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## KEY FACTS

- A thorough physical examination and complete history are important in diagnosing heatstroke in dogs because severe mentation abnormalities are associated with an unfavorable outcome.
- Heatstroke should be considered in any dog that presents with a core body temperature higher than 106°F (41.1°C).
- Systemic infections with fever usually do not cause panting and hypersalivation; therefore, clinical signs may give clues to help identify animals with heatstroke.
- The mortality of heatstroke in dogs can be significantly decreased if owners institute cooling measures before transporting their pet to the veterinarian.

# Heatstroke in Dogs: Clinical Signs, Treatment, Prognosis, and Prevention\*

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**ABSTRACT:** Heatstroke is an acute, life-threatening emergency with a complex pathophysiology—the key clinical features of which include metabolic acidosis, oliguric renal failure, coagulation abnormalities, and neurologic disturbances. Physical examination is marked by excessive panting, hyperemia, hypersalivation, tachycardia, and various neurologic signs. Common laboratory changes associated with heatstroke are hemoconcentration, elevated liver enzymes, electrolyte changes, prolonged clotting times, azotemia, and hypoglycemia. Rapid cooling of the core body and support of vital organs are essential factors in the management of heatstroke and prevention of further secondary sequelae. Prognosis worsens if severe neurologic signs develop and persist throughout the course of treatment. Owners of heatstroke animals can decrease mortality if the animal is cooled before being transported to the veterinarian. Prevention of heatstroke is achieved primarily by educating owners about proper acclimatization times, exercising during cooler periods of the day, and providing adequate shade and cool water for dogs confined outdoors.

**H**eatstroke is an acute, progressive life-threatening emergency characterized by a nonpyrogenic elevation in body temperature that results in direct thermal injury to body tissues. The disorder happens more often during the summer, especially in the southeastern United States. There are several diseases or certain physical conditions that could perpetuate hyperthermic events in dogs,<sup>1-3</sup> but heatstroke most often occurs after exercise or confinement in an enclosed area with poor ventilation, such as in an automobile.<sup>4,5</sup> Heatstroke requires early detection, aggressive therapeutic intervention, and continuous critical care monitoring to avoid any secondary complications and death.

Although its pathophysiology is complex, the physiologic state is precipitated by alterations in normal cooling functions resulting in improper thermoregulation and the body's inability to properly dissipate heat.<sup>3,6,7</sup> Key features of the syndrome include cardiovascular collapse, global endothelial damage, coagulation alterations, central nervous system dysfunction, loss of gastrointestinal integrity, acid-base disturbances, and sometimes sepsis and/or septic shock. This article discusses the clin-

\*See companion article on p. 410. The opinions or insertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.



Figure 1A



Figure 1B

**Figure 1**—Petechial hemorrhages on the dorsal antebrachium (A) and medial pinna of the ear (B) of a dog experiencing DIC during heatstroke.

ical presentation, the clinicopathologic findings, prognosis, prevention, and treatment of heatstroke in dogs.

### CLINICAL PRESENTATION

A complete physical examination should be performed on all dogs suspected of heatstroke. Because severe mentation abnormalities are reported to be associated with a poor outcome, particular attention should be directed toward determining the level of consciousness.<sup>8</sup> Heatstroke should be considered in any animal that presents with a core body temperature higher than 106°F (41°C), that has a consistent history of environmental exposure, and when other causes of hyperthermia have been excluded. However, it should be pointed out that some animals may have a normal or even subnormal temperature at the time of examination; this occurs especially if the owners have initiated treatment to cool down the animal before presentation or if the patient is in an advanced stage of shock.<sup>8</sup>

The most common clinical sign in patients with heatstroke is excessive panting.<sup>8,9</sup> The oral cavity and mucous membranes are usually tacky due to panting and extreme dehydration. Mucous membranes may also be darkened or hyperemic due to systemic vasodilation. The capillary refill time may be immediate or nondetectable. Dogs may exhibit ataxia, loss of consciousness, cortical blindness, seizures, or even coma. Dogs with cerebral edema can be initially stuporous and progress to involuntary paddling, course tremors, and obtundation. Brain stem reflexes (e.g., pupil, cornea) may also be diminished. The mucous membranes or sclera may reveal icterus due to massive hemolysis or hepatic dysfunction. In terminal stages of

heatstroke, shallow respirations and apnea may occur from neurologic dysfunction.

