Chapter 36

The pediatric burned patient

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Introduction

Burn injuries are the third most common injury causing death in children, following motor vehicle crash and drowning accidents.[1] Burn injuries have the greatest length of stay (average of 7.8 days) for all hospital admissions due to injuries.[2] Approximately 1.25 million people are burned in the United States every year. Fortunately, most burns do not require hospitalizations; however, 60 000 to 80 000 patients sustain severe enough burns to demand hospital admission.[3,4] Of those, 40% are children younger than 15 years.[5] About 5500 burn patients die each year,[3] and approximately 2500 of them are children.

House fires injure or kill over 10 000 people per year. House fires are among the leading causes of burn-related deaths in children at a rate of 12%; they are the leading cause of injury-related deaths for black children between the ages of 1 and 9 years. Children between 0 and 5 years of age are at a greater risk as a disproportionate number of fire deaths occur in homes. Deaths among preschool children are at a rate of more that twice the national average (29.6 deaths/million children) or an average of 20% of the total percentage of all home fire deaths.

Scalds are another common burn injury in children 2 years and under. Scald injuries may be due to household accidents or deliberate abuse. These may include spilling hot coffee or water, children reaching up to counter tops pulling pot handles or cords attached to cooking appliances and spilling the contents onto themselves, unknowingly putting body parts under a hot water faucet or climbing into a hot tub without realizing the water was too hot, and intentionally or unintentionally being placed into or brought in contact with a hot substance by another individual.

Great improvements have been made in the reduction of mortality related to thermal injuries over the past few decades. Advances in fluid resuscitation, early surgical excision and grafting of the burn wound, infection control, treatment of inhalation injury, nutritional support, and support of hypermetabolic response to injury have contributed to a 50% decline in burn-related deaths and hospital admissions in the United States.[3] This overall improvement in mortality is most perceptible in the pediatric population. In 1949, Bull and Fisher reported the expected 50% mortality rate for 49% total body surface area (TBSA) burn size in children aged 0 to 14.[6] This has improved to 50% expected mortality in 98% TBSA burn in the same population group.[7]

The burn injury produces overwhelming physiological and psychological challenges to a child. The unique anatomical and physiological attributes of the child require the attention of physicians and nurses who are trained not only in burn care but also in the specifics of pediatric care. The most obvious differences between adults and children are in size and body proportion. Shorter lengths, tighter angles, and smaller diameters of various anatomical structures and spaces make certain manipulations more difficult. These differences also require the provision of special equipment and supplies, which reflect the configurations of pediatric anatomy. In addition to anatomical differences there are also many physiological differences between children and adults, which must be considered and will be discussed concerning the treatment of the pediatric burn patient.

Initial evaluation

A patient must be immediately removed from the source of burn and clothing and jewelry removed immediately as these burning items can prolong the burning process. Pouring cool water to minimize the depth of the burn can cause hypothermia in large burns and should be avoided. After the burning process is stopped, the patient should be kept warm by wrapping with a sterile sheet or blanket, if available, or clean sheet or blanket. If the burn involves chemical burns, the patient should be removed from the chemical immediately and the wound irrigated with copious amount of water at least for 30 minutes to dilute the chemicals.

Burn patients should be treated as a trauma patient and any other traumatic injuries diligently ruled out. Any potential life-threatening injuries should be identified and treated.

The airway should be assessed first: 100% oxygen should be administered and oxygen saturation monitored using pulse oximetry. Arterial blood gas and carboxyhemoglobin are obtained when appropriate. A full-thickness circumferential chest burn can interfere with ventilation. Chest expansion should be observed to ensure equal air movement. If the patient is on a ventilator, airway pressure and P_{CO_2} should be monitored. If ventilation is compromised, escharotomy of the chest should be performed to allow better chest movement and improve ventilation. Wheezing, tachypnea, stridor, and hoarseness indicate an impending airway problem due to inhalation injury or edema, and immediate attention is required.

A cuff blood pressure measurement may be difficult in patients with burned extremities. These patients may need an arterial line to monitor their blood pressure especially if they require a long transfer. A radial arterial line may not be reliable in patients with extremity burns and may be difficult to secure. A femoral line may be more reliable and easy to secure. These should be secured with sutures. Persistent tachycardia should alert a practitioner for a missed injury. Accurate and rapid determination of burn depth is vital to the proper management of burn injury.

A nasogastric tube is placed in all patients with major burns, as the majority of children develop gastric distension or ileus. A bladder drainage catheter is placed to monitor urine output accurately as a measure of successful resuscitation.

Resuscitation

In the hours after a serious burn, there is a systemic capillary leak that increases with burn size. Capillary usually regains competence after 18–24 hours if resuscitation has been successful. Intravenous (IV) access should be established immediately for the administration of resuscitative fluid. Increased times to beginning resuscitation of burned patients results in poorer outcomes, and delays should be minimized.[8] It is crucial that venous access be obtained early postburn, even though such access may be extremely difficult to obtain. Due to the small circulating volume, delays in resuscitation for periods as short as 30 minutes can result in profound shock. Peripheral IV access is preferred and may need to go through burned skin if necessary. When peripheral IV is not available due to extremity burns, a central venous line may be secured. Either subclavian or femoral line can be obtained but femoral venous access may be easier to obtain in edematous patients. Small-bore catheters limit the rates at which fluid can be administered; therefore, children with large burns require two large-bore IVs so that sufficient fluid can be given. The presence of two IVs also provides a safety margin if one infiltrates to allow continued resuscitation while the 'safety' line is re-established.

When vascular access is unobtainable in small children (less than 6 years old), the intraosseous route is a viable option and it is relatively easy to obtain. Children can be administered fluid volumes in excess of 100 mL/h directly into the bone marrow.[9] Intramedullary access can be utilized in the proximal tibia until IV access is accomplished. There is a very rare incidence of embolic complications with this procedure. A 16–18 gauge bone marrow aspiration needle can be used to cannulate the bone marrow compartment, although spinal needles and even butterfly needles can be pushed through the soft bone of a child. Although previously advocated only for children younger than 3 years of age, intraosseous fluid administration can be safely performed in children under 6 years of age, if the bone is sufficiently soft to allow needle penetration.[10] The anterior tibial plateau, medial malleolus, anterior iliac crest, and the distal femur are preferred sites for intraosseous infusion. The needle should be introduced into the bone, taking care to avoid the epiphysis, either perpendicular to the bone or at a 60° angle with the bevel facing the greater length of bone (Figure 36.1). The needle has been properly inserted when bone marrow can be freely aspirated. Fluid should be allowed to infuse by gravity drip. The needle should be well secured to prevent inadvertent removal. The use of pumps should be discouraged in case the needle is dislodged from the marrow compartment.

