Management of the Heat Stroke patient

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Introduction
Heat stroke is defined as a severe illness characterised by core temperatures greater than 41°C due to external factors, central nervous system dysfunction and a systemic inflammatory response syndrome that can lead to disseminated intravascular coagulation (DIC). Immediate medical management of a heat stroke patient is vital for the patient’s survivability. Therefore, it is important to understand the pathogenesis and pathophysiology of heat stroke to medically manage such a patient.

Pathogenesis
The ultimate goal of any living organism is to maintain homeostasis by ensuring all systems remain balanced. If one system becomes unbalanced then all other body systems will be affected. Thermal homeostasis is maintained by the balance between heat gaining and heat dissipating mechanisms, controlled by temperature sensitive centres in the hypothalamus. Heat gain occurs through oxidative metabolism of food, exercise and increased muscle or metabolic activity, and elevated environmental temperature. Heat dissipating mechanisms include behavioural changes (seeking a cooler location), peripheral vasodilation and changes in circulation, evaporative cooling primarily in the form of respiratory heat exchange, radiation and convection. This mechanism is called negative feedback (figure 1), because an effector (increased cutaneous circulation, increased cardiac output, vasodilation, panting) activated by the control centre (hypothalamus) either opposed or eliminated the stimulus (normal temperature restored). In many cases the initiation of an effector
will require a trade off with other systems. For example, the shunting of blood to the periphery is a trade off with blood supply to the viscera (intestines and kidneys). When homeostatic regulation fails as in the case with heat stroke, other body systems become compromised.

Heat stroke is usually associated with a warm humid environment (nonexertional heatstroke) or excessive exercise (exertional heatstroke) as they may cause extreme hyperthermia even in animals with functional heat dissipating mechanisms. In companion animals the main type of heat dissipation is through cutaneous vascular dilation and respiratory evaporation. Animals’ respiratory evaporative heat loss may be diminished by humid climatic conditions, closed confinement with poor ventilation (dogs in cars), and upper respiratory abnormalities such as brachycephalic confirmation, laryngeal paralysis or masses, or collapsing trachea. In the case of dogs locked in cars, the dog is unable to affectively use heat dissipation as the air surrounding the animal is saturated with water, therefore water molecules will not vaporise and cool the body. Diminished radiation and convective heat loss from the skin may occur as a result of hypovolemia due to any cause, poor cardiac output, obesity, or lack of acclimatisation to heat. The patient is unable to dissipate heat therefore an elevated core body temperature is the result. This will cause tachycardia, increases in cardiac output, and increase minute ventilation.

There is a sequence of events in the progression of heat stroke at a cellular level (figure 1.2). Nearly all cells respond to sudden heating by producing heat-shock proteins or stress proteins. Heat stress induces thermoregulation. The normal cardiovascular adaptation to severe heat stress is an increase in cardiac output and a shift of heated blood from the core circulation to the peripheral circulation. An inability to increase cardiac output because of salt and water depletion, cardiovascular disease, or a medication that interferes with cardiac function can impair heat tolerance and result in increased susceptibility to heat stroke. The local and systemic insults associated with heat stress, such as visceral hypoperfusion, alter the immunologic and barrier function of the intestines. This alternation allows leakage of endotoxins and increased production of inflammatory (pyrogenic) cytokines. Therefore it is thought that the gastrointestinal tract fuels the inflammatory response.

Pathophysiology
Heat stroke and its progression to multiorgan-dysfunction syndrome (MODS) is due to a complex interplay among the acute physiological alterations associated with hyperthermia (e.g. circulatory failure, hypoxia, and increased metabolic demand), the direct cytotoxicity of heat, and the inflammatory and coagulation responses of the host. This leads to direct cellular and tissue injury, destruction and denaturation of enzymes, and alternations in blood flow in the microcirculation. At extreme temperatures (49°C to 50°C) all cellular structures are destroyed and cellular necrosis occurs in less than five minutes. Common systems affected include the CNS, gastrointestinal, cardiovascular, hepatobiliary, renal/urelogic, hematologic and muscular.