Examination of the mucous membranes, pinna, and vulva may reveal petechial hemorrhages or ecchymosis, indicating possible disseminated intravascular coagulation (DIC; Figure 1). Tachycardia with thready pulses is usually present due to extreme hypovolemia.<sup>10</sup> Pulse deficits may be noted if there is an arrhythmia. Melena, bloody diarrhea, or mucosal sloughing may be detected on rectal examination.<sup>5</sup> In addition, dark urine described as “machine-oil” or “coke-colored” may be present, indicating myoglobinuria.

Clinical presentation can sometimes give clues as to whether the animal's elevated temperature is pyrogenic or nonpyrogenic. Pyrogenic hyperthermia includes infectious and noninfectious systemic inflammatory diseases. Systemic inflammatory diseases characterized by elevated temperatures usually do not cause panting and hypersalivation in animals.<sup>2</sup> Pyrogenic animals also will usually be ambulatory, whereas many heatstroke animals are unwilling or unable to rise.<sup>8</sup>

A thorough history is very important in determining the reason for the hyperthermic event. Careful questioning may reveal that the dog was confined in a car on a hot day. Dog handlers and trainers may reveal that the dog collapsed after a working or athletic event. There also may be other pathologic diseases present that prevent proper heat dissipation, such as laryngeal paralysis or upper airway, neurologic, or cardiovascular disease. Loud breathing sounds may be observed on inspiration, suggesting underlying anatomic defects or diseases of the upper airway. Owners may report a recent change in the dog's bark, suggesting laryngeal disease.

Some dogs may be presented for hyperthermia with a syndrome that affects muscle metabolism. For example, eclampsia and other causes of hypocalcemia can be associated with elevated body temperature, muscle tremors, and altered mental state.<sup>3</sup> Ingestion of tremorgenic toxins, such as hexachlorophene, mycotoxins on moldy cheese or walnuts, organophosphates, or metaldehyde, has been reported to cause hyperthermia.<sup>11</sup> Both untreated hypocalcemia and toxin ingestion can cause the same sequelae as heatstroke because of excessive muscle activity from tremors. Recently, hyperthermia was reported in dogs with macadamia nut toxicosis.<sup>12</sup> Malignant hyperthermia is another syndrome that may be confused with heatstroke.<sup>3</sup> This syndrome causes a myopathy resulting in extreme muscle rigidity that is sometimes associated with administration of pharmacologic agents, such as halothane and succinylcholine chloride. There is also a genetic predisposition to the development of malignant hyperthermia in some dogs.<sup>13</sup>

### CLINICOPATHOLOGIC FINDINGS

Initial assessment tests of patients with clinical signs related to heatstroke include packed cell volume, total solids, peripheral blood smear evaluation, blood glucose, and estimation of whole blood urea nitrogen (BUN). Hemoconcentration (elevated hematocrit and total solids) associated with dehydration is commonly seen. Low total solids and anemia may be found in some dogs as the result of direct hyperthermic damage, gastrointestinal losses, vasculitis, or renal losses. One study reported poorer outcomes in dogs presented for heatstroke with hypoproteinemia; however, blood samples in that study may have been drawn after fluid resuscitation.<sup>8</sup> Blood glucose concentration is often decreased because of increased metabolic demands, hepatic dysfunction, or even sepsis.<sup>8</sup> BUN and creatinine levels may be elevated, especially during an acute renal failure crisis. In addition, prerenal factors may contribute to the azotemia through dehydration, poor perfusion, and hemoconcentration. Urine specific gravity also should be evaluated to assess urine-concentrating ability. Urine sediment should be examined for casts indicating renal tubular damage. Myoglobinuria is occasionally noted on urinalysis and indicates rhabdomyolysis. Hepatocellular damage usually results in elevated liver enzyme concentrations, particularly alanine transaminase, aspartate transaminase, and alkaline phosphatase.<sup>8,14</sup> Mild hyperbilirubinemia may also occur.<sup>8</sup> High levels of creatine phosphokinase indicate rhabdomyolysis and may peak at 24 to 48 hours before declining.

