. Medications
3. Which is the natural tendency of the body to maintain a steady and normal internal environment?
 - A. Homeostasis
 - B. Thermodynamics
 - C. Homeopathy
 - D. Normostasis
4. Thermal gradient is the difference in temperature between the
 - A. highest and lowest normal body temperature.
 - B. lowest and highest tolerable temperatures.
 - C. hypothermia and hyperthermia.
 - D. environment and the body.
5. Thermoregulatory thermogenesis is the production of heat resulting from the
 - A. high environmental temperatures.
 - B. increase in the rate of cellular metabolism.
 - C. exercise of muscles working effectively.
 - D. processing of food and nutrients.
6. Which mechanism of heat loss occurs when the body's surface has direct contact with another, cooler object?
 - A. Convection
 - B. Conduction
 - C. Radiation
 - D. Evaporation
7. Which mechanism of heat loss enables an unclothed person to lose approximately 60% of their total body heat at room temperature?
 - A. Convection
 - B. Conduction
 - C. Radiation
 - D. Evaporation
8. Which brain structure is responsible for temperature regulation?
 - A. Pituitary gland
 - B. Hippocampus
 - C. Hypothalamus
 - D. Corpus callosum

9. Which IS NOT a clinical sign suggesting that the body is trying to dissipate heat?
- A. Diaphoresis
 - B. Increased skin temperature
 - C. Flushing
 - D. Cyanosis

10. **Case 1**

An 18 y/o conscious male is found in a neighborhood park where he and several friends have been playing basketball. 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 to play. 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

What is your paramedic impression?

What is the pathophysiology which led to his presenting physical signs? What electrolyte has been lost?

What treatment should you initiate per SOP?

What can be done to relieve this patient's pain?

11. **Case 2**

A 60 y/o conscious male is found on the golf course propped up under a tree. Bystanders state that he had been complaining of weakness, lightheadedness, headache and nausea after the first nine holes. Outdoor temperature is 92°. The patient has a history of diabetes and heart disease and takes Inderal.

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: Cool, pale, diaphoretic
VS: BP: 98/60; P: 72 weak and thready; R: 24; T: 99° F

What is your paramedic impression?

Why is this patient at high risk for developing a heat disorder?

What mechanism of heat dissipation has his body attempted?

What is the pathophysiology that led to his current condition?

What interventions should you initiate per SOP?

12. **Case 3**

You find a 70 y/o unconscious female in her apartment two days after a blistering 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:	Bilateral crackles
CV:	ECG: ST with PVCs
GI:	Soft, no guarding, masses
Skin:	Hot, flushed and dry
VS:	BP: 80 palp; P: 112; R: 32; T: 106° F

What is your paramedic impression?

Why is her skin dry?

Why does she have an altered mental status?

What neurological complications should you anticipate?

What signs and symptoms would alert you to the development of those complications?

What type of heart failure is imminent?

Why does she have crackles?

What medication should you anticipate to treat her ↓ CO?

What electrolyte disorder should be anticipated?

What portion of her ECG would alert you to a change in this electrolyte?

How should her airway be secured?

How should oxygen be delivered to this patient?

What type of fluid therapy appears indicated?

What could happen to her BP if the dehydration was corrected?

How should you attempt to cool this patient?

You should lower her temperature to no lower than _____.

What is her general prognosis?