NOTE: Wounds of the distal extremities that are sutured under tension generally are immobilized with a cast or splint bandage.

EXUBERANT GRANULATION TISSUE

- Wounds located on the limb below the carpus and tarsus, with large tissue deficits, are predisposed to the development of exuberant granulation tissue (see Fig. 12-18).
- Factors believed to be involved in the formation of exuberant granulation tissue include the following:
 - Excessive contamination/chronic inflammation (often caused by the presence of a foreign body)
 - Increased movement (e.g., wounds located on the extensor and flexor surfaces of joints and in the heel bulb region)
 - Lack of soft tissue coverage (the absence of an epithelial cover promotes the excessive formation of granulation tissue, while epithelialization is inhibited, physically and chemically, by exuberant granulation tissue)
 - Poor vascular perfusion/hypoxia that results in chronic inflammation; deregulated fibroplasia with continued synthesis of extracellular matrix components; and lack of differentiation of the proliferative fibroblast into a contractile phenotype
 - Body size: Individuals >140 cm in height and weighing more than 365 kg seem predis-



Figure 12-18 Exuberant granulation tissue (*EGT*), elevated above the skin edges and projecting over the advancing border of epithelium. (Photo courtesy Pr. Olivier Lepage, École Nationale Vétérinaire de Lyon.)

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posed; ponies have a more efficient inflammatory response to wounding, improved fibroblast orientation within the wound granulation tissue, and faster wound contraction.

- Aberrant cytokine profile, in favor of fibrogenic transforming growth factor beta₁ in wounds located on the distal limb: This growth factor stimulates fibroblast proliferation and synthesis of extracellular matrix components while limiting the disappearance of dermal fibroblasts by apoptosis (programmed cell death).
- The use of bandages and casts, which stimulate angiogenesis and fibroplasia, possibly via an effect on wound oxygen levels and cytokine profile
- Prevention
- Careful examination of the wound is critical to exclude stimuli such as bone sequestrum or frayed tendon ends.
 - Pressure bandages can be applied to young, edematous granulation tissue when the wound is located on the limb.
- Treatment
 - Débride the wound and then apply a steroidantibiotic ointment and a pressure bandage. **NOTE:** Steroids applied to newly formed granulation tissue have little effect on wound healing when applied more than 5 days after trauma.
 - Granulation tissue protruding above the surrounding skin surface forms a fibrogranuloma and is surgically excised; a pressure bandage or cast is applied. The silicone gel dressing effectively prevents the development of exuberant granulation tissue in experimental limb wounds.
 - Caustics and astringents effectively remove and prevent the formation of granulation tissue through chemical destruction. However, chemicals are not cell-selective and may thus destroy the migrating epithelial cells, causing prolonged healing times, increased inflammation, and excessive scarring.

BURNS AND ACUTE SWELLINGS

Earl M. Gaughan, R. Reid Hanson, and Thomas J. Divers

THERMAL INJURY (BURNS)

Thermal injury to a horse is rare. Most cases involve barn fires, lightning, electricity, caustic chemicals, or friction. Most burns are superficial, easily managed, and 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. 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. Longterm care is required to prevent continued trauma, for burn wounds are often pruritic and selfmutilation is common. Burned horses frequently are disfigured, preventing them from returning to full function.

Management of these severe and extensive burns is difficult, expensive, and time consuming. Before treatment, it is recommended that the patient be carefully examined with respect to cardiovascular status, pulmonary function (smoke inhalation), ocular damage (corneal ulceration), and extent and severity of the burns, and that prognosis be discussed with the owner.

History and Physical Examination

A complete history helps determine the cause and severity of burns. The extent of the burn depends on the size of the area exposed, and the severity relates to the maximum temperature the tissue attains and the duration of overheating. This explains why skin injury often extends beyond the original burn. Skin typically takes a long time to absorb heat and a long time to dissipate the absorbed heat. Therefore the longer the horse is exposed, the poorer the prognosis.