Coagulation abnormalities caused by thrombocytopenia, coagulation factor disruption or depletion, and DIC are common complications in canine heatstroke.<sup>10,15</sup> Thrombocytopenia, decreased fibrinogen levels, prolongation of activated partial thromboplastin time, prothrombin time, increased fibrin degradation products, and prolonged activated clotting time can be seen individually or in combination during DIC. Schistocytes may be present on a blood smear, lending support to a presumptive diagnosis of DIC. There may be increased leukocyte numbers; however, severely affected dogs may exhibit marked leukopenia. In addition, blood smears may reveal nucleated red blood cells; however, this finding is transient.

Various electrolyte abnormalities commonly occur with canine heatstroke. Hypernatremia is frequently present due to pure water loss.<sup>16,17</sup>

A mild hyperkalemia may also be present.<sup>10</sup> Hypophosphatemia and hypocalcemia may occur as well, although the mechanism of these changes is unknown.<sup>1</sup>

Blood gas analysis may reveal respiratory alkalosis reflecting hypocarbia secondary to excessive panting or metabolic acidosis reflecting lactic acid production associated with poor tissue perfusion or excessive muscle activity. Metabolic acidosis in dogs with heatstroke may also be caused by acute renal failure or diarrhea. In addition, mixed acid–base disorders (e.g., respiratory alkalosis, metabolic acidosis) commonly occur. Frequent monitoring of blood gases or total carbon dioxide is recommended during the initial resuscitation of heatstroke patients.

## TREATMENT

The primary goal of treatment for patients with heatstroke is to lower the body temperature quickly enough to prevent further damage to vital organ tissues, but not so fast as to cause hypothermia and induce heat-producing mechanisms. Rapid surface cooling can also produce peripheral vasoconstriction, which inhibits cooling mechanisms and shunts warm blood to core body organs. After cooling, sequelae that are secondary to heatstroke may develop and complicate patient status. It is imperative that the patient be monitored continuously for at least 24 to 48 hours after initial presentation.

### Measures Instituted by Owners

Heatstroke is a medical emergency; therefore, owners should institute treatment immediately by taking steps to progressively cool the animal to normal body temperature. If possible, the dog should be sprayed down by the owner before being transported to the veterinary hospital. Evaporation can be enhanced by driving with the windows open or placing the dog by the air conditioning vent as this will help with convective heat dissipation. One study showed a mortality rate of 49% for dogs that were not cooled by their owners versus 19% for those that were cooled before being transported to their veterinarian.<sup>8</sup> The following steps are recommended in treating heatstroke victims; however, steps A through D should take place simultaneously, particularly in unstable patients. Dosages for pharmacologic recommendations are listed in Table 1.

### Veterinary Treatment

**A. Ensure patent airway and provide oxygen.** On presentation to the clinic, the dog should be quickly evaluated for patent airway and oxygen should always be supplemented. Sedation may be necessary for dogs with laryngeal paralysis or for tracheal intubation. Intubation and positive pressure ventilation are necessary

for patients that are hypoventilating or in respiratory arrest. Oxygen cages are contraindicated because they can contribute to overheating.

**B. Provide external cooling.** The dog should be sprayed down with water or immersed in a cool water bath and placed in front of fans. Massaging may help with cooling by increasing blood flow and vasodilation. Ice water baths are contraindicated because they may actually cause vasoconstriction, decreased cutaneous blood flow, and capillary sludging, which promote DIC. Furthermore, ice water may cause a shivering response, which is a heat-producing mechanism. Internal cooling, such as cold water enemas and gastric lavages, has been suggested and may help decrease core body temperature.<sup>18</sup> However, these cooling methods are not practical and they impair ability to monitor temperature properly. A few ice packs may be placed on the dog's head to hasten cooling of the brain. Aggressive cooling efforts should be discontinued when the patient's temperature reaches 103°F (39.4°C) because it may continue to drop precipitously.

**C. Obtain a minimum database.** An IV catheter should be placed, and a minimum database that includes hematocrit, total solids, BUN, blood glucose, urine dipstick, and urine specific gravity, should be obtained. Pretreatment blood samples for complete blood cell count, serum chemistry profile, blood gas, and coagulation profile should be drawn at this time.

