

Compendium

Heatstroke: Clinical Signs, Diagnosis, Treatment, and Prognosis

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Abstract: Heatstroke is a complex disease process that, in its most severe form, can result in multiorgan dysfunction and death. Heatstroke stems from the failure of the body's thermoregulatory mechanisms, resulting in cellular damage and death. The organ systems most commonly affected in this disease process include the gastrointestinal tract and the coagulation, renal, cardiac, pulmonary, and central nervous systems. Heatstroke is diagnosed based on the patient history, physical examination, and clinicopathologic findings. Treatment should be instituted immediately to improve patient outcome and includes active cooling, fluid resuscitation, and supportive care. Patients with altered mental status, hypoglycemia, prolonged prothrombin time, and prolonged activated partial thromboplastin time at admission have increased mortality rates. Additional negative prognostic indicators include elevated serum creatinine level, delayed admission to the hospital, seizures, and obesity.

For more information, please see the companion article, "Heatstroke: Thermoregulation, Pathophysiology, and Predisposing Factors."

Heatstroke is the most severe form of heat-related illness. Recently, a new definition of heatstroke for human patients has been proposed: "A form of hyperthermia associated with a systemic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates."¹ A similar disease process has been seen in veterinary patients. Regardless of the definition, this disease process is often progressive and can be life threatening.

Multiorgan dysfunction is the hallmark of heatstroke. Affected organ systems often include the gastrointestinal tract and the coagulation, renal, pulmonary, and central nervous systems. Common sequelae of heatstroke include disseminated intravascular coagulation (DIC), acute lung injury or acute respiratory distress syndrome, and acute kidney injury. This article discusses the clinical signs and diagnosis of, as well as treatment options and prognosis for, heatstroke.

Clinical Signs and Diagnosis History and Patient Assessment

Patients suffering from heat-related illnesses have clinical signs that vary greatly depending on the severity of the disease. Therefore, a thorough history is key in the initial stages of heatstroke management. Often, the history includes exposure to increased environmental temperatures or strenuous exercise without acclimatization.

During the initial evaluation, owners should be questioned about underlying medical conditions and concurrent medication administration. Known predisposing factors or heatstroke include obesity, cardiovascular disease, neurologic/neuromuscular disease, upper airway abnormalities, strenuous exercise, and confinement with poor ventilation.

Patient assessment and treatment should be performed simultaneously. Initial assessment of the patient using the CAB (cardiovascular-airway-breathing) approach is essential for all patients that are potentially suffering from a heat-related illness. The patient should be assessed for the presence of a heartbeat, a patent airway, and normal respiratory excursions. After the initial assessment is done, a second, more comprehensive examination of all body systems should be performed and appropriate therapies initiated (**FIGURE 1**).

Evaluation of the patient's cardiopulmonary systems to determine if signs of shock are present should include a visual assessment in conjunction with thoracic auscultation and assessment of perfusion parameters (mentation, pulse quality, mucous membrane color, capillary refill time; **TABLE 1**).

Apnea or agonal breathing is an indication for immediate intubation to secure the patient's airway. Breathing patients should be examined closely for evidence of upper airway obstruction (inspiratory stridor), abnormal respiratory patterns, or both, as upper and lower airway disease can be concurrent in patients with heatstroke. Systematic auscultation of the thoracic cavity aids in identifying cardiac arrhythmias, murmurs, and adventitious breath

Table 1: Perfusion Parameters and Potential Stages of Shock⁴

Mucous Membrane Color	Capillary Refill Time	Pulse Quality	Stage of Shock
Normal	Normal or prolonged	Normal to hyperdynamic	Compensatory
Pale	Prolonged	Fair to poor	Decompensatory
Hyperemic	Fast to normal	Variable	Decompensatory

sounds, further directing treatment and diagnostic recommendations. Most heatstroke patients are tachycardic and tachypneic, but heart and respiratory rates vary depending on the presence and stage of shock. Potential cardiac arrhythmias should be evaluated further by electrocardiography; based on a 2006 retrospective study,² up to 25% of patients presenting with heatstroke have confirmed arrhythmias.

