Hypothermia and Hyperthermia
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OVERVIEW

The mammalian body is designed to function best within a very small temperature range at the core. This core body temperature (CBT) is so vital, that the autonomic system will respond to changes of less that 1°C. When the CBT is outside of the individual patients normal range, serious deleterious effects occur to the cells and organs of that patient.

The CBT is controlled by the main thermostat of the body, the hypothalamus. Temperature sensors are located in the hypothalamus and secondary sensors within the skin, organs and great veins. Appropriate physiologic and behavioral responses then attempt to return the CBT back to the functional range.

HYPOTHERMIA

Hypothermia can be either primary (environmental) or secondary (disease, trauma, medication, surgery) in nature. Regardless of the underlying cause, the effects on the body are the same. Historically, the classification of hypothermia was based purely on the CBT. Unfortunately, this system does that appropriately classify the severity of the hypothermia. A more useful classification is based on the clinical signs produced as a consequence of the degree of hypothermia.

Classification of Hypothermia Based on Clinical Signs

<table>
<thead>
<tr>
<th>Classification</th>
<th>Temperature</th>
<th>Clinical Signs</th>
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</thead>
<tbody>
<tr>
<td>Mild</td>
<td>90-99F</td>
<td>Shivering, ataxia, vasoconstriction</td>
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<tr>
<td>Moderate</td>
<td>83-90F</td>
<td>Decreased level of consciousness, hypotension, +/- shivering</td>
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<tr>
<td>Severe</td>
<td>&lt;83F</td>
<td>Loss of shivering, dysrhythmias, profound CNS deficits</td>
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Note: temperature range based on the "classic" system

HEAT LOSS MECHANISMS

- Convection: body heat transferred to surrounding air
- Conduction: body heat transferred to objects (table, floor)
- Radiation heat transfer: photon energy transferred to surround structures (walls)
- Evaporative heat transfer: body heat lost through moisture evaporation (respiration)
PHYSIOLOGIC EFFECTS

- Cardiovascular
  - The primary complications include bradycardia, hypotension, dysrhythmias, decreased cardiac output and eventual asystole.

- Respiratory
  - Decreases in respiratory rate and depth, pulmonary tissue injury and oxygen dissociation disturbances are associated with profound hypothermia.

- Neuromuscular
  - Decreased cerebral blood flow results in level of consciousness changes, muscle stiffness/rigidity, and decreased/absent reflex.

- Acid-Base
  - Primarily a mixed respiratory and metabolic acidosis.

- Coagulation
  - Severe coagulation complications include an apparent thrombocytopenia, platelet and coagulation factor activity dysfunction and disruption of fibrinolysis.
  - Of important note, a coagulation panel can be “normal” because the blood has been warmed to run in the machine, yet the patient could be severely coagulopathic.

- Renal
  - Cold diuresis leads to significant hypovolemia initially, which may be followed by acute renal failure as hypothermia progresses.

REWARMING

The rewarming technique should be chosen based on the individual patients clinical consequences, not just the CBT.

- Passive external rewarming
  - This is augmentation of the patients own intrinsic ability to create and retain heat. This is accomplished through insulation to prevent heat loss from the four mechanisms of heat transfer.

- Active external rewarming
  - This is application of exogenous heat directly to the skin via forced-air surface rewarming (3M Bair Hugger), resistive heating (Hot Dog Warming Blanket).
  - Thermal injury is very high when heat is applied directly to the skin (hot water bottles, electric heating pads) and should be avoided if possible.
  - Rewarming shock can be a consequence when peripheral vasodilation leads to a relative hypovolemia.
  - Afterdrop results when colder peripheral blood returns to the core.
  - Rewarming acidosis results when the acidotic peripheral blood returns to circulation.

- Active core rewarming
○ This is application of exogenous heat directly to the vital (core) organs. Examples of this include warmed humidified air, heated infusions and heated peritoneal or thoracic lavage.
○ Gastric and colonic lavage with warmed water is ineffective due to the limited amount of surface area.

TREATMENT

1. Prevent further decreases in CBT.
2. Institute a rewarming strategy.
3. Stabilize and maintain the cardiopulmonary system. Pay particular attention to fluid therapy and fluid shifts.
4. Physiologic support for glucose, electrolytes (particularly potassium) and acid-base changes.
5. Anticipate and attempt to prevent hypothermic complications such as coagulopathies and dysrhythmias.

HYPERTHERMIA

Hyperthermia is the condition in which the CBT is above the normal homeostatic range for that individual animal. Just like hypothermia, whenever the CBT is not held within the normal “operating range”, complications and injury to body systems occur. The cause of the hyperthermia is an inability to lose (or transfer rapidly enough) the heat produced, induced or stored within the body.

