



Burns are a leading cause of preventable hospitalization and mortality in children both in the US and worldwide, with more than 6000 children hospitalized every year for burn-related injuries. Great strides in both the prevention and treatment of burns have been made since the turn of the twentieth century. Early excision and grafting, prompt fluid resuscitation, and, when appropriate, transfer to specialized burn centers have improved mortality rates in even the most extensive burns. Still, according to the CDC, burns are among the top 4 causes of unintentional injury-related deaths in children, with some 7800 lives lost since the year 2000.

It is essential to consider the mechanisms and situations where children are burned and how these differ from adults. Whereas adults typically present following an industrial or occupational accident, children spend most of their time in and around the home. Scald injuries are the most common burn mechanism in young children. It is easy to imagine a child being submerged in water too hot for their sensitive skin. The kitchen is a particularly high-risk area where curious children may reach for hot cookware on the counter above them or bump into a parent's hot beverage. When a child turns 16, flame burns become the predominant burn mechanism, as they are in adults. The flame burns are more likely to cause deeper burns, lead to concomitant inhalational injuries, and require inpatient management than other burn

mechanisms. Male children of all ages experience burns at a higher rate than females.

Children of low socioeconomic status are at higher risk for burns. Factors such as rented living accommodation, depressed income, low parental education status, and being a member of an ethnic minority are predictors of sustaining a burn injury. Children may be intentionally burned either as a form of abuse or inflicting self-injury. Rates of non-accidental burns in children have been reported to be as high as 10%. These intentional burns carry a significantly higher mortality rate than accidental burns. It is crucial to recognize patterns of non-accidental burns in children as abuse recurs in up to half of cases.

The anatomic and physiologic differences of children require special consideration when managing a burn victim. Children under the age of 2 have thinner skin, placing them at higher risk for more severe burns given the same thermal insult. When insulating skin and subcutaneous tissue are lost, children are vulnerable to hypothermia. A larger surface area-to-body mass ratio in children contributes to their higher risk of hypothermia and increased insensible fluid losses following a burn. Immature kidneys less adept at concentrating urine necessitate comparatively larger fluid volumes for adequate resuscitation. Alterations in physiology mediated by a hypermetabolic state persist for years following a severe burn in children. Just as prompt fluid resuscitation and early surgical intervention have improved mortality, early management of the hypermetabolic state is essential to minimizing long-term disability.

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Initial Evaluation

In the prehospital setting, first responders' immediate goal is to halt the burn process while taking the utmost caution to prevent themselves from becoming victims. Prompt removal of burned clothing, even following scald burns, appears to reduce further morbidity. Likewise, the anticipation of edema should prompt the removal of items that could later form a

tourniquet, such as rings or other jewelry. Contact with the victims' possessions may be hazardous due to retained heat, the presence of caustic chemical agents, or bodily fluids. Caustic chemicals should be brushed away if still present on the skin, which is then irrigated copiously with water. Attempts to chemically neutralize the caustic agent can generate heat and should be avoided.

It is common for burn victims to attempt to cool the burned area. While there is debate over the ideal method and duration of cooling, for minor burns under 5% TBSA, cooling the area with tap water is acceptable as long as it does not delay care or transfer to a hospital. Attempts to cool extensive burns in children place them at an even greater risk for hypothermia and must be avoided. The use of ice or cold packs is not recommended and risks further tissue damage. Once safely removed from the scene, the victim can be covered in a clean sheet or blanket to help diminish heat loss while transferred to an appropriate facility.

Burn patients are trauma patients. Assessing the victim's airway is the most urgent priority. Burns can lead to airway compromise by a variety of mechanisms and victims may rapidly deteriorate. Patients with suspected inhalational injury should receive humidified 100% oxygen initially and preparations for intubation made before a patient begins to show signs of respiratory distress. Suspected cervical injuries should be immobilized. Pulse oximetry will display falsely optimistic readings in the setting of carbon monoxide poisoning due to its inability to differentiate carboxyhemoglobin and oxyhemoglobin. The pediatric airway is more prone to obstruction given the smaller cross-sectional area of the trachea. Thus, children are more susceptible to upper airway edema due to thermal injury or fluid shifts during resuscitation. The child with no signs of respiratory distress might rapidly develop stridor and hoarseness, signaling impending airway obstruction. Scald injuries should not be ignored as possible sources of upper airway thermal injury as hot liquids are sometimes ingested at the time of the cutaneous injury. The victim's clothing should be removed, and the chest examined for adequate expansion and the presence of circumferential burns.

Prompt and aggressive fluid resuscitation has improved survival in burn patients. Patients with extensive burns should have two large-bore intravenous lines placed in anticipation of fluid administration. It is ideal to start lines through unburned skin when possible but burns to the extremities may necessitate starting lines through burned skin. In significant burns estimated to be greater than 20% TBSA, the ABA recommends fluids can be given at an initial rate before precise burn extent and depth calculations. For children five years and younger, an initial rate of 125 mL/h of LR is recommended, in children aged 6–13, 250 mL/h, and in children aged 14 and older, 500 mL/h. Due to the focus on fluid administration, children can arrive at burn centers showing

signs of fluid overloads, such as pulmonary edema. Age-appropriate fluids should be used, understanding that more fluids do not always mean better outcomes for the patient. Insertion of a urinary catheter is prudent as urine output will be used to guide fluid resuscitation. Electrical injuries and hypothermia may lead to cardiac arrhythmias and continuous cardiac monitoring should be considered in some instances.

Burn victims will often first arrive at the nearest emergency department or hospital for stabilization before being transferred to a specialized burn center if necessary. It is here where more invasive interventions often take place to assure adequate stabilization. Patients with suspected inhalational injuries or those in respiratory distress will benefit from an arterial blood gas and carboxyhemoglobin level for confirmation. Additional testing such as serum lactate and the base deficit will set a benchmark for fluid resuscitation and help determine the need for possible intubation. Patients initially without respiratory distress might now be showing signs of airway edema in response to fluid resuscitation. Escharotomy is warranted if chest expansion becomes compromised due to circumferential chest burns. Sternal retraction during inspiration can be a sign of respiratory distress in children. In some cases, peripheral intravenous lines may not have been obtainable in the field. Central lines may need to be placed when the patient arrives, or an intraosseous line started in if vascular access is unobtainable. Venous cut-downs are typically reserved as a last resort when other intravascular or intraosseous methods have failed. Blood pressure measurements in burn patients are often unreliable, and elevated catecholamines result in ubiquitous tachycardia. Arterial lines may be required for an accurate assessment of blood pressure and are often placed in children with extensive burns for continuous hemodynamic monitoring.

In the setting of severe burns, it becomes clear that the patient will meet ABA criteria for transfer to a specialized burn center (Table 22.1). The referring hospital then works to stabilize the patient for transfer and coordinate care with the nearest burn center. Before transfer, patients should have two well-functioning intravenous access sites. A foley should be placed to monitor urine output; a goal of 1 mL/kg/h is reasonable. With their clothing removed, steps must be taken to keep children warm. A clean sheet is appropriate, but a blanket or warm fluids may be needed, especially in children with extensive burns. A nasogastric tube is placed in all intubated patients. Communication with the burn center regarding the severity of burns and travel distance will dictate the method of transportation and accompanying medical personnel.

Following the management of immediately life-threatening injuries and the primary survey, attention next should be turned to detailing the history and extent of the burn. The extent of partial and full-thickness burns is used to

Table 22.1 Burn center referral criteria (Adapted from Fundamentals of Pediatric Surgery Table 20.1)

1. Partial-thickness burns of >10% of the total body surface area
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints
3. Third-degree burns in any age group
4. Electrical burns, including lightning injury
5. Chemical burns
6. Inhalation injury
7. Burns injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
8. Burns and concomitant trauma (such as fractures) when the burn injury poses the greatest risk of morbidity or mortality. If the trauma poses the greater immediate risk, the patient's condition may be stabilized initially in a trauma center before transfer to a burn center. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols
9. Burns in children—children with burns should be transferred to a burn center verified to treat children. In the absence of a regional pediatric burn center, an adult burn center may serve as a second option for the management of pediatric burns
10. Burn injury in patients who will require special social, emotional, or rehabilitative intervention

Data from Guidelines for Trauma Centers Caring for Burn Patients, Resources for Optimal Care of the Injured Patient 2014, Committee on Trauma, American College of Surgeons, pp. 100–106

guide initial fluid resuscitation and can be calculated in adults using the rule of nines, where areas of the body correspond to well-defined percentages of total body surface area. Children's body surface area is distributed differently from adults, with the head contributing comparatively more and the extremities less (Fig. 22.1). The Palmer method refers to the victim's hand corresponding to 1% of their body surface area and is most appropriately used for small, scattered burns. The Lund and Brower chart aids the clinician in systematically evaluating each body part to yield the total body surface area (TBSA) affected. Accurate assessment of TBSA is crucial for guiding fluid resuscitation and for determining patient prognosis. It is increasingly likely that computer-aided evaluation of TBSA will become commonplace, as they have continually been shown to produce reliably accurate estimates and can easily be incorporated into the clinician's workflow. Many mobile applications have been developed for this purpose as well. The Rapid Burn Assessor application, developed in Austria, utilizes everyday objects' surface area to determine TBSA. Burn Med developed by Johns Hopkins is another mobile application available in the US. Many applications also incorporate fluid resuscitation formulae to minimize mathematical errors (Fig. 22.2).

