Hyperthermia and Heatstroke in the Canine

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HYPERTHERMIA

Hyperthermia is an elevation in body temperature that results when heat production exceeds heat loss. Core body temperature rises above the established normal range (99.8-102.8°F/ 37.6-39.3°C) in the homeothermic canine.

A. Classifications of Hyperthermia

1. Pyrogenic – with fevers, endogenous or exogenous pyrogens (e.g. virus, bacteria, cytokines) act on the hypothalamus to raise body temperature, creating a higher set point. As a normal response acute phase response to infection and inflammation, this rarely raises body temperature higher than 105.5 °F/ 40.8°C, not putting the patient at a severe health risk, and may be beneficial in mitigating morbidity and mortality of infectious diseases.

2. Non-pyrogenic – heat stroke, a severe form of heat-induced illness. This results in central nervous dysfunction and multi-systemic tissue injury secondary to a systemic inflammatory response.

   a) Heatstroke is classified as exertional or non-exertional. Exertional heatstroke is typically seen in late spring and early summer, before acclimatization has occurred.

   b) Predisposing factors that impair ability to dissipate heat (i.e. via effective panting) or increase heat production further define a patient with heat stroke: anatomy, laryngeal paralysis, obesity, endocrine disease, and exogenous factors.
B. Normal Thermoregulatory Responses to Hyperthermia

1. Hypothalamic Thermoregulatory Center – a rise in core body temperature stimulates central and peripheral temperature receptors, which activate hypothalamic cooling measures via physiologic responses.

2. Physiologic Responses
   a) Evaporative cooling – panting for evaporation of saliva and fluid in upper respiratory tract, may account for up to 60% of heat dissipation
   b) Peripheral vasodilation and increased cardiac output – contributes to cooling via conduction, convection, and radiation. More than 70% of a dog’s body surface heat loss may occur by these mechanisms, up to 40% via radiation alone
   c) Release of pro-inflammatory and anti-inflammatory cytokines – an acute phase response
   d) Heat shock proteins – play a role in protecting cells from heat damage

3. Behavioral Responses
   a) Seek cool surfaces to lie upon
   b) Seek shade, getting out of direct sun
   c) Seek water
   d) Seek breezes
   e) Minimize/avoid activity

C. Risk Conditions Associated with Heatstroke (Non-Pyrogenic Hyperthermia)

1. Factors that inhibit heat dissipation
   a) Lack of acclimatization to the weather – late spring, early summer; dogs may require up to 20 days to acclimate to warmer weather
   b) High/hot ambient temperatures
   c) Humidity
   d) Confinement with poor ventilation
   e) Water deprivation, dehydration – associated with vasoconstriction and may interfere with hypothalamic function
   f) Brachycephalic breeds (Pugs, Boston Terriers, English Bulldogs) with compromised upper respiratory anatomy (stenotic nares, elongated soft palate, laryngeal edema/collapse, everted saccules, trachea hypoplasia)
   g) Obesity
   h) Extremes of age – very young, very old
   i) Thick, dense coat – insulating effect, although this also reflects short-wave radiation better and protects body surface from heat radiation exposure
   j) Muzzling
   k) Drugs - antihistamines
2. Factors that contribute to heat production
   a) Simple exposure to excessive environmental temperatures
   b) Exercise – minimal in hot, humid environment to strenuous in moderate environment. Muscle metabolism accounts for up to 80\% of the body’s overall heat production during exercise.
   c) Anxiety
   d) Dark coat color
   e) Drugs – aspirin, thyroid supplements

3. Medical conditions that predispose to heatstroke – apparently healthy search canines may not be clinical or yet diagnosed
   a) Cardiovascular disease
   b) CNS disease, hypothalamic disease, seizures
   c) Laryngeal paralysis
   d) Hypokalemia
   e) Endocrine disorders: hyperthyroidism, diabetes mellitus, Addisonian crisis, pheochromocytoma
   f) Prior heatstroke – mechanism unknown
   g) Rebound hyperthermia following hypothermia episode

4. Other
   a) Fatigue - canine is not moving as efficiently and must work harder to perform the same job.
   b) Ground surface – higher ground temperature, especially asphalt or sand which also reflect heat back up to canine

D. Degrees of Hyperthermia

1. Mild - <104 °F / <40 °C does not normally require advanced treatment
2. Moderate – 104-106 °F / 40-41 °C
3. Severe - >106 °F / >41 °C
E. Physiologic Effects of Hyperthermia – Causes of cell destruction and organ system dysfunction are multifactorial, and include thermal destruction of cell membrane lipids and chemical bonds, denaturation and inactivation of enzymes, and development of tissue hypoxia leading to acidosis.

