# Heatstroke: HOT DAWG (or cat)!

*Jo-Annie Letendre, DMV, DACVECC*
*Clinical lecturer in Emergency and Critical Care*
*Faculté de médecine vétérinaire, Université de Montréal*

## Introduction

Body temperature must be maintained within a relatively narrow range, despite variations in environmental temperature, for the organism to function properly. When body temperature exceeds the normal range for a species, the body depends on heat dissipation mechanisms to cool itself down. When this thermoregulatory capacity is exceeded, heat-related illnesses occur. This leads to thermal issue damages, and potentially fatal, multi-systemic consequences.

## Thermoregulation

Normal body temperature for cats and dogs is 37.2-39.2°C. Body temperature is maintained within this narrow range by a balance between thermal load (ambient heat and heat produced by muscular activity and metabolism) and heat dissipation mechanisms. The hypothalamus contains thermoreceptors and is thus the control center for thermoregulation. When heat load is high, body temperature rises. This rise in temperature is perceived by the hypothalamus. Like a thermostat, the hypothalamus is set to a target temperature. If body temperature exceeds the target temperature, heat dissipation mechanisms are activated to restore the temperature to the normal range. There are also peripheral thermoreceptors in the skin that detect variations in skin temperature. Heat dissipation can take place via 4 mechanisms: evaporation, radiation, conduction, and convection. Evaporation is the process of transforming a fluid into vapor (perspiration, panting). Radiation is the process of releasing heat into the environment. Conduction occurs when the body is in contact with a cooler surface (examination table). Convection is the transfer of heat to air or water moving around the body. Under normal environmental conditions, most heat in dogs is dissipated by radiation and convection. As the ambient temperature rises and approaches body temperature, radiation and convection decrease, and evaporation becomes the most important heat dissipation mechanism.

## Definitions

Hyperthermia is defined as a body temperature above the normal value. Hyperthermia is not synonymous with fever. Fever is a rise in body temperature caused by an upward shift in the target temperature of the thermostat (hypothalamus) induced by cytokines. Fever is under the influence of pyrogenic substances. This is known as pyrogenic hyperthermia. Fever occurs with infectious, inflammatory, or neoplastic diseases. Heat-related disorders occur when thermogenesis and/or ambient temperature exceed the body's ability to dissipate heat. There are several types of heat-related illnesses, ranging from the least to the most severe: heat rash, heat syncope, heat cramps, heat exhaustion, and heat stroke. One proposed definition of heat stroke is "a form of severe and life-threatening hyperthermia, associated with a systemic inflammatory response, leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates." More simply, we can say that heat stroke is a significant rise in body temperature that can have various serious consequences!

## Causes

Heat-related illnesses can occur when the animal is exposed to a hot, humid environment (classic or exposure heatstroke) and/or when the heat generated by exercise or involuntary muscle activity, such as spasms or epileptiform seizures, is excessive (exertional heatstroke). Several risk factors have been described. Physical or anatomical characteristics, such as obesity, dense coat, breed, and upper respiration tract abnormalities (tracheal collapse, laryngeal paralysis, brachycephalic syndrome) can predispose the animal to heat-related illnesses, by interfering with the body's cooling mechanisms. These characteristics can increase the risk if heatstroke if the animal is kept in an excessively hot environment and/or low if it exercises (even in normal environmental conditions). Ambient conditions such as high temperature, high humidity, and/or low ventilation, which interfere with evaporation, convection, conduction, and radiation, contribute to the heat load. Most dogs suffering from heat-related illnesses are presented at the onset of hot, humid weather (spring/summer in Quebec). Despite increasing hot days at the end of summer, heat-related illnesses become less frequent. This may be linked to the time available for acclimatization. Acclimatization induces adaptive physiological and behavioral changes that improve the body's ability to cope with heat. Acclimatization can take up to two months to be complete. It is associated with improved cardiac performance, sodium conservation by the kidneys (Activation of the renin-angiotensin-aldosterone system), increased sweating capacity (greater in humans than in cats and dogs), plasma volume expansion, increased glomerular filtration rate, and enhanced ability to resist to rhabdomyolysis.