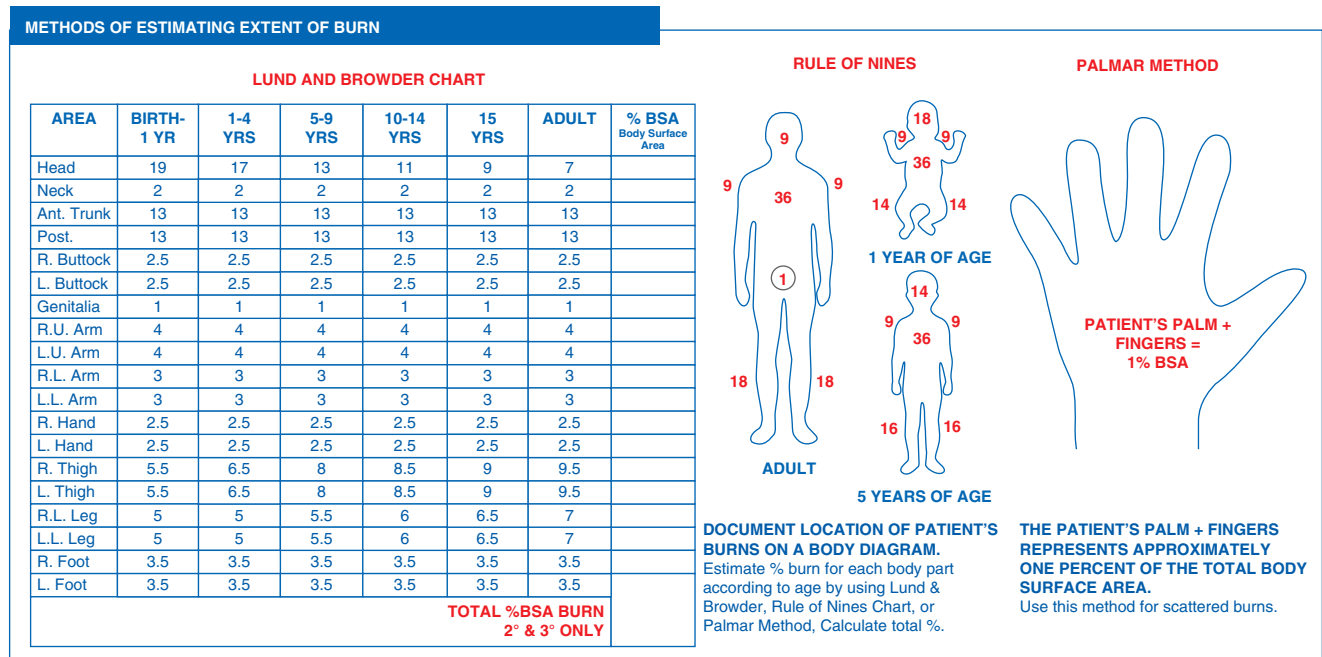


Fig. 22.1 Methods of estimating extent of burn. (Adapted from Fundamentals of Pediatric Surgery Fig. 20.1)

Accurately determining the severity of the burn, or rather its depth, is crucial for guiding surgical management and resuscitation efforts (Fig. 22.3). The skin is composed of two distinct layers differing in their regenerative capacity. Superficial, or first-degree, burns are those that only involve the epidermis. These burns will be erythematous and often

are exquisitely painful. Within a few days, the dead areas are replaced with no long-term sequelae. Superficial burns are not considered when calculating TBSA and fluid resuscitation efforts. And they do not require surgical management.

Burns extending into the dermis are classified as second-degree burns. These burns typically blister, although blistering may not have taken place at the time of initial evaluation. The dermis is composed of a more superficial papillary layer and a deeper reticular layer. Burns extending only into the papillary dermis are superficial second-degree burns. These burns are often painful and erythematous and before blistering can be mistaken for first-degree burns. Superficial dermal burns will heal without scarring and generally do not require surgical management. If a burn extends into the reticular dermis, it is considered a deep dermal or deep second-degree burn. These burns are often less sensate than more superficial burns and are less likely to be erythematous or blanch with pressure. Unlike more superficial burns, deep dermal burns will not heal on their own and require surgical management. Burns extending through the dermis into the subcutaneous tissues are known as full-thickness or third-degree burns. These burns are insensate and do not blanch. If not visibly charred, they may appear white or leathery. Early excision and grafting of burned areas remove a nidus for infection and helps diminish the likelihood of hypertrophic scars. Deeper burns into adipose tissue, muscle, and even bone are catastrophic third-degree burns, sometimes termed fourth through sixth-degree burns (Fig. 22.4).

Determining the proper management of second-degree burns is challenging for even experienced burn surgeons. Superficial second-degree burns that retain the ability to heal do better with more conservative management, while deep second-degree burns require excision and grafting. The burn injury is a dynamic process in the first few days following the initial insult. Adequate fluid resuscitation and infection prevention minimize the conversion of at-risk tissue into tissue requiring excision. Serial clinical assessments of second-

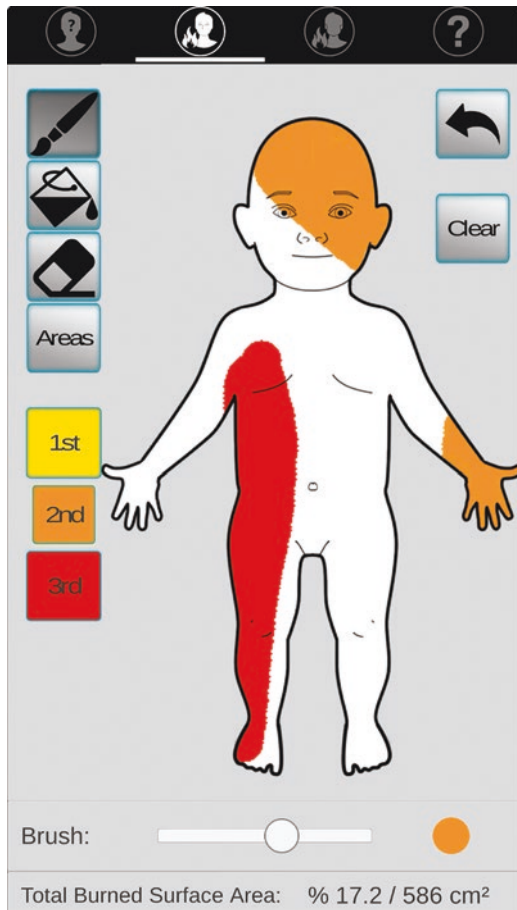


Fig. 22.2 E-burn mobile application. (Source: E-burn application in iOS App Store)

Depth	Clinical characteristics	Prognosis
Superficial (1°)	Dry, erythematous, painful, no blistering	Heals spontaneously within 1 week without scarring
Superficial partial-thickness (2°)	Weeping blisters, exposed dermis bright pink/red with brisk capillary refill, very painful	Heals spontaneously within 1–2 weeks, usually without scarring; pigmentation may be irregular
Deep partial-thickness (2°)	Some blisters, drier, exposed dermis paler/whiter, mottled	Heals spontaneously, but usually after 3 weeks, with high risk of hypertrophic scarring. Autografting usually required
Full-thickness (3°)	White, brown, black, gray or yellow, with dry, leathery, insensate eschar; sometimes may have cherry red color due to carbon monoxide poisoning	Healing by secondary intention and wound contraction with high risk of hypertrophic scarring and functional problems. Autografting required
Subdermal (4°)	Similar to full-thickness burns; thrombosed veins may be visible through eschar	Healing by secondary intention and wound contraction with high risk of hypertrophic scarring and functional problems. Autografting required

Fig. 22.3 Burn depth categories. (Adapted from Fundamentals of Pediatric Surgery Table 20.2) Illustration by Amicus Visual Solutions



Fig. 22.4 Depth (degree) of burn injury: (a) mostly superficial (1°) scald burn of thigh and leg with small area of superficial partial-thickness burn at superior/left margin; (b) mixed superficial partial-thickness and deep partial-thickness (2°) scald burns of posterior trunk; (c)

mixed deep partial-thickness (2°) and full-thickness (3°) intentionally inflicted immersion burn of the foot with classic stocking pattern; (d) mixed full-thickness (3°) and subdermal (4°) flame burns—note the thrombosed veins visible in the anterior thigh

degree burns for healing potential allow for more limited excision and improved outcomes. Scald burns, in particular, have been shown to improve after a period of watchful waiting. When burned tissue has failed to demonstrate an ability to heal, it must be promptly excised.

The gold standard for assessing burn severity is serial tissue biopsy and examination by skilled histopathologists. The drawbacks here are apparent: the need for onsite expert personnel, repeated biopsies acting as new injuries, and the potential for sampling bias in the biopsy sites. Newer methodologies have been developed to serve as adjuncts to the surgeon's clinical judgment. Imaging modalities that allow the surgeon to visualize microcirculation in the burn field have shown promise. Laser Doppler Flowmetry (LDF) was an early adjunctive technology, and based on differences in

microcirculatory blood flow patterns, demonstrated the ability to differentiate between wounds that would heal spontaneously and those that would not. Still, LDF is limited by the small field able to be visualized at one time and the need for direct contact between the probe and wound bed. Laser Doppler Imaging (LDI) improved upon many of the drawbacks of LDF. The probe no longer needed to contact the wound bed, and the imaging field was widened. LDI blood flow values appear to correlate with burn wound depth verified by biopsy. Many have confirmed the reliability and accuracy of LDI, making it a promising technology likely to become more widespread as associated adoption costs decrease over time. Other laser-based methods such as Laser Doppler Line Scanner (LDLS) and Laser speckle contrast imaging (LSCI) are also in use (Fig. 22.5).

