Heat illnesses are among the leading causes of morbidity and mortality during Hajj, particularly in summer. Temperatures in Makkah can rise higher than 45ºC. Lack of acclimatization, high intensity physical exertion poor physical fitness, and exposed spaces produce heat illnesses in many pilgrims. Accordingly, Clinicians, who care for pilgrims with severe heat illness, need to be aware of the basic physiologic principles of thermoregulation, the spectrum of heat illness, strategies for prevention, careful evaluation at the field and hospital, rapid cooling, and must remain with high clinical suspicion to detect developing complications, and treat them as early as possible.

HEAT STROKE

Definition:

Heat stroke is defined as a core body temperature usually in excess of 40ºC. (104ºF) with associated central nervous system dysfunction in the setting of a large environmental heat load that cannot be dissipated.

There are two types of heat stroke:

- **Classic (nonexertional) heat stroke** – Classic heat stroke affects elderly individuals with risk factors that impair thermoregulation, prevent removal from a hot environment, or interfere with access to hydration or attempts at cooling.
- **Exertional heat stroke** – Exertional heat stroke generally occurs in young, otherwise healthy individuals who engage in heavy exercise during periods of high ambient temperature and humidity.
Pathophysiology:

Body temperature is maintained within a narrow range by balancing heat load with heat dissipation. The body's heat load results from both metabolic processes and absorption of heat from the environment. As core temperature rises, the preoptic nucleus of the anterior hypothalamus stimulates efferent fibers of the autonomic nervous system to produce sweating and cutaneous vasodilation.

Evaporation is the principal mechanism of heat loss in a hot environment, but this becomes ineffective above a relative humidity of 75 percent. The other major methods of heat dissipation—radiation (emission of infrared electromagnetic energy), conduction (direct transfer of heat to an adjacent, cooler object), and convection (direct transfer of heat to convective air currents)—cannot efficiently transfer heat when environmental temperature exceeds skin temperature.

As the temperature reach 42°C. The cells of the body start to breakdown and lose their functional capability, resulting in multi-organ system failure and disseminated intravascular coagulation (DIC).

Risk factors:
Conditions that impair thermoregulation, prevent removal from a hot environment, or interfere with access to hydration or attempts at cooling, these conditions include:

- Extremes of age, especially pilgrims above 65 years old.
- Cardiovascular disease or hypertension.
- Diabetes.
- Neurologic, psychiatric disorders or Central nervous system medications.
- Liver diseases.
- Kidney diseases or diuretics.
- Obesity.
- Anhidrosis.
- Physical disability.
- The use of recreational drugs, such as alcohol or cocaine.
- Anticholinergic agents.
- Lack of acclimatization.
Acclimatization:
Acclimatization is the body’s ability to improve its response and tolerance of heat stress over time, and it is the most important factor that determines how well an athlete withstands extreme heat. Thus, allowing sufficient time and using optimal training strategies that enable pilgrims to acclimatize are critical for improving performance and mitigating the risk for exertional heat illnesses. Observational studies have found that the first week of athletic practice in high heat and humidity is the period of greatest risk for developing exertional heat illnesses. Full acclimatization requires at least 10 to 14 days of exercise at an intensity that raises body temperature to at least 38.51°C for at least 60 minutes. This can be accomplished in either hot environmental conditions or cooler conditions if clothing or equipment is worn and exercise intensity is high. However, any improved tolerance of heat stress generally dissipates within 2 to 3 weeks of returning to a more temperate environment. The major physiologic adjustments that occur during heat and humidity acclimatization include:
1. Plasma volume expansion.
2. Improved cutaneous blood flow.
3. Lower threshold for initiation of sweating.
4. Increased sweat output.
5. Lower salt concentration in sweat.
6. Lower skin and core temperatures for a standard exercise.

These adaptations allow for better dissipation of heat during exertion and limit increases in body temperature compared with pilgrims who have not acclimatized.

Risk factors for increased mortality:

Patients who present to the hospital with heat stroke have high mortality. Which is directly correlated to the duration of core temperature elevation, time to initiation of cooling measures, and the number of organ systems affected.

Diagnosis:

Clinical features:

- **History:**
  - History of exposure to severe environmental heat.
  - If they can respond coherently, patients with heat stroke may complain of weakness, lethargy, nausea, or dizziness.