## Diagnosis

Heat-related illnesses are diagnosed by the presence of a rise in body temperature without infectious, inflammatory, or neoplastic cause, i.e. without the rise in temperature being due to fever. Generally, the history suggests circumstances that have led to excessive heat production (physical exercise, strenuous activities, severe muscle spasms, tremors, long-lasting seizures/status epilepticus) or exposure to extreme environmental conditions that interfere with thermoregulatory capacities (limited access to water and/or shade, confinement in an enclosed space, with limited or absent ventilation, animal kept in the car). It's important to remember, however, that the animal's temperature may be normal or even decreased at the time of presentation, if the owner has implemented cooling measures. The clinical signs of heat-related illnesses depend on its severity. In heatstroke, we always observe, by definition, altered mental status (depression, lethargy, agitation, delirium, loss of consciousness). We can also observe panting, tachypnea, increased respiratory, dyspnea, tachycardia, hyperemia or cyanosis, dry mucous membranes, very rapid or absent CRT, pronounced or decreased/absent femoral pulses, pulse deficits, ataxia, loss of vision, seizures, petechiae/ecchymoses, salivation, vomiting, diarrhea (sometimes hemorrhagic), epistaxis, hematuria, tremors, stridor, hypotension, etc.

## Cooling measures

The higher the body temperature and/or the longer the hyperthermia lasts, the greater the systemic repercussions. It is therefore important to cool the animal quickly. Several cooling methods of varying effectiveness have been described: administration of cold intravenous fluid, ice bath, spraying the patient with tepid to cold water, use of a fan, application of ice to the extremities, wetting the paws/coat with alcohol, cold water gastric lavage or enema, peritoneal lavage. Immersion in an ice-water bath (at 0°C) is recommended in human medicine. It is the quickest and most effective (although controversial) cooling measure, reducing temperature by 0.2°C per minute. However, this method is not widely used in veterinary medicine. The method recommended by the author is to thoroughly wet the animal's body with fresh water and direct a

fan towards it. Covering the animal with a wet towel is not recommended. In fact, it's better not to cover the animal's coat, but simply wet it abundantly and then use a fan to create a current of air. This cools the body by using evaporation and convection mechanisms. If the patient is placed on the examination table, conduction will also help to cool the body. Icepacks placed under the armpits, and/or in the cervical and/or inguinal areas (where there are large blood vessels) can also help to cool the body. Wetting the paws with cold water or alcohol is much less effective, given the small body surface area involved. Avoid using alcohol to wet the animal's coat. Alcohol is of no benefit compared to water, the smell is unpleasant for both the animal and the staff, and there is a risk of burns if the defibrillator is needed in the even of a cardiopulmonary arrest with a shockable rhythm. During cooling, temperature must be closely monitored to avoid causing hypothermia. Indeed, cooling must be stopped when the temperature reaches 39.5°C. Restoring normal body temperature following hyperthermia does not lead to cessation of the inflammatory response, but it does prevent further direct thermal consequences.

**Consequences**

When body temperature rises to extreme values, multisystemic consequences can occur. Indeed, multiorgan failure is the most serious complication following heatstroke. All organs can be affected. These consequences arise from the exposure of cells to heat, causing direct thermal damage, but also from the physiological changes that take place during hyperthermia. These include vasodilation in the skin, and vasoconstriction in the kidneys and splanchnic circulation, shifting circulation to the periphery to allow heat dissipation. Eventually, splanchnic vasodilation will occur, resulting in venous blood pooling and cardiovascular collapse. Perfusion of the various organs is thus compromised to a greater or lesser extent. Hyperthermia activated coagulation and fibrinolysis, ultimately leading to microthrombi formation, consumption of coagulation factors, and possibly disseminated intravascular coagulation (DIC). Finally, an inflammatory response is triggered. This inflammatory response is beneficial when there is a balance between pro- and anti-inflammatory mechanisms. During heatstroke, the inflammatory response can become exaggerated, chaotic, and anarchic, contributing to the development of multisystemic lesions.