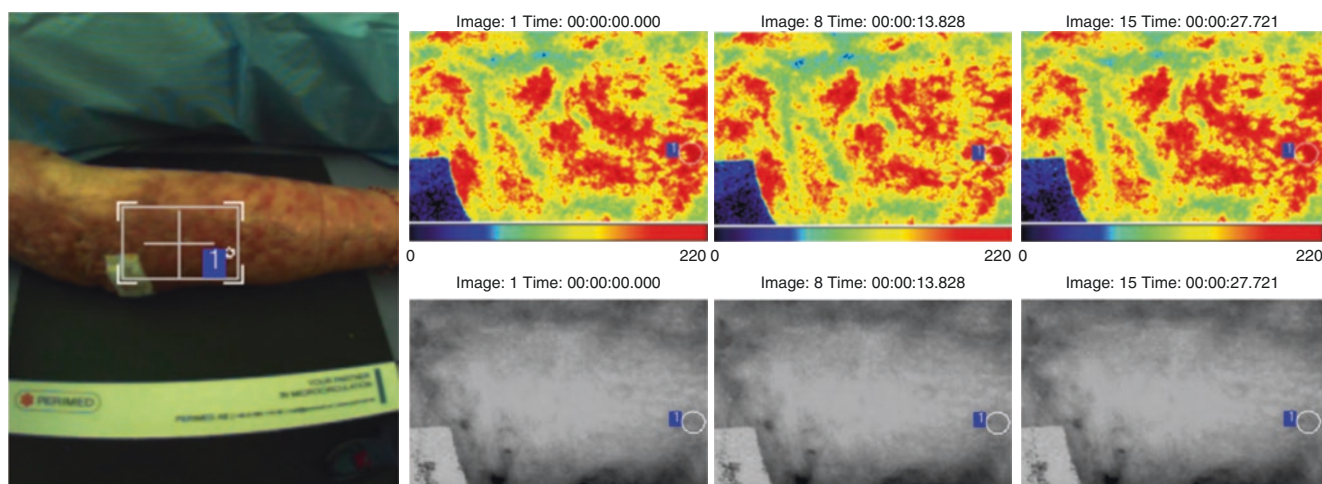


Fig. 22.5 Laser speckle imaging depicting microcirculatory blood flow patterns (Provided by Ludwik Branski, MD)

Airway Management and Inhalational Injury

The pediatric airway differs from that of an adult in several ways, making them more susceptible to airway collapse in the setting of burn-related trauma. The trachea's cross-sectional area is decreased in children such that edema can emergently result in airway obstruction. Edema may initially not be apparent but rapidly develop once fluid resuscitation begins. New-onset stridor, wheezing, hoarseness, or other signs of respiratory distress indicates impending airway obstruction. The time between these warning signs and loss of airway can be exceedingly fleeting. It is prudent to consider intubation before signs of distress in anticipation of airway obstruction. If an inhalational injury is suspected, flexible bronchoscopy should be performed to evaluate the airway. At that time, endotracheal intubation can be considered, with the endotracheal tube placed over the bronchoscope. Estimating ETT size using Broselow tape has been proven reliable, although other anatomic methods and formulae for determining ETT length exist. Pediatric patients will often be intubated in the field, and conversion to a nasopharyngeal airway will be performed at the burn center under bronchoscopy.

Once intubated, the ETT must be secured in place, a challenge in traumatized children. Typical methods involve securing the tube around the back of the head with tape or, in some cases, sublingually. Commercial tube holders are available but are not reliably superior to the more readily available tape. Many different techniques have been described for securing the ETT in the setting of facial burns, in which case swelling occurs in the first few days after injury in response to trauma and fluid resuscitation. Inflexible tape that was once secure can become overly restrictive as swelling progresses or become loose when swelling decreases. Adhering

tape to burned skin is irritating and serves as a nidus for infection. We prefer nasopharyngeal intubation and secure the ETT around the bony nasal septum. The tube is anchored off burned skin, minimizing irritation and maximizing access for burn wound care (Fig. 22.6). The bony nasal septum is traversed using 8–10 Fr red-rubber catheters placed in the nares, which are then retrieved from the oropharynx under direct laryngoscopy. Umbilical tape is tied to the catheters, which are then passed back out the nares, taking the loop of umbilical tape with them. After checking to ensure the uvula was not ensnared, the ends of the tape can be tied down tight enough to secure the tube but not so tight that surrounding structures are made ischemic. While a concern with nasopharyngeal intubation, sinusitis has not been a significant complication at our institution in over 25 years of utilizing this method.

Inhalational injury refers to damage to the airway and lung parenchyma due to heat or noxious chemicals generated during combustion. Inhalational injuries are estimated to occur in up to 20% of burn victims and are a significant, independent predictor of mortality. Burn mortality rates in children may be increased 20-fold if inhalational injuries are present. Burn history can make one suspect inhalational injury—flame burns are more likely to cause inhalational injuries. Inhalational injuries should also be considered in children found unconscious or trapped in enclosed spaces, such as a house fire. Burns to the face, singed nasal vibrissae or carbonaceous sputum raise suspicion of inhalational injury. Patients may also present with altered mental status raising suspicion of carbon monoxide poisoning. Flexible bronchoscopy can be performed to definitively diagnose upper airway inhalational injuries demonstrating soot, erythematous mucosa, mucosal blistering, and hemorrhage. Bronchoscopy can miss late changes to the airway due to a

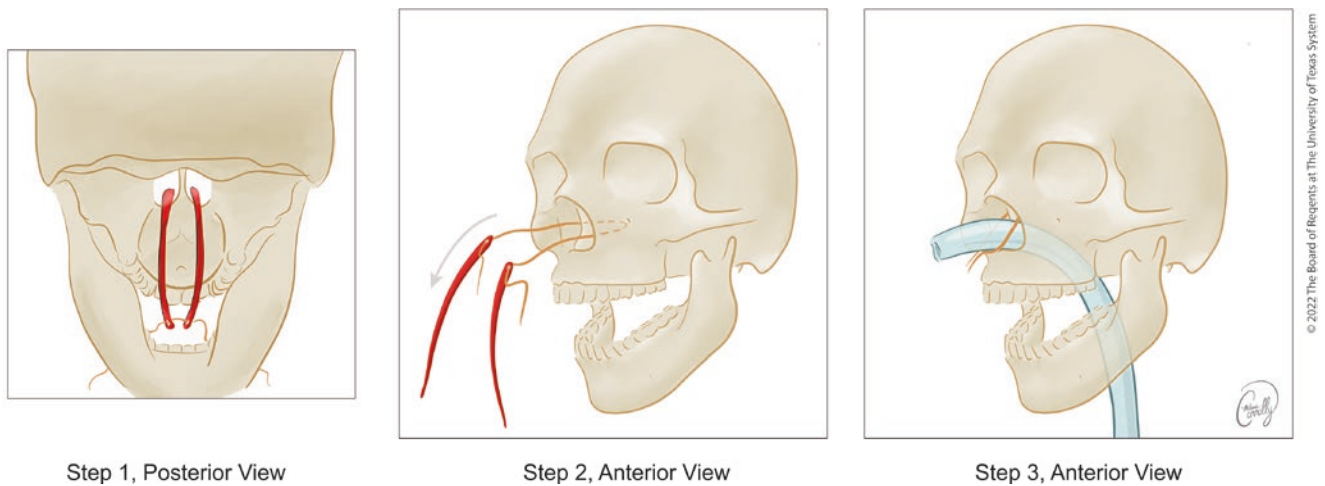


Fig. 22.6 Method of securing nasopharyngeal intubation in facial burns

delayed reaction to chemical irritants. Children with extensive cutaneous scald wounds can also experience acute lung injury and require intubation when no inhalational injury is present.

Burn victims found in enclosed spaces are at risk for carbon monoxide (CO) and cyanide (CN) poisoning. CO is an odorless, tasteless gas responsible for many fatalities occurring at the site of a fire. It is formed by the incomplete combustion of many common organic substances found around the home. CO causes tissue hypoxia by binding to hemoglobin with an affinity 200 times greater than that of oxygen. Systemic signs and symptoms correlate to blood carboxyhemoglobin levels. Tissues most sensitive to hypoxia, such as the brain, are affected first. Patients rarely present with the cherry-red appearance classically associated with CO poisoning. Likewise, patients may not be tachypneic as carbon dioxide expiration is not impaired. Cyanosis is similarly absent as the amount of oxygen dissolved in the plasma is unaffected. Arterial blood gas and carboxyhemoglobin levels should be drawn in patients with suspected CO exposure, such as those in a house fire or other enclosed space. Laboratory derangements will reflect tissue hypoxia, and patients with severe poisoning can be expected to have increased base deficits and lactate levels. Patients are frequently placed on supplemental oxygen during transit to the hospital, delaying the presentation of CO poisoning. A nomogram can be used to estimate the actual degree of CO exposure based on the time that has passed since being removed from the scene of the fire. Treatment with 100% oxygen decreases the half-life of CO. The role of hyperbaric oxygen therapy is unclear but is sometimes considered in patients with signs of severe CO poisoning unresponsive to 100% oxygen. Hyperbaric oxygen therapy is not widely available and should not be initiated if it delays transfer to a burn center.

The combustion of synthetic household products such as plastic, carpet, and upholstered furniture produces hydrogen

cyanide (CN). The gas is colorless and classically described as having the odor of bitter almonds. The presence of other volatile compounds at the scene of a fire makes detection of this scent unlikely. CN disrupts oxidative phosphorylation by binding to mitochondrial cytochrome c oxidase. Tissue anoxia then results, and victims may be rapidly rendered incapable of escape, contributing to mortality. CN toxicity possibly contributes more to mortality than CO does, but its true prevalence is unknown given the inability of routine laboratory testing to detect blood cyanide levels. CN presentation may be subtle and easily masked by other life-threatening injuries, widespread burns, or suspected CO poisoning. Disruption of oxidative phosphorylation first impacts the cardiovascular and central nervous systems. Clinical suspicion of CN poisoning should be high in the patient with persistent metabolic acidosis and elevated serum lactate, unresponsive to oxygen therapy. These patients are candidates for empiric treatment with a hydroxocobalamin antidote kit. Laboratory confirmation of blood cyanide level is unlikely to be available. The ABA suggests hydroxocobalamin be used in unresponsive patients and thus undergoing CPR. There is no high-level evidence establishing appropriate hydroxocobalamin dosing in pediatric patients. Previously a dose of 70 mg/kg has been recommended, but caution should be taken with all patients, monitoring them for signs of systemic toxicity.

Inhalational injuries are anatomically divided between those that occur above or below the glottis. Reflex laryngeal closure often protects subglottic structures from direct thermal damage; however, this may not be the case in the setting of explosions or steam-related inhalational injuries, which are capable of forcing the larynx open. Supraglottic edema may rapidly compromise the airway after the onset of fluid resuscitation, and early intubation is warranted if an inhalational injury is suspected. Injury below the glottis is almost always due to noxious chemicals. These toxins found in

smoke directly damage the epithelium and trigger a host of pathophysiologic changes that compromise ventilation. Increased mucus secretion, generation of free radicals, activation of inflammatory pathways, and epithelial sloughing contribute to the formation of fibrin plugs and airway obstruction. Epithelial sloughing may be delayed as much as 5 days after an accident leading to delayed respiratory collapse.

