CHAPTER 33
BURNS

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Introduction

A burn wound is a wound resulting from physical heat (thermal), chemical agents, or electric current applied to any part of the body. Burn injuries are common, complex injuries of cutaneous and underlying structures that are particularly difficult to manage in Africa due to inadequacies in infrastructure, resources, and staff. Factors such as poverty, illiteracy, urban migration, and the development of slums and shanty towns contribute to the high incidence of burn injuries in African children.

Burn injuries produce significant morbidity and mortality, particularly in children younger than 5 years of age. Prevention of burn injuries is of great importance because the consequences of burn injury in a child are scars that affect the child's life in a variety of ways.

Demographics

Although burn injuries are quite common, exact statistics are not available. Extrapolation from population-based studies suggests that the incidence of hospitalised paediatric burn patients is highest in Africa and lowest in the Americas, Europe, the Middle East, and Asia. However, hospital-based data vastly underestimate the true incidence of burn injuries because many children are seen in outpatient settings with minimal documentation.

Children younger than 5 years of age are at greatest risk of burn. Children younger than 2 years of age have more than twice the mortality rate of older children and adults with equivalent injuries.

Aetiology

Burn injuries may result from hot liquids (scalds), hot objects, flames, explosives, chemicals, friction, and electrical current. Scald burns are the most common, contributing up to 80% of burn injuries in some series. In comparison, in the United States, the leading burn injury mechanisms among children younger than 4 years of age are also scalds, followed by hot objects and outdoor fires. Kerosene is the most common source of flame burns in Africa.

Most paediatric burn injuries in Africa occur in the home environment, often while the child is under the care of a nonparental caregiver. In some cases, burn injury is a manifestation of child abuse. Nonaccidental burns are also seen in some cultures where therapeutic burns are practiced as a means of treating febrile convulsions and epilepsy, based on the belief that heat will terminate the convulsion. Bilateral symmetrical burn of the feet from the immersion of both feet in hot water is a characteristic pattern in such therapeutic burns.

Pathophysiology

The depth of a burn injury depends on the temperature and duration of exposure to the heat source as well as the patient's age. For example, the immersion time needed to induce a burn injury following exposure to water heated to 54°C is 30 seconds in an adult, 10 seconds in a child, and less than 5 seconds in an infant.

The initial local effect of a burn injury is divided into three histological zones (Figure 33.1). An intermediate zone of stasis surrounds a central zone of tissue coagulation composed of irreversibly injured tissue, and both are surrounded by the zone of hyperaemia. Increased vascular permeability in the zone of hyperaemia/inflammation causes transudation of fluid into the interstitial space, leading to oedema. The extravasation continues for 24–48 hours. In extensive burns, this may lead to hypovolaemia and shock, if untreated.

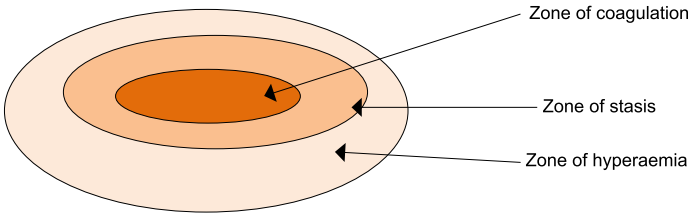

Figure 33.1: The histological effect of burn injury at the site of injury.

Appropriate cooling, fluid resuscitation, and maintenance of tissue perfusion may reverse the changes in the zone of stasis, allowing it to revert to normal. If not properly managed, continued tissue injury in this region may lead to an increase in the clinically apparent area of necrosis of the zone of coagulation.

Oedema

Increased capillary permeability in injured tissue, protein leakage, and the resultant hypoproteinaemia lead to increased osmotic pressure in burnt tissue, hence the oedema. In general, oedema is maximal at 24–48 hours, resolving in 3 to 4 days. However, in children with large burn wounds, the inflammatory response and tissue oedema may be significantly prolonged.

Hypermetabolism

The basal metabolic rate may increase up to 200 times, leading to a hypermetabolic phase associated with increased levels of catecholamines and catabolic hormones. Hypermetabolism slows down with treatment and resolves upon wound closure. The hypermetabolic response leads to increases in oxygen consumption, basal metabolic rate, urinary oxygen excretion, lipolysis, protein catabolism, and decreased synthesis, along with weight loss, that are directly proportional to the size of the burn. Early enteral feeding may attenuate the hypermetabolic response.

Many ongoing studies are focused on modulation of catecholamines in order to decrease oxygen demand, cardiac rate, and energy expenditure. Some of the promising agents include beta adrenergic blockers, insulin, and the anabolic steroid oxandrolone.

Classification of Burns

Burn injuries are classified into first- (superficial thickness), second- (partial thickness), and third-degree (full thickness) burns (Table 33.1). Second-degree burns are further subclassified into superficial and deep second-degree burns.