A focused neurologic examination should be performed at initial presentation and include the evaluation of patient mental status, behavior, and cranial nerves. Mental status can range from normal to obtunded or comatose. Heatstroke patients can also present with ongoing seizure activity or in a postictal state. In a recent retrospective study of canine heatstroke patients, neurologic abnormalities were common; 35% of patients presented with seizure activity at or before presentation. Of this population, 47% had an obtunded mental status and 24% presented in a comatose state.² The presence of seizure activity, altered mental status, or both is associated with an increased risk for death in canine heatstroke patients. A complete neurologic assessment, including gait (if ambulating), postural reactions, and spinal reflexes, should be performed once the patient is stable. Serial neurologic exams can be used to monitor the response to treatment and detect deterioration in

the patient's status. In patients with neurologic abnormalities, a modified Glasgow Coma Scale is recommended to facilitate serial neurologic exams.³

The rectal temperature of patients with heatstroke can indicate hypothermia, normothermia, or hyperthermia.⁴ It is important to remember that patients that are affected severely by heat-related illnesses may have a normal or subnormal presenting temperature. This may be caused by previous treatments by owners or referring veterinarians or by the presence of varying stages of shock.

Hydration should be assessed to guide fluid therapy planning. Evaluation of the patient's mucous membranes (dry versus moist) and skin turgor can help determine the degree of dehydration. Mucous membranes and unhaired skin should be examined for evidence of hemorrhage (petechiae or ecchymoses) because of the risk of developing coagulopathies or DIC caused by direct thermal cytotoxicity. A rectal exam should be performed to evaluate for evidence of overt gastrointestinal hemorrhage, mucosal sloughing, or melena.⁵ Abdominal palpation can help identify underlying medical conditions through detection of intraabdominal neoplasia, organomegaly, ascites (palpable fluid wave), or pain. These findings can help guide diagnosis and treatment recommendations.

Diagnostics

Blood should be collected during the initial assessment and can be drawn from an IV catheter during placement to provide baseline values for parameters to be monitored throughout treatment. Immediate point-of-care tests, including packed cell volume/total solids, blood glucose, electrolytes, blood gases, lactate, and kidney values (blood urea nitrogen/creatinine) can be used to guide the

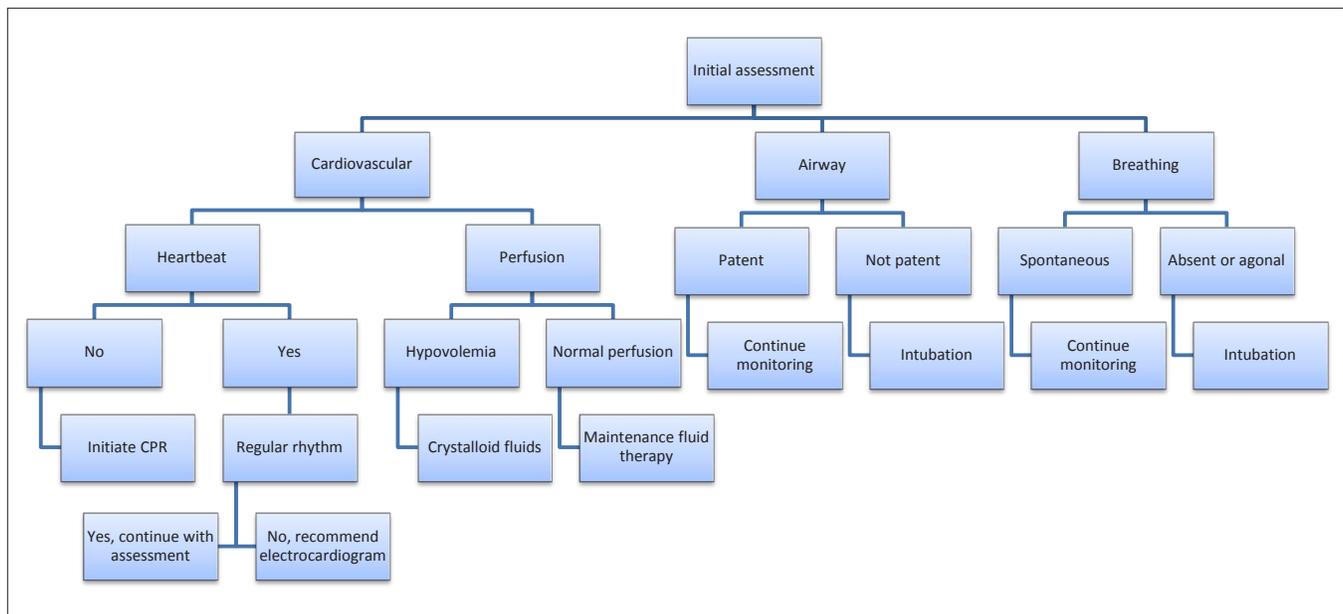


Figure 1. Algorithm for initial patient assessment.