**D. Administer IV fluids.** IV fluid therapy is an important component in cooling core body temperature. Commence IV fluid therapy of room-temperature fluids to counteract cardiovascular shock. Crystalloid fluids (e.g., balanced electrolyte solutions) are usually the initial fluids of choice because of the need to rehydrate the interstitium. Colloids (e.g., hetastarch, dextran, plasma) may be used during the initial resuscitation period; however, some crystalloid therapy must follow shortly thereafter.<sup>19</sup> When both types of fluids are used together, the dose of crystalloids should be reduced 40% to 60%.<sup>20</sup> Patients that are hypoalbuminemic and have decreased colloid osmotic pressure may benefit from colloids. Because heatstroke victims suffer from hypotension and hypoxia, the strong colloidal and oxygen-carrying capabilities of the polymerized bovine hemoglobin glutamer-200 (Oxyglobin; Biopure Corporation) may benefit some dogs that are anemic and have low oncotic pressure.<sup>21,22</sup> Whole blood or packed red blood cell transfusions may be necessary to treat anemia after initial resuscitation. Fluids are administered to effect because alterations in fluid types and administration rates must be based on serial patient evaluations (e.g., heart rate, pulse quality, mucous membranes and capillary refill time, arterial blood pres-

**Table 1. Recommended Pharmacologic Treatments for Stabilization/Complications Associated with Heatstroke in Dogs**

<i>Treatment/Clinical Problem</i>	<i>Drug</i>	<i>Dose Regimen</i>
Sedation	Butorphanol	0.2–0.4 mg/kg IV
	Diazepam	0.1–0.5 mg/kg IV
	Propofol	5–7 mg/kg IV
IV fluids	Crystalloids (lactated Ringer's solution, Normosol-R); if sodium >160 mEq/dl, use 0.45% sodium chloride and 2.5% dextrose	90 ml/kg/hr (during initial resuscitation), then 2–6 ml/kg/hr
	Colloids (e.g., hetastarch, plasma)	10–20 ml/kg/day
	Oxyglobin	10–30 ml/kg; 10 ml/hr
Antibiotics	Ampicillin	22 mg/kg q8h IV
	Enrofloxacin	5–7 mg/kg diluted 50:50 in saline q12h IV
	Cefazolin	22 mg/kg q8h IV
Gastrointestinal protection	Cimetidine	5–10 mg/kg q8h IV
	Ranitidine	0.5 mg/kg q12h IV
	Sucralfate	Large dogs: 1 g PO tid Medium dogs: ½ g PO tid Small dogs: ¼ g PO tid
DIC	Fresh-frozen plasma	10–20 ml/kg q4h IV
	Heparin	50–100 U/kg q8h SC; taper dose over several days
Metabolic acidosis	Sodium bicarbonate	Calculated dose (mEq) = $0.3 \times \text{wt (kg)} \times \text{base deficit}$ . Give 25%–50% of calculated dose slowly IV; repeat blood gas 30 min later
Hypokalemia (20 mEq/10 ml)	Potassium chloride	Give no more than 0.5 mEq/kg/hr IV
Ventricular arrhythmias	Lidocaine (2%)	2–4 mg/kg slow IV until arrhythmias resolve, 50–80 µg/kg/min IV CRI
	Procainamide	10–30 mg/kg q6h PO, 8–20 mg/kg IV or IM, 25–50 µg/kg/min IV CRI
Oliguric renal failure	Mannitol	0.5–1.0 g/kg IV
	Dopamine	3 µg/kg/min IV
	Furosemide	2 mg/kg IV; 1 mg/kg/hr
Pulmonary edema	Furosemide	2–4 mg/kg IV
Central neurologic treatment	Mannitol	1 g/kg IV
	Diazepam	0.5–1.0 mg/kg IV
Shock or hypotension	Dopamine or dobutamine	5–10 µg/kg/min IV CRI
	Dexamethasone SP	2–8 mg/kg IV
	Prednisolone sodium succinate	10–25 mg/kg IV

CRI = constant-rate infusion.

sure, central venous blood pressure). Central venous blood pressure monitoring is helpful in assessing intravascular volume and preventing excessive fluid administration.<sup>23</sup>

**E. Administer antibiotics.** The use of prophylactic antibiotic therapy is controversial because of the potential to induce resistance and worsen endotoxemia. If considered, administration of parenteral antibiotics that provide a bactericidal combination effective against most gram-negative, gram-positive, and anaerobic bacteria is recommended.

