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Treatment and Management of Major Burns

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The patient in this case was a six year old female dachshund named Penny. Four weeks previously, the referring veterinarian performed a spay on the dog and due to post-surgery complications, placed her on an electric heating pad. The result was a third degree burn involving 20% of the body area, located entirely on the dog's back. An eschar had formed and this was partially removed by the referring veterinarian. The dog was presented with a large amount of granulation tissue filling in the wound site. A general physical and basic blood work revealed no other complications. A treatment/management regime was discussed and executed. The dog was placed in a tub of warm circulating water each day to clean and soften the wound. More of the eschar was debrided until the entire wound was bordered by viable tissue. After drying off, mafenide (Sulfamylon) was applied to the granulating bed twice daily; no other antibiotics were used. The progress of the wound was carefully monitored, with the original intention of performing a skin graft when the granulating bed was ready. During the 14 day stay in the clinic the wound had progressed very well. There was substantial regrowth of epithelium occuring from the wound edges and the wound underwent noticeable daily contraction, decreasing the initial size of the defect considerably. With this prognosis, it was decided to send the dog home to allow further reduction and contraction of the burn wound with the possibility of a pedicle graft in the future (none was performed).

Thermal injuries are relatively uncommon in animals. The potential source of burns to pet animals is almost entirely man-made, hence an unnatural type of traumatic injury. However, the pathophysiology and treatment of burns in pets is every bit as complex as that of humans. In reviewing literature for this paper, I found many parallels between human and veterinary burn therapy.

Thermal injuries can occur in a variety of forms:

Direct heat: common accidents of this nature are a cat walking across a heated stove burner, dogs licking a hot grill, or animals left unprotected on heating pads or electric blankets (i.e., post-surgery patients).

Flame: House fires are a common cause of direct flame injuries. Sadistic torture of animals is becoming, unfortunately, a growing source.

Scald: This is often the result of hot water spilled on pets near a stove in the kitchen. Scalding is a common method of experimentally producing burns for study.

Friction: This type of thermal injury is especially associated with hit-by-car patients which dragged across pavement or asphalt.

Caustic chemicals: Household compounds are the usual source.

Electrical: Young puppies chewing on cords represent the most common case of electrical burns.

Lightning: Unfortunately, this is usually fatal.

Burns are classified according to the extent and severity of the lesion. First degree burns damage the epithelium resulting in erythema, mild swelling, and pain to the touch. Treatment is minimal and healing usually occurs rapidly with no tissue distortion. Second degree burns affect the entire epidermis and variable parts of the dermis. Blistering occurs as well as considerable pain and swelling. If wound sepsis can be avoided, second degree burns usually heal in two to four weeks with minimal tissue distortion. Third degree burns are the most serious and life threatening. Full thickness of the skin is

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completely destroyed, with varying amounts of subcutaneous damage. Skin glands and hair follicles are lost, and the eschar eventually sloughs, leaving an ulcer. Infection commonly occurs and healing is often slow, with permanent disfigurement. Pain sensation is gone.

The gravity of the situation and the degree of therapy which will be required are related to the type of burn and the amount of area burned. First degree burns are considered to be minor and require only minimal topical treatment. Second and third degree burns are also considered minor when involving less than 10% of the body. Combinations of second and third degree burns covering more than 20% of the animal's body are considered to be major and require intensive emergency care and long term treatment and management. It is the objective of this paper to discuss the treatment and management of major burns.

Although burns are relatively uncommon in animals, few injuries cause more damage to tissue and metabolism. Distinct changes in the integrity of the skin, and the physiology of the entire body take place following severe burns. Following will be a description of the pathophysiology of thermal injury and treatment which can be instituted to save the animal's life and reduce physical distortion of the wound.

Pathophysiology of Severe Burns

The degree of pathologic alterations to the skin and physiology is related to the severity of the burn. Immediately following a severe burn, the effects of epinephrine and the sympathetic nervous system cause an increase in arterial pressure and a slight increase in heart rate. This is followed by a period of dilatation of local and regional vasculature, as well as an absolute decrease in cardiac output. This decrease in cardiac output is felt to be related to the release of "burn factor" by the affected skin, which is a myocardial depressor. This "burn factor" has been shown experimentally to decrease contractility of isolated papillary muscle. The sudden drop in cardiac output may be as high as 50% normal resting values, and when combined with the characteristic drop in plasma volume (discussed later), can add up to a loss of 80% normal resting cardiac output. The heart, at this stage, is refractory to pressor drugs so that in animals which eventually die, cardiac output continually declines, whereas in animals that will live, the output will gradually return to normal in 24-36 hours. A study by Ferguson and associates with scalded guinea pigs showed that in the first 75 minutes post-burn, cardiac output decreased up to 58%. The skin received a reduced fraction of the normal cardiac output and there was an absolute decrease in perfusion to almost all organs.