The plasma levels of inflammatory cytokines and anti-inflammatory cytokines are elevated in patients with heat stroke; cooling the body to a normal temperature does not result in the suppression of these factors. Therefore, patients who have experienced a heat stroke episode may not show side-effects of the cytokines straight away. The imbalance between the two types of cytokines may result in either inflammation-associated injury or refractory immunosuppression. This may explain why many heat stroke patients have a high incidence of infection. The increase in the levels of the inflammatory cytokines is associated with high intracranial pressure therefore cerebral oedema, decreased cerebral blood flow and neuronal injury. This may result in altered levels of consciousness or seizures (especially in the cooling down phase). In 20% of cases, patients with damage to the thermoregulatory centre are predisposed to subsequent hyperthermic episodes.

The pulmonary system develops increased vascular resistance and cellular injury, leading to damage of the pulmonary endothelium. This causes noncardiogenic pulmonary oedema and acute respiratory distress syndrome. The damage to the pulmonary endothelium, compounded with the decreased cardiac output and perfusion, creates a decrease in oxygen uptake by the tissues. Patients with non-exertional heat stroke usually have respiratory alkalosis.

All patients have tachycardia and hyperventilation and 25% have hypotension. Many patients are dehydrated and hypovolemic therefore compounding the thermoregulatory dysfunction by reduced volume. Patients are usually hypophosphatemic and hypokalemic due to hypovolemia, thus further compounding the cardiac function. Hypercalcemia and hyperproteinemia, reflecting hemoconcentration may also occur. Endothelial-cell injury and diffuse microvascular thrombosis are prominent features of heat stroke. Therefore, disseminated intravascular coagulation and alterations in the vascular endothelium may be important pathologic mechanisms in heat stroke.
The most profound, and often the most life-threatening, effects of heatstroke and thermal injury are to the renal system. Acute renal failure is common, especially in the dehydrated animal. Rhabdomyolysis, due to muscle necrosis, is common and can exacerbate the acute tubular necrosis via dehydration, hypoperfusion, and pigment desposition.

**Clinical Presentation**

The most common clinical signs reported by owners are excessive panting and dark coloured (brick red) mucous membranes. Other signs may include vomiting, ataxia, ptysialism (hypersalivation), diarrhoea, muscle tremors, with an episode of collapse or extreme lethargy. Clinical signs may not become apparent for several days after the incited event. A thorough history is required to reveal any predisposing cause in the animal’s inability to dissipate heat. Such causes include an animal recently moved to a warmer climate, locked in a car, cardiac medication, excessive exercise, laryngeal paralysis, brachycephalic or a previous episode of heat stroke.

On physical examination the patient will have an elevated rectal temperature above 40.7°C, hyperaemic mucous membranes, fast (due to vasodilation) or slow (cardiovascular failure) capillary refill time, increased respiratory effort, altered mentation, tachycardia with weak or irregular femoral
pulse and possibly cutaneous or mucosa petechiae indicating disseminated intravascular coagulation (DIC). A presumptive diagnosis of nonpyrogenic hyperthermia can be made with an animal with a body temperature greater than or equal to 40.7°C with no obvious evidence of infection⁷.

The patients airway should be assessed immediately and oxygen supplementation should be initiated using an oxygen mask. Many patients who present with heat stroke develop severe upper airway obstructions due to laryngeal oedema. Oxygen supplementation improves tissue perfusion and decreases the risk of ischemia; therefore, it is beneficial even if the patient shows no signs of requiring oxygen.⁹

Clinical Pathology
It is important to obtain base level haematology and urine analysis to ascertain where the imbalances lie. Remember that these patients are predisposed to coagulation problems and increased intracranial pressure, therefore do not take your blood sample from the jugular vein. A jugular puncture could bleed profusely, placing pressure on the trachea causing a tracheal obstruction. When occluding the jugular vein you inadvertently increase intracranial pressure, therefore placing more pressure on the cerebrum.

A complete blood count (CBC), serum biochemistry panel, urinalysis, serial blood gas determinations and serial coagulation profiles should be performed in all animals⁷. Common findings are elevations in packed cell volume (PCV) and total protein due to severe dehydration, elevation in blood urea nitrogen (BUN) and creatinine (Cr) as seen in acute tubular necrosis⁷ and usually peaks within 24 to 48 hours¹. Serum alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphate and bilirubin may be elevated due to hepatocellular damage⁸. Rhabdomyolysis may cause marked increases in creatinine. Blood glucose is often extremely low, although this is an inconsistent finding⁷.

Evidence of DIC is often present (thrombocytopenia, increased fibrin degradation products [FDP’s], prolonged prothrombin time [PT], and activated partial thromboplastin time [APTT])⁷. If PT, APTT and platelet count cannot be performed, then an activated clotting time (ACT) should be performed and a blood smear may detect schistocytes (red blood cell fragments) (figure 3) and give an estimation of platelet count¹. In general, there should be at least 3–5 platelets per oil immersion field on a well performed blood smear⁹.