Table 33.1: Classification of burn injuries.

	First degree	Second degree		Third degree
	Superficial	Superficial	Deep	Full thickness
Cause	Very short flash, ultraviolet exposure	Short flash, spill scald	Flame, scald	Flame, immersion scald, chemical contact, electric current
Injured tissue	Epidermis only	Entire epidermis and part of dermis; dermal appendages intact	Entire epidermis and part of dermis; very few dermal appendages intact	Epidermis and dermis destroyed; no dermal appendages
Clinical appearance	Skin erythematous	Blisters, bullae, oedema	Blisters, bullae, oedema, pseudo-eschar	Leathery, charred skin with thrombosis of vessels
Pain	Pain gone in 48–72 hours	Painful	Painful	No pain
Healing time	1 week	2–3 weeks	More than 3 weeks	Requires grafting to heal
Results of healing	No scarring	Minimal or no scarring; dyspigmentation may occur	Large scar, hypertrophic may develop into keloids and contractures	Chronic wound, incapable of healing without intervention; contractures result

Eschar

An eschar is the necrotic tissue resulting from a burn. It separates slowly from underlying viable tissue and can serve as the substrate for invading microorganisms. Left untreated, it becomes colonised and eventually infected. Infection attracts white blood cells, which digest the interface and cause separation of the eschar from the underlying viable tissue. Circumferential eschars around limbs may impair blood circulation, and if unrelieved may cause distal ischaemia. Immediate relief is obtained by performing an escharotomy by placing vertical incisions through the eschar along the sides of the limb. Chest and abdominal eschars restrict respiration and may also require escharotomies along the sides of the chest wall. There should be no hesitation in early escharotomy if physiologic compromise is suspected.

Blisters

Burn blister management is controversial. Small blisters may be left alone to serve as biological dressings. Larger blisters require debridement to prevent an impairment of function and release the fluid that is rich in potentially deleterious proinflammatory substances.

Initial Resuscitation and Management

As with any trauma, the principles of Advanced Trauma Life Support (ATLS®) must be implemented to ensure that all life-threatening injuries are prioritised and managed. The ABCDE of ATLS must be followed:

Airway: Early intubation should be considered in patients with extensive burns requiring intensive care unit (ICU), those with extensive facial burns, and those with inhalation injuries. Progressive airway oedema is common in these situations.

Breathing: Deep chest and abdominal burns, especially when circumferential, severely impair chest wall breathing and ventilation. Escharotomies should be performed urgently when indicated. Associated chest and abdominal injuries may also impair chest wall excursion.

Circulation: Large-bore intravenous (IV) access should be placed through nonburned tissues. Venous cutdowns are frequently necessary for IV access for initial resuscitation. As an alternative route, intraosseous (IO) access may be used in the paediatric population. The doctor providing burn care must be conversant with the relevant techniques and anatomy. Isotonic salt solutions, most commonly lactated Ringers solution, should be used for resuscitation and maintenance.

Depth of Burn and **D**isability: Assessment of the depth of the burn is discussed under "Secondary Survey" in this chapter. A thorough neurological examination sets a baseline, especially in the setting of an associated head injury. Mental status changes or a history of loss of consciousness in the setting of a flame burn is most likely due to carbon monoxide poisoning. Administration of 100% oxygen, or hyperbaric oxygen where available, may be life saving.

Extent of Injury(s) and controlled **E**xposure of body: The full extent of the burn should be determined and the child examined for additional injuries. The child should be kept warm at all times.

An "**F**" should be added to ABCDE for paediatric patients:

For children:

- Children have larger heads and smaller limbs in terms of body surface area (BSA) compared to adults.
- Hypothermia is more common due to larger evaporation because the total BSA (TBSA)-to-height ratio in children is higher.
- Children have smaller glycogen stores, so hypoglycaemia is a risk.
- Adequate tetanus prophylaxis must be ensured.
- For any unusual injury patterns, consider child abuse.

Inhalational Injuries

The possibility of inhalational injury should be considered early during burn resuscitation because such patients may require early intubation. Inhalation injury results from exposure of the respiratory tract to superheated steam or air, toxic gases, chemicals, and particulate matter of smoke. Clinical diagnosis is difficult, but inhalational injury should be suspected when the child had been trapped in a closed space and in burns involving the head and neck. Characteristic symptoms indicating severe upper airway injury include hoarseness, change in voice, complaints of throat pain, and odynophagia. The child may cough up carbonaceous sputum and may demonstrate tachypnoea, wheezing, crepitations, rhonchi, and use of accessory respiratory muscles. When available, early diagnostic bronchoscopy will identify most victims.

The presence of inhalational injury is the major predictor of morbidity and mortality after burn injury. The pathogenesis can be differentiated into direct pulmonary and upper airway inhalation injury, and secondary (indirect) pulmonary injury due to activation of the systemic inflammatory response. In addition, secondary delayed pulmonary injury can be caused

by sepsis and pneumonia. Ventilator-associated lung injury may be an important contributing iatrogenic factor.