- **Physical findings:**
  - Elevated core body temperature (generally >40ºC [104ºF]), some patients with heat stroke may not exceed 40ºC, particularly if cooling measures were initiated prior to the patient's arrival at the hospital, A thermometer (rectal or esophageal) that is accurate at high temperatures must be used when assessing heat stroke patients.
  - Central nervous system dysfunction (eg, altered mentation, slurred speech, irritability, inappropriate behavior, agitation, ataxia and other signs of poor coordination, delirium, seizures, and coma).
  - Lack another explanation for their hyperthermia (eg, infection).
  - Vital sign abnormalities (eg, tachycardia, tachypnea, hypotension).
  - Other physical findings may include flushing (cutaneous vasodilation), diarrhea, crackles due to noncardiogenic pulmonary edema and aspiration pneumonia.
  - The skin may be moist or dry, depending upon underlying medical conditions, the speed with which the heat stroke developed, and hydration status, not all victims of heat stroke are volume-depleted.
  - The presentation of elder adults with heat stroke may be subtle and nonspecific early in the course of the disease.
  - The severity of a heat illness may not be apparent during the initial presentation.
Frequently encountered complications include acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), acute kidney injury (ie, acute renal failure), hepatic injury, hypoglycemia, and rhabdomyolysis.

Differential diagnosis of heat stroke:

The differential diagnosis of severe hyperthermia is extensive and includes infectious, endocrine, central nervous system, toxic, and oncologic etiologies.

Diagnostic evaluation:

No single diagnostic test definitively confirms or excludes heat stroke. Furthermore, laboratory study abnormalities may overlap in patients with heat stroke and with hyperthermia due to other conditions.

- Continuous monitoring of rectal temperature should be obtained in all patients suspected of heat stroke.
- A chest radiograph may demonstrate pulmonary edema.
• The electrocardiogram may reveal dysrhythmias, conduction disturbances, nonspecific ST-T wave changes, or heat-related myocardial ischemia or infarction.
• Laboratory studies to obtain in the patient with heat stroke Including but not limited to Complete blood count, serum electrolyte concentrations, Arterial or venous blood gas, serum lactate, blood urea nitrogen (BUN) and creatinine concentrations, liver function test, cardiac enzymes, Prothrombin time (PT) and partial thromboplastin time (PTT), septic screening. the common laboratory features :
  o Leukocytosis may reach as high as 30,000 to 40,000/mm³.
  o Hypokalemia, hyponatremia, hyperglycemia,
  o Metabolic acidosis and respiratory alkalosis are the most common abnormalities.
  o Acute renal failure: high urea and creatinine with hemogranular casts and proteinuria.
  o Features of Rhabdomyolysis: (eg, serum creatine kinase, urine myoglobin) and its complications (eg, hypocalcemia, hyperphosphatemia, myoglobinuria, and BUN and creatinine). Myoglobinuria should be suspected in any patient with brown urine supernatant that is heme-positive and clear plasma. Urinalysis may reveal other evidence of renal injury, including protein, blood, renal tubular casts, and increased specific gravity.
  o Heat-induced liver damage: Hepatic transaminase concentrations. Transaminase concentrations are rarely normal in patients with heat stroke; however, in patients with severe liver injury marked elevations may not appear for 24 to 48 hours.
  o DIC (low platelet, low Hb, high PT, high PTT, high D-Dimer (or FDP), low fibrinogen).
  o Toxicologic screening may be indicated if a medication effect is suspected. Drugs that may contribute to hyperthermia and for which tests are often available include ethanol, amphetamines, cocaine, salicylates, hallucinogens, and lithium.
  o A head CT and analysis of the cerebrospinal fluid should be performed as indicated if central nervous system causes of altered mental status are suspected.

