REPRESENT A SPECTRUM OF DISORDERS, INCLUDING HEAT CRAMPS, HEAT SYNCOPE, HEAT EXHAUSTION AND HEAT STROKE.

• The differentiation is somewhat blurred & patients can rapidly progress from relatively benign to life-threatening disease.

HEAT STROKE IS SEEN LESS AMONG PERSONS WHO LIVE IN COUNTRIES WHERE HOT SUMMERS ARE COMMON BECAUSE OF PHYSIOLOGICAL ACCLIMATISATION

## PATHOPHYSIOLOGY:

#### **MECHANISMS OF HEAT TRANSFER:**

Body temperature is regulated through the delicate balance of HEAT PRODUCTION, ACCUMULATION AND DISSIPATION

METHODS OF DISSIPATION:

- RADIATION;
  - transfer by electromagnetic waves from warmer object to cooler one
- CONDUCTION;
  - heat exchange between two surfaces in direct contact
- CONVECTION;
  - heat transfer by air or liquid moving across the surface of an object
- EVAPORATION;
  - heat loss by vaporisation of water

When temperature is <35C, radiation accounts for  $\sim$ 60% of heat dissipation and evaporation for 30%

EFFECT OF WIND ON HEAT LOSS --> approximately proportional to square root of the wind velocity

When temperature rises to >35C, the body can no longer radiate heat to the environment and becomes dependent on evaporation for heat transfer.

AS HUMIDITY INCREASES, THE POTENTIAL FOR EVAPORATIVE HEAT LOSS DECREASES

• Hence the combination of HIGH TEMPERATURE AND HIGH HUMIDITY blocks the two main physiologic mechanisms that the body uses to dissipate heat.

## **RESPONSE TO HEAT STRESS:**

- Native thermal regulation mechanisms begin to fail at core temperatures >35-40C
  - It is possible to maintain a core temperature of 40-42C for short periods without adverse effects

# • PHYSIOLOGIC RESPONSE TO HEAT STRESS OCCURS THROUGH FOUR PRIMARY MECHANISMS:

- Dilatation of blood vessels
  - Especially in the skin
- Increased sweat production
- Decreased heat production
- Behavioural changes --> find a cool environment
- As body temperature rises, the sympathetic outflow of the posterior hypothalamus is inhibited;
  - unopposed anterior hypothalamic sympathetic outflow
  - decreased vascular tone throughout the body, especially in the cutaneous circulation
  - substantial CV stress from this response, because CO increases by ~3L/minute for each 1C above core temperature
  - · Can result in myocardial ischaemia, arrhythmias & CHF

#### ACCLIMATISATION:

- Human body has incredible capacity to respond to heat stress
- Primary acclimatisation involves changes in onset and volume of sweating, improvement in cutaneous vascular flow and overall CV function (Can be achieved over 7 days)

#### PATH TO HEAT INJURY:

- Heat exhaustion and heat stroke occur when the body's thermoregulatory responses are impaired or OVERWHELMED and are no longer capable of maintaining homeostasis
- Excessive heat is DIRECTLY TOXIC TO CELLS;
  - Causes an acute phase reaction and release of inflammatory cytokines, heat-shock proteins & stress proteins.
  - Proteins are denatured & cellular processes are interrupted.
  - Damages vascular endothelium
- Massive activation of clotting-cascade occurs --> DIC.
- Classic heat injury occurs during high environmental heat stress.
  - Takes hours --> days.
- HIGH RISK POPULATIONS:
  - Elderly
  - The young
  - Psychological, physiologic & pharmacologic impairments.
- Exertional heat injury usually affects individuals who are physically fit and participating in athletic events or performing jobs under conditions of high heat stress (military, firefighters)
- Without an efficient cooling mechanism, progressive dehydration and hyperpyrexia continue to the level of CARDIOVASCULAR & METABOLIC FAILURE.

# **CLINICAL FEATURES AND TREATMENT:**

## HEAT OEDEMA:

- Self-limited. Mild swelling of feet, ankles and hands that appears in first few days of hot weather.
- Due to cutaneous vasodilatation and orthostatic pooling of interstitial fluid
- Oedema is exacerbated by heat-induced release of ADH and aldosterone
- No special treatment is necessary. Diuretics not effective

## **PRICKLY HEAT:**

- A pruritic, maculopapular, erythematous rash over normally clothed areas (aka. heat rash)
- It is an acute inflammation of the sweat ducts caused by blockage of the sweat pores by macerated stratum corneum
- · Wearing clean, light and loose-fitting clothing is the cure



## HEAT CRAMPS:

- Painful, involuntary and spasmodic contractions of skeletal muscles, usually those of the calves (can effect thighs & shoulders)
- Usu. occur in those who are SWEATING PROFUSELY and replace fluid losses with water or other hypotonic solutions
- Most often occur during a rest period after several hours of vigorous physical activity.
- Do not involve enough muscle mass to cause rhabdomyolysis.
- Check electrolytes: May have hyponatraemia & hypokalaemia.
- TREATMENT: fluid and salt replacement (PO or IV) and rest in a cool environment. Consider sports drinks.

