

1 Heatstroke

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2 Outline

- Definitions
- Pathophysiology
- Physical Exam
- Diagnostic Tests
- Treatment
- Prognosis

3 Definition

- Heat cramp
 - Muscle cramps from Na and Cl loss
- Heat exhaustion
 - Fatigue, weakness, muscle tremors, vomiting, diarrhea
- Heatstroke
 - Severe CNS dysfunction

4 Heatstroke- defined

- Core body temperature >104F (40C)
- CNS dysfunction
 - T>105.8F may cause permanent brain damage
 - T> 109.4F may cause severe organ damage, markedly increased mortality
 - T>120F for 5 min cellular necrosis occurs
- Organ dysfunction

5 Heatstroke types

- Exertional
 - Caused by exercise
- Nonexertional = Classic
 - Secondary to environmental factors
 - Ambient temp of 75° F, closed car, direct sun = 120° F in 20 minutes, death in one hour

6 Acclimatization

- Takes 20-60 days, explains why HS happens early in summer, not on the hottest days
- Mechanisms
 - Increased cutaneous circulation
 - Increased CO
 - Peripheral vasodilation
 - Salt conservation and volume expansion

- Resistance to rhabdomyolysis

7 **Predisposing factors**

- Environment
 - Warm, humid environment
 - Closed confinement with poor ventilation
- Exercise
- Underlying respiratory compromise
 - Brachycephalic conformation
 - Laryngeal paralysis
 - Collapsing trachea

8 **Predisposing factors**

- Cardiac disease
 - Decreased ability to increase cardiac output
 - Furosemide therapy → hypovolemia, electrolyte abnormalities
- Dark or thick haircoat
- Obesity
- CNS disease

9 **Hyperthermia vs. Fever**

- Hyperthermia
 - Inappropriate balance of heat dissipation compared to heat load
 - Patient will feel hot
- Fever
 - Cytokine mediated resetting of thermoregulation set point
 - Appropriate physiologic response
 - Active cooling not indicated

10 **Protective mechanisms**

11 **Thermoregulation**

12 **Thermoregulation- Heat physiology**

- Heat dissipation
 - Convection- by movement of air or fluid over the animal
 - Conduction- contact with a cooler surface
 - Radiation- natural heat release of body into environment
 - Evaporation- by turning liquid into a gas (panting)
- With increasing ambient temperature and humidity, these become less effective

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14 **Acute phase response**

- Activated by inflammation
 - Protects against tissue injury
 - Promotes repair

- Pro-inflammatory cytokines
 - IL-1, TNF, IL-8
 - Leukocytosis
 - Acute phase proteins stimulated
 - Fever
 - WBC and endothelial cell activation
- Anti-inflammatory cytokines
 - IL-6

15 Heat shock proteins

- Protective role from thermal damage
 - Cellular level
 - Prevents denaturation of intracellular proteins
 - Protein chaperones and molecular guardians
 - Systemic level
 - Regulation of baroreceptor response
 - Protect enzyme function
 - Reduces oxidative stress
- Varying levels may explain genetic predisposition to HS

16 Pathophysiologic sequence

- Muscle release IL-1 and IL-6
- Increased endotoxin from GI tract
- Leads to excessive activation WBC and endothelial cells
- More pro-inflammatory and anti-inflammatory cytokines released
- Activation of coagulation (hypercoagulable)
 - Microthrombi
 - Progressive tissue injury
- Inhibition of fibrinolysis

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18 Sequelae

- Coagulation abnormalities
 - DIC (Disseminated Intravascular Coagulation)
- Direct heat injury
 - MODS (Multiple Organ Dysfunction Syndrome)
 - Respiratory, CV, renal, GI, CNS, coagulation, hepatic

19 Physical Examination- TPR

- Temperature
 - Decreased, normal, elevated
 - Perfusion dependent
 - If cooling methods have been initiated
- Pulse
 - Tachycardia
 - Compensatory sinus tachycardia
- Respiration
 - Tachypnea, panting
 - Increase heat dissipation
 - Generally not a result of respiratory disease

20 Cardiovascular System

- Hyperdynamic state

- Hyperemic MM
- Increased CRT
- Weak pulses
 - Hypovolemia (evaporative fluid loss, vomiting, diarrhea, vasodilation)
- ECG
 - Sinus tachycardia
 - Intermittent ventricular arrhythmias
 - Associated with a worse outcome