1. Cardiovascular
   a) Increased metabolic rate and oxygen consumption
   b) Decreased cardiac output causing poor organ tissue perfusion, acidosis, muscle degeneration
   c) Myocardial necrosis, pulmonary hypertension
   d) Sinus tachycardia, ventricular arrhythmias from to all of the above
   e) Hypovolemic shock, distributive shock secondary to vasodilation

2. Respiratory
   a) Parenchymal disease (harsh, crackles auscultated) secondary to hemorrhage from DIC or aspiration pneumonia
   b) Laryngeal edema
   c) Acute Respiratory Distress Syndrome associated with DIC – systemic inflammatory response, sepsis secondary to bacterial translocation for the GIT, and primary parenchymal injury

3. Neurologic
   a) Cerebral edema
   b) Neuronal degeneration, necrosis, petechial hemorrhage
   c) Seizures

4. Gastrointestinal – direct thermal injury to GI mucosa and hypoperfusion
   a) Mucosal barrier breakdown
   b) Bacteria translocation and bacterial endotoxemia
   c) GI hemorrhage

5. Renal
   a) Acute tubular necrosis from thermal injury, intravascular thrombosis, hypoperfusion, hypoxia, and myoglobinuria
   b) Obstructive uropathy secondary to crystallization of myoglobin and uric acid

6. Hepatic
   a) Hepatocellular necrosis
   b) Cholestasis
   c) Immune compromise as the reticuloendothelial system fails

7. Musculoskeletal
   a) Rhabdomyolysis from direct thermal injury
   b) Hypoperfusion
8. **Biochemical**
   a) Coagulopathy – vascular endothelial damage, capillary permeability, platelet destruction, impaired clot factor synthesis, fibrinolysis
   b) Acid Base
      1. Early respiratory alkalosis from excess panting
      2. Later metabolic acidosis with dehydration leading to hypotension, poor tissue perfusion, causing lactic acidosis
   c) Electrolytes
      1. Hypokalemia from vomiting and panting resp alkalosis
      2. Hyperkalemia later from acidosis, tissue destruction, renal compromise
      3. Hypernatremia and hyperchloridemia from dehydration
      4. Hypophosphatemia and hypomagnesemia (unknown mechanism)

F. **Summary of Signs Indicative of Hyperthermia**

1. **Early Stages**
   - Tachypnea, hyperventilation, panting
   - Tachycardia, hyperdynamic femoral pulse
   - Hyperemia, dry mucous membranes
   - Hypersalivation
   - Hematochezia (bloody stool, early sign)
   - Altered mentation: depression, stupor, coma
   - Dark red mucous membranes
   - Seizure (late stage)
   - Hypotension
   - Weak, collapse
   - Vomiting
   - Diarrhea
   - Hemorrhage
   - Cap refill <1 sec

2. **Severe or Protracted Heatstroke**
   - Weak femoral pulses
   - Pale, gray mucous membranes
   - Shallow respirations, progression to apnea
   - Vomiting, diarrhea – often bloody
   - Seizure, coma

3. **Delayed Signs** – as late as 3-5 days after apparent recovery
   - Oliguria
   - Icterus
   - Cardiac arrhythmias
   - Seizures
   - DIC
   - ARDS
G. Diagnosis

1. History
2. Physical exam findings
   a) Elevated body temperature, however this may be normal depending on intervention before taking temperature and time to transport
3. Laboratory
   a) Hemogram – high PCV, anemia later (hemorrhage, hemolysis), thrombocytopenia, leukocytosis
   b) Biochemistry
      \( \uparrow \) Increases: BUN, creat, liver/muscle enzymes, \( \text{Na}^+ \), \( \text{Cl}^- \), \( \text{K}^+ \) (late)
      \( \downarrow \) Decreases: glucose, \( \text{Ca}^{2+} \), \( \text{K}^+ \) (early), \( \text{PO}_4^{3-} \), \( \text{Mg}^{2+} \)
   c) Urinalysis – proteinuria, hematuria, myoglobinuria, tubular casts
   d) Coagulogram
      - Prolonged PT, PTT, ACT
      - Elevated FDPs
      - Decreased fibrin and platelets

H. Treatments

*Initial stabilization* should focus on decreasing body temperature to prevent further heat induced injury, maximizing oxygen delivery to tissue by restoring tissue perfusion and arterial oxygen concentration, and minimizing further neurologic injury

1. Normalizing Body Temperature

   **Surface Cooling Techniques** – in the field/during transport
   a) Remove animal from the hot environment
   b) Wet down with cool/room temperature water
   c) Place on cool surface
   d) Use fan to blow air over the patient or place before air conditioner
   e) Ice packs may be placed to neck, axillary, and groin areas (large vessel areas: jugular, brachial, and femoral)
   f) Isopropyl alcohol applied in very small amounts to hairless areas (axillae, inner ear pinnae, abdomen, inguinum) for its evaporative and vasodilation properties used by some
   g) Stop cooling methods once body reaches 103-104 °F / 39.4-40 °C as temperature will continue to fall. If hypothermia occurs, patient warming may be necessary

   **Internal Cooling Techniques in the Field** – in the field, during transport
   h) Cold intravenous isotonic fluid administration
   i) Cold water enema –will lose accurate rectal temperature monitoring
   j) Other options generally reserved for in-hospital care, including gastric lavage, open body cavity, peritoneal dialysis
**NOTES:**

Ice water baths have historically been discouraged due to concerns that (1) vasoconstriction will reduce heat transfer out of the body and actually raise internal temperature and (2) the shivering would generate more heat.

