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A Study of Heat Stroke and Heat Exhaustion in the Dog

Susan M. Stanley*

Summary

A review of thermoregulation in the dog is presented, which is followed by a study of heat stroke and heat exhaustion. The etiology, clinical signs, secondary complications, treatment, predisposing factors, and prevention of heat stroke and heat exhaustion are discussed. Two clinical cases are then presented to illustrate both disorders.

Introduction

Heat stroke (sunstroke, heat hyperexia, insolation) is the most common heat disorder in the dog.¹ It is caused by forced confinement in a hot environment, such as a parked car with the windows rolled up. When the animal's thermoregulatory mechanisms become ineffective at dissipating the body's extra heat, heat stroke occurs. In man heat stroke is also due to ineffective thermoregulation. It is caused by a cessation of sweating, the main thermoregulatory mechanism of man.² Unlike dogs, heat stroke occurs only rarely in man, where heat exhaustion is the more common disorder.² However, in both species, heat stroke is extremely serious and often rapidly fatal. The main clinical signs of heat stroke are extremely high body temperature and sudden collapse.³

Heat exhaustion (heat prostration) is due to prolonged exposure to high temperatures, which causes failure of the peripheral circulation.¹ Extreme physical exertion of an animal in a hot environment can also cause heat exhaustion. Normally, high environmental temperatures cause peripheral vessels to dilate, allowing more blood to flow to the skin and dissipate heat. The brain and

muscles have a concurrent need for increased blood flow. If the vascular system can not meet these demands, heat exhaustion occurs.²

Physiology of Thermoregulation

Temperature is perceived by thermoreceptors found peripherally and in the central nervous system.⁴ All heat-loss mechanisms in conscious mammals are activated by the local warming of the anterior hypothalamus, which stimulates the thermal receptors found there.⁴ These mechanisms are both physiological and behavioral. There are also thermal receptors in the spinal cord which pass information concerning the spinal temperature to the anterior hypothalamus.⁴

Thermal stability exists when heat gain equals heat loss. The heat load on the body is the total of environmental and metabolic heat.⁵ Heat production by the organs of the body is fairly constant, however heat produced by skeletal muscle is dependent on muscular activity. As much as 80% of the body's heat can be produced by working skeletal muscle.⁴ Radiation, conduction, and convection provide a means of heat loss and heat gain to the body. Body heat is also lost by evaporation of water from the skin and respiratory passages, and by the excretion of feces and urine.

Homeotherms, or warm-blooded animals, have thermoregulatory mechanisms which work to maintain a normal body temperature. The activity of these regulatory mechanisms is very dependent on external temperature. A thermoneutral zone exists where circulatory adjustments alone are capable of maintaining heat balance.⁴ Above and below this zone additional mechanisms are required to maintain a thermal steady state. Sympathetic vasoconstrictor nerves control the cutaneous vasomotor reactions to

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temperature changes.⁴ Inhibition of the sympathetic vasoconstrictor tone causes peripheral vasodilation, and this increases heat loss.⁴ However, when the external temperature is above 31°C, heat is no longer lost by cutaneous vasodilation.⁴ The inhibition of vasoconstrictor tone is mediated by an increase in hypothalamic temperature or reflexly through thermoreceptors in the skin.⁴

Normally the insensible water loss, or that lost by evaporation from the skin and respiratory passages, accounts for 25% of the heat produced by a resting mammal under conditions of average temperature and humidity.⁴ The panting mechanism is an efficient way of increasing the evaporative heat loss. It also is the main mechanism of temperature reduction in the dog.³ The panting mechanism, like cutaneous vasodilation, may also be initiated reflexly and centrally, by an increase in body or external temperature and by local warming of the anterior hypothalamus.⁴ Increased salivation during panting greatly increases the evaporative cooling.⁶ Sweating is relatively unimportant as a means of heat-dissipation in the dog. However, in man as much as 1,000 Cal. of heat can be lost per hour.⁴ A dog's thermal equilibrium begins to deteriorate when its body temperature surpasses 104°F.⁶ The brain becomes involved at 106°F, and sustains permanent damage at 108°F or more.⁶ Cats are able to withstand atmospheric temperatures 5°F higher than dogs.⁶