In patients with DIC the platelet count is reduced.

Blood gas analysis is often variable and will depend on the stage of heatstroke. As heat stress progresses respiratory effort becomes more pronounced and a respiratory alkalosis develops⁷. Patients can also have metabolic acidosis or a mixed acid base disturbance⁸.

Urinalysis should be performed preferably prior to fluid therapy to assess renal function or damage¹. Specific gravity (USG) should be measured and a urine dipstick performed. The USG is usually high due to the patients hydration and perfusion status and the dipstick is often positive for protein as well as haemoglobin. On urine sediment stain the presence of renal casts may indicate significant renal tubular damage⁷.

Treatment
Once the patient has been diagnosed as non-pyrogenic hyperthermia treatment should be commenced immediately. The goal of heatstroke treatment is to cool the patient down to a normothermic body temperature, provide cardiovascular support and to prevent further organ damage.

Cooling
The first treatment should commence with the initial phone call from the owner. It is imperative that the owner is informed to initiate treatment by cooling that patient down with cool water (the use of ice water is contraindicated) before transporting the patient to the veterinary hospital. One study reported that the mortality rate of dogs cooled by their owners was 19% as compared to a rate of 46% for those not cooled by their owners⁸. Advise the owner to drive with the windows down or turn the air conditioner on to enhance radiant and convection cooling. On arrival, the most important aspect of treatment is to lower core temperatures⁷.

It is important not to overcool the patient as this could initiate the bodies normal response to hypothermia, shivering. This must be avoided as it will continue to increase the core body temperature by metabolic heat from muscle contractions due to shivering.

Once the patient is admitted to hospital the cooling process should continue whilst an intravenous catheter is placed

Figure 3: Arrows pointing to schistocytes. Courtesy of Dr. Eloise Jillings, Massey University
and baseline bloods are taken. You will need enough blood for a CBC, biochemistry, PCV/TP and coagulation profile (or ACT if this is not available). The cooling process should continue with the temperature measured every five minutes if you do not have a constant temperature monitor which has an anal probe, to avoid rebound hypothermia. To assist with cooling down the patient, ice packs can be placed over large superficial vessels in the inguinal and axillary areas. Some literature suggests administering cool intravenous fluids and cold water enemas to assist with the cooling process, though enemas interfere with monitoring the patients temperature. It is important to cool the patient to 39.4°C within 30 to 60 minutes of initial presentation, though overcooling must be avoided. Because external cooling results in cutaneous vasoconstriction, vigorous massaging of the skin is recommended.

The use of antipyretic drugs such as aspirin and carprofen is contraindicated, as these drugs will alter the hypothalamic thermoregulation set point and heat stroke patients already have a normal hypothalamic set point.

**Fluid Therapy / Cardiovascular**

Many patients present in hypovolemic shock, therefore it is important to begin aggressive cardiovascular support through volume expansion, which will decrease blood viscosity and improve blood flow. If cardiovascular disease is unlikely, then administration of crystalloid fluids intravenously is the initial fluid of choice, which can be given in doses up to 90ml/kg/hr for the first 1 to 2 hours. Continuously assess perfusion status and titrate the rate and volume of fluids to effect. If several boluses of crystalloid intravenous fluids does not improve tissue perfusion and blood pressure, then administration of colloids should be considered and/or use of positive inotropes or pressure agents (such as dobutamine, dopamine, or adrenaline). Synthetic colloids should be considered if total proteins are below 35 g/l. Judicious use of IV fluids is warranted with care given to avoid fluid overload. Fluid needs of the patient presented with heat stroke are individual and needs to be balanced with treatment response, central venous pressure (CVP), electrolyte balance, acid-base imbalances, blood pressure monitoring, lung resuscitation and urine output rather than a strict fluid volume end-point goal.

Sodium bicarbonate may be administered if the patient's acid base disturbance can not be corrected by the initial fluid therapy.