ETIOLOGY

- Reset of hypothalamic set point
  ○ True “fever” is caused by release of endogenous pyrogens that raise the CBT as part of the acute-phase response.
- Inadequate heat-dissipation
  ○ Heat stroke is the most common type of severe hyperthermia and is caused by an inability to transfer the excess heat faster than it accumulates.
  ○ Exercise or tonic-clonic muscle activity can also be associated with significant hyperthermia.

HEAT STROKE

Heat stroke is the most severe form of acute hyperthermia and is associated with profound morbidity and mortality. This typically occurs when the heat loss mechanisms can no longer provide enough heat transfer. In dogs, this normally occurs due to a either pathologic disturbance (laryngeal paralysis) or an environmental (trapped in car) cause. In dogs, as the
hypothalamus notes the increased CBT, the respiratory center is stimulated to increase dead space ventilation allowing for significant heat loss through evaporation. Secondarily, the cardiac output is increased and the periphery vasodilates to allow further heat transfer to the environment. If the CBT continues to rise, the patient will start to display signs of **Heat Exhaustion**, such as weakness, vomiting and diarrhea. As the CBT rises, **Heat Stroke** ensues, with the classic signs of CNS dysfunction and multi-organ dysfunction syndrome (MODS).

**PHYSIOLOGIC EFFECTS OF HEAT STROKE**

- **Cardiovascular:** After the initial hyperdynamic state, continued volume loss and loss of vascular tone can lead to shock. Sinus tachycardia followed by ventricular dysrhythmias can occur due to damaged myocytes.
- **Pulmonary:** Upper airway disease is very common in many heat stroke patients and can be the root cause of the condition. Aspiration pneumonia from vomiting and ARDS due to MODS are also complications.
- **CNS:** Mentation changes secondary to cerebral edema, direct neuronal injury and death or intracranial hemorrhage are common.
- **Renal:** Renal tubular cell injury and severe hypovolemia can lead to acute renal failure. Secondarily, rhabdomyolysis can lead to additional renal injury.
- **Gastrointestinal:** In dogs, the GI tract is extremely sensitive to poor perfusion. As the enterocytes fail, an increased risk of endotoxemia from translocation can occur.
- **Hepatic:** Hepatocytes can be injured directly or secondary to poor perfusion. This can lead to glycogen depletion, hypoglycemia or clotting factor dysfunction.
- **Coagulation:** DIC is extremely common in heat stroke due to endothelial injury, the inflammatory state and venous stasis. This leads to a hypercoagulable state (early DIC) followed by the hypocoagulable state (late DIC).
- **Acid-Base:** Progression of aerobic glycolysis to anaerobic leads to an increase in lactic acid and an oxygen debt, causing a metabolic acidosis.

**THERAPY**

Treatment of heat stroke includes decreasing the CBT, supporting and maintaining vital organs, and prevention of complications.

- **Decrease the CBT**
  - Enhancement of heat loss through evaporation is the most effective way to decrease the CBT. This is accomplished by wetting the animal with tepid water and using a fan to move air across the wet to enhance evaporation.
    - Avoid cold water/ice as this causes vasoconstriction and will slow heat loss.
    - Alcohol on the footpads, cold water lavage (stomach, colon) and cold fluid infusions are ineffectual and should be avoided.
  - Stop cooling techniques when the CBT drops to 103°F. Since the hypothalamus is injured, the temperature will continue to drop. Therefore, once within the
appropriate temperature range, dry the patient and prevent temperature drops below normal.

● Supportive
  ○ Intravenous fluids are the mainstay of cardiovascular support. There is typically a severe hypovolemia which must be corrected. Initially, start with 20-30 ml/kg of any balanced intravenous fluid over 15-20 minutes and monitor for response. Follow up volumes will be required based on individual patient needs.
  ○ Vasoactive substances (dopamine, dobutamine, norepinephrine, vasopressin) to increase cardiac output or blood pressure may be needed if volume loading is not enough.
  ○ Oxygen support should be used to maximize oxygen delivery initially, then tailored as needed for pulmonary injury.
    ■ If laryngeal paralysis is present, endotracheal intubation or tracheostomy may be required.
  ○ Dextrose infusions may be required as hypoglycemia is common.
  ○ Empiric broad spectrum antibiotics should be instituted in patients severely affected.
  ○ Plasma as needed for treatment of a hypocoagulable state.
  ○ Glucocorticoids should be avoided.

● Preventative
  ○ Aggressive patient monitoring of blood pressure, volume status is the best defense against further injury.
  ○ Goals should include keeping the MAP above 80mmHg, SaO2 above 96% and urine production above 1ml/kg/hr.
  ○ Provide gastrointestinal protection (sucralfate, proton pump inhibitors).

PROGNOSIS

The prognosis of heat stroke depends on several factors. Intuitively, patients with MODS or severe CNS disturbances have a worsened prognosis. In a review of 54 cases, there was a 50% mortality, with significant risk factors being DIC and ARF.

REFERENCES
