13 Burn Injuries

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Introduction

Burns are uncommon in horses, with the majority resulting from barn fires. Thermal injuries may also result from contact with hot solutions; electrocution or lightning strike; friction as in rope burns, abrasions, and radiation therapy; and chemicals such as improperly employed topical drugs or maliciously applied caustic agents.[1,2]

Most burns are superficial, easily managed, inexpensive to treat, and heal in a short time. Serious burns, however, can result in rapid, severe burn shock or hypovolemia with associated cardiovascular changes. Smoke inhalation and corneal ulceration are also of great concern.[1,2] Management of severe and extensive burns is difficult, expensive, and time consuming. The large surface area of the burn dramatically increases the potential for loss of fluids, electrolytes, and calories. Burns covering up to 50% or more of the body are usually fatal, although the depth of the burn also influences mortality. Massive wound infection is almost impossible to prevent because of the difficulty of maintaining a sterile wound environment. Long-term care is required to prevent continued trauma, because burn wounds are often pruritic and self-mutilation is common. Burned horses are frequently disfigured, preventing them from returning to full function. Therefore, it is imperative that prior to treatment the patient be carefully examined, with particular attention paid to cardiovascular function, pulmonary status, ocular lesions, and the extent and severity of the burns. Cost of treatment and prognosis should be thoroughly discussed with the owner.[1–4]

This chapter will review the classification and mechanism of burn injury. The pathophysiology of burn injury and pulmonary injury will be discussed as they relate to the physical exam findings and appropriate treatment for immediate disorders as well as long-term wound care. Complications and nutritional needs will also be addressed.

Classification of Burns

Burns are classified according to the depth of the injury.[1–3] First degree burns involve only the most superficial layers of the epidermis. These burns are painful and characterized by erythema, edema, and desquamation

of the superficial layers of the skin. The germinal layer of the epidermis is spared and the burns heal without complication (Figure 13.1).[4]

Second degree burns involve the epidermis and can be superficial or deep. Superficial second degree burns involve the stratum corneum, stratum granulosum, and a few cells of the basal layer. Tactile and pain receptors remain intact. Because the basal layers of the epidermis remain relatively uninjured, superficial second degree burns heal rapidly, within 14–17 days with minimal scarring (Figure 13.2).[5] Deep second degree burns involve all layers of the epidermis, including the basal layers. These burns are characterized by erythema and edema at the epidermal-dermal junction, necrosis of the epidermis, accumulation of white blood cells at the base of the burn zone, and eschar (slough produced by a thermal burn) formation, and they cause minimal pain (Figure

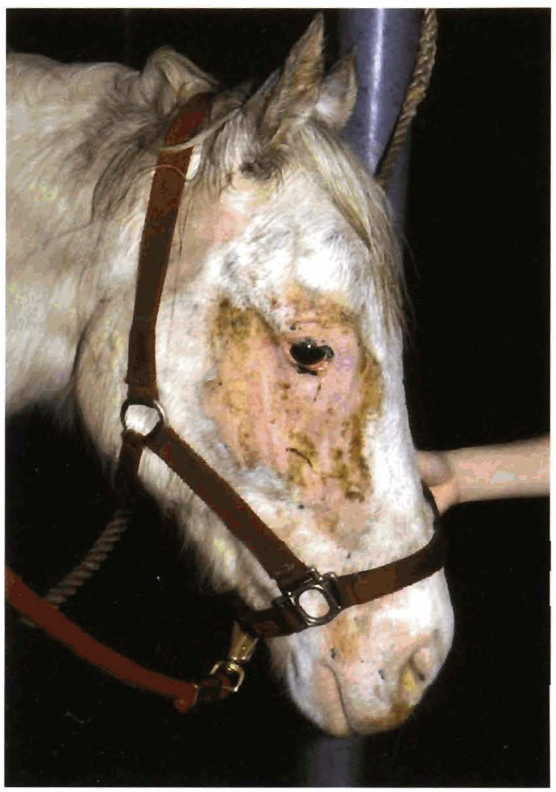

Figure 13.1. First degree burn of the right facial and periocular regions. This type of burn involves only the most superficial layers of the epidermis. These burns are painful and characterized by erythema, edema, and desquamation of the superficial layers of the skin. The germinal layer of the epidermis is spared and the burns heal without complication. Reprinted from Veterinary clinics of north america, Vol 21, R. Reid Hanson, Management of burn injuries, p. 106, (2005), with permission from Elsevier.

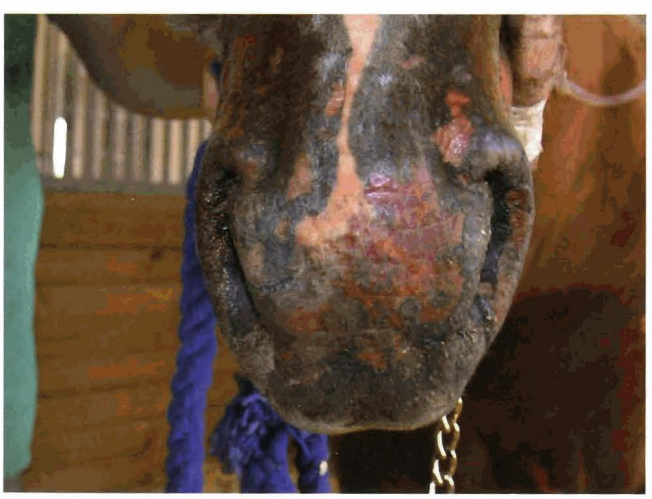

Figure 13.2. Superficial second degree burn of the nose. Tactile and pain receptors remain intact. Because the basal layers of the epidermis remain relatively uninjured, superficial second-degree burns heal rapidly, within 14–17 days with minimal scarring.

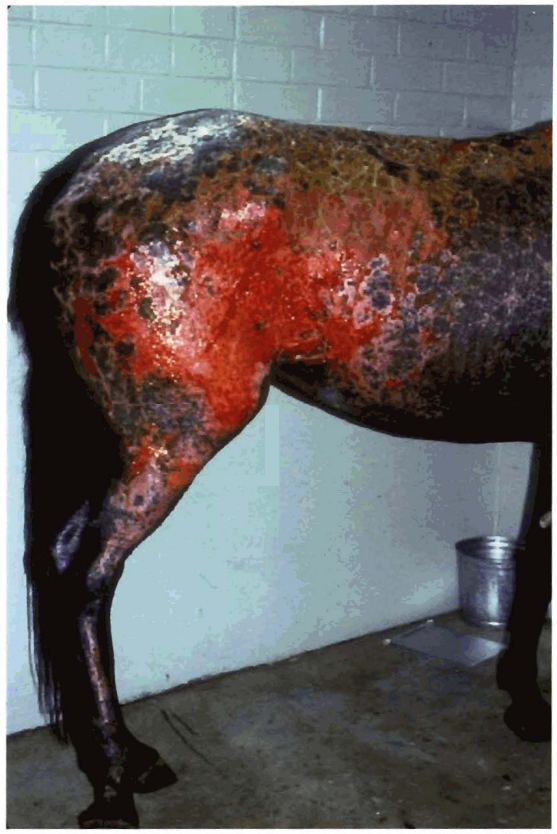

Figure 13.3. Deep second degree burn of the right dorsum and right hind limb. Deep second degree wounds may heal spontaneously in 3 to 4 weeks if care is taken to prevent further dermal ischemia that may lead to full-thickness necrosis. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 107, (2005), with permission from Elsevier.

