Thermal Burns

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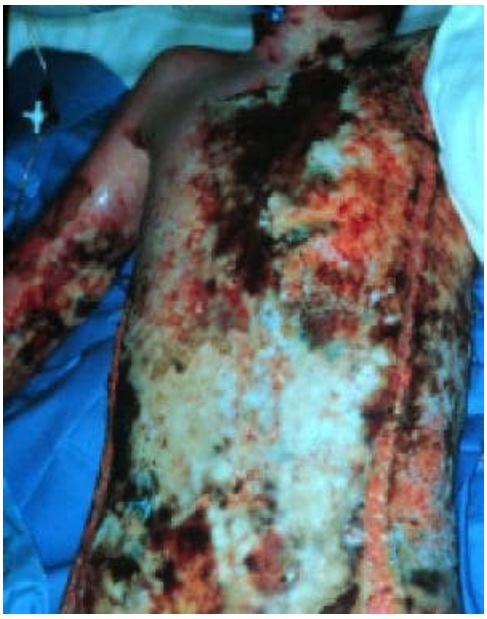
OVERVIEW

Overview

Burn wounds can be classified into 6 separate groups based on the mechanism of injury: scalds, contact burns, fire, chemical, electrical, and radiation. [1] The first 3 types of burns are addressed in this article. Scald burn injuries can be caused by liquids, grease, or steam. Liquid scalds can be further divided into spill and immersion scalds. Fire burn injuries can be divided into flash and flame burns. The mechanism of burn injury can be used as a predictor of outcome. For example, patients with flame burns and electrical burn injuries often require hospitalization. In contrast, most patients with burns caused either by contact with hot surfaces or sun exposure are managed as outpatients.

Burns exert a catastrophic influence on people in terms of human life, suffering, disability, and financial loss. [2] Burns are estimated to cause approximately 180,000 deaths annually worldwide, mostly in low- to middle-income countries. [3] Burns or corrosions accounted for the primary diagnosis in 489,000 visits to emergency departments (EDs) in the United States in 2017, [4] while in 2016, there were approximately 40,000 burn-related hospitalizations in the United States, 30,000 of which were at specialized burn centers. [5] Work-related burns account for 20-25% of all serious burns. [6] See the image below.



Invasive burn wound infection implies that bacteria or fungi are proliferating in eschar and invading underlying viable tissues. These wounds display a change in color, new drainage, and often a foul odor. These infections are life-threatening.

A retrospective, cross-sectional study by Shields et al found that in the United States between 2016 and 2018, the direct inpatient health-care costs for initial encounters with tap water scald burns was $206.69 million, with emergency department (ED) visits costing $29.79 million. The report used the National (Nationwide) Inpatient Sample (NIS) and Nationwide Emergency Department Sample (NEDS) databases. [7]

Risk factors

People of all ages are susceptible to minor burn injury. The highest incidence occurs during the first few years of life and in persons aged 20-29 years. The mechanism of minor burn injury varies considerably with the age of the victim. Minor burns in children younger than 4 years are caused primarily by contact with hot surfaces and by liquid scalds, with scalds accounting for approximately 75% of burns in children under age 5 years.

At later ages, a large number of heat sources (eg, hot surface, liquid scald, grease scald, radiation, chemical) cause burn injury. These varied heat sources reflect the many different daily experiences of older children and adults. [8, 9, 10]

A study by Palilonis et al, using the 2014 Nationwide Emergency Department Sample, found that among patients aged 13-25 years, 12.9% of 20,787 burn injury–related emergency department visits involved a codiagnosis of intoxication. The investigators also determined that within this age group, burn-related visits with a codiagnosis of intoxication were 1.34 times more likely to be associated with flame burns. [11]

In the pediatric population, thermal injury is a leading cause of morbidity and mortality related to inadvertent trauma. [12] A British study, by Stewart et al, found that risk factors for scald injuries in children younger than 5 years old include the presence of hot drinks within reach of these youngsters and a lack of education for these children concerning “rules about climbing on kitchen objects," “what to do or not do when parents are cooking,” and “hot things in the kitchen.” [13] In a large US study, from 2016 to 2018, Bentivegna et al showed that tap water warmer than 49ºC (120ºF) is a significant cause of scald burns in children under 5 years, with the majority of victims being boys, non-Hispanic Whites, children on public insurance, and youngsters from urban locations. Primary prevention and education targeting caregivers remain the best methods for avoiding pediatric burns. [14]

Frequency and incidence

Serious burn injuries occur most commonly in males (67%). [5] The highest incidence of serious burn injury occurs in young adults (20-29 y) followed by children younger than 9 years. Individuals older than 50 years sustain the fewest number of serious burn injuries (2.3%). Major causes of severe burn injury are flame burns (37%) and liquid scalds (24%). For children younger than 2 years, liquid scalds and hot surface burns account for nearly all serious burn injuries. After age 2 years, flame burn is the most common cause of serious burn injuries, accounting for nearly one third of all serious burns.

A British study, by Battle et al, looking at 1387 cases presenting to the ED of a regional burn center, found the most common thermal injuries to be reported in children under age 16 years to be scalds and contact burns (41.0% and 40.6%, respectively). [15]

Similarly, using emergency department and inpatient database figures (2005-2013) obtained through California’s Office of Statewide Health Planning and Development, Sheckter et al determined that of 16,480 minor burn encounters in patients under age 18 years, 76.3% involved scald or contact burns. [16]

In much older persons (80 y and older), hot surface exposure is a major cause (22%) of serious burns.

Of hospitalized burn patients, 5% die as a result of their burn injuries; most of these deaths are from flame burns. Liquid scald burns account for the second largest number of deaths.

The aforementioned study by Shields and colleagues reported that in the United States between 2016 and 2018, tap water scald burns were responsible for 52,088 (weighted) ED visits, 7270 (weighted) hospitalizations, and 110 hospital-based deaths. Of inpatient and ED visits, 35.4% and 16.1%, respectively, involved multiple body surfaces. [7]

In structural fires, approximately one half of all burn victims, many with only moderate burns of less than 40% body surface area, die of asphyxiation or carbon monoxide poisoning before reaching the hospital.

Flame and flash burn injuries

Flame burn injuries are associated with recurring scenarios regarding the most likely burn victims, the circumstances surrounding the burn, the burned victim's response to the situation, and the role of garments in the burn injury. Duration of exposure to flame, associated trauma, and inhalation injury contribute to the severity of flame burns. Flash and flame burns are the most common causes of hospital admission for burns in adults. The white population is most commonly involved (67%), and the highest incidence occurs in those aged 15-29 years. A flammable liquid is involved in most cases (66%); gasoline is the most common liquid (63%). [17] The high incidence of gasoline burns during the summer months reflects the increased use of gasoline products for farming or recreational purposes (eg, bonfires, burning leaves, boating, yard work). The most common contributing factor in flame burn injuries is the consumption of alcohol (26%).

Patient or bystander response

The patient's or bystander's response to burn incidents has considerable influence on the magnitude of burn injury. A timely and effective response reduces the magnitude of burn injury (except when flammable liquids are involved). When flammable accelerants are present, the burning process persists even when the victim is rolling on the ground. In this setting, removal of the burning garments or smothering the flames is more likely to be an effective measure.

Recent decrease in deaths from burn injuries

During the past 2 decades, the overall median length of hospital stay and number of emergency department (ED) visits, as well as deaths from burn injuries, have declined. This decline has been attributed to improved firefighting techniques and improved emergency medical services. The use of smoke detectors has significantly reduced the severity of burn injuries with an estimated 80% reduction in mortality and 74% decline in injuries from residential fires. Educational programs reminding homeowners to lower the thermostat on water heating units as well as teaching children to extinguish flaming cloth by stopping, dropping, and rolling have had a significant impact. Consequently, all medical leaders agree that the best treatment of burn injuries is prevention.

Burn injuries are extremely complex and elicit physiologic and metabolic interactions involving all major organ systems. These pathophysiologic changes occur in a time-dependent manner. One of the major goals of this chapter is to describe a system of care of burn injuries and to review current modes of surgical therapy with discussions of wound care and modern burn dressings.

Emergency medical technicians, firefighters, and ED health workers should be wearing emergency medical examination gloves that meet the stringent standards of the National Fire Protection Association (NFPA). [18] The authors strongly prefer powder-free latex-free emergency medical examination gloves. On September 24, 2008, 13 health professionals filed a Citizen's Petition with the US Food and Drug Administration (FDA) to ban cornstarch powder on all medical gloves. [19] Cornstarch has been documented as promoting wound infection and causing serious peritoneal adhesions and granulomatous peritonitis, and it is a well-documented vector of the latex allergy

epidemic. In January 2017, an FDA rule took effect banning the use of powdered surgeon's gloves, powdered patient examination gloves, and absorbable powder used for lubrication of surgeon's gloves. [20]

Pathophysiology

Skin Anatomy and Function

Skin is the largest organ of the body. It has 3 major tissue layers.

Epidermis

The outermost layer, the epidermis, is composed of stratified epithelium. Epidermis has 2 components, an outer layer of anucleate cornified cells (stratum corneum) that covers inner layers of viable cells (Malpighian layers) from which the cornified surface cells arise by differentiation. The stratum corneum acts as a barrier to impede the entrance of microorganisms and toxic substances while allowing the body to retain water and electrolytes. Malpighian layers provide a continuous production of cornified cells. Malpighian layers can be further subdivided into the germinal basal cell layer, stratum spinosum, and stratum granulosum.

Dermis

Beneath the epidermis is the dermis, which is composed of a dense fibroelastic connective-tissue stroma containing collagen and elastic fibers and an extracellular matrix termed ground substance. This amorphous matrix is made up of an acid mucopolysaccharide protein combined with salts, water, and glycoproteins; it may contribute to salt and water balance, serve as a support for other components of the dermis and subcutaneous tissue, and participate in collagen synthesis. The dermal layer contains an extensive vascular and nerve network, special glands, and appendages that communicate with the overlying epidermis.

The dermis is divided into 2 parts.

1. The most superficial portion, the papillary dermis, is molded against the epidermis and contains superficial elements of the microcirculation of the skin. It consists of relatively cellular, loose connective tissue with smaller, fewer collagen and elastic fibers than the underlying reticular dermis. Within the papillary dermis, dermal elevations indent the inner surface of the epidermis. Between the dermal papillae, the downward projections of the epidermis appear peglike and are termed rete pegs.

