

Chapter 133 – Heat Illness

Episode overview

- 1. List 10 predisposing factors for heat illness
 - a. Including discussion of heat illness pathophysiology
- 2. List the four types of minor heat illness include their clinical features and management.
 - a. Prickly heat
 - b. Heat cramps
 - c. Heat edema
 - d. Heat syncope
- 3. Describe the types of major heat illness.
- 4. Differentiate between minor heat illness, heat exhaustion and heat stroke clinically
- 5. Compare exertional and classic heatstroke
- 6. Describe the diagnostic features of heatstroke
- 7. List 5 cooling measures for heat stroke + 3 adjuncts to therapy
- 8. List 8 DDx for hyperthermia/hyperthermia (*temp* and altered)

Wisecracks

- 1. What is meant by wet bulb globe temperature?
- 2. List etiologies of hyperthermia.
- 3. List six types of heat-related illnesses.
- 4. Describe the pathophysiology of heat exhaustion.
- 5. Describe the pathophysiology of heat stroke.
- 6. How do you calculate the free water deficit?
- 7. What are the targets of cooling?

Rosen's In Perspective:

Heat illness usually occurs in two population groups: the athlete/military person or the elderly / destitute.

- Football athletes are the highest risk athlete group for heat illness
- Older adults and the poor, who often lack adequate air conditioning and nutrition, and those with preexisting disease are prone to heat illness during environmental extremes. It is estimated that at least 10 times as many heat-aggravated illnesses occur in patients with comorbid conditions, such as coronary artery disease, cerebrovascular disease, and diabetes.
 - During heat waves, many deaths are prevented due to air conditioning units.
- Let's talk about HEAT PRODUCTION
 - Occurs due to the body's biochemical furnace which is an exothermic reaction; this increases during strenuous exercise
 - Environmental heat adds to the body's heat load and prevents heat dissipation



- Heat transfer in humans occurs via:
 - Conduction: the thermal conductivity of water is at least 25 times that of air.
 - Convection: Once the air temperature exceeds the mean skin temperature, heat is gained by the body. Convective heat loss varies directly with wind velocity.
 - Radiation: heat transfer by electromagnetic waves. Although radiation accounts for approximately 65% of heat loss in cool environments, it is a major source of heat gain in hot climates. Up to 300 kcal/hr can be gained from radiation when someone is directly exposed to the hot summer sun.
 - **Evaporation:** As the ambient temperature rises, evaporation becomes the dominant mechanism of heat loss.
- Let's discuss HEAT REGULATION
 - Regulated by:
 - Thermosensors on the skin and in the hypothalamus (sweating or behaviour change)
 - Central integrative area the body's thermostat or set-point regulator
 - Thermoregulatory effectors leading to sweating and peripheral vasodilation (increased cardiac output)
- Normally our body is good at adapting to heat after 7-14 days of daily exposure (acclimatization)
 - an earlier onset of sweating (at a lower core temperature), increased sweat volume, and lowered sweat sodium concentration.

Although many similarities exist among thermoregulatory responses to heat and exercise, the well-conditioned athlete is not necessarily heat-acclimatized. For heat- and exercise-induced adaptive responses to be maintained, heat exposure needs to continue intermittently, at least on 4-day intervals. Plasma volume decreases considerably within 1 week in the absence of heat stress.

- Rosen's 9th Edition, Chapter 133

Core questions

1) List 10 predisposing factors for heat illness

Non-exertional heat illness increase the risk for classic heatstroke during periods of high heat and humidity:

- advanced age,
- psychiatric conditions,
- chronic disease,
- obesity,
- medications



- Pump or circulation problem:
 - Beta blockers, CCBs
- Dehydration
 - Diuretics
- Inhibition of sweating
 - Anticholinergics, stimulant drugs

Exertional heat illness:

- Lack of acclimatization
- Inadequate hydration (cool, flavored liquids) goal body weight within 1% of previous day's weight
- Weight class / category athletes
- Wet bulb globe temperature > 25

a. Including discussion of heat illness pathophysiology

Car analogy. Coolant (blood) is circulated by a pump (heart) from the hot inner core to a radiator (skin surface cooled by the evaporation of sweat). Temperature is sensed by a thermostat (CNS), which alters coolant flow by a system of pipes, valves, and reservoirs (vasculature). Failure of any of these components can result in overheating. (See Rosen's 133.2 for a visual representation of this analogy)

2) List the four types of minor heat illness - include their clinical features and management