Due to their small body weight to body surface area ratio, fluid losses are proportionally greater in children. Normal blood volume in children is approximately 80 mL/kg body weight and neonates, 85–90 mL/kg, compared to the adult whose normal blood volume is 70 mL/kg. Evaporative water losses in a 20% TBSA burn in a 10 kg child are 475 mL or 60% of the circulating volume, while the same size burn in a 70 kg adult causes the loss of 1100 mL or only 25% of the blood volume. Although fluid losses after a burn injury are directly proportional to the burned surface area, the commonly used

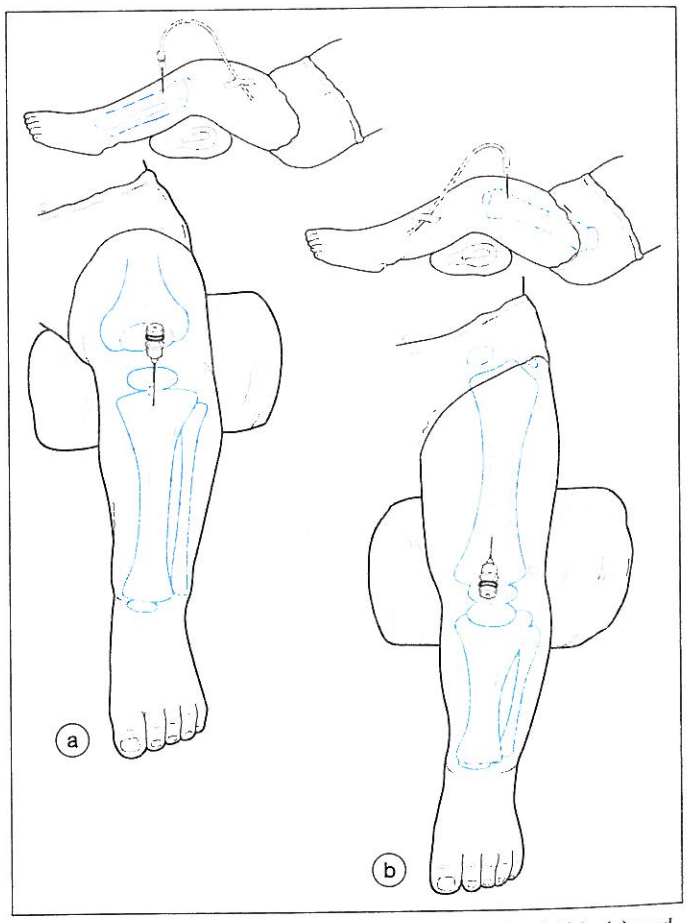

Fig. 36.1 Intraosseous line placement in the proximal tibia (a) and distal femur (b). (Redrawn with permission from Fleisher and Ludwig.[60])

'rule of nines', useful in adults, adequate in adolescents, does not accurately reflect the surface area of children under 15 years of age (Figure 36.2). The standard relationships between surface area and weight in adults do not hold true in children, as infants possess a larger cranial surface area with less area in the extremities than adults. Most routinely used resuscitation formulas were developed using adult patients and are almost exclusively weight-based. Since the linear relationship between weight and surface area does not exist in children (surface area varies to weight as a 2/3 function), use of these formulas in children results in under- or over-resuscitation (Table 36.1).

Therefore pediatric burned patients should be resuscitated using formulas based on body surface area, which can be calculated from height and weight using a standard nomogram (Figure 36.3) or formulas (Table 36.2). The most commonly

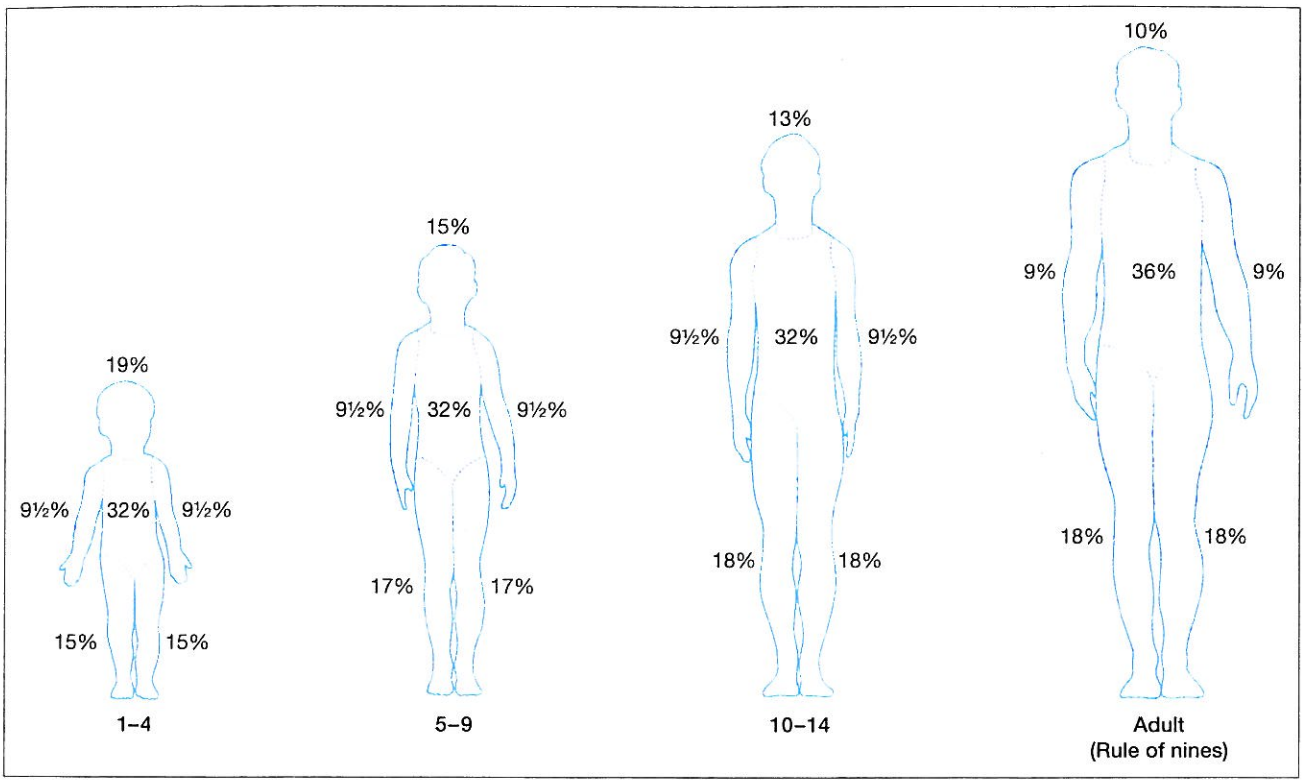

Fig. 36.2 The 'rule of nines' altered for the anthropomorphic differences of infancy and childhood.

