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Heatstroke in Dogs: Pathophysiology and Predisposing Factors*

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KEY FACTS

- Although the upper lethal body temperature in dogs is approximately 109.4°F (43°C), permanent brain damage may develop at temperatures as low as 105.8°F (41°C).

- Dogs that are confined to automobiles or tethered outdoors lack convection cooling in the development of heatstroke.

- Conduction accounts for only approximately 3% of total heat loss in humans; however, it plays a greater role in dogs.

- Cardiovascular disease and drugs greatly predispose dogs to heatstroke because of their inability to mount a proper cardiovascular response and low-circulating plasma volume.

ABSTRACT: Heatstroke is a rapidly progressive life-threatening emergency resulting from direct thermal injury to cardiovascular, gastrointestinal, renal, hepatic, endothelial, musculoskeletal, and central nervous tissues. Thermal injury to cells causes generalized cellular necrosis through denaturization of proteins, inactivation of enzyme systems, destruction of cell membrane lipids, and alteration of mitochondrial function. Heatstroke is precipitated by the collective inability of the body to maintain normal thermoregulation through proper cooling and heat dissipation mechanisms. Classic (or nonexertional) heatstroke most commonly develops when dogs are confined in an overheated enclosure. Exertional heatstroke is associated with muscular activity and is most common in dogs with such predispositions as obesity, laryngeal paralysis, and brachycephalic conformation. Common complications of heatstroke include oliguric renal failure, disseminated intravascular coagulation, cardiac arrhythmias, septic shock, and seizures.

Heatstroke is a commonly recognized syndrome in dogs occurring most often in the summer months, especially in the southern United States. It is a heat illness caused by exposure to extreme environmental temperatures resulting in elevated body temperature and direct hyperthermal injury to body tissues. Alterations in normal cooling functions interfere with thermoregulation and the body's inability to properly dissipate heat and can precipitate heat-induced illness.[1–3] Several diseases and physical conditions in dogs can perpetuate these hyperthermic events.[3–5]

In humans, heat-induced illnesses are classified according to the type and severity of clinical signs[2,3]:

- **Heat cramps**, the least severe of the heat illnesses, is characterized by extreme dehydration, muscle cramps, and sodium depletion. It is the most common heat-induced illness among humans and is rarely detected or recognized as a problem in veterinary patients.

*See companion article on p. 422. 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.

- **Heat exhaustion** is the inability to perform work due to lethargy caused by extreme heat conditions. It is an advanced form of heat-induced illness that is often described by affected people as having "the chills" or clammy skin during high physical activity in extreme temperatures.

- **Heat prostration** is more severe and is characterized by headache, vomiting, tachycardia, and hypotension.

- **Heatstroke**, which results in central nervous system (CNS) dysfunction, circulatory insufficiency, and breakdown of normal enzymatic and cellular functions, is the most severe form of heat-induced illness.

Most of the clinical information regarding canine heatstroke has been derived from human medicine or a limited number of studies involving experimentally induced heatstroke in dogs.[6–8] Dogs are considered unsuitable models for heat-related disease in humans because of differences in responses to high temperatures.[9] However, one study in dogs reported that histopathologic changes following hyperthermia paralleled the same irreversible lesions described in humans who have died from heatstroke.[10] Few reports have described clinical observations and treatment in canine heatstroke.[11–13] One retrospective study correlated clinicopathologic findings, history, physical examination, and outcome in 42 dogs with heat-induced illness.[14]

PHYSIOLOGY AND THERMOREGULATION

Normal homeostatic mechanisms operate to maintain body temperature within a very narrow range called the set-point,[2] which acts as a "trigger" for the body to activate physiologic processes to elevate or decrease body temperature. For example, when an animal's body temperature decreases below the set-point, shivering, increased voluntary activity, increased catecholamine secretion, cutaneous vasoconstriction, postural changes, piloerection, and an increase in thyroxine production result in elevated body temperature. When body temperature is elevated, cutaneous vasodilation, increased respiration, panting, anorexia, and sweating cool the body and lower its temperature.[2,4]