- a) Prickly heat
 - aka: Miliaria rubra, lichen tropicus, heat rash
 - Acute inflammatory skin disorder: the blockage of sweat gland pores by macerated stratum corneum and secondary staphylococcal infection.
 - intensely pruritic vesicles on an erythematous base. The rash is confined to clothed areas. Rash can persist for weeks
 - Chlorhexidine in a light cream or lotion is the antibacterial treatment of choice during the acute phase. Salicylic acid, 1% tid, can be applied to localized affected areas to assist in desquamation,
 - Prickly heat can be prevented by wearing light, loose fitting, clean clothing and avoiding situations that produce continuous sweating. The routine use of talcum or baby powder should be avoided.
- b) Heat cramps
 - Not the same thing as exercise associated muscle cramps



- Heat cramps are brief, intermittent, and often severe muscle cramps occurring typically in muscles that are fatigued by heavy work. Heat cramps appear to be related to a salt deficiency.
- They usually occur during the first days of work in a hot environment and develop in persons who produce large amounts of thermal sweat and subsequently drink copious amounts of hypotonic fluid.
- Clinical Features: athletes, roofers, steelworkers, coal miners, field workers, and boiler operators are among the most common victims of heat cramps. Heat cramps tend to occur after exercise, when the victim stops working and is relaxing
- Usually have total body hyponatremia and hypochloremia.
- Most cases respond well to PO salt intake (tablets dissolved in oral solution). IV NS may be needed in severe cases.
- Essentials of diagnosis (Box 133.2)
 - Cramps of most worked muscles
 - Usually occur after exertion
 - Copious sweating during exertion
 - Copious hypotonic fluid replacement during exertion
 - Hyperventilation not present in cool environment
- c) Heat oedema
 - hydrostatic pressure and vasodilation of cutaneous vessels, combined with some degree of orthostatic pooling, lead to vascular leak and accumulation of interstitial fluid in the lower extremities.
 - Increase aldosterone levels further encourage fluid retention
 - Swollen feet/ankles
 - o Usually in adults with existing chronic disease
 - Important DDx in this population CHF, liver disease, infections, or DVT's
 - Managed expectantly.
 - Usually the edema resolves with acclimatization
 - No role for diuretics
 - Support stockings and leg elevation may help
- d) Heat syncope
 - In warm environments an increased volume of blood is in the peripheral circulation and decreases central circulation
 - This is worsened by long periods of standing → placing people at risk for inadequate central venous blood return and a decrease in cerebral perfusion leading to a LOC
 - Usually a multifactorial disorder, but elderly people are more fragile and at risk for it
 - o Do that full hx, physical and let that guide your workup
 - True heat syncope is self-limited and can be prevented with acclimatization, active muscle movement, compression garments, and a euvolemic state



3) Describe the types of major heat illness

a) Heat exhaustion

- A clinical syndrome of volume depletion during heat stress
 - Usually individuals working in a hot environment who don't drink enough water and electrolytes to replace the losses
- Two main types (but usually it is a mixed type) :
 - Salt depletion relative salt deficit due to excessive free water intake (slower to develop)
 - It differs from heat cramps in that systemic symptoms occur. Symptoms are similar to those seen in water depletion heat exhaustion; the body temperature usually remains nearly normal.
 - Water depletion type loss of primarily free water
- **Sx**:
 - Vague.
 - weakness, fatigue, frontal headache, impaired judgment, vertigo, nausea and vomiting and, occasionally, muscle cramps. Orthostatic dizziness and syncope can occur. Sweating persists and may be profuse. The core temperature is only moderately elevated, usually below 40°C (104°F), and signs of severe CNS dysfunction (eg, altered mental status) are not present.

• Heat exhaustion: diagnosis (Box 133.3)

- Vague malaise, fatigue, headache
- Core temperature often normal; if elevated, <40C
- Mental function essentially intact; no coma or seizures
- Tachycardia, orthostatic hypotension, clinical dehydration (may occur)
- Other major illnesses ruled out
- If in doubt, treat as heatstroke
- Labs:

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- HypoNa
- HypoCl
- CPK elevation
- Heat exhaustion: management (Box 133.4)
 - Rest
 - Cool environment
 - Assessment of volume status orthostatic changes, BUN level, Hct, serum sodium concentration
 - Fluid replacement normal saline to replete volume if patient is orthostatic; replace free water deficits slowly to avoid cerebral oedema