TABLE 36.1 RESUSCITATION BY THE PARKLAND FORMULA ONLY COMPARED TO MAINTENANCE FLUID REQUIREMENTS ALONE

		Calculated needs		Replacement burn loss	
Example	% Burn	Resuscitation*	Maintenance†	mL	mL/kg/%
1 year old	15	600	800	−200	−1.33
10 kg	30	1200	800	400	1.33
0.48 m² BSA	60	2400	800	1600	2.67
	90	3600	800	2800	3.11
4 years old	15	990	1200	−210	−0.85
16.5 kg	30	1980	1200	780	1.58
0.68 m² BSA	60	3900	1200	2760	2.79
	90	5940	1200	4940	3.33
12 years old	15	2400	2250	1150	1.92
40 kg	30	4800	2550	2550	2.12
1.13 m² BSA	60	9600	2250	7350	3.06
	90	14400	2250	12150	3.38

*4 mL/kg/% burn.
†2000 mL/m² BSA.

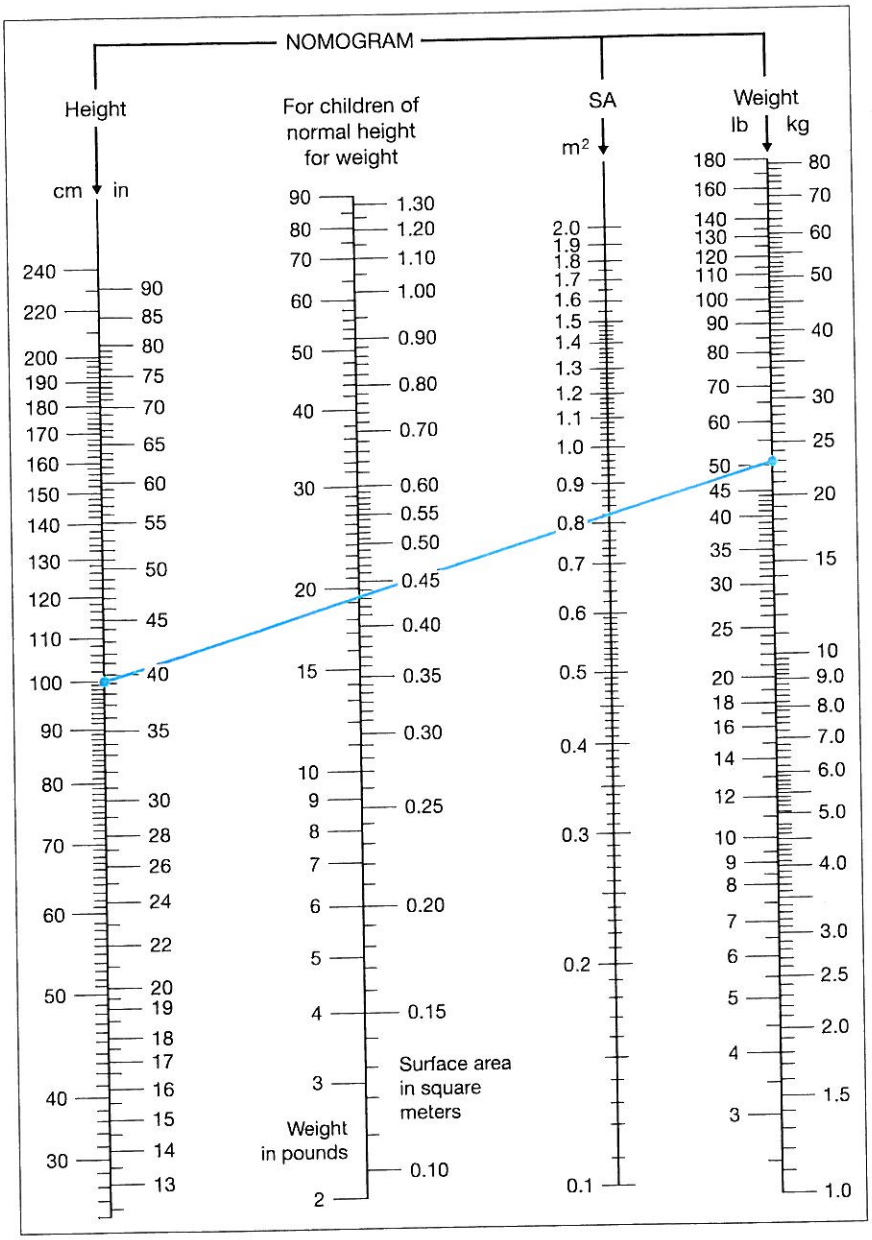

Fig. 36.3 Standard nomogram for the determination of body surface area based on height and weight. The example depicted is for a child of 100 cm in height and 23 kg in weight. (Reprinted with permission from Eichelberger.[61])

TABLE 36.2 FORMULAS FOR CALCULATING BODY SURFACE AREA (BSA)	
Dubois formula	BSA (m^2) = ht (cm)$^{0.725}$ × wt (kg)$^{0.425}$ × 0.007184
Jacobson formula	BSA (m^2) = [ht (cm) + wt (kg) − 60]/100

used resuscitation formula in pediatric patients calls for the administration of 5000 mL/m^2 total body surface area (TBSA) burned plus 2000 mL/m^2 TBSA for maintenance fluid given over the first 24 hours after burn, with half the volume administered during the first 8 hours and the second half given over the following 16 hours.[11] The subsequent 24 hours, and for the rest of the time their burn is open, call for 3750 mL/m^2 TBSA burned or remaining open area (for evaporation from wound) plus 1500 mL/m^2 TBSA (for maintenance requirements). This need decreases as a patient achieves more wound coverage and healing. As in the adult patient, resuscitation formulas offer a guide to the initial starting point for the amount of fluid necessary for replacing lost volume in children and the amount of fluid should be titrated according to effects.

Loss of renal medullary concentrating capacity is usual secondary to washout of the medulla during resuscitation and immature kidneys' inherent inability to concentrate. Hyponatremia is a frequently observed complication in pediatric patients after the first 48 hours post-injury. Frequent monitoring of serum sodium is necessary to guide appropriate salt or water supplementation. Children of less than 1 year of age may require more sodium supplementation due to higher urinary sodium losses. Further, potassium losses should usually be replaced with oral potassium phosphate rather than potassium chloride as hypophosphatemia is frequently observed in this population.[12] Calcium and magnesium losses must also be supplemented.

An indwelling urinary catheter is essential for burns greater than 20%. During the early phase of resuscitation urine output should be assessed as frequently as every 15 minutes and titrated appropriately. Fluid administration should be titrated to achieve a urine output of 1 mL/kg/h in children and 2 mL/kg/h for infants. If the patient is making more than that, IV fluid should be titrated down. Other endpoints should also be followed, such as mental status, heart rate, blood pressure, and capillary refills. One can also follow the trend of either lactic acid or base deficit and see the resolution. Initial fluid boluses should be administered in amounts appropriate for the size of the child and should represent no more than 25% of the total circulating volume (10 mL/kg). If urine output is less than minimal, it may be more appropriate to increase the rate of IV fluid rather than giving bolus.

