Pathophysiology and Current Management of Burn Injury

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The author has disclosed that she has no significant relationships or financial interests in any commercial companies that pertain to this education activity.

Wolters Kluwer Health has identified and resolved all faculty conflicts of interest regarding this educational activity.

PURPOSE
To provide the physician and registered professional nurse with an overview of the pathophysiology and current management of burn injuries.

TARGET AUDIENCE
This continuing education activity is intended for physicians and nurses with an interest in learning about evidence-based prevention and management of burn wounds.

OBJECTIVES
After reading the article and taking the test, the participant should be able to:
1. Explain the pathophysiology of skin function.
2. Describe the different types of burn injuries.
3. Identify the treatment strategies for burn injuries.

Remendous advances have been made in the management of burn injury in the past decade. Mortality and morbidity have been markedly reduced due to overall major improvements in critical care, metabolic support, infection control, and wound management.1-8

As with any wound, many systemic factors impact burn wound healing, including metabolic response to injury, nutritional status, presence of systemic infection, and other systemic insults such as pain and stress. Fortunately, the advances in burn wound care have focused on addressing these factors. Aggressive surgical management of deep burns, improvement of the wound healing environment with use of silver release dressings, better pain control in partial-thickness burns, improved healing of partial-thickness burns with temporary skin substitutes, improved functional and cosmetic outcomes of massive burns with use of adjuvant therapies, and optimum

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management of major burns in burn centers are among the advances in burn care.  
A burn is defined as damage to the skin caused by excessive heat or caustic chemicals. The most common burn injuries result from exposure to heat and chemicals. Full-thickness burns usually develop, causing immediate cell death and matrix destruction, with the most severe damage on the wound surface. Additional heat and inflammation induce tissue injury beneath the nonviable surface, which can either progress over time to healing or can deteriorate to further necrosis, depending on the approach to treatment.

Managing a burn wound is challenging because treatment must be continuously adapted to the changing wound biology, dictated by the burn injury process, the host’s response to injury, and the wound environment. Flexibility in adapting care to the changing wound is essential.

SKIN FUNCTION

Understanding a burn injury requires recognition of the anatomy and physiology of the skin. The skin is a bilayer organ (Figure 1) with many protective functions essential for survival (Table 1). The outer epidermal layer provides critical barrier functions and is composed of an outer layer of dead cells and keratin, which present a barrier to bacterial and environmental toxins. The basal epidermal cells supply the source of new epidermal cells. The undulating surface of the epidermis, called rete pegs, increases adherence of the epidermis to the dermis via the basement membrane.

Table 1.

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<th>SKIN FUNCTION</th>
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The inner dermal layer has a number of essential functions, including continued restoration of the epidermis. The dermis is divided into the papillary dermis and the reticular dermis. The former is extremely bioactive; the latter, less bioactive. This difference in bioactivity within the dermis is the reason that superficial partial-thickness burns generally heal faster than deeper partial-thickness burns; the papillary component is lost in the deeper burns.

Loss of the normal skin barrier function causes the common complications of burn injury. These include infection, loss of body heat, increased evaporative water loss, and change in key interactive functions such as touch and appearance (Table 2).

BURN SEVERITY

Burn severity can be determined by burn depth, size, location, and patient age. Burn size is defined by the percentage of total body surface area (TBSA) that is burned. The Rule of Nines is a commonly used tool; it divides the surface area of the body into segments of 9% (Figure 2). Age is a major determining factor in a patient’s prognosis. Infants and the older adults have a higher mortality rate than older children and young and middle-aged adults.

Table 2.

<table>
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<th>COMPLICATIONS OF BURN-INDUCED SKIN BARRIER LOSS</th>
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The depth of heat injury depends on the degree of heat exposure and depth of heat penetration. Wet heat (scald) travels more rapidly into tissue than dry heat (flame) because water conducts heat 100 times greater than air. In addition, skin thickness is critical; the thinner the skin, the deeper the burn will be due to less residual dermis. Children and older adults have thin skin and, therefore, are at risk for deeper injury than younger adults from the same heat exposure.