Management of inhalational injuries is supportive and involves airway maintenance, clearance of secretions, and ventilation when appropriate. Meticulous pulmonary hygiene, early mobilization, and chest physiotherapy mobilize airway debris and reduce atelectasis, both of which are important in preventing pneumonia. Chest wall oscillation has been used with success to clear mucoid secretions, and bronchoalveolar lavage has been used to remove obstructions and collect microbiological samples when indicated. Inhaled agents to facilitate the breakdown of fibrin plugs and reduce free radicals have been used. Aerosolized heparin and N-acetylcysteine may decrease the duration of intubation and mortality in pediatric patients. Inhalational racemic epinephrine has also been shown to be a safe adjunctive in patients already receiving heparin and acetylcysteine and may improve respiratory status by reducing bronchospasm and mucus hypersecretion in pediatric inhalational injury patients.

Burn Resuscitation and Assessment

Burn shock refers to the severe cellular and physiologic derangements occurring shortly after the massive tissue injury in severe burns. Dysfunction at multiple levels works against victims. Intravascular fluid leaks into the interstitial space due to increased capillary permeability in response to inflammatory mediators. These inflammatory mediators coupled with a loss of preload lead to global cardiac dysfunction and decreased cardiac output. Peripheral vascular resistance becomes labile. Shortly after a burn, peripheral resistance is high, later developing into vasoplegia. Fluid resuscitation alone cannot entirely resolve this complex, systemic insult. Great strides in burn care during the twentieth century were due in part to the adoption of aggressive fluid resuscitation strategies. Although delays in resuscitation lead to end-organ failure and death, aggressive resuscitation is not without risk. The term *fluid creep* refers to a trend of administering more substantial fluid volumes than predicted by resuscitation formulae. Over-resuscitation risks the development of extremity, orbital, and abdominal compartment syndromes. The development of airway and pulmonary edema risk respiratory failure, and cerebral edema is an under-recognized complication. Thus, the challenge becomes to maintain end-organ function at a minimal physiologic expense. Adequate fluid

resuscitation is a careful balancing act that achieves organ perfusion while avoiding over-resuscitation.

Prompt fluid resuscitation saves lives. Peripheral IV access is preferred; starting two large-bore IV lines provides a safety net in the case of one dislodging or becoming non-functional. Central lines may be necessary should the extremities be compromised in the setting of severe burns. An intraosseous line can be lifesaving when vascular access is otherwise temporarily unobtainable. The proximal anterior tibia is often the preferred site, but the medial malleolus, anterior iliac crest, or distal femur are alternatives. A bone marrow aspiration needle, spinal needle, or specialized interosseous needle is used to cannulate the marrow, taking care to avoid epiphysis, with the bevel facing the longer length of the bone. The use of pumps should be avoided in case the needle becomes dislodged, with the fluid being infused by gravity.

Resuscitation with crystalloid fluids should begin immediately after IV access is established, preferably with LR. In patients who appear to have a greater than 20% TBSA burn, an initial fluid rate should be administered based on patient age. Once stabilized the patient can be weighed, and TBSA assessed as part of the secondary survey. Mechanism of burn and the potential of inhalational injury should be considered as these may influence the volume of fluid needed for adequate resuscitation. Formulae for burn resuscitation in children are based on total body surface area, calculated based on their weight and height (Table 22.2). A commonly used resuscitation formula calls for 5000 mL/m² TBSA burned plus 2000 mL/m² TBSA as maintenance fluid. This volume is calculated and administered over the first 24 h after a burn, with half the amount being administered in the first 8 h and the remaining half given over the next 16 h. In the following 24 h and for as long as the burn remains open, the suggested volume decreases to 3750 mL/m² TBSA burned plus 1500 mL/m² TBSA as maintenance fluid. Children under 30 kg should receive 5% dextrose at a maintenance rate to prevent hypoglycemia.

Electrolyte derangements are likely to develop in the post-burn period. Serum chemistries should be monitored

Table 22.2 Pediatric resuscitation formula (Adapted from Merrell SW et al. 1994)

Shriner's Cincinnati (for children)	LR: 4 mL/kg/% TBSA burn + 1500 mL/m ² , 1/2 given over the first 8 h and the remaining over the next 16 h (older children)
	LR: 4 mL/kg/% TBSA burn + 1500 mL/m ² + 50 mEq. sodium bicarbonate for the first 8 h, followed by LR alone in the second 8 h, followed by 5% albumin in LR in the third 8 h (younger children)
Galveston Formula (for children)	LR: 5000 mL/m ² burn + 2000 mL/m ² total, 112 volume in the first 8 h, followed by remainder in 16 h

regularly and electrolytes repleted promptly. Hyponatremia is typically the first abnormality observed in the first 48 h post-burn and should be supplemented accordingly. Young children require more supplementation due to increased urinary sodium losses. Hypophosphatemia develops due to a myriad of reasons, reaching a nadir 2–5 days post-burn, and can be repleted orally, or if severe, intravenously.

Burn resuscitation formulae generate an initial total volume estimate that must be titrated regularly to optimize fluid administration. While most formulae prescribe fluid volumes administered in 8- and 16-h increments, monitoring of clinical response to resuscitation should occur on an hourly basis. An indwelling catheter is essential for monitoring urine output to assess clinical response to fluid resuscitation. Sources differ regarding target urine output. The ABA recommends a target of 1 mL/kg/h of urine output in children up to 30 kg. Children 30 kg and heavier have the same target as adults, 0.5 mL/kg/h. Other institutions have higher goals, such as 1 mL/kg/h in children and 2 mL/kg/h in infants.

Laboratory studies are valuable for determining response to resuscitation. Improvements in lactate level and base deficit indicate adequate fluid resuscitation. Early investigations into assessing burn resuscitation focused on hemoconcentration as a measure of inadequate fluid administration. Hemodilution should be expected given the volume of fluid administered. Some patients are unresponsive to fluid resuscitation with appropriate volumes of crystalloids. Rather than using pressor support in these patients, colloids including albumin, fresh frozen plasma, or even PRBC may be initiated as early as 6 h post-burn to augment crystalloids already administered. Urine output lags in some burn patients who clinically show other signs of adequate fluid resuscitation. Early renal replacement therapy should be considered in these patients rather than increases in fluid volumes. When pressors must be used, epinephrine is preferred as it has the lowest impact on skin perfusion. When adequate fluid resuscitation and pressors do not maintain tissue perfusion, cardiac dysfunction may be present and should be augmented with dobutamine.

Certain situations can be anticipated where the predicted fluid resuscitation volume will be inadequate. Electrical burns mandate higher fluid resuscitation volumes and efforts are made to quickly dilute out myoglobin released from damaged muscles to prevent cast formation in the renal tubules. Inhalational injuries frequently require increased fluid volumes. The volume required can vary greatly and patients should be started on a standard formula-based volume and have output titrated accordingly in anticipation of volumes in excess of those predicted. Should the volume of fluid required for resuscitation significantly exceed the amount predicted, further evaluation for additional trauma, infection, or cardiovascular dysfunction should be performed.

Other adjuvant techniques have been used to monitor the hemodynamic status of burn patients. Pulmonary arterial catheters (PAC), once the standard of care for hemodynamic monitoring in critically ill patients have been replaced by less invasive methods of assessing cardiovascular function. Patients who fail to show clinical improvement with large fluid volumes often receive a transthoracic echocardiogram (TTE) to assess hemodynamic parameters. Despite improvements in imaging quality, it has been demonstrated that TTE may overestimate cardiac output in children. Pulse index Continuous Cardiac Output (PiCCO) has emerged as a promising method of accurately determining hemodynamic parameters in severely burned children. Making use of transpulmonary thermodilution (TPTD), cold saline is infused through a central venous line, and temperature changes are measured by a thermistor arterial catheter. Severely burned children often have arterial and venous access, so no additional lines need to be placed, and the increased risk is minimal. Important indicators of preload and pulmonary edema can be determined with PiCCO. Other noninvasive methods of assessing hemodynamics are in use but less validated. Near-infrared spectroscopy (NIRS) is noninvasive and can be performed at the bedside to estimate regional capillary venous hemoglobin saturation. Methods of estimating cardiac output based on bioimpedance have also been described.

Care must be taken to avoid over-resuscitation as its complications can prove deadly. The development of abdominal compartment syndrome necessitating laparotomy has been associated with near 100% mortality. Malfunctioning IV lines rapidly lead to the development of extremity compartment syndrome (ECS). Often the clinical signs commonly associated with ECS, pain, pallor, and pulselessness, are difficult to appreciate in the burned limb of a sedated child. Over-resuscitation can lead to right heart failure in pediatric patients as well. The use of colloid is generally limited in burn resuscitation as it is not superior to crystalloid solutions. After the 8–12 h mark, it has been determined that albumin is likely to remain in the intravascular space as the capillary leak begins to resolve. As such, *colloid rescue* has been employed to normalize resuscitation in pediatric patients.

Burn Wound Infection

Burn victims are susceptible to infection. The skin and its microbiome are significant components of the innate immune system, working to prevent invasive infections by pathogenic organisms. Infections are a leading cause of mortality in burn victims, be it in the burn wound itself or infections secondary to iatrogenic interventions such as mechanical ventilation or indwelling urinary catheters. The post-burn hypermetabolic state leaves patients immunocompromised, and vulnerable to

opportunistic infections. Preventing infection is a significant challenge for the burn surgeon and involves assiduous source control, prompt identification of microorganisms, and surgical excision and grafting.

Thermal injury to the skin creates a sterile environment, leaving behind no commensal or pathogenic microorganisms immediately following a burn. The vulnerable burn wound becomes colonized by organisms either from the environment or from the victim's own gut or nasopharyngeal microbiome. The ABA reached a consensus regarding burn wound colonization, defining it as low concentrations of bacteria (under 10^5 organisms per gram tissue) and a lack of invasive infection (Greenhalgh et al. 2007). In the resource-rich environment of the burn wound, a competition is then afoot between rapidly multiplying microorganisms and the host attempting to epithelize the wound and restore its defenses. The efforts of the surgical team are directed at augmenting the host response and sabotaging potentially invasive microorganisms. Briefly, excision of devitalized tissue dissuades microorganism proliferation. Temporary wound coverage or definitive grafting restores a natural barrier to infection and promotes reepithelization. Topical soaps and antiseptics reduce microorganism burden and promote a healing environment. Systemic antimicrobials neutralize invasive species and help stagnate proliferation in perfused areas of tissue. Hemodynamic support and adequate nutrition facilitate the host's own immune response.