Fluid Resuscitation

Advancements in fluid resuscitation of critically burned patients have made a major impact on patients' survival and have led to a general decrease in complication rates. Burn injury leads to a combination of hypovolaemic and distributive shock by means of generalised microvascular injury and interstitial third-space fluid accumulation.

Fluid resuscitation formulas are based on the child's weight and percentage of the TBSA burned. The goal is to replace ongoing fluid losses during the early postburn period. For burns larger than 15% TBSA, significant fluid losses occur and must be replaced aggressively. The most widely used fluid regimen is probably the Parkland formula or one of its several adaptations. Numerous resuscitation formulae are in use as guides to the initial resuscitation in hypovolaemic shock following thermal injury. Most use various combinations of crystalloid and colloid solutions, but they differ widely in the ratio of crystalloid to colloid as well as the rate of administration. Most formulae give approximately 0.52 mmol of sodium/kg body weight per % TBSA burn. Although no single fluid replacement formula is perfect, physicians should aim for a urine output of 1.0–2.0 ml/kg body weight per hour. This is proof of adequate resuscitation and perfusion.

With the Parkland formula, the child is given 2–4 ml/kg per % TBSA burn over the first 24 hours, with half administered in the first 8 hours and the second half in the next 16 hours. Different physiologic demands in children of various ages and the size of the burn require even more modification of the guideline formula. For children with burns of more than 15% TBSA and weight less than 20 kg, an additional maintenance fluid containing glucose should be administered.

For burns of less than 10% TBSA, oral fluids or maintenance (IV) fluid are usually sufficient. Children with burns between 10% and 15% TBSA generally respond appropriately to 1.5 times the normal calculated maintenance fluid. Maintenance glucose infusion should be given to children younger than 2 years of age, as they may easily become hypoglycemic due to limited glycogen stores.

Frequent measurements of vital signs, hourly urine output, and observation of general mental and physical response are best used to judge the adequacy of resuscitation. If available, monitoring of the central venous pressure is also a helpful guide to the adequacy of intravascular volume.

Children require more fluid for burn shock resuscitation than do adults with similar burns. The presence of inhalation injury increases the fluid requirements for resuscitation from burn shock after thermal injury. Continuous colloid replacement may be required to maintain colloid oncotic pressure in very large burns and in the paediatric burn patient. Serum albumin levels should be maintained above 2.0 g/dl.

Failure of Burn Shock Resuscitation

In some patients, failure of burn shock resuscitation still occurs despite administration of massive volumes of fluid. Such patients are characterised by extreme age, extensive tissue trauma, major electrical injury, major inhalation injury, a delay in initiating adequate fluid resuscitation, or underlying disease that limits metabolic and cardiovascular reserve. In such patients, refractory burn shock and resuscitation failure remain major causes of early mortality. Additional data implicate a myocardial depressant factor as a contributor to early burn shock, despite adequate volume resuscitation.

The Parkland formula, discussed in the last subsection, is well known and is used as an example in this chapter. Crystalloids are preferred with this formula, as they are also cheaper than other fluids, but some centres use colloids or even hypertonic saline. Because of the increased capillary leak, colloids may potentially worsen postburn oedema.

In electrical injuries (high-voltage, including lightning strikes), the goal for urine output should be 2.0 ml/kg per hour, and alkalinisation of the urine may be necessary (add bicarbonate to the IV fluid). When available, electrocardiogram (ECG) monitoring and measurement of cardiac muscle enzymes and urine myoglobin levels are useful indicators of muscle damage. Urine output is the single most useful index of adequate intravascular replacement. In this regard, systemic blood pressure (BP) and central venous pressure (CVP) are unreliable. However, an overaggressive protocol may lead to complications, such as compartment syndromes and pulmonary oedema. Serial serum potassium and sodium levels are needed to monitor electrolyte changes.

Secondary Survey

Following initial resuscitation, a detailed history and head-to-toe examination should be conducted. The possibility of associated nonburn injuries or a precipitating event (e.g., epilepsy) should always be considered.

History

In addition to the history obtained during the primary survey, more detailed information is needed to determine:

- the cause of the burn injury (hot liquid, hot object, chemical, open flame, etc.);
- the time since injury;
- the duration and location of contact/exposure (a closed-space flame burn suggests a coexistent inhalation injury);
- any preexisting medical conditions, such as epilepsy, diabetes, mental handicap, and so forth;
- other coexisting injuries; and
- a vaccination history.

Physical Examination

Assessment of the burn wound should include the age, height, and weight of the patient; the depth of the burn wound; the extent (total body service area) of the burn; and the anatomical location of the injury.

Age, height, and weight

The age, height, weight, and calculated TBSA are needed to determine the appropriate doses of fluid and medications.