Burn depth is defined by how much of the skin’s 2 layers are destroyed by the heat source, and it is the primary factor that dictates wound management. In the past, burn wounds were categorized by degrees, namely, first through fourth degrees. However, it is more accurate to refer to burn wound depth by the anatomic thickness of the skin involved, as follows:

- A **superficial burn** is confined to the outer epidermal layer.
- A **partial-thickness burn** involves the epidermal layer and part of the inner dermis.
- A **full-thickness burn** involves destruction of both layers.
- A **subdermal burn** involves destruction of both layers and extends to the tissue below, including fat, tendons, muscle, and bone.

Burn wounds are dynamic and can evolve into deeper injuries over time, depending on the initial injury and subsequent environmental insults. Burn wounds are composed of an outer layer of nonviable tissue, known as the **zone of necrosis**.

This involves both layers of skin in a full-thickness burn. In a partial-thickness burn, the viable tissue beneath the layer of necrosis is still injured—known as the **zone of injury**—and can become nonviable over time, depending on the degree of injury and subsequent insults, such as infection. This process is known as **wound conversion**.

Several significant differences exist between management of a burn wound and management of a nonburn wound. First, infection is of greater concern with a deep burn due to its impaired blood flow and, in part, historic patterns of care. Topical antimicrobials are routinely used from the onset, compared with more selective use in nonburn wounds. Second, deep burn wounds are typically surgically excised and closed with a skin graft or skin substitute early in their course. By comparison, healing by secondary intention is more common in nonburn wounds.

Third, less attention is paid to moist wound healing in a burn wound, although desiccation is prevented by the use of temporary skin substitutes in superficial burns and use of moist dressings over excised or grafted burns. Hydrocolloids and alginates are less commonly used on burn wounds.

**BURN WOUND MANAGEMENT**

**Superficial burn**

A superficial burn is confined to the epidermis and is not considered to be a significant burn. No barrier functions are...
altered. The most common form of superficial burn is caused by ultraviolet radiation from the sun (sunburn). It generally heals by itself in less than a week without scarring. Skin moisturizers can be used to treat a superficial burn.

Partial-thickness burn

A partial-thickness burn involves the destruction of the epidermal layer and portions of the dermis; it does not extend through both layers. There are 2 depths of partial-thickness burns—superficial partial-thickness and deep partial-thickness—and each corresponds with a predictable healing time, treatment modality, and outcome.

- A superficial partial-thickness burn involves destruction of the entire epidermis and no more than the upper third of the dermis (Figure 3). The microvessels perfusing this area are injured, leading to leakage of large amounts of plasma. This lifts off the heat-destroyed epidermis and causes a blister to form (Figure 4). The resulting wound is pink, wet, and painful. These are the most painful burns because the nerve endings of the skin are exposed to air. Remaining blood flow is adequate and the infection risk is low. Despite loss of the entire epidermis, the zone of injury is relatively small and conversion is uncommon except with extremes of age or presence of chronic illness. Rapid healing occurs in 1 to 2 weeks. Scarring is uncommon unless the wound is grossly contaminated.

Treatment begins with cleansing and debridement of loose epidermis and remaining large blisters from the wound surface. Large blisters should remain intact for no more than 2 days, as the infection risk is increased. A topical antibiotic is not required. Areas such as the face and ears are treated open, without a dressing; an ointment such as bacitracin is generally used to maintain wound moisture and control the predominantly Gram-positive bacteria on the face. Open areas are gently cleansed daily with a dilute chlorhexidine solution to remove crust and surface exudate. Areas such as the hands, upper and lower extremities, and trunk can be treated with petrolatum-impregnated gauze. A petrolatum-impregnated gauze is covered with several layers of dry absorbent gauze. If the petrolatum-impregnated gauze appears well adhered to a superficial partial-thickness burn wound with no underlying exudate, the gauze does not need to be changed. If some exudate is present, the dressing should be removed, the wound gently cleansed, and the dressing reapplied. Silver sulfadiazine cream is not recommended for treatment of a superficial partial-thickness burn because the cream retards healing. An antibiotic ointment is a better choice. Exceptions include a dirty wound that has not been cleansed of initial debris or a perineal or buttock wound, for which a silver-based topical antibiotic is typically required (Table 3).

