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Hypothermia, Hyperthermia & Thermal Injury

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Hypothermia

Primary hypothermia occurs in the presence of normal heat production, and usually results from exposure to a cold environment.

Secondary hypothermia occurs from illness, injury, or drug-induced alterations in heat production and thermoregulation. Severe or critical secondary hypothermia may increase morbidity and mortality in critically ill animals. Primary and secondary hypothermia occur during commonly encountered clinical scenarios, including:

- Surgery or general anesthesia
- Trauma
- Hypovolemia
- Environmental exposure

Therapeutic efforts are aimed at rapidly rewarming patients during fluid resuscitation as well as reducing additional heat loss. Resuscitative efforts should not contribute to the hypothermia. Rewarming hypothermic animals can be accomplished by several different methods, including:

- Passive Surface (e.g., blankets)
- Active Surface (e.g., Bair hugger, heated water bed)
- Active Core Rewarming (e.g., peritoneal and pleural lavage)

It has been recommended to warm the animal by at least 1-2 degree Celsius per hour; however, faster rates may be necessary. Moderate intravascular volume support is recommended during active rewarming in hypovolemic shock; this will support mean arterial blood pressure (MAP) and resolve most cases of hypothermia-induced hypotension, bradycardia, hypoventilation, and coagulopathy while avoiding volume overload. Electrolytes and acid-base status should be monitored and alterations addressed. The ECG, MAP, and blood gases should be monitored closely in severely hypothermic patients. Surface rewarming should always accompany active core rewarming to reduce core-to-peripheral temperature gradients. During external heating, care must always be taken to prevent skin burns by controlling the temperature of the external heating devices or placing a barrier between the heat source and the patients.

A patient's core body temperature may continue to drop for a period of time after the onset of rewarming. This condition, referred to as the "afterdrop" is caused by the return of cold peripheral blood to the body core and movement of blood from the warmer core to the periphery. A second important complication to anticipate is the development of rewarming shock. Rapid rewarming will cause a great metabolic burden on patients as well as significant vasodilation that may overwhelm an already compromised circulatory system.

Heatstroke

Heatstroke is a life-threatening emergency that should be considered in any dog that presents with a core body temperature $>104^{\circ}\text{F}$ and has a history of environmental exposure. Heat dissipation occurs via four different mechanisms:

1. Convection: transference of heat from the body as air passes over it
2. Conduction: occurs when the body comes into contact with a cooler surface, to allow heat transference
3. Radiation: a natural process by which the body releases heat into the environment
4. Evaporation: an endothermic process that involves fluid changing into a vapor

In our patients, evaporation (panting) becomes the most important mechanism for dissipating body heat as the environmental temperature increases. Factors that predispose to decreased heat dissipation include both exogenous and endogenous causes:

- Exogenous causes: lack of acclimatization, confinement and/or poor ventilation, increased humidity, water deprivation, drug administration (furosemide, negative inotropic drugs, phenothiazines).
- Endogenous causes: brachycephalic anatomy, laryngeal paralysis, overweight/obesity, cardiovascular disease, neurologic/neuromuscular disease, age, and hair coat/color.

Predisposing factors that increase the body's heat production include exogenous (amphetamines, metaldehyde, macadamia nuts,

organophosphates, halothane) and endogenous (exercise, pyrexia, hormonal hyperthermia, seizures, eclampsia) causes. Core body temperature elevation activates an inflammatory cascade and yields the release of several substances that produce systemic injury that may induce multiple organ failure, coagulopathies, and ultimately DIC.

If owners call prior to a their pet's presentation to the hospital, they should be advised to begin cooling at home. The animal should be sprayed down with room temperature water before transporting to the hospital. Driving with the windows down and/or placing the dog in front of an air conditioning vent may enhance evaporation. Bruchim et al (*J Vet Intern Med*, 2006) showed an overall mortality rate of 50% in dogs presenting with heatstroke, but mortality rate was 38% in those cooled at home versus 61% for those who were not.

Initial patient assessment should include a performing a primary survey and obtaining a minimum database (MDB) and blood pressure. Physical exam findings consistent with heatstroke include an elevated rectal temperature, altered level of consciousness, brick red mucous membranes, tachypnea/dyspnea, and petechiae/ecchymoses. A recommended minimum database is PCV/TS, platelet count, peripheral blood film evaluation, lactate measurement, creatinine, blood glucose, electrolyte evaluation, urine specific gravity, and PT/PTT.

The veterinarian should provide supplemental oxygen and must ensure the patient has a patent airway. External cooling should be provided by spraying the patient with water (particularly inguinal, axillary and dorsal cervical regions) and placing the patient in front of a fan. Submerging a patient's body in a cold bath, using topical alcohol (i.e.: applied to foot pads), and cold gastric lavage are NOT recommended methods for cooling heatstroke patients. Cooling methods should be discontinued when the body temperature reaches 103°F to prevent rebound hypothermia. An IV catheter should be placed and blood samples should be obtained before administering IV fluids (using room temperature fluids). Appropriate resuscitative doses of isotonic crystalloids should be utilized initially, and if warranted, a synthetic colloid (i.e.: Hetastarch) may be added.