The thermoregulatory center controls the critical set-point and is located in the preoptic region of the anterior hypothalamus.[2] Numerous thermostatic receptors act as temperature sensors exist in this preoptic region. When core body temperature increases, warm blood supplying the hypothalamus stimulates compensatory cooling mechanisms. In addition, afferent signals originating throughout the body (e.g., skin, viscera, CNS) detect the temperature of circulating blood and converge in the caudal hypothalamus. In the caudal hypothalamus, the signals from the preoptic area and the periphery converge, initiating neurogenic compensatory responses to decrease body temperature. The initial compensatory mechanism in dogs is activation of the panting center in the pons.[2]

Thermoregulation is simply the balance between heat production and dissipation. Body heat is produced by three main processes: 1) basal metabolism, 2) muscular activity, and 3) metabolism of nutrients (oxidation). Body heat is dissipated by several means, including: 1) evaporation, 2) radiation, 3) conduction, and 4) convection. In humans, evaporation by sweating and radiation of infrared heat waves account for the majority of heat loss from the body.[2,3] In dogs, the primary cooling mechanisms are radiation, convection, and evaporation
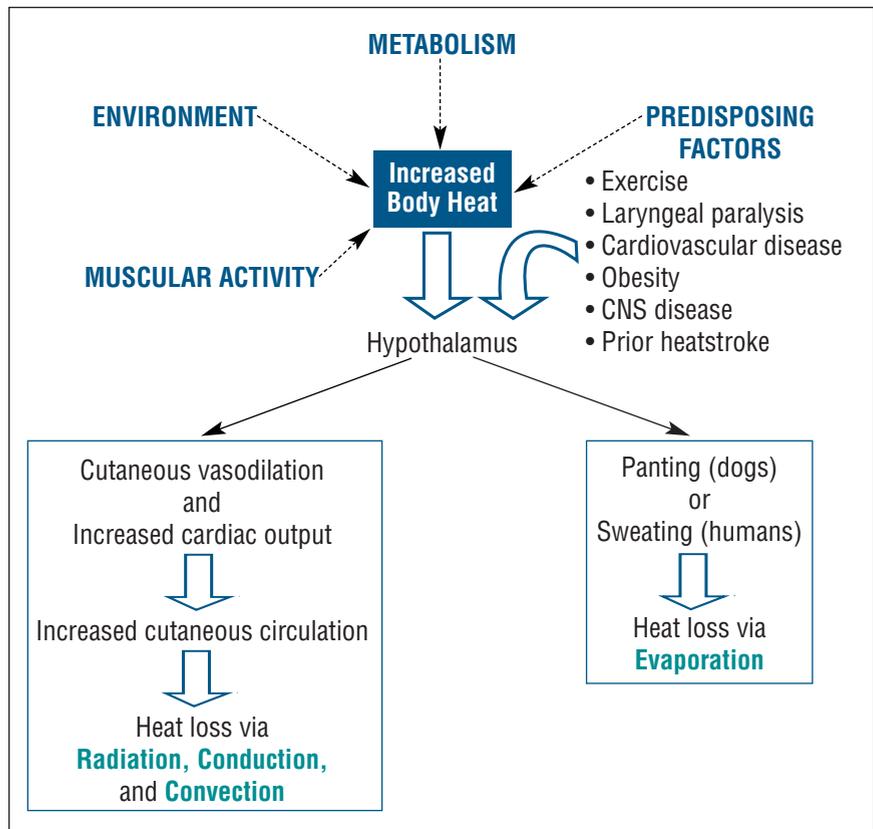

Figure 1—Normal physiologic response to heat stress. (Adapted from Johnson KE: Pathophysiology of heatstroke. *Compend Contin Educ Pract Vet* 4[2]:142, 1982)

by panting rather than sweating (Figure 1).[2,7,15,16] A very small amount of heat is lost in association with excretion of feces and urine.

