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A Review of Heat Stroke and Its Complications in the Canine

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SUMMARY

A review of heat stroke and its complications is presented. The etiology, physiology, clinical signs, secondary complications, diagnosis, treatment, necropsy results and prevention of heat stroke are discussed. A clinical case is then presented to illustrate the disorder of heat stroke.

INTRODUCTION

Adult dogs can survive in an environment in which there are wide variations of temperature because of their ability to regulate their internal temperature.[1] The internal temperature, however, must be maintained within the range of 5°C above normal to 15°C below the normal temperature of blood to avoid cellular injury or death. Thermal homeostasis occurs when there is a balance between "heat load" and heat dissipation. Heat load is defined as the summation of environmental and metabolic heat.[2] Heat stroke occurs when heat load markedly exceeds the ability of body compensatory mechanisms to promote heat loss. In man heat stroke is also due to ineffective thermoregulation. It is caused by the cessation of sweating, which is the main thermoregulatory mechanism. Heat stroke is characterized by hyperthermia (above 105°F), often complicated by alterations in many systems and organs such as acid-base balance, kidney, liver, cerebral edema, and the blood clotting mechanism.

ETIOLOGY

Several factors are necessary for, or contribute to, the induction of heat stroke. The most obvious prerequisite is a high environmental temperature. When the ambient temperature increases above 86°F, a rise in internal body temperature results. Dogs can tolerate rising environmental temperature quite well. However, when the body temperature exceeds 104°F a breakdown of the animal's thermal equilibrium begins. At 106°F the brain becomes involved and permanent damage may develop.[3]

Inadequate ventilation is one of the most critical factors in heat stress development. Greater than 70% of the total body heat loss in dogs and cats is due to radiation and convection from the body surface.[3] Static air around the body is soon elevated to body temperature, and surface heat loss is blocked. Animals placed in an enclosed environment with no air flow, such as an automobile, can develop serious problems.

Dogs that are confined by chain outdoors have been known to develop heat stroke. Often these dogs are unable to get into the shade or have no drinking water available. In these cases, excitement and exercise associated with animal fights appear to have precipitated heat stroke.[4] Although exercise and excitement can significantly contribute to the induction of heat stroke in confined dogs, heat stroke is rare in dogs that run free, regardless of exercise and air temperature.

In contrast to human heat stress, humidity has less of an effect on canine heat stress, primarily due to the poor development of canine sweat glands.[3] However, high humidity may contribute to the likelihood of heat stroke because evaporation of water from the oral and nasal cavities is reduced in spite of maximal panting.[4]

Other predisposing factors include lack of available water, brachycephalic anatomy, length of hair coat, obesity, specific dynamic

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action of food, and decreased heat tolerance associated with young and old age.

PHYSIOLOGY

Signals from temperature-sensitive organs in skin, viscera, and central nervous system converge in the caudal hypothalamus[7] (Fig. 1). The central effector signal is mediated through the endocrines, the autonomic nervous system, and by neuromuscular activity.[5] The initial compensatory response to increased ambient temperature is panting.[4] The panting mechanism may be initiated reflexively and centrally, by an increase in body temperature or external temperature and by local warming of the anterior hypothalamus. This mechanism of dissipating heat is efficient and involves a partial circle system of air passing into the nasal cavity and out the oral cavity.[6] This unidirectional flow maximizes evaporation and heat loss because of the greater evaporative surface area of the nasal turbinates to which the air is exposed. Increased salivation during panting greatly increases the evaporative cooling. Even though the animal becomes dehydrated by 6–10%, salivation continues at the same rate, resulting in a decreased plasma volume and flow. Panting, however, is not without drawbacks. Hyperventilation occurs which may cause respiratory alkalosis as carbon dioxide is blown off. The respiratory alkalosis induced in pentobarbital-anesthetized dogs subjected to high temperature, however, is eventually modified by metabolic acidosis presumably due to muscle activity associated with panting.[4] The acid-base status of the individual patient can be determined only if blood gas analysis is done.

Sympathetic vasoconstrictor nerves control the cutaneous vasomotor reactions to temperature changes. Inhibition of the sympathetic vasoconstrictor tone causes peripheral vasodilation, which increases heat loss. However, when external temperature exceeds 31°C, heat is no longer lost by cutaneous vasodilation. This inhibition is mediated by an increase in hypothalamic temperature or reflexively through thermoreceptors in the skin.[8] As blood is shunted to the peripheral vessels (capillaries), there is a large decrease in circulatory blood volume. Eventually, if there is no compensatory increase in blood volume, dilation of the heart and impaired cardiac efficiency, and finally impairment of respira-

tory centers occur. Tissue edema and hypoxia can develop which are most serious in such areas as the lungs and brain. The kidney can also be affected by shock-induced ischemia.