initial therapeutic plan. A complete database should also be performed, including a complete blood count (CBC), serum chemistry panel, urinalysis, and coagulation testing.

Common abnormalities on the CBC include leukocytosis or leukopenia, anemia, hemoconcentration/polycythemia, the presence of nucleated red blood cells (nRBC), and thrombocytopenia. Leukocytosis could indicate an acute inflammatory response from the episode of heat stress, a response to infection, or an underlying bone marrow disease. Leukopenia can be seen secondary to sepsis or bone marrow disease. Anemia could indicate acute blood loss from hemorrhage or anemia of chronic disease associated with a preexisting medical condition. A blood smear should also be evaluated for the presence of nRBC. In a 2009 retrospective study, 90% of dogs with heatstroke had nRBC noted on a blood smear.⁶ This finding is unique to dogs and has not been noted with human heatstroke patients. The presence of nRBC was speculated to be caused by direct thermal cytotoxicity to the patient's bone marrow.⁶ Thrombocytopenia was seen in 83% of canine heatstroke patients in a 2006 retrospective study.² The authors of this paper proposed the following causes for thrombocytopenia in the study's population: secondary platelet consumption caused by vasculitis, gastrointestinal bleeding, and hyperthermia-induced platelet aggregation.²

Chemistry panel abnormalities can include elevations of alanine aminotransferase, total bilirubin, blood urea nitrogen, creatinine, creatine kinase, and glucose levels. Canine patients with heatstroke commonly have elevations in alanine aminotransferase and bilirubin levels caused by direct hepatocellular hypoxia.^{4,5} Elevations of blood urea nitrogen and creatinine levels can be prerenal as a result of dehydration, renal as a result of direct cytotoxicity and ischemic insults, or both. Rising creatinine levels during fluid therapy have been linked with increased mortality in canine heatstroke patients.⁴ Creatine kinase elevation is expected as a result of diffuse muscle damage (rhabdomyolysis).⁴ Alterations in the patient's blood glucose can be multifactorial. Hypoglycemia can be secondary to sepsis from gastrointestinal bacterial translocation, hepatocellular injury, prolonged seizure activity, or an unrelated preexisting condition (e.g., insulinoma).^{4,5} Persistent hypoglycemia despite treatment was associated with increased mortality in canine heatstroke patients.^{4,7}

Acid-base and electrolyte abnormalities can include metabolic acidosis with compensatory respiratory alkalosis⁵ and alterations in sodium, potassium, phosphorus, and calcium. Metabolic acidosis can be caused by global hypoxia that can lead to lactic acid production from anaerobic metabolism, or by uremic acids in patients with impaired renal function.⁸ A patient's sodium level can be highly variable with heatstroke. Hyponatremia can be caused by pure water loss and dehydration, while hyponatremia can be caused by increased hypertonic fluid loss (e.g., vomiting, diarrhea).^{8,9} Hyperkalemia can be seen as a result of cellular death, particularly with rhabdomyolysis.¹⁰ Hypophosphatemia and hypocalcemia may be seen; however, the underlying mechanism is unknown.^{8,11}

A urinalysis should be completed to evaluate the patient's urine concentrating ability and to detect the presence of myoglobin,

bilirubin, or casts within the urine. Confirming a normal to elevated urine specific gravity at admission can serve as a baseline for evaluation of the patient's hydration status and current renal function. Isosthenuria at admission may indicate a preexisting or secondary kidney injury.