Depth of burn

The depth of the burn may be determined by clinical wound inspection and the pinprick test (see Table 33.1). The depth of the burn is the primary determinant of the patient's long-term appearance and function. It is critical to differentiate between superficial and deep second-degree burns. Whereas superficial second-degree burns heal within 2–3 weeks, deep second-degree burns require early tangential excision and skin grafting to permit relatively uncomplicated healing and a return to normal life.

Extent of burn

The extent of the burn surface involved is determined by careful observation, and should be graphically represented to aid in diagnosis, treatment, prognosis, and epidemiologic surveillance. It is calculated as a percent of total body surface area (% TBSA) using any of the following:

- Wallace's "rule of nines" (Table 33.2), which allows rapid estimation;
- Lund and Browder normogram for a more precise estimation (this table is described in several references);
- the "rule of tens", which is more appropriate for estimation of paediatric burns; or
- the patient's palm (~ 1% of their body surface area), which is useful for children with smaller burns.

Anatomical location

The location of burns has an important bearing on specific treatment, reconstruction, and rehabilitation. The hands, feet, face, eyelids, perine-

Table 33.2: Wallace's "rule of nines" for estimating % TBSA involved in burns.

Anatomic area	% TBSA
Head and neck	9
Anterior trunk	18
Posterior trunk	18
Right upper extremity	9
Left upper extremity	9
Right lower extremity	18
Left lower extremity	18
Perineum & external genitalia	1

um, genitalia, and joints are considered primary areas. They must be given appropriate care to optimise wound healing and prevent cosmetic and functional problems.

Investigation

Burn patients presenting acutely should be resuscitated as described above. The initial therapy aims at restoring normal physiologic parameters and the prevention of life-threatening complications. It is guided by the weight of the patient and the % TBSA injured.

Initial blood samples should be drawn for blood grouping and cross-match, total blood count, electrolytes, glucose, and urea nitrogen. Arterial blood gases and pH are obtained whenever inhalation injury is suspected.

Radiological investigations are generally not necessary except where inhalation injury is suspected or in the multiple trauma patient. Where possible, an initial baseline chest radiograph is useful for later comparisons.

Hospital Care

An assessment of the severity of the burn (Table 33.3) should be established early, as it gives a useful guide of the prognosis and the amount of resources that will be required to care for the child. The following steps should be initiated once the child has been resuscitated

Table 33.3: Classification of burn severity.

	Minor burns	Moderately severe burns	Major burns
BSA	<5%	5–15%	>15%
Special areas involved	No	No	Yes
Full thickness burns	None	None	Present
Comorbidities present (medical or trauma)	None	None or present	Present
Electrical or chemical injury	None	None	Present
Management	Outpatient	Hospital	Hospital

1. Clean the burns with normal saline and dress with saline gauzes, or cover with gauze dressing.

2. Adequate anaelgesia must be administered.

3. Administer tetanus prophylaxis.

4. Prophylactic antibiotics, oral or intravenous, are not indicated. Their use, prophylactically, is indicated only in the following three scenarios:
   - early administration of antistreptococcal drugs in a high-risk patient to prevent burn wound cellulitis;
   - perioperative administration of antibiotics; and
   - administration of broad-spectrum antibiotics pending return of culture information in febrile or hypotensive patients.

Ideally, children with severe burns should be managed in a burn centre or a hospital with an ICU. Guidelines include:

- children with burns >10% BSA require IV resuscitation;
- children with burns >30% BSA require central line placement;
- resuscitate crystalloids initially, with possible subsequent inclusion of colloids; and
- kaliuresis is common, and K^+ losses must be supplemented; however, this should be done with care because the damaged tissue may release large amounts of potassium.

Nutrition

During days 2 and 3 following thermal injury, treatment is directed toward fluid resuscitation and maintenance of haemodynamic stability and electrolyte balance. Starting on postburn days 3 to 5, metabolic expenditure in the thermally injured patient begins to increase and is paralleled by an accompanying increase in nutritional demands. This increased metabolic drive is directed toward support of the healing burn wound by both local and systemic hormonal mechanisms. Due to the catabolic effect of catecholamines and increased energy expenditure, a high-calorie and high-protein diet or nutritional supplementation should be initiated as soon as possible after injury.

The goals of nutritional support are to maintain and improve organ function, prevent malnutrition, and improve overall outcomes. Nutritional support is not without potential complications, which may include sepsis, glucose, and osmolar intolerance, and the mechanical hazards of the administration techniques.

A number of different formulae that may be used to calculate caloric needs for burn patients exist. The Curreri formula is one example:

$$\text{Calories/day} = (\text{wt in kg})\,(25) + (40)\,(\%\ \text{BSA})$$

This formula probably overestimates caloric needs, and needs periodic recalculation as healing occurs.