## HEAT TETANY:

- Consists of typical hyperventilation resulting in:
  - · Respiratory alkalosis
  - · Paraesthesia of the extremities
  - Circumoral paraesthesia
  - Carpopedal spasm
- Remove from heat and decrease the RR.

#### **HEAT SYNCOPE:**

- A variant of postural hypotension & results from the cumulative effect of:
  - VOLUME DEPLETION
  - PERIPHERAL VASODILATATION
  - DECREASED VASOMOTOR TONE
- Occurs early in heat exposure
- Elderly most at risk.
- Exclude metabolic, CV and neurologic disorders that cause syncope.
- Remove from heat and rehydrate.

#### **HEAT EXHAUSTION:**

- Occurs in two different ways --> water depletion or sodium depletion, but is OFTEN A COMBINATION OF BOTH
- Presents with headache, N+V, malaise, dizziness and muscle cramps as well as signs of dehydration such as tachycardia and orthostatic hypotension.
- DIAGNOSIS OF EXCLUSION
- On exam; temperature may be normal or elevated, BUT NOT >  $40^{\circ}$ C.
  - Patients DO NOT MANIFEST CNS IMPAIRMENT !
- Lab studies;
  - Haemoconcentration, specific electrolyte abnormalities depending
     on fluid and electrolyte losses to intake
  - No fluid intake --> hypernatraemia
  - Partially rehydrate with salt-containing solutions --> isotonic hypovolaemia
  - Hyponatraemia if rehydration with hypotonic solution
- TREATMENT:
  - Volume and electrolyte replacement and removal from heatstressed environment
  - If patients do not respond to 30 minutes of fluid replacement, aggressive cooling is mandated.

#### Table 204-1 Signs and Symptoms of Heat Emergencies

Heat Cramps	Heat Exhaustion	Heat Stroke
Muscle cramps Normal to mildly elevated temperature Sweating	Symptoms seen in heat cramps plus: Normal to elevated temperature [<40°C (<104°F)] Nausea, vomiting, headache, malaise, dizziness Orthostatic hypotension	Symptoms seen in heat exhaustion plus: Elevated temperature [>40°C (>104°F)] Neurologic abnormalities: inappropriate behavior, confusion, delirium, ataxia, coma, seizures Anhidrosis or sweating

## **HEAT STROKE:**

An acute, life-threatening emergency with mortality rates as high as 30-80% and is UNIVERSALLY FATAL IF LEFT UNTREATED

## CARDINAL FEATURES OF HEAT STROKE:

- HYPERTHERMIA (> 40\*C)
- ALTERED MENTAL STATUS
  - Absence of sweat is NOT DIAGNOSTIC as sweat is present in over half patients with heat stroke.
- · CNS is particularly vulnerable to heat stroke, esp the CEREBELLUM
  - Ataxia is an early finding
  - Virtually any neurologic abnormality can be ascribed to heat stroke
  - SEIZURES ARE QUITE COMMON (esp. during cooling)
  - Neurologic injury is a function of maximum temperature reached and the duration of exposure
  - Can progress to plantar responses, decorticate and decerebrate posturing, hemiplegia, status epilepticus and coma
- Medications often interfere with heat-removal mechanisms
  - Anticholinergic drugs, diuretics, phenothiazines, β-blockers, CCB & sympathomimetic agents are most notable
- IMMEDIATE COOLING AND SUPPORT OF ORGAN SYSTEM FUNCTION IS THERAPEUTIC
  - A delay in cooling increases the mortality rate
- Evaluation needs to EXCLUDE OTHER PROCESSES:
  - See DDx list below.

Table 204-2 Differential Diagnosis of Heat Stroke	
	Neurologic
Infectious	Hypothalamic bleeding or infarct
Sepsis syndrome	Cerebrovascular accident
Meningitis	Status epilepticus
Encephalitis	Toxicologic
Malaria	Anticholinergic toxidrome
Typhoid	Sympathomimetic overdose
Tetanus	Salicylate overdose
Endocrine	Serotonin syndrome
Thyroid storm	Malignant hyperthermia
Pheochromocytoma	Neuroleptic malignant syndrome
Diabetic ketoacidosis	Withdrawal syndromes—alcohol and benzodiazepine withdrawal

#### Table 204-2 Differential Diagnosis of Heat Stroke

#### LABORATORY EVALUATION:

- Diagnostic studies are directed toward detecting end-organ damage & excluding other disease processes
- ABG, CK, coagulation profile, ECG, CXR
- Patients with exertional heat stroke often have a lactic acidosis
- Hypoglycaemia may occur

#### TREATMENT OF HEAT STROKE:

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Goals of therapy are IMMEDIATE COOLING AND SUPPORT OF
ORGAN SYSTEM FUNCTION
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PREHOSPITAL;

- Remove from the environment while standard resuscitation measures are undertaken.
- Cooling with removal of clothing and spray with warm water and place wet towels or sheets over the patient's body or ice packs over groin, neck and axilla.