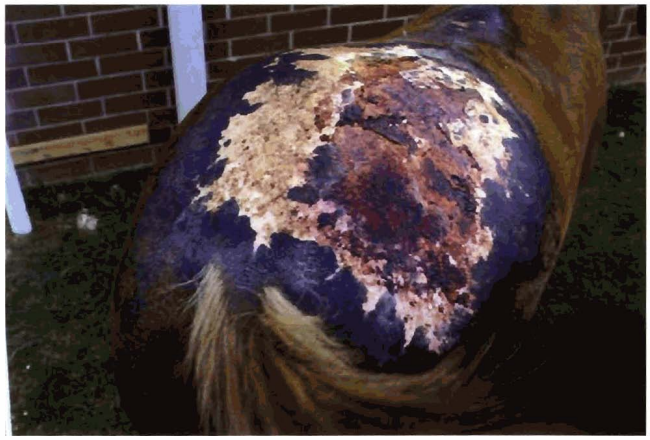

Figure 13.4. Third degree burn of the dorsal gluteal region incurred during a barn fire due to hot asphalt roof shingles falling on the horse. The central burn area is surrounded by deep and superficial second degree burns. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 108, (2005), with permission from Elsevier.

13.3).[1,3] The only germinal cells spared are those within the ducts of sweat glands and hair follicles. Deep second degree burn wounds may heal spontaneously in 3 to 4 weeks if care is taken to prevent further dermal ischemia that may lead to full-thickness necrosis. In general, deep second degree burn wounds, unless grafted, heal with extensive scarring.[5]

Third degree burns are characterized by loss of the epidermal and dermal components, including the adnexa. The wounds range in color from white to black (Figure 13.4). There is fluid loss and a marked cellular response at the margins and deeper tissue, eschar formation, lack of pain, shock, wound infection, and possible bacteremia and septicemia. Healing occurs by contraction and epithelialization from the wound margins or acceptance of an autograft. These burns are frequently complicated by infection. Fourth degree burns involve all of the skin and underlying muscle, bone, ligaments, fat, and fascia (Figure 13.5).[6]

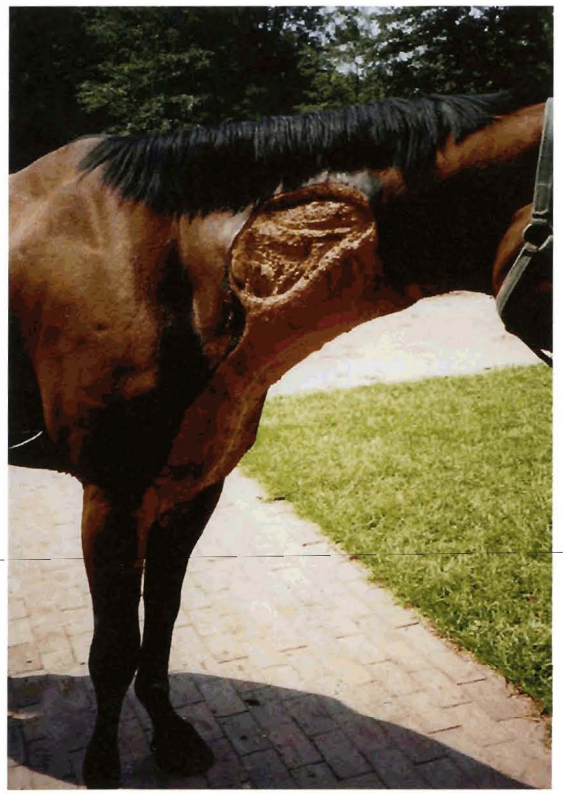

Figure 13.5. Fourth degree burn of the right cervical region and pectoral area due to acid. Fourth degree burns involve all of the skin and underlying muscle, bone, ligaments, fat, and fascia. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 108, (2005), with permission from Elsevier.

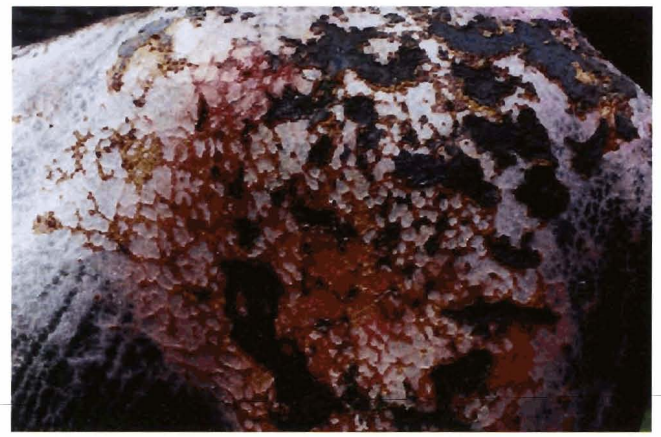

Figure 13.6. Deep second degree burn of the left hind limb. The central burn area is surrounded by less severe skin burns, illustrating the dissipating radiating effects of the heat and damage to the skin. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 109, (2005), with permission from Elsevier.

Mechanism of Burn Injury

The extent of tissue destruction depends upon the temperature of the heat source, duration of exposure, blood supply, and local environment of the wound.[6] At onset there are three levels of injury: the central zone of coagulation, the intermediate zone of vascular stasis, and the outer zone of hyperemia. The central zone of coagulation corresponds to the area that was closest to the heat source. At temperatures >45°C, protein denaturation exceeds the capacity for cellular repair and cell death ensues. The severity of injury decreases radially from this center as heat is dissipated (Figure 13.6).

Adjacent to the coagulation core is an intermediate zone of vascular stasis caused as dermal vessels thrombose during the initial 24 hours following injury. The damaged cells in this zone will survive only under ideal

circumstances. The use of heparin and thromboxane inhibitors may moderate the amount of tissue damage in this zone by limiting the number of thromboses developing in the affected area.[7] The outer region is the zone of hyperemia where epidermis is lost but the dermis remains intact and cellular recovery is rapid.