Physical criteria used to evaluate burns include erythema, edema and pain, blister formation, eschar formation, presence of infection, body temperature, and cardiovascular status. In general, erythema, edema, and pain are favorable signs because they indicate that some tissue is viable, although pain is not a reliable indicator for determining wound depth. Often, time must elapse to allow further tissue changes to occur for an accurate assessment of burn severity to be made.

It is important that the entire patient be examined, not just the burns. Burn patients frequently become severely hypovolemic and "shocky" and have respiratory difficulty; thermal injuries may cause serious suppression of the immune system.

Clinical Signs and Findings

- · Skin burns most common on the back and face
- Erythema, pain, vesicles, and singed hair
- · Increase in heart and respiratory rates
- Abnormal discoloration of mucous membranes
- Blepharospasm, epiphora, or both, which signify corneal damage (Fig. 12-19)
- Coughing, which may indicate smoke inhalation
- Fever signals or confirms a systemic response
- Special attention should be taken to identify injury to major vessels of the lower limbs and the presence of eye, perineal, tendon sheath, and joint involvement.
- Euthanasia should be recommended for animals with deep partial-thickness to full-thickness burns involving 30% to 50% of the total body surface area.

Laboratory Findings

 Shock (decreased cardiac output, low total solids and blood volume, increased vascular permeability)



Figure 12-19 Blepharospasm, epiphora, and severe etythema with loss of epithelium of the muzzle of a horse because of a barn fire.

- Anemia that may be severe and steadily progressive
- Hemoglobinuria
- Hyperkalemia early but hypokalemia later, often associated with large volume fluid therapy

Classification of Burns

First-Degree (Superficial) Burns

The germinal layer of the epidermis is spared. Burns are classified by the depth of the injury. Firstdegree burns involve only the most superficial layers of the epidermis. These burns are painful and are 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 complications (Fig. 12-20). Prognosis is excellent unless there is ocular or respiratory involvement.



Figure 12-20 First-degree burn of the right facial and periodular area. This type of burn involves only the most superficial layers of the epidermis. These burns are painful and are 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 complications.

Second-Degree (Partial-Thickness) Burns

Second-degree burns involve the epidermis and can be superficial or deep.

Superficial Second-Degree Burns

- Superficial second-degree burns involve the stratum corneum, stratum granulosum, and a few cells of the basal layer. Typically, these burns are painful because the tactile and pain receptors remain intact. Because the basal layers remain relatively uninjured, superficial second-degree burns heal rapidly with minimal scarring, within 14 to 17 days (Fig. 12-21).
- Prognosis is good.

Deep Second-Degree Burns

- 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 basal layer of the burn, eschar (slough produced by a thermal burn) formation, and minimal pain (Fig. 12-22). The only germinal cells spared are those within the ducts of sweat glands and hair follicles. 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.
- Prognosis: In general, deep second-degree wounds, unless grafted, heal with extensive scarring.



Figure 12-21 Superficial second-degree burn of the nose. Tactile and pain receptors remain intact. Because the basal layers remain relatively uninjured, superficial second-degree burns heal rapidly with minimal scarring, within 14 to 17 days.



Figure 12-22 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.



Figure 12-23 Third-degree burn of the dorsal gluteal region incurred during a barn fire when hot asphalt roof shingles fell on the horse. The central burn area is surrounded by deep and superficial second-degree burns.



Figure 12-24 Fourth-degree burn of the right cervical neck region and pectoral area. Fourth-degree burns involve all the skin and underlying muscle, bone, ligaments, fat, and fascia.

Third-Degree (Full-Thickness) Burns

- Burns are characterized by loss of the epidermal and dermal components, including the adnexa, and damage to underlying tissue structures.
- No cutaneous sensation occurs.
- The wounds range in color from white to black (Fig. 12-23). Fluid loss and a significant cellular response at the margins and deeper tissue, eschar formation, lack of pain, shock, wound infection, and possible bacteremia and septicemia also occur. Healing is by contraction and epithelialization from the wound margins or acceptance of an autograft. These burns are frequently complicated by infection.
- Prognosis can be poor, depending on extent.

