



HEATSTROKE IN DOGS

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Heatstroke is a common problem in pets during the summer months, especially in hot, humid climates. This life-threatening condition can affect dogs of any age, breed, or gender.

Heatstroke in dogs is defined as a *nonpyrogenic increased body temperature above 104°F (40°C), with a spectrum of systemic signs*.¹ The ability to rapidly recognize and begin treatment of heatstroke is vital to maximize the chances of saving the animal's life.

HYPERTHERMIA

Maintenance of normal body temperature is an important aspect of preserving homeostasis and preventing interruption of normal metabolism.

The thermoregulatory center, located in the anterior hypothalamus, maintains body temperature within a narrow range, or *set point*. A pyrogenic hyperthermia, or fever, may be induced by an increase in the set point in response to disease.

Differentiating between pyrogenic and nonpyrogenic hyperthermia is important, due to the variation in pathophysiology and treatment of each disease process.

Pyrogenic Hyperthermia

If a dog develops pyrogenic hyperthermia, the underlying cause guides treatment. Active cooling of a patient with pyrogenic hyperthermia causes metabolic stress and physical discomfort.²

In one human study, pyrogenic hyperthermia was induced via an injection of IL-2, a pyrogenic cytokine; then subjects were actively cooled, resulting in an increase in oxygen consumption and reported physical discomfort. Within that study, pyrogenic hyperthermia subjects exposed to forced air warming were most comfortable.²

Nonpyrogenic Hyperthermia

A nonpyrogenic hyperthermia, such as heatstroke, is not due to a change in the hypothalamic thermoregulatory set point, which allows us to perform active cooling without the consequences previously mentioned.

THERMOREGULATION

The body uses several methods to cool itself, which are described in **Table 1**.

TABLE 1. Methods of Cooling

EVAPORATION

- Occurs through panting, which dispels heat via evaporation of water from the surface of the tongue, and is one of the most important ways a dog thermoregulates
- Most effective when the ambient temperature reaches 89.6°F (32°C);¹ less efficient with high ambient humidity, which increases the risk for heatstroke

CONDUCTION

- Occurs through transfer of heat from one object to another
- In dogs, commonly achieved by lying with their sparsely haired abdomen in contact with a cool surface, allowing transfer of heat to this surface

CONVECTION

- Occurs by movement of air over the body to disperse heat, such as wind or air from a fan
- Plays an important role in treating heatstroke

RADIATION

- Occurs when heat from the body dissipates into the environment
- Less effective when ambient temperature reaches body temperature

Heat-related illness can vary in severity and, in humans, is classified based on that severity.

Heat stress is rarely recognized in veterinary patients. It includes some discomfort and physiologic strain. The core body temperature will be normal.³

Heat cramps are rarely recognized in veterinary patients. Muscle cramps occur secondary to the depletion of sodium and water. The core body temperature will be normal.^{3,4}

Heat exhaustion includes weakness, ataxia, fainting, and an inability to continue the work the person was performing. The core body temperature will be normal or slightly increased.^{3,4}

Heat stroke includes dysfunction of the central nervous system, hypovolemia, and cellular dysfunction. The core body temperature will be elevated.^{3,4}

A minimum nonpyrogenic body temperature elevation of 1.8°F (1°C) can activate heat receptors found in the periphery, triggering the thermoregulatory center, which in turn induces constriction of renal and splanchnic vessels, tachycardia, and cutaneous vasodilation. These processes result in increased blood flow to the skin, where it can be cooled.¹

The hypothalamus also stimulates tachypnea and panting to assist with cooling.¹

TYPES OF HEATSTROKE

Heatstroke is classified as *exertional* or *nonexertional* (classical).

