

Heat Stroke in Dogs – Pathophysiology, Clinical Manifestations and Intensive Care Management

Franziska Meyer, Dr. med. vet., DACVECC, FVH

Introduction

Heat stroke is a life-threatening syndrome characterized by **severe hyperthermia associated with systemic inflammatory response and multi-organ dysfunction**, in which neurological impairment predominates. It occurs when thermoregulatory mechanisms fail and body temperature exceeds approximately **40–41 °C** without a change in hypothalamic set point.

In dogs, heat dissipation relies predominantly on **panting and evaporative cooling**, as sweat glands are limited to the paw pads. Environmental conditions such as **high ambient temperature, humidity, confinement in hot vehicles, and intense physical exertion** represent common triggers. Predisposing factors include **brachycephalic conformation, obesity, dehydration, advanced age, and poor acclimatization to heat**.

Thermoregulation and Physiological Adaptation

Dogs dissipate heat through several mechanisms:

- **Evaporation:** panting and evaporation from the tongue
- **Conduction:** contact with cooler surfaces
- **Convection:** air movement across the body
- **Radiation:** passive heat loss to cooler surroundings

Adaptation to warm environments occurs over **weeks to months** through acclimatization processes. One important mechanism is the **carotid rete mirabile**, a vascular network that cools arterial blood supplying the brain through heat exchange with cooler venous blood. Additionally, **heat shock proteins (HSPs)** are synthesized in response to thermal stress, helping maintain protein integrity and cellular homeostasis.

Pathophysiology

Heat stroke results from a complex cascade involving:

- Direct cytotoxic effects of hyperthermia
- Oxidative injury
- Systemic inflammatory response
- Distributive and hypovolemic shock
- Increased metabolic demand and hypoxia

These mechanisms ultimately lead to **multi-organ dysfunction syndrome (MODS)** with predominant neurological involvement.

Key secondary complications include:

- Disseminated intravascular coagulation
- Rhabdomyolysis
- Acute kidney injury
- Cardiovascular collapse

Organ System Involvement

Neurological System

Neurological dysfunction is a hallmark of severe heat stroke.

Clinical manifestations include:

- Delirium
- Stupor
- Coma
- Seizures

Underlying lesions may include **cerebral edema, hyperemia, hemorrhage, and neuronal necrosis**. Contributing factors include direct thermal injury, shock, and metabolic disturbances such as hypoxemia or acid–base imbalances.

Gastrointestinal System

Intestinal injury occurs due to **splanchnic vasoconstriction and ischemia** followed by nitric-oxide mediated vasodilation and oxidative damage.

Consequences include:

- Mucosal barrier disruption
- Bacterial translocation
- Endotoxemia
- Increased risk of sepsis

Muscular System

Rhabdomyolysis frequently develops due to muscle hypoperfusion and direct thermal injury. ATP depletion leads to muscle cell necrosis and **myoglobin release**, which contributes to systemic toxicity and renal damage.

Cardiovascular System

Cardiac complications arise from:

- Direct thermal injury
- Inflammatory cytokine release
- Hypotension and hypovolemia
- Microvascular thrombosis

These mechanisms may cause **cardiac arrhythmias and myocardial dysfunction**.

Renal System

Acute kidney injury (AKI) is common and may initially be **subclinical**, requiring careful monitoring of renal parameters.

Mechanisms include:

- Myoglobin-induced nephrotoxicity
- Oxidative stress
- Microvascular thrombosis
- Ischemia and inflammation

Hemostatic System

Severe endothelial injury activates the coagulation cascade, resulting in:

- Platelet activation
- Release of von Willebrand factor and tissue plasminogen activator
- Microthrombosis

This process can progress to **disseminated intravascular coagulation (DIC)** and bleeding disorders, clinically manifesting as **petechiae, ecchymoses, and hematomas**.

Respiratory System

Pulmonary injury occurs secondary to inflammatory and oxidative mechanisms, leading to:

- Pulmonary edema
- Lung infarction
- Hemorrhage

Respiratory complications, particularly **acute respiratory distress syndrome (ARDS)**, are a major cause of death in dogs with heat stroke.

Clinical Presentation

Dogs with heat stroke may present with:

- Lethargy to coma
- Tachycardia and panting
- Dyspnea or tachypnea
- Dehydration and hypovolemia
- Petechial hemorrhages

Interestingly, patients may be **normothermic or even hypothermic at presentation** due to cooling attempts prior to admission.

Laboratory Findings

Typical abnormalities include:

- Increased CK, AST and ALT
- Myoglobinuria
- Hemoconcentration (elevated PCV and total solids)
- Thrombocytopenia
- Prolonged PT and aPTT
- Nucleated erythrocytes and botryoid neutrophils in blood smears.

Treatment

Immediate Cooling

Rapid reduction of body temperature is the cornerstone of therapy and is achieved with Immersion in water, and active air movement using fans.

Fluid Therapy

Initial stabilization involves **crystalloid resuscitation** with early consideration of **vasopressors**. Subsequent therapy should use **conservative fluid administration**, as these patients often exhibit vascular leakage and limited fluid responsiveness similar to septic patients. Excessive fluids may lead to pulmonary edema and worsen outcomes.

Management of Coagulopathy

Coagulation abnormalities require dynamic monitoring and should be treated promptly with fresh frozen plasma, and platelet transfusions. Anticoagulation may be considered if hypercoagulability has been demonstrated.

Cardiovascular support consists of the treatment of clinically significant arrhythmias, optimization of the circulating volume, and inotropic support (eg. Pimobendan, vasopressors) when indicated.

Respiratory Support depends on the severity of dyspnea and includes regular oxygen supplementation, high-flow oxygen therapy, and mechanical ventilation. When ventilating patients with ARDS, **lung-protective ventilation strategies with low tidal volumes** are recommended.

Prognosis

The prognosis for dogs with heat stroke is **guarded to poor**, with reported mortality rates approaching **50%**.

Favorable prognostic factors include:

- Rapid and effective cooling within **30–60 minutes**
- Absence of severe neurological signs
- Normal coagulation parameters
- Preserved renal function

Poor prognostic indicators include progression to **multi-organ dysfunction**, severe neurological impairment, and persistent renal injury. Long-term complications may include **chronic kidney disease, neurological deficits, and increased susceptibility to future heat stress**.