Tissue enzyme systems operate effectively over a narrow range of temperature. When this range is exceeded, widespread cellular damage and death of various tissues occurs. Central nervous system destruction, especially of Purkinje cells, and heart muscle destruction have been shown with heat stroke.[9] Renal tubular damage is probably a direct result of heat injury in cases not associated with shock. The liver is very susceptible, as heat itself causes liver cell death.

CLINICAL SIGNS, SECONDARY COMPLICATIONS, AND DIAGNOSIS

The onset of heat stroke in three-fourths of the cases is acute, with clinical signs developing fairly rapidly. The physical findings in dogs with heat stroke depend on duration and severity of the disease. Initial findings include panting, tachycardia, bright red oral mucosa and hyperthermia. The pulse is rapid and weak.[3] As the disease progresses the dog becomes stuporous due to the development of cerebral edema. The extremities become hot and dry to the touch. The bright red oral mucosa becomes pale, due to either peripheral vasoconstriction or decreased circulatory volume or both.[4] Massive diarrhea and vomiting may occur at this later stage. If the diarrhea becomes bloody, disseminated intravascular coagulation (DIC) may be a complication. Progression of cerebral edema to terminal stages leads to coma and respiratory arrest.

Survival from heat stroke depends heavily upon maintainence of a cardiac output sufficient to meet the elevated circulatory demand. Complications attributable to circulatory failure are myocardial damage and marked increase of pulmonary vascular resistance. That direct thermal injury is at least partly responsible for myocardial damage is suggested by widespread tissue injury in this condition.[10] Elevated pulmonary vascular resistance is suggested by vascular congestion and pulmonary edema, as well as right-sided dilation of the heart at necropsy.[11] Arrhythmias progressing from premature contraction and bradycardia to ventricular fibrillation and heart block may occur if hyperkalemia is present.
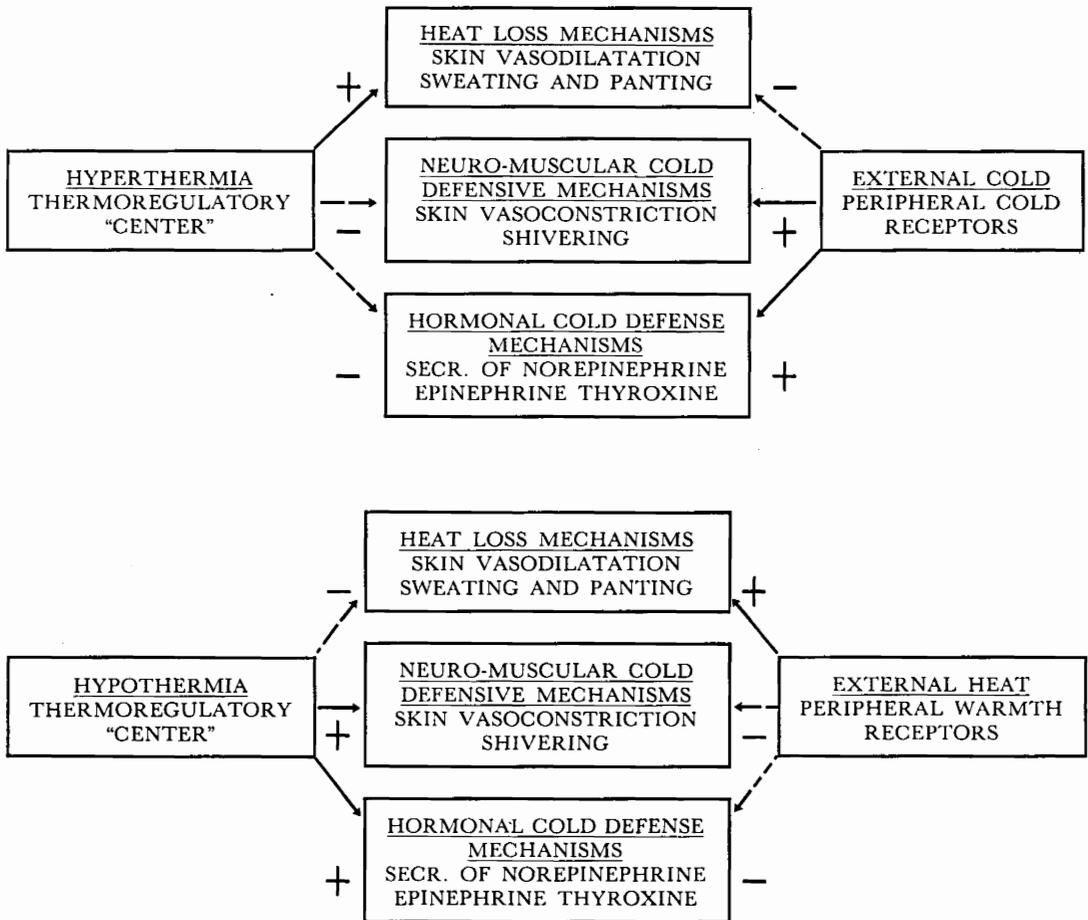
Figure 1. A tentative summary of temperature regulation in homeotherms. The upper part shows how an elevated body temperature (hyperthermia) may counteract the thermoregulatory effect of peripheral cold through the intermediation of the hypothalamic thermoregulatory center. The lower part illustrates the reverse of a subnormal body temperature (hypothermia). [Swenson, *Duke's Physiology of Domestic Animals,* 1977.]