Exertional Heatstroke

Exertional heatstroke occurs during exercise and is more common in dogs that have not been acclimated to their environment. If a period of temperature acclimation is allowed, dogs become less susceptible to heatstroke. Acclimation can take up to 60 days, although the animal is partially acclimated within 10 to 20 days.³

While exertional heatstroke can occur in working dogs, it is less common because handlers are typically more knowledgeable. For example, it is possible for military dogs to work in environmental temperatures reaching 140°F (60°C) without adverse effects. After racing, greyhounds can transiently have rectal temperatures as high as 107.6°F (42°C) without showing signs of heatstroke.⁴

Nonexertional Heatstroke

Nonexertional heatstroke results from exposure to increased environmental temperature in the absence of adequate means of cooling. This may be seen when a dog is enclosed in a parked car or left in a yard without shade and water.

RISK FACTORS

Several predisposing factors may decrease a dog's ability to cool itself; therefore, increasing its risk for developing

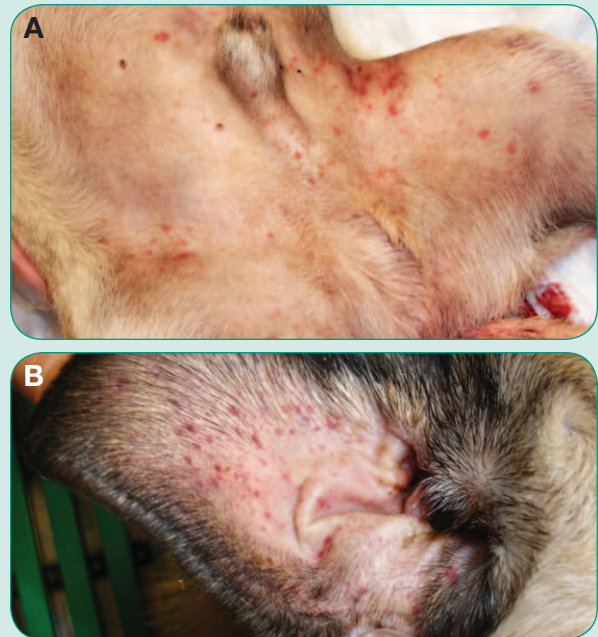


Figure 1. Petechial hemorrhage on (A) the abdominal wall, and (B) the ear pinna of a young pug with disseminated intravascular coagulation caused by heatstroke. Brachycephalic breeds are particularly at risk for heatstroke because of limited ability to thermoregulate as a result of partial upper airway obstruction.



Figure 2. Bloody urine in a pug with heatstroke may reflect intravascular hemolysis or hemorrhage into the urinary tract due to coagulopathy.



Figure 3. Emergency resuscitation of this comatose pug with disseminated intravascular coagulation due to severe heatstroke included administration of a blood transfusion.

Figures 1-3 courtesy of Dr. Lesley G. King

heatstroke. These factors are listed in **Table 2**. Dogs that have a history of heatstroke are more susceptible to developing it again.⁴

PATHOPHYSIOLOGY
Acute-Phase Response

The acute-phase response is a coordinated cellular response that occurs in inflammatory events, such as infection, surgery, trauma, burns, immune-mediated diseases, and nonpyrogenic hyperthermia.^{3,5} During nonpyrogenic hyperthermia, this response is stimulated in an attempt to protect tissues from excessive heat and promote repair.³

Acute-phase proteins are divided into 2 categories:

1. **Negative acute-phase proteins** decrease by 25% during an acute-phase response.^{5,6} In most species, albumin is a negative acute-phase protein, and levels may decrease during an acute-phase response due to gastrointestinal (GI) or renal loss or decreased production.
2. **Positive acute-phase proteins** increase by 25% during an acute-phase response.^{5,6} In dogs, positive acute-phase proteins include C-reactive protein and serum amyloid A.

TABLE 2. Examples of Factors That Predispose Dogs to Heatstroke

- Brachycephalic syndrome
- Cardiac disease
- Laryngeal paralysis
- Obesity
- Tracheal collapse

- » C-reactive protein activates the complement system, which promotes phagocytosis, and up- or down-regulates cytokine production and chemotaxis.
- » Serum amyloid A causes chemotaxis of several types of white blood cells and inhibits inflammation.⁶ Several other acute-phase proteins play important roles in the acute-phase response, including:⁶
 - » Haptoglobin: Reduces oxidative damage
 - » Ceruloplasmin: Scavenges free radicals
 - » Fibrinogen: Plays a role in tissue repair.