 Healthy young patients are usually treated as outpatients, consider admission if the patient is older, has significant electrolyte abnormalities, or would be at high risk for recurrence if discharged

b) Heat stroke

- This is a CATASTROPHIC life-threatening emergency when the body's thermoregulatory systems fail.
- Neurologic dysfunction is a hallmark of heat stroke, and cerebral edema is common.
 - Failure of compensatory peripheral vasodilation and central vasoconstriction leads to cerebral ischemia
- The cardiovascular system is also stressed leading to central vasoconstriction and peripheral vasodilation.
 - compensatory vasoconstriction of the splanchnic and renal vasculatures. The resulting splanchnic and renal ischemia may explain the nausea, vomiting, and diarrhea observed in runners after a marathon. Hepatic damage is a consistent feature of heatstroke, and its absence should cast doubt on the diagnosis.
- Hematological system dysfunction:
 - Abnormal hemostasis is manifested clinically by purpura, conjunctival hemorrhage, melena, bloody diarrhea, hemoptysis, hematuria, myocardial bleeding, or hemorrhage into the CNS. Diarrhea, probably caused by intense splanchnic vasoconstriction, is commonly seen. Cooling aggravates the diarrhea, creating an unpleasant treatment problem. Pancreatitis is described, with elevated serum amylase and lipase levels.
- Heatstroke: diagnosis (Box 133.5)
 - Exposure to heat stress, endogenous or exogenous
 - Signs of severe CNS dysfunction (coma, seizures, delirium)
 - Core temperature usually >40.5C, but may be lower
 - Hot skin common, and sweating may persist
 - Marked elevation of hepatic transaminase levels
- See Rosen's Figure 133.5 for pathogenesis of haemmorhage

4) Differentiate between minor heat illness, heat exhaustion and heat stroke clinically

These things exist on a spectrum: in reality heat exhaustion and heat stroke can have overlapping symptoms. - when in doubt treat as a the most severe form!

Minor heat illness	Heat exhaustion	Heat stroke
One of these presentations:	Vague malaise, fatigue,	Exposure to heat stress,
Heat rash	headache	endogenous or exogenous



 Heat edema Heat syncope Heat cramps 	 Core temperature often normal; if elevated, <40°C (104° F) Mental function essentially intact; no coma or seizures Tachycardia, orthostatic hypotension, clinical dehydration (may occur) Other major illness ruled out If in doubt, treat as heatstroke. 	 Signs of severe central nervous system dysfunction (coma, seizures, delirium) Core temperature usually > 40.5°C (105° F), but may be lower Hot skin common, and sweating may persist Marked tachycardia
Normal temperature No persistent CNS symptoms Localised symptoms	Vague CNS symptoms Intact mental function <40 deg. C. Can proceed heat stroke	Sudden onset altered LOC Hot skin +/- sweat Major CNS dysfunction Coma, seizures Temp > 40.5 (CORE temp) Multi-system tissue damage and organ dysfunction (major lab abnormalities) - Some say any temperature > 40 deg. C AND altered mental status or neurologic findings is sufficient to dx heat stroke

Mild heat exhaustion and full-blown heat stroke represent extremes of the spectrum of heat illness, and intermediate cases may prove difficult to differentiate. Nevertheless, heat exhaustion should not be diagnosed in the presence of major CNS dysfunction* (eg, seizures, coma) or severe hyperthermia (40.5°C [104.9°F]).

*Any type of dysfunction can occur: bizarre behaviour, opisthotonos, hallucinations, rigidity, cerebellar dysfunction

5) Compare exertional and classic heatstroke



Have different presentations and manifestations. CHS occurs during periods of sustained high ambient temperatures and humidity, such as during summer heat waves.

Victims are often older adults and poor and live in underventilated dwellings without air conditioning. Debilitated patients who have limited access to oral fluids may develop water depletion heat exhaustion, which progresses to heatstroke if untreated. Victims of CHS commonly suffer from chronic diseases, alcoholism, or schizophrenia, which predisposes to heat illness. Such patients are often prescribed medications (eg, diuretics, antihypertensives, neuroleptics, anticholinergics) that impair the ability to tolerate heat stress. Sweating ceases in most CHS patients. Factors such as advanced age, hypotension, altered coagulation status, and the necessity for endotracheal intubation on arrival at the ED predict a poor outcome, despite successful cooling measures.