The degree of hemoconcentration that occurs in heat stroke can be severe with packed cell volumes (PCV) as high as 75% recorded. Other blood changes include hyperkalemia, despite the developing alkalosis which under normothermic conditions is associated with hypokalemia. The liver and jejunum have been implicated as possible sources of potassium (K) released to the extracellular fluid. However, a few simple assumptions and calculations leave doubt that the liver and gastrointestinal tract could be the source of all the K changes observed.[12] As mentioned previously hyperkalemia can cause neuromuscular malfunction which eventually leads to muscular and respiratory paralysis. Of special concern are arrhythmias progressing to

ventricular fibrillation and heart block. Hypophosphatemia has been reported in experimental hyperthermic dogs (2.0 mg/dl) but the mechanism is unknown. Other alterations in serum electrolyte concentration known to be associated with experimental hyperthermia have been minor and probably are the result of hemoconcentration.[4]

Acute primary renal failure associated with necrosis of tubular epithelial cells is a common complication. The early phases of acute renal failure are associated with oliguria. The urine is turbid, scanty, and brownish.[13] If the patient survives, however, profound polyuria may develop.[2]

Disseminated intravascular coagulation (DIC) can also complicate heat stroke in the

dog. The triggering mechanisms are probably (1) platelet activation, (2) Hageman factor activation or (3) endothelial or tissue injury with intravascular release of thromboplastic substances.[13] The following mechanism has been proposed by Stefanini and Spicer[14] (Fig. 2). If bloody diarrhea or petechiae are present, DIC should be suspected. Coagulation test results vary with the duration of the disease making accurate diagnosis of DIC difficult. The initial phase of DIC is a hypercoagulable state, characterized by increased levels of procoagulants. The secondary phase is consumption coagulopathy, which can then proceed to a severe defibrination syndrome and resultant hemorrhagic state.[6,15] Consumption coagulopathy is classically characterized by thrombocytopenia, hypofibrinogenemia, and reduced levels of clotting factors V and VIII. The thrombin is usually prolonged because of the fibrinogen defect. Fibrin (fibrinogen) degradation products are present in the serum due to the activation of the fibrinolytic state. It is essential that this syndrome be differentiated from primary fibrinolysis, in which only the fibrinolytic system is activated. This is especially important in terms of treatment.

Another complication of heat stroke is cerebral edema. Although the mechanism of cerebral edema is unknown, it is an often seen complication. It has been hypothesized that failure of cerebral metabolism could result from a limited supply of nucleotides (cytidine diphosphate and uridine diphosphate from liver) required in cerebral glucose transport.[16] The decreased supply of nucleotides could be the result of liver injury. Body cells operate within certain thermal limits, and above these limits cellular degeneration begins. The brain is especially vulnerable to cellular damage because once neural cells are destroyed they are replaced by glial cells and not viable neural tissue. Almost all patients have cerebellar symptoms.[17] Dogs with cerebral edema are initially stuporous. Involuntary paddling and coarse tremors are often present and the dogs appear to be unaware of their surroundings. Seizures have occurred in all fatal cases.[18] As the edema progresses, the menace reflex is lost, and the dogs lapse into a coma. As stated previously the panting reflex is abolished, respiratory rate markedly decreases and death occurs due to respiratory arrest.