**F. Correct hypoglycemia.** Supplement dextrose in IV fluids as needed.

**G. Protect the GI tract.** An H<sub>2</sub> blocker, such as cimetidine or ranitidine, may be given 1 hour after administration of sucralfate. Administration of oral medications in debilitated patients with potentially compromised swallowing reflexes may cause aspiration and, therefore, should be used with caution.

**H. Treat for DIC.** Fresh-frozen plasma should be administered in dogs with DIC.<sup>24</sup> Consider incubating the plasma with heparin for 30 minutes before administration.<sup>24</sup> Unless active hemorrhage is occurring, heparin may also be given parenterally.

**I. Correct acid-base and electrolyte abnormalities.** Correction of perfusion deficits often leads to improvement in the degree of acidemia. However, if acidemia persists after adequate resuscitation, sodium bicarbonate can be given to correct metabolic acidosis (pH <7.2). For correction of hypokalemia, administer potassium chloride with maintenance IV fluids after initial resuscitation.

**J. Monitor for arrhythmias.** If ventricular arrhythmias should arise causing hemodynamic compromise, then treatment should be considered. Electrolyte abnormalities should be corrected before administration of antiarrhythmics because cardiac arrhythmias may be difficult to manage if electrolytes are imbalanced. Lidocaine is commonly administered as a slow IV bolus followed by a constant-rate infusion for 48 to 72 hours until arrhythmias subside.

**K. Monitor urine output.** Because acute renal failure is a potential sequela, urine output should be monitored. If urine output becomes oliguric (<1–2 ml/kg/hr) after the patient is well hydrated, administration of mannitol, dopamine, and furosemide should be considered. Prior to diuretic administration, the patient must be well hydrated and should have adequate mean arterial blood pressure to ensure effective renal perfusion. After urine flow is initiated, fluid therapy should be continued at a rate of 4 to 6 ml/kg/hr and tapered off, depending on serial patient evaluations (e.g., heart rate, pulse quality, mucous membranes and capillary refill time, arterial blood pressures, central venous blood pres-

ures). Urine sediment, BUN, and creatinine should be rechecked frequently. A closed urinary collection system will help measure urine production, ensure that fluid therapy is adequate, and facilitate patient management.

**L. Treat pulmonary edema.** Respiratory sounds should be auscultated frequently during the initial resuscitation period. If pulmonary edema is suspected or confirmed with radiography, central venous pressure should be monitored and furosemide administration should be considered.

**M. Monitor neurologic function.** The patient's neurologic status should be reevaluated constantly. If it is deteriorating, mannitol should be considered. Hypoglycemia also should be corrected. Seizures should be controlled with diazepam as needed.

**N. Treat hypotension.** If mean blood pressure falls below 60 mm Hg after fluid resuscitation, dopamine or dobutamine hydrochloride should be considered. Doses should be adjusted according to blood pressure monitoring and clinical response. Septic shock should be considered if the animal remains hypotensive after fluid resuscitation and is septic.

## Drugs Used for Treatment

Corticosteroid use is controversial; however, corticosteroids (prednisolone sodium succinate, dexamethasone sodium phosphate) may prevent cerebral edema and stabilize membranes in some patients experiencing shock. Complications associated with corticosteroids include gastric ulceration to an already underperfused gastrointestinal system, immunosuppression, and exacerbation of heatstroke-induced renal damage.<sup>1,16,25</sup> One study recently demonstrated that glucocorticoid administration reduced interleukin-1 concentrations and resulted in neuroprotective effects in rats with heatstroke.<sup>26</sup>

NSAIDs, such as dipyrone and flunixin meglumine, are contraindicated for heatstroke because they can cause severe hypothermia, gastrointestinal ulceration, prolonged bleeding times, and bone marrow suppression.<sup>27</sup> Antiprostaglandins are generally only effective in lowering body temperature in dogs with a true fever and are not indicated with environmentally induced hyperthermia.