The acute-phase response also initiates production of heat shock proteins (HSPs).⁷

Heat Shock Proteins

When cells are exposed to an increased temperature, HSPs protect against protein denaturation.⁸ They prevent disaggregation of denatured proteins, refold denatured proteins, protect against endotoxin leakage across the intestines, and reduce cerebral ischemia.⁷

Effects by Body System

Heatstroke has multiple deleterious effects within the body (**Table 3**). Direct cytotoxicity occurs at body temperatures ranging from 106.7°F to 107.6°F (41.5°C–42°C). At these temperatures enzymes and proteins are denatured.¹ The severity of these effects depends on both degree of body temperature elevation and duration of time it is elevated.

TABLE 3. Heatstroke Effects by Body System

BODY SYSTEM	HEATSTROKE EFFECTS
Central Nervous	<ul style="list-style-type: none"> • Susceptibility to cytotoxicity is increased, resulting in injury and death of neurons. • Cerebral edema, hemorrhage, and necrosis can occur.³
Coagulation	<ul style="list-style-type: none"> • Endothelial damage—incited by direct cytotoxicity—releases thromboplastin and factor XII, activating the coagulation cascade.³ • Disseminated intravascular coagulation may then occur (Figures 1 through 3).
Cardiovascular	<ul style="list-style-type: none"> • Initially, cardiac output, peripheral vasodilation, and central vasoconstriction are increased. • When these compensatory measures fail, venous blood pools, and central vasodilation leads to decreased circulating blood volume, hypotension, and shock.¹ • Blood is unable to adequately circulate, resulting in electrolyte derangements, acidosis, and production of microthrombi. • Ventricular arrhythmias may occur due to ischemia of myocardium and exacerbate perfusion abnormalities.⁴
Pulmonary	<ul style="list-style-type: none"> • Direct injury to the pulmonary vascular endothelial cells can lead to pulmonary edema, increased pulmonary vascular resistance, and acute respiratory distress syndrome.⁴
Gastrointestinal	<ul style="list-style-type: none"> • Direct cytotoxicity, prolonged hypotension, hypovolemia, and production of microthrombi cause decreased perfusion and ischemia, compromising the integrity of the GI tract.³ • Cellular hypoxia produces reactive oxygen and nitrogen species and stimulates inflammatory cells that further damage GI mucosa.⁹ • Increased permeability of GI mucosa can potentially result in translocation of bacteria and bacterial products, such as endotoxin. • Bacteremia, endotoxemia, and sepsis can cause further ischemia, cardiac dysfunction, shock, and eventually death.⁴
Renal	<ul style="list-style-type: none"> • Direct thermal injury and protein denaturation lead to tubular necrosis. • Indirect injury is secondary to vasoconstriction, dehydration, and myoglobinuria secondary to rhabdomyolysis.^{3,4}

TABLE 4. Clinicopathologic Findings: Heatstroke Patients

COMPONENT	RESULT	CAUSE
Serum Biochemical Profile¹⁰		
Alanine aminotransferase	May be increased	Hepatic injury ¹⁰
Alkaline phosphatase	May be increased	Hepatic injury ¹⁰
Aspartate aminotransferase	May be increased	Hepatic injury, rhabdomyolysis (due to direct thermal damage to muscle or increased muscular activity) ¹⁰
Creatinine	May be increased	Renal (direct thermal damage, myoglobinuria due to rhabdomyolysis) and prerenal azotemia (dehydration, vasoconstriction) ¹⁰
Creatinine kinase	May be increased	Rhabdomyolysis (due to direct thermal damage to muscle or increased muscular activity) ¹⁰
Hypoglycemia	May be observed	Increased use (due to increased ATP demand) or decreased production (due to hepatic damage) of blood glucose ¹⁰
Lactate	May be increased	Hypovolemia and decreased perfusion leading to tissue hypoxia ¹⁰
Complete Blood Cell Count & Differential¹⁰		
Thrombocytopenia	Commonly observed	Secondary to platelet consumption (caused by GI hemorrhage) and platelet aggregation (due to disseminated intravascular coagulation). ¹⁰
Packed cell volume	Commonly increased	Dehydration
Hemoglobin concentration	Commonly increased	Dehydration
Nucleated red blood cells	May be observed	Secondary to direct thermal damage to bone marrow causing premature release of cells ¹⁰
Coagulation Panel		
Activated partial thromboplastin time	May be prolonged	Direct thermal injury to endothelium and consumption of coagulation factors ¹⁰
Prothrombin time	May be prolonged	Direct thermal injury to endothelium and consumption of coagulation factors