Heat stroke must be differentiated from eclampsia, hypoglycemia, encephalitis, convulsions, and similar conditions.[6] The sudden onset of signs with high rectal temperatures (excess of 105°F) is usually sufficient for diagnosis.

TREATMENT
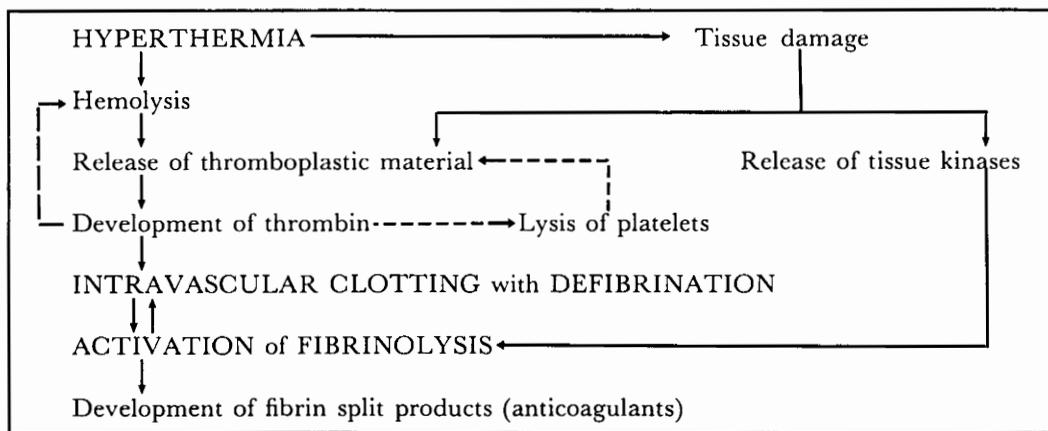The rapid progression of heat stroke dictates

Fig. 2.   Pathogenesis of intravascular defibrination, fibrinolysis, and hemolysis in heat stroke. Hyperthermia causes tissue and erythrocytic damage. Then: (a) release of thromboplastic material into the bloodstream initiates the formation of thrombin and results in intravascular defibrination; (b) release of tissue kinases triggers the fibrinolytic mechanism, the activation of which causes the appearance of anticoagulants; (c) an autocatalytic chain of events also results when thrombin causes agglutination and lysis of platelets and these, in turn, release additional thromboplastic material; (d) hemolysis may be enhanced by the direct effect of thrombin on the erythrocytes. [Stefanini and Spicer, "Hemostatic Breakdown, Fibrinolysis, and Acquired Hemolytic Anemia in a Patient with Fatal Heatstroke," 1971.]

that its treatment be prompt and intensive. The primary consideration of therapy is to lower body temperature. Total submersion in cold or ice water can cause shock, so one should douse legs, belly, nose, and neck with cool, not cold, water to avoid shock. The water must be sufficiently cold to remove heat from the body at a rate faster than it is produced by metabolism.[19] An alternative method of cooling is to sprinkle the dog with cold water and place the dog in the breeze of a fan. When rapidly cooling the dog, the threat of hypothermia is a very real one because the thermal set point may be altered as a result of possible brain edema involving the hypothalamus. Consequently, the rectal temperature should be monitored every ten minutes and the dog removed from the cold water and wrapped in blankets when his temperature reaches 103°F to prevent hypothermia. Also, the temperature should be monitored every ten minutes for the next thirty minutes as hyperthermia may recur.[4] Cold water enemas have been used for cooling but have the disadvantage of interfering with temperature monitoring. The dog's extremities should be rubbed during cooling to promote peripheral circulation.

The next main consideration is the prevention of cerebral edema. Glucocorticoids should be administered in an anti-edema dose (Dexamethasone 1.0–2.0 mg/kg body weight). This dose may also be beneficial to treat and prevent shock. Mannitol (2.0 gm/kg body weight as a 20% solution over a 10 minute period) may be used if the patient is stuporous or comatose but should be administered cautiously if DIC is suspected.[4]

IV fluids, such as lactated Ringer's solution, are indicated if hemoconcentration, peripheral circulatory failure, shock, or DIC are present. Fluids must be administered cautiously so as not to induce pulmonary edema or aggravate cerebral edema.[8] There is insufficient data to recommend using calcium gluconate for possible hypocalcemia resulting from hyperventilation and alkalosis.

If hemorrhagic diarrhea, excessive bleeding from venipuncture sites, or hemorrhage elsewhere is present, DIC may have occurred. Coagulation studies, as stated above, help verify the presence of DIC. Therapy for DIC should be initiated with the IV fluids. The administration of heparin (50–150 IU/kg body weight) which has been used in the past, is very controversial and probably doesn't work.