Fourth-Degree Burns

- Fourth-degree burns involve all of the skin layers and the underlying muscle, bone, ligaments, fat, and fascia (Fig. 12-24).
- Prognosis is grave.

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Management

First-Degree Burns

WHAT TO DO

- · Typically, first-degree burns are not lifethreatening (unless there is severe ocular and/or respiratory involvement).
- . Immediately cool affected area with ice or cold water to draw heat out of tissues and decrease continued dermal necrosis.
- If there is minimal ocular and respiratory involvement, apply topical water-soluble antibacterial creams: aloe vera or silver sulfadiazine cream.

• Silver sulfadiazine:

Broad-spectrum antibacterial agent able to penetrate the eschar

- Active against gram-negative bacteria, especially Pseudomonas, with additional effectiveness against S. aureus, Escherichia coli, Proteus, Enterobacteriaceae,
- and Candida albicans TF
 - Relieves pain, decreases inflammation
- Causes minimal pain on application but 1.16 must be used twice a day because it is inactivated by tissue secretions
 - Decreases thromboxane activity

• Aloe vera:

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- Gel derived from a yuccalike plant
- · Has antithromboxane and antiprostaglandin properties.

· Relieves pain, decreases inflammation, 10 stimulates cell growth, and kills bacteria 1 =0and fungi

- May actually delay healing once the 12 initial inflammatory response has resolved
- 1. Pain control: flunixin meglumine (Banamine), phenylbutazone (Butazolidin),
- 71 ketoprofen (Ketofen).

Second-Degree Burns

WHAT TO DO

- Typically, second-degree burns are not lifethreatening.
- Manage the burn the same as for superficial burns.
- Burn is associated with vesicles and blisters.

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- Vesicles should be left intact for the first 24 to 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, partially excise the blister and apply an antibacterial dressing to the wound or allow an eschar to form (Fig. 12-25).

Third-Degree Burns

WHAT TO DO

- Because third-degree burns are potentially life-threatening, treatment of shock and/or respiratory distress should be the first priority.
- Destruction of the dermis leaves a primary collagenous structure called an eschar.



Figure 12-25 Deep second-degree and third-degree burns of the dorsum and left hind limb 8 days after injury. Significant erythema and early eschar formation are present.

SECTION 1 Organ System Examination and Related Diagnostic and Therapeutic Procedures

- 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. Therefore, the most effective and practical therapy for large burns in horses is leaving the eschar intact, with continuous application of antibacterial agents.
- Initially, the surrounding hair should be clipped and the wound débrided of all devitalized tissue. 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.
- A water-based antibiotic ointment 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 débridement allows removal of necrotic tissue as it is identified, thereby preventing possible removal of healthy germinal layers by mistake.
- The eschar is allowed to remain intact with gradual removal, permitting it to act as a natural bandage until it is ready to slough. Devitalized areas that appear necrotic or fetid should be débrided.
- Because bacterial colonization of large burns in horses is not preventable, the wound should be cleaned 2 or 3 times daily, and a topical antibiotic should be reapplied to reduce the bacterial load to the wound.
- Occlusive dressings should be avoided because of their tendency to produce a closed wound environment that may encourage bacterial proliferation and delay healing. A shroud sheet soaked in antiseptic solution (PI or CHD) and draped over the topline of the horse works well to protect burn areas in this region. Dry flakes of sterile starch copolymer can be mixed with silver sulfadiazine (Silvadene) and applied as a bandage anywhere on the body.
- Systemic antibiotics do not favorably influence wound healing, fever, or mortality and can encourage the emergence of resistant microorganisms in human beings; in horses, it may not be the same. 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.

Burn Shock: Life-Threatening

Burns exceeding 15% of body surface area are likely to require fluid therapy. Large volumes of lactated Ringer's solution may be needed. An alternative is to use hypertonic saline solution, 4 ml/kg, with plasma, Hetastarch, or both, followed by additional isotonic fluids. If there has been inhalation (smoke or heat) injury, then crystalloids should be limited to the amount that normalizes circulatory volume and blood pressure.