Patients should receive a broad-spectrum antibiotic to limit complications from bacterial translocation. Gastroprotectants, including proton pump inhibitors and pro-motility agents, are also recommended. The administration of corticosteroids and/or non-steroidal anti-inflammatory drugs is not recommended in heatstroke patients due to the alterations of gastric mucosal permeability and reduction of gastrointestinal prostaglandin production. Adjunct therapies that may be indicated, based on laboratory test results, include dextrose supplementation and fresh frozen plasma administration.

Heatstroke patients should be monitored around-the-clock for at least 24-48 hours following presentation, paying particular attention to respiratory status, cardiac rhythm, coagulation times, perfusion parameters (including blood pressure and lactate), acid/base status, electrolytes, urine output, and neurologic status. Retrospective studies show approximately 25-50% of patients die. Those presented within 90 minutes had a 27% mortality rate compared to 62% for those presented after 90 minutes. Other negative prognostic factors include coma or hypothermia, progressive neurologic deterioration, persistent hypoglycemia, worsening of azotemia or oliguria despite adequate fluid resuscitation, evidence of DIC, refractory hypotension, hyperbilirubinemia, ventricular dysrhythmias, persistent hypoproteinemia, labored respiration, and pulmonary edema. Aroch et al (*J Vet Intern Med*, 2009) showed number of nucleated red blood cells (NRBC) correlated with survival. Median relative NRBC was 24 cells/100 leukocytes (range 0-124) and was significantly higher in non-survivors versus survivors. A cut-off point of 18 NRBC/100 leukocytes corresponded to a sensitivity and specificity of 91 and 88% for death. Death typically occurs within the first 24 hours of presentation, while patients alive after 48 hours typically survive to discharge.

Thermal Injury

Thermal injury is characterized by depth of affected tissue. A superficial burn involves only the epidermis. The skin is hyperemic, painful, non-blistered, and will often heal in 5 days without scarring. A partial thickness burn involves the epidermis and the dermis, and is further divided into superficial partial thickness and deep partial thickness burn. A superficial partial thickness burn involves the epidermis and one half of the dermis. It is characterized by blisters, pain, blanching upon pressure, intact hairs, and will take 2-3 weeks to heal. A deep partial thickness burn leads to destruction of deep dermal layers, and may be dry or moist and blistered; the area will not blanch, and the hair will fall out easily. The burn will heal slowly to result in scarring and potential loss of function. A full thickness involves all dermal layers. The skin is often dry and leathery, and may appear white or charred, results in loss of sensation, and therefore may be non-painful although the tissue has been irreversibly damaged. A third degree burn will heal by contracture and epithelial migration or via excision and grafting. Third degree burns may result in eschar formation, the hard leathery product of a deep, full thickness burn.

Thermal injury is often characterized by the amount of body surface area that has been affected. In human medicine, the Rule of 9's is used to characterize burns as follows:

- Head/neck: 9%
- Each arm: 9%
- Each leg: 18%
- Thorax: 18%
- Abdomen: 18%

In patients with burns involving greater than 50% of their TBSA, prognosis is poor to guarded, and euthanasia should be discussed with the owner.

Severity of a thermal injury depends on the temperature of the medium, the concentration of heat, and the duration of contact. Jackson et al outlined three different zones evident on visual examination immediately following a burn injury: the zone of coagulation, the zone of stasis, and the zone of hyperemia. The zone of coagulation is the area in a burn nearest to the heat source. This zone suffers the most damage, resulting in clotted blood and thrombosis of vessels. Tissue properties are irreversibly altered secondary to extensive protein denaturation. The zone of stasis surrounds the zone of coagulation, and is characterized by decreased blood flow (blood stasis). There is active edema formation due to vasodilation and increased microvascular permeability. Excessive local edema is followed by hypoperfusion, further exacerbating local tissue ischemia. The tissue in this zone is potentially salvageable, and is therefore the focus of most burn resuscitation. Therapies are aimed at restoring perfusion to the affected area in order to prevent irreversible damage. The zone of hyperemia is the peripheral area around the burn, and is characterized by increased blood flow. The tissue in this zone will likely recover as long as sepsis or prolonged hypotension does not ensue.

The body's response to thermal injury may be described in four stages:

- Stage 1 (Emergent phase): occurs within minutes to hours of the initial injury, and is characterized by a pain response, catecholamine release, and subsequent tachycardia, tachypnea and mild hypertension

- Stage 2 (Fluid shift phase): lasts for 18-24 hours. During this phase, damaged cells initiate the inflammatory response. This local phenomenon results in a shift of fluid from the intracellular space to the extracellular space to cause capillary leakage and substantial edema
- Stage 3 (Hypermetabolic phase): may persist for days to weeks, and may be thought of as a period of profound increase in nutritional needs for proper tissue healing.
- Stage 4 (Resolution phase): results in scar formation with a gradual return to normal tissue function.