Treatment:
Treatment Goals:
• Normal vital signs.
• Euhydration state.
• Full consciousness.
• Normal investigations.
• Avoid and treat complications.
Algorithm for Management of heat stroke

- Ensure airway, breathing and circulation
  - Mechanical ventilation if indicated
- Initiate resuscitative measures and evaporative cooling method immediately with continuous rectal temp monitoring, No antipyretics should be used.
- Chest x-ray, ECG and lab investigations including but not limited to (CBC, chemistry, ABG, lactate, LFTs, RFTs, cardiac enz., PT, PTT) to rule out other diagnosis and complications.
- Look for complications and treat them accordingly
- Stop cooling measures whenever rectal temp reaches 39°C

Resuscitative measures and monitoring:

Establish Airway and maintain Breathing:
- Tracheal intubation and mechanical ventilation are needed for patients unable to protect their airway or with deteriorating respiratory function to overcome the oxygen demand.

Circulation:
- Establish an IV line.
- Check blood pressure (BP), if low give IV bolus of NS 500 ml then maintenance IVF NS at 125 ml/hour.
- Central venous pressure (CVP) monitoring may be useful for assessing volume status and determining the need for fluid resuscitation. A target CVP of 8 to 12 mmHg is an appropriate target. However, CVP readings may be inaccurate if heat-related cardiac dysfunction develops (e.g., acute right heart failure). Alpha-adrenergic agonists should be avoided, since the resultant vasoconstriction decreases heat dissipation.
- Cardiac and hemodynamic monitoring.
- Foley’s catheter (to monitor urine output).
- Monitor: vital signs, O₂ saturation.
- Input output monitoring and charting.
Rapid cooling:

- For hyperthermia (Rectal temperature > 40º C): Start evaporative Cooling measures (Fan and body water spray). With evaporative cooling, the naked patient is sprayed with a mist of lukewarm water while fans are used to blow air over the moist skin. This Evaporative cooling is the method used most often to treat heat stroke because it is effective, noninvasive, easily performed, and does not interfere with other aspects of patient care. When used to treat elderly patients, evaporative cooling is associated with decreased morbidity and mortality.

- Other effective cooling methods are less commonly used in patients with heat stroke:
  - Special beds called body cooling units have been made for this purpose is an alternative method that allows greater access to the patient, in which the patient is placed supine on a porous stretcher, alternate warm and cold mist with air used till temperature reaches 39ºc.
  - Applying cold compresses to the glabrous (smooth, hairless) skin surfaces of the cheeks, palms, and soles.
  - Applying ice packs to the axillae, neck, and groin (areas adjacent to major blood vessels, but may be poorly tolerated by the awake patient.
  - Immersing the patient in ice water (cold water immersion) is an efficient, noninvasive method of rapid cooling, but it complicates monitoring and intravenous access, and may be harmful to elder patients.

- Continuous core temperature monitoring rectally and cooling measures should be stopped once a temperature less than 39ºc has been achieved in order to reduce the risk of iatrogenic hypothermia.

- Pharmacologic therapy — Pharmacologic therapy is not required in heat stroke. There is no role for antipyretic agents such as acetaminophen or aspirin in the management of heat stroke, since the underlying mechanism does not involve a change in the hypothalamic set-point and these medications may exacerbate complications such as hepatic injury or disseminated intravascular coagulation (DIC). Salicylates can contribute to hyperthermia by uncoupling oxidative phosphorylation. Dantrolene is ineffective in patients with severe temperature elevation not caused by malignant hyperthermia.

Exclude other causes of hyperthermia:

- Rapid improvement with active cooling suggests that heat stroke is the primary diagnosis. However, clinicians should investigate alternative causes for hyperthermia, and detect heat stroke complications as early as possible.

Treatment of complications:

Severe hyperthermia may lead to a wide range of complications. These often resolve as cooling measures take effect, but this depends upon the degree and duration of hyperthermia. Complications may include the following:
Respiratory dysfunction:
Patients with heat stroke often develop pulmonary complications, which can include aspiration, bronchospasm, noncardiogenic pulmonary edema, acute respiratory distress syndrome, pneumonitis, pulmonary infarction, and pulmonary hemorrhage. Tracheal intubation and mechanical ventilation are often necessary to protect the airway and to address increased metabolic demands (ie, provide supplemental oxygen and increased minute ventilation.)
- **Convulsions:**
  Seizures are common in patients with heat stroke. Initial treatment consists of short-acting benzodiazepines such as:
  - Midazolam 0.1-0.2 mg/kg IV, to a maximum dose 4 mg, has an onset of action one to five minutes and duration of action of one to six hours.
  - Lorazepam 0.1 mg/kg IV, to a maximum dose 4 mg, is a second-line option, as the duration of action may be prolonged from 12 to 24 hours.
  - Barbiturates should be avoided.
  - Rapid cooling measures.