WHAT TO DO

- Use lactated Ringer's solution unless ectrolyte values dictate otherwise.
- Administer flunixin meglumine, 0.25 to 1.0 mg/kg IV q12-24h.
- Administer pentoxifylline, 7.5 mg/kg PO or IV q12h.
- Carefully monitor hydration status, lung sounds, and cardiovascular status.
- Administer plasma, 2 to 10 L per adult.

NOTE: As a general rule, for a 450-kg adult, 1 L of plasma increases the total solids 0.2 ./L.

- DMSO, 1 g/kg IV for the first 24 hour, may decrease inflammation and pulmonary edema.
- If pulmonary edema is present and is unresponsive to DMSO and furosemide treatment, administer dexamethasone, 0.5 mg/kg IV once only. If there is rapid loss of plasma protein and pulmonary edema, 25% human albumin (1 ml/kg) can be administered along with furosemide.
- If there are respiratory signs or smoke inhalation is suspected (most burns to the face have smoke or heat inhalation injury), begin systemic antimicrobial therapy. Administer penicillin intramuscularly to protect against oral contaminants colonizing the airway. Broad-spectrum antimicrobial therapy may encourage fungal growth. If respiratory signs deteriorate, transtracheal aspiration should be performed and additional broadspectrum antimicrobial therapy administered according to the results of Gram strin, culture, and sensitivity.

Smoke Inhalation

See Chapter 19, p. 460.

For severe upper airway injury, a tracheotomy may be required. Perform the procedure only if an

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obstruction is anticipated. (See tracheotomy procedure, p. 441.)

WHAT TO DO

- Endoscopy of the trachea should be performed for prognostic purposes. If there is obvious sloughing of the mucosa, aspiration should be performed. Aspiration should last no longer than 15 seconds intervals because prolonged aspiration leads to hypoxemia.
- Supplemental humidified oxygen should be provided through an intranasal catheter. (See nasal oxygen insufflation procedure, p. 439.)
- Nebulization with albuterol, amikacin (1 ml), and acetylcysteine should be performed every 6 hours.
- Systemic antioxidant therapy should include orally administered vitamins E and C.
- The mouth should be rinsed every 4 hours with 0.05% CHD solution.
- Whether to use systemic antibiotics is controversial. One choice is penicillin alone as for burn shock. Another choice is ceftiofur (Naxcel), 2 to 4 mg/kg IV q12h, and metronidazole, 15 to 25 mg/kg PO q6-8h.
- Flunixin meglumine, 0.25 to 1 mg/kg IV q12h, should be administered for both antiinflammatory effect and in the goal of decreasing pulmonary hypertension.

Corneal Ulceration and Eyelid Burns

WHAT TO DO

- If the lids are swollen, apply ophthalmic antibiotic ointment to the cornea every 6 hours. Examine the cornea for ulceration initially and then twice daily.
- If damaged, débride the necrotic cornea after tranquilization and application of a topical anesthetic.
- Apply antibiotics and cycloplegics (atropine) topically. Do not use corticosteroids.
- A third eyelid flap may be needed to protect the cornea from a necrotic eyelid.
- Silver sulfadiazine can be used around the eyes.

WHAT NOT TO DO

Do not use chlorhexidine around or in the eye!

Nutritional Needs

Assessment of adequate nutritional intake is performed with a reliable weight record. Weight loss of 10% to 15% during the course of illness is indicative of inadequate nutritional intake. Nutritional support can include parenteral and enteral routes, with the latter being superior. 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 subsequent sepsis.

WHAT TO DO

- Gradually increase the grain, add fat in the form of 4 to 8 oz vegetable oil, and offer free-choice alfalfa hay increases caloric intake.
- An anabolic steroid may be used to help restore a positive nitrogen balance.
- 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 with good ventilation provided.

Complications

Wound Infection

Severe burns become infected. Most infections are caused by normal skin flora.