Coagulation testing should be pursued in all heatstroke patients, especially those with suspected active blood loss (hematochezia, hematemesis, petechiae/ecchymoses, anemia with hypoproteinemia). Baseline coagulation testing includes a partial thromboplastin time, prothrombin time, and platelet count. If platelet dysfunction is suspected, a buccal mucosal bleeding time should be performed. If available, a thromboelastogram is useful in evaluating all aspects of coagulation and identifying both hypo- and hypercoagulable patients. As mentioned earlier, heatstroke patients are at risk for development of DIC. Early and serial evaluations of a peripheral blood smear are recommended. The presence of schistocytes may support a diagnosis of DIC.⁵ Additional hematologic changes commonly associated with DIC that may help to confirm the diagnosis include thrombocytopenia, prolonged prothrombin time, prolonged activated partial thromboplastin time, hypo- or hyperfibrinogenemia, low antithrombin levels, and the presence of fibrin degradation products or D-dimers.¹²

Thoracic radiography should be performed for any patient with cardiopulmonary dysfunction, such as abnormal lung auscultation (adventitious breath sounds), persistent hypoxemia, irregular breathing patterns, heart murmur, or cardiac arrhythmia. Patients with respiratory compromise can benefit from serial pulse oximetry readings, blood gas analyses, or both to identify and characterize underlying hypoxemia and assess response to oxygen therapy. Additional advanced diagnostic testing (e.g., abdominal/thoracic ultrasonography, magnetic resonance imaging) should be considered in geriatric patients or those presenting with non-exertional heatstroke without a known predisposing factor(s).

Treatment

Treatment should begin as soon as possible to increase the chance of survival and should be prioritized to address the most critical clinical abnormalities identified on the primary survey (TABLE 2).

Treatment Options for Owners at Home

Owners who contact a hospital with a patient with possible heatstroke should be encouraged to actively cool the patient while en route to the hospital. At-home cooling should not delay arrival to the hospital but rather be performed simultaneously. Active cooling can be accomplished by applying cool water to the patient, either with a hose or other water source or with presoaked towels placed over the patient. This assists in heat dissipation through evaporation and convection when used in conjunction with open windows or air conditioning while driving to the hospital.⁸ If this cannot be accomplished safely, it should be avoided and the patient should be brought to the hospital immediately. Patients should not be submerged in a cold-water bath due to the risk of drowning if neurologically impaired; in addition, extreme peripheral vasoconstriction may impair heat dissipation.

Hospital-initiated Treatment

Patients that are obtunded or comatose at presentation should be intubated immediately and their airway secured. Supplemental oxygen and intermittent positive-pressure ventilation should be administered until spontaneous breathing is deemed adequate. Initial treatment should be targeted at reducing ongoing hyperthermia and correcting cardiovascular collapse to minimize further organ injury.

Active Cooling

Active cooling is an essential part of treatment for heatstroke patients and is achieved by maximizing heat dissipation mechanisms. Cooling techniques should be instituted, including applying cool water to the patient while increasing air circulation over the body with fans to improve evaporation. Care should be taken in soaking patients with thick haircoats because wet hair can act as an insulation barrier and limit heat dissipation. Thinly haired areas (ventral abdomen, axilla, and inguinal regions) should be cooled by applying towels soaked with cool water. Ice packs should be avoided to prevent cutaneous vasoconstriction that may cause shunting of heated blood to central visceral organs.⁵ Massaging extremities is a technique used in human medicine to improve peripheral circulation during active cooling.¹³ Active cooling should be stopped when the patient's temperature is 39.5 °C (103.1°F) because cooling will continue and there is a risk of progression to hypothermia. A 2007 study reported that cold-water immersion in human patients with exertional heatstroke improved morbidity and mortality but may be detrimental in cooling patients with nonexertional heatstroke.¹⁴ Immersion is not currently recommended in veterinary patients due to the high prevalence of nonexertional heatstroke. Otherwise, no means has proven superior to immersion in reducing core body temperature while reducing mortality/morbidity in human heatstroke patients.¹⁵