**Central Nervous System**

During the initial clinical examination the patient should have a full neurological examination to establish a baseline for future reference. The patients level of consciousness (LOC) can deteriorate rapidly and some patients may present in an already stuporous or comatose state. Any electrolyte, body water and glucose abnormalities should be corrected to reduce the possibility of affecting the central nervous system. Hypoglycaemia is not unusual in the severely compromised dog with heat stress, due to the increased metabolic activity which consumes vast amounts of glucose as well as the livers inability to produce enough glucose. Therefore, it is possible for the patient to go into a hypoglycaemic coma. An intravenous bolus of 0.25 to 0.5g per kilogram of body weight of 25% dextrose should be administered as a bolus. Dextrose should be added to the intravenous fluids to a 2.5% to 5% concentration if hypoglycaemia is present. If the patient continues to show abnormal signs of mentation after the correction of the hypoglycaemia then cerebral oedema may be present. Mannitol (0.5 grams/kg body weight slow IV, ensuring a syringe filter is used) should be considered. Some literature suggests that anti-inflammatory doses of dexamethasone should be considered, though care must be taken to reduce the possibility of further immune suppression, gastric ulceration and ischemic injury to the kidneys.

**Renal**

Because of the risk of acute renal failure a urinary catheter should be placed at presentation for monitoring of urine output in the more severely affected dog. A closed collection urinary system will be required to monitor the urine output. The patients urine output should be maintained to at least 2ml to 4ml/kg body weight per hour depending upon the amount of fluid being administered.

**Gastrointestinal**

Direct thermal damage and poor visceral perfusion may result in gastrointestinal mucosal sloughing and ulceration. This may lead to vomiting and diarrhoea as well as allowing leakage of endotoxins thus absorption, and bacterial translocation. This can lead to systemic inflammatory response syndrome (SIRS) and sepsis. Broad-spectrum antibiotics such as the second generation Cephalosporin Cefoxitin, Ampicillin with enrofloxacin, and sometimes metronidazole should be administered to decrease bacteraemia. Non-nephrotoxic antibiotics should be administered, as renal compromise is a serious concern in patients with heat stroke.

Sucralfate and H2 blockers such as ranitidine and cimetidine will help treat gastric ulceration, where as an antiemetic such as metoclopramide can be added to the fluids at a rate of 1 to 2 mg/kg/day.

**Coagulation**

The onset of heat stroke coincides with the activation of...
coagulation and endothelial-cell injury. The endothelium controls vascular tone and permeability, regulates leukocyte movement, and maintains a balance between procoagulant and anticoagulation substances. Hyperthermia enhances vascular permeability, and increases the ability of cells to adhere to the vascular walls causing alterations in blood flow. The activation of the coagulation cascade is exaggerated and platelets are consumed in large quantities, leading to thrombocytopenia.

Therapy for DIC is controversial. The removal of the underlying cause is essential therefore it is import to cool the patient, give cardiovascular support through fluid therapy and reduce any further complication. Symptomatic therapy may include fresh frozen plasma intravenously and low dose heparin therapy (100IU/kg every 4-6 hours) subcutaneously. Alternatively, 20 IU/kg body weight of heparin incubated with fresh frozen plasma may be administered.

Role of the Veterinary Nurse

The veterinary nurse plays a vital role in the heat stroke patient’s initial treatment, ongoing care and monitoring. Heat stroke patients must be monitored closely as they can deteriorate rapidly and become compromised by multiple organ dysfunction and failure. Monitoring of the heat stroke patient must continue for several days after the initial episode as the patient may not show clinical signs of deterioration for several days.

Thermoregulatory

Whilst the patient’s receiving cooling therapy the veterinary nurse must be diligent with constant body core temperature monitoring. The temperature must be monitored at least every 5 minutes until the patient’s temperature reaches 39.4°C, ensuring the patient does not have a hypothermic incident. As the patient’s thermoregulatory centre in the hypothalamus may be altered, the patient may have difficulties with maintaining a constant normothermic body temperature. Once the temperature goal has been met the monitoring should not discontinue. The patient will need to be monitored every 5 minutes until they have a constant normothermic temperature, this could take up to a further 25 to 30 minutes.

In the cooling process the act of placing cool water on the patient will lower the skin temperature; this may trigger cutaneous vasoconstriction and shivering. To overcome this the patient may be vigorously massaged, sprayed with tepid water (40°C), or exposed to hot moving air (45°C), either at the same time as cooling methods are applied or in an alternating fashion.