A superficial partial-thickness burn can also be managed with a temporary skin substitute, which protects the wound surface and provides moist wound healing (Figure 5). The outer layer of gauze needs to be changed when it becomes saturated with plasma from the wound surface. When the wound no longer oozes, the skin substitute can be left open to heal.

### Table 3.

<table>
<thead>
<tr>
<th>ADVANTAGES OF SILVER DRESSINGS</th>
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<tbody>
<tr>
<td>• Release pure silver that is nontoxic to tissue</td>
</tr>
<tr>
<td>• Release silver over days in antimicrobial quantities</td>
</tr>
<tr>
<td>• Decrease mechanical trauma to the wound with no need for frequent dressing changes</td>
</tr>
<tr>
<td>• Maintain moist wound healing</td>
</tr>
</tbody>
</table>

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**Figure 4.**

SUPERFICIAL PARTIAL-THICKNESS BURN CAUSING BLISTERS

![Image](image1.jpg)

**Figure 5.**

SKIN SUBSTITUTE COVERING A SUPERFICIAL PARTIAL-THICKNESS BURN

![Image](image2.jpg)
A deep partial-thickness burn involves destruction of most of the dermal layer, with few viable epidermal cells remaining (Figure 6). Reepithelialization is slow, sometimes taking months. Blisters do not generally form because the dead tissue layer is thick and adheres to the underlying viable dermis (eschar). The wound appears white and dry. Blood flow is compromised, making the wound vulnerable to infection and conversion to a full-thickness injury. The wound is often a mixed partial- and full-thickness injury. Direct contact with a flame source is a common cause; most chemical burns are also deep partial-thickness injuries. Pain is reduced because the nerve endings have been destroyed. It is difficult to distinguish between a deep partial-thickness and a full-thickness burn wound by visualization; however, the presence of sensation to touch indicates that the burn is a deep partial-thickness injury.

Deep partial-thickness burn wounds heal in 4 to 10 weeks (sometimes longer). Wound breakdown is common because the new epidermis is thin and not well adhered to the dermis due to the lack of rete pegs.

The focus of treatment includes removing eschar and using topical antibiotics during the debridement process or until surgical wound closure occurs. Excision and grafting is the preferred treatment because dense scarring is seen when these wounds are allowed to heal through primary intention.

The predominant antimicrobial used in deep partial-thickness burn wounds is an agent containing silver, either in the form of a cream or silver-impregnated membrane used as a dressing on the wound surface. The cream must be removed and reapplied at least once a day. Silver dressings continuously release silver over several days, minimizing the need for frequent dressing changes. These dressings need to be kept moist to activate release of the silver; the wound fluid from the burn injury is often sufficient. Moist wound healing is preserved under the silver membrane. A dry gauze dressing is used over the silver cream or dressing.

Full-thickness burn

A full-thickness burn results in complete destruction of the epidermis and dermis, leaving no residual epidermal cells to repopulate (Figure 7). Initially, the dead avascular burn tissue (eschar) appears waxy white in color. If the burn produces char or extends into the adipose layer due to prolonged contact with a flame source, a leathery brown or black appearance can be seen, along with surface coagulation veins. Direct exposure to a flame source is the usual cause of a full-thickness burn injury; however, contact with hot liquids, such as grease, tar, or caustic chemicals, will also produce a full-thickness burn. Similar to a deep partial-thickness burn, a full-thickness burn is also painless. One
major difficulty is distinguishing a deep partial-thickness burn from a full-thickness burn; however, treatment is similar for both.

Treatment of a full-thickness burn wound includes early surgical debridement and wound closure with a skin graft or permanent skin substitute. Before the planned surgery, the wound is treated with a silver-based antibiotic cream or dressing, using a closed dressing technique.

