Common Diseases of Urban Wildlife

GENERAL DISEASES – Myopathy and Trauma

The Australian Registry of Wildlife Health

MISSION STATEMENT:
The Australian Registry of Wildlife Health is committed to contributing to the preservation of Australia's biodiversity through increased understanding of the interactions among animals, the environment, and disease causing agents.

Taronga Zoo | Western Plains Zoo
Zoological Parks Board of New South Wales

1 General Diseases

1.1 Shock

Many animals that have suffered a serious injury or are debilitated by disease are found in a state of shock. Shock is defined as acute circulatory failure that results in multisystemic hypoperfusion and hypoxia of tissues (Ware, 1992). Clinical signs of shock are often related to hypotension. The mucous membranes of an animal in shock may be pale or muddy and the peripheral blood vessels are collapsed or provide a weak pulse. The heart rate may be weak and rapid. Animals in a state of shock are often weak, depressed, tachypnoic, and have reduced urine output. Animals suffering from endotoxic shock, may have bright red mucosa.

Dehydration often contributes to the lack of peripheral perfusion. An animal is severely dehydrated when the eyes are sunken, the capillary refill time is very slow, the mucous membranes are dry and tacky, and the skin has lost its elasticity.

There are four mechanisms that result in shock; hypovolaemia, loss of vasomotor tone and massive peripheral vasodilation, severe reduction in cardiac output, or obstruction of blood flow.

The neuroendocrine cascade that is initiated during shock is initially protective, but over time energy reserves are depleted and peripheral vasoconstriction contributes to hypoperfusion of tissues. The heart, lungs, liver, gastrointestinal tract, pancreas, and central nervous system are most susceptible to damage induced by hypoxia.

Pulmonary effects of shock can include consolidation of tissue, and increased risk of bacterial infection. The effects of shock on the lung can be highly species specific. Some species experience “Acute Respiratory Distress Syndrome”, also known as shock lung, which is manifested as pulmonary oedema and decreased activity of alveolar macrophages.

Acute necrosis of the proximal renal tubules and periacinar (centrolobular) regions of the liver occurs under conditions of prolonged hypoperfusion. Mucosal ulceration and decreased mobility occur with gastrointestinal ischaema. These gastrointestinal lesions can allow bacteria or bacterial toxins to enter the blood stream (Mann and Helmick, 1996). Cells exposed to hypoxia initially undergo degenerative change, but once cell death has taken place, the changes induced may be irreversible. Animals that are treated in this phase of shock may respond to initial fluid therapy, but succumb to acute renal tubular necrosis (urate nephrosis and visceral gout in birds), gastrointestinal ulceration or sepsis three to five days later. If hypoperfusion continues, pancreatic ischaema can result in the release of vasoactive substances and myocardial depressant factor. Ultimately, cerebral ischaemia causes nerve cell death (Ware, 1992).

1.2 Bite Wounds

Predation is an everyday occurrence in wildlife. Bite wounds inflicted by feral or domestic pets account for a large proportion of the animals admitted to wildlife care centres. Bite wounds caused by canine and feline predators are most often centred over the neck, shoulders, and dorsal thoracic region. Puncture wounds caused by feline predators are often very fine. These wounds can be difficult to see, and often the only outward sign of attack is moist or matted fur over the shoulders. Canid-inflicted bite wounds do not necessarily break the skin. The mild outward appearance of predator-induced lesions often masks very serious internal injuries. Feline bite wounds can puncture deep into the tissues, and felids have the potential to break bones or reduce the underlying muscle to pulp. Canine bite wounds are most often associated with circular subcutaneous contusions over the dorsal thorax, and crushing injury to the chest. Canine bite wounds often cause extensive pulmonary contusion and fractured ribs. Measuring the distance between puncture wounds can be used to estimate the inter-canine tooth distance, which can help to differentiate wounds inflicted by cats or foxes (18-22 mm inter-canine distance) from those inflicted by large dogs (>25 mm inter-canine distance)

Feline bite wounds are often heavily contaminated with *Pasteurella multocida*, and sepsis is a very common sequela. Canine bite wounds may be contaminated with a wide variety of gram negative and anaerobic bacteria. The prognosis for any animal receiving predator bite wounds, however, is most often guarded to poor.