- **Arrhythmia and cardiac dysfunction:**
  Potential cardiac complications include acute decompensated heart failure and myocardial injury associated with reversible cardiac biomarker increase and ST-segment changes on electrocardiogram (ECG). The biomarker and ECG changes are believed to be caused by an increase in catecholamine levels due to heat stroke, causing a stress-induced cardiomyopathy. Other ECG abnormalities that have been reported in association with heat stroke include sinus tachycardia and other tachyarrhythmias, conduction abnormalities, prolonged QT interval, transient Brugada pattern, and nonspecific ST-T changes.
  - Rapid cooling is essential; cardiac dysfunction and tachyarrhythmias generally resolve with cooling.
  - Antiarrhythmics are seldom necessary, and electrical cardioversion should be avoided until cooling is achieved, unless necessary to treat ventricular defibrillation or pulseless ventricular tachycardia.

- **Hypotension:**
  Hypotension associated with heat stroke results from peripheral vasodilation, cardiac dysfunction, and volume depletion. Treatment consists of:
  - IV fluid boluses of isotonic crystalloid solution 250 to 500 ml, then monitor according to vital signs and urine output. Given the risk of pulmonary edema, excessive fluid administration should be avoided.
  - If no response, start Dopamine 3-10 µg/Kg/min and increase up to 20 µg/Kg/min according to vital signs and CVP measurement.
  - Avoid Alpha-adrenergic agonists, which impairs cooling due to vasoconstriction.

- **Bleeding:**
  Disseminated intravascular coagulation (DIC) can develop during the first three days of illness and coagulation studies should be monitored during this period. Replacement of:
  - Lost blood with Packed RBCs.
  - Clotting factors with fresh frozen plasma and platelets. Cryoprecipitate and fibrinogen may be necessary.

- **Acute renal failure:**
  Heat stroke can cause acute kidney injury. Renal function studies and serum electrolyte concentrations should be followed closely over the first few days of illness:
Renal replacement therapy or Hemodialysis for overload, may be needed.
Correction of hyperkalemia and acidosis.

- **Rhabdomyolysis:**
  The combination of muscle injury, volume depletion, and acute kidney injury can lead to rhabdomyolysis in patients with heat stroke.
  - R/O Rhabdomyolysis by CK and treat with IV fluid.

- **Acid base and electrolytes imbalance:**
  Metabolic acidosis and respiratory alkalosis are the most common abnormalities.
  - Correct and replace accordingly.

- **Hepatic injury:**
  Hepatic injury due to heat stroke is generally self-limited but in some cases may progress to acute liver failure, with a subset of patients requiring liver transplantation.

- **Hyperglycemia:**
  - Monitor RBS and K level.
  - No insulin initially unless patient is known to be diabetic.

- **Sepsis:**
  In cases where the etiology of the patient’s hyperthermia is unclear initially and infection remains a possibility, empiric administration of an initial dose of antibiotics, following collection of appropriate cultures, is prudent, while cooling measure is implemented.

- **Diarrhea:**
  - Only fluid replacement.

**Discharge criteria:**
- Normal vital signs.
- Fully conscious.
- Well hydrated.
- Normal investigations.
- Free of complications.

**Follow up:**
- Long-term outpatient therapy may be required when chronic renal failure develops and when irreversible damage to the CNS, heart, and liver occurs.
HEAT EXHAUSTION

Definition:
Heat exhaustion is defined as a heat illness with core body temperature elevation, usually less than 40ºC. (104ºF) with normal central nervous system function in the setting of strenuous physical exertion and environmental heat stress.

Pathophysiology:
Inability to maintain adequate cardiac output due to loss of salt and water, in unacclimatized patient.

Clinical features:
- **History:**
  - Most often Heat exhaustion manifests as physical collapse during exercise.
  - Body temperature elevation.
  - Headache, nausea, vomiting, dizziness, weakness and cramps.

- **Physical findings:**
  - Patient is presented with normal or increased core body (rectal) temperature < 40ºc., (milder than with heat stroke or heat injury).
  - Sweating, postural hypotension.
  - The central nervous system is not affected.

