PATHOLOGY OF TRAUMA

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Trauma is a common cause of injury and death in both domestic and wild mammals. Trauma is a broad category of injuries that include physical injury (fractured bones, muscle trauma, gunshot, electrocution/lightning strike, and drowning – predation, hit-by-vehicle, fighting, and accidents all cause physical injury and fall into fractured bones or muscle trauma depending on the predator and the prey) and temperature-related injury (burns, frostbite, hypothermia, and hyperthermia).

Trauma causes injury at the molecular (cellular disruption, injury, or death), gross (muscle, bone, or organ), and systemic (shock) levels. The history of a case and gross pathology (necropsy) are normally enough to diagnose cause of death as different types of trauma have characteristic gross lesions. It also is possible to determine the species of predator that killed an animal if enough of the carcass is remaining and someone has done a thorough investigation in the field at the kill location. There will be more information on this topic later in the notes.

TERMINOLOGY

There are specific terms used to describe trauma that one should be familiar with:

- **Trauma** = an injury produced by a sudden, violent force that results in compression, stretching, torsion, or penetration of the tissues
- **Abrasion** = tearing away of epidermal cells by friction or crushing (scrape)
- **Laceration** = a split or tear caused by excessive stretching of tissue (NOT a cut)
- **Incision** = a wound caused by the pressure or friction against skin or other tissues by an instrument with a sharp edge; longer than deep (cut)
- **Penetrating injury** = the impact of an appropriately shaped object against the skin that causes wound that is deeper than long (stab)
- **Contusion** = the force of an impact is transmitted through the skin into the underlying tissue and disrupts the walls of small blood vessels, causing interstitial bleeding without disruption of epidermis (bruise)
- **Fracture** = mechanically produced disruption in the continuity of bone (broken bone)

SEQUELAE LEADING TO INJURY AND DEATH

**Cellular response to injury** depends on the type of injury the cell incurs. Cellular injury due to physical injury (blunt, sharp, or penetrating trauma – e.g. hit-by-vehicle or gunshot) causes cells to die from direct rupture or disruption of blood supply (hypoxia). Extreme cold damages cells directly through intracellular ice crystal formation that destroys the integrity of the cell or indirectly through impaired blood flow. This phenomenon results from vasoconstriction
of extremities to supply blood to critical internal organs and the brain. Extreme heat results in increased metabolism, resulting in disruption of substrate, water, and pH levels. In addition, the function and structure of cells are negatively impacted through denaturing of enzymes and proteins due to temperatures outside of the viable living range. Cellular response to injury depends on the type of cell affected (labile, quiescent, or permanent). Skin or muscle cells are labile and will be repaired over time through the body’s healing process. Nerve cells, which are permanent, are not replaced through healing and thus lead to permanent injury (e.g., paralysis).

Local response to injury includes hemorrhage of blood via lacerated blood vessels, internal organ damage via direct laceration or loss of blood supply (hypoxia), inflammation (the body’s response to injury), or infection. Primary infection occurs because injury disrupts the integrity of the innate immune system, often through laceration of the skin, which provides a portal of entry for bacteria to infect the site of injury. Secondary infection of other organ systems can occur if bacteria enter the blood system and results in systemic infection. There are numerous types of bacteria that can result in infection depending on the source (soil, skin, or mucous membranes).

An extremely serious and life-threatening sequelae of trauma is shock, or cardiovascular collapse. Shock is circulatory disruption due to loss of circulating blood volume (hypovolemic shock), reduced cardiac output of blood (cardiogenic shock), and/or inappropriate peripheral vascular resistance through vasodilation (maldistribution or distributive shock). Hypovolemic shock occurs as a direct result of loss of blood through internal or external bleeding. Extreme fluid loss through diarrhea, vomiting, or burns can indirectly lead to inadequate blood volume and result in hypovolemic shock. While there are three types of shock, the underlying events are stereotypical. Hypotension (low blood pressure) leads to decreased tissue perfusion and thus hypoxia of cells → cells shift to anaerobic metabolism → increased lactic acid and decreased oxygen, glucose, and ATP in cells → cellular degeneration and swelling → cell death and ultimately death of the organism. In the early stages of shock, the body compensates for the hypotension through vasoconstriction, fluid movement into plasma, and increased heart rate. The absolute loss of blood volume determines the outcome of shock. If less than 10% of blood volume is lost than the organism returns to homeostasis, if greater than 10% of blood volume is lost than the result is loss of pressure and tissue perfusion, and if greater than 35% of blood volume occurs than pressure and cardiac output fall drastic leading to death. Cardiogenic shock can result from an underlying disease process or an injury such as electrocution that disrupts the normal rhythm of the heart, which decreases blood flow. Electrocution, stress, allergic reactions, or endotoxemia can all result in maldistribution or distributive shock via vasodilation (not blood loss!) resulting in inadequate blood volume compared to intravascular capacity.