At ambient temperatures below 89.6°F (32°C), convection, conduction, and radiation contribute to the maintenance of normothermia. As the environmental temperature increases and approaches body temperature, evaporation becomes more important in maintaining normothermia. Evaporation occurs primarily through panting. The nasal turbinates provide a large surface area for the loss of water from the moist mucous membranes. Panting increases evaporation but requires respiratory muscle activity that in itself generates heat.

More than 70% of the total body heat loss in dogs and cats is due to radiation and convection from the body surface.[16] The continuous movement of cooler air adjacent to the skin allows conduction of heat to the air, or convection, a significant cooling mechanism in dogs. Conduction is the exchange of heat between two objects in direct contact with one another. Although conduction accounts for only approximately 3% of total heat loss in humans, conduction probably plays a greater role in dogs.[2] Animals often lie down on cool surfaces, allowing the relatively hairless skin of the ventral abdomen to lose heat by transfer or conduction to the surface. Heat loss through conduction is aided by cardiovascular responses during warm ambient temperatures. Peripheral vasodilation and elevated cardiac output increase cutaneous circulation, allowing greater heat loss by conduction (Figure 1).[7]

HYPERTHERMIA

Hyperthermia can be described as a pyrogenic or nonpyrogenic elevation in body temperature.[2] Fever, or pyrogenic hyperthermia, is characterized by an increased body temperature mediated by functional thermoregulatory mechanisms in response to noninfectious systemic inflammation (e.g., pancreatitis, trauma, immune-mediated disease) or infections producing pyrogens. Pyrogens (e.g., interleukin-1, tumor necrosis factor, viruses, bacteria) act on the anterior hypothalamus to raise the set-point to a higher temperature range, producing fever, which rarely puts veterinary patients at risk. However, nonpyrogenic hyperthermia occurs when heat-dissipating mechanisms cannot compensate adequately for heat-producing mechanisms, thereby leading to an increase in body temperature above the set-point and sometimes to heatstroke.

HEATSTROKE

During extreme temperatures or high humidity, there is a limit to the rate at which a dog can lose heat by panting, conduction, or convection. This temperature threshold probably varies with individual animals, depending on several factors (e.g., physical status, metabolic rate, hydration status). During hyperthermia, elevated core body temperature stimulates an increase in metabolic rate and endogenous heat production, thereby perpetuating hyperthermia.[3] This cycle of increasing core temperature continues and the hypothalamus becomes affected, resulting in depression and weakening of thermoregulatory function.[17] Hypothalamic dysregulation results in diminished central thermoregulation and abnormal activation of peripheral compensatory cooling mechanisms, allowing core body temperature to further increase, resulting in heatstroke.

Heatstroke can be categorized as exertional or nonexertional.[6] **Exertional heatstroke** is more likely to occur in late spring or early summer before dogs become acclimatized to the high environmental temperatures. Exertional heat stroke is uncommon in working and athletic dogs despite activity in hot, humid environments. Racing greyhounds in the southern United States rarely exhibit heatstroke, although rectal temperatures often exceed 107.6°F (42°C) after a 400-m race.[18] Similarly, military working dogs work for hours in temperatures exceeding 140°F (60°C) in desert conditions without developing heatstroke. Athletes and working dogs are well hydrated, acclimated, and prevented from overexertion during activities by handlers. **Nonexertional heatstroke** most commonly develops when dogs are confined in an overheated enclosure or chained outdoors and deprived of water and/or shade.[4]