Intravenous resuscitation fluid should be isotonic and replace lost electrolytes. Lactated Ringer's (LR) is the most commonly used solution for the first 24 hours postburn. Electrolyte loss in the wound correlates better with LR. Children less than 1 year of age should also receive maintenance fluid containing dextrose solution to prevent hypoglycemia as their glycogen stores are limited.

Mortality

In a review of 103 children with >80% TBSA burns over a 15-year period, it was found that 69 survived with an overall mortality of 33%. Mortality was greatest in children under 2 years of age and in burns >95% TBSA (Figures 36.4 and 36.5).[8] Another major predictor in mortality was the length of time to intravenous access (Figure 36.6). Burns that received resuscitation fluids within the first hour had a significantly higher chance of survival.[8] The mortality rate also increases significantly with smoke inhalation injury. In no pediatric patient, no matter how large the burn, how young or with what type of inhalation injury could it be accurately predicted whether they would live or die at the time of admission.[8]

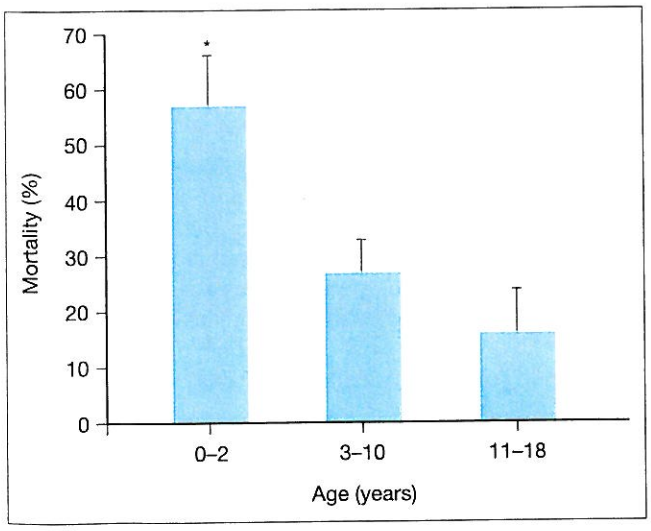

Fig. 36.4 Mortality in burns >80% total body surface area for various ages.

Assessment of resuscitation

Evaluation of the efficacy of resuscitation is difficult in children. The routine clinical signs of hypovolemia for the adult burn patients – low blood pressure and decreased urine output – are late manifestations of shock in the pediatric patient and tachycardia is omnipresent. Children have remarkable cardiopulmonary reserve, often not exhibiting clinical signs of hypovolemia until more than 25% of the circulating volume has been lost and complete cardiovascular decompensation is imminent. Mental status, pulse pressures, arterial blood gases, distal extremity color, capillary refill, and body temperature reflect volume status. Capillary refill is a good indicator of volume status in burned children. Decreased capillary refill should warn a practitioner of imminent danger. A child with normal blood pressure and acceptable heart rate, but with cool clammy extremities, obtundation, and a delayed

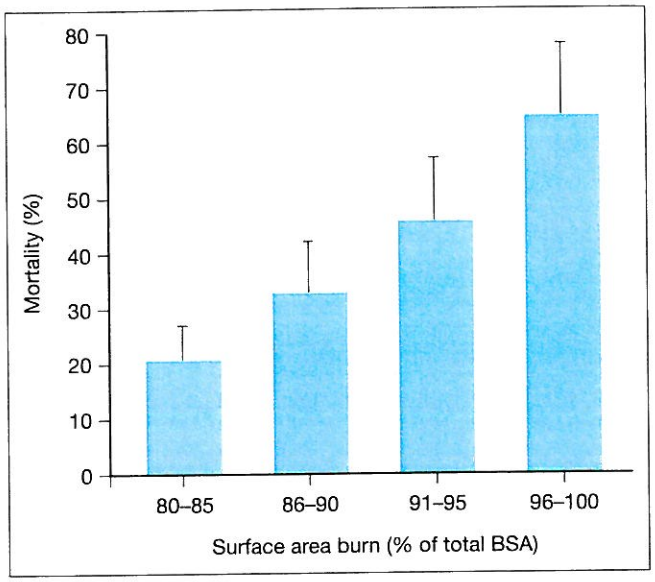

Fig. 36.5 Mortality for increasing burn size.

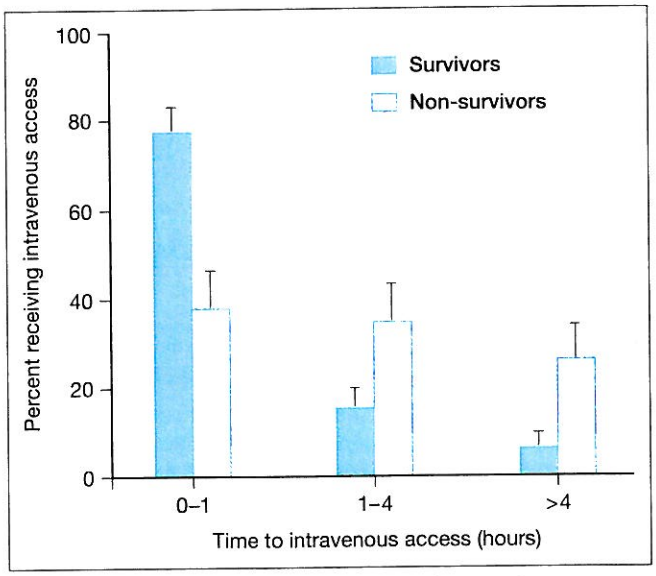

Fig. 36.6 Time to intravenous access in survivors and non-survivors. Mortality increases with delays in starting an intravenous line and instituting volume resuscitation.

TABLE 36.3 NORMAL PEDIATRIC VITAL SIGNS

Age	Minimum heart rate (beats/min)	Systolic BP (mmHg)	Respirations (breaths/min)	Minimal hemoglobin (g/dL)	Minimal hematocrit (%)
<2 years of age	100–160	60	30–40	11.0	33.0
2–5 years of age	80–140	70	20–30	11.0	33.0
6–12 years of age	70–120	80	18–25	11.5	34.5
>12 years of age	60–110	90	16–20	12.0	36.0

capillary refill, is a child in dire danger. Measurement of arterial blood pH with attention to base deficit or lactic acid is of particular importance in this age group, reflecting decreased tissue perfusion.

Children frequently develop a reflex tachycardia after even the most trivial injury due to an overexuberant catecholamine response to the trauma or anxiety. Systolic blood pressures of less than 100 mmHg are common in children younger than 5 years of age (Table 36.3). Young children with immature kidneys have less tubular concentrating ability than adults, and urine production may continue in spite of hypovolemia. Arterial pH, lactic acid, or base deficit can be followed to assist resuscitation.