In humans, the total body surface area affected and the depth of the injury correlate highly with the degree of morbidity and mortality associated with thermal injury. New blood markers, such as serum cholinesterase and inflammatory cytokines, have been introduced to assist in the prognosis of morbidity and mortality, beyond the traditional vital signs. At this time, however, these are available only for research purposes.[8]

Pathophysiology

Burn Injury

Following severe burns there is a dramatic cardiovascular effect termed burn shock, which resembles hypovolemic shock. A dramatic increase in local and systemic capillary permeability occurs in response to heat and the release of cytokines, prostaglandins, nitric oxide, vasoactive leukotrienes, serotonin, histamine, and oxygen radicals.[9] Local tissue damage results from massive protein coagulation and cellular death. In the immediate area of the burn, arteries and venules constrict and capillary beds dilate. Capillary wall permeability is increased in response to vasoactive amines released as a result of tissue damage and inflammation. These vascular responses result in fluid, protein, and inflammatory cells accumulating in the wound; ensuing vascular sludging, thrombosis, and dermal ischemia lead to further tissue damage. Tissue ischemia continues for 24–48 hours after injury and is caused by the local release of thromboxane A_2.

Before any change in blood or plasma volume, there is a dramatic drop in cardiac output due to circulating levels of myocardial depressant factors. Fluid loss into the extravascular space leads to an acute reduction in blood volume. With reductions in blood volume and cardiac output, peripheral and pulmonary vascular resistance increase, peripheral tissue perfusion decreases, and organ failure ensues.[9]

The extent of fluid loss parallels the severity of the burn. Fluid losses result in increased heat loss from evaporation and an increased metabolic rate. The heat loss is in part responsible for increased oxygen consumption and metabolic rate as the horse tries to generate heat. Depletion of fat stores and some endogenous protein supplies are two means whereby metabolic compensation is achieved. In turn, this hyper-metabolic rate leads to weight loss, a negative nitrogen balance, and delayed wound healing. Thus, the nutritional condition of the patient prior to injury is a prime prognostic consideration.

In burn injury the vascular compartment remains permeable to proteins up to 15 nm in size, including albumin.[10] With moderate thermal injury, up to 2 times the total plasma albumin pool can be lost from the vascular compartment. Loss occurs both through the open wound and into the extravascular space. Protein concentration can reach 3 g/dl in the extracellular fluid, which is sufficient to cause large fluid shifts because of differences in osmotic pressure.[11] The resultant burn edema is clinically recognized within 60 minutes of injury.[12]

Electrolyte disturbances accompany the fluid and protein shifts. Immediately after a burn, hyperkalemia may occur because of cellular disruption and potassium leakage. When counterbalanced by increased mineralocorticoid secretion, the urinary sodium-potassium ratio is reversed and a subsequent potassium deficit may develop 3 to 5 days after injury.[13] Simultaneously, hypernatremia may result as sodium is reabsorbed after restoration of vascular membranes. Protein concentrations, circulatory volume, and electrolyte concentrations should be measured frequently during replacement therapy.[13]

Anemia is not usually a significant concern immediately following a burn. Anemia can, however, become a problem in patients with burns exceeding 30% of the total body surface area. Early anemia resulting from red cell hemolysis and splenic sequestration may be present but is often masked by hemoconcentration. Initial anemia is caused by the immediate destruction of red blood cells by heat and wound hemorrhage. Subsequently, erythrocyte loss occurs from both intravascular and extravascular removal of damaged cells, as well as during eschar removal. Thrombocytopenia may result from platelet aggregation on damaged capillary endothelium. If damage is extensive, a hemorrhagic diathesis may result from exhaustion of clotting factors.

Immunoglobulin levels in the serum drop; the lowest values are reached 2 days following a burn. Serious defects in neutrophil function such as inefficient chemotaxis, impaired phagocytic rate, and bactericidal capacity have also been observed in severely burned horses. Their combined effect leads to a compromised host that is prone to infection.[14] This translates clinically to the fact that most deaths in severely burned human patients are

due to burn wound sepsis; burn patients are also at risk for developing sepsis secondary to pneumonia, catheter-related infections, and suppurative thrombophlebitis.[15]

Host metabolic rate increases in a curvilinear fashion in proportion to the size of the thermal injury exceeding 10% of the total body surface area. This causes a 1°–2°C increase in core body temperature and increases in oxygen consumption, fat degradation, and protein and glucose utilization.[16] Caloric expenditure and protein catabolism are greater in burn injury than in any other physiologic stress state. In patients with burns exceeding 30% of the total body surface area, energy expenditure doubles and fuel substrates are metabolized at 2 to 3 times the normal rate.[16] Caloric and protein intakes must be rapidly adjusted to avoid the rapid depletion of skeletal muscle, delayed wound healing, and impaired cellular defense mechanisms.[16,17] Environmental temperatures should be kept between 28° and 33°C to minimize the metabolic expenditure required to maintain the elevated core temperatures.[16,17]

Inhalation Injury

Inhalation injury is a common sequella of closed-space fires. It develops through three mechanisms: direct thermal injury, carbon monoxide poisoning, and chemical insult. Direct thermal injury causes edema and obstruction of the upper airway, but because of the efficient heat exchange capacity of the nasopharynx and oropharynx, superheated air is cooled prior to entering the lower respiratory tract.

Carbon monoxide interferes with oxygen delivery in several ways.[18] It has a 230–270 times greater affinity for oxygen, thus shifting the oxygen-hemoglobin curve to the left. The resultant carboxyhemoglobin is incapable of oxygen transport. Carbon monoxide also binds to myoglobin, thereby impairing oxygen transport to muscles.[18] Carbon monoxide is excreted by the lungs at a rate related to ambient oxygen tensions. In room air, carbon monoxide has a half-life of 3 to 4 hours. An increase in oxygen tensions promotes the dissociation of carbon monoxide and hemoglobin; thus 100% oxygen therapy reduces the half-life to 30–40 minutes. Hyperbaric oxygen therapy at 2.5 atmospheres further decreases the half-life to 22 minutes.[19]

Chemical insult depends on the material that was burned.[20] Combustion products such as hydrogen cyanide, hydrochloric acid, phosgene, sulfuric acid, and aldehydes may induce severe tracheobronchitis when combined with the moisture in the airways. Initially, only erythema may be present, but chemical injury continues as long as chemical-covered carbon particles remain attached to the airway mucosa; particle size determines where damage will occur within the respiratory tree (Figure 13.7). Combustion products cause increased pressure within the pulmonary artery, peribronchial edema, mucosal sloughing, bronchoconstriction, decreased mucociliary transport, and bacterial clearance, as well as altered surfactant activity.[20] Significant pulmonary ventilation/perfusion mismatches may subsequently develop.[21]

Pulmonary infection is a potential complication of smoke inhalation. Alveolar macrophages, the primary cellular defenders in the lung, are increased in number after injury but display decreased phagocytic and bactericidal functions. Susceptibility to pulmonary infection, pulmonary edema, and lung dysfunction increases greatly in patients that also suffer cutaneous thermal injury. The relationship between inhalation and surface