During conditions of high environmental humidity, panting is less effective in dissipating heat. Dogs exposed to 35% humidity and 129.9°F (54.4°C) showed significant differences in heart rate, venous pH, rectal temperature, and venous partial pressure of carbon dioxide compared with dogs exposed to 15% humidity at the same temperature.[10] Static air also prevents the movement of warm air, decreasing convection and further loss of body heat. Animals placed in an enclosed environment with no airflow, such as an automobile, suffer from extreme lack of convection cooling. Vehicular entrapment is a common cause of heatstroke in dogs.[4] One study suggested that 50% of hyperthermic dogs trapped in a car may have a survival time of as few as 48 minutes with an ambient temperature of 84.2°F (29°C) and 90% humidity.[19] In the past, heat-induced hyperthermia was a major problem in shipping animals during the summer months.[10] Prior to temperature-controlled guidelines, animals could be exposed to temperatures exceeding 129.2°F (54°C) in airline cargo compartments. In addition, in most communities, leash laws require dogs to be confined. Dogs that are tethered outdoors or whose movement is greatly limited are predisposed to heat-

stroke when restricted from shade and available water. Moreover, heatstroke is rarely found in dogs that roam free, regardless of exercise, temperature, or humidity.

PREDISPOSING FACTORS

Factors that could predispose dogs to severe hyperthermia can be divided into the following two categories: 1) decreased ability to dissipate heat and 2) increased heat production (see box at *right*). Exogenous and endogenous stimuli can affect both aspects of thermoregulation.

Exogenous conditions resulting in decreased heat dissipation include lack of acclimatization, water deprivation, high ambient humidity, and confinement to compartments with poor ventilation. Common drugs that may cause idiosyncratic hyperthermic events or predispose dogs to heatstroke include phenothiazine derivatives (e.g., acepromazine, chlorpromazine), diuretics (e.g., furosemide), cardiac drugs, and inhalation anesthetics (e.g., halothane).[3,5,20,21] Halothane-induced malignant hyperthermia, a rare anesthetic complication, causes a myopathy, resulting in increased muscle rigidity and production of excessive body heat. Diuretics promote fluid and electrolyte depletion, resulting in decreased circulating blood volume and improper electrical activity in cardiac and skeletal muscles. Phenothiazine derivatives have been implicated in producing heatstroke because of their ability to alter heat-dissipating mechanisms through hypohidrosis and autonomic instability in humans. Use of negative inotropic drugs such as β-blockers (e.g., atenolol, propanolol) may further impair cardiac output during hyperthermia.

Common endogenous factors impairing heat dissipation include brachycephalic anatomy, upper airway disease, cardiovascular and neurologic disease, obesity, age, and haircoat. Brachycephalic breeds with elongated soft palates and stenotic nares may experience inadequate airflow in their upper respiratory system, hindering panting mechanisms. Similarly, dogs with upper airway disease, such as laryngeal paralysis or tracheal collapse, have inefficient ventilation.

Cardiovascular disease causes decreased cardiac output and leads to decreased perfusion to the peripheral vasculature and cutaneous circulation. During hyperthermia, decreased perfusion to the skin can impair heat loss via radiation and convection. Cardiac insufficiency can be associated with decreased circulating blood volume and hypotension resulting in decreased mucous hydration in the nasal turbinates for evaporative cooling during panting. One human study reported that 78% of patients with compensated heart failure developed overt heart failure in as little as 2 hours after exposure to temperatures of 89.9°F (32.2°C) and 75% relative humidity.[22]

Factors Predisposing Dogs to Heatstroke[a]

Heat Dissipation
1. Exogenous
   a. Lack of acclimatization
   b. Confinement/poor ventilation
   c. Humidity
   d. Water deprivation
   e. Drugs (e.g., furosemide)
2. Endogenous
   a. Brachycephalic anatomy
   b. Obesity
   c. Laryngeal disease (paralysis)
   d. Cardiovascular disease
   e. Central or peripheral nervous system disease
   f. Age (geriatric)
   g. Prior heatstroke
   h. Haircoat

Heat Production
1. Exogenous—Drugs/toxicity
   a. Amphetamines
   b. Macadamia nuts
2. Endogenous
   a. Exercise
   b. Pyrexia (febrile disease)
   c. Hormonal hyperthermia (hyperthyroid)
   d. Seizures
   e. Eclampsia

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[a]Adapted from Johnson KE: Pathophysiology of heatstroke. *Compend Contin Educ Pract Vet* 4(2):142, 1982.

