Heat related illnesses are common reasons for presentation to the emergency department, particularly during the summer months. Symptoms range in severity and patients are categorized based upon their clinical signs and body temperature, with heat stroke being the most severe form of heat related illness.

Both endogenous and exogenous factors affecting either the ability to dissipate heat or increase heat production can predispose patients to development of heatstroke. In an attempt to reduce the risk of hyperthermia during an episode of heat stress, cellular and systemic compensatory mechanisms are activated. Thermoregulation, acclimatization, the acute phase response and induction of heat shock proteins all play a role in this compensatory response, however, when these compensatory systems become overwhelmed, progression to heatstroke and its associated pathophysiologic derangements occurs.

Pathophysiologic derangements include increased metabolic demand, hypoxia, circulatory shock, cytokine/chemokine release and activation, endothelial cell damage, endotoxemia, activation and amplification of inflammation, and activation of coagulation and fibrinolysis. These massive alterations can progress to multiple organ dysfunction, with circulatory collapse, acute renal failure, encephalopathy, coagulopathy, hepatic failure, intestinal ischemia/infarction, acute respiratory distress syndrome, myocardial injury and endothelial dysfunction all possible sequelae.

Clinical signs vary greatly depending upon the severity of the disease. Initial rapid patient assessment should consist of evaluation of the airway, breathing and circulation. Upper and lower airway disease is common in patients with heatstroke, and close attention should be paid to the respiratory character and pattern. Intubation may be necessary for airway control, particularly in patients with obstructive upper airway disease, or in those who present obtunded or comatose. Most patients present with significant perfusion deficits and fluid resuscitation is vital in initial stabilization. At a minimum, an emergency database should be collected during initial assessment (packed cell volume/total solids, blood glucose, electrolytes, lactate, creatinine +/- blood gases) and can be used to help guide initial resuscitation.

Active cooling is an integral part of treatment and can be initiated by owners even during transport to the clinic. Patients with thick haircoats should not be soaked in water, as wet hair can form a barrier to heat dissipation—thinly haired areas should be cooled by applying towels soaked in cool water. Increasing air circulation over the body will also help improve evaporation. Using alcohol as a coolant should be avoided and cold water enemas and gastric lavage are
contraindicated. Active cooling should be discontinued when the patient’s temperature reaches 103.1°F, as there is a risk of progression to hypothermia if excessive cooling continues.

Comprehensive laboratory analysis is indicated in all patients with heat related illness. Common CBC abnormalities include white blood cell alterations, hemoconcentration or anemia, thrombocytopenia and the presence of nucleated red blood cells. Chemistry panel abnormalities can vary, with elevations in ALT and total bilirubin a common finding, along with azotemia, creatinine kinase elevations, and hypoglycemia. Varying electrolyte abnormalities are also common. Urinalysis is warranted to evaluate the patient’s concentrating ability and to detect myoglobin, bilirubin or casts within the urine.

Baseline coagulation testing, to include a platelet estimate, partial thromboplastin time (PTT), and prothrombin time (PT), is indicated in all heatstroke patients, particularly those with suspicion for active blood loss. Heatstroke patients are at risk for development of DIC and close monitoring for changes supportive of this diagnosis is warranted.

Balanced crystalloid solutions are recommended as the initial fluid of choice, administered as incremental large volume boluses (20ml/kg for dogs) via rapid infusion (over 10-15 minutes). Normal end parameters (normal heart rate, blood pressure, improved mentation) should be targeted. If necessary, synthetic colloid solutions may also be added to attain normal perfusion in patients who are refractory to large-volume crystalloid infusion, however, keep in mind the potential for development of potential coagulopathy and acute kidney injury with use of synthetic colloids. If hypotension persists despite adequate fluid resuscitation, vasopressor therapy may be necessary. Continued fluid therapy to meet ongoing losses and maintenance needs should be determined based upon frequent patient reassessment.

Additional components of therapy include dextrose supplementation if hypoglycemia is present and broad spectrum antibiotic therapy in all patients with signs of sepsis or those with evidence of gastrointestinal mucosal disruption (hematochezia, hematemesis, melena) because of the risk of bacterial translocation. Gastroprotectant therapy and implementation of enteral nutrition is also indicated. If intracranial hypertension is suspected, mannitol or hypertonic saline therapy should be utilized. Blood product therapy may also be necessary, with the specific product to be administered based upon the patient’s underlying needs. Routine use of corticosteroids is discouraged, as benefit has not been proven and increasing risks of gastrointestinal ulceration and immunosuppression exist.

Frequent reassessment of the patient is necessary, monitoring blood pressure, heart rate, perfusion parameters, respiratory status, neurologic evaluation and repeating laboratory analysis as indicated by initial abnormalities. Admission to a facility with 24 hour care is warranted as the condition of these patients can change rapidly, necessitating prompt intervention.

Outcome is largely dependent upon initial presenting severity and the patient’s response to aggressive treatment within the first 12-24 hours. Negative prognostic indicators include delayed
admission to the hospital (>90 minutes), seizure activity, obesity, and elevated serum creatinine (>1.5mg/dL). Published mortality rates range from 50%-56% in the veterinary literature.