Cold-water enemas and gastric lavage have been suggested in literature. These techniques have been associated with adverse outcomes; gastric lavage is associated with an increased risk of aspiration pneumonia, and both techniques may further gastrointestinal permeability. Therefore, these techniques cannot be recommended at this time. Peritoneal lavage has been investigated in animal studies with mild increase in survival versus control groups but is not currently recommended in human medicine.^{15,16} Pharmacologic agents to hasten cooling or reduce core temperature (e.g., NSAIDs, dantrolene) have not been proven beneficial and are discouraged because of potential negative effects on organ function (e.g., acute kidney injury, gastric ulceration).^{9,17} Other novel and experimental methods of active cooling for human exertional heatstroke patients have included noninvasive external cooling systems, hyperbaric oxygen therapy, and endovascular cooling devices, but they have yet to be validated in veterinary patients.^{18–20}

Cardiovascular Support

Heatstroke is characterized by a state of distributive shock with absolute or relative volume deficit. As a result, fluid resuscitation

should be instituted during the process of active cooling. Restoring the patient's circulating volume improves cutaneous vasodilation and perfusion of visceral organs.

The initial goal of fluid therapy is to quickly restore perfusion. Balanced electrolyte crystalloid fluids are recommended as the initial fluid choice. Fluids should be administered as incremental boluses (e.g., 10 to 20 mL/kg over 10 to 15 min) with subsequent patient reassessment to guide further administration. Supplementation with synthetic colloid boluses should be considered in hypoproteinemic patients or those refractory to large-volume crystalloid administration. Excessive crystalloid therapy may lead to dilutional coagulopathy and worsen already low colloid osmotic pressure in hypoalbuminemic patients.

If hypotension persists despite adequate fluid therapy, vasoactive agents (dopamine, vasopressin, norepinephrine, dobutamine) should be considered, as distributive shock secondary to bacterial sepsis is common. End points of resuscitation should include improved mentation with normalization of heart rate, respiratory rate, temperature, blood pressure, and hyperlactatemia, if measured. Once the patient is hemodynamically stable, its hydration status should be assessed and a fluid plan instituted to replace any ongoing deficits.

Glucose supplementation may be required either on presentation or later during hospitalization in response to hypoglycemia. Blood glucose levels should be checked frequently and supplemented using 50% dextrose as an initial bolus followed by a continuous infusion if hypoglycemia persists. Acid-base and electrolyte abnormalities are pervasive in patients with heatstroke and dynamic throughout the course of treatment. Most abnormalities do not resolve immediately; therefore, monitoring of changes and trends is recommended to guide treatment adjustments.

Antibiotics should be instituted in patients with signs of sepsis as well as patients with hypoperfusion and evidence of gastrointestinal mucosal disruption (hematochezia, hematemesis, melena, or prolonged anorexia) because of the increased risk of bacterial translocation. Broad-spectrum bactericidal antibiotic coverage that is effective against gram-negative, gram-positive, and anaerobic bacteria is recommended.⁹ Coverage should be continued until the patient's cardiovascular status is stable, enteral intake is good, and gastrointestinal signs are resolving.

Based on the patient's red blood cell count, platelet count, and coagulation testing results, the use of blood products may be

Key Facts

- Heatstroke, the most severe form of heat-related illness, is characterized by a core temperature >104°F (>40°C) and central nervous system abnormalities.
- Patients with altered mental status have increased mortality rates compared with those with non-altered mental status.
- Patients can be severely affected by heat-related illnesses but have a normal or subnormal presenting temperature.
- Early initiation of active cooling by owners during transport to the hospital may reduce the risk of death from heatstroke.