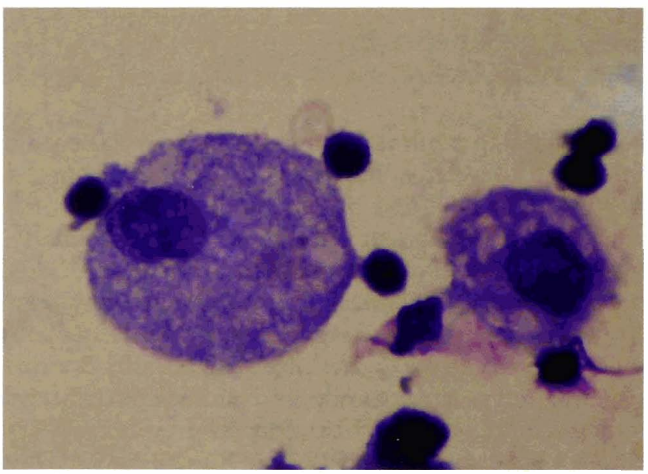

Figure 13.7. Carbon particles associated with alveolar macrophages in the bronchoalveolar lavage due to smoke inhalation injury. Chemical injury continues as long as chemical-covered carbon particles remain attached to the airway mucosa; the size of the particles determines where damage will occur within the respiratory tree. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 112, (2005), with permission from Elsevier.

burns is unclear but appears to be additive. Major cutaneous burns alone have been reported to cause pulmonary dysfunction in as many as 25% of patients, whereas inhalation injury increases the morbidity and mortality rates for a given cutaneous thermal injury.[22]

Exam Findings

Physical Exam Findings

Because heat is slow to dissipate from burn wounds, it is often difficult to accurately evaluate the amount of tissue damage in the early period following injury. The extent of the burn depends on the size of the area exposed, while the severity relates to the maximum temperature the tissue attains and the duration of overheating. This explains why skin injury often extends beyond the boundaries of the original burn.[23] A complete physical examination should be performed on any burned animal before the wound is evaluated. Only after the patient's condition is stable should the burn wound be assessed.

Physical criteria used to evaluate burns include erythema, edema, and pain; blister formation; eschar formation; presence of infection; body temperature; and cardiovascular status.[23] In general, erythema, edema, and pain are favorable signs because they indicate that some tissue is viable, although pain is not a reliable indicator of wound depth.[23] Often, time must elapse to allow progression of tissue changes in order for an accurate evaluation of burn severity to be made (Figures 13.8 and 13.9).

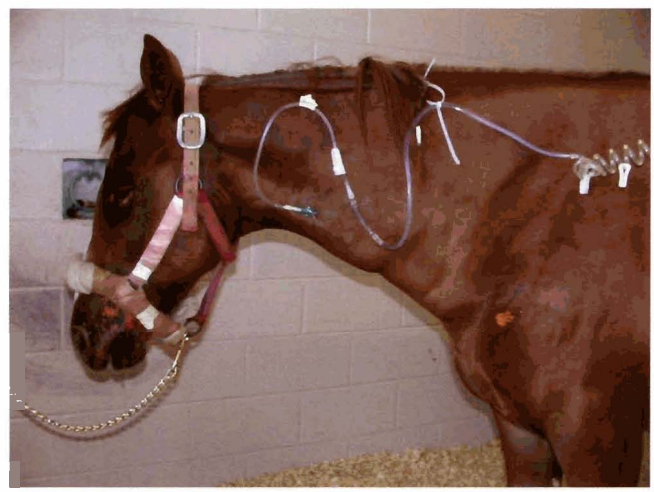

Figure 13.8. Severe burn edema along the ventral neck region 24 hours after injury due to a barn fire. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 114, (2005), with permission from Elsevier.

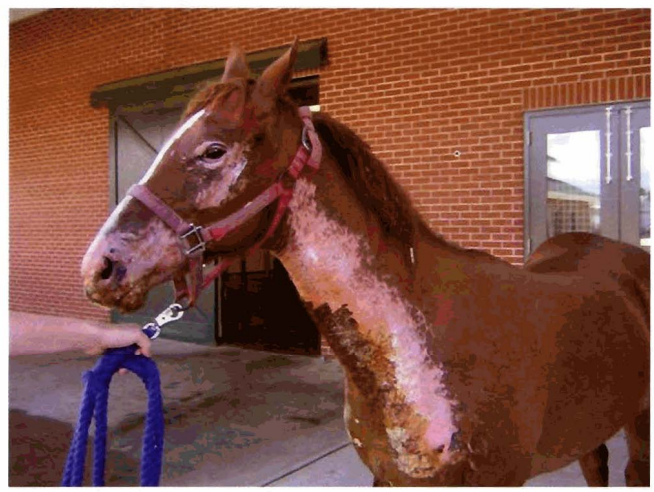

Figure 13.9. Same horse as in Figure 13.8. The extent of the burn is more evident after the skin has sloughed in response to latent thermal injury to the skin. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 114, (2005), with permission from Elsevier.

Burns are most commonly seen on the back and face. Erythema, pain, vesicles, and singed hair are present, depending on the extent of the injury (Figure 13.10). Increases in heart and respiratory rates accompany abnormal discoloration of mucous membranes. The burned horse may exhibit blepharospasm, epiphora, or both, which signify corneal damage (Figure 13.11). Coughing may indicate smoke inhalation while a fever signals or confirms a systemic response to injury.

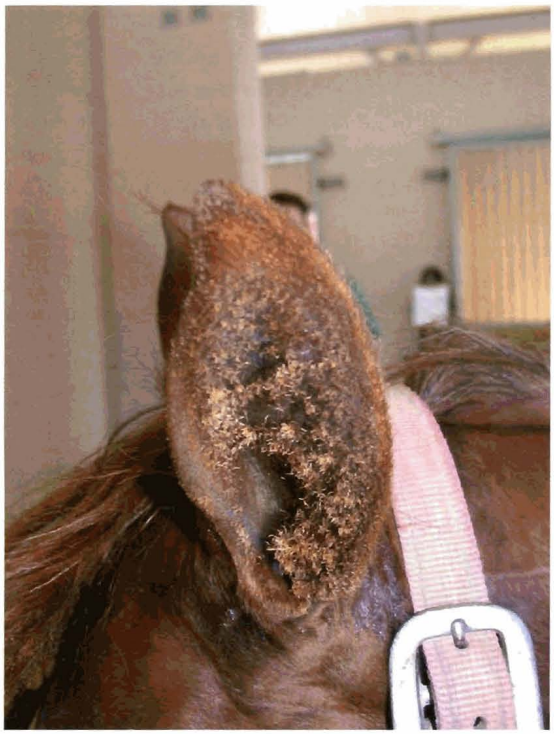

Figure 13.10. Singed hair due to heat generated from a barn fire.

