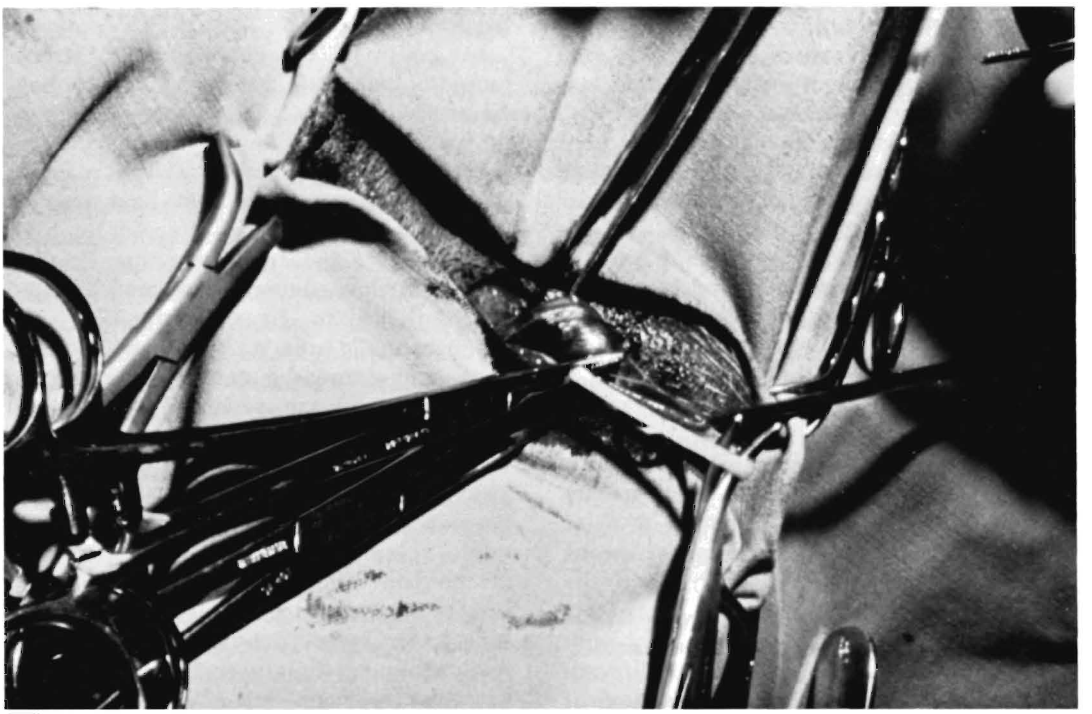
sutured with 4-0 chromic gut and the cord was returned to its original position. The procedure was repeated on the opposite cord and the skin incision was closed with simple interrupted vetafil sutures. A minimal amount of post operative swelling in the scrotal area was noted one day following surgery.



The vas has been isolated prior to ligation and transection.

Effect of Heat on Canines and Felines

by
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Exposure of dogs and cats to high environmental temperatures may have serious and often fatal consequences. This article will briefly discuss the harmful effects of heat, known as heat stress (heat strokes, hyperpyrexia, sun stroke), which is characterized by acute onset, high body temperature, and collapse.

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PHYSIOLOGY OF HEAT LOSS

The first response of a dog or cat to temperature rise is vasodilation of the skin by inhibition of the sympathetic

vasoconstriction tone of blood vessels (5). About 73% of all body heat is lost via skin radiation and circulation (2). Vasoconstriction tone decrease is mediated by either the hypothalamic temperature control centers or reflexly by thermoreceptors in the skin (2). Above an atmospheric temperature of 86°F, skin vasodilation no longer results in heat loss and other mechanisms are used (2).

The second major mechanism for heat loss is hyperventilation (panting). Studies have shown that cooling occurs in the upper respiratory passage and pharynx and not the lungs (2). This factor should be considered when placing an endotracheal tube in a heat stressed animal because by doing so a major source of heat loss is eliminated.

ETIOLOGY

Heat stress results largely from confinement in an overheated enclosure for a period of time (5). Many factors are involved in stressing an animal to a point where heat stress occurs, the most obvious being high atmospheric heat. When the environmental temperature increases above 86°F a rise in internal body temperature results. Depending upon additional environmental and physiological factors, dogs can tolerate rising environmental temperatures quite well. Cats can tolerate atmospheric temperatures 5°F. higher than dogs. However, when a combination of factors results in an increase in body temperature greater than 104°F., breakdown of the animal's thermal equilibrium begins. At 106°F. the brain becomes involved and permanent damage may develop. At body temperatures of 108-109°F. severe and massive central nervous system and cardiac changes are definitely occurring (1,2,5).