2. In the reticular portion of the dermis, collagen and elastic fibers are thicker and greater in number. Fewer cells and less ground substance are found in the reticular dermis than in the papillary dermis. Thickness of the dermis varies from 1-4 mm in different anatomic regions and is thickest in the back, followed by the thigh, abdomen, forehead, wrist, scalp, palm, and eyelid. Thickness varies with the individual's age. It is thinnest in the very old, where it is often atrophic, and in the very young, where it is not fully developed.

Subcutaneous tissue

The third layer of skin is subcutaneous tissue, which is composed primarily of areolar and fatty connective tissue. This layer shows great regional variations in thickness and adipose content. It contains skin appendages, glands, and hair follicles. Hair follicles extend in deep narrow pits or pockets that traverse the dermis to varying depths and usually extend into the subcutaneous tissue. Each hair follicle consists of a shaft that projects above the surface and a root that is embedded within the skin.

Apocrine and eccrine sweat glands

There are 2 types of sweat glands in skin: apocrine and eccrine.

Apocrine glands are epitrichial because they have a duct that opens into a hair follicle. Apocrine glands are largely confined to the axillary and perineal region and do not become functional until just after puberty.

Eccrine glands are simple, coiled, tubular glands usually extending into the papillary dermis. Eccrine glands are atrichial because their duct opens onto the skin surface independently of a hair follicle. Eccrine glands are found over the entire body surface, except the margins of the lips, eardrum, inner surface of the prepuce, and glans penis.

Sebaceous glands are simple or branched alveolar glands, usually connected to the hair follicles. Sebaceous glands unconnected with hair follicles occur along the margin of the lips, in the nipples, in the glans and prepuce of the penis, and in the labia minora. Depending on the depth of burn injury, epithelial repair can be accomplished from local epithelial elements and skin appendages.

When skin is burned, the damaged stratum corneum allows the invasion of microorganisms, and the Langerhans cells, which mediate local immune responses, also are damaged. In burn patients with severe injuries, their systemic immune response is diminished, making them susceptible to serious infections.

Heat transfer from heating agent to skin

Severity of burn injury is related to the rate at which heat is transferred from the heating agent to the skin. Rate of heat transfer depends on the heat capacity of the agent, temperature of the agent, duration of contact with the agent, transfer coefficient, and specific heat and conductivity of the local tissues.

1. Heat capacity: Capacity of a material to hold heat energy is determined by both the specific heat and the heat capacity of the material.

2. Specific heat of a material: This is defined as the ratio of the amount of heat required to raise a specific mass of the material 1 degree in temperature, to the amount of heat required to raise an equal mass of a reference substance (usually water) 1 degree in temperature.

3. Heat capacity: This refers to a quantity of heat a material contains when it comes in contact with skin. Quantity of heat stored depends on the specific heat of the material and the amount and temperature of the material.

The importance of heat capacity as a determinant of severity of burn injury is best illustrated by comparing the amount of heat stored in 10 g of 2 different materials (copper and water) heated to the same temperature (100°C). Specific heat of water is 4.2178 W x sec/g x K (watt times seconds of heat per gram mass times degrees Kelvin). If these 2 materials come in contact with skin, they give up their heat by cooling while skin accepts the heat by increasing its temperature.

If the temperature of each material decreases by 60°C, water gives up 2530 W x sec of heat, whereas copper transfers only 230 W x sec of heat. Even if the initial temperatures of the 2 materials are identical, heat available from water is much more likely to produce a severe injury. The specific heat of water (most common cause of scald burns) is the highest of all the gases, metals, and solids tested to date, with the exception of ammonia and ether.

Temperature

Initial temperature of a material at the instant of contact is also an important determinant of burn severity. Many materials (eg, water) cannot be heated beyond a certain temperature without changing state. Water can only be heated to 100°C at atmospheric pressure before it ceases to be a liquid and vaporizes. Because more joules are required to produce steam, this additional heat transfer accounts for the severe burns caused by steam injury. When other liquids reach a specific temperature, they ignite or oxidize by combining with oxygen.

The flash point of the liquid is the temperature at which the vapors of a volatile liquid mixed with air spontaneously ignite. A flammable liquid is defined as any liquid having a flash point less than 37.8°C. Liquids with a flash point above this temperature are considered combustible. In addition to their high temperatures, burning liquids also may ignite the victim's clothing, thereby further exacerbating severity of the injury.

Duration of contact

Human skin can tolerate temperatures as high as 44°C (111°F) for a relatively long time (6 hours) before irreversible injury occurs [21] . Temperatures greater than this level cause an almost logarithmic increase in tissue destruction. Duration of contact between a liquid and skin depends on both the viscosity of the liquid and the manner in which it is applied to the victim's skin. When hot liquid is splashed on a person, as in a spill scald, it usually flows down the body in a rate of descent that depends on the fluid's viscosity. Although water streams to the ground unless impeded by clothing, viscous oils and greases usually cling to a victim's skin, prolonging duration of exposure and extent of injury.

In immersion scalds, duration of contact between the hot liquid and the skin is considerably longer than that with spill scalds, thereby increasing the severity of injury. Certain populations are at high risk of suffering immersion scald burns, including children younger than 5 years, older persons (65 years and older), and disabled persons. Individuals in these high-risk groups tend to have a slower reaction time and a physical inability to escape from hot water. Immersion burns commonly cover a large percentage of total body surface area (TBSA), almost twice that of other scald burns, which contributes to their high rate of morbidity and mortality. [22, 23]

Child abuse and immersion scald burns

Child abuse accounts for a large proportion of immersion scald burns. Nonaccidental burn injuries can occur due to a caretaker's maltreatment or negligence and are associated with greater mortality and longer hospitalization than accidental burns. Immersion burns caused by child abuse can be distinguished from accidental burns by the pattern and site of the burn, histories given by the caretaker and patient, and a medical history of scars representing previous abuse. Nonaccidental burns are often characterized by a larger total body surface area (TBSA; usually >10%), greater severity (third-degree burns), clear-cut edges, and a "stocking" or "glovelike" pattern; the child's lower extremity is frequently involved. [24] Spill scald burns, on the other hand, more often have uneven, fuzzy edges as a result of the victim's attempts to escape the hot liquid. Burns from abuse tend to occur on the back of hands and feet, the buttocks and perineum, and legs. Accidental burns, such as those caused by a child spilling a cup of coffee, more often cause burns on the head, trunk, and palmar surface of hands and feet. Physical evidence of previous injuries, such as craterlike cigarette burn scars or bruises, also suggests abuse.

Heat transfer

Even when a substance possesses sufficient heat to cause a burn injury, it will not do so unless its heat can be transferred to the skin. This ability to transfer heat between 2 different materials is regulated by the heat transfer coefficient, which is defined as the amount of heat that passes through a unit area of contact between 2 materials when the temperature difference between these materials is 1 degree.

Three different methods of heat transfer exist: conduction, convection, and radiation. The simplest method of heat transfer is conduction, which occurs when a hot solid object comes in direct contact with the skin. Convection is the transfer of heat by a material that involves the physical movement of the material itself and is determined by heat conduction and by energy storage and mixing motion. Convection is most important as the mechanism of energy transfer between skin and a heated liquid or gas. Hot water spilling on skin transfers heat by convection between the water droplets and the skin surface. Steam or very hot air also transfers heat to the skin by convection.

Tissue conductivity

Conductivity of the specific tissue involved has a significant influence on the extent of burn injury. Heat transfer within skin is influenced by the thermal conductivity of the heated material, the area through which heat is transferred, and the temperature gradient within the material. Water content, natural oils or secretions of the skin, and the presence of insulating material (eg, cornified keratin layer of skin) influence tissue conductivity. In addition, alterations in local tissue blood flow produce a profound effect on heat transfer and distribution. Inability to conduct heat away from a contact point efficiently results in varying degrees of tissue injury.

Because skin is a relatively poor conductor of heat, it provides an extensive barrier to heat injury. The degree to which it resists injury depends on its anatomic configuration. Its uppermost layer, the epidermis, is relatively uniform in thickness in all body regions (0.07-0.12 mm) except for the soles and palms, where it attains a greater thickness (0.8-1.4 mm). The rarity of full-thickness injury to the palms and soles of the feet can be attributed to their thick epithelial cover.

The ultimate outcome of a burn injury also is influenced by the depth of epidermal appendages in the burned tissue, which varies according to the age of the patient. Very young and old individuals have superficial appendages, which make both groups more susceptible to full-thickness burn injury. By contrast, the epidermal appendages of the human scalp and male beard are very deep, making these sites more refractory to severe burn injury.

Burn wound injury

During the first day after burn injury, 3 concentric zones of tissue injury characterize a full-thickness burn: zones of coagulation, stasis, and hyperemia. [25] The central zone of coagulation has the most intimate contact with the heat source. It consists of dead or dying cells as a result of coagulation necrosis and absent blood flow. It usually appears white or charred. The intermediate zone of stasis usually is red and may blanch on pressure, appearing to have an intact circulation; however, after 24 hours, circulation through its superficial vessels often has ceased. Petechial hemorrhages may be present. By the third day, the intermediate zone of stasis becomes white because its superficial dermis is avascular and necrotic. The outer zone of hyperemia is a red zone that blanches on pressure, indicating that it has intact circulation. By the fourth day, this zone has a deeper red color. Healing is present by the seventh day.

Transformation of the zone of stasis to coagulation occurs and has been related to many factors, including progressive dermal ischemia. Experimental studies have implicated prostaglandins, histamine, and bradykinin as the chemical mediators of this progressive vascular occlusion. They can produce edema by altering endothelial cell and basement membrane function to enhance permeability. When this ischemia persists, the zone of stasis eventually becomes a full-thickness burn injury.

When Robson et al discovered various prostaglandin derivatives in burn wounds, they suggested that an imbalance in the vasoconstrictive and vasodilatory prostanoids produces a progressive tissue loss in the zone of stasis. [26] In acute burn wounds, an increased level of oxygen free radicals, such as xanthine oxidase, appeared to be involved in the formation of burn edema. This edema formation can be attenuated by pretreatment with xanthine oxidase inhibitors. [27]

Systemic inflammatory response

In patients whose burns exceed 30% of TBSA, cytokines and other mediators are released into the systemic circulation, causing a systemic inflammatory response. Because vessels in burned tissue exhibit increased vascular permeability, an extravasation of fluids into the burned tissues occurs. Hypovolemia is the immediate consequence of this fluid loss, which accounts for decreased perfusion and oxygen delivery. In patients with serious burns, release of catecholamines, vasopressin, and angiotensin causes peripheral and splanchnic bed vasoconstriction that can compromise in-organ perfusion. Myocardial contractility also may be reduced by the release of inflammatory cytokine tumor necrosis factor-alpha.