\* \* \*

The key to successful treatment and recovery of heatstroke is early recognition and treatment. Once the animal's temperature is reduced and its condition is stabilized, frequent monitoring of acid-base status, packed cell volume, white blood cell count, total solids, BUN, and platelet count is necessary. Serial patient evaluations are also critical to identify subtle hemodynamic changes that may occur secondary to complications. Further-

more, diagnostic tests should be conducted to determine any underlying disease process that may have precipitated the hyperthermic event. Although heatstroke presents a therapeutic challenge to veterinarians, the survival rate in one series of cases was reported to be as high as 64%.<sup>8</sup>

## PROGNOSIS

There are specific unfavorable criteria that have been associated with poorer outcomes in dogs with heatstroke<sup>28</sup> (see box on page 430). One study reported that dogs that died from heat-related illness usually died during the initial 24 hours of treatment.<sup>8</sup> Length of hospitalization depends on how quickly the animal responds and can range from several days to weeks if complications arise. In one study, dogs that were hospitalized more than 72 hours all survived, despite the fact that many had evidence of multiorgan involvement.<sup>8</sup> Animals that recover are usually those whose temperatures are returned to normal early in the course of the disease because the longer the animal remains severely hyperthermic, the greater is the damage that occurs to vital organ systems. Owners need to be aware of potential developments associated with permanent damage to the kidneys, heart, and liver. Heatstroke victims may have residual neurologic deficits and are probably predisposed to repetitive heat injuries in the future.

## PREVENTION

In order to prevent heatstroke, owners must be educated about the risk of confining or exercising dogs in hot environments. To prevent exertional heatstroke in working animals, outdoor duties or events should be scheduled during the cooler parts of the day, if possible. Only well-conditioned animals should be subjected to short periods of vigorous activity in hot environments. It has been demonstrated in humans that 7 to 21 days are required to partially accli-

mate to a hot environment.<sup>1,29,30</sup> It seems likely that a similar time frame would be required in dogs; although, it may take up to 2 months for full acclimation to occur in some dogs.<sup>28</sup> Improper acclimatization may be the reason why more cases of heatstroke in dogs were reported in the early summer.<sup>8</sup> Furthermore, heat stress can be prevented by simply providing adequate ventilation, a place where the dog can get out of direct sunlight, and free access to cool drinking water.

## SUMMARY

Clinical signs and clinicopathologic findings associated with heatstroke vary depending on the length of

### Unfavorable Prognostic Criteria for Heatstroke

1. Animals that present in a coma or show progressive neurologic deterioration
2. Hypothermia on presentation
3. Persistent hypoglycemia
4. Worsening azotemia despite adequate fluid therapy
5. Evidence of DIC
6. Refractory hypotension
7. Elevated total bilirubin
8. Ventricular arrhythmia event
9. Persistent hypoproteinemia
10. Labored respiration, pulmonary edema
11. Persistent oliguria despite adequate fluid loading

time the patient is exposed to hyperthermia. Rapid diagnosis is critical to ensure that therapy can be started immediately and complications prevented. The primary treatment goal for patients with heatstroke is to lower the core body temperature with effective cooling measures quickly enough to prevent further damage to vital organ systems. Initial support also includes aggressive fluid therapy, gastrointestinal protection, and correction of electrolyte and acid-base disorders. Continuous critical monitoring of heatstroke patients is vital for the first 24 to 48 hours, and additional pharmacology and therapy may be necessary to treat secondary complications.

As in humans, the prognosis for dogs with heatstroke depends on the duration and severity of hyperthermia before treatment is instituted. Prognosis also depends upon the presence or absence of any underlying disease. The potential for developing life-threatening complications is great; thus the prognosis is guarded for most dogs initially. Owners can improve their dog's chance for survival by following effective cooling instructions provided by their veterinarian prior to transporting their dog to the hospital. Heatstroke can be easily prevented if measures are taken to allow for proper acclimation, adequate ventilation, and free access to shade and cool drinking water.