Hypermetabolism is a characteristic physiological response to major injury, and there is a direct relationship between the magnitude and duration of the hypermetabolic response and the severity of the sustained trauma. The hypermetabolic response to burn injury is not temperature dependent, and has been postulated to be mediated through the hypothalamic temperature centre. The reset hypothalamus triggers an increased metabolic rate by elevating the plasma levels of three hormones: catecholamines, glucagon, and cortisol. Because the skin plays a large part in thermoregulation, extensive damage due to burns impairs the body's thermoregulatory capacity.

There is also a marked catabolic response that accompanies severe burns; it is associated with weight loss; poor wound healing; and negative nitrogen, potassium, sulfur, and phosphorus balance. It is also associated with increased levels of glucagon and catecholamines in plasma as well as depressed levels of insulin.

The increased metabolic expenditure persists for several weeks until the burn wound either spontaneously heals or is closed by skin grafting. However, even wound closure does not immediately return metabolic expenditure to normal, and thus increased nutritional support must continue even after closure of the wound surface.

Adequate nutritional support is best monitored by daily measurement of body weight. Postburn weight loss of up to 10% is well tolerated, provided the patient was not nutritionally compromised before the burn. Weight loss exceeding 10% of the preburn weight is associated with increased morbidity. A progressive physical therapy programme enhances the deposition of protein into lean muscle mass, allowing the performance of kinetic work required for the maintenance of normal function.

Enteral feedings are recommended over parenteral feedings in burn patients because they are more physiological and less costly, and they help to preserve gut structure and function, thereby reducing

translocation of bacteria and/or toxins. As a result, the incidence of sepsis is lower in enterally fed burn patients. Due to the high incidence of gastric ileus in burn patients, nasoduodenal or nasojejunal tubes may be used for administration of feedings.

Despite the benefits, enteral feeding still carries significant risks, with the potential for disastrous complications if not well managed, including:

- mechanical complications (aspiration pneumonia, sinusitis, nasoalar, oesophageal and gastric mucosal irritation and erosion, tube lumen obstruction);
- gastrointestinal (GI) complications, such as diarrhaea and faecal impaction; and
- metabolic complications (dehydration, hyperglycaemia, hyper- or hyponatremia, hyper- or hypophosphataemia, hypercapnia, hyper- or hypokalaemia).

Pain Management

Burn injuries cause significant pain. Untreated, the pain exacerbates the hypermetabolism. This pain can be constant, therefore requiring continuous analgesia, including the use of narcotics and sedative agents. It is vital to provide adequate pain relief, especially during dressing changes, when ketamine may be useful. If narcotics are used for pain alleviation, the physicians must remember that tolerance may develop if therapy is prolonged. Sedation and analgesia should not be administered until hypoxia and hypovolaemia have been excluded and/or treated because they both produce anxiety and disorientation in the patient. When given, they must be kept at an absolute minimum to avoid cardiopulmonary depression and to allow evaluation of the sensorium, an important indicator of adequate resuscitation. Analgesics should be given intravenously because intramuscular absorption is erratic and unpredictable. Discontinuation of opiates should be anticipated and tapered as wounds heal.

Burn Wound Management

The goals of local wound management are the prevention of viable tissue desiccation and control of bacterial loads by use of topical antimicrobial agents and/or biological dressings.

Second-degree wounds usually present as vesicular lesions that should be punctured and the nonviable skin removed to allow for the application of topical chemotherapeautic agents to the underlying viable dermis.

Topical Antibiotics

Several topical antimicrobial agents are available, as shown in Table 33.4. Modern antibacterial topical therapy for burn injuries was advocated by Moyer and co-workers in the early 1960s. They used aqueous silver nitrate 0.5% solution.

Silver nitrate is effective against most gram-positive organisms and most strains of *Pseudomonas*, although it has limited effectiveness against other gram-negative bacteria, such as *Klebsiella* and *Enterobacter*. Silver nitrate (0.5%) soaks are also effective in preventing microbial penetration of the eschar when treatment is begun immediately after the burn. Because silver nitrate does not readily penetrate the eschar, however, it has limited ability to control the proliferation of microorganisms already colonising the eschar. Soaks of 0.5% silver nitrate are generally reserved for use in patients allergic to sulfonamides.

Sulfamylon® was introduced in the mid-1960s, and is effective against a wide spectrum of gram-positive and gram-negative organisms, as well as anaerobes. Sulfamylon is an 11.1% suspension of mafenide acetate in a hydrophilic base. The solubility and the high activity of mafenide against gram-negative organisms, particularly *Pseudomonas aeruginosa*, make Sulfamylon burn cream particularly effective in limiting the proliferation of bacteria that have penetrated the eschar and preventing the development of invasive burn wound infection. However, by inhibition of the carbonic anhydrase enzyme, it may induce acid-base derangements. It is also associated with pain on application, as well as occasional hypersensitivity reactions (5–7% of patients).