In contrast, patients with EHS are usually young and healthy individuals whose heat-dispelling mechanisms are overwhelmed by endogenous heat production. Athletes and military recruits are typical victims. Rhabdomyolysis and acute renal failure, rarely seen in patients with CHS, are common in patients with EHS. Sweating is present in 50% of cases of EHS. Hypoglycemia may occur as the result of increased glucose metabolism and hepatic damage, resulting in impaired gluconeogenesis. Coagulopathy is common;

- Rosen's 9th Edition, Chapter 133

Exertional	Classic	
Healthy	Predisposing factors or medications	
Younger	Older	
Exercise	Sedentary	
Sporadic	Heat wave occurrence	
Diaphoresis	Anhidrosis	
Hypoglyclaemia	Normoglycaemia	
DIC	Mild coagulopathy	
Rhabdomyolysis	Mild CK level elevation	
Acute renal failure	Oliguria	
Marked lactic acidosis	Mild acidosis	
Hypocalcaemia	Normocalcaemia	
Can Deserve Table 422.4 for existingly some existing table		

Characteristics of classic vs. exertional heatstroke

See Rosen's Table 133.1 for original comparison table

6) Describe the diagnostic features of heatstroke

• Heatstroke: diagnosis (Box 133.5)

- Exposure to heat stress, endogenous or exogenous
- Signs of severe CNS dysfunction (coma, seizures, delirium)
- Core temperature usually >40.5C, but may be lower
- Hot skin common, and sweating may persist



- Marked elevation of hepatic transaminase levels
- *Any type of CNS dysfunction can herald heat stroke: bizarre behaviour, opisthotonos, hallucinations, rigidity, cerebellar dysfunction
- *Hepatic damage is consistently featured in heatstroke. Look for elevated AST and ALT in the patient with altered mental status

7) List 5 cooling measures for heatstroke + 3 adjuncts to therapy

Immediate cooling is the cornerstone of treatment. Patients who present to the hospital with heatstroke have high mortality rates ranging from 21% to 63%, and mortality increases significantly when cooling is delayed. **-Rosen's Chapter 133**

Key management: (one of two options!)

- 1. Evaporative cooling
 - a. Strip all clothing, spray tepid water on patient, have fans blow air continuously over the patient
- Immersion ice water cooling = fastest way of dropping a person's body temperature (10-40 mins)

a. Adds challenges to resuscitation!

Stop cooling once the temperature reaches 39 deg.

There are some other "adjuncts" to the cooling process, but aren't as effective at dropping the body temperature rapidly - which is why they are call adjunctive!

- Application of ice packs to high heat transfer areas (eg, neck, groin, axillae) is commonly used.
- Cooling blankets may be a useful adjunct but will not produce rapid cooling if used exclusively.
- Cold irrigant gastric or rectal lavage will not provide significant heat exchange if used as the primary cooling modality.

In addition to cooling, there are other adjunctive therapies that need to be performed for these very sick patients with heat stroke:

- Resuscitation of the ABC's
 - Securing the airway (aspiration risk, control oxygenation and ventilation)
 - Targeted volume resuscitation
 - Some patients are profoundly hypotensive and volume deplete, while others can have right sided heart failure or pulmonary edema - so reassess and resuscitate prn
 - Hemodynamic resuscitation: focused on cooling
 - A variety of tachyarrhythmias commonly occur during heatstroke. These usually resolve with cooling, and electrical cardioversion should be avoided until the myocardium is adequately cooled. The use of α-



adrenergic agents such as norepinephrine is not recommended because they promote vasoconstriction without improving cardiac output or perfusion, decrease cutaneous heat exchange, and may exacerbate ischemic renal and hepatic damage. Atropine and other anticholinergic drugs that inhibit sweating should be avoided.

- Treat metabolic complications:
 - NO acetaminophen or ASA
 - Maintain urine output 2 ml/kg/hr especially with rhabdomyolysis
 - IV benzodiazepines for excessive shivering or seizures

Antipyretics are NOT on this list – they only work with febrile illnesses and NOT environmental illness

Cooling modalities to lower body temperature in heatstroke

- Preferred
 - Evaporative cooling with large circulating fans and skin wetting ice water immersion
- Adjuncts
 - Ice packs to axillae and groin
 - Cooling blanket
 - Peritoneal lavage (unproven efficacy in humans)
 - Rectal lavage
 - Gastric lavage
 - Cardiopulmonary bypass

8) List 8 DDx for hyperthermia/hyperthermia (*îtemp* and altered)