Subdermal burn
A subdermal burn entails complete destruction of the epidermis and dermis, with extension into underlying tissue, such as connective tissue, muscle, and bone. The wound appears charred, dry, and brown or white without sensation; typically, the affected digit or extremity has limited or no movement. Treatment often requires amputation of the involved area.

BURNS REQUIRING SPECIAL CARE
Burns to the face, eyes, ears, hands, feet, and perineum have a greater risk of complications and potential functional and cosmetic disabilities. These burns should be managed in a burn care facility.23,24

Face
Burns to the face are at high risk for cosmetic and functional disability. For superficial burns, gentle and frequent cleansing to remove all devitalized tissue is followed by application of an antibiotic ointment such as bacitracin 2 to 3 times a day to prevent desiccation and control residual Gram-positive organisms. The face is treated open. Temporary skin substitutes can be useful because they help protect the wound and eliminate pain.25

Deeper facial burns require a more aggressive approach to help prevent infection, including the use of silver products and frequent debridement of loose necrotic tissue. Surgical management is typically needed.

Ears
Superficial ear burns are managed similarly to facial burn injuries; however, external pressure should not be applied to the injured helix. The cartilage in this area is already poorly vascularized and any compression will potentiate further injury. Pressure control includes removing pillows or pressure while sleeping. Deeper burns need more potent topical therapy, usually with a silver or mafenide cream. Chondritis, or cartilage infection, is a major complication and leads to loss of cartilage and permanent deformity. Systemic antibiotics are required.

Hands and feet
Burns to the hands and feet can cause functional disability. Superficial burns are managed with a petrolatum-impregnated gauze or skin substitute. Skin substitutes help provide wound protection and pain control, especially in wounds on the feet. Deeper burns require therapy with silver-based products. When the hands and feet are injured, each digit should be individually wrapped to minimize functional disability, such as web space contractures, and to allow for continued movement and aggressive physical therapy.

Perineum
A perineal burn is at high risk for developing infection and requires thorough cleansing and reapplication of topical agents after urination or defecation.

Major burns
Major burns can lead to significant morbidity or mortality; treating them requires expertise in burn care. They are defined as follows:
- partial-thickness burns greater than 10% of TBSA
- burns that involve the face, hands, feet, genitalia, perineum, or major joints
- full-thickness burns in any age group, over 1% of body surface
- electrical burns, including lightning injury
- chemical burns
- inhalation injury
- a child with any of the above burn injuries
- burn injury in patients with preexisting medical disorders that could complicate management
- any burned child, if the hospital initially receiving the patient does not have qualified personnel or equipment for children.

Patients with major burns are usually admitted to the hospital and should be treated in a burn center.23,24

BURN WOUND INFECTION
Most burns are at risk for infection. Burn wound infection is defined as bacterial invasion into viable tissue beneath the burn eschar.16,17,26,27 Because all burns are colonized with bacteria, a positive swab culture does not mean infection is

<table>
<thead>
<tr>
<th>Table 4. LOCAL SIGNS OF BURN WOUND INFECTION</th>
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<tbody>
<tr>
<td>• Black or brown focal areas of discoloration</td>
</tr>
<tr>
<td>• Enhanced sloughing of burned tissue</td>
</tr>
<tr>
<td>• Partial-thickness wound converting to full-thickness wound</td>
</tr>
<tr>
<td>• Increasing edema around the wound edges</td>
</tr>
<tr>
<td>• Softening of focal subeschar</td>
</tr>
</tbody>
</table>
present. Infection is diagnosed by quantitative culturing of a small full-thickness biopsy of the burn wound, including some viable tissue. A bacterial count exceeding $10^5$ organisms per gram of tissue indicates infection because this amount of bacteria typically overwhelms local immune defenses. Infection can also be diagnosed clinically, although this method is less reliable.

Table 4 describes some of the local signs of burn wound infection. Although systemic signs of increased fever and other signs of sepsis provide valuable information when evaluating for infection, fever and elevated white count are commonly seen in burn patients without infection because of the inflammatory response to burn tissue.