Neurologic impairment of respiratory function or intracranial disease affecting the thermoregulatory center can impair physiologic cooling mechanisms.[23–25] Peripheral nerve disorders, such as botulism, acute polyradiculoneuritis (Coonhound paralysis), and tick paralysis, can result in ventilatory failure (respiratory paralysis). Intracranial diseases include brain tumors and central neurologic lesions affecting the thermoregulatory center in the hypothalamus.

Excess fat, which causes increased insulation in obese animals, impairs normal heat dissipation.[26] In humans, elderly patients suffer from an increased risk of heatstroke because of their inability to sweat, poor acclimatization, deficient voluntary control, compromised cardiovascular response, and the consumption of drugs that adversely affect thermoregulation.[27] Older dogs may have similar problems functioning in hot environments.[28] There are no specific breeds that seem to be at an increased risk; however, in one study purebreds (bulldogs, Saint Bernards, Australian shepherds, weimaraners)

were overrepresented.[14] Also, longhaired and dark-coated animals may be at an increased risk.[29] There is no gender predilection reported in dogs with heatstroke.

All dogs regardless of size, age, or breed require a transitional period for acclimatization to hot weather because unconditioned dogs forced to exercise (e.g., jogging with owners) may be at an increased risk for heatstroke. Endogenous conditions that increase physiologic heat production, such as pyrexia[5] and hormonal hyperthermia (e.g., hyperthyroidism, pheochromocytoma),[30,31] may act synergistically to cause heat-induced illnesses. Eclampsia and status epilepticus result in extreme muscle activity that can lead to significant heat production resulting in severe hyperthermia.[32,33] Amphetamine and macadamia nut intoxication can cause hyperthermia in dogs as well.[34,35]

PATHOPHYSIOLOGIC EFFECTS OF SEVERE HYPERTHERMIA

Once a critical temperature is reached, permanent intracellular alterations and cell membrane instability lead to multiple tissue and organ deterioration. Specifically, thermal injury causes generalized cellular necrosis through denaturization of proteins, inactivation of enzyme systems, destruction of cell-membrane lipids, and alteration of mitochondrial function.[10,36] Global ischemia coupled with temperature-induced massive cellular destruction of cells can propagate multiorgan dysfunction syndrome. Multiorgan dysfunction or failure can involve the cardiopulmonary, central nervous, gastrointestinal (GI), renal, hematologic, and coagulation systems.

Cardiovascular and Pulmonary Systems

Initially, hyperthermia is associated with increased cardiac output and decreased systemic vascular resistance. The baroreceptor response to low systemic vascular resistance is peripheral vasoconstriction to maintain normal blood pressure and circulating plasma volume to vital organs. However, during extreme hyperthermia, vasoconstriction is prevented by inhibition of the sympathetic centers in the hypothalamus.[2] As core body temperature continues to rise, cutaneous vasodilation persists and splanchnic arteriolar vasodilation leads to venous pooling. The selective loss of splanchnic vasoconstriction appears to be a key point in the pathogenesis of heatstroke.[24,37,38] Furthermore, hypodynamic shock coupled with preexisting dehydration results in low circulating plasma volume and hypoperfusion. Low plasma volume prevents core body heat to circulate and reach the periphery for heat dissipation mechanisms.[39] Thus the core body temperature continues to rise, and the cellular events underlying heatstroke progress.

