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Canine Heatstroke

Heatstroke is defined as a state of extreme hyperthermia (106°–109° F) resulting in thermal injury to tissues, which occurs when heat generation exceeds the body’s ability to dissipate heat.

Heat dissipation occurs through 4 mechanisms: radiation, convection, conduction, and evaporation. Heat loss through radiation and convection occurs by cutaneous vasodilatation and increased cardiac output. Conductive heat loss occurs through contact with cool surfaces. Evaporation occurs through sweating in humans, horses, and cows, and by panting in dogs.

When the ambient temperature is below 89.5° F, heat loss may occur through conduction, radiation, and convection. As the ambient temperature increases and approaches body temperature, evaporative heat loss becomes much more important. In addition, evaporative heat loss becomes less efficient with increased ambient humidity.

Pathophysiology

Inability to dissipate heat may result from endogenous or exogenous sources. *Endogenous factors* that contribute to a decrease in the ability to dissipate heat include obesity, brachycephalic conformation, laryngeal paralysis/upper airway obstruction, cardiovascular or respiratory disease, extremes in age, and central nervous system disease. Animals with a history of heatstroke tend to be predisposed to further episodes, even at lower temperatures and humidity. *Exogenous factors* that limit heat dis-



Severe ecchymosis on the ventral abdomen of a dog with disseminated intravascular coagulation secondary to heatstroke caused by a grooming dryer

sipation include water deprivation, confinement in a poorly ventilated area, lack of acclimatization, and high humidity.

Increased heat production leading to heatstroke may be caused by exercise, febrile disease, hormonal hyperthermia (hyperthyroidism, pheochromocytoma), seizures, or severe muscle

fasciculations. In addition, malignant hyperthermia due to abnormal calcium metabolism may occur in predisposed breeds exposed to specific anesthetic drugs. Often, heatstroke is caused by a combination of a decreased ability to dissipate heat and increased heat production.

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In the initial stages of heat stress, cardiac output increases due to peripheral vasodilatation and decreased vascular resistance. As hyperthermia progresses, splanchnic blood pooling occurs and causes a decrease in circulating blood volume, resulting in hypotension. Cardiac output then starts to decline due to decreased vascular resistance and hypovolemia. With a decrease in circulating blood volume, heat loss through the mechanisms of radiation and convection fail, causing an elevation in body temperature and then heatstroke.

Global thermal injury causes multisystem organ dysfunction; organ damage occurs at temperatures above 109° F. At these higher temperatures, uncoupling of oxidative phosphorylation occurs, enzymes are denatured, and cell membrane functions are disrupted. The coagulation, renal, hepatic, gastrointestinal, and central nervous systems may all be affected, with mortality rates reported in dogs from 36% to 50%.^{1,2}

Types of Complications

Coagulation System

Thermal injury to the endothelium causes widespread vascular damage and initiates coagulation to help repair the injured vessels. In addition, extensive cell necrosis in multiple organs stimulates the release and consumption of coagulation factors and platelets. The severity of the thermal injury contributes to development of DIC in dogs with heatstroke. Direct thermal injury to bone marrow causes early release of nucleated red blood cells, which elevates the number of these cells in circulation.

Thrombocytopenia is a common result of thermal injury and may be due to direct heat damage to platelets or may be associated with DIC. In 2 retrospective studies of canine heatstroke, thrombocytopenia was a common finding, but it was not associated with a worse outcome. In 1 study, elevated PT and aPTT were associated with increased mortality.² In this study, DIC was diagnosed in dogs that also had thrombocytope-

nia and at least 2 of the following: prolonged PT, prolonged aPTT, and clinical signs compatible with DIC (petechiae, ecchymoses, hematochezia, hematemesis, hematuria). Dogs with a diagnosis of DIC, based on these parameters, were also more likely to die.²

Renal System

In the kidney, direct thermal injury damages the renal tubular epithelium. Decreased renal blood flow from hypoperfusion and microthrombosis secondary to DIC also contributes to the pathogenesis of renal failure. Depending on the severity of renal damage, oliguric or anuric renal failure may ensue.

Elevated creatinine levels were significantly associated with decreased survival in 2 studies.^{1,2} Azotemia may worsen for the first 24 to 48 hours, despite aggressive fluid therapy due to continued renal damage from excretion of excessive myoglobin, produced from thermal injury to muscle tissue. This derangement is manifested by elevations in serum creatinine kinase levels, which have been documented in many cases of canine heatstroke.