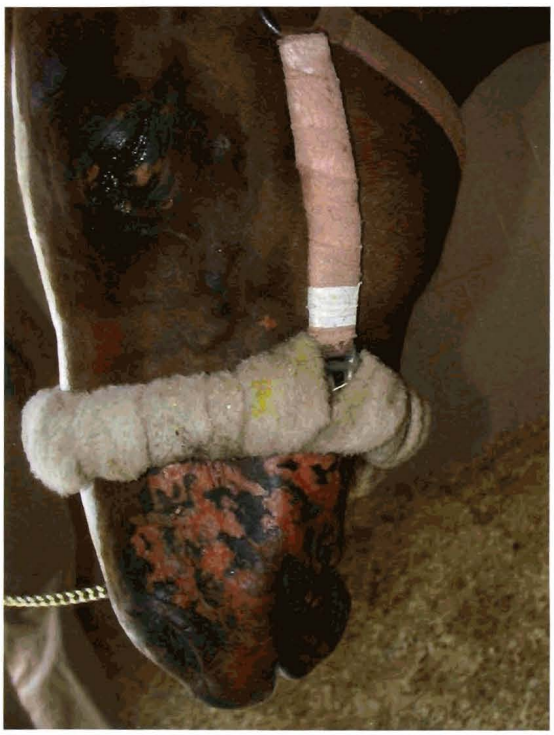

Figure 13.11. Blepharospasm, epiphora, and severe erythema with loss of epithelium of the muzzle due to a barn fire.

Laboratory Findings

Initial laboratory data including complete blood count, clotting profile, serum chemistry, urinalysis, arterial blood gas, and carbon monoxide concentration as well as chest radiographs and bronchoalveolar lavage are helpful in the preliminary evaluation.[6]

Laboratory findings may reveal a low total protein concentration with anemia that may be severe and steadily progressive. Hemoglobinuria may be detected. Hyperkalemia may be present initially but hypokalemia is more likely later in the course of the condition and is often associated with fluid therapy. Evidence of hemolysis or hemoglobinuria in a horse that has suffered cutaneous burns is indicative of severe burn trauma and potential complications such as pigment nephropathy and renal failure.[24]

Percentage Body Surface Area Burn Related to Prognosis

The percentage of total body surface area involved usually correlates with mortality, whereas the depth of the burn determines morbidity.[11] The rule of 9 is used in humans to estimate the total body surface area involved. This method allows an estimation of prognosis according to the extent of the burn. Each arm represents 9%, each leg 18%, the head with neck 9%, and the thorax and abdomen each 18% of body surface area.[4] Special care should be taken to identify injury to major vessels of the lower limbs and presence of eye, perineal, tendon sheath, and/or joint involvement.

Although specific guidelines do not exist for burned large animals, euthanasia should be recommended for those with deep partial-thickness to full-thickness burns involving 30%–50% of the total body surface area.[13,25] The availability of adequate treatment facilities, cost of treatment, and pain experienced by the horse during long-term care should be considered when deciding whether or not to treat. Because convalescence may take up to 2 years, euthanasia is often an acceptable alternative.[26]

Treatment

Burn Shock

With burn shock, large volumes of balanced electrolyte solution are generally chosen unless serum electrolyte analysis dictates otherwise. In patients with burns exceeding 15% of the total body surface area, intravenous fluid therapy is required to avoid circulatory collapse.[27,28] Inadequate fluid resuscitation results in decreased renal and gastrointestinal perfusion that could lead to gastrointestinal bacterial translocation and sepsis.[27,28] Administration of isotonic fluids at a rate of 2 ml–4 ml/kg for each percentage of surface area burned is recommended, but fluid resuscitation is best titrated to maintain stable and adequate blood pressure.[16] An alternative is to use hypertonic saline solution (4 ml/kg) with plasma, hetastarch, or both, followed by administration of additional isotonic fluids.

If there has been smoke or heat inhalation injury, crystalloid administration should be limited to the amount that normalizes circulatory volume and blood pressure. Continuation of the same rate of administration of electrolyte solutions following the resolution of burn shock leads to edema in excess of any improvement in cardiovascular dynamics.[28] Two to 10 liters of plasma is an effective albumin source as well as an exogenous source of antithrombin III against coagulopathies. Hydration, lung sounds, and cardiovascular status should be monitored carefully during fluid administration by clinical assessment and PCV/TP measurement.

Flunixin meglumine (0.25 mg–1 mg/kg IV q 12–24 h) is an effective analgesic. Cyclooxygenase (COX)-2 inhibitors are a new class of nonsteroidal anti-inflammatory drugs that selectively block the COX-2 enzyme, which impedes the production of prostaglandins that cause pain and swelling. Because the COX-2 enzyme does not play a role in the normal function of the stomach or intestinal tract, medications which selectively block COX-2 do not present the same risk of injuring the stomach or intestines. Firocoxib (0.1 mg/kg PO SID) is a newly developed COX-2 inhibitor for the horse. Although COX-2 inhibitors would seem beneficial in the management of burn patients, Firocoxib is currently approved only for use in musculoskeletal pain. Pentoxyfylline (8 mg/kg IV q 12 h) is used to improve the flow properties of blood by decreasing its viscosity. Administration of dimethylsulfoxide (DMSO) (1 g/kg IV) for the first 24 hours may decrease inflammation and pulmonary edema. If pulmonary edema is present and is unresponsive to DMSO and furosemide treatment, dexamethasone can be administered once at 0.5 mg/kg IV.

Pulmonary Injury

Maintenance of airway patency, adequate oxygenation and ventilation, as well as stabilization of hemodynamic status are the cornerstone of therapy for smoke inhalation injury. Intervention and respiratory support are essential, even before the diagnosis of respiratory injury is confirmed. Nasal or tracheal insufflation with humidified 100% oxygen counteracts the damaging effects of carbon monoxide and facilitates clearance by decreasing its half-life in the blood. Oxygen insufflation rates of 15–20 l/minute can be achieved through a tracheostomy and should be continued until the patient is able to autonomously maintain normal oxygenation. Humidification can relieve excessive airway drying or mucous plugging.

Nebulizing with N-acetyl cysteine and heparin and using humidified air will reduce the formation of pseudomembranous casts and aid in the clearance of airway secretions.[21,22] If there is respiratory distress with low arterial oxygen tensions, a tracheostomy is indicated to attempt to remove large obstructive pseudomembranous tracheobronchial casts that have formed.[29] Nebulized DMSO will help decrease lung fluid formation.[21,22] The beta adrenergic agonist albuterol can be aerosolized to reduce bronchospasm. DMSO and heparin may protect against airway damage caused by smoke.[30–33] Maintenance of optimal fluid status is essential. Patients with concurrent surface burns and inhalation injury require 2 ml per percentage burn per kg more fluid than those with cutaneous burns alone to support adequate cardiac and urine output.[34]

Antibiotics and corticosteroids do not influence survival rates and should not be routinely administered to smoke inhalation patients. Systemic antimicrobials are indicated only for proven infections, the incidence of which increases 2 to 3 days after smoke inhalation. Intramuscular penicillin is effective against oral contaminants colonizing the airway. Gentamicin was shown to improve hemodynamics in ovine septic shock after smoke inhalation injury.[35] If signs of respiratory disease worsen, a transtracheal aspirate should be submitted for culture and sensitivity testing, and the antibiotic regimen adapted accordingly.[30,34]

Patients with suspected significant smoke inhalation should be observed closely for several hours and hospitalized in the presence of extensive burns. Therapy must be adjusted according to the clinical response and the results of serial blood gas analyses, complete blood counts, chest radiographs, airway endoscopy, and cultures. Successful treatment depends on continuous patient reassessment as well as early and aggressive patient care.