Myocyte injury secondary to hyperthermia, decreased perfusion, acidosis, electrolyte abnormalities, and thromboembolism contributes to cardiac dysfunction.[10,37] Myocardial ischemia and tachyarrhythmia have been observed during hyperthermia in humans.[37] Major tissue changes observed in experimental canine hyperthermia include severe fragmentation of the myocardium and loss of myofibrillar striations.[10] Myocardial and Purkinje fiber damage predisposes the heart to myocardial conduction defects and ventricular arrhythmias. Isolated canine Purkinje fibers exposed to ischemia (e.g., hypoxia, acidosis, elevated lactate) in vitro produce myocardial conduction defects and ventricular arrhythmia.[40]

Marked increases in pulmonary vascular resistance and direct thermal injury to the pulmonary endothelium may lead to cor pulmonale or acute respiratory distress syndrome (ARDS) in humans.[3,37,38] Significant pulmonary injury during heatstroke often leads to disseminated intravascular coagulation (DIC), noncardiogenic pulmonary edema, or ARDS.[3,41] Although pulmonary hemodynamics during hyperthermia have not been evaluated in dogs, it seems likely that they may be at risk for developing ARDS as well. Experimentally, fibrinogen deposition and microembolization in the lungs have resulted in severe noncardiogenic pulmonary edema in dogs.[42] In addition, histopathologic lung lesions from dogs after heatstroke included marked alveolar hemorrhage or edema and septal thickening and congestion.[10]

Central Nervous System

Severe hyperthermia results in massive neuronal injury and cell death. Experimental evidence suggests that temperatures as low as 105.8°F (41°C) may cause permanent brain damage.[16] Cerebral edema, hemorrhage, infarction, and cerebellar dysfunction are common consequences of severe hyperthermia in dogs.[8] The exact mechanisms resulting in cerebral edema are unknown. Dopamine, serotonin, and cytokines (e.g., interleukin-1, interleukin-6, tumor necrosis factor–α) have been implicated as mediators of cerebral edema, neuronal tissue damage, and decreased cerebral perfusion pressure during hyperthermia.[43] The Purkinje cells in the cerebellum are also very susceptible to hyperthermia.[10] The development of cerebral and cerebellar edema and localized areas of necrosis due to intracranial hemorrhage can lead to disorientation, seizures, coma, and sometimes death. Altered uptake of essential amino acids (e.g., glutamine) during hyperthermia has been implicated as a cause for disorientation and coma.[44] Excessive temperatures in the hypothalamus can damage the thermoregulatory center, leading to impairment

of temperature regulation.[17] Permanent abnormalities in the thermoregulation center can predispose animals to subsequent hyperthermic episodes.[45]

Recently, the induction of heat shock protein and the antagonism of interleukin-1 resulted in protection against heatstroke-induced arterial hypotension and cerebral ischemic injury in rats.[46] These data imply that arterial hypotension and cerebral ischemia may also be implicated in the development of canine heatstroke.

Gastrointestinal System

In humans, gut injury plays a pivotal role in the pathogenesis of death due to heatstroke.[47] Hypovolemia, blood sludging, and microthrombi cause decreased perfusion and alter peripheral circulation to the GI tract, producing ischemia that leads to loss of the structural integrity of the GI tract mucosa. Altered splanchnic blood flow to the GI tract has been reported in laboratory animals with heatstroke; however, similar changes may not occur in dogs.[37,48] After GI wall integrity is compromised, bacterial translocation or endotoxin leakage can occur and incite gram-negative or positive bacteremia, endotoxemia, and sepsis.[49,50] Bacterial translocation and septic shock can propagate, leading to further cardiovascular dysfunction, global ischemia, and often death.[51,52] In dogs with septic shock, splanchnic pooling and maldistribution of blood flow can result in GI villous ischemia and endotoxemia.[53] Coagulation abnormalities associated with septic shock can range from mild changes in platelet count and subclinical alterations in clotting time to fulminant DIC.[54]

Hyperthermia-associated hepatocellular vascular degeneration with centrilobular necrosis and cholestasis has been reported in humans and dogs.[11,14,15] Heatstroke-induced hepatic damage results from direct thermal injury to hepatocytes and prolonged splanchnic hypotension.[6] Serum chemistry profiles of hyperthermic canine patients commonly exhibit elevations of hepatic enzymes (alanine transaminase and alkaline phosphatase) and bilirubin concentrations, suggesting both cholestasis and hepatic damage.[14] Elevated serum bilirubin has been reported in up to 50% of dogs with heatstroke.[14]