Diagnosis of infection in burn patients presents a challenge. Systemic signs and symptoms commonly associated with infection such as fever and tachycardia will be ubiquitous in extensive burns. Physician examination and clinical judgment then become indispensable. Burn wound erythema is the result of inflammatory mediators from the burned tissue leading to a cellulitic appearance in the surrounding tissue and is easily mistaken for infection. Clinical suspicion of an infectious etiology should be raised in the patient with erythema accompanied by induration and new-onset tenderness. If cellulitis is suspected, systemic antibiotics are warranted targeting the most likely species. Should tissue become nonviable due to infection, surgical debridement becomes necessary. Invasive burn wound infections typically manifest with changes in wound color and odor. Ideally, histologic examination of the tissue would prove microorganism invasion; in practice, a preliminary diagnosis is made clinically and confirmed with quantitative tissue cultures. Systemic antimicrobial therapy should be sufficiently broad until susceptibilities are available, at which time therapy may become more tailored.

Gram-positive bacteria commonly infect burn wounds. *Staphylococcus aureus* is of particular concern given its propensity to disseminate hematogenous and high rates of resistance. Streptococci are also common culprits of infection and graft failure and are more susceptible to penicillins and cephalosporins than resistant strains of *S. aureus*. Enterococci

are less prevalent, but vancomycin-resistant strains may necessitate less frequently used antibiotics such as linezolid or quinupristin/dalfopristin.

Of the Gram-negative bacteria associated with burn wounds, *Pseudomonas aeruginosa* is the most prevalent and the most associated with lethal. Infected wounds classically develop a yellow-green discoloration and fruity odor. Interestingly, *P. aeruginosa* may be acquired not only from the environment but also via translocation from the patient's gastrointestinal tract. Piperacillin/tazobactam, cefepime, or carbapenems are typically used empirically, but the rate of MDRO is on the rise.

Fungal burn wound infections are becoming increasingly problematic, due in part to the use of broad-spectrum antibiotics. Patients are colonized with fungal spores at the time of the burn or through a nosocomial source. Fungal cultures may take considerably longer to identify the organism and direct targeted antifungal therapy, making an early diagnosis that much more critical. The presence of invasive mold in a burn wound is a true surgical emergency mandating radical debridement combined with high potency topical and systemic antifungals.

Infection control measures in the burn unit limit nosocomial infections, the use of antibiotics, and the development of drug-resistant organisms. Contact precautions are standard for all patients. Broad-spectrum antimicrobials are acceptable when organisms and susceptibilities have yet to be determined but should be narrowed appropriately. Cooperation with institutional infectious disease experts and knowledge of local antibiograms is essential for maximizing treatment effect and minimizing the risk of drug resistance.

Hypermetabolism

Severe burns over 30% TBSA cause a systemic stress response. This hypermetabolic state acutely affects every organ system. This stress response is mediated by increased levels of catecholamines, cortisol, and proinflammatory cytokines that may be persistently elevated years post-burn. Inflammation, muscle wasting, cardiac dysfunction, growth retardation in children, impaired insulin sensitivity, and hormonal dysfunction are hallmarks of the hypermetabolic state. Children, in particular, suffer long-term morbidity affecting their physical and emotional quality of life. Elevated levels of catecholamines profoundly stress the cardiovascular system, raising resting heart rate and cardiac output in a phenomenon that persists post-burn. The chronic hyper-sympathetic state is detrimental to cardiac myocytes. Myocardial oxygen consumption increases and may outpace delivery. In response, focal degeneration of the myocardium may occur, manifesting as fibrosis, hypertrophy, or dilatation. Heart failure, local myocardial hypoxia, and cardiac

death also may result. The hypermetabolic state causes a pro-motes catabolic environment, resulting in wasting of lean body mass (LBM). The body transitions to a prolonged state of increased resting energy expenditure (REE), associated with dysregulation of glucose, lipid, and protein metabolism. The rate of protein breakdown surpasses protein synthesis, resulting in a net loss of muscle protein. Increased energy expenditure and protein catabolism lead to a loss of LBM and changes in whole-body composition. This state of chronic catabolism and loss of LBM is also persistent. Therapies and agents targeting the hypermetabolic state are essential in mitigating long-term sequelae in burn survivors and typically involve early initiation of pharmacologic agents and optimal nutrition.

Temperature Regulation

When a significant surface area of skin is lost, thermoregulation becomes a challenge on multiple levels. The body is deprived of insulation, and evaporative cooling occurs through the burn wound. Inflammatory cytokines raise the hypothalamic temperature setpoint, and resting energy expenditure is increased. Care should be taken to minimize a child's exposure to a cool environment. The ambient room temperature should be maintained at 30–33 °C to reduce energy demands and evaporative water losses. In addition to blankets, radiant warmers and warmed intravenous fluids may also be warranted. Situations, where the child is exposed, should be minimized and done under optimized environmental conditions. Tub rooms and operating rooms should be appropriately warmed to minimize heat loss during bathing and surgical procedures. Early wound coverage, either with autograft or temporary occlusive dressings, helps to reduce evaporative losses and is a critical component of addressing the hypermetabolic state.

Several serious consequences are associated with hypothermia. The myocardium is more susceptible to changes in electrolyte concentrations, and arrhythmias may develop. Altered mental status, respiratory depression, coagulopathy, and loss of peripheral vasomotor tone have also been reported. Notably, recent evidence suggests that mitochondrial thermogenesis is a significant component of hypermetabolism. New technologies for wound coverage, such as synthetic skin products, drug therapies, and environmental strategies, should all be explored as methods to further attenuate hypermetabolism after burn injury.

Nutrition

Optimal nutrition in the post-burn period plays a vital role in mitigating muscle wasting and supplementing the protein needed for the wound healing process. Nutritional support

should be started within 1–2 h of admission, and a nasoduodenal or nasojejunal tube should be placed in children with significant burns to facilitate early alimentation. Early enteral feeding supports intestinal blood flow and motility and begins to ameliorate the hypermetabolic response. Evidence suggests that bacterial translocation from the gut is minimized by early feeding. If enteral feeding is not feasible or unable to adequately nourish the patient, then total or supplemental parenteral nutrition should be initiated. Enteral feeding can continue safely throughout dressing changes and operative procedures with little risk of aspiration if feeding tubes are placed beyond the pylorus.

The hypermetabolic response demands an increase in caloric intake. Resting energy expenditure (REE) has been repeatedly found to be 1.3–1.5 times higher in burn patients. Matching these needs allows patients to better thermoregulate in the post-burn period and compensate for the increased metabolic demands mediated by elevated catecholamine, cortisol, and proinflammatory cytokines. Patients should be weighed daily, and indirect calorimetry should be completed weekly to determine REE and guide caloric goals. Oxygen consumption and carbon dioxide production are determined, and energy expenditure is calculated. Different formulae have been developed to estimate caloric needs in pediatric burn patients based on TBSA and REE (Table 22.3).

The macronutrient composition of feedings is critical for facilitating healing and preventing complications. Protein must be supplemented with requirements up to 4 g/kg being recommended. Feedings containing high levels of carbohy-

Table 22.3 Formula estimating caloric requirements in pediatric burn survivors (Adapted from Al-Mousawi A et al. 2009) BSA, Body surface area; RDA, recommended dietary allowance

Formula	Sex/age (years)	Equation (daily requirement in kcal)
WHO	Males 0–3	$(60.9 \times W) - 54$
	3–10	$(22.7 \times W) + 495$
	10–18	$(17.5 \times W) + 651$
	Females 0–3	$(61.0 \times W) - 51$
	3–10	$(22.5 \times W) + 499$
	10–18	$(12.2 \times W) + 746$
RDA	0–6 months	$108 \times W$
	6 months to 1 year	$98 \times W$
	1–3	$102 \times W$
	4–10	$90 \times W$
	11–14	$55 \times W$
Curreri junior	<1	$RDA + (15 \times \% \text{BSAB})$
	1–3	$RDA + (25 \times \% \text{BSAB})$
	4–15	$RDA + (40 \times \% \text{BSAB})$
Galveston infant	0–1	$2100 \text{ kcal/m}^2 \text{BSA} + 1000 \text{ kcal/m}^2 \text{BSAB}$
Galveston revised	1–11	$1800 \text{ kcal/m}^2 \text{BSA} + 1300 \text{ kcal/m}^2 \text{BSAB}$
Galveston adolescent	12+	$1500 \text{ kcal/m}^2 \text{BSA} + 1500 \text{ kcal/m}^2 \text{BSAB}$

drates provide fuel and spare supplemented proteins for wound healing. Supplemental feedings high in fat are associated with hepatic steatosis and increased mortality. Essential fats must be supplemented, but the overall fat content of feedings should be modest. Findings from a study in 14 children with greater than 40% TBSA burns showed that low-fat (3% of energy), high-carbohydrate (82% of energy) enteral formulas reduced muscle wasting by 40% compared with formulas with more typical fat (15% of energy) and carbohydrate (70% of energy) compositions (Hart et al. 2001). Rarely will a pediatric burn patient receiving enteral feeding not experience diarrhea. It is prudent to decrease the osmolarity of feedings at this time. Electrolytes should be monitored regularly in order to adjust for free water substitution.

Pharmacologic Regulation of the Hypermetabolic Response

A number of pharmacologic agents have been studied to modulate the hypermetabolic response. Children, in particular, will benefit from early initiation of these therapies to minimize long-term sequelae such as growth delay and cardiovascular dysfunction. Propranolol is one of the most studied drugs in the management of severe burns and is the standard of care in our pediatric burn patients. Non-specific β -adrenergic blockade with propranolol has been shown to have widespread benefits in burn survivors. Cardiac work and resting energy expenditure are decreased, manifested by a decreased resting heart rate. Protein metabolism is also revised, resulting in decreased protein catabolism and improvements in LBM at 12 months post-burn. Recombinant human growth hormone (rhGH) has been shown to have similar benefits to propranolol—tamped hypermetabolic response, lower cardiac output, improvements in LBM, and protein metabolism. Additionally, it appears to augment the body's healing response, decreasing skin graft donor site healing times.