In deep third-degree burns, hemolysis may be encountered, necessitating blood transfusions to restore blood loss. A decrease in pulmonary function can occur in severely burned patients without evidence of inhalation injury from the bronchoconstriction caused by humoral actors, such as histamine, serotonin, and thromboxane A2. A decrease in lung and tissue compliance is a manifestation of this reduction in pulmonary function. Burned skin exhibits an increased evaporative water loss associated with an obligatory concurrent heat loss, which can cause hypothermia.

A significant proportion of the morbidity and mortality of severe burns is attributable to the ensuing hypermetabolic response. This response can last as long as a year after injury and is associated with impaired wound healing, increased infection risk, erosion of lean body mass, impaired rehabilitation, and delayed integration of the burn patient into society.

Pharmacologic and nonpharmacologic strategies are being used to reverse the catabolic effect of thermal injury. Nonpharmacologic approaches include early excision and wound closure, aggressive management of sepsis, elevation of the environmental temperature, continuous high carbohydrate/high protein enteral feeding, and early institution of resistive exercise programs. Pharmacologic modulation of the postburn hypermetabolic response has been achieved through administration of recombinant human growth hormone, low-dose insulin infusion, use of synthetic testosterone analog (oxandrolone), and beta blockade with propranolol.

Nutritional support

Because burn injury causes a hypermetabolic state that is characterized by a dramatic increase in resting energy expenditure, nutritional support is essential, especially via the enteral route, to reduce intestinal villous atrophy. In patients with severe burns, the Toronto formula can reliably be used to predict energy expenditure. [28]

Deitch et al reported a syndrome of decreased bowel mucosal integrity, capillary leak, and decreased mesenteric blood flow, that allowed bacterial translocation into the portal circulation. [29] These translocated bacteria significantly alter hepatocyte function and spread systemically to cause systemic sepsis. Adequate resuscitation that ensures mesenteric blood flow can prevent potential development of multisystem organ failure. Enteral nutrition with glutamine has a tropic effect on the enterocytes that preserve mucosal integrity.

Infection

In patients with major burn injuries, infection remains the major cause of death. Immune consequences of this injury have been identified and are specific deficits in neutrophil chemotaxis, phagocytosis, and intracellular bacterial killing. Cell-mediated immunity, as measured by skin testing, also is compromised and has been related to both decreased lymphocyte activation and suppressive mediators present in the serum of burn patients. A reduction in immunoglobulin synthesis also has been encountered in these seriously ill patients.

In terms of infection treatment in critically ill patients, the substantially altered physiology remains a challenge, since this causes an extreme decrease in antibiotic concentration in tissue, leading to failure to achieve a therapeutic dose and resulting in antibiotic resistance. [30]

A retrospective study by Fochtmann et al indicated that in patients with extensive burns, risk factors for candidemia include female sex, gastrointestinal complications that require surgery, nongastrointestinal thromboembolic complications, inhalation trauma, and younger age (possibly because younger patients survive longer with extensive burns than do older individuals). The study involved 174 patients, 20 of whom developed candidemia a median of 16 days after admission to an intensive care unit. [31]

Burn shock

Severe burn injury causes a coagulation necrosis of tissue that initiates a physiologic response in every organ system that is directly proportional to the size of the burn. Tissue destruction results in increased capillary permeability with profound egress of fluid from the intravascular space to the tissues adjacent to the burn wound. Inordinate amounts of fluid are lost by evaporation from the damaged surface that is no longer able to retain water. This increase in capillary permeability, coupled with evaporative water loss, causes hypovolemic shock.

Other physiologic changes

Other physiologic changes seen with thermal injury are, to a large extent, a response to diminished circulating blood volume. Immediate cardiovascular response to thermal injury is a reduction in cardiac output accompanied by an elevation in peripheral vascular resistance. In the absence of heart disease, ventricular ejection fraction and velocity of myocardial fiber shortening are actually increased during thermal injury. [32] With replacement of plasma volume, cardiac output increases to levels that are above normal. This hyperdynamic state is a reflection of the hypermetabolic flow phase of thermal injury.

Alterations in pulmonary function after burn injury are similar to those seen with other forms of traumatic injury. Minute ventilation usually increases immediately. After resuscitation, respiratory rate and tidal volume progressively increase, resulting in minute ventilation that may be twice normal. Pulmonary vascular resistance also increases after burn injury, which may be a manifestation of the release of vasoactive amines and other mediators. [33] This increase in pulmonary vascular resistance may provide a protective effect during fluid resuscitation by reducing pulmonary capillary hydrostatic pressure and lowering susceptibility to pulmonary edema. In the absence of inhalation injury, no significant change occurs in pulmonary capillary permeability after cutaneous thermal injury. [34]

In the immediate postburn period, glomerular filtration rate and renal blood flow are reduced in proportion to the reduction in intravascular volume. GI dysfunction also appears to be proportional to the magnitude of thermal injury. In patients with burned areas in excess of 25% TBSA, gastroparesis is commonly noted until the third to fifth postburn day.

Burn shock may be complicated by an acute erythrocyte hemolysis caused by both direct heat damage and by a decreased half-life of damaged red blood cells (RBCs). In major burns, the RBC mass may be reduced 3-15%. RBCs also exhibit a decreased half-life because of a microangiopathic hemolytic anemia that may persist for up to 2 weeks.

Quantifying Burn Severity

Burn Depth

Burned patients' survival is related to the following factors: burn size/depth, age, presence of inhalation injury, and patient comorbidity. Depth of burn injury is usually classified according to degrees.

First-degree burns

In first-degree burns, minor epithelial damage of the epidermis exists. Redness, tenderness, and pain are the hallmarks of this injury. Blistering does not occur, and 2-point discrimination remains intact. Healing takes place after several days without scarring. Because the epidermal barrier remains intact, metabolic response and risk of infection are minimal. Most common causes of first-degree burns are flash burns and sunburns.

Second-degree burns

Superficial partial-thickness and deep partial-thickness burns are the 2 types of second-degree burns. In these burn injuries, some portion of the skin appendages remains viable, allowing epithelial repair of the burn wound without skin grafting. Superficial partial-thickness burn involves the epidermis and superficial (papillary) dermis, often resulting in thin-walled, fluid-filled blisters. These burns appear pink, moist, and soft and are exquisitely tender when touched by a gloved hand. They heal in approximately 2-3 weeks, usually without scarring, by outgrowth of epithelial buds from the viable pilosebaceous units and sweat glands residing in the papillary and reticular dermis.

Deep partial-thickness burns extend into the reticular dermis. Skin color is usually a mixture of red and blanched white, and capillary refill is slow. Blisters are thick-walled and commonly ruptured. Two-point discrimination may be diminished, but pressure and pinprick applied to the burned skin can be felt. Superficial partial-thickness burns usually re-epithelialize 7-10 days after injury. Risk of hypertrophic

scarring is very small. For deep partial-thickness burns, tissue may undergo spontaneous epithelialization from the few viable epithelial appendages at this deepest layer of dermis and heal within 3-6 weeks (if no infection arises).

Because these burns have less capacity for re-epithelializing, a greater potential for hypertrophic scar formation exists. In deep partial-thickness burns, treatment with topical antimicrobial dressings is necessary to prevent infection as the burn wound heals. Contraction across joints, with resulting limitation in range of motion, is a common sequela. Splash scalds often cause second-degree burns.

Third-degree burns

Third-degree burns are full-thickness burns that destroy both epidermis and dermis. The capillary network of the dermis is completely destroyed. Burned skin has a white or leathery appearance with underlying clotted vessels and is anesthetic. Unless a third-degree burn is small enough to heal by contraction (< 1 cm), skin grafting always is necessary to resurface the injured area. Immersion scalds, flame burns, and chemical and high-voltage electrical injuries cause third-degree burns.

Fourth-degree burns

Fourth-degree burns cause full-thickness destruction of the skin and subcutaneous tissue, with involvement of the underlying fascia, muscle, bone, or other structures. These injuries require extensive débridement and complex reconstruction of specialized tissues and invariably result in prolonged disability. Fourth-degree burns result from prolonged exposure to the usual causes of third-degree burns.

Burn size

Accurate assessment of initial burn injuries is important to guide subsequent burn wound treatment. If it is anticipated that a burn wound will take longer than 21 days to heal spontaneously, surgical débridement and grafting are required to reduce the chance of hypertrophic scarring.

The "rule of nines" is a practical technique for estimating the extent of TBSA involved in a burn injury. This approach divides the major anatomic areas of the body into percentages of TBSA. For the adult, it allots 9% of the TBSA to the head and neck and to each upper extremity, 18% each to the anterior and posterior portions of the trunk, 18% to each lower extremity, and 1% to the perineum and genitalia. The patient's palm area represents approximately 1% of TBSA and can be helpful in calculating scattered areas of involvement.

In estimating the extent of burn injury, the extent of involvement of each anatomic area (eg, an arm or leg) must be calculated separately, and the total is derived from the simple addition of the burned anatomic sites. A small difference between TBSA of the adult and infant reflects the size of the infant's head (18%), which is proportionally larger than that of the adult, and the lower extremities (14%), which are proportionally smaller than those of the adult. Lund-Brower charts with age-appropriate diagrams can be used to better estimate the area of burn injury in children. Remember that first-degree burns are not included in the calculation of burn size.

A study by Swords et al suggested that in children transferred from a referring institution to a burn center, the referring institution often overestimates the patient’s burned TBSA, leading to overresuscitation. The investigators found that the average burned TBSA estimated by referring institutions was 15.5%, compared with an average of 9.5% at the burn center in the study to which pediatric patients were sent. In addition, overestimation of burned TBSA of at least 5% was found to be associated with overresuscitation by at least 10 mL/kg. [35]

Age and burn size

A direct but inverse relationship exists between age and survival for any burn size. While the mortality of a 40% TBSA burn in a 20-year-old patient is approximately 8%, the mortality of this same injury in someone older than 70 years is 94%. The higher mortality of older patients with burn injuries is attributed to their preexisting medical conditions, including cardiac, pulmonary, renal, and hepatic dysfunction. Similarly, children younger than 1 year survive large burns at a reduced rate.