Systemic antibiotics plus topical antibiotics specific to the cultured organisms are used to treat burn wound infection. Eschar is also debrided to help remove the source of infection while providing better access to the remaining infected viable tissue for topical antibiotics. The topical antibiotic mafenide (Sulfamylon) is often used either as a solution or cream to treat burn wound infections because it has better tissue penetration than available silver products. Silver-based agents can be used after debridement has removed most of the infected tissue.

Table 5.
TEMPORARY SKIN SUBSTITUTES

<table>
<thead>
<tr>
<th>Product</th>
<th>Origin of Tissue</th>
<th>Layers</th>
<th>Category</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human (allograft)</td>
<td>Human cadaver</td>
<td>Epidermis and dermis</td>
<td>Split thickness</td>
<td>• Temporary coverage for large excised wounds</td>
</tr>
<tr>
<td>Pig skin (xenograft)</td>
<td>Porcine dermis</td>
<td>Dermis</td>
<td>Dermis</td>
<td>• Temporary coverage for partial-thickness and excised burns</td>
</tr>
<tr>
<td>Biobrane</td>
<td>Synthetic with added denatured bovine collagen</td>
<td>Blayer product: outer silicone layer; inner nylon mesh with collagen</td>
<td>Synthetic epidermis and dermis</td>
<td>• Superficial partial-thickness burns • Temporary coverage for excised burns</td>
</tr>
<tr>
<td>TransCyte</td>
<td>Allogenic dermis</td>
<td>Blayer product: outer silicone layer; inner nylon seeded with neonatal fibroblasts</td>
<td>Bioactive dermal matrix components on synthetic dermis and epidermis</td>
<td>• Superficial partial-thickness to mid-partial-thickness burns • Temporary coverage for excised burns</td>
</tr>
</tbody>
</table>

ADVANCED PRODUCTS FOR BURN CARE

Silver-release products
Silver has been used for centuries to prevent and treat a variety of diseases, most notably infections. Silver has extremely potent antimicrobial properties, with levels in solutions exceeding 10 parts per million. Silver ions appear to kill microorganisms instantly by blocking their respiratory enzyme system (energy production) and altering microbe deoxyribonucleic acid (DNA) and the cell wall; they have no toxic effect on human cells in vivo. However, the available delivery systems—often in the form of a salt—have been limiting factors to successful biologic use of this noble metal in burn wound care.

Silver nitrate and silver sulfadiazine have been used to deliver antimicrobial silver for the past 40 years. However, they impair fibroblast and epithelial proliferation, which impairs healing.

Silver itself is considered to be nontoxic to human cells in vivo. The only reported complication is the cosmetic abnormality argyria, which is caused by precipitation of silver salts in the skin and results in a bluish gray discoloration. Clinical evaluations have found no tissue toxicity. The major complications attributed to silver compounds are due to the
complex, or anion—namely, nitrate and sulfadiazine—and not the silver itself.

Pure silver present in current silver dressings has been shown not only to have potent antimicrobial activity, but also to lack toxicity to wound cells. Some data also indicate prohealing and anti-inflammatory properties of pure silver, including blocking excess matrix metalloproteinase (MMP) activity.31-34 Current silver dressings release pure silver ions in antimicrobial concentrations from a membrane surface over a period of days. Sustained release of silver is important in reducing bacterial burden. Silver nitrate must be applied every 2 hours to be effective, and the cream base in silver sulfadiazine reacts with serous exudate to form a pseudo-eschar that must be removed before the cream can be reapplied. Current silver dressings can be left in place for up to 7 days. The wound does not have to be manipulated during this period, which decreases trauma to new epithelial growth and reduces the wound bacterial burden. A thin moisture layer beneath the silver dressing also maintains a moist healing environment. The available hyperosmolar creams, which have a short period of silver activity, can also cause surface desiccation.