Growth delay is a serious concern in the pediatric burn survivor and is a readily apparent manifestation of the persistent hypermetabolic state. Long-term rhGH administration has been shown to support anabolism and growth in children, improving height percentiles well after the burn. Oxandrolone, a testosterone analog, has also been studied on the basis of combating protein catabolism post-burn. A 5-year follow-up of children with major burns randomly assigned receive to either oxandrolone for 12 months showed accelerated growth, improvements in LBM, bone mineral content, and heart percentiles at the two-year mark (Porro et al. 2012). Generally, oxandrolone is preferred to rhGH given cost concerns, especially if ongoing courses are required.

Wound Management

Historically, full-thickness burn wounds were serially debrided, removing only small amounts of eschar at a time. This left devitalized eschar present in the burn wound for extended periods of time, acting as a nidus for infection and contributing to a high bacterial burden resulting in wound infections and burn sepsis. Early surgical intervention of even massive burn wounds can be safely performed in children and is associated with improved clinical outcomes and overall mortality (Fig. 22.7). Partial-thickness burns are the most common burn injuries in children. Typically, these wounds retain healing potential and will re-epithelize without intervention. The burn wound should be washed and debrided of devitalized tissue if necessary. Traditionally, topical antimicrobial agents were used for the treatment of partial-thickness burn wounds to create a moist, antiseptic environment for healing to take place. However, these agents required frequent application and dressing changes, are locally painful, and may have toxic systemic effects. Silver sulfadiazine, once a mainstay of burn care, has been replaced by synthetic and biological wound dressings. The development of wound dressings requiring less frequent changes was a marked advancement in pediatric burn care and improved patient comfort. Biobrane, an early flexible biosynthetic wound dressing used in partial-thickness burns, is shown to be safe in children and improve pain. Pediatric patients treated with Biobrane experienced shorter hospital stays and healing times compared to topical antimicrobials. The next-generation product AWBAT (Advanced Wound Bioengineered Alternative Tissue) has now largely replaced Biobrane on the market. Other commonly used dressings are Mepitel and Mepilex Ag. Both also have been shown to reductions in healing time and pain and require infrequent dressing changes. Other synthetic wound dressings include Suprathel, UrgoTul, and Allevyn. Biological tissues are also available for temporary wound coverage in partial-thickness burns, such as porcine xenograft skin. Xenografts become adherent and provide the benefits of temporary wound coverage, such as pain control, while the underlying wound re-epithelializes. The human amniotic membrane is used in many parts of the world as a relatively cheap temporary dressing for superficial wounds and may reduce lengths of stay, frequency of dressing changes, and pain.

Partial-thickness burns may appear to be an indeterminate depth shortly after injury due to a mosaic of superficial second-degree burns, which retain the ability to heal, and deep second-degree burns, which do not. Indeterminate-depth burns wounds covering less than 20% TBSA are best managed conservatively rather than with early excision. Unless the wound is clearly full thickness, the burn wound can reasonably be

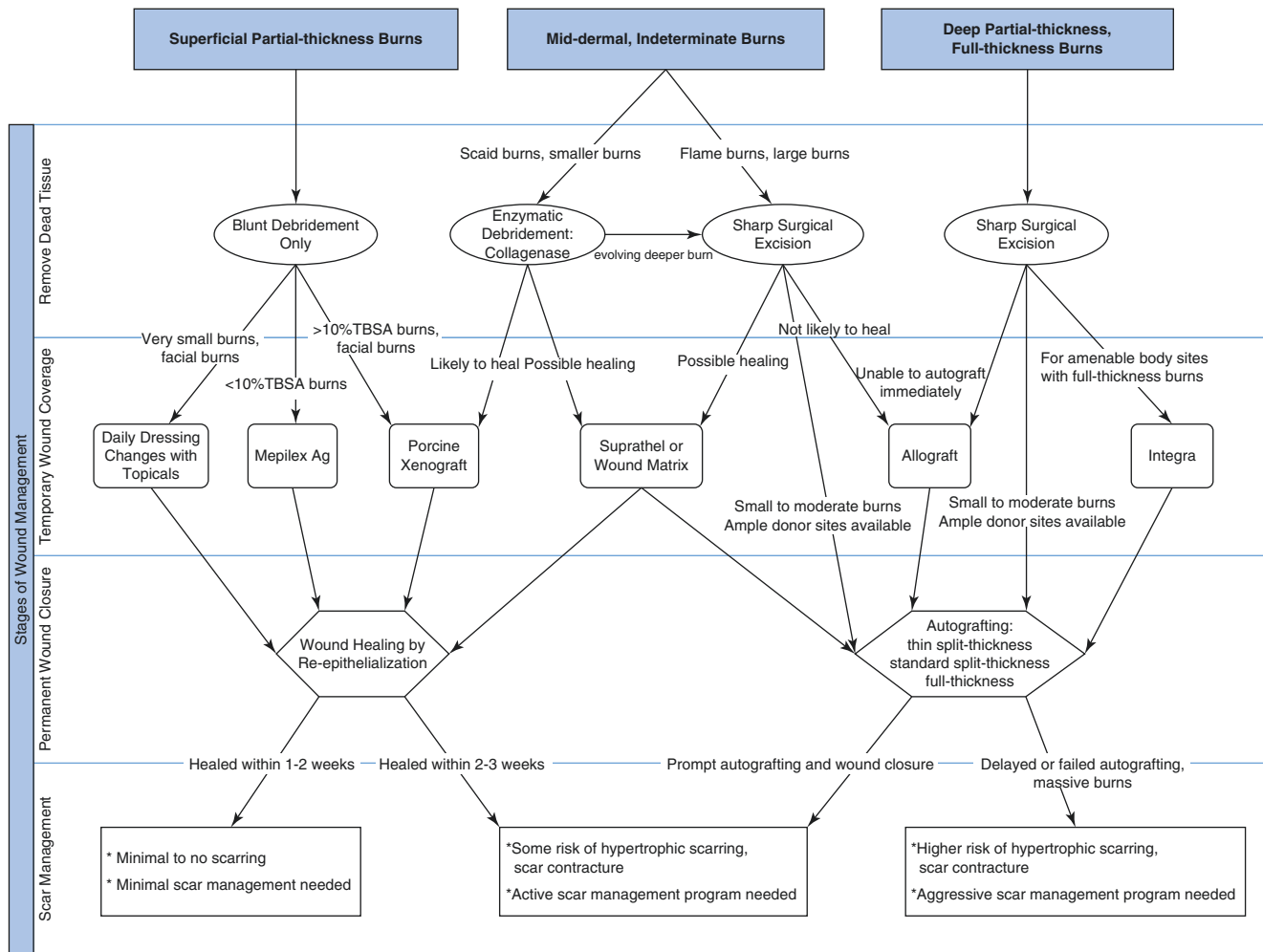


Fig. 22.7 Algorithm for pediatric burn wound management. (Adapted from Fundamentals of Pediatric Surgery Fig. 20.4)

given time to demonstrate healing potential before excision. This strategy minimizes the amount of tissue excised. Burns that heal within 21 days generally do so without the development of hypertrophic scars or functional impairment. Burn wounds healing longer than 21 days often result in unsightly hypertrophic scars, affecting physical function and mobility.

Surgical Treatment

The surgical treatment of full-thickness burns involves escharotomy and fasciotomy when indicated, followed by burn eschar excision. Early excision improves mortality and should take place promptly once the patient has been adequately stabilized for the operating room. Operative excision of a burn wound is indicated for all full-thickness burns and a deep-partial thickness burn that have failed to demonstrate healing

potential. Commonly, burn wounds are excised by tangential excision. Thin layers of burn eschar and nonviable dermis are shaved from the wound until healthy tissue with brisk punctate bleeding is reached. The most commonly used tools for tangential excision are the Goulian knives, the Watson knife, and the VersaJet Hydrosurgery System. Burn excision can result in significant blood loss and it is prudent to anticipate the need for blood products in patients with extensive burns. Adjuvant agents for reducing blood loss include tumescent solutions containing epinephrine and tourniquets.

Autografts

Once the burn wound is excised, autograft skin can be used for coverage. Autografts can be harvested by different techniques, depending on burn extent and location. Autografts are classi-

fied as split-thickness or full-thickness, depending on the depth of tissue harvested. Full-thickness autografts, those that contain the entire dermis, have better cosmetic results compared to split-thickness grafts. Full-thickness grafts are primarily used for deep facial, hand, and genital burns, as they function as an ideal replacement for burned skin. Full-thickness donor sites are limited to areas that can be closed primarily. The inguinal region is a common choice in pediatric patients.

Split-thickness grafts are used for major burns covering an extensive surface area. In small burns, split-thickness grafts can be applied unmeshed in sheets, which results in a better cosmetic result. Sheet grafts can be used in areas such as the face, neck, and hands, where cosmesis and function are of the greatest concern. A disadvantage of sheet grafts is the potential risk of developing seromas or hematomas underneath, impairing adherence to the wound bed. Fluid accumulations between the graft and wound bed should be managed by aspiration with a 25- or 27-gauge needle. Skin grafts can be secured by sutures, staples, Steri Strips, or by application of a bolster dressing, which can reduce shearing of the skin graft. In extensive burns, autografts are limited; therefore, to increase surface area, grafts are meshed to a ratio of 2:1 or 4:1. Large areas can be grafted with a ratio of 4:1; however, they require coverage by a nonexpanded allograft skin to decrease the risk of graft loss, applied over the meshed skin graft in a sandwich pattern. Another common technique for grafting extensive burns is the Meek technique: skin grafts are cut into micrografts, which are pressed onto pre-folded pleated gauze that is expanded in all four directions. These autografts can be expanded at a ratio of up to 1:9 and provide a sufficient expansion ratio enabling grafting of patients with burns up to 75% TBSA (Kreis et al. 1993).