Cardiovascular

Heat stroke patients have increased cardiac output, hypovolemia, electrolyte and acid/base imbalances this can lead to cardiac arrhythmias, ischemia and multiple infarctions due to DIC. Therefore, the patient’s physical parameters should be monitored continuously and at set intervals. The patient should have a continuous ECG and blood pressure monitoring, central venous pressure (if available), mucous membrane colour, capillary refill time, pulse rate, rhythm and quality and mentation status. Aggressive fluid therapy should improve the patient’s cardiovascular parameters; if this is not the case then sympathomimetics such as dopamine and dobutamine should be considered.

Haematology and biochemistry should be continued throughout treatment to ascertain the success of treatment.

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Dose Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antiarrhythmic</strong></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>CRI: 2mg/kg bolus then 40-80 ug/kg/min</td>
</tr>
<tr>
<td><strong>Antibiotics</strong></td>
<td></td>
</tr>
<tr>
<td>Ampicillin</td>
<td>22mg/kg IV tid</td>
</tr>
<tr>
<td>Cefazolin</td>
<td>22mg/kg IV tid</td>
</tr>
<tr>
<td>Enrofloxacin</td>
<td>5-20 mg/kg IV,IM, or PO q 24hr or divided and given bid</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>15mg/kg IV bid</td>
</tr>
<tr>
<td><strong>Diuretics</strong></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>For acute renal failure: 2mg/kg IV; if no response in 1 hr, then give 4mg/kg IV. If no response in the next hr, then give 6mg/kg IV. For general purposes: 2.2-4.4mg/kg IV tid-qid</td>
</tr>
<tr>
<td>Mannitol</td>
<td>0.25 – 0.5g/kg IV over 20 minutes</td>
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<tr>
<td><strong>GI Protectants</strong></td>
<td></td>
</tr>
<tr>
<td>Ranitidine</td>
<td>0.5-2mg/kg PO, IV, or IM bid-tid</td>
</tr>
<tr>
<td>Surcralate</td>
<td>Small dogs: 0.5 g PO tid; Large dogs: 1g PO tid</td>
</tr>
<tr>
<td><strong>Hypotensive Medications</strong></td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>CRI: 5-15ug/kg/min IV</td>
</tr>
<tr>
<td>Dopamine</td>
<td>CRI: 1-3 ug/kg/min IV</td>
</tr>
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*Table 1.1 Medical Management of Heat stroke (adapted from Tabor, 2007)*
Respiratory
Oxygen supplementation should continue until it has been determined that the dog can maintain arterial oxygenation.

If the oxygen therapy is delivered through a nasal oxygen line, ensure the oxygen is humidified, as non-humidified oxygen can deplete the body water further through evaporation via the nasal cavity. The delivery of oxygen in an oxygen cage or humidicrib/incubator is contraindicated as the increased temperature and water vapour in the surrounding area affects heat dissipation.

Oxygen saturation levels should be monitored. The SpO₂ to be above 95% if patient is not on oxygen support or around 98% on supplement oxygen. Blood gas analysis baselines and continual monitoring is important as many heat stroke patients will be tachypnoeic and may develop respiratory acidosis.

Other parameters to be assessed include auscultation of the chest, respiratory rate and effort, and mucus membrane colour is warranted in the heat stroke patient.

Central Nervous System
The patients LOC could change rapidly due to electrolyte imbalances, hypoglycaemia, increased intracranial pressure and/or oedema and in final stages cerebral micro-emboli and hepatic encephalopathy. Constant monitoring of the patients LOC, papillary size and light response, breathing pattern, posture and correction of imbalances should be instituted. The patients gag reflex should be assessed intermittently to ascertain if this is lost at any stage.

The patients head should be slightly elevated to avoid occluding the jugular veins as occlusion increases intracranial pressure.

Renal
As previously mentioned heat stroke patients are at risk of acute renal failure and they should have a urinary catheter placed to measure urine output (UO). The most accurate way to measure UO is a closed collection system attached to the urinary catheter. The normal urinary output for a healthy patient is 1-2ml/kg/hr, though heat stroke patients will be receiving aggressive fluid therapy therefore the inputs and outputs should equal. Generally we want the patient to be producing around 2-4ml/kg/hr. If the patients “ins” are exceeding the “outs” or the “outs” exceed the “ins” the fluid therapy must be revised. In both instances the patient may be at risk of fluid overload or dehydration.

The patient should be weighed daily as this can assess fluid balance. If a patient has a dramatic change in body weight, either an increase or decrease this may indicate fluid losses or gains.