Renal System

Acute renal failure due to tubular necrosis occurs as a result of direct thermal injury, hypoxia, and microthrombosis associated with DIC.[7,10,14,15,55] The early phases of acute renal failure are associated with oliguria and elevated blood urea nitrogen and creatinine. Azotemia often has a prerenal and renal component in dogs with heatstroke because of severe dehydration and direct renal tis-
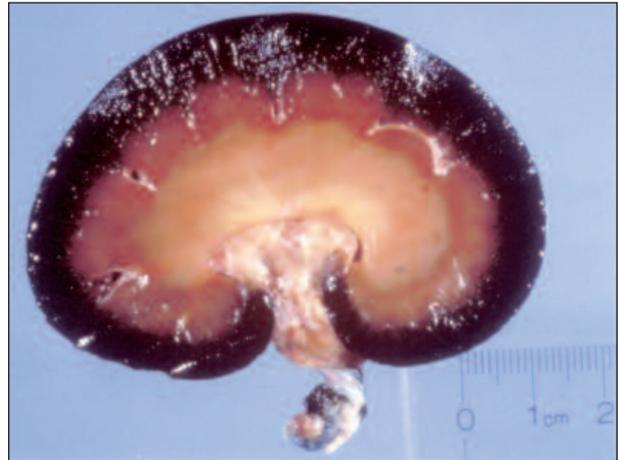

Figure 2—Longitudinal cut through a canine kidney discolored by hemoglobinuria and myoglobinuria.

sue damage, respectively. One review reported higher creatinine values and decreased renal function in nonsurvivors compared with that of survivors.[14] The filtration of myoglobin resulting from massive rhabdomyolysis associated with muscle necrosis is nephrotoxic and may exacerbate renal failure (Figure 2).[7] Although potentially reversible with rapid intervention, permanent renal insufficiency or profound polyuria may develop following recovery from heatstroke.[11,56,57]

Hematologic and Coagulation System

Severe dehydration and hemoconcentration in patients with heatstroke has been associated with hematocrits as high as 82%.[14] Dehydration most likely results from GI fluid loss and respiratory evaporative lost. Patients may also be anemic despite significant hemoconcentration. Proposed mechanisms of anemia during heatstroke include: 1) decreased survival time and hemolysis of the red blood cells, 2) hemorrhage into the GI tract, and 3) increased capillary permeability during DIC.[1,8,15,47,58,59] Hemoconcentration of circulating red blood cells creates sludging of the blood and increased viscosity.[60] Sludging or vascular stasis in capillaries decreases oxygen delivery and uptake by peripheral tissues, which propagate cellular hypoxemia. Sludging also contributes to microthrombi development and further exacerbates ischemia.

The capillary and venous endothelium are very susceptible to direct thermal injury. Damaged endothelium releases thromboplastic substances (i.e., thromboplastin, factor XII), which activate the clotting cascades and complement, producing an inflammatory response and widespread or systemic coagulation. This inflammatory and coagulation response includes increased adherence of platelets and leukocytes to the damaged

endothelium. Hyperthermia-induced platelet activation, coagulation factor and platelet damage, and widespread endothelial disruption result in the depletion of platelets and coagulation factors.[4,60–63] In addition, decreased hepatic synthesis of coagulation factors and widespread fibrinolysis result in DIC.[4,11,14,15,64] Massive hemorrhagic diathesis due to DIC is a common necropsy finding in nonsurvivors of heatstroke.[14] Megakaryocytes are very susceptible to high temperatures causing decreased release of platelets from the bone marrow.[4] The resulting thrombocytopenia may be delayed by several days and should not be confused with the low platelet counts seen earlier in the hyperthermic process.

Musculoskeletal System

Muscle degeneration and necrosis occur as a direct result of extremely high temperatures. Significant elevations in muscle enzymes, such as creatinine phosphokinase and hypermyoglobinemia, can be observed.[7,14] Severe rhabdomyolysis is more common in patients that have an exertional component to heatstroke compared with dogs with classic heatstroke.