Pseudomonas aeruginosa, S. aureus, E. coli, beta-hemolytic streptococci, other Streptococcus spp. organisms, Klebsiella pneumoniae, and Proteus, Clostridium, and Candida organisms are commonly isolated.

It is appropriate to change antibacterial creams as needed to control infection.

Silver sulfadiazine is effective against gramnegative organisms such as *Pseudomonas* and has some antifungal activity.

Aloe vera is reported to have antiprostaglandin and antithromboxane properties (e.g., to relieve pain, decrease inflammation, and stimulate cell growth), in addition to antibacterial and antifungal activity.

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Laminitis

See Chapter 29, p. 627.

Pruritic Wounds

Healing burn wounds are pruritic.

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 during the inflammatory phase of repair and during eschar sloughing.

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WHAT TO DO

To prevent extreme self-mutilation, the patient must be cross tied and/or sedated (e.g., acepromazine except in breeding stallions) during this time. Antihistamines may be effective in some cases. Reserpine can be effective in decreasing the urge to scratch by successfully breaking the itch-scratch cycle.

Other Short-Term Complications

- Habronemiasis
- Because scarred skin is hairless and often depigmented, solar exposure should be limited.

ACUTE SWELLING: EDEMA

Acute edematous conditions in the horse most commonly result from increased hydrostatic pressure, septic inflammation, or a local or general immune response. Acutely occurring hypoproteinemia is a less common cause. Inflammatory conditions, septic and immunologic, usually are painful to the touch. Edema resulting from increased hydrostatic pressure is less painful and, in many cases, nonpainful.

Purpura Hemorrhagica

- Consider purpura hemorrhagica with any unexplained vasculitis and edema.
- Edema is most common in the limbs and ventral abdomen and often moderately painful to the touch. Edema forms elsewhere in the body, causing respiratory distress (laryngeal swelling and pulmonary edema), colic, heart failure (distress and trembling), or myositis (stiffness).
- Fever and petechiae of mucous membranes occur in approximately 50% of cases.

• Often the horse has a history of respiratory infection or exposure to *Streptococcus equi* (most frequent) or *S. zooepidemicus* in the preceding 2 to 4 weeks.

Diagnosis

- Diagnosis is based on a complete blood cell count, measurement of creatine kinase and aspartate aminotransferase, platelet count, measurement of serum immunoglobulin A, and serologic testing for serum streptococcal M protein antibody and immune complexes (performed at Gluck Equine Research Center, University of Kentucky).
- A skin specimen from an edematous area obtained with a 6-mm Baker biopsy punch^{hh} can be submitted in formalin to examine for vasculitis. Detection of immunoglobulin deposition is rare, and submission in special medium (Michel's) or snap freezing is recommended. The biopsy specimen should not be harvested from an area over an important structure (e.g., tendon).
- Mature neutrophilia occurs, and creatine kinase and aspartate aminotransferase levels frequently are elevated with or without signs of myositis.
- A normal platelet count >90,000 cells/ml is expected.
- An elevation in plasma protein measurement is usual, as are an elevated immunoglobulin A level and a high antibody response to streptococcal M protein. However, a high antibody response to streptococcal M protein also occurs in some healthy individuals.
- Severe proteinuria and even hematuria occur in some patients. Severe myopathy, mostly involving the hind limbs, may, also occur in some horses (see Chapter 16, p. 350).

Differential Diagnosis

Equine viral arteritis (EVA), equine herpesvirus, equine infectious anemia, Anaplasma phagocytophilum infection, and Lyme disease are differential diagnoses. Be careful interpreting positive Lyme titers. Many normal horses in endemic areas have a titer to Borrelia. An indirect fluorescence antibody titer greater than 1:1280 is considered suspect for Lyme disease, and additional testing with kinetic enzyme-linked immunosorbent assay (>300 units), immunoblots, and polymerase chain reaction (PCR; performed at Cornell University Diagnostic Laboratory) may be indicated. Most Standardbreds are

^{hh}Baker Cummins Pharmaceuticals Inc., Miami, Florida.