1.3 Burns

Wild animals sustain thermal injuries from contact with any number of hot items in an urban environment. Wildlife may also sustain severe thermal injury during forest fires. Chemical burns have been suspected as the cause of melting ulcerative wounds in wildlife, where the wounds were localised to parts of the body where contact with caustic agents may occur.

Clinical signs of burn wounds may vary from charred feathers or fur to widespread necrosis and secondary suppurative inflammation. Animals may suffer concurrent inhalation pneumonia or traumatic injury. When greater than 25% of the animal's skin surface is burnt the animal is highly likely to succumb to systemic illness, such as shock, sepsis, renal failure, and anaemia (Muller, et al., 1989). Australian fauna with less than 15% of their body affected by burns are considered to have a reasonable prognosis. The prognosis deteriorates when greater than 15 % of the skin surface area is burnt, and when the head and joints are affected (Spielman, 1994).

Burns occurring in animals are most often categorised as first degree, second degree or third degree burns. First-degree burns involve incomplete destruction of the epidermis, and healing is achieved rapidly through re-epithelialisation. Second-degree burns are defined as damage to the epidermis and variable quantities of dermis. These injuries heal primarily through re-epithelialisation from adnexal remnants. The degree of residual alopecia will depend upon the number of follicles completely damaged. Third degree burns involve the complete destruction of the epidermis, adnexa, and nerve endings. Healing of full-thickness burns occurs slowly through wound contraction, and migration of epidermal cells from the wound margins. Animals suffering second or third degree burns to greater than 50% of the skin surface have a very poor prognosis for recovery and euthanasia is recommended (Spielman, 1994).

Burn injuries break the barrier protection offered by skin and provide a route for either localised or systemic invasion by bacteria. These wounds are often initially colonised by gram-positive bacteria; however, over the course of three to five days, gram-negative bacteria tend to invade. *Pseudomonas aeruginosa* is a prevalent pathogen involved in the opportunistic infection of burns.

Electrocution is another source of thermal tissue damage. Most animals that are electrocuted die acutely as a result of ventricular fibrillation and pulmonary oedema (Cooper, 1996). Animals that are electrocuted have lesions that can be traced along the flow of current, often with a distinct entry and exit wound. Thus, simultaneous wing and foot injuries are common in electrocuted birds and bats. Electrocuted animals also tend to have a characteristic odour. If the animal survives the acute effects of electrocution, they will usually have thermal injuries that become progressively more severe and exudative. It is not unusual for electrocuted animals to be found with debility and an unusual odour, but few lesions. Then over a course of three to five days ulcerative wounds develop along the path of the current. This progression of clinical signs is most likely the result of ischaemic tissue necrosis stemming from thermal injury to blood vessels.

1.4 Ocular Injuries

Ocular injuries are not uncommon in injured wildlife. Hyphaema and the presence of blood in the oral cavity are indicators of significant cranial injury. Diagnosis and treatment of ocular injuries in wildlife are very similar to that of domestic animals; however, the likelihood of return to full ocular function must be assessed. The release of blind or partially blind animals may be questionable, especially when there is no possibility of post-release monitoring.

1.5 Skeletal Trauma

Traumatic injury that is sufficient to fracture a bone is often accompanied by substantial soft tissue injury. It is important to assess the integrity of the vascular and nervous supply and to evaluate the joints prior to immobilising a limb. Animals that

have sustained nervous deficits or joint injury will have a very poor prognosis for recovery of full function. Rib fractures can be very painful. Concurrent pneumothorax or pulmonary contusion is common in animals presenting with fractured ribs.

Fractures intended for repair should be assessed radiographically, to ensure the fracture is not secondary to another primary condition such as infection, neoplasia, or metabolic bone disease.