Dermal Replacement

If donor sites for skin grafts are not available, skin substitutes such as allograft, porcine xenograft, Suprathel, Integra, or BTM can be used for temporary coverage until donor sites are available again (Fig. 22.8). Temporary coverage has numer-

ous advantages, including minimizing fluid loss, pain relief, and reduced scarring. Suprathel represents a solid, reliable epidermal skin substitute, which can be used to cover deep dermal burn wounds and to save split-thickness grafts from donor sites for the coverage of full-thickness burned areas. The most commonly used dermal replacement is Integra, a bilaminar composite composed of a neodermis of bovine collagen held in a matrix of shark cartilage chondroitin-6-sulfate. The matrix is fully incorporated into the wound bed in 2–3 weeks, at which time a split-thickness skin graft can be applied over the top. The RECELL System is a device that produces a suspension of spray-on skin cells using a small sample of the patient's own skin. This suspension contains the cells necessary to regenerate the outer layer of natural, healthy skin.

Burn Reconstruction

Unsightly scars, hypertrophic scarring, and burn scar contracture are common complications requiring reconstructive procedures in burn patients. It is vital to create a realistic plan regarding potential cosmetic and functional outcomes and the timing of operations. Some patients are troubled by the appearance of burn scars, while others less so. Understanding a patient's goals is crucial to successful reconstruction. Before a scar can be reconstructed, the initial burn wound should be treated and allowed to heal fully. During this time, the scar should be treated conservatively using pressure garments, silicone sheets, and sunscreen. Physical and occupational therapy are involved early, guiding exercise programs that minimize joint contracture and can effectively reduce the need for surgical interventions. Standard surgical techniques for burn reconstruction involve Z-plasty, local fascio-cutaneous flaps, and tissue rearrangement. Full-thickness skin grafts may be placed to relieve areas of contracture. Adjuvants such as tissue expansion, fat grafting, and various lasers may be employed as well. Other procedures higher up on the reconstructive ladder may also be necessary.

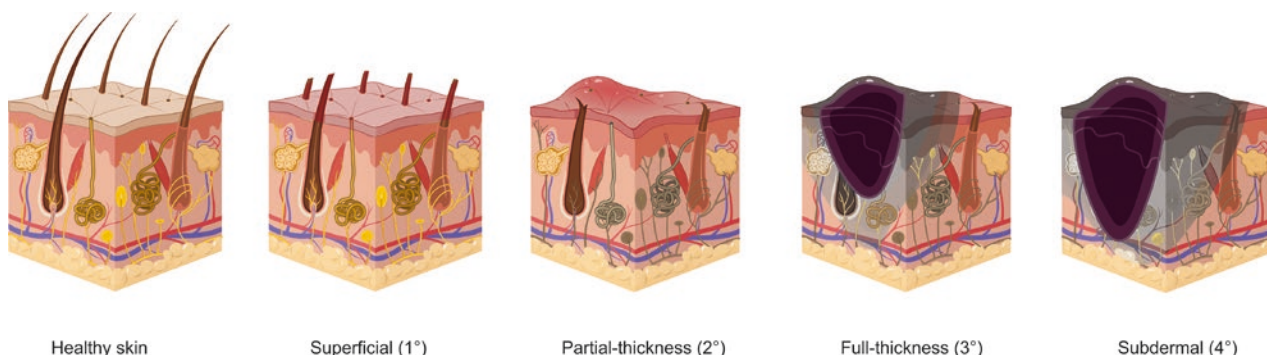


Fig. 22.8 Common dermal replacements

Pain Management

Burns are painful not only during the initial insult but also during routine interventions that are hallmarks of burn care. Patients experience substantial pain during surgical procedures, bathing, dressing changes, and physical activity. Tissue damage releases cytokines resulting in pain in the burned area and in surrounding tissue. Second-degree burns result in variable amounts of pain depending on the amount of destruction to the dermis. Full-thickness burns are initially anesthetic due to the complete destruction of the dermis and its rich network of nerve endings. Over time children begin to complain of intense pain during reintervention and manipulation in the burned area (dressing changes). Pruritus is another common complaint in these patients that may be particularly debilitating. Pain management is a critical element of burn care, especially with the knowledge that uncontrolled pain is correlated with feelings of anxiety, depression, anorexia, fatigue, and helplessness. Furthermore, poorly managed pain risks the development of dysesthesia and chronic pain.

The subjective experience of pain is quantified using validated tools and scales. Assessment has to be individualized and consider the child's age, clinical condition, and preferences. Verbal pain scales that assess pain severity are the most commonly used assessment tools. Studies have shown that burned children preferred face and color scales over visual analogs and adjective scales. After the assessment and quantification of discomfort, a treatment regime can be considered and planned. In general, there are three options/modalities for pain management: surgical, pharmacological, and behavioral. From a surgical standpoint, grafting of open burn wounds significantly reduces pain, and open burn wounds should be grafted as soon as possible. There are also several topical agents available, which have been reported to reduce pain, such as Cellulose dressings or Suprathel.

Pharmacologic management of burn pain is a mainstay of therapy. In general, analgesics are most effective when given on a regular schedule to prevent pain rather than given in response to breakthrough pain. Furthermore, the pain medication should not be given as an intramuscular injection since injections themselves cause pain and anxiety. Children should be reassessed regularly and dosing titrated carefully to avoid undertreatment or complications of overdose. The intravenous route is the preferred mode of analgesic administration in the acute phase post-burn. Morphine sulfate is one of the most commonly used analgesics and has been described as the gold standard in pediatric burn patients. Fentanyl is another opioid analgesic with a shorter duration and rapid onset of action, which makes it a suitable choice for analgesia during painful procedures. Furthermore, it is available as a flavored Oralet lozenge and has been used successfully in the outpatient setting for dressing changes and

Table 22.4 Challenges of sedation and analgesia in pediatric burn patients

• Intense but brief pain requiring rapid onset and short duration of drug effects to avoid overdose or delayed recovery
• Lack of intravenous access, especially for outpatient care
• Nutritional requirements for large burns preclude frequent fasting periods for deep sedation
• Exaggerated anxiety states
• Staffing resources may limit availability of personnel credentialed to provide deep sedation

wound care. For burn debridement and dressing changes, patients receive either oral oxycodone and either minimal sedation with oral or intravenous morphine and midazolam or moderate sedation with intravenous midazolam and ketamine. Most outpatient children who are scheduled for reconstructive procedures are treated with hydrocodone/acetaminophen, while those undergoing major surgical interventions require longer-acting narcotics such as methadone. Several other factors have an impact on sedation and analgesia in burn procedures (Table 22.4).

Burn Prevention

National burn prevention and education efforts have significantly decreased the number of pediatric burns each year (Fig. 22.9). The most significant risk of thermal injury for children is in the home. The kitchen represents a dangerous environment, especially for children under the age of five years who are at higher risk for contact burns and scalds. Children often unconsciously reach for hot objects. Therefore, cups with hot drinks should never be placed at the edge of tables. Pots and pans should have the handle facing away from where a child may reach up and grasp it. Adults should avoid carrying children and hot beverages at the same time. Lowering the temperature set point on water heaters and educating families to check the bathwater temperature before placing a child in the bath reduces scald burns. The importance of placing smoke detectors in multiple areas of the house cannot be overstated. Children should never be left alone with candles or open fires, and access to matches and lighters should be impossible. Certain aerosol sprays are highly flammable, and care should be taken to keep them away from children and open flames. Current prevention education focuses on children and especially infants who are not able to remove themselves from a fire. Educating children as early as possible that fire is dangerous is imperative. Providing safe environments for children and providing appropriate education is the responsibility of caregivers, the community, and health-care providers.



PEDIATRIC **SCALDS: A Burning Issue**

DO

- Set water heater temperature to no higher than 120°F/48°C, or just below the medium setting
- Create a “no kid zone” in the kitchen around stoves, ovens and hot items
- Keep hot drinks away from the edge of tables and counters
- Use a travel mug with a tight-fitting lid for all hot drinks
- Place pots and pans on the back burner with handles turned away from the edge of the stove

THE FACTS

- ✓ The average annual cost of scald injuries is \$44 million
- ✓ Over 136,000 children were seen in emergency rooms for burn injuries in 2011
- ✓ 1,100 children die each year from fire and burns



DON'T

- Leave a child unattended in the bathtub; if you must leave, take the child(ren) with you
- Allow young children to adjust the water temperature or sit near faucet handles
- Set anything hot on tabletops within reach of young children who can pull them down
- Allow appliance cords (slow-cookers, deep-fryers, coffeemakers) to dangle over the counter edge

THE FIX

If a burn injury does happen...

- 1) Cool the burn with COOL (not cold) water to stop the burning process
- 2) Remove all clothing and/or diaper from the injured area
- 3) Cover the area with a clean dry sheet or bandages
- 4) Seek medical attention



American Burn Association
www.ameriburn.org | 312.642.9260

This material is for information purposes only. It is not a substitute for professional medical advice, diagnosis, or treatment, which you should seek from your physician. The ABA does not endorse any specific product, service or treatment.

Fig. 22.9 Pediatric scalds: a burning issue. (Source: The American Burn Association, ABA)

Editor's Comments

In addition to the ABCs of trauma care and management of the burn wound itself, care of the burned child demands careful consideration of many simultaneous and sometimes competing issues: social and legal concerns, the psychological and emotional care of the child, alleviating pain and anxiety and, of course, ruling out other injuries. Making matters worse is the fact that for the severely injured child, the care provided in the trauma bay is transitional, as they are accepted by the first responders and safely prepared for transfer to a pediatric burn center. Transfers of care increase the risk of errors and omissions, highlighting the importance of exceptional attention to detail and the risk of potential complications. One should never assume that the care provided at another institution or in the field was adequate or sufficient—always check endotracheal tube position by auscultation and chest X-ray, be sure that the cervical spine is properly immobilized, and perform a careful physical assessment, including the calculation of the TBSA, yourself. Likewise, if the patient is being transferred, it is important to think ahead as to what will be needed on the receiving end: rather than applying an opaque cream, cover the burns with clear ointment and dry or simple sterile dressings that can be easily removed for a proper assessment of the depth and extent of the injury; secure adequate intravenous access and hydrate the patient well; avoid long-acting muscle relaxants to allow an accurate assessment of neurologic status after transfer, and be sure that copies of all films and medical records accompany the patient.