Clinical Signs and Secondary Complication of Heat Stroke

Initially the dog is panting, hyperthermic, and has a bright red oral mucosa.⁷ Its pulse is rapid and strong.⁷ Gradually the dog's cerebral function decreases due to development of cerebral edema.⁷ The dog may even paddle involuntarily.⁷ Progression of the cerebral edema causes the dog to enter a coma and lose its panting reflex.⁷ The respiratory rate also slows, and the dog may die of respiratory arrest.⁷ The mucous membranes become pale due to peripheral vasoconstriction or decreased circulatory blood volume or a combination of both.⁷ The extremities will feel noticeably warm.⁷ Dyspnea, profuse salivation, vomiting, and of course extreme weakness or collapse are other signs.³

Hemoconcentration can be very severe. Packed cell volumes as high as 75% have been recorded.⁷ Other blood changes are hyperkalemia, thrombocytopenia, and hypothermia.³ It has been found that liver and jejunum contribute to the increase in extracellular K concentration.⁸ However, it is unusual that hyperkalemia develops. The hyperthermic dog is in a state of respiratory alkalosis due to panting blowing off CO₂. In a normothermic dog alkalosis causes a hypokalemia. Hyperkalemia is a special problem because it causes neuromuscular malfunction. The most important malfunction is cardiac arrhythmias leading to heart block.

Disseminated intravascular coagulation (DIC) can also complicate heat stroke. If the dog voids bloody diarrhea or if petechiae are present you should suspect the possibility of DIC.⁷ It has been theorized that DIC is initiated by release of thromboplastin from necrotic cells and by activation of clotting factor XII, due to endothelial cell damage.⁵ Theories on the mechanisms of cell death in heat stroke are: 1) heat inactivation or denaturation of intracellular enzymes or lipids, and 2) hypoxia associated with hypotension.⁵ In fact, the shock-induced ischemia can cause total renal shutdown.⁶ Therefore BUN and urine production should be monitored closely. If treatment is not given, coma and respiratory arrest will result.⁷

Clinical Signs of Heat Exhaustion

The main clinical signs of heat exhaustion are related to the peripheral circulatory collapse. They are weakness, muscle tremors, and cyanosis.¹ There is usually not an elevated body temperature.¹ If treatment is initiated right away, heat exhaustion is usually non-fatal.¹

Treatment

Heat exhaustion should be treated by placing the patient in a cool environment and administering IV fluids and electrolytes.² Treatment usually rapidly improves the patient's condition.

On the other hand, heat stroke must be treated much more vigorously. The mortality rate in heat stroke cases is directly proportional to the height and duration of the fever.¹ A guarded prognosis should be given with temperatures of 106°F or more.¹ Tem-

peratures of 108°F and above are usually fatal.¹ The importance of lowering the dog's body temperature as rapidly as possible can not be overemphasized. It can be most effectively accomplished by placing the dog in a tub of ice water. An alternative method of cooling the dog is to sprinkle it with cold water and then place the dog in the breeze of a fan. The dog's extremities should be massaged during the cooling to treat the peripheral stagnation that is present.⁹ The rectal temperature should be monitored every 10 minutes so that the dog can be removed from the ice water when its temperature has dropped to 103°F.⁷ Be careful not to overcool the dog and cause hypothermia. Temperature monitoring should be continued for the next half-hour or longer, because hyperthermia may recur.⁷

The next main concern when treating heat stroke is the prevention of cerebral edema.⁷ This may be accomplished by administering IV dexamethasone at 1.0-2.0 mg/Kg BW as a 20% solution over a 10 minute period, as long as blood loss or DIC are not complicating factors.⁷

IV fluids of a balanced electrolyte solution, such as Ringer's, are indicated when hemoconcentration, peripheral circulatory failure, DIC, or shock are present. However, they should be administered slowly so as not to cause pulmonary edema or increase the problem of cerebral edema.⁷ If bleeding tendencies are present, assume that DIC is present and treat with IV heparin at a rate of 50-150 IU/kg BW.⁷