Differential diagnosis of heatstroke

- CNS haemorrhage
- Toxins, drugs
- Seizures
- Malignant hyperthermia
- Neuroleptic malignant syndrome
- Serotonin syndrome
- Thyroid storm
- High fever, sepsis
- Encephalitis, meningitis

Examples include

- Cerebral malaria, typhoid fever, typhus
- Graves' disease or thyroid storm



- Anticholinergic poisoning (note that they should have mydriasis*)
- Overdose of sympathomimetics, stimulants (amphetamines, cocaine, PCP)
- ASA or plavix overdose
- Dietary supplement use in a hot environment ephedrine or ergogenic aid creatine
- NMS: muscle rigidity, severe dyskinesia or akinesia, hyperthermia, tachycardia, dyspnea, dysphagia, and urinary incontinence.
- Serotonin syndrome: mental status changes, autonomic hyperactivity, and neuromuscular abnormalities secondary to increased CNS serotonergic activity (clonus).
- Delirium tremens
- Hypothalamic hemorrhage

Wisecracks

1) What is meant by wet bulb globe tempearture?

"The wet bulb globe temperature heat index is an excellent meteorologic measure of environmental heat stress. It includes the effects of temperature, humidity, and radiant thermal energy from the sun.

When climatic conditions exceed 25°C (77°F) wet bulb, even healthy people are at high risk during

exercise. Above 28°C (82.4°F), exercise and strenuous work should be avoided or limited to extremely short periods.

WBGT = 0.1 x Dry Bulb Temperature (DBT) + 0.7 x Wet Bulb Temperature (WBT) + 0.2 x Globe Temperature (GT)

DBT represents the ambient air temperature, WBT the relative humidity, and GT the radiant heat. <u>The equation for the WBGT reflects the critical importance of evaporative cooling for</u> <u>managing heat stress</u>, as judged by the relative weight given to WBT. Measurements to determine the WBGT should be obtained about three to four feet off the ground on the playing field where the training session or sporting event will take place.

Given the close association between WBGT and exertional heat illnesses, this measurement should be used to guide and modify the intensity and duration of exercise, the use of equipment (eg, football helmets and padding), the frequency of rest breaks, and hydration needs. Any person or group responsible for these kinds of decisions should establish an accurate method for determining WBGT on site and should not rely upon local weather stations or news reports. However, it is acceptable to use WBGT measurements performed regularly by experts within close proximity (approximately 10 miles or 16 km) of the site of athletic activity (eg, WBGT calculated daily by local airport meteorologists)."

Uptodate



The heat strain index is widely accepted as an example of an index that includes environmental and physiologic factors. There are several variations and modified heat strain indices, with varying ease of use and accuracy.

Cooling is best achieved by evaporation from the body surface; sweat that drips from the skin does not cool the body. Each liter of completely evaporated sweat dissipates 580 kcal of heat. The ability of the environment to evaporate sweat is termed atmospheric cooling power and varies primarily with humidity, but also with wind velocity. **As humidity approaches 100%, evaporative heat loss ceases.**

- Rosen's 9th Edition, Chapter 133 (see Rosen's Box 133.1)

2) List aetiologies of hyperthermia

- Fever = your body raises the set-point "the thermostat increases"
 - Elevated levels of prostaglandin E2 (PGE2) in the hypothalamus appear to be the trigger for raising the set-point. Once the hypothalamic set-point is raised, this activates neurons in the vasomotor center to commence vasoconstriction and warm-sensing neurons to slow their firing rate and increase heat production in the periphery.

• Hyperthermia = your thermostat is set at 36 degrees, but your house is on fire

- Despite your body trying to lose as much heat as possible, it is unable to dissipate the heat or some external factor is overwhelming the system.
- Aetiologies
 - Environmental
 - Heat stroke
 - o Exertional
 - o Classic
 - Excited delirium
 - Endocrine:
 - Hyperthyroidism
 - Drugs**
 - Ecstasy
 - Serotonin syndrome
 - Malignant hyperthermia
 - ASA overdose
 - Iron overdose
 - DNP

Although the vast majority of patients with elevated body temperature have fever, there are a few instances in which an elevated temperature represents hyperthermia. These include heat stroke syndromes, certain metabolic diseases, and the effects of pharmacologic agents that



interfere with thermoregulation. In contradistinction to fever, the setting of the thermoregulatory center during hyperthermia remains unchanged at normothermic levels, while body temperature increases in an uncontrolled fashion and overrides the ability to lose heat. Exogenous heat exposure and endogenous heat production are two mechanisms by which hyperthermia can result in dangerously high internal temperatures. **... Rosen's 9th Edition, Chapter 119**

3) List six types of heat-related illnesses

This is a review from today:

- Minor
 - Heat rash
 - Heat cramps
 - Heat syncope
 - Heat edema
- Major
 - Heat exhaustion
 - Heat stroke
 - Exertional vs. Classic

*classic heatstroke include predisposing factors or medication, older population, sedentary lifestyle, anhidrosis, normoglycemia, mild coagulopathy, mild elevation in creatine kinase level, oliguria, mild acidosis, and occurrence during heat waves.