PHYSICAL INJURY

Fractured bones are a common result of physical injury. Bone fractures are classified as: a) closed or simple fracture – clean break of the bone with no protrusion of the bone through the skin but with associated internal hemorrhage; b) comminuted fracture – bone shatters resulting in multiple bone fragments and extensive damage to surrounding muscle tissue; and c) open or compound fracture – the broken bone penetrates through muscle and skin and is exposed to the external environment. Fractured bones result in loss of function of the limb or surrounding area, loss of structural support, soft tissue damage, and hematoma (blood clot around fracture).
Anemia and hypovolemic shock also can result from severely or untreated fractured bones. Common causes of fractured bones include hit-by-vehicle, fighting, predation, gunshot or arrow, striking objects, capture, or accidents. Fractures heal by first forming a blood clot at the site of the fracture followed within days by infiltration by fibroblasts via neovascularization and formation of collagen by Mesenchymal cells (later these cells will form cartilage and bone through metaplasia) to lay the framework for callus formation (necrotic tissue also helps form the callus). Also within days macrophages and platelets at the site of the blood clot release growth factors that encourage osteogenic tissue formation resulting in woven bone (visible within 36 hours). Primary callus formation occurs through the unorganized meshwork of woven bone formation that bridges the gap at the fracture site. Within weeks a secondary callus forms by replacing woven bone with stronger lamellar bone. Within years this secondary callus will decrease in size by osteoclasts, resulting in the normal shape of the bone.

**Muscle trauma** occurs from a variety of causes, including crush injury, laceration (possibly from fractured bones), surgical incisions, tearing from excessive stretching or exercise, burns, gunshot/arrows, and predation. Healing occurs through fibrosis formation. As mentioned previously, **predation** is a common cause of muscle trauma. The species of predator can be determined by signs at the kill site, stereotypic kill patterns (e.g., mountain lions prefer to bite their prey from behind at the base of the skull, resulting in cervical dislocation and paralysis), or distance between canine punctures in the skin or underlying muscle tissue (measure the distance in millimeters and compare distance to reference chart of predatory species). **Gunshot** often results in extensive muscle damage. The faster the bullet is traveling and the greater the mass of the bullet, the more damage that will occur (Force =MV^2/2g). Gunshot entrance wounds are normally the approximate diameter of the bullet, circular, with a rounded abrasion of the skin (normally seen best after skinning the carcass and looking at the subcutaneous layer of the skin). The extent of the injury is greater than the diameter of the bullet due to temporary wound cavity formation from the radially projecting force of the bullet. Not only is muscle damage caused, but also fractured bones, lacerated blood vessels, and organ damage. If the bullet exits, the exit wound is often larger than the entrance wound, irregularly shaped due to bullet mushrooming and possible bone fragments penetrating the exit wound.

**Electrocution** occurs if a circuit is completed between two conductors, including two body parts. Electricity travels the most direct path through the body and causes injury or death differently depending on which part(s) of the body are affected (heart or lung failure, muscle contractions, burns, etc.). Large birds of prey (I know they are not mammals but these are the most common wildlife species affected) can be electrocuted by touching two power lines – this is more common than you might think! In domestic animals, poor electrical wiring in barns can result in electrocution of animals. **Lightning strikes** are a specific cause of electrocution and occur in both wild and domestic animals. If one can see the skin, then arborescent (tree-like) cutaneous hyperemia is a common gross lesion. In furred mammals, singed hair is a common gross lesion.

**Drowning** is the final physical trauma that will be discussed. Both domestic and wild animals drown. Common causes include being swept away in current, breaking through ice, exhaustion, commercial fishing, artificial or natural flooding, oil spills, or secondary problems such as illness, injury, or anesthesia. One may survive drowning depending on the duration of submersion, water temperature, and speed of resuscitation. Water temperature affects survival because cold water results in vasoconstriction of extremities leading to most of the blood flow going to critical internal organs and the brain, which results in longer survival time (diving
reflex). Drowning results in water in the respiratory tract (blocking bronchioles) and alimentary tract (resulting in no harm or vomiting). If freshwater is inhaled then water may be pumped by the heart throughout the blood, resulting in hemolysis. If saltwater is inhaled the lungs are quite heavy due to fluid from the blood diffusing into the lungs (outcome is dehydration and pulmonary edema). Ultimately death is caused by hypoxia and respiratory acidosis (due to anaerobic metabolism) leading to cardiac arrest.