Wound Care

First degree burns are generally not life-threatening and thus are simply managed. Topical therapy in the form of cool compresses, cold-water baths, and wound coverings may provide relief from pain. Additional pain control can be achieved with nonsteroidal anti-inflammatory drugs or narcotics.

Second degree burns are associated with vesicles and blisters. These vesicles should be left intact for the first 24–36 hours following formation, because blister fluid provides protection from infection and the presence of a blister is less painful than the denuded exposed surface. After this interval the blister is partially excised and an antibacterial dressing such as silver sulfadiazine (Silvadene, Par Pharmaceutical, Inc., Spring Valley, NY) is applied to the wounds while an eschar is allowed to form.[1,2,11]

Third degree burns can be difficult to manage. The patient's condition should be stabilized as rapidly as possible prior to undertaking wound management. Destruction of the dermis leaves a primary collagenous structure called an eschar. Dry exposure is a treatment method that respects the principle that bacteria do not thrive on a dry surface. The goals of therapy are to keep the wound dry and protected from mechanical trauma. Heat and water loss from the uncovered wound, however, are a disadvantage of this approach.

There are several methods to treat burn wounds in the horse, and the choice depends on the extent and location of the injury. Full-thickness burns can be managed by occlusive dressings (closed technique), continuous wet dressings (semi-open technique), eschar formation (exposed technique), or excision and grafting.[23]

The closed method relies on the use of occlusive artificial dressings. Wound cleansing and debridement are performed at each of the frequent dressing changes. Temporary dressings can, by adhering to the underlying wound bed, decrease the bacterial population, reduce heat and water loss, protect the bed of granulation tissue, and hasten wound healing. However, with large burns, frequent bandage changes and debridement can be painful, and extensive bandaging may not be feasible or affordable in some patients.[23]

With the semi-open method, the eschar is left in place but kept covered with an antimicrobial-soaked dressing such as silver sulfadiazine (Silvadene, Par Pharmaceutical, Inc., Spring Valley, NY). The dressing provides protection against trauma, bacterial contamination, and evaporative losses. The wet dressings enhance eschar removal.[23]

With the open technique the wound is left exposed to the air to form its own biologic barrier composed of exudate, collagen, and layers of dead skin, known as the burn eschar. The eschar does not prevent bacterial

contamination nor heat nor water evaporation, and the depth of tissue destruction may be marginally increased during the drying process. The eschar is covered with an antibacterial agent twice daily. Wound contraction does not occur while the eschar is intact. The eschar is sloughed by bacterial collagenase activity within 4 weeks.[36] The exposed bed can then be grafted or allowed to contract.

Eschar excision and grafting are useful for smaller burns but cannot be used for large burns because of lack of donor skin. Commercially available xenografts (porcine skin) can be used to cover large defects following excision; however, the cost can be prohibitive.[1,11] Amnion, however, functions as a protective barrier for the wound, prevents fluid and protein loss, controls the growth of bacteria, and reduces pain at the wound site. The physical structure of amnion is similar to that of skin and it contains growth factors that enhance fibroplasia; therefore, it is a viable alternative for burn wounds that are treated with the closed method. Thus, while early excision of the eschar has substantially decreased the incidence of invasive burn wound infection and secondary sepsis in humans, eschar excision and open treatment are not practical for extensive burns in horses because of the likelihood of environmental contamination and massive losses of fluid and heat.[23] The most effective and practical therapy for large burns in horses is the open method, leaving the eschar intact, combined with continuous application of antibacterial agents.[1,2,23]

Initially, the surrounding hair should be clipped and the wound debrided of all devitalized tissue.[11] Attempts should be made to cool the affected skin using an ice- or cold-water bath. Copious lavage with a sterile 0.05% chlorhexidine solution should be performed.[11] A water-based antibiotic ointment such as silver sulfadiazine should be applied liberally to the affected areas to prevent heat and moisture loss, protect the eschar, prevent bacterial invasion, and loosen necrotic tissue and debris. This slow method of debridement allows removal of necrotic tissue as it is identified, thereby preventing erroneous removal of healthy germinal layers. The eschar is allowed to remain intact with gradual removal, permitting it to act as a natural bandage until it is ready to slough.

A calcium alginate wound dressing with acemannan hydrogel (CarraGinate, Carrington, Irving, TX) absorbs 30 times its weight in exudate, prevents further eschar formation by keeping tissues moist, and will not interfere with the activity of topical antibiotics, which can be applied before the gel or mixed with it.

Although bacterial colonization of large burns in horses is not preventable, the wound should be cleansed 2 or 3 times daily and a topical antibiotic reapplied to reduce the bacterial load to the wound. Occlusive dressings should be avoided because of their ability to produce a closed wound environment, which may encourage bacterial proliferation and delay healing.

Systemic antibiotics do not favorably influence wound healing, fever, or mortality, and can encourage the emergence of resistant microorganisms. Additionally, circulation to the burned areas is often compromised, making it highly unlikely that parenteral administration of antibiotics can achieve therapeutic levels at the wound. Short-term prophylactic intravenous antibiotic therapy may be indicated in the immediate post-burn period if quantitative biopsy cultures or a more rapid slide dilution method yield more than 100,000 cells/g of tissue.

The most commonly used topical antibacterial for the treatment of burns is silver sulfadiazine in a 1% water miscible cream. It is a broad-spectrum antibacterial agent that can penetrate the eschar. Silver sulfadiazine is active against Gram-negative bacteria, especially *Pseudomonas*, with additional effectiveness against *S. aureus*, *E. coli*, *Proteus*, *Enterobacteriaceae*, and *C. albicans*.[11,23,28] It causes minimal pain upon application but must be used twice a day because it is inactivated by tissue secretions.