Inhibitors of fibrinolysis, such as epsilonaminocaproic acid (EACA), should never be given to patients with DIC, as fibrinolysis is secondary in this disorder; however, bleeding caused by a primary fibrinolytic state requires EACA to control hemorrhage.[20] A systemic, broad-spectrum, bacteriocidal antibiotic is often administered on the assumption that patients are predisposed to infection. Affected animals should always be maintained in a cool (70°F) oxygen chamber for 24 hours after recovery.[21]

Only a guarded-to-good long-term prognosis can be offered to patients that respond to therapy, because permanent brain damage may have occurred and will not become evident until later.

NECROPSY RESULTS

Mortality due to heat stroke is difficult to assess because the criteria for diagnosis and staging are not uniform.[9] Mortality has been reported to vary from 10–80%. Autopsy findings generally include hemorrhage, congestion, and cell death in all organs of the body.[22]

The following organs, in order of decreasing severity, show histopathologic changes: cerebellum, cerebral cortex, heart, kidney and lung.

In the cerebellum, Purkinje cells are markedly degenerated; they exhibit deformity, hyperchromatic cytoplasm, loss of nuclear definition, and cytolysis,. There are also hydropic changes in deep cerebellar white matter and microhemorrhages are present.

The neurons of the cerebral cortex show deformity, hyperchromatic cytoplasm, loss of nuclear definition and glial infiltration.[23]

The heart has numerous petechial hemorrhages and degenerative changes in the myocardium. The myofibrillar striations are poorly defined, and fraying and separation of muscle fibers are frequently seen.

Kidneys show hemorrhage and various degrees of tubular necrosis.[24] In many specimens, glomeruli are hypercellular and Bowman's capsule is dilated to some degree.

Sections from lungs show great engorgement of vessels with hemorrhage into interstitial tissue and alveoli.[25] Edema is also present most of the time, as evident by frothy hemorrhagic fluid which lungs and bronchial tree yield on section.

PREVENTION

Heat stress can be prevented by providing adequate ventilation, a place where the dog can get out of the sun's direct rays, and free access to cool drinking water. Finally, dogs should not be forced to exercise during times of high environmental temperature.

CLINICAL USE

A 12 year old spayed female toy poodle was presented to a Minneapolis area veterinary hospital at 5:30 p.m. on July 1. The dog had been in a car with all windows shut for about four hours. On physical exam the dog was semi-comatose, panting, extremely hot to the touch, and had a rapid pulse. The dog had cataracts but otherwise was free of problems. She was not on any medication or heartworm preventative. Her temperature was 106°F on presentation. 24 mg of Dexamethasone were given immediately.

The dog was put in an ice bath for 2 minutes until her temperature reached 103°F. Her limbs were massaged to increase peripheral circulation. The temperature fell to 97°F and a PCV was run at this time which read 60. The temperature was taken every 10 minutes and after 30 minutes it started to increase.

At 7:00 p.m., the dog was up and alert and the temperature was 102°F. She was removed from the incubator into which she had been placed after her bath. At 7:20 p.m., she began paddling with her front legs but had no reflexes or pain response in her back legs and no anal tone. The PCV was again taken and read 68. An IV catheter was hooked up and fluids started. The temperature had reasonably stabilized at 101°–101.5°F. Bloody diarrhea began and also petechial hemorrhages were seen on the oral mucous membranes. DIC was suspected but no tests were run. Heparin was given at the dose of 100 IU/kg. At this time, the dog developed tremors and became semi-comatose with no pain response. Intermittent seizures started accompanied by yellow vomitus.

At 9:00 p.m., 24 mg of Dexamethasone were given. The diarrhea was less bloody and more mucoid. At 11:45 p.m., the dog entered severe respiratory distress and all efforts to revive her failed. Necropsy was not performed due to owner request.

CONCLUSIONS

Most stroke is a complicated disease that affects many organs of the body. Environmental temperature, ventilation, and confinement seem to be major contributing factors. Clinical signs seem to be quite diagnostic but develop rapidly, so one must be attentive to the situation. The prognosis depends a lot on the development of secondary complications such as cerebral edema, circulatory failure, DIC, and renal failure. Treatment should be prompt and intensive to ensure the best chance of survival. Prevention is still the best medicine. Be sure to keep animals out of direct sunlight, give them plenty of cool water and don't force exercise in hot weather.

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