Heat Stroke: Putting Out the Fire

By Mitchell Fults, DVM, DACVECC

We are well into summer with temperatures in Seattle reaching the mid to high 90s F. Pets are not acclimated to this type of weather, which leads to an increase in frequency of heat stroke cases. Heat stroke is defined as hyperthermia resulting in a systemic inflammatory response and multi-organ dysfunction where encephalopathy predominates. Heat stroke results from an inability to dissipate heat adequately. This most often occurs in a hot and humid environment or due to strenuous physical exercise.¹

In healthy dogs and cats at ambient temperatures below body temperature, 70% of heat loss is by radiation and convection through the skin. An increase in body heat is detected by a variety of temperature sensors in the skin, viscera and central nervous system (CNS). The initial response to an increased temperature is an increase in cardiac output, which increases blood flow and vasodilates the skin, allowing heat to be lost through radiation, conduction and convection. However, this mechanism is only effective so long as the environmental temperature is less than the temperature of body. Once ambient temperature is near body temperature, evaporation through the respiratory tract becomes the predominant mechanism of heat loss and the brain initiates panting. Panting is an effective form of heat dissipation because the rate of panting (200 br/min) is close to the resonant frequency of the respiratory system, meaning no extra body heat is produced. The increased surface area of the nasal turbinates and ptyalism also contribute to heat loss with panting.

Humidity, poor ventilation and upper airway diseases all decrease the effectiveness of panting. As humidity increases, the evaporation efficiency is decreased, and at a humidity greater than 80%, evaporation is negated. Hypovolemia, poor cardiac output, obesity, thick hair coat and lack of acclimatization are also risk factors. The most common drugs used in dogs and cats that predispose them to heat stroke include acepromazine, furosemide and negative inotropes.¹

The pathophysiology of heat stroke is the result of an acute phase response initiated by an increased body temperature once cooling methods fail. Both pro and anti-inflammatory mediators are produced, along with intracellular heat shock proteins which protect cells against further heat insults by guarding against denaturation of intracellular proteins. The acute phase response also causes systemic release of endotoxins from the intestines and subsequently the excessive activation of white blood cells and endothelial cells, which in turn leads to the excessive release of inflammatory mediators, activation of the coagulation cascade and inhibition of fibrinolysis. Direct endothelial cell injury from heat combined with the initial hypercoagulable state results in microthrombosis and progressive tissue injury.

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Heat Stroke: Putting Out the Fire (Continued)

Once the pro-inflammatory and pro-coagulation process overpowers protective measures, multi-organ dysfunction results. On physical exam, panting and elevated temperature are the most common findings. Most patients are hyperdynamic with a rapid capillary refill time, weak pulses and sinus tachycardia. They may exhibit vomiting and diarrhea. An EKG should be performed during initial triage; presence of intermittent ventricular tachycardia indicates a worse prognosis. Temperature will be elevated in most cases, but can be normal or even low particularly with heat cramps and exhaustion. Mentation is often depressed. Some are cortically blind on admission, which usually resolves over a period of hours. Some may be ataxic from hypoglycemia, poor cerebral perfusion, direct thermal damage, cerebral edema or CNS hemorrhage.

Initial diagnostics should include CBC, Chemistry panel, urinalysis and clotting times. Hemoconcentration (elevated Hct and RBC count) and elevated BUN from decreased renal perfusion and/or GI hemorrhage are common. Excessive panting can quickly lead to hypernatremia due to loss of free water. ALT and creatinine typically peak within 24 to 48 hours. Initiation of the coagulation cascade caused by direct thermal injury to tissues and endothelium result in consumption of platelets and clotting factors leading to a decrease in platelets and prolonged clotting times. The presence of acute renal failure and DIC are known to increase mortality as is the finding of hypoglycemia. Interestingly, an elevation in nucleated RBC with a cutoff of 18/100 WBC has a 90% sensitivity and specificity for nonsurvival.

Treatment for heat stroke patients involves restoring normal body temperatures, restoring hydration and correcting electrolyte derangements as necessary. When trying to cool heat stroke patients, we try and keep it simple. Whole body wetting combined with muscle massage and blowing fans is the most beneficial method (in human heat stroke patients as well as animals). This can be started before the patient reaches the hospital; if an owner calls and says they are bringing their pet in for heat stroke, they should hose their pet down prior to arrival as this can lead to a 30% reduction in mortality. Once in hospital, patients are soaked with cool water from shoulder to hind end and placed in front of a fan. You may also use wet towels, but it is important to move the towels regularly as a static wet blanket can act as an insulator and retard heat loss. We typically don’t apply alcohol on the body pads as there is no evidence it is better than water. Whole body alcohol bathing should be avoided, as it poses a fire hazard and can cause local vasodilation and absorption leading to toxicity. Other techniques such as iced gastric lavage, cool water enemas and iced peritoneal lavage can result in serious complications and have been shown in various studies to provide no additional benefit. I typically do not apply ice packs, but if you do they should be wrapped to prevent burns and applied in the groin and axillary regions.