Following severe burn, blood cells passing in the vicinity of the burn sludge. This results in a decrease of venous return to the heart, and coupled with the already compromised cardiac output, results in endothelial anoxia. This effect of anoxia, plus histamine, serotonin, and catecholamine release cause an increase in capillary permeability. This change in capillary permeability allows a severe loss of proteins from the vascular compartment to the interstitial space, and with it the oncotic pressure necessary to maintain adequate plasma volume. The major plasma protein lost is albumin—up to two times the total plasma pool in four days. Along with its oncotic contribution, the transport ability of albumin is lost as it leaves the intravascular compartment. Other proteins lost from plasma are fibrinogen and clotting factors. Continued permeability of the capillary membranes and free movement of proteins across them make accordance to Starling's Law impossible and the outcome is a severe hypovolemia. Combined with the already decreased cardiac output, the net effect is inadequate tissue perfusion in almost all tissues and profound shock. As discussed later, the integrity of capillary permeability begins to return after about 24 hours and this has an important influence on the type of fluid therapy at that time.

The increase of plasma proteins in the interstitium results in a flux of fluids from the vascular compartment. Pressure in the tissue follows this swelling and edema, obstructing veins and lymphatics. Eventual thrombosis of these vessels occurs, producing more edema and extravasation of formed elements of blood and lymph. Dermal ischemia is the end result.

It takes about 3 weeks for much circulation to return to the burn wound, and most of this is only in granulation tissue at the burned/unburned tissue interface.

A number of changes occur in blood cells following severe burn. Damages in red blood cell membranes result in sequestration and hemolysis. The amount of hemolysis is variable but usually less than 10% (most of the decrease in plasma volume is related to plasma protein loss). An indication of ongoing hemolysis is the presence of spherocytes. Hemoglobinuria and hemoglobinemia are also evident soon after the burn as well as an increase of urobilinogen excretion. A negative nitrogen balance during early convalescence depresses hemoglobin formation so that about 8 weeks are required to reach normal hematocrits again. White blood cell values may reach 36,000 in less than 12 hours, with a neutrophilia, lymphopenia, and eosinopenia. The neutrophilia will persist through convalescence and normal values are present again in about 6 weeks. Immediately after the burn, a marked eosinopenia exists; eosinophil counts should start to rise again by the third day or a grave prognosis is indicated. Another phenomenon which has been noted is the decreased immunocompetence of neutrophils in burn victims. This presents an important problem as sepsis is often the lethal factor in burns.

Renal function is usually a victim of shock associated with severe burns. Thermal trauma causes a reflex constriction of renal vessels, and, coupled with hypovolemia and decreased cardiac outout, results in tubular damage.

A non-specific hepatitis may sometimes occur in burn victims. The hepatocellular damage is due to anoxia from a combination of decreased cardiac output, increased blood viscosity, and splanchnic vasoconstriction.

Pneumonitis is often a complicating factor in post-burn situations. The alveolar macrophage fails to increase production, permitting the harboring and growth of septicemic invaders. The most common invader in burns is *Pseudomonas* which causes marked destruction and permeability of the air-blood barrier. Since the monocyte is the precursor to the alveolar macrophage, a defect of monocyte production, release, distribution or migration is suspected. Studies have shown a definite depression of phagocytic activity of the reticuloendothelial system in major burn cases.

Physical destruction of a large area of skin creates serious problems in the animal's ability to retain body heat. Burning destroys a lipid in the skin, resulting in transmission of water four times normal (this is, of course, proportional to the depth and extent of the wound). This results in a considerable amount of heat lost from evaporation. This heat loss is in part responsible for an increase in oxygen consumption and metabolic rate as the victim tries to generate heat. Protein catabolism and depletion of fat stores are two means by which metabolic compensation is achieved. Since this negative nitrogen balance may exist for some time with continuous protein catabolism, the condition of the patient at the onset of the burn has much to do with the prognosis. In any case, metabolic compensation results in a severe weight loss and retarded wound healing.

