

Pacific Tide

An informational newsletter

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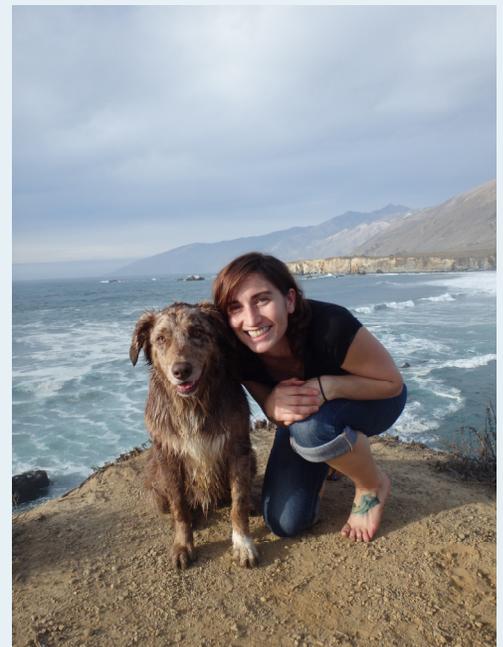
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About our Author

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Dr. Heidelberger graduated from UC Davis School of Veterinary Medicine in 2012 and completed a rotating small animal internship at VCA Emergency Animal Hospital in San Diego the following year. She loves wildlife medicine as well as emergency medicine, and enjoys volunteering at an exotics spay/neuter clinic and the Marine Mammal Center. She spends her spare time exploring the world with her Aussie pup Charlie, and enjoying beach life to the fullest.



**Sara Heidelberger,
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Managing Heat Stroke

By: Sara Heidelberger, DVM

What is heat stroke?

Heat stroke is a common medical emergency seen in small animal practice. It is a condition of extreme hyperthermia resulting from the body's inability to dissipate heat. Core temperatures often exceed 106°F, causing patient compromise. Heat stroke is classified as classical (resulting from environmental exposure), exertional (associated with exercise), or a combination of both. In all cases, the high internal temperature causes direct thermal injury to all the cells in the body, resulting in diverse and serious disease.

Presentation:

In most cases of heat stroke, animals have been exposed to either excessive exercise or high temperatures, although occasionally we get animals that are clinical despite only moderate exercise or environmental temperatures. Brachycephalic breeds are over-represented, as are dogs that are obese or have underlying clinical disease (eg. laryngeal paralysis, collapsing trachea, heart disease). Elevated body temperature is typically present. Other physical exam findings may include panting, lateral recumbency, collapse, weakness, altered mentation, tachycardia, pale or injected mucous membranes, and weak pulses. Hypotension is extremely common in these cases. GI signs such as vomiting and diarrhea are frequently present, with or without signs of GI bleeding. Rarely, petechia and ecchymoses are seen on initial presentation, although more commonly these signs develop later as the disease progresses and patients go into fulminant DIC.

Initial Stabilization:

The most important initial therapies in an overheated animal are to initiate active cooling and support the cardio respiratory system. Methods of cooling:

- Cool water soaks

- Fans over patient

- Constant rectal temperature monitoring

- Discontinue active cooling once the patient reaches rectal temperature of 103.0 °F or less. This will prevent rebound hypothermia and shivering

NOT RECOMMENDED: Ice baths and Inguinal and Axillary ice packs. These can cause significant peripheral vasoconstriction, making heat dissipation difficult.

IV catheter placement and IV fluid therapy is essential:

- Crystalloid boluses: 20-40mls/kg. Additional boluses are often required.

- Crystalloids at approx. 2 x maintenance. These patients may have significant ongoing fluid losses and distributive hypotension; thus they may require high rates of fluids initially.

- Hetastarch: consider initial bolus of 5mls/kg followed by maintenance at 1-2 ml/kg/hr. Only start hetastarch in hypotensive patients that have been appropriately rehydrated with crystalloids first

- Vasopressors: Occasionally necessary in refractory hypotension.