All veterinary burn patients should be provided with oxygen, intravenous fluids, and analgesia. The patient should be assessed for shock and treated accordingly. If inhalant injury is suspected, oxygen therapy should be continued and fluid therapy should be more conservative to prevent exacerbation of possible pulmonary capillary leak syndrome resulting from smoke inhalation. Additionally, the corneal epithelium is sensitive to temperature, and corneal ulcers are common. Therefore the eyes should be treated with lubricating and antibacterial ophthalmic medications.

Immediately following a thermal burn, the affected area should be cooled with water or damp towels for at least 10 minutes in order to prevent further thermal injury in the absence of heat. Even once the heat is removed, tissue may continue to burn. Cold water or ice should be avoided, as they may cause vasoconstriction and increased wound depth. Patients with burns involving more than 20% of their TBSA may develop severe metabolic derangements, and appropriate diagnostic samples should be obtained once the patient is stabilized. A recommended minimum database is PCV/TS, platelet count, peripheral blood film evaluation, lactate measurement, electrolyte, glucose, creatinine, urine specific gravity, and PT/PTT.

The greatest amount of fluid loss from thermal injury patients occurs during the first 24 hours secondary to increased microvascular permeability. Quantification of urine output, urine specific gravity (USG), body weight, packed cell volume/total solids (PCV/TS), lactate, blood urea nitrogen (BUN), creatinine, electrolytes and central venous pressure (CVP) should be used to better assess patient hydration status and fluid therapy needs.

Although early wound closure is ideal, it should not take place for the first 3-7 days to allow a wound to fully declares itself. Wound should first be cleaned with a povidone-iodine solution and then silver sulfadiazine should be applied. Alternatively aloe vera cream may be used for its antithromboxane effects to prevent vasoconstriction and thromboembolic seeding of the microcirculation. Unpasteurized honey has also been used with favorable outcomes in thermal injury management. Honey reportedly decreases inflammatory edema, accelerates sloughing of necrotic tissue, and provides a good energy source for development of granulation tissue. Sterile gloves should be worn at all times to protect the patient, and the wound should be cleaned on a daily basis until excision and closure can take place. Provision of moisture to the affected area(s) following the application of topical agents is crucial, and may be accomplished by applying a bandage with a non-adherent, porous inner layer allowing passage of fluids and exudates followed by an absorbent gauze/pad outer layer. Full thickness burns should be excised at the end of the first week if possible.

Conservative management of burn wounds prior to excision should entail hydrotherapy, removal of necrotic tissue, topical therapy and bandaging. This will help to prevent wound sepsis and SIRS in addition to decreasing morbidity and mortality and down regulating the hypermetabolic response. Another option for more conservative management is enzymatic debridement. This procedure entails the use of topical agents such as trypsin-balsam of Peru castor oil to soften, loosen, and digest necrotic tissue. This will facilitate easy removal of necrotic tissue with gentle lavage. Enzymatic debridement should only be considered in the very early stages of burn management, and must be discontinued once a healthy granulation bed has formed. Full thickness burns typically require skin grafts or flaps in order to achieve complete closure.

Significant thermal injuries induce a state of immunosuppression that predisposes patients to infectious complications. Most deaths in severely burned human patients are due to burn wound sepsis. Topical antibiotics are the antimicrobial of choice in veterinary patients, and systemic broad-spectrum antibiotics are indicated if there is evidence of immunosuppression, pneumonia, and/or sepsis. Due to their extreme state of catabolism, nutritional support in burn patients is vital for a positive outcome. Enteral nutrition may decrease the risk of translocation by promoting GI motility.

Inadequate pain management is detrimental to burn patients. Analgesia should be provided with pure μ agonists. Drugs such as fentanyl (2-8 mcg/kg/hr IV CRI), hydromorphone (0.025 mg/kg/hr IV CRI), and ketamine (0.15-0.6 mg/kg/hr IV CRI) are recommended. Ketamine has been used for over 40 years in the treatment of burn pain in humans. Ketamine inhibits GABA, and may also block serotonin, norepinephrine, and dopamine in the central nervous system. Ketamine can inhibit NMDA receptors in the CNS, and can decrease wind-up effect. Lidocaine may be used as an adjunctive analgesia and as a free radical scavenger (1.5-3 mg/kg/hr IV CRI). Gabapentin (5-10 mg/kg PO q8-12 hr) should also be considered in the thermal injury patient. Gabapentin is an anti-hyperalgesic drug that selectively affects central sensitization. Appropriate analgesia in the thermal injury patient should be multimodal, incorporating antagonism of both nociceptive and neuropathic pain pathways.

References

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