Table 2. Pharmacologic Treatment Recommendations^{12,26}

Category of Medication	Indication	Drug	Dose
IV fluids (crystalloid)	Hypovolemia	Lactated Ringer solution Normosol-R Plasmalyte 148	10 mL/kg bolus (can be repeated, being cautious if exceeding patient's blood volume)
IV fluids (crystalloid)	Hydration	Lactated Ringer solution Normosol-R Plasmalyte 148	Patient weight <2 or >50 kg: (BWkg ^{0.75})70 mL/day Patient weight 3–49 kg: (BWkg × 30) + 70 mL/day
IV fluids (colloid)	Hypotension Hypovolemia	Hetastarch 450/0.7 Vetstarch 6% 130/0.4	5 mL/kg bolus (can be repeated, being cautious if exceeding 20 mL/kg/d)
Antibiotics	Bacterial infection	Ampicillin	22 mg/kg q8h
		Enrofloxacin	5–10 mg/kg q24h
		Cefazolin	22 mg/kg q8h
Plasma	Coagulopathy		10 mL/kg
Osmotic agents	Cerebral hypertension	Hypertonic saline	3–5 mL/kg bolus
		Mannitol	1 g/kg over 30 minutes
Gastrointestinal protectants	Mucosal injury	Famotidine	0.5–1 mg/kg q24h
		Cimetidine	5–10 mg/kg q8h
		Sucralfate	0.25–1 g q8h
Sodium channel inhibitor	Ventricular arrhythmias	Lidocaine	2 mg/kg IV bolus (dogs)
Vasopressors	Hypotension	Dopamine	3–10 µg/kg/min IV
		Vasopressin	0.5–4 mU/kg/min IV
		Norepinephrine	0.05–2 µg/kg/min IV
		Dobutamine	2–20 µg/kg/min IV (dogs) 1–5 µg/kg/min IV (cats)
Dextrose	Hypoglycemia	50% Dextrose	0.5 mL/kg diluted 1:1 as a bolus and supplementation added to crystalloid fluids
		25% Dextrose	1 mL/kg diluted 1:1 as a bolus and supplementation added to crystalloid fluids
Electrolyte supplementation	Hypokalemia	Potassium chloride	Supplementation is based on the patient's potassium level but should not exceed 0.5 mEq/kg/hr IV

indicated. The product required is based on the patient's underlying needs. Coagulopathic patients may benefit from fresh frozen plasma, whole blood, or platelet-rich plasma transfusions. Anemic patients should be monitored closely and evaluated on an individual basis to determine if a transfusion is indicated. Indications for transfusions should be based on the patient's hematocrit (<25%) in conjunction with abnormal perfusion parameters. Anemic patients benefit most from packed red blood cell or whole blood transfusions. A

complete discussion of transfusion medicine is beyond the scope of this paper, but there are established resources regarding transfusion medicine.²¹

Frequent reassessment of patient cardiopulmonary status should include monitoring of blood pressure, heart rate, respiratory rate and effort, thoracic auscultation, and perfusion parameters. Cardiac arrhythmias can be present at the time of admission or develop during hospitalization and should be identified and

characterized immediately. Continuous ECG monitoring should be instituted if frequent and persistent arrhythmias are noted. Before initiating antiarrhythmic medication, underlying hypovolemia, pain, anxiety, acid-base disturbances, and electrolyte abnormalities should be ruled out or appropriately treated. Thoracic radiography, pulse oximetry, and arterial blood gas analysis are useful in identifying and addressing changes in pulmonary status. Supplemental oxygen is indicated in any patient with pulmonary dysfunction.

Placement of an indwelling urinary catheter is indicated in heatstroke patients with suspected kidney injury. This allows quantitative measurement of urine output to tailor ongoing fluid therapy and assessment of urine quality (hematuria, myoglobinuria, casts). Early identification of oliguria or anuria can improve outcome through judicious use of IV fluids in order to avoid overhydration. Maintaining a sterile, closed system is essential to reduce the risk of nosocomial infections. Indwelling urinary catheters should be limited to patients that are at risk of oliguria or anuric renal failure. Universal placement of urinary catheters in all heatstroke patients could place these patients at undue risk of ascending urinary tract infection.

Gastrointestinal support should be provided due to the increased prevalence of mucosal injury. Antiemetic therapy may be warranted based on patient history and clinical signs. Proton pump inhibitors or H₂ blockers should be considered to decrease the risk of stress-related mucosal disease.²² Sucralfate is indicated if gastric ulceration is suspected but is ineffective in preventing gastric ulcers. Administration of oral medications should be reserved for patients that have an intact swallow reflex. Nutritional support is essential to restoring gastrointestinal integrity and should be instituted as soon as possible, but only after the patient's cardiovascular status is stable. This is best accomplished through enteral feeding. If the patient cannot tolerate enteral feeding or is neurologically compromised (i.e., comatose or absent swallow reflex), parenteral nutrition is indicated.