Volume overload must be avoided. Volume overload can lead to pulmonary edema, right heart failure, abdominal deep muscle compartment syndromes, and cerebral edema in burn patients. Although children possess a large cardiopulmonary reserve, the young heart is less compliant, and stroke volumes plateau at relatively low filling pressures, shifting the Starling curve to the left. Cardiac output is almost completely dependent upon heart rate, and the immature heart is more sensitive to volume and pressure overload. Cardiac output can be measured using a PiCCO monitor, which is less invasive than a Swan–Ganz catheter and only requires an arterial line and a central venous line. Transthoracic or transesophageal echocardiograms should be utilized early to assess the cardiac function in patients who are failing or not responding to the conventional therapy. Children are particularly prone to the development of edema from both vasogenic and hydrostatic sources. Vasogenic edema occurs within the early postburn period when vascular integrity is impaired. Of particular concern is the development of cerebral edema. Care should be taken in order to maintain head of bed elevation, particularly during the initial 24–48 hours postburn and avoid hypercarbia. The maintenance of intravascular osmotic pressures decreases the likelihood of edema development. Salt-poor albumin can be expected to remain in the intravascular space if administered after 8 hours postburn in amounts necessary to maintain serum albumin levels at more than 2.5 g/dL. Albumin deficit can be calculated using the formula:

$$[2.5\,\text{g/dL} - \text{current serum albumin (g/dL)}] \times [\text{wt (kg)} \times 3]$$

The deficit can be administered as 25% albumin and given in three divided doses gradually.

Evaluation and management of airways

The smaller aperture of the pediatric trachea predisposes it to obstruction. Equal amounts of airway edema in pediatric and adult airway results in significantly disproportionate increases in amounts of resistance and decreases in cross-sectional area. A 1 mm increase in tissue thickness of a 4 mm diameter pediatric trachea results in a 16-fold increase in resistance with a 75% decrease in cross-sectional area. The same edema in an adult airway would increase the airway resistance threefold and reduce airway area by 44%.[13] Therefore early intubation is advocated. As edema develops promptly following injury, airway evaluation and management must be given priority in pediatric patients. During emergent conditions when edema is present, inability to secure the airway with an endotracheal tube is a clear indication of the need for surgical airway control.

Potential hemorrhage and edema formation make emergency intubation difficult. Early intubation should be considered when a long transfer is anticipated or a patient has a large burn which likely will develop airway edema with a large amount of fluid resuscitation. Concurrent placement of an endotracheal tube over the bronchoscope should be considered at the time of bronchoscopy. A readily available estimate of airway diameter is the width of the patient's little finger, an age-based formula (age + 16)/4 or the use of Broselow tape.

Following placement of the ET tube, it must be adequately secured. With a child, oozing wounds and moist dressings, this can be a difficult task. One successful approach is to attach the ET tube with tape around the back of the head both above and below the ears. An additional piece of tape over the top of the head, secured to the tape behind the head, will prevent accidental extubation in most children.[14]

Inhalation injury

Inhalation injury, and its sequelae of infection and pulmonary failure, are major determinants of mortality after burn injury.

The mortality rate of children with isolated thermal burns is 1–2%, but it increases to approximately 40% in the presence of in-halation injury.[15,16] Carbon monoxide poisoning coupled with hypoxia is the most frequent cause of death due to 'smoke inhalation'. Any flame-related injury, particularly if it is confined in closed space, should be evaluated for an inhalation component. If inhalation injury is suspected, arterial blood gas and carboxyhemoglobin level should be obtained and the patient placed on 100% oxygen. Children may be spared some of the overt signs of inhalation injury due to their short stature and proximity to the floor-level cool air. As with adults, the only definitive method to diagnose an inhalation injury is through direct visualization of the airway and early examination of arterial carboxyhemoglobin level. Signs of potential inhalation injury include facial burns, singed nasal hairs, carbonaceous sputum, abnormal mental status (agitation or stupor), respiratory distress (dyspnea, wheezing, stridor, hoarseness), or elevated carboxyhemoglobin level >10%.[17] Carboxyhemoglobin levels must be calculated from the time drawn, back to the time of the accident or for O_2 given as described in the chapter on this subject. A carboxyhemoglobin level >60% has more than 50% chance of mortality (Table 36.4).

Common treatment modalities for inhalation injury include airway maintenance, clearance, and pharmacological management (Table 36.5). A recent study has shown that a group of children treated with a regimen of nebulized heparin and acetylcysteine had a significant decrease in reintubation rates, atelectasis, and mortality when compared to a control group.[18]

Hypermetabolism

Children with burn injury demonstrate a remarkable increase in metabolic rate. No other disease state produces as dramatic an effect on the metabolic rate as burn injury.[19,20] This hypermetabolism is thought to slow wound healing and prolong generalized weakness. This prolonged metabolic dysfunction can lead to loss of lean body mass and increase morbidity. Marked wasting of lean body mass occurs within a few weeks of injury.

The hypermetabolic response increases with increasing burn size. There is an upregulation of catabolic agents, such as cortisol, catecholamine, and glucagons,[21] which induces a hyperdynamic cardiovascular response, an increased oxygen consumption, energy expenditure, proteolysis, lipolysis, and glycogenolysis, loss of lean body mass and body weight, delayed wound healing, and immune depression.[22,23]

Pharmacological agents have been used to attenuate catabolism and to stimulate growth in burn injury. To further minimize erosion of lean body mass, administration of anabolic hormones such as growth hormone, insulin, insulin-like growth factor-1 (IGF-1)/IGF-binding protein-3 (IGFBP-3), oxandrolone or testosterone and catecholamine antagonists such as propranolol have been used in pediatric burns. These agents contribute to maintenance of lean body mass and promote wound healing.[24–29]

Thermoregulation

Core body temperature is consistently elevated after a major burn. A hypothalamic reset induced by various inflammatory cytokines and pain causes this elevation even in the absence of infection. The temperature reset is thought to be an adaptive mechanism to bolster host defense against potential pathogens. Burned patients strive for temperatures of about 38°C. Depressed or 'normal' temperatures are more likely indicative of overwhelming sepsis or exhausted physiological capabilities to maintain temperature and should be viewed as an ominous sign. After major thermal injuries, routine methods of heat conservation are inadequate due to the extensive heat loss through convection and evaporation. Infants and toddlers, with their increased surface area/volume ratios, less insulating fat, and lower muscle mass for shivering, are particularly susceptible to hypothermia.