When possible, make an attempt to further subdivide TBSA into partial-thickness and full-thickness percentages to facilitate patient categorization and subsequent management. Depth of burn injury can be evaluated by numerous techniques, including burn wound biopsy, vital dyes, ultrasound studies, fluorescein fluorometry, thermography, light reflectance, MRI, and laser Doppler flowmetry. Most of these techniques are not used in standard practice, but laser Doppler and light reflectance show promise in measuring depth of burn injury.

Patient categorization

New guidelines provided by a panel of experts place burn injuries into three categories: (1) severe burn, (2) burn with complication risk, and (3) nonsevere burn. [36]

Classification depends on [36] :

- Extent, depth, and location of the burn injury
- Age of patient
- Etiologic agents involved
- Presence of inhalation injury
- Coexisting injuries or preexisting illnesses

Treatment recommendations are based on severity. [36]

Severe burn

In adults, the following characteristics are involved [36] :

- TBSA >20%
- Third-degree burn TBSA >5%
- Smoke inhalation
- Deep burns in areas of functional risk (face, hands, feet, perineum)

In pediatric patients, the following characteristics are involved [36] :

- TBSA >10%
- Third-degree burn TBSA >5%
- Patient age < 1 year
- Severe comorbidities
- Smoke inhalation injuries
- Deep burns in locations that are function sensitive (face, hands, feet, perineum, flexure lines)
- Circular burns
- Electrical or chemical burns

Burn with risk of complication

In adults, the following characteristics are involved [36] :

- TBSA >10% to < 20%
- Third-degree burn TBSA 3-5%
- Patient age >75 years
- Severe comorbidities
- Known or suspected smoke inhalation
- Deep circular burn
- Superficial burns in areas that are function sensitive (face, hands, feet, perineum, skin folds).

In pediatric patients, the following characteristics are involved [36] :

- TBSA >10%
- Third-degree burn TBSA >5%
- Patient age < 1 year

Nonsevere burn

In adults, the following characteristics are involved [36] :

- Second-degree burn TBSA < 10%
- Third-degree burn TBSA < 3%
- Patient age < 75 years
- No circular burns
- No burns on locations that are function sensitive (face, hands, feet, perineum)

In pediatric patients, the following characteristics are involved [36] :

- Thermal burn with < 5% second-degree TBSA
- Patient age >1 year
- No comorbidities
- No circular burns
- No burns on function-sensitive areas.

Prehospital Care

Optimal management of burn victims is provided by an echelon system of burn care that is developed on a regional basis. Organization of burn care should begin at the site of injury and continue through prehospital care and transportation to the closest burn center, or to the closest ED with advanced life support capability, followed by transfer to a burn center when appropriate. Education in prehospital care should be prioritized and should address timely transfer, cooling of the wound, wound coverage, fluid resuscitation, pain control, and airway management. [37]

Inhalation injuries and intubation

Fires usually emit smoke, which victims may inhale, especially in closed spaces. Smoke inhalation can cause pulmonary parenchymal damage and carbon monoxide and other toxic poisonings, which may have life-threatening consequences. The prehospital care provider should look for signs of inhalation injury (eg, dyspnea, burns of the mouth and nose, singed nasal hairs, sooty sputum, brassy cough). If one or more of these signs is present, administer humidified oxygen with a nonrebreathing reservoir mask at a rate of 10-12 L/min. A patient who is not breathing should be intubated and ventilated with 100% oxygen. (See also Emergent Management of Thermal Burns.)

Accurate initial estimation of TBSA and inhalation injury are a crucial part of prehospital assessment and management. While the primary team tends to intubate the more severe burns, inhalation injury and the depth of burn injury are often not accurately estimated. Harish et al found that estimation of TBSA was accurate in only one third of all transferred patients. [38] Consequently, prehospital intubation is done

unnecessarily in more than one third of patients before arrival. Generally, prehospital intubation has no significant impact on overall mortality. [37]

Corticosteroids are no longer used for inhalation injuries in the prehospital period. A retrospective study on 2509 patients in Germany also showed no significant influence of prehospital administration of corticosteroids on mortality or pulmonary complications. [37]

Treatment of burn shock

Treatment of burn shock in the prehospital setting should consist of elevating the patient's legs 12 inches off the ground and administering humidified oxygen. If rescue personnel have advanced life support capability and transport time may be prolonged, these treatments are complemented by intravenous (IV) fluid administration. Fluid resuscitation need not be initiated if patient is transported to the hospital in less than 30 minutes. When transport time is longer than 30 minutes, the indications for fluid resuscitation, following the Parkland formula, are thermal injuries involving greater than 20% of TBSA or evidence of burn shock. [36] (See also Guidelines)

Fluid resuscitation is not recommended for children at the scene of the accident because of the difficulties encountered in cannulating small veins. When fluid resuscitation is indicated in an adult, administer 20 mL/kg lactated Ringer solution though a large-bore percutaneous catheter, preferably inserted through unburned skin. The arm is the preferred site for cannulation. Determine IV flow rates by the patient's clinical status.

Cool water treatment

Cold water historically had been administered to acute burn injuries. Physiologically, heat increases capillary permeability and the release of histamine by damaged mast cells. This pathway was inhibited in rat models by cooling, which essentially controls the bradykinin-mediated vascular reaction.

In the absence of shock, adults with burn TBSA of less than 20% and children with burn TBSA of less than 10% should be cooled. Application of cold water within 3 hours post injury for at least 20 minutes, maintaining the surface temperature below 44ºC, leads to pain relief and fast reepithelization and scar reduction. More importantly, cooling the patient before arrival at the ED has a significant positive effect on reducing admission rate, length of hospitalization, and the need for skin graft. Cooling does not contribute to hypothermia in the patient, but it has been observed, nonetheless, that the technique is underused in many healthcare systems. [37]

Wound coverage

To prevent contamination, nonadherent dressing can be applied to the burn wound before transportation. However, most studies show that the majority of patients arrive without any wound dressing, signifying a "scoop and run" approach by first responders. Reports show no change in mortality whether patients arrive in their own clothes or with the burn covered by any sort of dressing.

Emergency Department Treatment

Primary survey

All burn patients arriving at the ED should receive a thorough advanced trauma life support (ATLS) workup. When the patient reaches the ED, perform a rapid initial assessment of respiratory and cardiovascular status (ABC) and intervene if required. Then, establish the extent of the burn injury, and determine the need for special procedures. ED treatment focuses on airway and respiratory care as well as fluid resuscitation. [39]

Secondary survey

When ventilatory and circulatory competence is restored, perform a secondary survey. Wash all burned clothing and skin with cool water. Immerse the burn wound in cold water (1-5°C) for approximately 30 minutes if transport cannot be undertaken immediately. This must be initiated as soon as possible because cooling has no therapeutic benefit if delayed more than 30 minutes after the burn injury. Local cooling of less than 9% of TBSA can be continued longer than this 30-minute interval to relieve pain; however, prolonged cooling of a larger TBSA can cause severe hypothermia, which may result in cardiac arrest. Do not apply ice directly to the burn wound because it may result in increased tissue injury through frostbite.

Inhalation injury

Inhalation injury has a significant impact on the survival of burn patients. It has three components: upper airway swelling, acute respiratory failure, and carbon monoxide intoxication. The natural history of upper airway burn injury is the development of edema that narrows the airway 12-24 hours after injury. Consequently, intubation rather than observation is recommended in patients with signs of upper airway injury, such as stridor, inspiratory grunting, wheezing, or tachypnea. [40]

However, burn victims are at high risk for unnecessary intubation in the prehospital setting due to fear of airway loss. Guidelines recommend avoiding routine intubation of patients with burns involving the face or neck. If burns involve the entire face, consider intubation if the patient also demonstrates at least one of the following features: 1) a deep, circular neck burn; 2) symptoms indicating airway obstruction, such as a change in voice, stridor, and laryngeal dyspnea; 3) a total burned BSA of 40% or greater. [36]

Fiberoptic bronchoscopy is a simple, safe, and accurate method of diagnosing acute inhalation injury. It reveals the anatomic level and severity of large airway injury; identification of supraglottic and infraglottic involvement is helpful in predicting ultimate pulmonary complications.

Fiberoptic bronchoscopy also may aid in intubating patients with inhalation injury. Because fiberoptic bronchoscopy has been associated with the development of severe hypoxemia, especially in elderly burn patients, administer supplementary oxygen through the bronchoscope. The ventilatory system for fiberoptic bronchoscopy can deliver a warmed, humidified, and measured fraction of inspired oxygen either for spontaneous or mechanically assisted ventilation.

Further treatment

Once the airway is secured and the patient is oxygenated, further treatment includes maintenance of pulmonary toilet, relief of mechanical restriction of chest wall motion, and prevention of respiratory failure. When collagen is burned, it loses its elasticity, shortens its fibers, and becomes rigid. This can occur very quickly in fourth-degree and severe third-degree burns. When combined with accumulation of burn edema in interstitial spaces, respiratory insufficiency or ischemia of an extremity may result.

The compressive effect of a full-thickness burn of the neck may contribute to airway compromise. Without tracheostomy, tight neck eschar accentuates pharyngeal edema and draws the neck into flexion, compressing the pharyngeal airway. A vertical incision through the eschar extending from the sternal notch to the chin helps maintain a patent airway. If respiratory insufficiency is caused by a constricting eschar of the anterior thorax that limits respiratory excursion, escharotomy is imperative. Lateral incisions are made in the anterior axillary lines that extend 2 cm below the clavicle to the 9th or 10th rib. The top and bottom of the incisions are then joined to form a square across the anterior chest.

Mechanical ventilation

If respiratory failure ensues, mechanical ventilation is necessary. Airway resistance is often increased after inhalation injury resulting from edema, debris within the airway, or bronchospasm. The goal of mechanical ventilation should be to accept a slightly acidic environment (pH >7.32) to minimize the mean airway pressure required for ventilation. To keep airway pressures to a minimum, ventilator settings may need to be adjusted to slightly higher respiratory rates (16-20 breaths/min) and smaller tidal volumes (7-8 mL/kg).

Experimental evidence has demonstrated in baboons with moderate smoke inhalation that the barotrauma index (rate times pressure product) is significantly increased during regular ventilation compared with high-frequency flow interruption ventilation. Significantly greater histologic damage of pulmonary parenchyma also occurred in the group treated with conventional ventilation.