Pathology

Pathologic findings in dogs with heatstroke are extensive and severe. Necropsy of 11 dogs with fatal heatstroke revealed severe pulmonary edema, splenomegaly, and hepatomegaly. Hemorrhage was observed within the myocardium, peritoneum, mesentery, and throughout the GI tract. The brains of 9 dogs had edema and necrosis. The kidneys in all 11 dogs revealed lesions, glomerular congestion, and tubular degeneration and necrosis. The small intestine of 2 dogs, large intestine of another, and liver of a third could not be examined due to autolysis.⁹

DIAGNOSIS

History & Clinical Signs

History often includes recent exercise or confinement in an area without access to water or shade.

The most common clinical signs in one study were collapse, tachypnea, shock, inappropriate mentation, and signs of coagulopathy,¹⁰ but may also include tachycardia, hyperemia, and hyperdynamic to nonexistent pulses. Concurrent central nervous system (CNS) signs can range from slight ataxia to seizures or coma.

Damage to the GI tract quickly leads to severe diarrhea. The diarrhea is often bloody (hematochezia) when it comes from the lower GI tract, or it may be black (melena) if originating from the upper GI tract.

Although hyperthermia is usually present, a patient with

heatstroke may present normothermic or hypothermic due to cooling efforts prior to arrival at the hospital, a prolonged travel time to the hospital, or secondary to shock.

Common Diagnostic Findings

Upon presentation, perform a urinalysis and blood analysis, including:

- Serum biochemical profile, including electrolytes, blood glucose, and lactate
- Complete blood count and differential (manual)
- Coagulation panel, including prothrombin time (PT) and activated partial thromboplastin time (aPTT).

Common findings in patients with heatstroke are listed in Table 4.

TREATMENT

Implementing treatment as soon as possible—even before the patient reaches the hospital—significantly improves the patient's prognosis.

Cooling

Active cooling is the most important aspect of treatment, although it may not prevent deleterious effects of heatstroke. If possible, instruct the owner to begin cooling his or her dog during travel to the hospital. One study revealed dogs actively cooled before arriving at the hospital had a lower mortality rate (19%) than dogs not cooled prior to arrival (49%).¹¹

What to AVOID During Treatment

Avoid use of ice as it causes peripheral vasoconstriction, preventing cooling of blood via shunting to the periphery.¹⁴ Ice may also damage the skin and make treatment uncomfortable for the patient.

Avoid gastric lavage or cold water enemas, if possible. The risk of aspiration may outweigh the benefits of gastric lavage, and cold water enemas may further damage the already compromised GI tract.¹⁵

Avoid shivering in the patient; however, one human study states that shivering does not contribute to hyperthermia because the heat produced is minor relative to the amount of cooling achieved during emergency treatment for heatstroke.¹⁵

The goal of active cooling is to return the dog to a normal body temperature, while avoiding further organ damage.¹² An effective cooling method that combines evaporative and convective cooling is directing a fan toward the dog and applying cool or tepid water to the skin. Monitor the temperature every 5 minutes and, once body temperature reaches 103.5°F to 104°F (39.7°C–40°C), discontinue active cooling in order to avoid rebound hypothermia.¹

Respiration

In addition to active cooling, evaluate the airway for patency and, if necessary, intubate the patient or perform an emergency tracheostomy. Provide supplemental oxygen via a mask or flow-by for dogs that are breathing unassisted. Note that older oxygen cages that do not have temperature and humidity controls may exacerbate hyperthermia.¹³

Fluid Therapy

Intravenous fluids are considered a cornerstone of heatstroke treatment.