1.6 Exertional Myopathy

Aetiology

Exertional myopathy, capture myopathy, or exertional rhabdomyolysis, are terms used to describe a syndrome characterised by damage to skeletal or cardiac muscle following a period of intense physical activity. Exertional myopathy occurs in a wide variety of mammals and birds; however, some species appear to be more susceptible to developing clinically apparent disease than others. There is no association between sex or age and the development of exertional myopathy; however, elevated environmental temperature may increase the likelihood of an animal developing exertional myopathy (Williams and Thorne, 1996). Exertional myopathy is well documented in Australian macropods and occurs occasionally in birds (Shepherd, 1983).

Exertional myopathy, red kangaroo.

Clinical signs

The clinical signs associated with exertional myopathy will vary depending on the types of muscles that have been damaged and the degree of damage sustained. The onset of this syndrome is usually precipitated by activities such as chemical or physical restraint, transport, or chase. Peracute death may follow such an incident, or clinical signs may begin days to weeks after the incident. Initial clinical signs include: increased heart rate, increased respiratory rate, and increased body temperature. The

animal may then go on to develop depression, ataxia, fever, pulmonary oedema, unsteady or stiff gait, muscle tremors, and myoglobinuria. Recumbency and death may ensue in severely affected animals.

Clinical pathological changes that are associated with this syndrome include those related to systemic metabolic acidosis. Blood urea nitrogen concentration is elevated when substantial renal impairment has occurred as a result of ischaemia or myoglobinuria. Creatine kinase (CK), aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) concentrations are also markedly elevated in the serum. Serum concentrations of CK increase rapidly in response to cardiac or skeletal muscle damage, yet the enzyme has a short half-life and tends to persist in very high concentrations only in the face of ongoing muscle damage. Serum concentrations of AST and LDH are less specific to muscle damage, since they may originate from multiple organs. Serum AST concentrations rise more slowly after an initial insult to muscle compared with serum CK concentrations. Serum AST has a longer half life and persists longer at elevated concentrations compared with CK. Serial monitoring of AST and CK may provide some insight into the duration and degree of muscle damage. Isoenzymes of CK are monitored in human patients to differentiate elevations of serum concentrations of CK originating from damage to cardiac versus skeletal muscle. This test would be useful as a prognostic indicator for animals suffering from exertional myopathy. Unfortunately, the use of CK isoenzymes has not been sufficiently investigated and standardised for use in Australian native fauna.

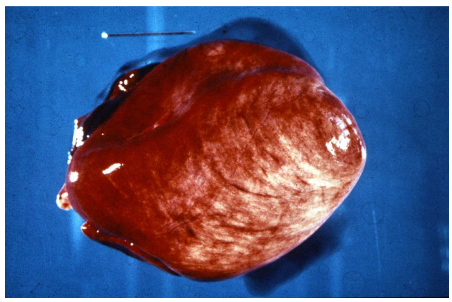
Pathogenesis

Sub-classifications of exertional myopathy include peracute death, muscle damage, muscle rupture, delayed peracute syndrome, compartment syndrome, and myoglobinuric nephrosis.

Peracute death syndrome is primarily the result of severe metabolic imbalance and shock. It is suspected that severe metabolic acidosis and cardiac fibrillation contribute to the pathogenesis of peracute death syndrome.

Muscle damage as a result of exertional myopathy is primarily due to a combination of reduced tissue perfusion, acidosis and depletion of ATP reserves. Damage to the muscle cells is degenerative rather than inflammatory. The sympathetic response primarily functions to direct blood flow away from some organs and maintain adequate blood flow to the brain and skeletal muscles. The peripheral arterioles constrict under sympathetic stimulation. Repetitive stimulation and increasing lactic acid concentrations cause the nerves supplying the peripheral arterioles to become refractory to stimuli and the arterioles dilate. The nervous supply of the peripheral venules is more resistant to the effects of increased lactic acid concentrations. Thus, venules continue to contract under sympathetic stimulation, while the arterioles dilate. This combination of events results in congestion, hypoxia, tissue damage, and circulatory collapse (shock) through decreased cardiac return and cardiac output (Sprayker, 1993).