Burn wound sepsis is the most common complication following burns. Septicemia is the most common cause of post-burn death. *Pseudomonas aeruginosa,* staphylococcus, streptococcus, and micrococcus are common isolates from burn wounds. *Candida* infections occur in wounds involving the oral cavity. Burn tissue is a rich media for growth of bacteria, especially *Pseudomonas aeruginosa* which is an opportunist type pathogen. In a full thickness burn, the presence of bacteria causes thrombosis and tissue death. This is especially evident in burns less than full thickness where circulation can be restored as early as 48 hours in the absence of sepsis. Gamma globulin catabolism is greatly increased in sepsis. If the infection breaks through first line defense at the wound, dissemination often occurs to the lungs where *Pseudomonas* may be harbored for a long time due to impaired alveolar macrophage function. Impaired neutrophil chemotaxis as well as decreased phagocytic activity of the reticuloendothelial system further promote the likelihood of sepsis.

Management of Burn Patients

Major burns result in not only severe tissue trauma, but also a series of pathophysiological events not encountered in other kinds of injuries. Therefore, early and aggressive therapy as well as long term management is mandatory if the animal is to survive. The goals of severe burn therapy are to save the animal's life, relieve pain, early wound closure, and to minimize deformities. The problems to be dealt with are multiple: shock,

sepsis, immunoincompetence, metabolic changes, malnutrition and a host of other organ complications.

The shock associated with burn has already been described. Decreased vascular tone, cardiac output, and an absolute loss of plasma volume indicate the need for immediate fluid therapy. But even before fluids are administered, careful evaluation of the respiratory system should be made if steam or smoke inhalation is suspected. Severe edema of the larynx and respiratory epithelium may occur, requiring intubation and oxygen administration. If infection is a threat, systemic gentamycin will be required. High levels of corticosteroids are also indicated to relieve edema of the respiratory passages. Alleviation of pain is usually required with morphine or one of its derivatives.

In the first 24 hours, fluid therapy is aimed at keeping up with the loss of fluids from injured tissues. The appropriate fluid for replacement during the first 24 hours is lactated Ringer's. This preparation restores electrolytes lost in the translocation of intravascular fluids. The amount of lactated Ringer's administered is calculated as follows; 4 ml/kg/% body burned. One half of this should be administered the first 8 hours and one fourth for each of the next 8 hours. Colloid containing fluids aren't very useful in the first 24 hours since capillary permeability is increased and the proteins are lost anyway. An interesting comparison was made between American and British fluid therapy in the first 24 hours post burn: British doctors gave colloids at a slower rate resulting in reduced cardiac output and greater instance of renal shutdown. Renal function at this time is critical and a balanced electrolyte solution maintains it best. Blood transfusions are also contraindicated during the first 24 hours as it will be extravasated, and albumin escaping into interstitial tissues will result in increased fibrosis during healing.

The strategy of fluid therapy in the second 24 hour period changes. The integrity of the capillary is improved and proteins are retained better intravascularly. Colloid containing fluids are administered to increase the plasma volume, cardiac output, and tissue perfusion. Less fluids should be needed at this time as Starling's Law is abided by again with the increase of intravascular oncotic pressure. Whole blood transfusions are useful also now, to partially restore lost colloids, increase oxygenation, dilute intravascular toxins, and enhance the repair of damaged vascularity. Electrolyte solutions in the second 24 hour period tend to aggravate the existing tissue edema and should be restricted. Oral water should be withheld the first few days due to risk of dilutional hyponatremia and aspiration into the lungs. Again, renal function is imperative during fluid therapy. If anuria persists, I.V. administration of isotonic sodium sulfate solution has been shown to be the most effective agent with which to reestablish renal function. Once diuresis has started, potassium excretion may be excessive so it may be necessary to supplement this ion in oral or parenteral fluids in the form of potassium phosphate or potassium chloride.