TEMPERATURE RELATED TRAUMA

**Burns** result from exposure to excessive heat. There are three categories of burns, including first degree, second degree, and third degree burns. First and second degree burns are classified as partial-thickness burns while third degree burns are more serious full-thickness burns. First degree burns affect the epidermis only, second degree burns affect the epidermis and the first layer (superficial layer) of dermis, and third degree burns affect all of the epidermis and the dermis. Ultraviolet light, heat (e.g. brush, grass, or forest fires), chemicals, or friction all cause burns. First degree burns result in erythema and edema via acute inflammation. Second degree burns progress to vesicle formation due to fluid accumulating between the epidermis and dermal layer due to inflammatory response. Third degree burns are characterized by desiccation and charring of epidermis with amorphous accretion of connective tissue. Edema and vesicle formation is caused by: capillaries and small blood vessels become dilated as tissue temperature rises → capillary wall permeability increases → fluid components of blood leave vessel into interstitial space → edema. Hypovolemic shock can result from extensive burns due to fluid loss from the plasma during edema formation. In addition to the direct damage caused by the heat source and inflammatory response, burn sites are extremely prone to infection by opportunistic bacteria leading to adjacent tissue infection or systemic infection. Infection occurs because the integrity of the skin (part of the innate immune system!) is disrupted, leaving the burn victim susceptible to infection. The degree of healing depends on the type of burn. First degree burns heal completely with minimal scarring because of the intact dermis. Second degree burns result in dermal scarring but adnexa (appendages such as hair and sweat glands) are maintained. Third degree burns are very serious and result in permanent scarring and loss of adnexa (unless a skin graft is performed). Third degree burns heal through macrophages infiltrating the tissue resulting in the sloughing of necrotic tissue. The wound is filled in with granulation tissue.

**Frostbite** is very common in captive animals, occurring frequently in range animals. Song birds also are prone to frostbite, especially during unexpected spring snow storms. The cause of frostbite is exposure to extreme cold, resulting in vasoconstriction, dehydration, ice crystal formation in cells, thrombosis, and metabolic disruption leading to ischemia. Frostbite progresses from frostbit (epidermal layer only) → superficial frostbite (epidermal layer and top layer of the dermis) → deep frostbite (epidermal and full dermal layers affected). Deep frostbite can result in sloughing of necrotic tissue, loss of appendages, and dry gangrene (coagulation necrosis due to infarction – tissue death from lack of oxygen).

**Hypothermia** occurs from exposure of a large surface area to cold temperatures rather than local exposure (frostbite). Hypothermia is caused by low body temperatures that disrupt circulation (vasoconstriction) when the body temperature falls below 20° centigrade; this occurs more rapidly with wind or cold water exposure. Exposure to low temperatures occurs when the mammal is unable to seek adequate shelter, if they are caught in a trap, or an unexpected drop in temperature or a storm occurs. Right sided cardiac dilation (due to blood flow backing up from
the lungs) and pulmonary edema (due to inflammation of lungs due to breathing in cold air, ice crystal formation, and decreased blood flow due to low body temperature) are the only lesions that may be present due to death from hypothermia.

**Hyperthermia** results from the body temperature rising above a critical safe temperature. If hyperthermia is not resolved quickly then irreversible changes occur resulting in death. Causes of hyperthermia are extensive, but a short list includes overexposure to the sun, capture/physical restraint, inadequate shelter in hot ambient temperatures, being locked in the house or car during hot days with inadequate ventilation, or overcrowding. Acute heat stress may result in burns along with damage to kidneys, liver, heart, and brain that lead to excess fluid loss and ultimately shock (which kind?). Chronic heat stress in domestic animals results in decreased production due to down-regulation of metabolism (the mechanism is not known). Rise in body temperature results in vasodilation with reduction in effective blood volume (maldistribution shock). To compensate the heart rate increases and the heart dilates, reducing cardiac efficiency. To try to cool the body, tachypnea (abnormally fast breathing) occurs followed by irregular breathing and finally breathing stops, resulting in death.

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