High-frequency flow interruption ventilation appears useful in its ability to recruit damaged, collapsed alveoli and keep them open in expiratory ventilation. Maintaining alveolar recruitment at low mean alveolar pressures helps minimize barotrauma and allows improved distribution of ventilation. Two retrospective studies demonstrate a decreased incidence of pneumonia and mortality in patients with inhalation injury when high-frequency percussive ventilation is used, compared with conventional "volume-limited" ventilation. Oscillating ventilator, which superimposes high-frequency ventilation onto conventional tidal volume breaths, may be an even better method of ventilation after smoke injury. This method reduces barotrauma and aids in the removal of airway casts by causing vibratory air movement. Airway cast and plug formation can be decreased by nebulized heparin treatments (5000 U in 10 mL of normal saline every 4 hours), which inhibit fibrin clot formation in the airway.

Circumferential full-thickness burns

The presence of circumferential full-thickness burns on any area of the body necessitates escharotomy. The burned skin will effectively act as a leash, compressing the underlying structures, an effect that will be exacerbated in the setting of edema resulting from fluid resuscitation. The resulting increase in pressure can cause compartment syndrome. Escharotomies can be performed with ease at the bedside using electrocautery or a scalpel. Incisions should be made on either side of limbs, effectively bivalving the burned skin. A crosshatch pattern across the chest is effective for releasing the eschar. It is important when performing escharotomies to incise only through the burn eschar to avoid damaging underlying structures.

Carbon monoxide

Carbon monoxide (CO) is present in smoke and has 280 times the affinity for hemoglobin as oxygen. Obtain a CO level in all patients with suspected inhalation injury. Patients should receive 100% oxygen until their carboxyhemoglobin (COHb) level is less than 10% because the elimination half-life for COHb depends on oxygen tension. In room air, the half-life of CO-bound hemoglobin is 4 hours. Under 100% oxygen, the half-life of CO-bound hemoglobin decreases to 45 minutes. Administration of 100% oxygen increases the gradient for oxygen binding to hemoglobin, and unbound CO is exhaled through the lungs.

Patients who have elevated COHb levels associated with a pH of less than 7.4 may be treated with hyperbaric oxygenation, although administration of hyperbaric oxygen is not routinely recommended for cases of suspected CO poisoning after smoke inhalation.

If CO poisoning is suspected or confirmed following smoke inhalation, the patient should undergo oxygen therapy without delay, via a high-flow mask, or be treated with 100% fraction of inspired oxygen (FiO_2) for 6-12 hours, if mechanically ventilated. [36]

Cyanide poisoning

Specific therapy for cyanide poisoning in patients with inhalation injury is another consideration. Cyanide causes tissue hypoxia by uncoupling oxidative phosphorylation via binding to mitochondrial cytochrome a-a3. Consider empiric treatment for cyanide toxicity for patients with unexplained severe metabolic acidosis associated with elevated central venous oxygen content, normal arterial oxygen content, and a low COHb level.

Consider avoidance of routine hydroxocobalamin administration after smoke inhalation. Use of hydroxocobalamin does not improve survival after smoke inhalation, and the agent is associated with nephrotoxicity. Restrict administration of hydroxocobalamin to adults suffering from smoke inhalation in whom there is a high suspicion of severe cyanide poisoning (as in patients with cardiac or respiratory arrest, shock, or coma) and to children with smoke inhalation and signs of moderate to severe cyanide poisoning. [36]

Fluid resuscitation

Percent TBSA

All patients with a major burn injury must be subjected to fluid resuscitation that is influenced by the percent TBSA as well as the presence of inhalation injury. Patients with burn wounds smaller than 20% TBSA can be treated with a combination of oral and IV fluid. For larger burns, the Parkland formula and its variations have become the standard method for resuscitating the burned patient.

Moderate burn victims should have at least one large-bore intravenous line placed through unburned skin, and severe burn victims should have at least 2 lines initiated. If necessary, venous catheters may be placed through burned skin or via venous cutdown using the saphenous vein at the groin or ankle. When a burn patient requires considerable fluid resuscitation or has evidence of cardiopulmonary disease, a central venous line is indicated. Patients with massive burns or respiratory injury and elderly patients with severe burns or cardiac disease should be monitored with a Swan-Ganz catheter to avoid fluid overload or inadequate replacement of volume.

Microvascular injury caused by a burn leads to increased vascular permeability with edema formation that results in ongoing plasma volume loss. Maximal edema formation occurs at 8-12 hours after burn injury for small burns, and 24-48 hours for large burns. The purpose of fluid resuscitation is to restore effective plasma volume, avoid microvascular ischemia, and maintain vital organ function. The amount of fluid required varies with the patient's age, body weight, and extent of burned TBSA.

Ideally, weigh the patient on a scale. In the absence of this measurement, obtain an estimate of the patient's weight from the patient, a relative, or the patient's driver's license. Carefully map the burned areas over the entire body, including the back, to estimate fluid requirements during the first 48 hours after injury. Typically, burns greater than 20% of TBSA require intravenous fluid resuscitation because the accompanying GI ileus precludes sufficient oral intake.

Different formulas for fluid resuscitation

Several different formulas for fluid resuscitation have been recommended, although all uniformly emphasize that adequate resuscitation is evidenced by a normal urinary output (1 mL/lb/h in children younger than 2 years, 0.5 mL/lb/h in older children, at least 30-40 mL/h in adults), a normal sensorium, and stable vital signs.

A survey of burn units in the United States and Canada demonstrated that 78% of the centers used the Parkland formula to estimate resuscitation volume and that lactated Ringer solution was the most popular type of fluid. [41] The Parkland formula for fluid resuscitation of burn patients is employed as follows: lactated Ringer solution (4 mL/kg/% TBSA burned) is administered intravenously in the first 24 hours, one half given in the first 8 hours, and the other half administered over the next 16 hours. Calculate fluid loss from the time of injury, and take into account the fluid administered by prehospital personnel for fluid replacement.

Administration of human albumin is recommended for severe burn patients with a burn TBSA of over 30%, after the first 6 hours of fluid resuscitation. Enough albumin should be administered to patients with severe burns to maintain a level of over 30 g/L (albumin dose: 1-2 g/kg/day); this prevents iatrogenic injuries caused by the administration of very high volumes of crystalloids. [36]

Strict adherence to a formula for fluid resuscitation does not guarantee successful fluid therapy. If the patient does not appear to be responding to resuscitation or signs of impending cardiac failure are present (eg, progressive central venous pressure elevation, pulmonary congestion, increasing edema with decreased urinary output), insertion of a Swan-Ganz catheter for measurement of pulmonary artery pressure and cardiac output is advisable.

During resuscitation, the most common error is overhydration, which increases the risk of acute respiratory distress syndrome developing 3-5 days postburn. In burn patients with concomitant large TBSA burns and inhalation injury, the Parkland formula may result in unnecessarily large fluid loads. To avoid overhydration, resuscitate patients with inhalation injuries with substantially less than formula predictions, with acceptance of a urinary output in the range of 0.3-0.5 mL/kg/h.

Protein losses

After a burn injury, significant intravascular protein is lost through endothelial leaks in the burned vessels. When endothelial integrity is restored 24 hours postinjury, some clinicians favor the administration of 5% albumin at 0.5 mL/kg/% TBSA to maintain dynamic forces between the extracellular spaces and the intravascular system. In addition, a low-dose dopamine infusion (3-5 mcg/kg/min) is beneficial in restoring renal and splanchnic blood flow in patients with major burn injury.

Hypertonic saline solutions

Resuscitation with hypertonic saline solutions reduces the required fluid volume. The volume of fluid administered using hypertonic fluid solutions is notably less, yet fluid requirements and percent weight gain have not decreased with hypertonic saline compared with Ringer solution. The anticipated benefits of fewer escharotomies and limited ileus have not been uniformly encountered. On the contrary, hypertonic saline resuscitation has been associated with an increased occurrence of acute tubular necrosis and hyperchloremic metabolic acidosis, which can exacerbate the metabolic acidosis of hypovolemic shock. Therefore, hypertonic saline is not currently recommended for resuscitation of burn patients.

When not to resuscitate

When patient survival is extremely unlikely after burn injury, the clinician must be encouraged not to begin fluid resuscitation. Elderly patients with large burns (>80% TBSA) will not survive. This decision must be made after thoughtful communication with family members. When resuscitation is not undertaken, make patients pain-free, keep them warm, and allow them to remain in a room with family members.

Fluid resuscitation

Most of the burn care in children is similar to that in adults, yet some relevant physiologic differences need to be considered in the care of burned children. The Parkland formula is effective in estimating fluid loss in adults, yet it underestimates the evaporative fluid loss and maintenance needs in children. Compared to adults, children have a larger TBSA relative to weight than do adults and generally have somewhat greater fluid needs during resuscitation. Use the Galveston formula for fluid resuscitation in children as follows: 5% dextrose in lactated Ringer (5000 mL/m^2 of TBSA burned plus 2000 mL/^{2}) is administered intravenously in the first 24 hours. One half is given in the first 8 hours, and the other half is given over the next 16 hours. Add dextrose to the resuscitation fluid in children to prevent hypoglycemia, because children have smaller glycogen stores than adults.

In infants younger than 6 months, temperature is regulated by nonshivering thermogenesis, a metabolic process by which stores of brown fat are catabolized under the influence of norepinephrine, which requires large amounts of oxygen. Consequently, prolonged hypothermia in burned infants may result in excessive lactate production and acidosis. After 6 months, infants and children are able to shiver. Because they have a greater evaporative water loss relative to weight than do adults, infants and children are especially prone to hypothermia; therefore, keep the ambient temperature high to decrease radiant and evaporative heat loss from burned infants and children to the environment.

Renal function differences

Differences in renal function between infants and adults may have important therapeutic implications in treating burned children. The glomerular filtration rate in infants does not reach adult levels until 9-12 months because of an imbalance in maturation of tubular and glomerular functions. During this interval, infants have approximately half the osmolar concentrating capacity of adults, and a water load is handled inefficiently. The rate of water excretion is time dependent and decreases as water loading continues. During the first several weeks of life, infants are likely to retain a larger portion of a water load administered as part of burn resuscitation. The hyposmolarity of lactated Ringer solution, when used in accordance with the Parkland formula, already accounts for the free water needs of infants during the first 24 hours postburn. Additional water often results in fluid overloading.