One *in vitro* study found that a 1% silver sulfadiazine/0.2% chlorhexidine digluconate product (Silvazene, Sigma Pharmaceuticals, Melbourne, Australia) was more cytotoxic to the tissue when compared to a 1% silver sulfadiazine (Flamazine, Smith & Nephew Healthcare, Hull, UK) and a silver-based dressing (Acticoat, Smith & Nephew Healthcare, Hull, UK).[37]

Recent evidence suggests that the compound delays the wound healing process and that silver may have variable but serious cytotoxic activity in a range of host cells. It is recommended in human burn patients that silver levels in plasma and/or urine be monitored.[38] A review of 410 papers comparing the effectiveness of silver sulfadiazine cream to normal dressings in promoting healing in burn patients without infection found no direct evidence of improved healing or reduced infection with silver sulfadiazine.[39] Although pseudo-eschar formation that may preclude wound evaluation as well as transient leukopenia, skin hypersensitivity, and the development of bacterial resistance have all been reported in humans, silver sulfadiazine has few systemic effects and provides good results in the horse.[23,28,40]

Aloe vera is a gel derived from a yucca-like plant that has antithromboxane and antiprostaglandin properties.[41] It is reported to relieve pain, decrease inflammation, stimulate cell growth, and kill bacteria and fungi.

Although used successfully in the acute treatment of burns, it may actually delay healing once the initial inflammatory response has resolved.[41] Aloe vera and silver sulfadiazine are good first choices in antibiotic therapy for burns and are used extensively in human medicine.

Other effective topical antimicrobials include mafenide acetate, chlorhexidine, povidone iodine, and gentamicin sulfate ointment. Nitrofurazone (Furacin, Phoenix Pharmaceuticals, Inc, St. Joseph, MO) has a fairly narrow range of antibacterial activity, it may induce resistance, and it does not penetrate the eschar very well. Chlorhexidine (Nolvasan, Fort Dodge Animal Health, Fort Dodge, IA) is active in vitro against a broad spectrum of Gram-positive and Gram-negative vegetative bacteria. A potential drawback is that *Proteus* and *Pseudomonas* have developed or possess an inherent resistance to this product and it has no effect against fungi or Candida.[42] Because of its cationic nature, chlorhexidine binds strongly to skin, mucosa, and other tissues; therefore, it is very poorly absorbed, thus minimizing excessive systemic absorption and toxicity. Chlorhexidine can be applied as a cream or solution.

Povidone-iodine (Betadine, Purdue Frederick Co., Norwalk, CT) causes some patient discomfort but is effective against bacteria, yeast, and fungi. Its hyperosmolality causes severe hypernatremia and acidosis due to water loss such that it should not be used on extensive burns where systemic absorption is likely.[34] Depression of the immune system has also been reported in humans.[34] Gentamicin (Gentamicin sulfate ointment USP, Clay-Parks Labs, Inc., Bronx, NY) is excellent for serious Gram-negative infections but should be used only in selected cases because resistance can develop and it may be nephrotoxic in patients with renal problems.

Topical aqueous antibacterial preparations have also been used to treat burns. The solution (mixture of nitrofurazone, glycerin, and distilled water) can be applied to the wound as a mist from a spray bottle several times a day.[1] The nitrofurazone kills bacteria while the moisture loosens the eschar and promotes debridement. Other agents that are occasionally used topically include neomycin, bacitracin, and polymixin B. Their use is generally associated with the rapid development of bacterial resistance and systemic toxicity. For this reason they are not recommended for routine use in long-term wound care.[40]

It is appropriate to change antibacterial creams according to clinical results. In large burns, quantitative wound biopsy analysis is advantageous. Wound flora densities of more than 10^5 organisms/g tissue predispose the patient to bacterial invasion of healthy tissue.[36] Conversion of superficial wound sepsis to full-thickness infection with the risk of systemic sepsis is prevented by administering local antibiotics. The use of systemic antibiotics is not recommended because they are ineffective in penetrating the avascular eschar, where the risk of contamination is greatest.[40]

Many burn patients suffer pruritis such that measures must be taken to prevent self-mutilation of the wound. Reserpine, normally used in the horse as a long-acting tranquilizer, can be effective in decreasing the urge to scratch by successfully breaking the itch-scratch cycle.

Hyperbaric Oxygen

Hyperbaric oxygen (HBO) is designed to increase oxygen delivery to local ischemic tissue and, by a variety of primary and secondary mechanisms, to facilitate wound healing (Figure 12.8). At normal atmospheric pressure (atm), most of the oxygen in blood is carried by hemoglobin, with minimal additional oxygen dissolved in the plasma. By administering high concentrations of oxygen under increased pressure (2 atm–2.4 atm), the dissolved oxygen in the blood can be significantly increased, resulting in a ~30% increase in oxygen-carrying capacity. At a standard treatment pressure of 2.4 atm, an arterial PO_2 of 1,500 mmHg can be achieved, which increases the driving pressure for diffusion of oxygen into the tissue and increases the diffusion distance by 3- to 4-fold. Even though treatment sessions are relatively brief, oxygen tensions may remain elevated in subcutaneous tissue for several hours after exposure.[43,44]

The postulated mechanisms of a beneficial effect of HBO on burn wounds are decreased edema due to hyperoxic vasoconstriction, increased collagen formation, and improved phagocytic killing of bacteria.[45] In a trial comparing burn treatment with and without HBO in 16 human patients, the mean healing time was significantly shorter in the group receiving HBO.[44] On the other hand, among 266 patients with burns who were treated with HBO and 609 who were not, there were no significant differences in mortality and length of hospital stay.[46] Preliminary results of a randomized, controlled trial using HBO at a burn center were reported recently; among 125 patients randomly assigned to usual burn care or usual burn care plus HBO, the outcomes were virtually identical.[47]

Nutritional Needs/Requirements

Assessment of nutritional intake is performed with a reliable weight record. Weight loss of 10%–15% during the course of illness indicates inadequate nutritional intake. Nutritional support can include both parenteral and enteral routes; the latter is superior.[22] Early enteral feeding not only decreases weight loss but also maintains intestinal barrier function by minimizing mucosal atrophy. This reduces bacterial and toxin translocation and the potential for subsequent sepsis.[22]

Gradually increasing the grain, adding fat in the form of 4 oz–8 oz of vegetable oil, and offering free-choice alfalfa hay increases caloric intake. Stanozolol (Winstrol-V, Pfizer, New York, NY; 0.55 mg/kg IM) is an anabolic steroid that may be used to help restore a positive nitrogen balance by improving appetite and promoting weight gain. It can be given on a weekly basis for up to 4 weeks. If smoke inhalation is a concern or there is evidence of burns around the face, the hay should be water-soaked and fed on the ground in a well-ventilated environment to facilitate mastication and minimize aspiration.[23]

Complications

Burn wounds are very pruritic.[2,23] Significant self-mutilation through rubbing, biting, and pawing can occur if the horse is not adequately restrained or medicated. Usually the most intense pruritic episodes occur in the first weeks following injury, during the inflammatory phase of repair and eschar sloughing. To prevent extreme self-mutilation the animal must be cross-tied and/or sedated during this time. Other complications include habronemiasis, keloid-like fibroblastic proliferations, sarcoids, and other burn-induced neoplasia.[2,26] Hypertrophic scars, which commonly develop following deep second degree burns, generally remodel in a cosmetic manner without surgery, within 1 to 2 years. Because scarred skin is hairless and often depigmented, solar exposure should be limited. Chronic non-healing areas should be excised and autografted to prevent neoplastic transformation. Delayed healing, poor epithelialization, and complications of second intention healing may limit the return of the animal to its previous uses.