Investigations:
- If febrile; including but not limited to CBC, serum chemistry, septic screening and Chest x-ray.
- Common Laboratory Features; Hypokalemia, hyponatremia.

Treatment:
Goals: Normal vital signs, Euhydration state and Normal investigations.
- **Rest in a cool environment.**
  - Remove the patient from the hot place and move him to a shaded or air-conditioned area.
  - Place the patient supine and raise his legs.
  - Remove excess clothing and equipment.
  - Cool the patient until the patient starts shivering by running ice or cool water over him or using evaporative cooling measures.
  - Continuously observe and frequently monitor heart rate, blood pressure, respiratory rate, rectal temperature, and mental status.
- **Transport the patient** to an emergency department if rapid improvement does not occur despite appropriate treatment.
HEAT INJURY

Definition:
Exertional heat injury is defined as a progressive multisystem disorder with hyperthermia, less than 40ºC. (104ºF) following vigorous activity that is associated with end-organ damage (eg, kidney, liver, muscle) in the absence of significant neurologic injury.
Pathophysiology:
• Exertional heat injury is similar to Heat stroke, but the patient's central nervous system is not affected and core body temperature does not have to exceed 40°C.
• It is unlike heat exhaustion. There is clear evidence of end-organ injury such as rhabdomyolysis, acute kidney injury, disseminated intravascular coagulation, or acute liver failure.

Clinical features:
• History:
  o Most often Heat injury manifests as physical collapse during exercise.
  o Body temperature elevation.
  o May complain of weakness, lethargy or nausea.
  o No significant neurologic manifestation.
• Physical findings:
  o Patient is presented with normal or increased core body (rectal) temperature < 40°C.
  o Sweating, postural hypotension.
  o The central nervous system is not affected.
  o Lack another explanation for hyperthermia (e.g., infection).
  o Vital sign abnormalities (e.g., tachycardia, tachypnea, hypotension).
  o The severity of the heat injury may not be apparent during the initial presentation.
  o Frequently encountered complications include acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), acute kidney injury (i.e., acute renal failure), hepatic injury, hypoglycemia, and rhabdomyolysis.

Differential diagnosis:
The clinical distinction between heat injury and exertional heat stroke is made based upon a careful assessment of central nervous system dysfunction (e.g., seizure, altered mental status, abnormal behavior). In practice, this distinction is often made after the initial treatment of the patient including rapid cooling, and is based on a careful review of the event with the patient, witnesses, and other treating clinicians.

Management plan:
• Rapid cooling: using any of the methods suitable for heat stroke.
• Continuous core temperature monitoring rectally and cooling measures should be stopped once a temperature less than 39°C has been achieved in order to reduce the risk of iatrogenic hypothermia.
• Monitoring of vital signs and urine output.
• Initial care is largely supportive.
• Laboratory studies including but not limited to urinalysis, urine myoglobin and creatine kinase. Complete blood count, serum electrolyte concentrations, Arterial or venous blood gas, serum lactate, blood urea nitrogen (BUN) and creatinine concentrations, liver function tests (serum
HEAT SYNCOPE

Definition:
Heat syncope is a transient loss or near-loss of consciousness due to the indirect effects of high ambient temperature that generally occurs during the first few days when a pilgrim is exposed to high environmental temperatures, before acclimatization is complete.

Pathophysiology:
Exertion associated collapse in unacclimatized pilgrims, due to heat induced peripheral vasodilatation and pooling of blood, with subsequent loss of consciousness.

Clinical features:
- **History:**
  - Most often Heat syncope manifests as physical collapse during or after exertion, with a feeling of heaviness in the legs, Blurred vision, Confusion, Feeling warm or hot, Lightheadedness, dizziness, a floating feeling, Nausea, Sweating, Vomiting or Yawning.
- **Physical findings:**
  - There may be a drop in blood pressure and weak pulse.

Treatment:
- Move the patient to a shaded area.
- Have the patient lay supine in a cool environment.
- Raise the legs of the patient.
- Give fluids to drink.
- The patient should avoid sudden or prolonged standing until fully recovered.
The patient should recover within 15 to 20 minutes with these maneuvers; failure to improve should prompt further evaluation, including a rectal temperature. Patients at higher risk for dangerous causes or adverse outcomes and those who do not completely recover within 20 minutes should be evaluated in the emergency department using the approach for any patient with syncope.