Acid–Base Status

A common acid–base disturbance in dogs with heatstroke is a mixed acid–base disorder characterized by respiratory alkalosis and metabolic acidosis.[6,65] Respiratory alkalosis is caused by excessive panting that reduces blood carbon dioxide concentration through hyperventilation. A metabolic acidosis (titrational) is produced by increased tissue demands, hypoxemia, and anaerobic metabolism resulting in lactic acid production.[4] Acidosis has been shown to significantly increase cell death during hyperthermia.[66] Acid–base derangements can complicate cardiovascular, electrolyte, and GI effects of hyperthermia.

SUMMARY

Thermoregulation involves the balance between heat loss and heat production. Heatstroke is precipitated by the collective inability of the body to maintain normal thermoregulatory balance through proper cooling and heat dissipation. Heat dissipation mechanisms in dogs include radiation, conduction, convection, and evaporation by panting. Environmental temperature and humidity, ventilation, and confinement are major contributing factors. In addition, underlying diseases and physical conditions can predispose dogs to developing hyperthermia.

Subcellular lesions associated with thermal injury include generalized cellular necrosis through denaturization of proteins, inactivation of enzyme systems, destruction of cell membrane lipids, and alteration of mitochondrial function. Heatstroke can, therefore, affect many organs of the body, including the brain, heart, lungs, GI tract, liver, and kidneys. Common complications that veterinarians may encounter during heatstroke include oliguric renal disease, acid–base disturbances, coagulation alterations, cardiovascular collapse, and neurologic syndromes.

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ARTICLE #1 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. The most severe form of heat-induced illness reported in dogs that results in CNS dysfunction, circulatory insufficiency, and breakdown of normal enzymatic and cellular function is
   a. heat prostration.   c. heatstroke.
   b. heat exhaustion.   d. heat cramps.

2. In mammals, what area of the brain controls thermoregulation?
   a. cerebral cortex   c. thalamus
   b. pons   d. hypothalamus

3. At environmental temperatures above 89.6°F (32°C), the primary mechanism of heat loss in dogs is
   a. evaporation.
   b. conduction.
   c. convection.
   d. radiation.

4. Permanent damage to vital organ systems, including the brain, may occur if the body's core temperature reaches approximately
   a. 104°F.   c. 113°F.
   b. 106°F.   d. 118°F.

5. Key physiologic cardiovascular responses to hyperthermia are cutaneous
   a. vasodilation and increase in cardiac output.
   b. vasoconstriction and decrease in cardiac output.
   c. vasoconstriction and increase in cardiac output.
   d. vasodilation and decrease in cardiac output.

6. Which of the following is not a reported lesion in the CNS due to severe hyperthermia?
   a. cerebral edema
   b. cerebellar edema
   c. Purkinje cell damage
   d. paradoxical cerebrospinal fluid acidosis

7. Which of the following is not a currently reported complication of heatstroke in dogs?
   a. oliguric renal failure
   b. DIC
   c. septic shock
   d. hypertension

8. Which of the following are predisposing factors that can precipitate heatstroke in dogs?
   a. obesity, cardiovascular disease, water deprivation, antibiotics
   b. laryngeal disease, brachycephalic breeds, large-breed dogs, humidity
   c. lack of acclimatization, confinement, prior heatstroke, geriatric dogs
   d. furosemide, exercise, seizures, dolichocephalic breeds

9. The acid–base status in heatstroke is characterized as respiratory _____ and metabolic _____.
   a. acidosis; alkalosis   c. alkalosis; alkalosis
   b. alkalosis; acidosis   d. acidosis; acidosis

10. Which of the following is a permanent intracellular alteration in multiorgan tissues due to hyperthermia?
   a. denaturization of proteins
   b. inactivation of enzyme systems
   c. destruction of cell membrane lipids
   d. all of the above