In the dog, inadequate ventilation is one of the most critical factors in heat stress development (6). As previously mentioned, greater than 70% of the total body heat loss in dogs and cats is due to radiation and conduction from the body surface. Static air around the body is quickly elevated to body temperature, thus surface heat loss is blocked. Dogs kept outside in shade with a breeze can easily withstand ambient temperatures of over 100°F (1). However, when placed in an enclosed environment with no air flow, a serious problem can develop. The two most common situations which lead to heat stress in dogs or cats involve either a closed automobile or (unfortunately for the veterinary profession) confinement in an animal hospital cage.

In contrast to human heat stress, humidity has little effect on canine heat stress, primarily due to the poor development of canine sweat glands. However, evaporation of sweat plays a relatively important role in heat loss in felines, so cats will be affected by high humidity to a greater extent than dogs.

Certain dogs demonstrate an increased susceptibility to heat, particularly the brachycephalic breeds (2,5). Many of these dogs cannot breathe easily under normal conditions and are severely handicapped when stressed by heat. The extra effort to breathe often irritates pharyngeal and laryngeal tissue causing the tissue to become edematous, thus decreasing the tidal volume and respiratory cooling mechanism. The forced effort to increase respiratory volume also causes an increased metabolic heat production from the respiratory musculature. The long-haired breeds of dogs are obviously more susceptible to heat due to the insulation effect of the hair coat.

The specific dynamic action of food (also known as the calorigenic action of food) is many times an unrecognized factor contributing to heat stress. Specific dynamic action heat is the heat produced when food is metabolized. For example, 100 calories of fat or carbohydrates will produce only 104-106 calories of heat, while 100 calories of protein produce over 130 calories of heat! Alone, the specific dynamic action of food is rarely significant in causing heat stress but becomes more significant when combined with the other factors.

Excessive exercise also contributes to body heat. Even mild excercise such as walking can increase the body's heat production by 200% The availability of water and hydration status of the animal are also important considerations.

Concurrent diseases, especially of the cardio-vascular and respiratory systems, will greatly compromise an animal's ability to respond to heat. Dogs with valvular

insufficiency, congestive heart failure or chronic pulmonary insufficiency are particularly susceptible (1).

Other factors include obesity and the general state of excitement of nervous animals.

The normal response of the body to high atmospheric temperature, dilation of the peripheral vessels to dissipate excess body heat, is the starting point of the pathogenesis of heat stress. As blood is shunted to the peripheral capillaries, there is a large decrease in the circulatory blood volume. Unless there is a compensatory increase in the blood volume and constriction of splanchnic vessels, the venous return to the heart becomes less and less. There is decreased blood volume available for oxygenation of tissue and transport of the excessive body heat. Although the heart is working harder to pump blood, the pulse weakens and the blood trappped in peripheral vessels becomes more or less stagnant. Tissue edema and hpoxia develop which are most serious in such areas as the lungs and brains.

Pulmonary congestion and edema help to propagate the problem. There is decreased oxygenation of blood and decreased heat loss via pulmonary air exchange. Panting increases to compensate and, as previously mentioned, this adds to the problem by increasing the heat produced by the respiratory musculature.

Cerebral damage is the most serious consequence of heat stress. Body cells operate within certain thermal limits. Above these limits cellular degeneration begins as essential body proteins are coagulated. The brain is especially susceptible to cellular damage because once neural cells are destroyed they are replaced not by viable neural tissue but by scar tissue. Oxygen consumption of the brain increases with temperature (1), but the availability of cerebral oxygen decreases with cerebral edema and decreased cerebral blood flow.

The very heat which causes heat stress will often cause increased heat production by the body as well. Basal metabolism increases 6% for each degree F. rise because of the intrinsic effect of heat to increase the rate of chemical reaction.

Hyperventilation due to panting may cause respiratory alkalosis as carbon dioxide is blown off. Because cats are not as adapted as dogs to compensatory ventilation changes, they are more likely to develope this alkalosis. In response to the decrease in carbon dioxide, the kidneys decrease the reabsorption of bicarbonate, thus reducing the total bicarbonate buffer capacity and thereby predisposing the cat to development of a secondary metabolic acidosis (1).

Other organs can also be affected by shock induced ischemia. Total renal shut down can occur leading to serious consequences later in the disease course.

Hyperkalemia is also associated with hyperthermic animals, despite the developing alkalosis which under normothermic conditions is associated with hypokalemia. Liver and gastrointestinal tissue contribute to the extracellular rise in K, but there are also unknown causes for the K rise (7). Hyperkalemia causes neuromuscular malfunction which eventually leads to muscular and respiratory paralysis. Of special concern are arrhythmias progressing from premature contraction and bradycardia to ventricular fibrillation and heart block.

CLINICAL SIGNS and DIAGNOSIS

When a dog or cat is exposed to high temperature for only a short amount of time transient hyperventilation (panting) is the only noticeable clinical sign. The animal is still alert and the hyperventilation decreases as soon as the body temperature returns to normal. However, exposure of the animal to high temperature for extended periods of time usually results in fairly characteristic clinical signs of heat stress. Heat stress is usually an acute syndrome with the clinical signs developing fairly rapidly. Owners usually notice first the hyperventilation with rapid, shallow respirations. The animal may appear to have a dull, blank, staring expression and often will not respond to visual stimuli or vocal commands in a normal manner. The animal may be weak and ataxic, but collapse and coma don't usually occur until terminally. As the condition progresses, the animal may become hysterical.