Hypothermia produces numerous consequences. The heart is particularly sensitive to temperature, and ventricular arrhythmias are not uncommon. Hypothermia also increases the susceptibility of the myocardium to changes in electrolyte concentrations. The oxyhemoglobin dissociation curve is shifted to the left by lowered body temperature, impairing peripheral oxygenation. In extreme cases, hypothermia

TABLE 36.4 CARBON MONOXIDE POISONING

Carboxyhemoglobin (%)	Symptoms
0–10	Normal
10–20	Headache, confusion
20–40	Disorientation, fatigue, nausea, visual changes
40–60	Hallucination, combativeness, convulsion, coma, shock state
60–70	Coma, convulsions, weak respiration and pulse
70–80	Decreasing respiration and stopping
80–90	Death in less than 1 hour
90–100	Death within a few minutes

TABLE 36.5 AIRWAY MAINTENANCE, CLEARANCE, AND PHARMACOLOGICAL MANAGEMENT

Turn side to side	q 2h
Sitting or rocked in chair	As soon as physiologically stable
Ambulation	Early
Chest physiotherapy	q 2h
Suctioning and lavage (nasal/oral tracheal)	q 2h
Bronchodilators	q 2h
Aerosolized heparin/acetylcysteine	q 2h alternating
	Heparin 5000–10000 units with 3mL NS q 4h
	Alternated with acetylcysteine 20% 3mL q 4h

produces central nervous system and respiratory depression, coagulopathies, and loss of peripheral vasomotor tone.

Every effort should be made to reduce the heat loss experienced by pediatric patients. Ambient temperatures and humidity should be maintained at 30–33°C and 80%, respectively, in order to decrease energy demands and evaporative water losses. Wet dressings should be avoided or at least wrapped, and wet bedding promptly changed so as to decrease evaporative or conductive cooling. The patient should be positioned so that drafts are avoided, including the inlets and outlets for air-conditioning and heating ducts. Bathing or showering should be expeditiously completed, avoiding undue environmental exposure.

TABLE 36.6 NUTRITIONAL REQUIREMENTS FOR CHILDREN

	Galveston	Modified Curreri
Infant	2100 kcal/m^2 + 1000 kcal/m^2 burn	BMR + 15 kcal/% burn
Toddler		BMR + 25 kcal/% burn
Child	1800 kcal/m^2 + 1300 kcal/m^2 burn	BMR + 40 kcal/% burn
Adolescent	1500 kcal/m^2 + 1500 kcal/m^2 burn	

Nutritional support

Nutritional support of the hypermetabolic response in severely burned patients is best accomplished by early enteral nutrition. Early institution of enteral feeding can abate the hypermetabolic response to burn.[30,31] Patients with smaller burns are immediately placed on a high-protein, high-caloric diet to support their metabolic response. Those with larger burns (>30%) are placed on enteral feedings.

Almost immediate enteral nutrition can be initiated via a transpyloric feeding tube. Most children will tolerate enteral feedings as early as 1–2 hours postburn, if not immediately. Several studies have demonstrated the efficacy of early alimentation and the additional salutary effects.[32,33] Enteral feedings can be given through a flexible Silastic duodenal feeding tube, bypassing the stomach, which may be experiencing decreased peristaltic action.

Early enteral feeding preserves gut mucosal integrity and improves intestinal blood flow and motility.[30]

Milk has been demonstrated to be one of the least expensive and best tolerated of all enteral formulas. Additionally, it is palatable and easily recognized by children when they are able to take oral feedings. Because of the low sodium content of milk, sodium supplementation may be necessary. Hyperosmolar feedings, commercially available, should be diluted because of the high incidence of subsequent diarrhea if used full strength. It is diluted to 1/2 or 3/4 strength. Diarrhea is particularly troublesome in children because of their increased sensitivity to volume deficits.

Several formulas are available to estimate caloric requirement in burn patients. Since caloric demands are related to burn size, caloric support should be given in amounts calculated based on body surface area. A series of different formulas based on body surface area have been developed at Shriners Hospitals for Children, Galveston to meet the differing requirements of the various age groups.[34–36] The Curreri formula has likewise been amended to reflect the differing demands of the pediatric group (Table 36.6).

Growth

Hypermetabolism and muscle protein catabolism persist long after the wound is closed.[37] Protein breakdown continues 6–9 months after severe burn. There is almost a complete lack of bone growth for 2 years after injury. This results in long-term osteopenia, which may adversely affect peak bone mass accumulation in children.[38,39] Severely burned children with a burn size of >80% have a linear growth delay for years after injury.[40] In severely burned patients, nail and hair growth are attenuated during the acute postburn period, and bone growth is slowed.[41–43] Dampened height and weight gain velocities have been documented in children during the first 3 years postburn, thereby rendering these burned children slighter and shorter than their age-matched peers.[44]

Nutritional support of acute burn patients becomes an essential part of treatment during their hospitalization.

Wound closure

One of the more important advances in the last 20 years has been the development of early excision and early wound closure. Improvements in the treatment of burn wounds and utilization of antimicrobial dressings have dramatically decreased the incidence of fatal sepsis in burned patients.[45] Two decades ago third-degree burns were treated by removing small amounts of eschar at a time, approximately 10–15%, followed by grafting. Commonly, eschar was allowed to separate with lysis by bacterial enzymes. This led to a high incidence of invasive infection and wound sepsis and prolonged length of hospital stay with increased mortality. Today massive excision can be easily managed in children, providing results of decreased mortality and decreased length of hospital stay.[46,47] Early excision even in the first 24 hours is safe and effective.[48] Performing early excision within the first 48 hours can significantly reduce blood loss.[49] By using skin substitutes such as allograft skin, xenograft, and Integra (Figure 36.7), the burn wound can be covered and protected for many weeks until enough donor site is available for grafting (repeat autografting is performed when the donor site is healed for reharvesting). Cultured epidermal autografts (CEA) is available for massive burn injuries (Figure 36.8). Although it is an effective way to cover large burns with limited donor sites, it may not be the most cost-effective approach. A group of patients treated with CEA had greater hospital costs, a longer length of stay, and more reconstructive admissions than conventional treatment with meshed autograft skin.[46] Cultured skin substitute (CSS) is currently undergoing investigative study and holds promise. CSS, consisting of autologous cultured keratinocytes and fibroblasts attached to collagen-based sponges, may reduce the requirement for donor skin and number of autograftings in massive burns.

Scald burns in young children are best managed with delayed treatment. Unless the wound is clearly third degree, the scald injury should be conservatively managed for approximately 2 weeks to allow the wound to heal or demarcate (Figures

Fig. 36.7 Dermal and skin substitutes can be used as temporary cover for severe burns. Integra, a bilaminar skin substitute, can replace homografts as temporary cover. The Silastic superficial layer can be removed after 3 weeks and a super-then autograft then placed on top. The entire wound can be covered with Integra, which is subsequently autografted when donor sites are available. (Reprinted with permission from Barret J, Herndon DN, eds. Color atlas of burn care. London: WB Saunders; 2001: 107, Plate 6.95.)