To date, there are no scientific studies to prove or disprove these assumptions. In the field just use what you have, and monitor response.

Another concern is the use of large quantities of alcohol, which is generally discouraged, because significant vasodilation may promote/worsen shock and in some cases lead to uncontrollable decreases in temperature. Again, use what you have but limit alcohol use to the less haired areas for best effect with the least amount.

2. **Restoring and Maintaining Tissue Perfusion** – treating hypovolemic shock
   a) IV catheter placement
   b) Blood collection for baseline values ideal
   c) Isotonic electrolyte solution @ 20-40 ml/kg bolus; reassess and repeat until cardiovascular parameters normalize. Another fluid guideline alternative is 90 ml/kg/hr, reassess every 15 minutes during administration to adjust rate based on patient response
   d) If blood pressure does not improve with adequate fluid resuscitation, consider drug therapies, but these patients should be at a hospital facility by this time!
      (1) Positive inotrope dobutamine 5-10 µg/kg/min
      (2) Vasopressor therapy with dopamine 5-20 µg/kg/min
      (3) Vasopressor therapy with norepinephrine 0.1-20 µg/kg/min
   e) Colloid bolus (5-10 ml/kg) typically not indicated; pure water loss needs water replacement rather than colloids until DIC does develop; caution if coagulopathy suspected

**NOTE:** adequate fluid therapy is important, as vasopressors may redistribute blood away from the gut, leading to more severe GIT compromise

   f) Monitor HR (80-120), ECG, CRT (<2 sec), BP (120/80)
   g) Maintenance fluids (40-60 ml/kg/day) plus fluid losses once stabilized (canine should be in hospital by now!)
3. **Airway and Breathing**
   a) Oxygen therapy until respiratory evaluation and oxygen delivery efficiency evaluated
   b) Short term oxygen safe: minimize breathing effort, corrects hypoxemia
   c) Respiratory distress and inability to pant properly contributes to continuing hyperthermia despite cooling measures
   d) If airway patency is compromised, intubation may be needed

4. **Central Nervous System**
   a) Blood glucose (normal 60-110) check immediately in the presence of neurologic abnormalities. If hypoglycemic:
      (1) 50% dextrose bolus @ 0.25-0.5 g/kg
      (2) Add dextrose to maintenance fluids @ 2.5-5% concentration
   b) Altered mentation after restored tissue perfusion or other signs indicative of cerebral edema (seizures, cranial nerve deficits, paresis, miosis/mydriasis, inappropriate bradycardia, apnea):
      (1) Mannitol 0.5-1.0 g/kg over 20 minutes
      (2) Hypertonic saline 7% 3-5 ml/kg
      (3) Elevate head ~30 degrees
      (4) Seizures: Diazepam 0.5 mg/kg IV; phenobarbital 2-10 mg/hr

**Additional assessment and treatment**, after initial stabilization, focuses on the renal, gastrointestinal, hepatic, and coagulation systems while continuing to monitor cardiovascular, respiratory, and neurologic systems.

5. **Renal System**
   a) BUN, creatinine, and potassium evaluations are paramount
   b) Desired urine output once tissue perfusion is restored and fluid replacements achieved is 2 ml/kg/hr

6. **Gastrointestinal System**
   a) Control vomiting with anti-emetics:
      (1) Ondansteron 0.2 mg/kg IV; Dolasetron 0.5 mg/kg IV
      (2) NOTE: Cerenia not used if hepatic dysfunction suspected
   b) Treat gastric ulceration
      (1) Famotidine 0.5-1.0 mg/kg; Ranitidine 0.5-2.0 mg/kg
   c) Treat bacterial translocation, +/- sepsis, with broad spectrum antibiotic
      (1) Penicillin and fluoroquinolone; Cephalosporin and fluoroquinolone

7. **Coagulation System**
   a) DIC common sequelae: PT, PTT, platelets, FDPs, D-dimers monitored
   b) Fresh frozen plasma may be administered to control hemorrhage

8. **Hepatic System**
   a) Biochemical evaluation of liver enzymes
I. Prognosis
Heat stroke patients are given a guarded prognosis due to the systemic complications that may occur. One study found factors associated with increased risk for death included DIC, hypoglycemia, and acute renal failure. The mortality rate was 50%, however all of the dogs that survived had been treated by their owners and transported to a hospital within 90 minutes. Other risk factors identified include delayed admission to the hospital (>90 minutes), seizures, ventricular arrhythmias, and some abnormal blood values.