### REFERENCES

1. Curley FJ, Irwin RS: Disorders of temperature control: Hyperthermia, in Rippe JM, Irwin RS, Fink MP, Cerra FB (eds): *Intensive Care Medicine*, ed 3. Boston, Little Brown and Co, 1996, pp 859–874.
2. Ruslander D: Heat stroke, in Kirk RW, Bonagura JD (eds): *Kirk's Current Veterinary Therapy XI: Small Animal Practice*. Philadelphia, WB Saunders, 1992, pp 143–146.
3. Miller JB: Hyperthermia and hypothermia, in Ettinger SJ, Feldman EC (eds): *Textbook of Veterinary Internal Medicine*, ed 5. Philadelphia, WB Saunders, 2000, pp 6–10.
4. Gregory NG, Constantine E: Hyperthermia in dogs left in cars. *Vet Rec* 139:349–350, 1996.
5. Macintire DK: Heatstroke in canine athletes. *Bull Sports Med Prog Spring*:1–2, 1999.
6. Ganong WF: Central regulation of visceral function: Temperature regulation, in Ganong WF (ed): *Review of Medical Physiology*, ed 19. Stanford, CA, Appleton & Lange, 1999, pp 238–243.
7. Guyton A: Body temperature, temperature regulation and fever, in Guyton A (ed): *Textbook of Medical Physiology*, ed 8. Philadelphia, WB Saunders, 1991, pp 797–807.
8. Drobatz KJ, Macintire DK: Heat-induced illness in dogs: 42 cases (1976–1993). *JAVMA* 209(11):1894–1899, 1996.
9. Larson RL, Carithers RW: A review of heat stroke and its complications in the canine. *N Z Vet J* 33:202–206, 1985.
10. Johnson K: Pathophysiology of heat stroke. *Compend Contin Educ Pract Vet* 4(2):141–145, 1982.
11. Lipitz D, Chauvet AE: Shivering and trembling, in Ettinger SJ, Feldman EC (eds): *Textbook of Veterinary Internal Medicine*, ed 5. Philadelphia, WB Saunders, 2000, pp 139–141.
12. Hansen SR: Macadamia nut toxicosis in dogs. *Vet Med April*:274, 276, 2001.
13. Nelson TE: Malignant hyperthermia in dogs. *JAVMA* 198(6):989–994, 1991.
14. Magazinik A, Shapiro Y, Neuman F, Sohar E: Enzyme changes of exercise and heatstroke in dogs. *Israel J Med Sci* 15:622, 1979.
15. Rosenthal T, Shapiro Y, Seligsohn V, et al: Disseminated intravascular coagulation in experimental heat stroke. *Thromb Diath Haemor* 26:417–425, 1971.
16. Holloway SA: Heatstroke in dogs. *Compend Contin Educ Pract Vet* 14:1598–1604, 1992.
17. Krum SH, Osborne CA: Heatstroke in the dog: A polysystemic disorder. *JAVMA* 170(5):531–35, 1977.
18. Lee-Chiong Jr TL: Disorders of temperature regulation. *Compr Ther* 21(12):697–704, 1995.
19. Raffe M: Medical use of colloids, in Bonagura JD (ed): *Kirk's Current Veterinary Therapy XIII: Small Animal Practice*. Philadelphia, WB Saunders, 2000, pp 66–68.
20. Kirby R, Rudloff E: The critical need for colloids: Maintaining fluid balance. *Compend Contin Educ Pract Vet* 19(6):705–719, 1997.
21. Day TK: Canine heatstroke. *Compend Contin Educ Pract Vet* 23(Supp 7A):4–7, 2001.
22. Harringer W, Hodakowski GT, Svizzero T, et al: Acute effects of massive transfusion of a bovine hemoglobin blood substitute in a canine model of hemorrhagic shock. *Eur J Cardio-Thor Surg* 6:649–654, 1992.
23. Macintire DK: Hypotension, in Ettinger SJ, Feldman EC (eds): *Textbook of Veterinary Internal Medicine*, ed 5. Philadelphia, WB Saunders, 2000, pp 183–186.
24. Feldman BF, Kirby R, Caldin M: Recognition and treatment of disseminated intravascular coagulation, in Bonagura JD (ed): *Kirk's Current Veterinary Therapy XIII: Small Animal Practice*. Philadelphia, WB Saunders, 2000, pp 190–194.
25. Stanton ME, Bright RM: Gastroduodenal ulceration in dogs. *J Vet Intern Med* 3:238–244, 1989.
26. Liu CC, Chien CH, Lin MT: Glucocorticoids reduce inter-

leukin-1 concentration and result in neuroprotective effects in rat heatstroke. *J Physiol* 527(Pt 2):333–343, 2000.