The urine specific gravity (USG) should be measured several times a day as this can indicate if the patient has fluid overload or not receiving the required fluid therapy to maintain body water. A high USG is indicative of dehydration where a low USG is indicative of fluid overload. A urine sediment and analysis should be performed daily as renal casts are indicative of renal injury.

Haematology and biochemistry can assist with assessing fluid therapy as well as renal function. A PCV/TPP can assist with fluid therapy as a dehydrated patient will be haemoconcentrated therefore the PCV and TPP will be high. Creatinine, BUN and electrolytes will assess fluid therapy as well as renal function.

On physical examination the chest should be auscultated to rule out pulmonary oedema. All extremities should be examined for pitting oedema as this indicates fluid overload.

A patient who becomes oliguric must be reassessed immediately. The closed collection system should be checked to ensure that the clamp on the giving set has not been left on or that the urinary catheter is kinked, therefore occluding flow.

The urinary catheter must be cared for daily ensuring the procedure is performed as aseptically as possible as heat stroke patients are usually immune compromised.

Gastrointestinal
The heat stroke patient may be exhibiting signs of thermal damage to the mucosa via vomiting and diarrhoea. The patient must be kept clean to reduce fecal scalding and ensure that hygiene is kept to a high standard. The veterinary nurse could place a tail bandage to reduce soiling of the coat.

The patient will be treated medically with anti-emetics to reduce vomiting as continual vomiting will affect fluid balance and the process of vomiting further increases intracranial pressure.

Total parenteral or enteral nutrition may be instituted to fulfil the patient’s nutritional requirements. Remember to use the resting energy requirement calculation rather than the metabolic energy requirement as the patient does not require a high caloric intake.

The thermal damage of the GI tract will predispose the patient to translocation of bacteria that can develop into SIRS or septic shock. Therefore the patient will be receiving systemic antibiotic therapy. A patient that is in septic shock may exhibit the following signs, hypotension, tachycardia,
hypothermia or fever, low or high white blood cell count and signs of multiple organ involvement. If the patient starts to exhibit any of the above mentioned clinical signs the veterinarian must be notified immediately.

Coagulation

Coagulation problems occur in the heat stroke patient due to the activation of the coagulation cascade due to widespread endothelial damage and/or hypoalbuminemia due to protein loss in the gastro-intestinal tract. Excessive activation of the cascade and excessive consumption of clotting factors leads to DIC. Patients must be observed for signs consistent with DIC such as hypercoagulability, bleeding from venipuncture sites, petechiae and ecchymosis, which is usually found on the mucus membranes.

If a patient was diagnosed with DIC 5 to 10 years ago they were given a poor prognosis but today more is known about the pathophysiology of DIC therefore mortality rates are declining. Therapy for DIC may include administration of blood products and heparin.

Monitoring of platelets, activated clotting time and red blood cell lysis can be performed in the normal clinical setting. A complete coagulation profile must be sent off to a veterinary pathology laboratory for analysis.

Note: As BD no longer produce ACT tubes I have found another source in New Zealand called Celite ACT tubes (black flip-top) from Biolab in Auckland. C-ACT tubes contain a celite (diatomaceous earth) activator. The supplier suggests that you perform testing by adding 2.0ml of whole blood to the warmed tube (body temperature), mix, and insert tube into the Actalyke. Although I have found that you do not need the Actalyke to perform the test, just use it as you would the BD ACT tubes.

Conclusion

Heat stroke is a life-threatening medical emergency and the patient’s prognosis is highly dependent on immediate medical intervention. The veterinary nurse plays a vital role in the initial treatment of the patient with the first telephone call from the owner to the continuing medical treatment, monitoring and client education. All veterinary personnel should be familiar with the clinical signs and the patients who are predisposed to heat stroke as well as the environmental conditions that may influence the increased probability of heat stroke.

A complete patient history, physical examination and base line comprehensive blood work will help determine the extent of systemic damage, treatment and prognosis. It is important to remember that a patient may not show clinical signs of heat stroke until several days after the insult, therefore they should be closely monitored for an extended period of time. The patient may only show mild clinical signs such as tachycardia, tachypnoea and panting or severe clinical signs associated with multi-organ dysfunction syndrome.

Understanding the patho-physiology of heat stroke will assist the veterinary nurse with understanding the complications that may arise, therefore alerting the veterinarian to any changes in the patient’s status.

References

1. Drobatz, K.J. (2004), Heatstroke: Assessment and stabilization, Atlantic Coast Veterinary Conference Proceedings 2004