After the patient has been stabilized, the next major concern is control of sepsis. Post-burn sepsis is the leading cause of post-burn death. The objectives of sepsis control are early wound closure, topical antibiotics, and a clean environment. *Pseudomonas aeruginosa* is an opportunistic type of bacteria which commonly invades burn wounds. A Wood's light can be useful in detecting colonies of *Pseudomonas,* but culture and sensitivity is the preferred method. Proper cleansing and debridement of the wound is necessary for maximum efficacy of topical treatments. If the patient is in extreme pain, morphine sulfate or methadone hydrochloride should be administered. The wound is wet with saline and as much devitalized tissue removed as possible. If there is gross contamination, the wound can be sponged gently with a mild detergent. No hexachloraphene should be used as it will be rapidly absorbed systemically by the wound and produce a neurotoxicity. A useful method of debridement which was used on deep wounds of battle victims in Vietnam is the use of a mild Betadine solution pressurized by a Water Pic unit. Between dressing changes, hydrotherapy in a mild warm Betadine keeps the wound clean and promotes healing. Gauze dressing covered by a stockinette is almost essential to keep the topical ointment on the wound and prevent contamination.

A variety of topical ointments are available in the treatment of burns. A requirement of any topical ointment is that it be water

miscible; oils and greasy salves cause tissue maceration and delay healing. Silver sulfadiazine is considered to be an effective antimicrobial preparation for topical use. The silver ion is, in itself, quite bactericidal and in combination with sulfadiazine, prevents inhibition by paramino benzoic acid. Nitrofurazone also works well as a topical ointment. Silver nitrate solution has been used for a long time in human burn patients. A 0.5% solution of silver nitrate solution is applied through 5 cm thick gauze bandages. The silver ion is the bactericidal component, but upon reaction with chloride in burned tissues, precipitates and loses its effectiveness. The burn must be continually debrided and the silver nitrate solution reapplied. This constant application of solution causes leaching of ions from body tissues so I.V. electrolytes must be supplemented. Mafenide (Sulfamylon) in a 10% cream has a wide spectrum and deep wound penetration. It must be applied twice daily and causes considerable discomfort to the patient. Mafenide is not antifungal and is a carbonic anhydrase inhibitor resulting in hyperpnea and respiratory alkalosis. Garamycin (gentamycin) cream 1% is quite effective against most invaders however it is quite expensive. It would be a natural impulse to treat the animal prophylactically with systemic antibiotics. However, this has been found to be ineffective, and sometimes detrimental in burn cases. Treatment prophylactically may produce extremely resistent populations (especially in the case of gentamycin). It is perhaps best to treat as vigorously as possible at the local level, and reserve systemic therapy for use in cases of protracted infection. A useful technique for treatment at the local level where topical preparations have failed is to infiltrate the burn wound or area under the eschar with an antibiotic solution. Culture and sensitivity is performed, and the maximum daily dose of an appropriate antibiotic diluted with saline. This is injected under the burn eschar. Research has been done using specific antigens so that in the future, severe burn victims may be immunized against *Pseudomonas.*

After stabilization of the patient and administration of adequate sepsis control, the next step in the treatment of major burns is wound closure. Since large areas of tissue are usually destroyed, the only truly effective physiological closure is grafting. Early grafts decrease evaporative losses and pain, and increase phagocytosis of bacteria, stimulation of epithelial islands and granulation tissue, and increase the rate of healing. A number of types of grafts and grafting techniques have been used in human medicine and are now being used in animal burn therapy. Autografting utilizes the animal's own skin in closure of the defect. This graft is to be permanent, and is performed either soon after injury or later in the course of healing when a bed of granulation tissue has formed. In either case, the wound should be thoroughly debrided and bacterial infection under control before any grafting procedures. The most common graft used in veterinary medicine is the sliding pedicle graft. For adequate blood supply to be maintained to the pedicle, the length must not exceed three times the width. Another kind of autograft which promotes rapid wound closure is the mesh graft. Skin adjacent to the wound is cut with a special instrument to make many small parallel slits. The skin is then pulled across the wound easily due to greater expansibility from the many slits. The epithelium proliferates from all edges and rapid wound closure is achieved in 5 to 7 days. The punch graft works in a similar fashion to the mesh graft. Small patches of the animal's own skin are transplanted into the wound site and each serves as an island of reepithelialization. Hair follicles migrate to a degree into the new epithelium. Due to debilitation by the burn wound, autografts performed soon after the injury often are slow in taking.