HEAT OEDEMA

Definition:
Heat oedema is a condition characterized by dependent edema from vasodilatory pooling, due to the indirect effects of high ambient temperature that generally occurs during the first few days when a pilgrim is exposed to high environmental temperatures, before acclimatization is complete.

Pathophysiology:
- Heat in unacclimatized pilgrims induce peripheral vasodilatation and pooling of blood, with subsequent gravitational oedema of hands, feet and legs.

Clinical features:
- **History:**
  - Older adults and People visiting hot climates from colder climates have an increased risk of heat edema, especially if they have other medical conditions that affect their circulation.
- **Physical findings:**
  - Mild swelling of hands and feet.

Treatment:
- Move the patient to a shaded area.
- Have the patient lay supine in a cool environment.
- Raise the legs of the patient.
- Give fluids to drink.

Usually heat edema Disappears after acclimatization.

PRICKLY HEAT

Definition:
Prickly heat, also called miliaria, is a rash that can develop after a person sweats far more than usual and sweat glands become blocked.
Pathophysiology:
Heat rash begins with excessive perspiration, usually in a hot, humid environment. The perspiration makes it easier for dead skin cells and bacteria on the skin to block the sweat glands, forming a barrier and trapping sweat beneath the skin, where it builds up, causing the characteristic bumps. As the bumps burst and sweat is released, there may be a prickly, or stinging sensation that gives this condition its name.

Clinical features:
- Manifests as an itchy rash of small raised red spots with a prickling or stinging sensation.
- Usually affects parts of the body covered by clothes, such as the back, abdomen, neck, upper chest, groin or armpits.

Treatment:
- In most cases, heat rash will clear up on its own in a few days if the affected area is kept cool and dry.
- Advise the patient to avoid excessive heat and humidity and cool off with a fan.
- Advise the patient to take a cool shower or bath and let skin air dry.
- Avoid using any type of oil-based product, which might block sweat glands.
- Calamine lotion and/or hydrocortisone cream can relieve itching and irritation.
- If prickly heat does not go away within a few days, or if an infection developed give antibiotic (cloxacillin).

Prevention:
- It is best prevented by wearing light, loose clothing and avoiding heavy, continuous sweating by using fans and cool showers and baths to stay cool, or air conditioning if available.

SUNBURN

Definition:
Sunburn is defined as red, painful skin that feels hot to the touch due to exposure to sunshine.

Pathophysiology:
Sunburn usually appears within a few hours after too much exposure to ultraviolet (UV) light from sunshine. Many people don’t produce enough melanin to protect the skin well. Eventually, UV light causes the skin to burn, bringing pain, redness and swelling. Sunburn may take several days or longer to fade.
Clinical features:
Signs and symptoms of sunburn usually appear within a few hours after sun exposure:
- Pinkness or redness.
- Skin that feels warm or hot to the touch.
- Pain, tenderness or itching.
- Swelling.
- Small fluid-filled blisters, which may break.
- Headache, fever, chills and fatigue if the sunburn is severe.

Treatment:
- Pain relievers as MEPO gel or calamine lotion.
- Medications that control itching: skin corticosteroids, combined with pain relievers.

Prevention:
- Advise to avoid sun exposure between 10 a.m. and 4 p.m.
- Cover up with white clothes and umbrella.

Heat Cramps

Definition:
Heat cramps (which do not appear to be caused by increased ambient temperatures) are muscle cramps that occur during or after exertion.

Pathophysiology:
Exertion associated cramps in unacclimatized pilgrims, Due to fluid deficiencies (dehydration), electrolyte imbalances, neuromuscular fatigue, or any combination of these factors.

Clinical features:
- Painful involuntary muscle contraction, involving large muscles groups specially legs.
- Moist cool skin, a normal body temperature, and minimal distress.

Treatment:
- Relax in cool environment, stretch and massage the involved muscle to reduce acute discomfort.
- Hydrate the patient and replace sodium losses with oral salt solution, as in rehydration solutions, can be made by adding one fourth to one-half teaspoon of table salt to 1 L of water, to improve taste, add a few teaspoons of sugar and/or orange juice or lemon juice.
• IV isotonic saline therapy (rarely required). However, oral rehydration has consistently been shown to be as effective as IV rehydration when equal amounts of fluids are given.