These cooling methods can also be used for other causes of hyperthermia (slug bait toxicities, status epilepticus, etc), though it is important to note that cooling should not be applied to patients with a fever. Fevers are due to an increase in the temperature set-point in the brain, and active cooling will only force patients to burn more energy to maintain that temperature. Active cooling methods should be discontinued and patients towel dried once the temperature reaches 103.5F to prevent rebound hypothermia, which is a negative prognostic indicator.

IV fluid administration is paramount when treating heat stroke. I use room temperature fluids. These patients often require aggressive fluid therapy with crystalloids, colloids and sometimes vasopressors (e.g. norepinephrine or dopamine) to maintain blood pressure. If two to three 20 ml/kg crystalloid boluses do not normalize blood pressure, I bolus colloids. Supplemental O₂ should be given initially followed by assessment of pulse ox and arterial blood gases if available. Putting a few ice chips in the patient’s oxygen mask can be considered as it will significantly cool the inhaled air and help cool the nasal turbinates and increase patient comfort (make sure the ice is not blocking the oxygen hole). NSAIDs and high dose steroids should be avoided. However, steroids can be given to brachycephalic breeds with laryngeal edema compromising ventilation. In these cases low dose Dex SP 0.15mg/kg IV should be used. I otherwise don’t use steroids in heat stroke.

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In order to help provide the best recovery for pets who have had surgery or who are recovering from trauma, we now offer rehabilitation services under the direction of Jason Spina, DVM DACVS-SA and Holly Gambill, LVT CCRA. Rehabilitation services and exercises can help reduce pain and enhance return to function after surgery but can also be useful in pets who need to lose weight, pets with arthritis or pets with trauma that are not surgical candidates. Initial consults are scheduled with Dr. Spina and include a thorough history and evaluation of muscle strength, joint flexibility, areas of discomfort and tightness. A detailed individualized plan will be created to help with at-home appropriate structured exercise as well as potential application of in-clinic exercises, laser therapy, electrical stimulation and use of the underwater treadmill.

BluePearl Rehabilitation and Physical Therapy Services include underwater treadmill, cold laser therapy, guided range of motion exercises, manual therapy and heat and cold therapy.

For questions or consultation, please call BluePearl Seattle at 206.364.1660 and ask for Dr. Jason Spina.

Heat Stroke (Continued)

Hypoglycemia is not uncommon in severe patients, which may require boluses of dextrose 0.25-0.5g/kg. If hypoglycemia persists, dextrose should be added to the IVF.

Once perfusion is improved, mentation should be re-evaluated. If it is still abnormal, cerebral edema may be present justifying mannitol 0.5-1g/kg IV given over 20 minutes. In severe cases where mentation is not much improved after an hour of therapy, a urinary catheter should be considered to monitor urine output. A lidocaine CRI at 25mcg/kg/min can be started initially to help decrease inflammation and act as a free radical scavenger. Electrolytes, renal values, coagulation panels and vitals should be monitored closely. If PT and/or PTT is greater than 1.5X normal, I will start fresh frozen plasma at 5-10ml/kg. Sucralfate and pantoprazole 1mg/kg IV Q12h can be used for gastric ulceration. Breakdown of the GI mucosal barrier may result in bacteremia or endotoxemia, so broad spectrum antibiotics such as Unasyn 30mg/kg IV Q8h can be considered if bacterial translocation is a concern. I reserve antibiotics for the most severe cases and do not use them where the patient is much improved after the first few hours of treatment.

Prognosis for severe heat stroke has a 50% mortality based on a previous study. I believe we get a better sense of these patients by how they are doing within the first 12 to 24 hours. There can be permanent systemic damage and CNS damage at sustained temperatures as low as 105F. That said, dogs can and do survive temperatures in excess of 108F. This is in part due to intrinsic thermal resistance in the canine brain which has not been found in many other species. This is often why with aggressive management we can see heat stroke cases with extremely high body temperatures be neurologically normal and ready to go home within 12 to 24 hours.

References:
BluePearl Continuing Education Series 2015

CE for the Technician - Triage of the Emergency Patient
Presented by Melissa Hoemann, LVT, VTS(ECC)
Wednesday, September 9, 2015
7PM - Light Dinner/Discussion
Location: PIMA Medical Institute
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