### Skin substitutes

Major advances in patient care have resulted in a marked decrease in morbidity and mortality, especially with massive burns. In addition to survival, the current focus of burn wound care is on improving long-term function and appearance of the healed or replaced skin cover and the quality of life.35-50 The issue of quality of life has generated a significant interest in the use of skin substitutes to improve wound healing, control pain, create more rapid closure, improve functional and cosmetic outcome, and, in the case of massive burns, increase survival.

To more effectively address these issues, the new generation of skin substitutes is typically biologically active. The bioactivity can modulate the burn wound instead of only providing coverage. The new products have not displaced the more inert standard burn wound dressings. Instead, they are used in conjunction with these products and have specific indications. Skin substitutes can be classified as temporary wound coverings used to decrease pain and augment healing or permanent skin substitutes used to add or replace the remaining skin components.

- **Temporary skin substitutes** are used to help heal partial-thickness burns or donor sites and close clean excised wounds until skin is available for grafting (Table 5). There are typically no living cells present in temporary skin substitutes.

  Temporary skin substitutes typically feature a bilayer structure consisting of an outer epidermal analog and a more biologically active inner dermal analog. The purpose of a temporary skin substitute is twofold. The first objective is to close the wound, thereby protecting it from environmental insults. The second objective is to provide an optimal wound healing environment by adding dermal factors that activate...
and stimulate wound healing. Biologically active dermal components are typically naturally provided to the inner layer, which is then applied to the remaining dermis in a partial-thickness burn or an excised wound.

**Permanent skin substitutes** are used to replace lost skin by providing an epidermis, dermis, or both. They offer a higher quality of skin than a thin skin graft (Table 6). Most permanent skin substitutes contain viable skin cells as well as components of the dermal matrix.

The purpose of a permanent skin substitute is to restore full-thickness skin loss and improve the quality of the skin that has been replaced after a severe burn. Permanent skin replacement is a more complex process than the use of a temporary skin replacement product.

Two approaches are available to develop a permanent skin substitute. The first is the use of a bilayer skin substitute, with the inner layer being incorporated into the wound as a neo-dermis, rather than removed like a temporary product. The outer layer is either a synthetic to be replaced by autograft (epidermis) or actual human epithelial cells. The epithelial cells, which will form the epidermal barrier, are often not sufficiently developed at placement to immediately act in this capacity.

The second approach is to provide either an epidermal or dermal analog or a 1-layer tissue. These products are technically not permanent skin substitutes on initial placement because there is no bilayer structure, although they are often described as such.

One disadvantage of skin substitutes is the absence of active antimicrobial activity; however, early effective wound closure decreases the risk of bacterial growth.

### Topical negative pressure therapy

The use of topical negative pressure (TNP) in the form of vacuum-assisted closure (VAC) is a recognized treatment for wounds. Some of the effects of TNP therapy using VAC are listed in Table 7.

Because of its beneficial effects, TNP therapy has been introduced to various aspects of burn wound management with positive results. For example, TNP therapy has been used for the initial management of high-risk burns, such as moderately deep hand burns, to decrease edema and improve outcomes. Wound conversion appears less likely when TNP is used in these cases because it removes edema and improves dermal blood flow. TNP therapy has also been found to improve management of difficult burns, especially those on the perineum and buttocks. Increased healing of donor sites and improved skin graft take have also been reported.

### SUMMARY

Multiple factors affect burn severity and outcome. Burn depth, percentage of TBSA, age, chronic illness, overall health status, part of the body burned, and presence of smoke inhalation injury contribute to the rate of burn survival.

Management of a burn wound has made remarkable progress over the last 10 years. Statistics indicate that burn survival has markedly improved in recent years, provided that optimum care is available from the injury scene to discharge. These outcomes, in part, parallel the advances made in wound healing and general wound management. Improvements in technology, infection control, and skin substitutes have also contributed to the improvements made in burn wound care.

Although significant differences remain in the pathophysiology and treatment of burn wounds compared with nonburn wounds, the focus of wound care remains the same: rapid wound closure and universal infection control.

### REFERENCES


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