27. Mathews K: Nonsteroidal anti-inflammatory analgesics. Indications and contraindications for pain management in dogs and cats. *Vet Clin North Am Small Anim Pract* 30(4):783–804, 2000.
28. Rozanski EA, Boysen S: Heatstroke. *Stand Care: Emerg Crit Care Med* 3(2):4–8, 2001.
29. Senay LC, Mitchell D, Wyndham CH: Acclimatization in a hot, humid environment: Body fluid adjustments. *J Appl Physiol* 40:786–796, 1976.
30. Aoyagi Y, McLellan TM, Shephard RJ: Interaction of physical training and heat acclimation. The thermophysiology of exercising in a hot climate. *Sports Med* 23:173–210, 1997.

### ARTICLE #2 CE TEST

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. Choose the best answer to each of the following questions; then mark your answers on the postage-paid envelope inserted in *Compendium*.

1. All of the following physical examination findings are commonly seen in dogs with heatstroke except
  - a. excessive panting.
  - b. hypersalivation.
  - c. hyperemic mucous membranes.
  - d. tachycardia.
  - e. bradycardia.
2. Which of the following is not a clinical pathologic finding in canine heatstroke?
  - a. elevated alanine transaminase
  - b. decreased alkaline phosphatase
  - c. elevated hematocrit
  - d. hypoglycemia
3. DIC is suspected if there is
  - a. thrombocytopenia, prolonged prothrombin time, and elevated activated partial thromboplastin time.
  - b. prolonged prothrombin time, prolonged activated partial thromboplastin time, and elevated fibrin degradation products.
  - c. prolonged activated partial thromboplastin time, decreased fibrin degradation products, and decreased fibrinogen.
  - d. elevated fibrin degradation products, elevated fibrinogen, and prolonged activated partial thromboplastin time.
  - e. thrombocytopenia, elevated fibrin degradation products, and elevated fibrinogen.
4. The initial and primary goal of treatment for patients with heatstroke is to
  - a. lower the core body temperature as quickly as possible.
  - b. treat for electrolyte disturbances.
  - c. start gastrointestinal protection.
  - d. correct acid–base abnormalities.
5. Which of the following is not an accepted method of cooling the body down during heatstroke?
  - a. spraying down the body with water and placing the patient in front of a fan
  - b. immersing the body totally in ice cold water
  - c. placing ice packs on the patient's head
  - d. administering cool water enemas
6. Patients with oliguric or anuric renal failure are best treated with
  - a. dexamethasone SP.
  - b. rapid infusion of 0.9% sodium chloride.
  - c. dopamine and furosemide.
  - d. dipyrone.
7. Unfavorable criteria that have been associated with poorer outcomes of dogs with heatstroke include all of the following except
  - a. persistent hypoglycemia.
  - b. evidence of DIC.
  - c. coma presentation.
  - d. persistently elevated alanine transaminase.
8. Heatstroke in dogs can be prevented primarily by
  - a. educating owners concerning the risks of confining animals outdoors.
  - b. allowing full acclimatization for animals prior to exercise in hot environments.
  - c. providing adequate ventilation and shade and allowing free access to cool drinking water.
  - d. all of the above
9. It may take \_\_\_\_\_ for some dogs to fully acclimate to a hot environment.
 

a. 1 week	c. 2 months
b. 28 days	d. 45 days
10. \_\_\_\_\_ are common electrolyte changes that occur during heatstroke.
  - a. Hypernatremia and hyperchloremia
  - b. Hyponatremia and hyperchloremia
  - c. Hypokalemia and hyponatremia
  - d. Hyperkalemia and hyponatremia