Table 33.4: Properties of topical antimicrobial agents.

Topical antimicrobial	Fibroblast toxicity	Bacteriocidal	Bacteriostatic
Sodium hydrochloride	×	0.025%	
Povidone Iodine	✓	0.5%	
	✓	1.0%	
Hydrogen peroxide	0.3%	×	
	3.0%	✓	
Acetic acid	0.25%	×	
Silver nitrate	✓		10.0%
	×		5.0%
Silver sulfadiazine	✓		1.0%
	× (with aloe vera/ nystatin)		1.0%

Silver sulfadiazine as 1% suspension in a hydrophilic base (Silvadene®) has essentially the same spectrum of activity as mafenide acetate, but fewer side effects. It is widely used in Africa as well as in the Western countries.

Betadine is a water-soluble antiseptic, effective against a wide range of gram-positive and gram-negative organisms, as well as some fungi.

Clinical bacteriologic monitoring of the burn wound is imperative in order to diagnose incipient burn wound sepsis and effect immediate treatment.

Traditionally, topical antimicrobial agents have been applied to a burn wound débrided of devitalised skin in a form of ointment, cream, or solution. A secondary dressing should be applied to the burn wound over the antimicrobial agent. These include: gauze, xeroform (3% bismuth tribromophenate in a petrolatum-blend on fine gauze), aquaphor gauze, foam dressings, and polyurethane dressings. These types of dressings are quite painful and, particularly in children, associated with significant anxiety. Recent developments of new silver-based antimicrobial delivery systems have eliminated the disadvantages of daily dressing change. Examples of available products include, among many: Acticoat®, Aquacel Ag®, Mepilex Ag®, and Glucan Silver Matrix®. These products consist of silver-containing pads or hydrocolloid fibre sheets that provide a sustained delivery mechanism for silver and in addition function to absorb excessive exudate from a wound. Applied to the débrided wound surface, these products could be left in place for several days.

Tangential Excision

The current accepted practice involves early excision (3–7 days post-burn)—tangential excision of deep second- and third-degree wounds until viable tissue is reached, as evidenced by capillary bleeding. Tangential eschar excision and skin grafting 3–5 days after the burn injury offers several advantages over full-thickness (fascial) excision, such as removal of only necrotic tissue, salvage of injured tissue that otherwise would have progressed to necrosis, preservation of biological properties of the dermis, and prevention of contractures. The primary closure is achieved by immediate grafting with autograft, and temporary closure is performed with heterograft or homograft, or synthetic barrier dressings. Although technically easy to perform, this procedure requires experience in determining the level of adequate excision. The advantages are a shortened hospital stay and potentially improved function when the wounds extend across joints. Tangential excision offers nothing if

the burn wound is large and full-thickness. The major disadvantage is performing a major operation, with potential for a lot of blood loss, on a very sick patient, as well as the fact that it does not appear to materially change the pattern of the causes of death in those who die after 3 days of hospitalisation. Due to a lack of resources, in many hospitals in Africa, the eschar is often allowed to separate on its own, leading to an increased risk of infections and prolonged convalescence.

Wound Closure

Biologic dressing and biosynthetic products

Following spontaneous eschar separation or, preferably, after surgical removal by tangential or fascial excision, extensive wounds can be permanently covered with autograft or temporarily covered using a variety of techniques and dressings.

Biologic dressings, such as porcine xenograft or cadaveric allograft, are most commonly used. These provide early temporary wound closure, and therefore contribute to the prevention and control of infection, the preservation of healthy granulation tissue, and the maintenance of joint function. They decrease evaporative water loss and limit heat loss secondary to evaporation; they cover exposed sensory nerves, and thus decrease pain associated with the open wound; and they protect neurovascular tissue and tendons that would otherwise be exposed. The major drawbacks are their variable quality and, depending on donor age and harvesting technique, both have to be removed and both carry potential risk for viral infection. Amniotic membranes have also been used. Tissue engineering and advancements in biotechnology have provided several novel modalities to address those issues. Varieties of products are available, including skin, dermal, and epithelial substitutes.

Biosynthetic products used for temporary wound closure include Apigraf® (allogeneic bilayered skin equivalent, which consists of human keratinocytes and human fibroblasts in a lattice of bovine type I collagen); Biobrane® (nylon mesh coated with porcine collagen type I peptides and bonded to silicone rubber membrane); and TransCyte® (human neonatal fibroblasts seeded on coated nylon of Biobrane). The latter tissue substitute contains multiple growth factors and secreted matrix molecules, and is not only effective in treatment as a temporary closure of excised wound, it is also easy to handle and to remove with reduced bleeding as compared to allograft. Its drawback, however, is a significant cost of production.