Catheterize patient

Place a Foley catheter into the bladder to monitor the effectiveness of intravenous fluid replacement. Burns of the perineum also are best cared for with an indwelling Foley catheter to decrease urinary soilage of the wound. In patients with major burn injuries who require intravenous fluid resuscitation, pass a nasogastric (NG) tube for initial evacuation of fluid and air from the stomach and feeding access. Removal of the gastric contents prevents vomiting and aspiration, sequelae of the ileus that commonly occur soon after burn injuries involving more than 20% TBSA. Early feeding through the NG tube within 6-8 hours of the burn injury diminishes the hypermetabolic response and maintains intestinal integrity.

Patient transport

After stabilization of the burned patient in the ED, transfer patients with severe burn injury to burn centers. As mentioned previously, the American Burn Association has established criteria for optimal treatment of burn patients, including both indications for admission to a hospital and criteria for transfer to a burn center.

Ground, helicopter, or fixed-wing aircraft may transport burn patients. In addition to the condition of the patient, the mode of transportation (ambulance vs helicopter) depends on such factors as distance, terrain, and prevailing weather. The safety and costs of using helicopters in the transport of burn patients have been questioned. Helicopters transport patients more rapidly than ambulances, and because a nurse or physician usually staffs them, provide a higher level of medical expertise during transport. For burn patients, a helicopter offers little advantage over a ground ambulance if the distance is less than 30 miles. Helicopter transfers may be efficacious if the distance is 30-150 miles or if the transfer time is greater than 30 minutes. Over 150 miles, fixed-winged aircraft are best to transport patients.

When a burn patient is being transferred from an ED to a burn center, early physician-to-physician contact with the burn center is essential. A standard check sheet facilitates assessment of the patient's physiologic status by both the referring and receiving physician. Evaluate airway patency, intravenous access, and other injuries prior to transfer. Cover burn wounds with dry dressings but postpone use of antimicrobial creams until admission to the burn center.

Consider the burn wound a dirty wound, and institute tetanus prophylaxis accordingly. Tetanus vaccine without thimerosal should be administered to each patient. [42] A vaccine information statement, which outlines the complications of the vaccine, should be given to either the patient or legal guardian. [43] The Vaccine Injury Compensation Program should also be discussed. [43] Prophylactic antibiotics are not indicated. Treatment of the patient in the burn center involves 3 important considerations: supportive care, burn wound management, and nutritional support.

Advances in telemedicine have helped to limit inappropriate transfers, which carry an increased mortality risk. Telemedicine can also be used, in places where no specialist is available, to improve TBSA measurement and characterize burn severity. [36]

Supportive Care

Upon arrival at the burn center, each patient should receive a standard regimen of supportive care that involves pain management, as well as prevention of gastric erosions and renal failure.

The requirement of pain medication is inversely proportional to the depth of burn injury. Full-thickness burns, which appear white, brown, or leathery with clotted vessels, are painless because their intrinsic sensory nerves are damaged. In contrast, partial-thickness burns, in which the skin is red with blisters, have intact nerves and are extremely painful.

For more than 160 years, morphine has been advocated for the management of pain in burn patients. Its analgesic effect can be easily titrated with incremental intravenous doses. Morphine has 2 pharmacologic advantages for use in burn patients: a low amount of protein binding (30%) and a major active metabolite that is conjugated in the liver and removed by glomerular filtration. This rapid elimination may

require that doses as high as 50 mg/h be used in severely burned adults. Any respiratory depression caused by morphine can be rapidly reversed by small doses of naloxone.

Dressing changes are exceptionally painful for burn patients and require additional analgesia. Increased analgesia with short-acting agents such as midazolam and/or fentanyl are particularly useful during these short periods of greater pain.

Acute upper GI erosions and ulcers may occur in patients with severe burn injuries. These lesions are often termed stress ulcers or erosions (Curling ulcer). The most common clinical finding in such patients is painless GI hemorrhage. Blood loss is usually minimal but occasionally may be substantial. Treatment of acute stress ulceration is principally preventive. In high-risk patients, antacids can reduce the occurrence of stress ulcerations by neutralizing gastric contents, and H2-receptor antagonists can inhibit gastric acid secretion. This prophylaxis against acute stress ulceration usually is initiated immediately after admission to the burn center.

Renal failure can occur after burn injury, as manifested by an elevated serum creatinine and a fall in creatinine clearance. Prevention of this complication involves adequate resuscitation, treatment of infection in the wound and other sites, and avoidance of nephrotoxic drugs (eg, aminoglycoside antibiotics, vancomycin, loop diuretics). When renal function deteriorates with resultant fluid and electrolyte imbalance, dialysis may be indicated. Peritoneal dialysis or continuous venovenous hemodialysis are the preferred methods because of the need for frequent monitoring and adjustment of electrolyte levels.

Although sepsis is a risk for burn patients, antibiotic prophylaxis should be avoided. *Staphylococcus* infections are the most common source of infection, typically presenting about 1 week after the injury. Following this window, patients are at greater risk for *Pseudomonas* infection. It is important to recognize pneumonia and other sources of infection in the burn patient. If infection is suspected, antibiotic therapy should be culture directed rather than prophylactic.

Venous thromboembolic prophylaxis should be administered to all burn patients, since these individuals are in a hypercoagulable state and immobile and therefore at increased risk for venous thromboembolism.

Burn Wound Management

The burn team accomplishes all wound management using powder-free gloves because of the demonstrated toxicity of glove powders to tissue. Initial treatment of the burn wound involves cleansing the wound with saline or various commercially available surfactants. Ruptured blisters are removed with scissors. After wound cleansing, cover with a topical antimicrobial dressing. Topical antibiotics decrease microbial growth and reduce invasive infection. Prophylactic systemic antibiotics are not recommended because they do not prevent wound sepsis. Systemic antibiotics may be indicated when cellulitis is evident in surrounding unburned tissue.

Burn pain management

Pain control is crucial to the recovery of burn patients. Suboptimal control of pain delays the healing process, as fear and anxiety produce an elevation in stress hormones (such as glucocorticoids). Consequently, with lack of pain control, physical and psychological burdens last longer, as does the hospital stay. [44]

Three main mechanisms are known for burn pain: nociceptive, neuropathic, and inflammatory. Intravenous morphine is the fundament of pain management; shorter half-life opioids and nerve blocks remain alternative options. During the healing process, pain levels are substantially increased by wound debridement, physiotherapy, and dressing changes. Scar maturation and continued physical therapy decrease nociceptive pain levels. Agents such as pregabalin and gabapentin are widely used for neuropathic pain. [44]

Dressings

Wound dressings have been proved to help reepithelialization and to prevent wound contamination and infection, skin desiccation, and further skin damage. Dressings are categorized into four groups: biologic, conventional, synthetic, and antimicrobial.

Biologic dressings

Biologic dressings include cadaver allograft skin and xenografts, both of which are used as a temporizing measure. The drawback of biologic dressings is their inability to function as a permanent skin replacement, due to their immunologic disparities, inconsistent quality, and limited supply, as well as the risk of pathogen transfer. [44]

Conventional dressings

Conventional dressings, such as Vaseline gauze or silicone sheets, are also widely used. However, the frequent changes they require lead to traumatization of the newly epithelialized wound surface and potential healing delays.

Synthetic dressings

An enormous variety of synthetic dressings have been developed, although there are only a few synthetic skin substitutes on the market. Synthetic materials have greater integrity and resistance than natural products but have shown poor bioactivity compared with biologic materials. [44]

First-generation film dressings (eg, thin films, hydrocolloidal hydrogel foams) are based on the concept that epidermal regeneration occurs best in a moist environment. Second-generation microenvironmental wound dressings combine the fluid-retaining properties of film dressings with the absorptive properties of the hydrocolloid. In theory, a central membrane absorbs wound fluid through the porous inner layer. An external layer allows moisture vapor to escape, yet is impermeable to exterior fluids and bacteria.

Second-generation dressings have many desirable features but are relatively expensive. A simple, less costly alternative to these synthetic dressings has been proposed that can be reliably used in minor burns. After cleansing and débridement, strips of sterile fine meshed gauze (type 1) soaked in 0.9% sodium chloride are placed over the entire wound. This layer of gauze is then covered by multiple layers of fluffed 4 X 4 inch coarse mesh gauze (type 6) secured by an inelastic roller gauze dressing. The gauze dressing is attached to unburned skin using microporous tape.

Antimicrobial dressings

Antimicrobial dressings are useful for preventing wound infection, reducing colonization. They contain either silver (eg, Aquacel AG), nano-crystalline silver (eg, Acticoat), cadexomer iodine (eg, Iodosorb), or honey as an antimicrobial agent. The introduction of silver compounds to burn management was a revolutionary step in topical burn therapy, drastically reducing burn wound–induced sepsis and mortality. [44]

Dressing application and removal

Apply daily dressings to the wound continuously until complete healing occurs or surgical intervention is required for wound closure. In the past, many burn surgeons dressed wounds twice daily. This practice has changed to daily dressing changes, resulting in a significant decrease in costs, nursing time, and pain. This daily dressing change is especially suited for children with superficial scald burns.

Twice-daily dressing changes are still indicated in patients with wounds that are or have been infected or those with excessive amounts of exudate. During these dressing changes, cleanse the wound gently to completely remove the topical antibiotics. Loosely adherent eschar usually can be removed with a sponge, but forceps may be needed to facilitate bedside débridement. After wound cleansing, the wound may be covered with antibiotic cream or a fresh dressing. At the author's institution, it is commonplace to dress stable, clean burn wounds with silver-impregnated gauze and to change dressings every 2 days to minimize discomfort to the patient.

When possible, elevate the site of injury above the patient's heart. Elevation of the injured site limits accumulation of fluid in the interstitial space of the wound. A healing burned extremity with little edema resumes normal function more rapidly than does the markedly edematous extremity. Early mobilization of the injured area within 24 hours after injury limits the development of joint stiffness, a particularly challenging problem in both older persons and heavy laborers.

If the patient returns to the ED for a follow-up visit, use an aseptic technique to gently remove the outer layers of the dressing to visualize the bottom (fine mesh gauze) layer. If the fine mesh gauze adheres to a relatively dry and pink burn wound, it should be covered again by layers of 4 X 4 inch coarse mesh gauze secured by roller gauze dressing. Instruct patient to return in 5-7 days for reevaluation. Because most superficial partial-thickness burns heal in 10-14 days, spontaneous separation of the gauze from the healing burn wound should be evident at the time of the next dressing change.