For patients with signs of central nervous system dysfunction, serial neurologic evaluations in conjunction with blood pressure monitoring should be used. If intracranial hypertension is suspected and the patient is adequately hydrated, 7.5% hypertonic saline may be superior to mannitol because it reduces the risk of dehydration caused by osmotic diuresis.²³

Corticosteroid use is controversial in heatstroke patients; careful risk evaluation is needed before proceeding with administration. Although some benefits have been found with steroid therapy in experimental models, risks are also present, including gastrointestinal ulceration and immunosuppression. Routine use of corticosteroids is discouraged. Use of these agents should be decided on a case-by-case basis to confirm that the benefits of administration outweigh the risks.

Prognosis

Heatstroke patients have many variables that make it difficult to predict outcome. Published mortality rates range from 50% to 56% in canine heatstroke studies.^{2,6} Negative prognostic indicators at hospital admission that have been identified in veterinary patients

include altered mental status, hypoglycemia, prolonged prothrombin time (>18 seconds), and prolonged activated partial thromboplastin time (>30 seconds).^{2,6} Other reported negative prognostic indicators include elevated serum creatinine level (>1.5 mg/dL), delayed admission to the hospital (>90 minutes), seizure activity, and obesity.² Based on our clinical experience, a patient's response to aggressive therapy in the first 12 to 24 hours is likely a better determinant of true outcome. In human heatstroke patients, high levels of high mobility group box-1 (HMGB1) protein were noted to peak at 6 hours and correlated with a negative prognosis.²⁴ HMGB1 is a new topic of research in a variety of inflammatory conditions in canine patients and an area of further research for veterinary patients.²⁵

Summary

Heatstroke is common in human and veterinary medicine. The body is equipped with intricate mechanisms for thermoregulation, but with prolonged exposure or extreme hyperthermia, these mechanisms eventually fail. Clinical signs vary depending on the severity of the disease, presence of predisposing factors, and underlying disease processes. Early diagnosis of heatstroke in conjunction with rapid initiation of treatment is key to improving patient outcome. Treatment is centered on active cooling, restoration of patient volume status, and prevention of multiorgan failure. Therapies can be multifaceted and must be tailored to the patient. Outcome depends largely on response to treatment.

References

1. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med* 2002;346:1978-1988.
2. Bruchim Y, Klement E, Saragusty J, et al. Heat stroke in dogs: A retrospective study of 54 cases (1999-2004) and analysis of risk factors for death. *J Vet Intern Med* 2006;20:38-46.
3. Platt SR, Radaelli ST, McDonnell JJ. The prognostic value of the modified Glasgow Coma Scale in head trauma in dogs. *J Vet Intern Med* 2001;15:581-584.
4. Drobatz KJ, Macintire DK. Heat-induced illness in dogs: 42 cases (1976-1993). *J Am Vet Med Assoc* 1996;209:1894-1899.
5. Flourney W, Wohl JS, Macintire D. Heatstroke in dogs: pathophysiology and predisposing factors. *Compend Contin Educ Vet* 2003;25:410-422.
6. Aroch I, Segev G, Loeb E, et al. Peripheral nucleated red blood cells as a prognostic indicator in heatstroke in dogs. *J Vet Intern Med* 2009;23:544-551.
7. Johnson S, McMichael M, White G. Heatstroke in Small Animal Medicine: A clinical practice review. *J Vet Emerg Crit Care* 2006;16:112-119.
8. Flourney W, Wohl JS, Macintire D. Heatstroke in dogs: clinical signs, treatment, prognosis and prevention. *Compend Contin Educ Vet* 2003;25:422-431.
9. Holloway S. Heatstroke in dogs. *Compend Contin Educ Vet* 1992;14:1598-1604.
10. Johnson K. Pathophysiology of heatstroke. *Compend Contin Educ Vet* 1982;16:141-145.
11. Holowaychuk MK, Hansen BD, DeFrancesco TC, et al. Ionized hypocalcemia in critically ill dogs. *J Vet Intern Med* 2009;23:509-513.
12. Silverstein D HK. *Small Animal Critical Care Medicine*. St. Louis, Missouri: Saunders Elsevier, 2009.
13. Pease S, Bouadma L, Kermarrec N, et al. Early organ dysfunction course, cooling time and outcome in classic heatstroke. *Intensive Care Med* 2009;35:1454-1458.
14. Casa DJ, McDermott BP, Lee EC, et al. Cold water immersion: the gold standard for exertional heatstroke treatment. *Exerc Sport Sci Rev* 2007;35:141-149.
15. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. *Crit Care* 2007;11:R54.
16. Bynum G, Patton J, Bowers W, Leav I, Hamlet M, Marsili M, Wolfe D. Peritoneal lavage cooling in an anesthetized dog heatstroke model. *Aviat Space Environ Med* 1978;49(6):779-784.
17. Smith J WL. Cooling methods used in the treatment of exertional heat illness. *Br J Sports Med* 2004;39:503-507.