Dermal substitutes include: Integra® (bilaminate membrane, which consists of bovine collagen–based dermal analogue covered with silastic sheeting); AlloDerm® (an acellular dermal substitute from cryopreserved human cadaver skin that is deprived of cells of the epidermis and dermis, leaving dermal matrix and basement membrane); and Matriderm® (a bovine noncross-linked collagen/elastin matrix).

Definitive burn wound closure is the ultimate objective of all burn wound care. However, priorities of coverage are dictated by functional and cosmetic considerations. The hands, feet, face (especially the eyelids), neck, and joints should in general be covered prior to nonfunctional surfaces.

Cultured epithelium

The technique of cultured epithelium involves the tissue culture growth of epidermal cells obtained from the prospective recipient, who will require grafting. Often, patients with extensive thermal injury have a disparity between available donor sites and the areas requiring coverage. Additionally, due to the paucity of donor sites, multiple graft harvests from the uninjured areas may be necessary, yielding tissue of progressively inferior quality. Cultured autologous keratinocytes have been used successfully to cover patients with massive skin defects secondary to burn injury.

Use of this technique in major burns may be the only way to prevent major burn complications and the consequent contractures, but it is not without its downsides. Disadvantages include the immensely high cost of the graft as well as its interference with the physical therapy programme (after grafting, the patient has to be immobilised for 7–10 days), easy traumatisation and blistering, breakdown, and lack of long-term durability because of the abnormal histologic architecture.

Complications

Complications after a burn injury may be examined from different perspectives. A thorough knowledge of the potential complications on initial evaluation and admission of the child allows the physician to prevent those complications. Acutely, the most feared complication is death. Others are complications related to the burn injury itself and subsequent organ failure, including death.

Burn complications may be classified as infective and noninfective.

Infective Complications

Infection is the most common and most serious complication of a major burn injury. Sepsis accounts for 50–60% of deaths in burn patients today despite improvements in antimicrobial therapies. Infections include bronchopneumonia, pyelonephritis, thrombophlebitis, and invasive wound infection.

Microbial colonisation of the open burn wounds, primarily from an endogenous source, is usually established by the end of the first week.

After a burn injury, in the absence of topical chemotherapy, the superficial areas of the burn wound contain up to 10^7 organisms per gram of burn tissue within 48 hours following the injury.

Routine administration of prophylactic antibiotics is associated with an increased incidence of yeast colonisation of the gastrointestinal tract and the rapid emergence of resistant gram-negative organisms in the burn wound, although antibiotics do not decrease the incidence of early gram-positive cellulitis. Indeed, even a brief 5- to 7- day course of prophylactic penicillin hastens the emergence of resistant gram-negative organisms. The potential harm caused by widespread use of prophylactic antibiotics has been known since the 1970s, but this practice is still rampart in many African hospitals.

Antimicrobial therapy is directed by bacterial surveillance through routine tri-weekly sputum, urine, and wound cultures, and antibiotics should be given only to treat specific infections. For example, gram-positive cellulitis caused by beta-haemolytic streptococci should be treated with penicillin. It is noteworthy that bacterial counts of $<10^3$ organisms/gm are not usually invasive and allow skin graft survival rates of >90%, without the use of antibiotics.

Methods of diagnosis of burn wound infection include clinical examination, quantitative cultures of a burn wound biopsy, and burn wound histology.

Generic clinical signs of burn wound infection include any of the following:

- spreading peri-wound erythema;
- oedema and/or discoloration of unburned skin at wound margin (usually due to *Pseudomonas* infections);
- rapid eschar separation (bacterial wound sepsis, may be fungal in some environments);
- punctuate haemorrhagic subeschar lesions;
- conversion of partial-thickness burns to full-thickness wounds;
- black or brown patches of wound discoloration;
- green pigment (pyocyanin) visible in subcutaneous fat (*Pseudomonas* infection);
- ecthyma gangrenosa—violaceous or black, erythematous nodular lesions in unburned skin (typically progress to focal necrosis);
- burn wound cellulitis;
- invasive burn wound infection; and

- burn wound impetigo.

Burn wound sepsis can be difficult to distinguish from the usual hyperdynamic, hyperthermic, hypermetabolic postburn state. Blood cultures are commonly negative, and fever spikes are frequently not proportional to the degree of infection.

Clinical diagnosis of sepsis is made by meeting at least three of the following criteria:

- burn wound infection (>10^5 organisms/gm tissue with histologic or clinical evidence of invasion);
- thrombocytopaenia (<50,000 or falling rapidly);
- leukocytosis or leukopaenia (>20,000 or <3,000);
- unexplained hypoxia, acidosis, or hyper- or hypoglycaemia;
- prolonged paralytic ileus;
- hyper/hypothermia (>39°C or <36.5°C);
- positive blood cultures;
- documented catheter or pulmonary infection;
- altered mental status; and
- progressive renal failure or pulmonary dysfunction.