Vomiting is common and usually persists

even after the temperature is reduced. The animal's stools may be loose and terminally become bloody.

If the animal is presented immediately (within minutes) to your animal hospital, the above mentioned clinical signs are present or develop shortly thereafter. Also, there are a number of signs which might not have been observed by the owner.

The rectal temperature is usually above 106°F, and may so high that it doesn't register on the thermometer. The heart beat is extremely fast and sounds strong, but the pulse is very weak. The animal doesn't appear to be dehydrated (unless it was prior to the heat stress) and superhydration may be evident. The skin feels warm but dry.

The mucous membranes are first hyperemic (bright red), then rapidly become cyanotic as anoxia progresses. Dyspnea develops with pulmonary edema and congestion.

As the animal lapses into a comatose state, the heart and respiratory rates become weak and irregular. Such animals almost invariably die.

If the animal is not brought to the hospital until several hours after the exposure occurred, the temperature may be subnormal (5). Immediate appraisal of the animal is difficult at this time and clinical signs attributed to hypoxia and edema in essential organs (such as the kidneys) may not develop until hours or even days later (5).

To aid in diagnosis, a complete history should be taken with particular attention paid to any previous attacks as this syndrome is apt to recur for life after the initial attack.

TREATMENT

The rapid progression of the heat stress syndrome dictates that its treatment be both prompt and intensive. The primary consideration in the treatment of heat stress is the rapid reduction of body temperature. The fastest and easiest method to accomplish temperature reduction is immersion of the total body in very cold water (ice if possible). Special attention should be directed to cooling of the head as quickly as possible. A water hose shower or equivalent method should be used if bathing facilities are not available. The body temperature should be constantly monitored during cooling with a reduction in temperature below 103°F as the goal. If the temperature is not reduced to less than 103°F within 10 to 15 minutes, cold water enemas should be used. However, once an enema is given, the temperature cannot be monitored accurately by use of a rectal thermometer so the enema should not be used immediately in treatment (2,5). Once the temperature is decreased below 103°F it should continue to be monitored as the temperature may rise again later. On rare occasions the hyperthermic animal will become hypothermic and care should be taken to prevent hypothermia.

Vigorous massaging and flexing of the legs aids in increasing blood return to the central circulation.

Circulation of air around the animal with a fan aids in cooling. A patent airway should be maintained, but oxygen therapy is contraindicated because carbon dioxide loss apnea may develop.

Fluid therapy is controversial in the treatment of heat stress. Jackson in *Kirk V* says, "the need to reduce the PCV and start organ perfusion is so great that pulmonary edema should be risked. If the cardiovascular system permits the solutions are given in large quantities, (up to 50ml/# the first four hours)" (5). Armistead in *Canine Medicine* states, "fluids should not be administered parenterally unless definitely indicated because of the danger of pulmonary edema" (2).

After the animal begins to recover, it should be placed in an air-conditioned room and watched for 24 hours, as collapse and recurrence may suddenly occur (4).

Because of the latent effect on the kidneys, BUN, creatinine and urine production should be monitored daily until normal values are reached (5).

Mortality of untreated heat stress is high (5). With treatment, mortality is directly proportional to the height and length of fever (2). If the temperature is above 106°F, a grave prognosis is given because of the possibility of permanent brain damage. If the temperature reaches 108-109°F for even a brief period of time, the

animal will usually die. Armistead states that a 20% mortality rate is expected with prompt treatment (2). If the animal is exposed 1-2 hours, a 50% mortality rate should be expected (6).

If the PCV can be reduced to normal within four hours, the prognosis is good (5). However, if the PCV remains elevated, or if the frequent passage of loose, bloody stools persists, the outcome is fatal (5). Persistence of shock or coma after a return to normal temperature usually reflects irreversible brain damage (2).

Even if the patient recovers from heat stress, the owner should be cautioned that permanent brain damage may have occured and may not become evident until later (2).

PREVENTION

Heat stress can be prevented by: providing adequate ventilation, protecting the animal from direct rays of sun and giving free access to cool drinking water (with 0.2% salt added in very hot weather). Animals should be kept quiet and strenuous excercise avoided. Nervous animals may be tranquilized or kept away from excitable influences (such as active children or other animals) (2).

When an animal is taken from a cold climate to a hot climate, they are more susceptible to heat until acclimatization occurs. This involves an increase in body protein and blood volume, thus decreasing the effect of peripheral vascular dilation during heat exposure (2).

Feeding should be done during the early morning or evening to avoid the problem of specific dynamic action heat production combined with hot temperatures.

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