ED MANAGEMENT;

- IV fluids that ensures adequate urine output.
- Check glucose on arrival.
- COOLING TECHNIQUES:
  - ONLY PHYSICAL METHODS (NO ROLE FOR ANTIPYRETICS)
     They may be deleterious, dantrolene has no role.
- With all cooling methods (summarised below), the goal is to reduce the core temperature to 39C and then stop to avoid overshoot hypothermia

Table 204-3 Summary of Cooling Techniques				
Cooling Method	Advantages	Disadvantages	Recommendations	
Evaporative methods	Provides effective cooling	Can cause shivering	Strongly recommended	
	Readily available	Less effective in humid environments		
	Practical	Makes it difficult to maintain electrode positions		
	Well tolerated			
Ice water immersion	Provides effective cooling	Can cause shivering	Recommended	
		Poorly tolerated		
		Not compatible with resuscitation settings		
Ice packs on neck, axillae, and groin	Practical	Has limited cooling efficacy	Can be used as adjunct cooling method	
	Can be added to other cooling methods	Poorly tolerated		
Cardiopulmonary bypass	Provides fast and effective cooling	Invasive	Recommended in severe or resistant cases when available	
		Not readily available		
		Setup is labor intensive		
Cooling blankets	Easy to apply	Has limited cooling efficacy	Not recommended when other methods available	
		Impedes use of other cooling methods		
Cold water gastric, urinary bladder, rectal, or	ectal, or —	Invasive	Not recommended	
peritoneal lavage		Labor intensive		
		May lead to water intoxication		
		Human experience is limited		
Cold water IV infusion	-	Carries unjustified complication rate	Not recommended	

- Use warm water when spraying, as when the skin temperature drops below 30C, shivering will resulting more heat production and peripheral vasoconstriction will impair evaporation
- Cold water urinary bladder lavage and rectal lavage are adjuncts but are labour intensive and have the potential for water intoxication
- Bypass is invasive and is reserved for refractory cases

Table 204-4 Complications of Heat Stroke				
	Early	Late		
Vital signs	Hypotension	_		
	Hypothermic overshoot			
	Hyperthermic rebound			
Muscular	Rhabdomyolysis	_		
Neurologic	Delirium/coma	Cerebral edema		
	Seizure	Encephalopathy		
		Persistent neurologic deficit		
Cardiac	Heart failure	Myocardial injury		
Pulmonary	Pulmonary edema	Acute respiratory distress syndrome		
Renal	Oliguria	Renal failure		
GI	_	Intestinal ischemia or infarction		
		Pancreatic injury		
		Hepatic dysfunction		
Metabolic	Hypokalemia	Hyperkalemia		
	Hypernatremia	Hypocalcemia		
	Hyponatremia	Hyperuricemia		
Hematologic	_	Thrombocytopenia		
		Disseminated intravascular coagulation		

# TREATMENT OF COMPLICATIONS OF HEAT STROKE:

- Both early and late complications can occur with heat stroke.
- HYPOTENSION is common initially.
  - Usually responds to IV fluid bolus.
  - Inotropes that cause severe vasoconstriction impede cooling by redirecting blood flow away from the skin
    - use DOBUTAMINE INSTEAD.
- FLUID & ELECTROLYTE ABNORMALITIES
  - Vary depending on type of onset and duration of the disorder and underlying disorders/medications
- DEHYDRATION AND VOLUME DEPLETION
  - May not occur in classic heat stroke but are common findings in heat exhaustion
  - Hypokalaemia due to total-body depletion
  - Hypernatraemia --> free water deficit.
  - Hyponatraemia can be seen in those who hydrate with oral hypotonic solutions
- ABNORMAL HAEMOSTASIS
  - Purpura, petechia and haemorrhage.
  - Occurs due to thermal deactivation of plasma proteins resulting in a decreased level of clotting factors, DIC and fibrinolysis
- THERMAL INJURY TO THE LIVER is common
  - Peaks at 24-72 hours, but does not result in jaundice frequently.
  - CENTRILOBULAR NECROSIS.
  - Glucose homeostasis and clotting are affected. Almost always reversible with a full recovery expected.
- RENAL FAILURE
  - Direct thermal injury, rhabdomyolysis or volume depletion
  - Manifest with oliguria.
  - Early, aggression volume expansion is the key to avoidance.
- ADULT RESPIRATORY DISTRESS SYNDROME
  - May occur and require respiratory support while cooling occurs.
- MYOCARDIAL INJURY
  - Cardiac muscle injury, low cardiac output, with falling cardiac index and hypotension is associated with a poor prognosis
- SEIZURES.
  - May occur during cooling and can be controlled with benzodiazepines.
  - Persistent neurologic deficits occur in ~20% patients and are associated with high mortality