Fig. 36.9 Superficial and small areas of deep second-degree scald burns before topical treatment: 25% total body surface area. (Reprinted with permission from Barret J, Herndon DN, eds. Color atlas of burn care. London: WB Saunders; 2001: 79, Plate 5.39.)

Fig. 36.8 The cultured epidermal autografts are ready to use 18–21 days later. Extreme care with handling is needed because of the fragility of the cultured cells. (Reprinted with permission from Barret J, Herndon DN, eds. Color atlas of burn care. London: WB Saunders; 2001: 106, Plate 6.89.)

Fig. 36.10 Deep second-degree burns treated for 10 days with silver sulfadiazine. Note that the edges are regenerating. Pseudo-eschar challenges the evaluation of the wounds. Foul smell, discoloration, surrounding cellulitis, and eschar separation are signs of infection. (Reprinted with permission from Barret J, Herndon DN, eds. Color atlas of burn care. London: WB Saunders; 2001: 77, Plate 5.31.)

36.9 and 36.10). This conservative treatment results in less wound excised, less blood loss, and less apparent scarring.[50] Treatment methods for the conservative care of scald burns were analyzed. Scald injuries greater the 20% TBSA (mean 31%) were randomized to treatment with allograft skin versus topical antimicrobial therapy. Treatment with allograft led to decreased time to healing and decreased pain.[51] In another study, patients greater than 40% TBSA burn (mean 65%) were randomized to allograft skin or topical antimicrobial. Patients who received treatment with allograft skin had a significantly decreased length of stay.[52] The current recommended treatment for second-degree burns, primarily scalds less than 30% TBSA, is immediate application of Biobrane. Biobrane can be safely used in children, even in infants less than 2 years of age. When applied within 48 hours of injury there is no difference in infection between Biobrane and topical antimicrobials. Further, application of Biobrane versus topical antimicrobials leads to decreased pain, decreased length of hospitalization, and decreased healing time.[53,54]

Pain management

Children do not always express their pain in the same way as adults.[55] Children may display pain through behaviors of fear, anxiety, agitation, anger, aggression, tantrums, depression, withdrawal, and regression.[56,57] How the child's experience of pain from the burn injury and anxiety from the hospitalization are clinically managed will have lasting psychological effects for many months and years to follow. A severe burn injury brings many weeks of surgeries, dressing changes, and

exercises that can cause intense pain. Pain can also exist as a constant state throughout the hospitalization. Morphine sulfate is the most commonly used analgesia. It should be given intravenously, and not intramuscularly. Fentanyl can be also used. Fentanyl Oralet can be used successfully for dressing changes (10 μg per kg). Most outpatients are treated with hydrocodone/acetaminophen.

Rehabilitation

Rehabilitation is a key component to success of burn treatment and starts at the time of the admission. During the acute phase of burn care, splints are used to minimize joint deformities and contractures. Splints are used continuously except during a therapy session. They are fabricated to each patient's needs and used from day 1 of hospitalization. Bedside therapy, including passive and active range of motion, is started early. Early mobilization should be practiced. Early ambulation after the grafts has taken and early physical and occupational therapies are important to success of rehabilitation of burned children. Patients with leg grafting are kept bedrest after the operation, but on postoperative day 4, they are gotten out of bed and started on ambulation. Early therapy and ambulation are keys to success of long-term rehabilitation of burned children. When patients are discharged from their acute care hospitalization, they undergo rigorous therapies, including stretching, and range of motion and strengthening exercises.

In general, deep partial-thickness burn wounds requiring 3 or more weeks to heal will likely produce hypertrophic scars. The longer it takes to heal, the more likely are scars. Using dressings that exert constant pressure on the healing wound and pressure garments exerting constant pressure on the healed wound are the most effective ways of decreasing the incidence of hypertrophic scar formation. Pressure garments may be worn up to 2 years or until scars mature.

Prevention

Prevention remains the single best way to manage pediatric burn injuries. National prevention and education efforts have positively impacted the number of pediatric burns each year. Lowering the temperature set point on hot water heaters and teaching families to check the bath water temperature before placing a child in the bath has decreased hot water scald injuries. Prevention groups have worked with gas hot water heater companies and the Consumer Product Safety Commission (CPSC) to provide education to raise gas water heaters 12 inches off the ground, which significantly reduces the risk of accidental explosions and fires.[58]

Much work still needs to be done in the area of 'child fire play'. Three-fourths of 'child fire play' involves matches or lighters. All matches and other ignition sources must be placed out of reach of children. A positive step toward prevention occurred in 1994 when CPSC placed into effect a child-resistant lighter to protect children under 5 years of age. The importance of placing smoke detectors in multiple areas in a house has received much public education over the last several years. Current prevention education focuses on children, and especially infants that are not able to remove themselves from a fire. Since the CPSC has reduced flammability standards on children's sleepwear in 1997, there has been an increased incidence of sleepwear-related burn injuries in children.[59] One way to protect infants and children is to dress them in fire-resistant sleepwear and clothing to protect them from a burn injury if a fire does occur.

Children have no idea of the dangerous situations they place themselves in. Educating children as early as possible that fire is dangerous is imperative. Providing safe environments for our children and providing appropriate education to them is the responsibility of healthcare providers, the adults that care for them, and the community at large.

References

1. National Vital Statistics System. Deaths. Final data for 1997. Centers for Disease Control and Prevention 1999; 47(19):1–105.
2. National Center for Health Statistics. Hospitalizations for injury, United States 1996. Centers for Disease Control and Prevention 1996; (318):1–10.
3. Brigham PA, McLoughlin E. Burn incidence and medical care in the United States: estimates, trends, and data sources. J Burn Care Rehabil 1996; 17:95–107.
4. Pruitt BA, Goodwin CW, Mason AD Jr. Epidemiologic, demographic, and outcome characteristics of burn injury. In: Herndon DN, ed. Total burn care. London: WB Saunders; 2002:16–30.
5. Barillo DJ, Goode R. Fire fatality study: demographics of fire victims. Burns 1996; 22:85–88.
6. Bull JP, Fisher AJ. A study in mortality in a burn unit: standards for the evaluation for alternative methods of treatment. Ann Surg 1949; 130:160–173.
7. Herndon DN, Gore DC, Cole M, et al. Determinants of mortality in pediatric patients with greater than 70% full thickness total body surface area treated by early excision and grafting. J Trauma 1987; 27:208–212.
8. Wolf SE, Rose JK, Desai MH, et al. Mortality determinants in massive pediatric burns: an analysis of 103 children with 80% TBSA burns (70% full thickness). Ann Surg 1997; 225(5):554–569.
9. Tocantins LM, O'Neill JF. Infusions of blood and other fluids into the general circulation via the bone marrow. Surg Gynecol Obstet 1941; 73:281–287.
10. Fiser DH. Intraosseous infusion. N Engl J Med 1990; 322:1579–1581.
11. Caravajal HF. A physiologic approach to fluid therapy in severely burned children. Surg Gynecol Obstet 1980; 150:379–384.
12. Kruesser W, Ritz E. The phosphate depletion syndrome. Contrib Nephrol 1978; 14:162–166.
13. Ryan JF, Todres ID, Cotes CJ, et al., eds. A practice of anesthesia for infants and children. New York: Grune & Stratton; 1986:39.
14. Mlcak R, Cortiella J, Desai MH, et al. Emergency management of pediatric burn victims. Pediatr Emerg Care 1998; 14(1):51–54.
15. Herndon DN, Thompson PB, Traber DL. Pulmonary injury in burned patients. Crit Care Clin 1985; 1:79–96.
16. Thompson PB, Herndon DN, Traber DL, et al. Effect on mortality of inhalation injury. J Trauma 1986; 26:163–165.
17. Bennett JDC, Milner SM, Gheranrdini G, et al. Burn inhalation injury. Emerg Med 1997; 12(17):22–24, 31, 32.
18. Desai MH, Mlcak R, Richardson J, et al. Reduction in mortality in pediatric patients with inhalation injury with aerosolized heparin/N-acetylcysteine therapy. J Burn Care Rehabil 1998; 19(3):210–212.