Diagnostic Testing:

Although heat stroke is generally diagnosed based on history and clinical signs, diagnostic testing is still important, both to direct management as well as to provide prognosis for survival. After initial triage therapies are started, CBC, chemistry, and clotting times (PT/PTT) are recommended. Common findings include:

- Elevated PCV, TS, and lactate

- Azotemia: Pre-renal, renal, or both. A persistently or progressively elevated creatinine in the face of fluid therapy is suggestive of acute kidney injury (AKI), and is a poor prognostic indicator.

Hypoglycemia: Suggests either significant hepatic compromise or sepsis. If refractory to initial treatment, this is a poor prognostic indicator.
Thrombocytopenia and prolonged PT and aPTT: Consumptive, secondary to systemic inflammation. Prolonged PT and PTT at presentation is a poor prognostic indicator.
Leukopenia: Consumptive secondary to widespread systemic inflammation; can also be suggestive of GI bacterial translocation and/or sepsis. Does not necessarily have prognostic significance.
Elevated hepatic enzymes: Due to thermal damage to the liver.
Electrolyte abnormalities: Common, and often easily corrected with basic fluid therapy.

In addition to blood work, several additional tests are strongly recommended as they provide further information and direct ongoing therapy.

Blood pressure to help monitor for and treat persistent hypotension
ECG to monitor for cardiac arrhythmias. VPCs and ventricular tachycardia are the most common
USG, urine output, and recheck BUN/Cr levels to monitor for AKI
Thoracic radiographs to monitor for pulmonary edema and ARDS if there is concern for respiratory compromise

Pathophysiology and Ongoing Treatment:

The pathophysiology of heat stroke has many similarities to sepsis, as the high temperature triggers a systemic inflammatory response which may lead to multiple organ dysfunction syndrome (MODS). Common MODS manifestations include circulatory collapse, ARDS (Acute Respiratory Distress Syndrome), intestinal ischemia, AKI, DIC, hepatic failure, cerebral edema, myocardial dysfunction, pancreatitis, and rhabdomyolysis.

Circulatory Collapse: The body's initial response to hyperthermia is to initiate massive peripheral vasodilation, resulting in hypotension and distributive shock. Blood starts pooling in visceral organs, decreasing circulating blood volumes, and further compromising blood pressure. Crystalloid, colloid, and vasopressors such as dopamine may all be used to maintain blood pressures.

ARDS: ARDS can result due to the systemic inflammation caused by heat stroke, generally manifesting as pulmonary edema. Respiratory support is often indicated; some patients do well with flow by oxygen, nasal cannulas, or oxygen cages; some patient's lungs are so severely affected by ARDS that they require intubation and ventilation. In brachycephalic breeds intubation and sedation may be more frequently required to prevent continued respiratory distress and progression of clinical signs.

Intestinal Ischemia: The GI tract is the 'shock organ' of the dog with hypotension, affecting GI perfusion early in the course of illness with resulting gastrointestinal signs. Poor perfusion and thermal injury lead to mucosal sloughing, ulceration, cell necrosis, and a decreased epithelial barrier. This often results in hemorrhagic diarrhea, hematemesis, and high risk for bacterial translocation. GI supportive medications should be started and maintained until resolution of clinical signs. These may include a combination of:

Maropitant (1mg/kg IV/SQ q24h),
Ondansetron (0.5mg/kg IV q12r),
Metoclopramide (1mg/kg/d IV as CRI),
Pantoprazole (1mg/kg IV q24hr),
Famotidine (0.5mg/kg IV q12h), and
Antibiotics (ampicillin 20mg/kg q8hrs or equivalent).

Therapy may be warranted for bacterial translocation –

1. Meds for nausea,
2. Meds for xileus
3. MOST IMPORTANT – assume gastric ulcers & use PPIs or H2 blockers, x 1 week +/-1 carafate

AKI: Damage to the renal tubular epithelium from direct thermal insult, or secondary to hypotension and distributive shock, may lead to serious kidney damage and possibly permanent loss of function. The large volumes of fluid necessary to maintain blood pressure will help to diurese the patient and protect the kidneys from further acute injury. If anuria or oliguria develop, prognosis worsens and more aggressive therapy is needed.