Noninfective Complications

Noninfective complications may include any of the following:

- contractures—positioning and physiotherapy are preventive manoeuvres;
- hypertrophic scars and keloids—early wound closure and appropriate scar management are important in the functional and cosmetic outcomes;
- smoke inhalation syndrome;
- sterile multiorgan failure;
- anaemia;
- malnutrition;
- Curling's ulcers—H_2 blockers or proton pump inhibitors are effective in protecting against gastric ulceration and bleeding; and
- thrombo-embolic complications—estimated to affect between 0.4% and 7% of burn patients.

Additionally, long-term complications of burn scars include skin dyspigmentation, hypertrophic scars, keloid, and chronic nonhealing or unstable scars that may degenerate into squamous cell carcinomas (Marjolin's ulcers). Cutaneous horns may also develop from burn scars. Alopecia and burn syndactylys, digit or limb amputations, corneal perforations, and blindness are other possible postburn complications.

Prognosis and Outcomes

Prompt and appropriate treatment of burn injuries, including resuscitation and appropriate wound care, have led to a reduction in morbidity and mortality. Poor outcomes are the result of inadequate early management. Inadequate fluid resuscitation may lead to renal failure and needless death. Inappropriate triaging of patients leads to a waste of resources as well as the deaths of otherwise salvageable patients. Poor surgical wound management leads to wound infection, delay in wound closure, prolongation of the inflammatory/hypermetabolic phase, and significant malnutrition, especially in the child.

Delayed wound closure, with wound healing by secondary intention, leads to unsightly scars, dyspigmentation, keloids, and contractures. Resultant low self-esteem coupled with limited mobility may lead to children being ostracised from society. Unable to attend school or other social activities, children may be unable to develop to their potential, unable to fit into society, and unable to pursue their dreams. Such children are at risk of posttraumatic stress disorder (PTSD) and other psychological disorders; psychological assessment and treatment are important components of rehabilitation from major burn injury.

Prevention

An old adage holds that "prevention is better than cure". Nowhere else is this proverb more applicable than in trauma, and more specifically in burn injuries. The majority of burn injuries occur among the poor urban populations living under deplorable conditions. Poor infrastructure, including overcrowding, poorly planned housing, and no water access points, lead to rapid spread of fires in these shanty communities. Provision of appropriate housing and decent living conditions are important steps in reducing the scourge of burns to children

Education and government action will likely be needed to abolish child labor practices that place children at greater risk of burn injuries (e.g., underage children who handle fires or hot liquids while cooking). Fire drills in schools should be implemented to help avoid deaths among schoolage children, particularly in boarding schools. Finally, first aid should be taught, which will minimise the burn injuries when they do occur.

Ethical Issues

The management of paediatric burn injuries in the African environment, especially in rural areas, may be complicated by traditional beliefs and practices. Many traditional therapies, such as raw egg mixtures, flour, and liquid paraffin, among other practices, remain harmful and delay appropriate care. Consistent education is urgently needed to both prevent these injuries and improve their outcomes, should they occur.

Child abuse by guardians must also be considered where unusual burn injury patterns or suspicious histories are presented, and appropriate safety measures must be undertaken.

Evidence-Based Research

Table 33.5 presents a comparative study of the use of a biosynthetic skin replacement versus cryopreserved cadaver skin to temporarily cover excised burn skin.

Table 33.5: Evidence-based research.

Title	A multicentre clinical trial of a biosynthetic skin replacement, Dermagraft-TC (DG-TC), compared with cryopreserved human cadaver skin for temporary coverage of excised burn wounds
Authors	Purdue GF, Hunt JL, Still JM Jr, et al.
Institution	Department of Surgery, University of Texas, Southwestern Medical Center, Dallas, Texas, USA
Reference	9063788 (PubMed ID)
Problem	Coverage of excised burn skin.
Intervention	Biosynthetic skin replacement.
Comparison/ control (quality of evidence)	Randomised controlled trial, comparative study.
Outcome/effect	DG-TC was equivalent or superior to allograft with regard to autograft take at post-autograft day 14. DG-TC was also easier to remove, had no epidermal slough, and resulted in less bleeding than did allograft, while maintaining an adequate wound bed. Overall satisfaction was better with DG-TC.
Historical significance/ comments	Improvement in burn care, surgical technique of covering wounds and its quality.

Key Summary Points

1. When a child is burned, his or her life is in danger.
2. Burn injuries are preventable.
3. Early and appropriate management of burn injuries significantly reduces associated morbidity and mortality.
4. Tetanus prophylaxis *must* be administered to burn victims. Prophylactic antibiotics are *not* indicated, and should *not* be used.
5. Antibiotics in burn care should be used only in preoperative prophylaxis and in cases of established infection.
6. Making a list of all the potential complications each week—both acute and long term—and taking action to prevent them would improve the outcome. Early splinting of limbs, early tangential excision and skin grafting, and physical therapy should all be instituted promptly.

Suggested Reading

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