Delayed peracute death syndrome occurs when an animal dies suddenly after a second episode of exertion or stress. This syndrome may occur due to the presence of pre-existing cardiac lesions increasing the sensitivity of the animal to hyperkalaemia and acidosis associated with a second exertional episode.



Exertional cardiac myopathy, red kangaroo.

Compartment syndrome has been reported in human athletes, and has been seen in the hind-limb adductor muscles of macropods, and the deep pectoral muscle of birds that were subject to exertion. Compartment syndrome occurs primarily in muscles that are bound by a tight fascia. When oedema occurs secondary to lactic acid build up in these muscles, the internal pressure can be so great that it compresses the muscle's venous supply. Arterial walls are far more rigid than those of the veins and they withstand compressive forces for a longer period of time. The combination of arterial integrity and venous occlusion result in massive oedema, congestion, haemorrhage and ischemic necrosis throughout the affected muscle.

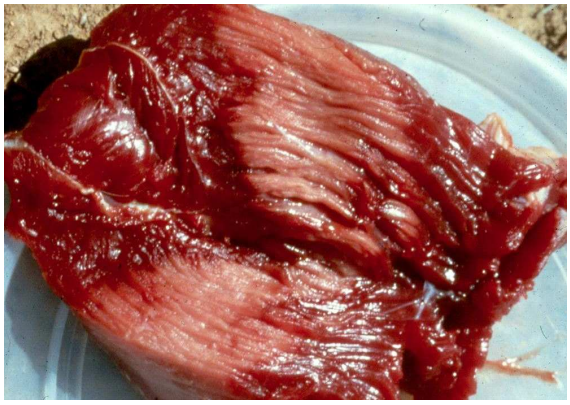
Myoglobinuric nephrosis often occurs when muscle break down results in the liberation of large quantities of myoglobin into the blood stream. This myoglobin is filtered through the glomeruli and is acutely toxic to the epithelial cells of the proximal tubules, and the loop of Henle. Shock often occurs in animals suffering exertional myopathy, and ischaemic damage may contribute to the process of acute tubular necrosis.

Pathology

Lesions evident on gross post mortem examination of an animal with exertional myopathy may include:

- haemorrhage or oedema throughout muscles,
- muscle pallor or pale streaking,
- gritty or chalky muscle texture (mineralisation of sub-acute to chronic injuries),
- swollen kidneys, with a dark cortex,
- dark brown urine,
- pulmonary congestion and oedema, and/or
- haemorrhage and congestion in the adrenal cortex

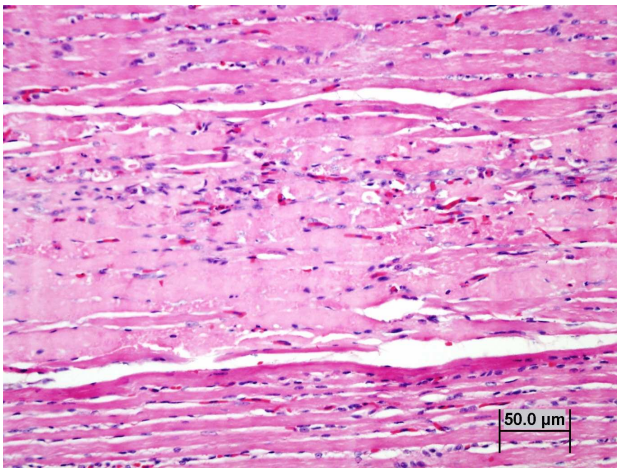
Any skeletal muscle may be affected; however, the hind limb adductor muscles are particularly prone to post-exertional degeneration. Muscles may appear swollen and haemorrhagic or soft, dry, pale and friable. Muscles affected by compartment syndrome are often markedly swollen, haemorrhagic, congested and muscle fibres are friable as a result of ischaemic necrosis.