Diaphoresis, hypoglycemia, disseminated intravascular coagulation, and marked lactic acidosis are characteristics of exertional heatstroke." - Rosen's 9th Edition, Chapter 133

4) Describe the pathophysiology of heat exhaustion

"Heat exhaustion is characterized by the inability to maintain adequate cardiac output due to strenuous physical exercise and environmental heat stress.

The body's ability to combat heat stress is overwhelmed - evaporation, conduction, convection, radiation - through vigorous activity, fitness level deficits, poor acclimatization to heat, heavy clothing/equipment, and compromised physiologic response (eg, degree of tachycardia).

This leads to Uncompensated heat stress (UCHS): when cooling capacity is exceeded and the athlete cannot maintain a steady temperature. Continued exertion in the setting of UCHS increases heat retention, causing a progressive rise in core body temperature and increasing the risk for severe heat illness.

The clinical criteria for heat exhaustion generally include the following:

•Athlete has obvious difficulty continuing with exercise

•Core body temperature is usually 101 to 104°F (38.3 to 40.0°C) at the time of collapse

•No significant dysfunction of the central nervous system (eg, seizure, altered consciousness, persistent delirium) is present



If any central nervous system dysfunction develops (eg, mild confusion), it is mild and resolves quickly with rest and cooling.

At risk medications:

- Anticholinergic agents
- Antiepileptic agents
- Antihistamines
- Decongestants
- Phenothiazines
- Tricyclic antidepressants
- Amphetamines
- Ergogenic stimulants (eg, ephedrine, dimethylamylamine)
- Lithium
- Diuretics
- Beta blockers
- Ethanol

- Uptodate

5) Describe the pathophysiology of heat stroke

"Large environmental heat load that can't be dissipated.

As we mentioned earlier there are two main forms of heat stroke:

- Classic form:
 - These elderly patients (usually >70 yrs) have a chronic illness that impairs their body's thermoregulation (they can't vasodilate, lose heat via sweating or increase their cardiac output); they are unable to get out of a hot environment/cool themselves/remain hydrated
 - Some young patients with psychiatric illness (clozapine use chronically) or drug addiction (ETOH and cocaine) are at risk for heat stroke.
- Exertional form
 - Occurs in otherwise healthy, usually young patients who exercise intensely in high temperatures and humidity. Remember that >75% humidity = heat can't be efficiently lost via evaporation.
 - Most of these patients have some risk factors for exertional heat illness

Temperature elevation is accompanied by an increase in oxygen consumption and metabolic rate, resulting in hyperpnea and tachycardia. Above 42°C (108°F), oxidative phosphorylation becomes uncoupled, and a variety of enzymes cease to function. A cytokine-mediated systemic inflammatory response develops, and production of heat-shock proteins is increased. Blood is shunted from the splanchnic circulation to the skin and muscles, resulting in gastrointestinal ischemia and increased permeability of the intestinal mucosa. Hepatocytes, vascular endothelium, and neural tissue are most sensitive to increased core temperatures, but all



organs may ultimately be involved. In severe cases, patients develop multi-organ system failure and disseminated intravascular coagulation (DIC)" - From UptoDate

Risk factors for poor outcomes in heat stroke:

- Elderly
- Hypotension
- Coagulopathy / DIC
- Need for intubation

6) How do you calculate free water deficit?

Total body water (TBW) refers to the estimated TBW, which is normally approximately 60 and 50 percent of lean body weight in younger men and women, respectively, and approximately 50 and 45 percent of lean body weight in older adult men and women, respectively.

Thus, in a nonobese, middle-aged, 60 kg woman with a serum sodium concentration of 168 mEq/L, TBW is approximately 40 percent of body weight, and the water deficit can be approximated from:

Uptodate

7) What are the targets of cooling?

In heat stroke the goal is to get to 39 degrees C.

Once you reach that target - we stop cooling so we don't "overshoot" and make the patient hypothermic.