**Treatment and monitoring**

Treating a patient with heatstroke is both simple and complicated. First, the patient must be cooled down using the cooling measures described above. This is to stop direct heat damage. Then, shock must be corrected by administering intravenous boluses of fluids (see section below: cardiovascular system). Restoration of a normal temperature and treatment of shock do not mean that the animal is out of the woods. In fact, the inflammatory response and damages already caused by the heat will still be present. We therefor need to manage the consequences and identify systemic lesions as soon as they appear so we can intervene as quickly as possible.

**Respiratory system**

As soon as a heatstroke patient is presented to the veterinary clinic, it is important to check saturation using a pulse oximeter and provide oxygen therapy if needed. In fact, oxygen supplementation should be provided in all animals that present with an increase in respiratory rate and/or effort until a thorough assessment of the respiratory system can be performed. In case of respiratory distress, sedation (butorphanol 0.1-0.3 mg/kg IV) may be necessary. If heatstroke occurs in a patient with anatomical anomalies (brachycephalic syndrome) or a respiratory condition (laryngeal paralysis, tracheal collapse), endotracheal intubation or even a tracheostomy may be necessary. Be prepared to sedate the animal quickly (propofol IV) and to perform intubation urgently if this becomes necessary. An emergency intubation cart with endotracheal tubes of all sizes and related accessories (laryngoscope, kling, stylet) should be easily accessible

and available. Heatstroke patients are also at risk of developing aspiration pneumonia, acute respiratory distress syndrome, pulmonary hemorrhage, or pulmonary thromboembolism. Respiratory rate, pattern, and effort, as well as saturation, need to be closely monitored. Thoracic radiographs (3 views) and/or thoracic POCUS should be performed if respiratory signs develop. Oxygen therapy should be provided if $SpO_2$ is 95% or less. In some cases, mechanical ventilation or high-flow nasal oxygen therapy is indicated.

**Cardiovascular system**

During the initial patient assessment, vital signs should be evaluated (TPR, mucous membrane color, CRT, pulse quality). Blood pressure should also be measured, and an ECG evaluated. Intravenous isotonic crystalloid fluids should be administered rapidly to restore blood volume and treat shock. To guide the volume of fluid to be administered, vital signs should be monitored closely (immediately after a bolus, and at least every 4 hours, or more frequently in critically-ill patients). Measurement of PCV/TP, electrolytes, blood glucose and blood lactate can help guide fluid therapy.

**Coagulation and hemostasis**

As body temperature rises, vascular damages occur, leading to the activation of the coagulation system. This results in hypercoagulability, which can lead to microthrombi formation, and eventually to DIC. Platelet aggregation is increased when platelets are exposed to heat. Hyperfibrinolysis may also occur. Thermal damage can cause liver dysfunction, exacerbating hemostasis disorders. Evaluation of coagulation times (activated partial thromboplastin time and prothrombin time), and examination of a blood smear to determine platelet count are recommended. The presence of petechiae, ecchymosis, epistaxis, hematuria, hematochezia, should be closely evaluated. In the event of coagulopathy (prolonged PT/aPTT, bleeding, reduced platelet count), transfusion of fresh-frozen plasma with or without packed red blood cells, depending on the presence or not of blood loss, may be necessary.