21 Respiratory system

- Rule out underlying airway disease
 - Laryngeal paralysis
 - Tracheal collapse
 - Upper airway obstruction
- Auscultation
 - Harsh airway sounds
 - Crackles
- Evaluate for aspiration pneumonia
 - Secondary to patient vomiting
- DIC
 - Pulmonary parenchymal hemorrhage (uncommon)

22 Central Nervous System

- 1**
 - Mentation
 - Depression most common
 - Also comatose, alert, stuporous
 - Variable pupil size
 - Pinpoint to dilated
 - Usually light responsive
 - Cortical blindness
 - Usually resolves
- 2**
 - Neurologic abnormalities
 - Head bobbing, tremors
 - Ataxia
 - Due to:
 - Poor cerebral perfusion
 - Direct thermal damage
 - Cerebral edema
 - CNS hemorrhage
 - Metabolic abnormalities

23 Renal System

- Bladder size
 - Monitor size change with fluid therapy for urine production
- ARF can be sequelae
 - Monitor urine output

24 Gastrointestinal System

- Diarrhea
 - Watery to hemorrhagic
 - Mucosal sloughing
- Vomiting
- Gastric ulceration

25 Coagulation system

- DIC
 - Initiated by direct damage
 - Thermal injury to tissues and endothelium

- Signs
 - Petechiation
 - Ecchymoses
 - Hematuria
 - Hematemesis
 - Hematechezia

26 **Laboratory evaluation**

- CBC
- Chemistry
- Coagulation
- Urinalysis
- Blood gas

27 **Treatment**

- Cool to 103° F then STOP
 - Avoid rebound hypothermia
- Surface cooling
 - Tepid water baths, fans, damp towels, ice packs wrapped in towels over large vessels
 - Avoid alcohol and excessively cold water (vasoconstriction inhibits heat loss)
- Internal cooling
 - IV fluids, cool rectal fluids (controversial), gastric lavage, peritoneal dialysis

28 **Treatment- CVS**

- Hypovolemic shock
 - 90ml/kg dog
 - 50ml/kg cat
- Blood pressure monitoring
- Colloids
 - Hetastarch 5-10ml/kg
- Pressors
 - Dopamine 5-20mcg/kg/min
 - Norepinephrine 0.2-2mcg/kg/min

29 **Treatment- Respiratory**

- Oxygen supplementation
 - Offer initially
 - Monitor SpO₂, MM color
- Auscult frequently
- ALI
- ARDS

30 **Treatment- CNS**

- Establish a baseline with full neurologic exam at presentation
- Biochemical evaluation
 - Glucose
 - Avoid creating hyperglycemia

- Acid Base status
- PCV/TP
- Seizure control

31 Treatment-CNS

- Correct perfusion abnormalities, avoid hypotonic fluids
- Cerebral edema
 - Mannitol 0.5-1g/kg over 20-30 minutes
 - Hypertonic saline
 - Elevate head 15-30 degrees above horizontal
 - Do not compress jugular veins
- Progression of neurologic signs despite therapy has a poor prognosis

32 Treatment- Renal

- Urinary catheter
 - Monitor ins and out
 - At least 2ml/kg/hr
 - Insensible loss 20ml/kg/day
- Complete urinalysis
 - Evaluate for casts
- Monitor BUN, Creat, Na+, K+
- Avoid steroids and NSAIDs

33 Treatment-Coagulation

- Get baseline PT, aPTT, and PLT count
- Plasma
 - 10-15ml/kg
 - Will NOT raise albumin
 - 22ml/kg plasma needed to raise albumin 0.5mg/dl
 - Provides clotting factors that may be deficient
- Heparin therapy in DIC is controversial

34 Treatment- GI system

- GI protectants
 - Sucralfate
 - Famotidine
 - Broad-spectrum antibiotics
 - for GI translocation
 - Ampicillin
 - Metronidazole
- Intussusceptions may develop

35 Prognosis

- Guarded
 - 2006 retrospective had a 50% mortality
 - Increased mortality: coagulopathy, hypoglycemia, azotemia, seizures, obesity
- Animals cooled prior to transport have a 19% mortality compared to 44% not cooled

- Delayed treatment worsens outcome

36 Acknowledgements

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