If the burn wound exhibits a purulent discharge, remove the fine mesh gauze and cleanse the burn wound with saline or poloxamer 188. One option is to apply silver sulfadiazine cream twice daily to the burn wound and dress the area with sterile roller gauze dressing. Instruct the patient to gently wash the burn wound in clean water to remove this cream before reapplying additional cream. If the topical cream is not removed completely at each dressing change, multiple layers of the cream accumulate on the burned skin and predispose the wound to infection. Alternatively, in the absence of surrounding cellulitis, the wounds can be treated with daily wet-to-dry dressings. Wounds with surrounding mild cellulitis should be treated similarly, with the addition of oral antibiotics.

Occlusive dressings

Occlusive dressings are an alternate method of managing burn wounds rather than antimicrobial creams. An occlusive dressing can be applied to a superficial partial-thickness burn that is clean and less than 24 hours postinjury. These products maintain a moist wound environment that enhances healing and eliminates the need for dressing changes. Currently, several different types of commercial occlusive dressings are available.

Biobrane is another biosynthetic wound dressing consisting of a silicon film with a nylon fabric embedded in the film. The fabric acts as a complex 3-dimensional structure of trifilament threads to which collagen has been chemically bound. When blood or sera clot in the nylon matrix, they firmly adhere to the dressing until wound epithelialization occurs. As the epithelium heals, the dressing separates from the wound. Products such as Biobrane are recommended for small, superficial burn wounds.

Indications for surgery are full-thickness burns or partial-thickness burns that are unlikely to heal within 3 weeks. If the burn fails to heal in 3 weeks, the risk for hypertrophic scar and contracture formation increases and the healed wound exhibits an aesthetically displeasing scar. In regions with a dense cross section of dermal appendages (such as the face, scalp, and ears), observe the burn wound for at least 3 weeks to clearly identify its healing potential. When circumferential burns of the extremity exist, emergency escharotomy can salvage an ischemic limb.

Negative pressure wound therapy

Negative pressure wound therapy (NPWT) is widely used by surgical teams in the management of acute complex burns and their chronic sequelae. Split- and full-thickness skin grafts take more easily in association with NPWT, permitting early patient mobilization. It also optimizes the environment, reduces wound infection, and eventually promotes wound healing. NPWT with instillation or continuous irrigation could play a major role in future burn wound management by reducing the number of treatment days, accelerating the clearance of infection and wound closure, and promoting the efficacy of antimicrobial solutions. [44]

Escharotomy

Full-thickness circumferential burn of an extremity can result in vascular compromise. Loss of Doppler ultrasound signals in the radial and ulnar arteries and digital vessels are indications for escharotomies of the upper extremity. Loss of dorsalis pedis or posterior tibial artery signals indicates the need for escharotomy of the lower extremity. Ordinarily, interstitial tissue pressure is slightly negative and the normal arterial capillary perfusion pressure is approximately 5-7 mm Hg.

After burn injury, a rise in interstitial tissue pressure first occludes venous outflow, then arterial capillary inflow. A period of 3-8 hours is required for edema to develop sufficiently to increase tissue pressure. When tissue compartment pressures are greater than 40 mm Hg, escharotomies of the full-thickness burn prevent this ischemic injury. Note that the most common cause of absent pulses in an extremity is hypovolemia with peripheral vasoconstriction, not increased interstitial pressure.

Escharotomies are performed on the medial and lateral aspects of the extremity and extend the length of the constricting eschar. Incisions are made using either a scalpel or high-frequency electric current, with release of the edematous tissues ensuring adequate depth. After prolonged vascular compromise, an escharotomy may cause reperfusion injury to the extremity with reactive hyperemia and edema of the compartment muscles. In this case, a fasciotomy is required to restore perfusion to the extremity.

For full-thickness circumferential burns of the upper extremity, first decompress the fingers by a digital escharotomy that is performed along each side of the burned finger, cutting down to fat. Decompress the palm by an incision along the palmar crease. At the wrist, continue the incision ulnarward to avoid injury to the palmar cutaneous branch of the median nerve. When intrinsic muscle involvement is suspected, decompress the interossei through short longitudinal skin incisions made in the intermetacarpal spaces carried down to the dorsal interossei. Decompress the leg by midmedial and midlateral incisions. Decompress each toe in a manner similar to that used for the fingers.

Surgical débridement and grafting

The standard of care for full-thickness burns is burn wound excision and grafting. The mortality of patients with massive burns is reduced by early tangential excision of the entire wound, followed by skin closure with an autograft from unburned areas on the patient or an allograft from a donor cadaver. In burns less than 30% TBSA, wound closure is accomplished in one operation using split-thickness skin grafts from unburned areas on the patient, taken in sheets or meshed either 1:1 or 2:1. In burns greater than 40% TBSA, split-thickness skin grafts are meshed 3:1 or 4:1, complemented by cadaver allograft used to temporarily cover the residual open wound areas. Cadaver allograft skin adheres to the wound and serves as a partial barrier to infection; however, it carries donor antigens and can transmit infectious disease.

Allograft, xenograft, or artificial coverings, such as Integra or Dermagraft-TC, are routinely used for burns involving over 40% TBSA. These burn dressings accomplish 4 functions: (1) protect the damaged epithelium, (2) splint the area into the desired position to maximize long-term function, (3) occlude the wound and prevent evaporative heat loss, and (4) provide comfort. Donor sites for autograft require 1-2 weeks to heal. At that time, temporary burn dressings are removed and residual open wound areas are closed with split-thickness skin grafts from these same donor sites. For larger than 90% TBSA burns, up to 10 cycles of autografting may be required to completely close the wounds.

Glycerol-preserved allografts (GPA) are widely used in Europe for the management of burn injury. Until recently, little has been known of the clinical usage and effectiveness of GPA. To gain more insight into the use of GPA, the Euro Skin Bank (ESB), which is the main provider of GPA, sent a questionnaire to 62 burn centers that had received GPA from the ESB in the last 5 years. Ninety percent of the correspondence used GPA regularly, although 24% of the centers preferred the use of cryopreserved allografts and 16% preferred fresh allografts. The uses for GPA included the following: as a temporary cover on freshly excised wounds, as an overlay on widely expanded autografts, as an improvement of the quality of the wound bed prior to autografting, and as a biologic dressing for partial thickness injuries.

One of the challenges of surgical débridement is operative blood loss. Several methods reduce operative blood loss. In some sectors, a tumescent fluid solution is used. The burn site is infiltrated with a 1:1,000,000 dilution of injectable epinephrine solution. Débridement is delayed for 10 minutes to allow the vasoconstrictive effect of epinephrine to occur. Use tourniquets during excision of all extremity burns. When comparing hemostatic effect of dilute solutions of phenylephrine to that of thrombin, both drugs decreased blood loss; however, phenylephrine proved to be the more effective hemostatic agent. Topical treatment of the excised burn wound with this vasoactive agent was not associated with systemic pressure effects or cardiac irregularities.

Cultured epidermal autografts

Cultured epidermal autografts, although controversial, are another approach for skin coverage of the excised burn wound. Most agree that the healed grafts are extremely fragile and susceptible to infection, antimicrobial agents, shear, and dressing changes. Limited data indicate that they do not enhance patient survival or reduce costs. Most burn surgeons believe in reserving cultured epidermal autografts for treatment of massive burn injuries.

Meek skin grafts

Meek skin grafts offer increased expansion of the donor site, covering up to nine times the original surface area. The Meek technique involves taking skin grafts, processing the grafts into small squares, applying them to an expandable foil, and then stapling the foil onto the burn wound. Meek grafts will heal in about 1 week with 1:4 expansion, 2-3 weeks with 1:6 expansion, and 1 month with 1:9 expansion. The Meek technique offers a more economical alternative to cultured epidermal autografts, with promising results achieved to date. [45]

Immobilize skin-grafted areas

Skin grafted areas must be immobilized postoperatively to prevent shear forces from disrupting the grafts. Splinting is a superb method of immobilization. Design splints to place joints at maximum stretch. Specific recommendations for positions of the grafted sites include the following: axilla, 90° horizontal with bedside troughs; wrists, slightly extended by 10º; metatarsophalangeal joints of the fingers, flexed; interphalangeal joints, fully extended; and thumb in 40-50° of abduction with interphalangeal joint extended.

Once the burn wound has healed, hypertrophic scar formation commonly occurs. Pressure garments designed to exert 25 mm of pressure are used to decrease the intensity of scar formation. Silicone sheets in direct contact with scars can further improve their appearance and decrease redness and itching.

Minor burns

First, cleanse all minor burns with sterile saline or poloxamer 188. The treatment of burn blisters remains controversial. Exposing an unbroken blister can lead to local wound infection but studies have demonstrated that burn blister fluid may be deleterious to wound healing, and undrained fluid confined by necrotic skin can act as a source for closed space infection. Most surgeons recommend leaving blisters on the palms or soles intact. Other blisters, particularly when large enough to preclude the application of an adequate dressing, should be aspirated sterilely. Alternatively, open blister with a No. 15 knife blade and remove the surface of the blister.

Provide tetanus prophylaxis if indicated. Prophylactic antibiotics are not recommended. Topical antimicrobial agents have little value in outpatient management of minor burns. Because these agents lose their antibacterial activity within 6-24 hours after application, frequent dressing changes are necessary. Unfortunately, removal and reapplication of the cream is both painful and time-consuming. As a result, many patients resort to reapplication of the cream without initial removal, an invitation to infection.

Treat burn wounds either by the open or closed technique. Open therapy of minor burn injuries usually is reserved for burns of the face. These burns are covered by bacitracin ointment, which is reapplied every 6 hours after gently washing the skin.

Burn wound infection

Challenges

Wound infection is the chief cause of mortality in burn-care patients. With the evolution of pathogens in tandem with antibiotic use, multi-drug resistant (MDR) bacteria and fungi have become major challenges in burn therapy. Most bacterial burn wound infections are gram negative, while *Staphylococcus aureus* is the main gram-positive bacterium in infected burn wounds. Infections with organisms such as *Pseudomonas aeruginosa*, *Klebsiella pneumonia*, *Escherichia coli*, and *Staphylococcus aureus* independently predict mortality. Methicillin-resistant *S aureus* (MRSA) has become the biggest pathogenic threat in some burn centers. [44]

Topical antimicrobials

Most partial-thickness burns of less than 10% TBSA respond satisfactorily to daily antibiotic dressings. Silver sulfadiazine (Silvadene, Flamazine) remains the most popular antimicrobial cream. This agent has broad-spectrum antibacterial activity and is associated with relatively few complications in these small burn wounds. The current formulation of silver sulfadiazine contains a lipid soluble carrier, polypropylene glycol, which has certain disadvantages, including pseudoeschar formation. When this antibacterial agent is formulated with poloxamer 188 the silver sulfadiazine can be washed easily from the wound because of its water solubility, making dressing changes considerably more comfortable. If the patient is allergic to sulfa, alternative medications include Polysporin, bacitracin, and Bactroban. While these agents are relatively inexpensive, they may induce allergies.