18. Broessner G, Beer R, Franz G, et al. Case report: severe heat stroke with multiple organ dysfunction—a novel intravascular treatment approach. *Crit Care* 2005;9:R498-R501.
19. Niu KC, Chang CK, Lin MT, et al. A hyperbaric oxygen therapy approach to heat stroke with multiple organ dysfunction. *Chin J Physiol* 2009;52:169-172.
20. Hong JY, Lai YC, Chang CY, et al. Successful treatment of severe heatstroke with therapeutic hypothermia by a noninvasive external cooling system. *Ann Emerg Med* 2012;59:491-493.
21. Tocci LJ. Transfusion medicine in small animal practice. *Vet Clin North Am Small Anim Pract* 2010;40:485-494.
22. Monnig AA, Prittie JE. A review of stress-related mucosal disease. *J Vet Emerg Crit*

Care (San Antonio) 2011;21:484-495.

23. Kamel H, Navi BB, Nakagawa K, et al. Hypertonic saline versus mannitol for the treatment of elevated intracranial pressure: a meta-analysis of randomized clinical trials. *Crit Care Med* 2011;39:554-559.
24. Tong HS, Tang YQ, Chen Y, et al. Early elevated HMGB1 level predicting the outcome in exertional heatstroke. *J Trauma* 2011;71:808-814.
25. Yu DH, Nho DH, Song RH, Kim SH, Lee MJ, Nemzek JA, Park J. High-mobility group box 1 as a surrogate prognostic marker in dogs with systemic inflammatory response syndrome. *J Vet Emerg Crit Care (San Antonio)* 2010;20(3):298-302.
26. Plumb D. *Plumb's Veterinary Drug Handbook*. Ames, IA: Wiley-Blackwell; 2011.



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1. Which neurologic abnormality(ies) at presentation are associated with increased mortality?

- a. tetraparesis
- b. presence of seizure activity
- c. altered mental status
- d. b and c

2. Which perfusion parameters should be assessed during the initial examination?

- a. capillary refill time
- b. mucous membrane color
- c. pulse quality
- d. all of the above

3. Leukocytosis is suspected to develop in heatstroke patients because of (an)

- a. acute inflammatory response.
- b. dehydration.
- c. response to infection.
- d. a and c

4. The presence of nucleated red blood cells in heatstroke patients is speculated to be caused by

- a. direct thermal cytotoxicity to red blood cells.
- b. chronic anemia.
- c. direct thermal cytotoxicity to the bone marrow.
- d. none of the above

5. The first treatment step to restoring perfusion in heatstroke patients is administration of

- a. gastrointestinal protectants
- b. antibiotics
- c. fresh frozen plasma
- d. fluid therapy (crystalloid and colloid).

6. The presence of schistocytes may be an early indicator of which sequela of heatstroke?

- a. acute renal failure
- b. acute respiratory distress syndrome
- c. acute liver failure
- d. disseminated intravascular coagulation

7. Active cooling should be discontinued at what core body temperature?

- a. 101.5°F (38.6°C)
- b. 103.1°F (39.5°C)
- c. 103.8°F (39.9°C)
- d. 104.5°F (40.3°C)

8. Air conditioning and open windows while driving to the hospital are two ways to increase heat dissipation through what mechanism(s)?

- a. conduction
- b. convection and evaporation
- c. radiation
- d. none of the above

9. Which is of the following is not a poor prognostic indicator?

- a. persistent hypoglycemia
- b. persistent increased creatinine
- c. decreased prothrombin time
- d. altered mental status

10. Patients that are adequately hydrated and are suspected of having increased intracranial pressure should be treated with

- a. antibiotics.
- b. hypertonic saline.
- c. mannitol.
- d. valium.