DIC: Thermal damage to endothelial cells causes systemic activation of clotting factors resulting in microthrombi within blood vessels. This hypercoagulable state causes an overall depletion in all coagulation factors. The body is unable to keep up with this rapid rate of consumption, and the patient may develop bleeding disorders. Replacement with fresh frozen plasma may be necessary to restore normal coagulation parameters. Frequently, repeat doses of plasma are required.

Hepatic Necrosis: Direct thermal damage results in hepatocellular necrosis and associated elevations in liver enzymes. Hypoglycemia can be seen. Fluid therapy, dextrose supplementation, and hepatic support medications such as denamarin are used to manage hepatic necrosis. Hypoglycemia does not indicate irreversible liver damage.

Cerebral Edema: Temperatures >109 can cause irreversible neuronal death, coma, and permanent brain damage. Even if the internal body temperature does not get this high, other complications of heat stroke include cerebral edema, cerebellar hemorrhage, and intracranial infarction/thrombi. These can result in obtundation, abnormal neurologic status, or seizures. If cerebral edema is suspected, Mannitol at 0.5-1g/kg slow bolus over 10-20mins can be considered, once euvolemic. Seizures are a poor prognostic indicator.

Myocardial Dysfunction: Cardiac arrhythmias (generally VPCs and ventricular tachycardia) are often seen but rarely need to be pharmacologically addressed. If VPC runs last for several minutes at a time at a heart rate greater than 180bpm, or if the patient's cardiovascular system is compromised by the arrhythmia, lidocaine is often a first line therapy. An initial lidocaine bolus of 2mg/kg IV slow, followed by a constant rate infusion of 25-75mcg/kg/min can be tried. Post-heat stroke arrhythmias are likely to be associated with myocardial thermal and ischemic damage, and their prognostic significance is uncertain.

Contraindicated Medications: In the past there have been discussions on the use of either steroids or NSAIDS as part of a complete therapeutic plan for managing heatstroke. The well-known adverse effects associated with these compounds at the renal, gastrointestinal, and platelet level make them strongly contraindicated and they should never be used in clinical heat stroke cases. One exception for steroid use is in cases of upper airway swelling, which may occur in brachycephalic dogs with laryngeal paralysis.

Prognosis and Prognostic Indicators:

Mortality in small animals with heat stroke has been reported to be around 50%. Several risk factors have been identified, but no clear cut-off points for mortality have been found, so aggressive therapy should be instituted as soon as possible for any patient presenting with heat stroke. More than half of fatalities occur during the first 24 hours, and according to one study death is unlikely if the animal survives the first 48 hours.

Poor prognostic indicators *at admit* include prolonged time before seeking treatment (>90 mins), obesity (not only a predisposing factor, but a poor prognostic indicator as well), hypothermia at admission, petechiation and/or prolonged PT/PTT, and hypoglycemia. Additional poor prognostic indicators during the course of treatment include development of seizures, an increasing creatinine in the face of ongoing, appropriate fluid therapy, and greater than 18 nucleated RBC's/100 leukocytes.

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Our Doctors

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Bryn Hoffman, MVB (Residency Trained in Internal Medicine)

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Lillian Good, DVM, DACVECC

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Radiology (VRS)

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Mark Lee, DVM, DACVR

Emergency

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Mark Saphir, DVM
Jessica Kurek, DVM
Sara Heidelberger, DVM

Behavior

Jan Brennan, DVM (practice limited to behavior)

About Our Hospitals

Pacific Veterinary Specialists was founded to provide high quality, specialized medical care to companion animal patients. Our practice is dedicated to serving the veterinary community as a partner in total patient care. We offer comprehensive specialized services including video endoscopy, Doppler ultrasound, surgery, 24-hour ICU care, and emergency and critical care. Our staff is committed to providing compassionate and thorough medical care that meets the needs of the patient, client, and referring veterinarian. In September 2011 we opened PVSM and currently offer internal medicine appointments and same day referrals, Tuesday through Thursday in Monterey. Behavior consultations by appointment are available on Mondays.

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