Hepatic Effects

The liver sustains direct thermal damage, causing widespread hepatic cellular necrosis. Hypoperfusion and microthrombosis from coagulation abnormalities may also contribute to hepatic dysfunction. Elevations in hepatic enzymes are so common that 1 author suggests that, in humans, normal levels of liver enzymes should place a diagnosis of heatstroke in doubt.³

Levels of ALT, AST, and ALP were elevated in 79% to 83% of dogs in 1 study.² Hepatic dysfunction may progress, evidenced by persistently elevated liver enzymes, elevated bilirubin, hypoglycemia, and hypocholesterolemia. Hypoglycemia is commonly found on presentation and may be due to hepatic dysfunction or increased use in response to an increased

demand for ATP in hyperthermic patients. Hypoglycemia on presentation was associated with a poorer outcome.²

Gastrointestinal System

Hypotension and thermal injury contribute to severe gastrointestinal epithelial damage and necrosis, resulting in hemorrhagic diarrhea, hematemesis, gastric ulceration, and potential for bacterial translocation into the bloodstream. In hypoperfused states, blood supply to the intestinal villus is shunted from the arteriole to the venule, thus decreasing oxygen delivery to the villus tip and intestinal mucosa. This results in significant hypoxia to the villi and mucosal lining, causing enterocyte death and intestinal mucosal sloughing.

Gastrointestinal ulceration and decreased absorptive capacity occur as a result of villus hypoxia. In addition, the gastrointestinal mucosa is an important barrier to significant bacterial translocation into the portal circulation. With intestinal mucosal damage, bacterial and endotoxin translocation is enhanced, increasing the risk for sepsis in these critically ill patients.

Central Nervous System

Dysfunction in the central nervous system may result from low cerebral perfusion, cerebral edema, or direct thermal injury to neurons, causing neuronal degeneration. Dogs often present in an altered mental state—disorientation, stupor, seizures, muscle tremors, and coma are common presenting signs.² In humans, the definition of heatstroke includes “hyperthermia with the presence of neurologic dysfunction”; however, it has been shown that the central nervous system in dogs is intrinsically resistant to thermal injury, allowing for higher core body temperatures before manifestation of central nervous system abnormalities.⁴

Prevention of Complications

Multiorgan dysfunction and failure are the sequelae to heatstroke in dogs, and the severity of organ malfunction is related to the degree

aPTT = activated partial thromboplastin time; ALP = alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; ATP = adenosine triphosphate; DIC = disseminated intravascular coagulation; PT = prothrombin time



A dog with multiple organ failure, including hepatic and oliguric renal failure, in a semicomatose state secondary to heatstroke

and duration of the hyperthermic state. Heatstroke most often occurs in the warm summer months and is especially common when humidity is high.

Owners of active dogs—including those involved in hunting, field trials, and scheduled runs—and working dogs should be educated about warning signs of heat exhaustion and heatstroke. Most owners are aware of the possibility of overheating and the need to rest their dogs when appropriate, provide plenty of fresh water, and cool them with shade and water hoses. Other preventable causes of heatstroke include prolonged exposure to cage dryers; confinement in a closed, unventilated area (such as a car); and extended muzzling.

Cooling the dog with possible heatstroke before presentation to a veterinarian is imperative. In 1 study, only 38% of dogs died when cooled before presentation compared with 61% that died if not cooled.² In another study, 16 of 42 dogs presenting with heatstroke were cooled by their owners, and rectal temperatures of these

dogs on presentation were significantly lower than those in dogs that were not cooled.¹ Owners should be instructed to use lukewarm water, as cold water or ice will cause peripheral vasoconstriction and lead to a decreased ability to lose heat through convective mechanisms. If severe signs of heatstroke are present, the owners should wet the dog down and immediately present it to a veterinary clinic for further therapy.

Factors associated with a poorer outcome include hypoglycemia, elevated PT and aPTT, delayed presentation (> 90 minutes after signs are evident), and elevated serum bilirubin and creatinine levels.^{1,2} Efforts should therefore be directed at preventing these complications, through aggressive cooling measures and optimizing organ perfusion.