J. Prevention Techniques
Most of these are conclusions based on the preliminary findings of the “Dubai Study”, supported by the International Rescue Dog Organisation’s Commission for Science and Research, 2009

1. Search periods should be as short as possible; 15 minutes has been shown to be the absolute upper limit in outside temperatures in excess of 86°F / 30°C
   a) Search at midday and in direct sunlight should be kept especially short
   b) If possible, avoid midday search and concentrate on early morning, early evening, or at night

2. Body temperature is the factor that limits performance during search in hot climates, therefore body temperature measurements are a good way of measuring heat stress levels on a canine
   a) Temperature of 104°F / 40°C should be removed from search; body temperature continues to rise for some time after physical exertion is over
   b) Canines have been known to continue to search even when their temperature had risen into the critical range of 105.8°F / 41°C. They literally would work until they dropped; handler recognition and monitoring are critical to determine when to stop a search
   c) Encourage some acclimatization rather than relying on air conditioning for the main down time. Although 20 days is needed for full acclimatization, even 4 days will lower the strain on rescue canines in a hot climate.
**NOTE:** Body temperature and degree of exhaustion are not the same in all dogs

- Greyhounds, considered sprinters, had temperatures ranging from 104-106°F.
- Labrador Retrievers, considered intermediate athletic performers, had temperatures between 102-107°F while hunting.
- During a long race, sled dogs, the endurance athletes of the dog world, often had temperatures between 104-108°F.

3. Sufficient rest period between individual search operations is required
   a) Fatigue and lack of sleep lead to a clear decline in mental and physical capabilities of these canines, risking that they miss a victim
   b) Rest period between should be at least 40 minutes, as body temperatures fall slowly
   c) Rest time must be adapted to ambient temperature: the hotter it is, the longer the rest time
   d) Shade and wind are the best places to rest, improving heat dissipation
   e) Free access to sufficient drinking water

4. Hydration
   a) Maintaining adequate hydration cannot be overemphasized
   b) Water intake maintenance guideline: 2-4 ml/kg/hr, or 1.5-3 liters/day for a 75# canine; work & heat stress add 1.25-2 times this amount
   c) 40-60 ml/kg/day is another formula
   d) Encourage to drink with training, flavored additives
   e) Oral electrolyte solutions of questionable value, though dogs that drool a lot may benefit

5. Other Considerations
   a) Wet down hair coat before, during, and after search
   b) Walk in the shade when at all possible, as ground temperatures may be higher than the air temperature especially when walking or working on asphalt or sand
   c) Check paws frequently, as these bear the brunt of physical abuse on hot surfaces (inflammation, cuts and abrasions)
   d) Hot, unfamiliar climes expose canines to unfamiliar bacteria which may increase risk of gastrointestinal stress. Maintain their normal feed and if possible use bottled water for drinking
What About Subcutaneous Fluids BEFORE Working in a Hot Environment?

Currently there is no published scientific data regarding potential benefits and/or contraindications for giving a working canine subcutaneous fluids in an effort to prevent and/or delay the onset of dehydration and other heat-related conditions.

Factors to consider against:

- Austere and unsanitary conditions increasing potential for infection or abscess introduced by the needle
- Volume needed to 'pre-load' a 30 kg (70 lb) canine against 5% dehydration is 1500 ml, a large amount at one time
- A normovolemic canine would not absorb the fluids very quickly as they are not needed by the body
- Large humps of fluids may interfere with harness or safety vests worn by some working canines
- Canine heat dissipation is mainly via respiratory tract (panting), a pure water loss. Most commonly 0.9% NaCl is the available subcutaneous fluid; this may exacerbate hypernatremia if the canine becomes dehydrated.
- Proper acclimation, enforced work-rest cycles, opportunity to orally hydrate, resting in shade or air-conditioning, healthy weight, and monitoring temperature are the best ways to maintain hydration or catch a problem early

Factors to consider for:

- Giving 500 ml may increase the amount of time a canine can work in a hot environment before becoming dehydrated
- No known infection/abscess occurrence in >500 military working dogs (MWDs) - Dr. Janice Baker, anecdotal
- Political situation: in Haiti, Urban Search and Rescue groups were concerned about giving the canines water in view of a populace that did not have enough potable water. Handlers were directed not to give their canines oral water, so the medics were giving them subcutaneous fluids.
- Handlers report better endurance, scenting, and recovery from hard work when they received prophylactic SC fluids compared to when they did not
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