**Digestive system**

Direct thermal damage and poor blood perfusion can lead to gastrointestinal signs. Damages to the digestive mucosa increases the risk of bacterial translocation and endotoxemia, and hence sepsis. Vomiting and diarrhea (often hemorrhagic) are not uncommon during heatstroke. Supportive treatments should be considered in the event of vomiting (maropitant 1 mg/kg q24h) or ileus (metoclopramide 1-2 mg/kg/day CRI) and to limit lesions (pantoprazole 1 mg/kg IV q12h, sulcrate). When digestive lesions are severe and there is a risk of bacterial translocation, antibiotics should also be considered (must should not be given in all cases as they are not always necessary). Finally, fluid therapy must be adjusted according to the severity of digestive losses.

**Urinary system**

Kidney cells can be directly damaged by heat, and dehydration, hypovolemia, shock, and microthrombi formation reduce perfusion and contribute to the development of acute kidney injury (AKI). This can also be compounded by rhabdomyolysis. During heatstroke, myocytes break down and release myoglobin into the circulation. The results is dark-brown urine (pigmenturia). In high concentrations, myoglobin is toxic to the kidneys and can contribute to the development of AKI. It is therefore important to measure creatinine on admission, and at least once a day. Evaluation of urine sediment is also important, as the presence of casts can indicate tubular damage. Depending on the severity of the heatstroke, it may be useful to monitor the patient's urinary output by placing a urinary catheter with a closed collection system. Finally, in case of AKI, significant electrolyte imbalances may be present, such as hyperkalemia, which needs to be treated aggressively depending on its severity.

**Nervous system**

Neurons are highly sensitive to thermal damages, and hemorrhages, necrosis, and edema are common in case of heatstroke. These lesions are associated with altered mental state or varying severity, seizures, tremors, blindness, etc. Hypoglycemia, which is common in dogs suffering from heatstroke, and hepatic encephalopathy, which can occur in case of severe liver damage and impaired liver function, can both contribute to neurological signs. Systemic hypertension with bradycardia (Cushing's reflex) may also be noted, indicating increased intracranial pressure. Mental status monitoring, cranial nerve assessment, and close monitoring of vital signs should therefore be included in the management of these patients.

**Blood tests**

Blood tests should be performed promptly to obtain an overall picture of the patient's initial condition on presentation to the emergency room. The following parameters should be assessed: PCV/TP, blood glucose, platelet count by blood smear, and PT/aPTT. Evaluation of creatinine and electrolytes is also important. Serum color should be noted to evaluate for the presence of jaundice, as well as urine color, to evaluate for the presence of pigmenturia. Urine specific gravity should also be measured. CBC/biochemistry is also useful to assess the extent of damage. Thrombocytopenia is frequently observed, as is the presence of nucleated red blood cells. In heatstroke, the bone marrow prematurely releases young red blood cells into the circulation under the direct effect of heat, or under the effect of cytokines from the inflammatory response. The presence of more than 18 nucleated red blood cells/100 white blood cells has been shown to be associated with a poor prognosis in dogs suffering from heatstroke.

## Prognosis

The prognosis for dogs diagnosed with heatstroke is guarded to poor. Death usually occurs within the first 24 hours, due to multiple organ failure. Reported mortality rates vary between 40 and 64%. Some prognostic factors associated with mortality have been reported: hypoglycemia, prolonged PT/aPTT, increased creatinine, late presentation (more than 90 minutes) to the hospital, seizures, and obesity. Successful treatment depends on rapid diagnosis and implementation of cooling measures. Referral to an ICU for close monitoring and intensive treatment of complications should be considered.

## Conclusion

Given the high cost of care and the risk of death, it is beat to prevent heatstroke. Owners of brachycephalic dogs or dogs with upper respiratory tract disorders need to be particularly vigilant. These animals should be kept in a cool environment and exercise avoided on hot spring/summer days. At the mildest sign of heat-related illness, cooling measures should be put in place. It is also important to make pet owners aware of the risk of leaving a pet in the cat in hot weather, even for a few minutes. Finally, as the prognosis can be affected by the speed of intervention, the veterinary team must be able to recognize heatstroke quickly and implement cooling and stabilization measures as soon as possible.

**References available upon request.**