Treatment of Complications

The progress of multiorgan failure depends on the duration of hyperthermia. Cooling measures should be aimed at lowering core body temperature while preventing hypothermia.

Cooling Methods

Methods of cooling include soaking with lukewarm water, covering the patient with towels soaked with lukewarm water, and placing a fan in front of patients to maximize heat loss through radiation and convection. Cool- and cold-water enemas have fallen out of favor, as further constriction of vascular beds of the gastrointestinal tract can worsen gastrointestinal ischemia and bacterial translocation.

Fluids & Blood Products

In every case of heatstroke, the goal of treatment is to increase organ perfusion. This is best accomplished through administration of intravenous fluids. Large doses of crystalloid fluids should be avoided in patients with altered mentation, as these dogs may be more prone to cerebral edema. However, cerebral perfusion must be maximized. To achieve perfusion while preventing tissue edema, both crystalloids and colloids can be used together.

The administration of fresh frozen plasma can help provide needed coagulation factors in the treatment and prevention of DIC. Many heatstroke patients require multiple doses of plasma over several days to treat coagulation abnormalities. A dose of 10 to 20 mL/kg/day should be used. PT, aPTT, and platelet levels should be checked on a daily basis.

Blood Pressure Support

Blood pressure should be supported and checked to assure maximum tissue perfusion. Vasopressors may be necessary in volume-resuscitated patients to maintain normal blood pressure. Dopamine, norepinephrine, dobutamine, and vasopressin are examples of medications used to support blood pressure in hypotensive, volume-resuscitated patients (**Table**).

Antibiotics

If signs of gastrointestinal compromise are present (vomiting, hemorrhagic diarrhea, ileus), broad-spectrum antibiotics may be administered to help prevent sepsis from bacterial translocation and may include a combination of antibiotics effective against gram-positive,

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Table. Medications for Blood Pressure Support

Drug	Dose	Comments
Dobutamine	2.5–10 µg/kg/min	<ul style="list-style-type: none"> • Selective β-1 agonist • Use with caution in cats
Dopamine	<i>Renal perfusion enhancement (dogs):</i> 2.5 µg/kg/min <i>Vasoconstriction:</i> 5–10 µg/kg/min	<ul style="list-style-type: none"> • Dopaminergic agonist at lower doses • α-1 and β-1 agonist at higher doses
Norepinephrine	0.05–1 µg/kg/min	Potent α-1 agonist
Vasopressin	0.001–0.004 U/kg/min	Vasoconstriction via V-1 receptors on vascular smooth muscle cells

gram-negative, and anaerobic bacteria. Examples include ampicillin and cefazolin (gram-positive and some anaerobes), enrofloxacin (gram-negative bacteria), and metronidazole (for anaerobes). Nonsteroidal antiinflammatory drugs and glucocorticoids should be avoided because of the potential for gastrointestinal ulceration and effects on renal perfusion.

Urine Output

Monitoring urine output is important in dogs with signs of oliguria or anuria accompanied by azotemia. A urinary catheter and closed-collection system should be placed, and urine should be collected and measured aseptically every 4 to 6 hours. If oliguria or anuria is present in a volume-resuscitated patient (urine output < 2 mL/kg/H), diuretics, such as furosemide or mannitol, can be administered as a constant-rate infusion to help stimulate urine production.

In summary, complications of heatstroke include DIC, acute renal failure, altered mentation, hepatic failure, and gastrointestinal dysfunction. Mortality ranges from 36% to 50%, highlighting the importance of early, aggressive therapy in dogs presenting with heatstroke. ■

See **Aids & Resources, back page, for references, contacts, and appendices.**

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TAKE-HOME MESSAGES

- Heatstroke may develop due to environmental or endogenous factors, is most commonly seen in the warmer months, and is often associated with higher humidity. However, exertional activity, confinement in an unventilated space, or prolonged exposure to dryers or other heating units can cause heatstroke at any time.
- Aggressive cooling measures should be instituted immediately, while avoiding hypothermia, as duration of hyperthermia is associated with a worse outcome and progression of organ failure.
- Prevention of complications of heatstroke include client and community education about avoiding situations associated with development of hyperthermia, and if heatstroke is present, maximizing tissue perfusion through the use of intravenous fluid therapy.

DIC = disseminated intravascular coagulation