Upon arrival, establish IV access in the patient and administer room temperature crystalloids.¹ A bolus of crystalloids is beneficial for hypovolemic and hemoconcentrated heatstroke patients. Crystalloids provide cardiovascular support by expanding intravascular volume, and increasing blood flow to the periphery, aiding with the cooling process. Once hospitalized, crystalloids should be continued to provide maintenance fluid therapy as well as replace continuous losses (seen with continued vomiting and diarrhea).

If crystalloids do not adequately maintain intravascular volume, consider administering colloids.¹ Colloids may also be considered if the patient is hypoalbuminemic.^{1,13,14} Fresh frozen plasma is recommended if the patient has prolonged PT and/or aPTT.

Medical Therapy

Antibiotics. Due to the high risk of bacterial translocation secondary to GI damage, consider broad-spectrum antibiotics (Table 5), while keeping the possibility of antibiotic resistance in mind.¹⁵ Once antibiotic therapy is initiated, continue until GI signs, such as hematochezia, melena, or hematemesis, resolve and the patient is eating.¹⁴

GI tract support. Antiemetics (metoclopramide, dolasetron, maropitant) are beneficial when GI tract damage or CNS abnormalities cause nausea. Administer GI protectants because GI ulceration is likely to occur. Proton pump inhibitors (omeprazole, pantoprazole) are beneficial to prevent or treat gastric ulceration, while famotidine acts as an H₂ receptor antagonist and also decreases the production of acid. Sucralfate is given orally and acts as a local protectant that binds to ulcers preventing further damage from acid, pepsin, or bile (Table 5).

CNS support. CNS abnormalities are common in heatstroke patients. In some cases, these abnormalities may result from increased intracranial pressure.

Mannitol—an osmotic diuretic that expands intravascular volume, decreases blood viscosity and intracranial pressure, and improves cerebral microcirculation—can be administered if the patient has neurologic signs (Table 5).¹ If mannitol is contraindicated (such as with dehydration, intracranial hemorrhage, pulmonary edema, or anuria secondary to renal disease) hypertonic saline (7.2%–7.5%) can be used instead. Hypertonic saline will increase cerebral, coronary, and microvascular blood flow as well as decrease intracranial pressure. As with mannitol, hypertonic saline is contraindicated with dehydration.¹

Additional nursing management of patients with suspected cerebral edema may include elevation of the head and avoidance of compression of the jugular veins.

Cardiac support. Ventricular arrhythmias are common in patients presenting with heatstroke because cardiac cells are susceptible to thermal injury and ischemia. Damage to the myocardium causes defects in conduction leading to ventricular arrhythmias, which can be treated with lidocaine.⁴

MONITORING

Initiate patient monitoring upon arrival, including:

- Baseline vital signs (heart rate, respiratory rate and effort, temperature, mucous membrane color, and capillary refill time)
- Blood pressure
- Electrocardiography (ECG)
- Pulse oximetry.

Although unlikely to be seen on presentation, multiple organ dysfunction syndrome (MODS) may develop during hospitalization. MODS occurs when 2 or more systems are affected and begin to fail. Because heatstroke affects multiple organ systems, the risk for developing MODS is high, and the patient should be monitored closely during hospitalization.