Other kinds of grafts can be used as physiological dressings in temporary wound closure. The homograft is skin which is obtained from another individual of the same species. It is held in place with bandages and is changed every 2-5 days. In humans these grafts used to be left on until rejection (about 10 days), but sepsis was more common and the patient often ill in comparison to cases where more frequent changes were made. The heterograft is used in a fashion similar to the homograft. The source is an animal of a different species. Pig skin is commonly used and commercially available in fresh, frozen, or lyophilized form. These skin preparations are almost as effective as homografts and can

be left on 5 days between changes. Research with rats has shown that using a dressing made from edible bovine corium enhances wound closure. This microcrystalline collagen is packed into the open wound and held in place with a gauze bandage, speeding healing 2-4 days over conventional dressings. The most significant gain was in the immediate post-burn period when fibroblasts were invading the area. Microcollagen dressings, if made available on a practical scale, could be a useful new kind of temporary graft to promote healing of the burn wound in preparation for later, more permanent grafts. Again, during the entire period that therapy is performed, asepsis should be a rule adhered to as much as possible. The animal is in a state of compromised immunocompetence and the finest plastic surgery technics will be wasted if a septicemia ensues. The environment where the patient is kept should have the same amount of attention in cleaning as the burn wound itself.

As mentioned before, leakage of fluids through the burn wound places a stress on normal metabolism through loss of heat by evaporation. The loss of proteins from the intravascular compartment also represents a potential nutritional debt as these colloids are replaced. The body's means of meeting the demands of hypermetabolism are protein catabolism and depletion of fat stores, resulting in emaciation and a negative nitrogen balance. The entire process of wound healing is greatly retarded. Obviously, these caloric and nitrogen demands exceed the amounts gained from a normal diet. It is recommended that the total calorie content in the diet of severe burn patients be increased 2 to 4 times, and the nitrogen content 3 to 5 times. The preferred route is orally. Such products as Nutri-cal offer a concentrated source of these nutrients to meet the increased nutritional plane. Plastic wrap around bandages can cut evaporation.

As more experimentation is performed with burns, more potentially useful knowledge is being gained. Recent studies with rats showed that serum harvested from convalescent burn rats and injected into newly scalded rats promoted serum detoxification, diminished proteolytic enzyme activity, and decreased morphological disturbance. Another group studying levels of the components of complement following severe burn found that serum levels of factors 3-9, factor B, properdin, and properdin convertase were deficient. All of these factors except properdin and properdin convertase soon return to normal. It was found that supplementing properdin and properdin convertase restored opsonic activity of the complement system significantly. Studies with topical treatments showed that by using inhibitors of proteolytic enzymes, prevention of secondary necrosis in deep burns could be achieved. Even more applicable to the clinician treating burn shock is the finding that treating burns with topical steroids maintained adequate capillary perfusion to prevent dermal ischemia in many cases, without appreciably suppressing the immune system and allowing bacterial invasion.

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Another Frontier: Veterinary Herpetology

Kimo Jow*

When I was a growing youngster, I often had "unusual" house pets—the "ant lion," Family Myrmeleóntidae (which I commonly called the 'doodle bug'), tadpoles, lizards and fuzzy caterpillars of all types. My favorite, though others had regarded it as repulsive, was the "horned toad," *Phrynosoma* sp. While they were fun to keep, my parents made sure they were outside housepets.

Today, there continues to be youngsters, as well as many adults, who as amateurs collect reptiles and amphibians. This constitutes an increasing number of people visiting their local veterinarian about a disease or injury of their herptile.

Outside of the zoological parks, professional veterinary care to herptiles has been limited—often from lack of understanding or interest. However, a tremendous amount of information has been compiled in the recent years, particularly by the veterinary profession, in efforts to improve the care and health of reptiles. A valuable number of materials and resources are becoming more available by experienced professional and lay individuals on herpetology.

As a measure of further interests locally, there is the Iowa Herpetological Society (IHS). With objectives of education and conservation of herptiles, their activities include workshops, public displays and various publications. IHS membership is from all ages and walks of life, and is of benefit to both the collector and the practitioner, since the importance of veterinary care is stressed and worthwhile information about herpetology is provided.

As sophisticated as modern veterinary practice is today, there can still be a great sense of satisfaction and accomplishment in treating the scaley creatures of this world. Though a lot still remains to be learned of such animals' husbandry and medicine, the application of a veterinarian's basic medical knowledge and training is the first step in treating herptiles. Hopefully in the near future we can look toward improved veterinary care for these animals. This is in keeping with the Veterinarian's Oath, ". . . a lifelong obligation (to) the continual improvement of (our) professional knowledge and competence."

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