Persistent or systemic cramping should prompt an assessment of the serum sodium to evaluate for exertional hyponatremia and raise the possibility of sickle cell crisis due to exertion.

**Prevention:**
Muscle cramps are thought to be prevented best through adequate conditioning, acclimatization, hydration, electrolyte replacement, and appropriate dietary practices.

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**EXERTION-ASSOCIATED HYPONATREMIA (EAH)**

**Definition:**
Exertion-associated hyponatremia (EAH) is defined by a serum or plasma sodium concentration below the normal reference range of 135 mmol/L that occurs during or up to 24 hours after prolonged physical activity (usually occur when activity exceeds 4 hours).

**Pathophysiology:**
Two mechanisms of Low serum-sodium levels are proposed:
- A combination of excessive fluid intake and inappropriate body water retention due to ingesting water or low-solute beverages well beyond sweat losses (also known as water intoxication).
- Sweat sodium losses are not adequately replaced.

Ultimately, the intravascular and extracellular fluid has a lower solute load than the intracellular fluids, and water flows into the cells, producing intracellular swelling that causes potentially fatal neurologic and physiologic dysfunction.

**Clinical features:**
- Affected pilgrims present with a combination of disorientation, altered mental status, headache, vomiting, lethargy, and swelling of the extremities (hands and feet), pulmonary edema, cerebral edema, and seizures.
- Excessive fluid intake with low sweat and urine losses.
- Exertional hyponatremia can result in death if not treated properly.

**Management plan:**
- In Hajj environments in which patient evacuation to definitive care is often greatly delayed. Rapid recognition and appropriate treatment are essential in the severe form to ensure a positive outcome. Failure in this regard is a recognized cause of event-related fatality.
It is important to differentiate Exertion-associated hyponatremia from Heat stroke, because the management is different, fluid therapy might kill the patient with Exertion-associated hyponatremia.

Algorithm for exertion-associated hyponatremia (EAH) field management:

Acute hospital assessment and management of EAH:
**Assessment:**
- Urgent measurement of blood sodium by the most rapidly available means.
- Assess for clinical signs suggestive of developing cerebral edema.
- Obtain and store specimens if possible for later analysis of blood serum osmolality and urine sodium and osmolality.
Management:
- Supplemental oxygen to maintain oxygen saturation above 95%
- Restrict fluids (both IV and oral) until onset of urination.
- Avoid IV normal saline until sodium correction is initiated.
- Thereafter normal saline may be required for hypovolemic shock or in renal protection therapy for rhabdomyolysis.
- In severe EAH (signs of cerebral edema or serum sodium less than 125 mmol/L) administer IV 3% hypertonic saline as a 100-mL bolus repeated twice at 10-minute intervals aiming to reverse cerebral edema.
- Aim to increase serum sodium by approximately 4 to 5 mmol/L or until neurological symptoms are reversed by active treatment, then allow the remaining correction to occur spontaneously via urinary free water excretion.

Prevention of Exertion-associated hyponatremia:
Exertion-associated hyponatremia can be prevented by matching fluid intake with sweat and urine losses and by rehydrating with fluids that contain sufficient sodium.

Preventive measures for heat illnesses:
Heat illnesses are often preventable. Important principles for developing a prevention program for exertional heat illnesses and specific measures for reducing risk, including:
- Institute prevention policies, including an emergency action plan.
- Educate staff and pilgrims about heat illness.
- Acclimatize gradually to exercising in hot and/or humid conditions; the process of heat acclimatization generally requires 7 days, under a heat stress comparable to the target competition. Training sessions for heat acclimatization should last at least 60 minutes per day, and induce an increase in core and skin temperatures, as well as stimulate sweating.
- Direct sun light exposure should be avoided.
- Remain at air-conditioned places as long as possible.
- Use white–colored umbrella whenever exposed to sun, and take rest at shady areas.
- Provide frequent breaks for hydration and cooling.
- Dress light cotton–made clothes and Minimize equipment and clothing that hinder heat loss in hot or humid conditions.
- Avoid activity during severe heat and/or humidity.
- Take all medications regularly.

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