TABLE 5. Medical Therapy for Canine Heatstroke Patients

INDICATION	DRUG NAME	DRUG CLASS	DOSAGE
Bacterial Translocation	Ampicillin	Bactericidal aminopenicillin	20–40 mg/kg IV Q 8 H
	Cefazolin	First generation cephalosporin	22 mg/kg IV Q 8 H
	Enrofloxacin	Bactericidal fluoroquinolone	5–20 mg/kg IV, IM, PO (once discharged) Q 24 H or divided Q 12 H
	Metronidazole	Antibacterial and antiprotozoal	10–15 mg/kg IV, PO (once discharged) Q 12 H
Gastrointestinal Damage	Dolasetron	Antiemetic (5-HT ₃ antagonist)	1 mg/kg IV Q 24 H
	Famotidine	H ₂ antagonist	0.5 mg/kg IV, IM, PO (once discharged) Q 12–24 H
	Maropitant	Antiemetic (NK ₁ receptor agonist)	1 mg/kg/day SC or IV, 2 mg/kg/day PO (once discharged)
	Metoclopramide	Antiemetic prokinetic	1–2 mg/kg/day IV as a CRI
	Omeprazole	Proton pump inhibitor	1 mg/kg PO (once discharged) Q 24 H
	Pantoprazole	Proton pump inhibitor	1 mg/kg IV Q 24 H
	Ranitidine	H ₂ antagonist	0.5–2 mg/kg IV, IM, PO (once discharged) Q 8–12 H
	Sucralfate	Gastric protectant	Small dog: 0.25 g PO Q 8 H Medium dog: 0.5 g PO Q 8 H Large dog: 1 g PO Q 8 H
Increased Intracranial Pressure	Mannitol	Osmotic diuretic	0.25–0.5 g/kg IV slowly over 20 minutes
Ventricular Arrhythmias	Lidocaine	Class 1B antiarrhythmic	2 mg/kg bolus IV, then 40–80 mcg/kg/min CRI
Cardiovascular Support	Dopamine	Catecholamine	3–15 mcg/kg/min CRI

Adapted from Plumb DC. *Veterinary Drug Handbook*, 4th ed. White Bear Lake, MN: PharmVet Publishing, 2002.

Placement of a urinary catheter and monitoring of urine production is beneficial in patients with renal insufficiency or failure.¹⁴ Normal urine output in a dog on IV fluids is at least 1 to 2 mL/kg/hour. A decrease in urine output may be due to renal failure, hypovolemia, or hypotension. If unable to place a urinary catheter, the patient's urination should be monitored closely.

Nutritional support is beneficial for the GI system. It improves the GI tract's integrity and allows the patient to replace important proteins, electrolytes, and other nutrients. If the patient is not willing to eat, a nasoesophageal feeding tube can be placed to allow for trickle feeding.¹⁴

Serial monitoring during hospitalization should include:

- Neurologic status assessment
- Heart rate, pulse quality, respiratory rate and effort, blood pressure, and ECG
- PT and aPTT
- Packed cell volume and total protein
- Blood glucose and electrolytes
- Lactate and acid–base balance
- Urine output.

Response to therapy can be monitored, in part, with serial lactate tests because lactate clearance is associated with resolution of tissue hypoxia.¹

PROGNOSIS

Prognosis is most dependent on length of time the patient was hyperthermic and highest body temperature experienced. When the body temperature reaches 109.4°F (43°C), marked organ damage and high mortality are seen. Cellular necrosis and destruction of all cellular structures occurs once body temperature has been maintained between 120.2°F to 122°F (49°C–50°C) for only 5 minutes.¹

Probability of survival increases with decreased time period of hyperthermia; however, it is important to monitor the patient for MODS and coagulopathies.¹² Negative indicators of prognosis associated with higher mortality include:

- Development of ventricular arrhythmias¹
- Severe coagulopathies¹⁰
- Abnormal neurologic signs that do not resolve once body temperature normalizes.

In humans, 20% of heatstroke victims suffer unresolved brain damage and CNS abnormalities, which have been linked to high mortality rates.¹² Therefore, return of normal CNS function is considered a positive prognostic indicator for dogs that have suffered heatstroke.

PREVENTION

Prevention of heatstroke relies heavily on owner education. Owners can prevent heatstroke several ways:

- Ensure availability of adequate shade and drinking water outdoors
- Exercise dogs only during cooler periods of the day
- Never leave dogs alone in closed vehicles
- Acclimate dogs to warm temperatures for up to 2 months
- Surgically address upper airway obstructions, such as brachycephalic airway disease or laryngeal paralysis, to decrease risk in individual susceptible patients.■

aPTT = activated partial thromboplastin time; CNS = central nervous system; ECG = electrocardiography; GI = gastrointestinal; HSP = heat shock protein; MODS = multiple organ dysfunction syndrome; PT = prothrombin time

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