Predisposing Factors

The basic predisposing factor to heat stroke and heat exhaustion is a high environmental temperature. High humidity is an additional factor in human medicine due to man's dependence on sweating to dissipate heat. However, humidity is relatively unimportant in dogs because they have less sweat gland development. In both man and the dog approximately 73% of the total body heat is lost by radiation and conduction from the surface of the body to the surrounding air.¹ With poor ventilation this mechanism of heat loss is blocked, because the temperature of the air surrounding the body rises to body temperature. Strenuous exercise is also a predisposing factor because muscular activity greatly increases the total body heat. The

brachycephalic breeds are predisposed to heat stroke and heat exhaustion because their anatomy interferes with panting, thus decreasing the effectiveness of this mechanism of heat loss. Any disease of the heart or lungs compromises the animal's ability to respond to heat.¹ In these cases just moderately high temperatures may cause heat stroke. Another physical condition that increases the chances of heat stroke is obesity. The subcutaneous fat decreases the heat that can be lost by conduction and radiation.¹ The specific dynamic action of food is only a problem when it accompanies other predisposing causes. Proteinic foods have the highest specific dynamic effect, with carbohydrates second, and fats third.¹

Prevention

With the above predisposing factors in mind, a list of ways to prevent heat exhaustion and heat stroke can easily be written. Dogs should be provided fresh drinking water free-choice. There should be some place where dogs can get out of the sun's direct rays. Adequate ventilation is very important. Finally, dogs should not be forced to exercise during times of high environmental temperatures.

A Clinical Case of Heat Stroke

A 7-month-old male English Setter was brought to the ISU Small Animal Clinic in mid-June. It was unconscious and in shock upon presentation, and had a temperature above 110°F. A cold-water bath was used to cool the dog. Fluid, steroid, and oxygen therapy were instituted immediately. The dog never regained consciousness. It died after about 4 hours of intensive therapy.

Necropsy results were as follows. The dog's coat was covered with vomitus. The peritoneal and thoracic cavities were filled with blood-tinged fluid. The epiglottis, larynx, and trachea had streaks of hemorrhage on the mucosal surface. The trachea and bronchi were filled with froth and vomitus. The lungs were moist and congested craniodorsally. There was also petechiae on the lung serosa. Both ventricles of the heart contained blood clots, and the endocardium was hemorrhagic. The entire colonic mucosa contained petechiae. Both the liver and kidney were congested. A note from the pathologist stated that the lesions were not

diagnostic for but were consistent with a clinical diagnosis of heat stroke.¹⁰

A Clinical Case of Heat Exhaustion

A 2½-year-old male Doberman was presented to the ISU Small Animal Clinic on July 1 with a history of collapsing suddenly after vomiting while it was eating. The dog had been playing before it was fed. When it arrived at the Clinic it was ataxic and weak, and had a temperature of 102°F. The pulse rate was 120/minute and the respiratory rate was 40/minute. The dog was started on IV lactated ringers, and both corticosteroid and antibiotic therapy. Recovery was uneventful.

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Veterinary Care of Ferrets, Raccoons and Skunks

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Purpose and Introduction

The continuing tendency of people to desire unique pets and the availability of non-domestic animals such as ferrets, skunks, and raccoons from local pet stores make it important for the practicing veterinarian to have some knowledge of routine daily and specialized veterinary care for these animals. Pets are also made of young skunks and raccoons taken from the wild. It is necessary for the veterinarian to have some background in the housing and nutritional needs of these exotic pets as well as knowledge of recommended vaccinations, specialized veterinary procedures, and problems associated with the ownership of these non-domestic pets. It may

be the veterinarian's duty to help a client make the decision for or against choosing one of these animals as a pet.

More and more frequently, people are choosing non-domestic or exotic animals as pets. A decade ago, it was estimated that 10,000 persons annually take in skunks as pets.¹⁵ The incidence of raccoon ownership is also increasing²⁸ and most recently, ferrets have become popular as unusual pets. The reasons why people choose an exotic animal over a dog or cat vary greatly.² For some, it is merely keeping pace with current trends. Others are attracted to the idea of keeping a wild animal in the home as a type of personal biological laboratory allowing them to monitor the lifestyle and habits of a wild animal at their leisure. The continuing popularity of Walt Disney nature movies and

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