Every surgical resident is taught about the Parkland formula, the fact that burn victims require a large amount of fluid, and that more fluid is always better, often leading to fluid overload and massive edema. It is nice to see that a more reasonable fluid-resuscitation approach that is physiologic and goal-based. Likewise, it has become clear that early enteral feedings—within hours rather than days of injury—improve outcomes. Finally, evidence-based approaches to infection prevention, treatment of sepsis, burn-wound management, and medical therapy of the typical post-burn hypermetabolic state are welcome additions to the modern care of the child with severe burns.

Silvadene has been the mainstay of burn therapy for many years, though many now prefer to use petrolatum-based antibiotic ointments, which are transparent, keep the wounds moist, and do not necessarily need to be removed or washed off before every application. The antibiotic concentration in most topical antibiotic preparations is too high for use in the eye, so in little children, it is best to use the ophthalmic-strength ointment for burns on the face or hands, where it might easily be transferred to the eyes.

Sadly, many burn wounds in children are due to child abuse (often a misguided attempt to punish) or simple negligence. It is therefore important to be skeptical, without being overtly accusatory, in all but the most obvious cases. It is far better to err on the side of caution and to involve child protective services or social work early in the process, before discharging a child into a potentially unsafe or abusive home environment. This is especially true for scald burns (not just classic dip burns), contact burns (from an iron or hot plate), and localized flame burns. When in doubt, the child should be admitted overnight for observation and further assessment.

Further Reading

- Al-Mousawi A, Branski LK, Andel HL, et al. Ernährungstherapie bei Brandverletzten. In: Kamolz LP, Herndon DN, Jeschke MG, editors. *Verbrennungen: diagnose, therapie und rehabilitation des thermischen traumas*, German edition. New York: Springer-Verlag; 2009. p. 183–94.
- Alcorta R. Smoke inhalation & acute cyanide poisoning. Hydrogen cyanide poisoning proves increasingly common in smoke-inhalation victims. *JEMS*. 2004;29(8):6–15.
- American Burn Association. National burn repository 2019 update: Report of data from 2009 to 2018. *Natl Burn Repos 2019 Updat Rep data from 2009–2018*. 2019; 60, 606(312).
- Armstrong M, Wheeler KK, Shi J, et al. Epidemiology and trend of US pediatric burn hospitalizations, 2003–2016. *Burns*. 2021;47(3):551–9.
- Barret JP, Herndon DN. Effects of Burn Wound Excision on Bacterial Colonization and Invasion. *Plast Reconstr Surg*. 2003;111(2):744–50.
- Breederveld RS, Tuinebreijer WE. Recombinant human growth hormone for treating burns and donor sites. *Cochrane Database Syst Rev*. 2014;15:CD008990.
- Carvajal HF. Fluid resuscitation of pediatric burn victims: a critical appraisal. *Pediatr Nephrol*. 1994;8(3):357–66.
- Chong HP, Quinn L, Jeeves A, et al. A comparison study of methods for estimation of a burn surface area: Lund and Browder, e-burn and Mersey Burns. *Burns*. 2020;46(2):483–9.
- Desai MH, Rutan RL, Herndon DN. Conservative treatment of scald burns is superior to early excision. *J Burn Care Rehabil*. 1991;12(5):482–4.
- Gee Kee EL, Kimble RM, Cuttle L, Khan A, Stockton KA. Randomized controlled trial of three burns dressings for partial thickness burns in children. *Burns*. 2015;41(5):946–55.
- Goldbaum LR, Orellano T, Dergal E. Mechanism of the toxic action of carbon monoxide. *Ann Clin Lab Sci*. 1976;6(4):372–6.
- Gore DC, Hawkins HK, Chinkes DL, et al. Assessment of adverse events in the demise of pediatric burn patients. *J Trauma Inj Infect Crit Care*. 2007;63(4):814–8.
- Greenhalgh DG, Saffle JR, Holmes JH, et al. American burn association consensus conference to define sepsis and infection in burns. *J Burn Care Res*. 2007;28(6):776–90.
- Hart DW, Wolf SE, Chinkes DL, Lal SO, Ramzy PI, Herndon DN. β -blockade and growth hormone after burn. *Ann Surg*. 2002;236(4):450–7.
- Herndon DN, Gore D, Cole M, et al. Determinants of mortality in pediatric patients with greater than 70% full-thickness total body surface

- area thermal injury treated by early total excision and grafting. *J Trauma Inj Infect Crit Care*. 1987;27(2):208–12.
- Herndon DN, Hart DW, Wolf SE, Chinkes DL, Wolfe RR. Reversal of catabolism by beta-blockade after severe burns. *N Engl J Med*. 2001;345(17):1223–9.
- Hoffman GM, Ghanayem NS, Tweddell JS. Noninvasive assessment of cardiac output. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu*. 2005;8(1):12–21.
- Hundeshagen G, Herndon DN, Clayton RP, et al. Long-term effect of critical illness after severe paediatric burn injury on cardiac function in adolescent survivors: an observational study. *Lancet Child Adolesc Heal*. 2017;1(4):293–301.
- Jaskille AD, Ramella-Roman JC, Shupp JW, Jordan MH, Jeng JC. Critical review of burn depth assessment techniques: part II. Review of laser doppler technology. *J Burn Care Res*. 2010;31(1):151–7.
- Jaskille AD, Shupp JW, Jordan MH, Jeng JC. Critical review of burn depth assessment techniques: part I. Historical review. *J Burn Care Res*. 2009;30(6):937–47.
- Jeschke MG, Chinkes DL, Finnerty CC, et al. Pathophysiologic response to severe burn injury. *Ann Surg*. 2008;248(3):387–401.
- Jeschke MG, Gauglitz GG, Kulp GA, et al. Long-term persistence of the pathophysiologic response to severe burn injury. *PLoS One*. 2011;6(7):e21245.
- Jeschke MG, van Baar ME, Choudhry MA, Chung KK, Gibran NS, Logsetty S. Burn injury. *Nat Rev Dis Prim*. 2020;6(1):11.
- Kahn SA, Bell DE, Stassen NA, Lentz CW. Prevention of hypophosphatemia after burn injury with a protocol for continuous, preemptive repletion. *J Burn Care Res*. 2015;36(3):e220–5.
- Kealey GP. Carbon monoxide toxicity. *J Burn Care Res*. 2009;30(1):146–7.
- Kendrick D, Smith S, Sutton AJ, et al. The effect of education and home safety equipment on childhood thermal injury prevention: Meta-analysis and meta-regression. *Inj Prev*. 2009;15(3):197–204.
- Khorasani EN, Mansouri F. Effect of early enteral nutrition on morbidity and mortality in children with burns. *Burns*. 2010;36(7):1067–71.
- Kreis RW, Mackie DP, Vloemans AFWP, Hermans RP, Hoekstra MJ. Widely expanded postage stamp skin grafts using a modified Meek technique in combination with an allograft overlay. *Burns*. 1993;19(2):142–5.
- Merrell SW, Saffle JR, Sullivan JJ, et al. Fluid resuscitation in thermally injured children. *Am J Surg*. 1986;152(6):664–9.
- Mlcak RP, Suman OE, Herndon DN. Respiratory management of inhalation injury. *Burns*. 2007;33(1):2–13.
- Mosier MJ, Peter T, Gamelli RL. Need for mechanical ventilation in pediatric scald burns. *J Burn Care Res*. 2016;37(1):e1–6.
- Porro LJ, Herndon DN, Rodriguez NA, et al. Five-year outcomes after oxandrolone administration in severely burned children: a randomized clinical trial of safety and efficacy. *J Am Coll Surg*. 2012;214(4):489–502.
- Porter C, Tompkins RG, Finnerty CC, et al. The metabolic stress response to burn trauma: current understanding and therapies. *Lancet*. 2016;388(10052):1417–26.
- Ratcliff SL, Brown A, Rosenberg L, et al. The effectiveness of a pain and anxiety protocol to treat the acute pediatric burn patient. *Burns*. 2006;32(5):554–62.
- Retrouvey H, Chan J, Shahrokhi S. Comparison of two-dimensional methods versus three-dimensional scanning systems in the assessment of total body surface area estimation in burn patients. *Burns*. 2018;44(1):195–200.
- Saffle JI. The phenomenon of “fluid creep” in acute burn resuscitation. *J Burn Care Res*. 2007;28(3):382–95.
- Schulte J. Anchoring endotracheal tubes on patients with facial burns. Review from Harborview Hospital Seattle, Washington. *J Burn Care Rehabil*. 8(3):235–6.
- Sheridan R. Skin substitutes in burns. *Burns*. 1999;25(2):97–103.
- Vloemans AFPM, Hermans MHE, van der Wal MBA, Liebrechts J, Middelkoop E. Optimal treatment of partial thickness burns in children: A systematic review. *Burns*. 2014;40(2):177–90.
- Walker PF, Buehner MF, Wood LA, et al. Diagnosis and management of inhalation injury: an updated review. *Crit Care*. 2015;19(1):351.
- Wang R, Zhao J, Zhang Z, Cao C, Zhang Y, Mao Y. Diagnostic accuracy of laser doppler imaging for the assessment of burn depth: a meta-analysis and systematic review. *J Burn Care Res*. 2020;41(3):619–25.
- Weinstein RA, Mayhall CG. The epidemiology of burn wound infections: then and now. *Clin Infect Dis*. 2003;37(4):543–50.
- Williams FN, Herndon DN, Jeschke MG. The hypermetabolic response to burn injury and interventions to modify this response. *Clin Plast Surg*. 2009;36(4):583–96.
- Williams FN, Jeschke MG, Chinkes DL, et al. Modulation of the hypermetabolic response to trauma: temperature, nutrition, and drugs. *J Am Coll Surg*. 2009;208(4):489–502.
- Wolf SE, Rose JK, Desai MH, Mileski JP, Barrow RE, Herndon DN. Mortality determinants in massive pediatric burns. *Ann Surg*. 1997;225(5):554–69.
- Zak AL, Harrington DT, Barillo DJ, et al. Acute respiratory failure that complicates the resuscitation of pediatric patients with scald injuries. *J Burn Care Rehabil*. 1999;20(5):391–9.