Keeping the above in mind, however, a meta-analysis reported silver sulfadiazine to have poorer healing outcomes than alternate dressings and to lack evidence of effectiveness in preventing wound infections. [46] This is believed to be due to a high requirement for regular dressing changes, poor eschar penetration, and, in a more recent issue, reported silver resistance in clinical organisms.

Acticoat has the beneficial antimicrobial properties of the silver ion by coating the dressing material with a thin, soluble silver film. This dressing appears to maintain antibacterial levels of silver ions in the wound for up to 5 days. Because Acticoat remains on the burn wound for up to 5 days, the patient is spared the pain associated with dressing changes as well as the expense. Additional studies are now underway to determine the ultimate benefit of this product.

The carrier in the burn cream can have a considerable impact on patient outcome. Because nitrofurazone burn creams contain polyethylene glycol, they provide an avenue for absorption of polyethylene glycol in patients with large TBSA burns. Absorption of polyethylene glycol results in increases in the anion gaps and serum osmolalities that result in patient death. It produces a syndrome similar to the more common poisoning with ethylene glycol but also includes an increased serum calcium with a concomitant decrease in ionized calcium. The cause of this high calcium gap appears to be the binding of calcium by dicarboxylic acid metabolites of polyethylene glycol. Consequently, avoid burn creams containing polyethylene glycol in patients with large TBSA burns.

Systemic antibiotics

Drug-resistant organisms (eg, MRSA) are normally treated with vancomycin. In light of a newly emerging strain, vancomycin-intermediate *S aureus* (VISA), new antimicrobials, including oxazolidinones, streptogramins, tigecycline, daptomycin, and dalbavancin, are important additions to the tools against such infections. [44]

Owing to a substantially altered physiology in critically ill patients after severe burn injuries, the main focus of systemic antibiotic treatment is now on new drug delivery innovations. This is because burn-related physiologic changes lower antibiotic tissue concentrations and lead to more resistance. In optimizing drug delivery, antibiotic concentrations can be regularly monitored, and antibiotics can be delivered by way of continuous infusion rather than bolus regimes. [44]

Fungal infection

Fungal wound invasion still represents a major cause of infection in burn centers, with the most common fungi in such infections being *Candida* species. About two thirds of invasive burn wound infections arise from fungi, with organisms such as *Aspergillus* being associated with high mortality. [30]

Burned tissue débridement and wound closure are the most effective preventive measures. Fungal burn wound infections can be treated with three available classes of systemic antifungal drugs: polyenes, azoles, and echinocandins. [30]

Fungal wound infection accompanies larger burns and is associated with mortality in burn patients, particularly in patients with total burn surface areas of between 30% and 60%. This association is independent of burn size, inhalation injury, and the patient's age. The use of newer diagnostic strategies, such as serology and molecular techniques, and newer, less toxic antifungal therapies means that accurate prediction models of fungal burn wounds are of paramount importance. [47]

However, despite the wide acceptance of topical antimicrobial agents, early excision, and patient isolation practices, there has been no change in the incidence of fungal wound infections. The difficulty met in preventing or eradicating fungal infections is not surprising considering the immunosuppression encountered in the most severely burned patients. [47]

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Nutritional Support

The degree of metabolic alteration experienced by burn patients is directly related to the extent of injury. "Ebb phase" is the initial decrease in cardiac output and metabolic rate. Following fluid resuscitation, cardiac output normalizes and then increases to above normal levels with a simultaneous increase in resting energy expenditure (flow phase). A severe burn can double the metabolic rate, which can be blunted by 40-60% using occlusive dressings and increased room temperature. Central temperature is reset to 38.5°C 5-15 days after the burn injury. In burns of more than 60% TBSA, the central temperature can remain elevated for up to 2 months from direct stimulation of the hypothalamus by inflammatory mediators and cytokines.

Burn injury causes the release of massive amounts of amino acids from muscle. This response is caused by increases in cortisol and decreases in growth hormone and insulin, with resultant increased proteolysis of muscle protein and release of amino acids. Anabolic growth hormone treatment is shown to increase protein synthesis in muscle, increase muscle mass, and accelerate wound healing after burn injury. Potential anabolic hormones (eg, insulinlike growth factor, insulin, dehydroepiandrosterone, oxandrolone) are being evaluated for their effects on wound healing.

Because basal energy expenditure is increased 3-fold above normal, early and aggressive nutritional support via the enteral route is important in preventing bacterial translocation from the gut and systemic sepsis. Nutritional support is initiated within 18 hours of admission using a Dobbhoff feeding tube. Although gastric feeds are safe in many patients, positioning the tip more distally prevents aspiration of food during anesthesia and allows the patient to be fed continuously. Passage of the tip of the tube beyond the pylorus can be facilitated by the administration of metoclopramide or erythromycin.

The patient's caloric requirements can be estimated using the Curreri formula (25 kcal/kg+40 kcal/% TBSA) or twice the Harris-Benedict estimate. Realizing that these estimates can be very inaccurate, many practitioners measure the resting energy expenditure by indirect calorimetry and then give 20% more calories than this number indicates. It is recommended that the resting energy expenditure be measured on admission and weekly thereafter. Clinical measurements of protein requirements for these patients indicate that they require a nonprotein kilocalorie-to-nitrogen ratio of 100:1 and at least 2 g of protein/kg/d. Measuring the prealbumin level is another approach to documenting the effectiveness of nutritional support.

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Scarring

Severe burn survivors will suffer from burn scars. Hypertrophic and keloid scarring are major causes of morbidity, with functional and cosmetic outcomes often being poor. Burn scars can lead to pain, pruritus, dyspigmentation, heat intolerance, and scar contraction (resulting in limited range of motion). Commonly used options for burn scar management include treatment with silicone, corticosteroids, botulinum toxin A, fat grafting, pulsed dye lasers, and neodymium-doped yttrium aluminum garnet (Nd:YAG) lasers. [44]

Postburn Dyspigmentation in Asian Skin

Dyspigmentation, in the form of either hyperpigmentation or hypopigmentation, is frequently a serious psychological problem for the self-image of patients. [48] This pigmentation is a challenging problem after a burn injury. The most common alteration in skin color is due to the result of changes in epidermal melanin of the underlying skin. In patients with postburn dyspigmentation disorders, the clinician must distinguish between the 2 changes (hypopigmentation and hyperpigmentation). The depigmented skin after burn injury has been reported to contain little melanin pigment in the basal cells and marked thickening in all skin layers. Hyperpigmentation has been described as resulting from injury to the melanin cells.

The surgical goals for effective treatment of patients with postburn dyspigmentation disorders are to remove scar tissue, to establish even coloration from abnormal vascular structures and injured melanin cells, and to produce healthy melanin cells. Although postburn dyspigmentation disorders of the skin are more common in non-Caucasian populations, and 80% of the world's population consists of individuals with pigmented skin, only limited studies are available on the surgical application of epidermal grafting procedures directly to treat postburn dyspigmentation in non-Caucasian patients.

In a study of 23 cases in 2007, Burm et al used superficial dermabrasion and simultaneous autologous epidermal grafting with suction blisters for the treatment of postburn dyspigmentation. [49] All epidermal grafts took completely within 4-5 days after surgery; no case exhibited hematoma or delayed wound healing. Skin color and surface irregularity were remarkably improved in all cases and blended in well with the surrounding skin. Postoperative complications, such as peripheral hypopigmentation, delayed hyperpigmentation, milia, inclusion cysts, achromic fissures, prolonged erythema, and scar deformity were not observed in any of the cases during the 2-year follow-up period. The study authors' method of combined modalities had a synergistic effect on the treatment of postburn dyspigmentation.

While many alternate procedures exist for the treatment of dyspigmentation of burned skin, one of the most important advantages of the method described in this article is the absence of scarring at the donor site. Other advantages include no need for general anesthesia, no visible border line of grafted epidermis, and few residual complications.

Guidelines

Guidelines released in 2020, from a panel of experts brought together by organizations that included the French Society of Anesthesia, Critical Care and Perioperative Medicine (SFAR), provided recommendations for the management of severe acute-phase thermal burns in adults and children. These included, but were not limited to, the following suggestions [36] :

- In hemodynamic management, use balanced crystalloid solutions
- In hemodynamic management, the formula used in estimating the initial crystalloid infusion rate should at least include the body weight and total burned body surface area (BSA)
- Basing changes on clinical and hemodynamic parameters, adjust the infusion rate as soon as possible in fluid resuscitation for severe burns
- Patients with severe burns in whom the total burned BSA is over 30% should receive human albumin after the first 6 hours of management
- Do not routinely intubate patients with burns involving the face or neck
- If burns involve the entire face, consider intubation if the patient also demonstrates at least one of the following features: 1) a deep, circular neck burn; 2) symptoms of airway obstruction (ie, change in voice, stridor, laryngeal dyspnea); 3) a total burned BSA of 40% or greater
- Do not routinely administer hydroxocobalamin after smoke inhalation
- Restrict hydroxocobalamin administration to adults suffering from smoke inhalation in whom there is a high suspicion of severe cyanide poisoning and to children with smoke inhalation and signs of moderate to severe cyanide poisoning
- In the absence of shock, burn cooling should be performed in adults with a total burned BSA of less than 20% and in children with a total burned BSA of less than 10%
- Routinely prescribe thromboprophylaxis for severe burn patients in the initial phase

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Federation of Burn Foundations

The mission of the Federation of Burn Foundations is to empower burn-related organizations to better serve their communities. Their specific purposes include the following: (1) communication and information exchange among member organizations, (2) promotion of public policy beneficial to burn prevention, care, research, and/or burn survivors, (3) liaison with other organizations with similar or comparable purposes, and (4) enhancing the capabilities of members in assisting in the development of burn foundations. The Federation of Burn Foundations provides a newsletter that should be read by all physicians involved in burn care treatment and prevention.

Burn Injuries: Complications and Care Options
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