Skin Grafts

Burns heal slowly and many weeks may be required for the wound to close by granulation, contraction, and epithelialization (Figures 13.12, 13.13, 13.14). Closure of the burn wound either by suturing or skin grafting

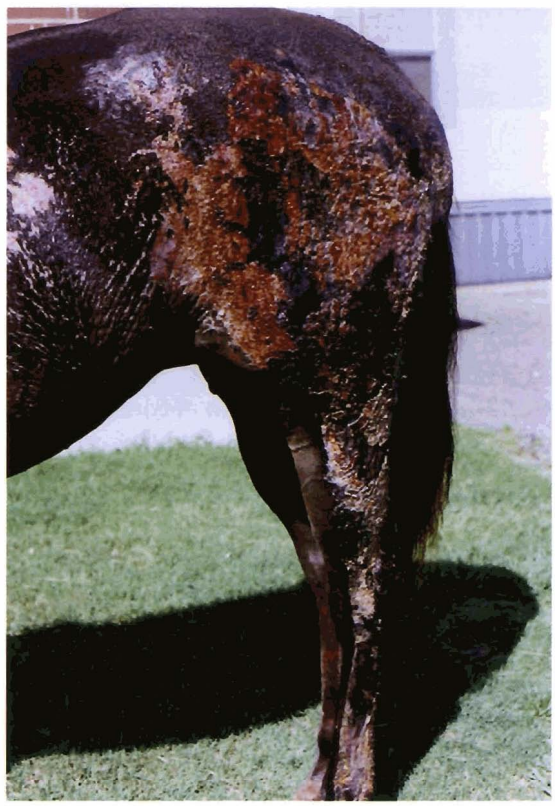

Figure 13.12. Deep second degree and third degree burns of the dorsum and left hind limb 8 days post injury. Marked erythema and early eschar formation are present.

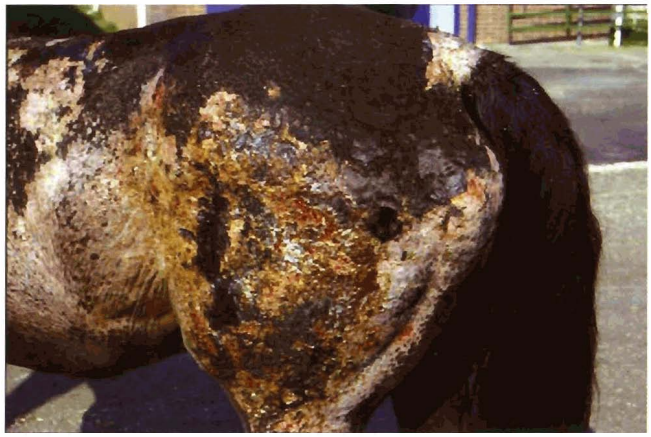

Figure 13.13. Same horse as in Figure 13.12, 5 weeks after injury. The eschar is still present centrally. The wounds are cared for with twice-daily application of silver sulfadiazine and removal of the loose eschar. Note the peripheral epithelialization of the wound.

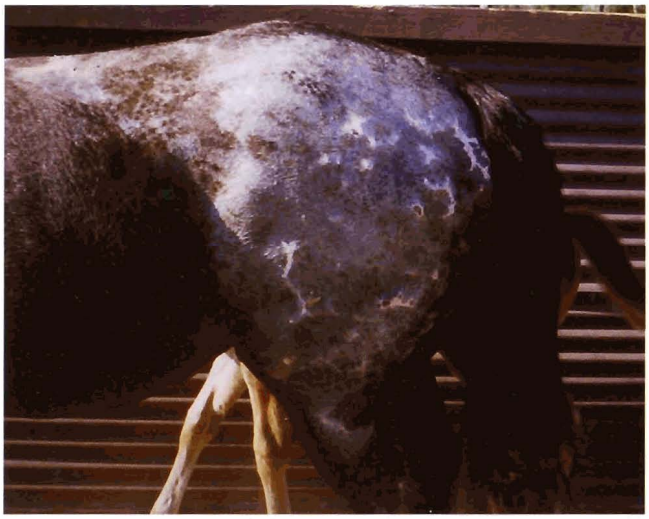

Figure 13.14. Same horse as in Figure 13.13, 7 months post injury. The entire wound has epithelialized. The skin is thin and brittle due to sparse subcutaneous tissue. Reprinted from Veterinary Clinics of North America, Vol 21, R. Reid Hanson, Management of burn injuries, p. 121, (2005), with permission from Elsevier.

following eschar removal allows for more rapid healing and superior pain relief, and prevents loss of heat, water, and protein-rich exudate from the wound surface. Burns involving only the superficial dermis heal well within 3 weeks and do not need grafting. Conversely, deep partial-thickness wounds require several months to heal, during which time bacterial contamination of the wound develops. Second-intention healing generates a thin and hairless epithelium which is vulnerable to trauma. Excision followed by grafting of the wound is recommended in these cases. Full-thickness grafts from a cadaver donor can be used as a dressing early in the clinical course of the burn to encourage healing, while split-thickness autogenous mesh grafts can be applied once healthy granulation tissue has formed. Early excision and grafting may also benefit horses that do not tolerate daily wound debridement and cleansing.

Conclusion

Extensive thermal injuries in horses can be difficult to manage. The large surface of the burn dramatically increases the potential for loss of fluids, electrolytes, and calories. Burns are classified according to the depth of injury: first degree burns involve only the most superficial layers of the epidermis; second degree burns involve the entire epidermis and can be superficial or deep; third degree burns are characterized by loss of the epidermal and dermal components; and fourth degree burns involve all of the skin and variable amounts of underlying muscle, bone, and ligaments.

Burns cause local and systemic effects. Routine use of systemic antibiotics is not recommended in burn patients. Topical medications should be water-based to facilitate cleaning and minimize toxicity. They are easily applied and removed, do not interfere with wound healing, and can be readily excreted or metabolized.

Weight loss of 10%–15% during the course of illness is indicative of inadequate nutritional intake. Gradually increasing the grain, adding fat in the form of vegetable oil, and offering free-choice alfalfa hay increase caloric intake.

During the past 2 decades, there have been many advances in burn therapy, including improved respiratory care, better use of topical and parenteral antibiotics, early debridement, and parenteral nutrition. HBO therapy is a promising treatment modality that has the potential to be an ideal supplemental therapy for burn injury and its associated complications in the horse.

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