Ischaemic necrosis Compartment syndrome, red-necked wallaby

Microscopic lesions in muscles will vary depending on the severity and duration of the insult. The primary pathological process evident is cell degeneration, with a regenerative response visible several days to weeks after the initial damage. Microscopic muscle lesions may include:

- haemorrhage or oedema throughout the interstitium of the muscle,
- multifocal to diffuse myofibre degeneration characterised by cell swelling, loss of myofibril striation, fragmentation of myofibrils, and cupping degeneration, and
- within more chronic lesions there may be mineralisation of affected fibres, satellite cell proliferation, infiltration of myocytes with mononuclear cells, phagocytosis of degenerate myofibrils, and possibly the initiation of interstitial fibrosis.

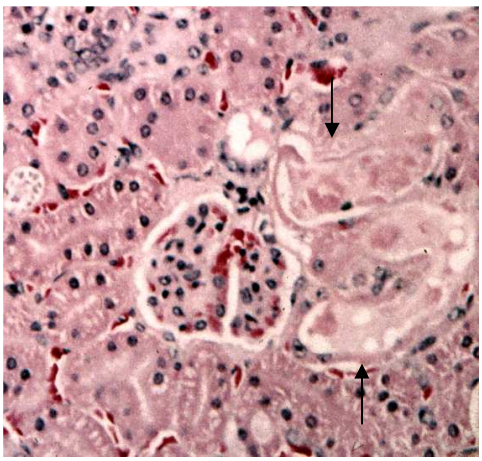


Extertional myodegeneration, H & E stain

These lesions may be evident within the cardiac musculature, skeletal musculature, and in severe cases in the striated muscle of the tongue, diaphragm, and oesophagus.

Microscopic renal lesions may include:

- orange to brown pigment within the cytoplasm of renal tubular epithelial cells,
- hydropic change, necrosis or regeneration of renal tubule epithelial cells, and/or
- granular casts or sloughed epithelial cells scattered throughout tubular lumina.


Acute renal tubular necrosis associated with exertional myopathy, bridled nailtail wallaby, H & E stain

Additional microscopic lesions are often related to ischaemia and shock, and may include:

- pulmonary oedema and congestion,
- periacinal hepatic necrosis (anoxic damage),
- foci of necrosis within the adrenal cortex,
- perivascular haemorrhages throughout the meninges and parenchyma of the brain, and/or
- lympholysis

Differential diagnoses

Exertional myopathy must be differentiated from nutritional, toxic, infectious, and parasitic myopathies. Exertional myopathy should also be differentiated from other causes of hind limb dysfunction, such as direct trauma, neural angiostrongylosis or other causes of central or peripheral nervous system dysfunction.

Treatment

The objectives of therapy for exertional myopathy are to reverse concurrent shock or hyperthermia, reverse metabolic acidosis, and stabilise cellular membranes. Fluid therapy and correction of acidosis through administration of sodium bicarbonate are initiated to increase tissue perfusion and prevent myoglobinuric nephrosis. Administration of steroids may contribute to stabilising cell membranes to prevent ongoing or irreversible cell degeneration.

Prevention

Myopathies are considered to be extremely painful, and associated mortality rates are very high. Animals that survive the initial episode of myopathy may go on to relapse upon subsequent exertion. Preventive planning is paramount to reducing the risk of exertional myopathy. The prevention of exertional myopathy should be a consideration each time a wild animal is to be handled. Factors such as increased environmental temperature, inexperienced staff members, traumatic restraint devices, and prolonged duration of restraint increase the risk of the animal developing exertional myopathy. The administration of vitamin E and selenium at the time of an

exertional episode has been proven, in some species, to reduce the risk of subsequent myopathy (Williams and Thorne, 1996). Supplementation of the diet with these nutrients in advance of an impending stressful event may be warranted. Diazepam has been shown to reduce the prevalence of myonecrosis in macropods when administered intramuscularly after capture (Shepherd, 1983).

2 References

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