19. Yarborough MF, Herndon DN, Curreri PW. Nutritional management of the severely injured patient: (1) thermal injury. Contemp Surg 1978; 13:15–20.
20. Wilmore DW. Nutrition and metabolism following thermal injury. Clin Plast Surg 1974; 1:603–619.
21. Fleming RYD, Rutan RL, Jahoor F, et al. Effects of recombinant human growth hormone on catabolic hormones and free fatty acids following thermal injury. J Trauma 1992; 32:698–702.
22. Lee JO, Herndon DN. Modulation of the post-burn hypermetabolic state. In: Cynober L, Moore FA, eds. Nutrition and critical care. Switzerland: Nestec; 2003(8):39–56.
23. Herndon DN. Mediators of metabolism. J Trauma 1981; 21:701–705.
24. Pierre E, Barrow R, Hawkins H, et al. Effects of insulin on wound healing. J Trauma 1998; 44:342–345.
25. Wolf SE, Barrow RE, Herndon DN. Growth hormone and IGF-I therapy in the hypercatabolic patient. Baillières Clin Endocrinol Metabol 1996; 10:447–463.
26. Herndon DN, Ramzy PI, DebRoy MA, et al. Muscle protein catabolism after severe burn: effect of IGF-1/IGFBP-3 treatment. Ann Surg 1999; 229:713–720.
27. Hart DW, Wolf SE, Ramzy PI, et al. Anabolic effects of oxandrolone after severe burn. Ann Surg 2001; 233:556–564.
28. Ferrando AA, Sheffield-Moore M, Wolf SE, et al. Testosterone administration in severe burns ameliorates muscle catabolism. Crit Care Med 2001; 29:1936–1942.
29. Herndon DN, Hart DW, Wolf SE, et al. Reversal of catabolism by beta-blockade after severe burns. N Engl J Med 2001; 345:1223–1229.
30. Mochizuki H, Trocki O, Dominioni L, et al. Mechanism of prevention of postburn hypermetabolism and catabolism by early enteral feeding. Ann Surg 1984; 200:297–310.
31. Dominioini L, Trocki O, Fang CH, et al. Enteral feeding in burn hypermetabolism: nutritional and metabolic effects at different levels of calorie and protein intake. JPEN J Parenter Enteral Nutr 1985; 9(3):269–279.
32. Enzi G, Casadei A, Sergi G, et al. Metabolic and hormonal effects of early nutritional supplementation after surgery in burn patients. Crit Care Med 1990; 18:719–721.
33. McDonald WS, Sharp CW, Deitch EA. Immediate enteral feeding in burn patients is safe and effective. Ann Surg 1991; 213:177–183.
34. Hildreth MA, Herndon DN, Desai MH, et al. Caloric requirements of patients with burns under 1 year of age. J Burn Care Rehabil 1993; 14(1):108–112.
35. Hildreth MA, Herndon DN, Desai MH, et al. Caloric needs of adolescent patients with burns. J Burn Care Rehabil 1989; 10(6):523–526.
36. Hildreth MA, Herndon DN, Desai MH, et al. Current treatment reduces calories required to maintain weight in pediatric patients with burns. J Burn Care Rehabil 1990; 11(5):405–409.
37. Hart DW, Wolf SE, Mlcak RP, et al. Persistence of muscle catabolism after severe burn. Surgery 2000; 128:312–319.
38. Klein GL, Herndon DN, Langman CB, et al. Long-term reduction in bone mass after severe burn injury in children. J Pediatr 1995; 126:252–256.
39. Klein GL, Wolf SE, Goodman WG, et al. The management of acute bone loss in severe catabolism due to burn injury. Horm Res 1997; 48:83–87.
40. Rutan RL, Herndon DN. Growth delay in postburn pediatric patients. Arch Surg 1990; 125:392–395.
41. Artz CP, Moncrief JA. Treatment of burns, 3rd edn. Philadelphia: WB Saunders; 1974.
42. Artz LP, Stern PJ, Wurick JD. Skeletal changes after burn injuries in an animal model. J Burn Care Rehabil 1988; 9:148–151.
43. Klein GL, Herndon DN, Rutan TC, et al. Bone disease in burn patients. J Bone Miner Res 1993; 8(3):337–345.
44. Rutan RL, Herndon DN. Growth delay in postburn pediatric patients. Arch Surg 1990; 125:392–395.
45. Merrell SW, Saffle JR, Larson CM, et al. The declining incidence of fatal sepsis following thermal injury. J Trauma 1989; 29:1362–1366.
46. Herndon DN, Parks DH. Comparison of serial debridement and autografting and early massive excision with cadaver skin overlay in the treatment of large burns in children. J Trauma 1986; 26(2):149–152.
47. Thompson P, Herndon DN, Abston S, et al. Effect of early excision on patients with major thermal injury. J Trauma 1987; 27(2):205–207.
48. Barret JP, Wolf SE, Desai M, et al. Total burn wound excision of massive paediatric burns in the first 24 hours post-injury. Ann Burns Fire Disasters 1999; XIII(1):25–27.
49. Herndon DN, Barrow RE, Rutan RL, et al. A comparison of conservative versus early excision therapies in severely burned patients. Ann Surg 1989; 209(5):547–553.
50. Desai MH, Rutan RL, Herndon DN. Conservative treatment of scald burns is superior to early excision. J Burn Care Rehabil 1991; 12:482–484.
51. Rose JK, Desai MH, Mlakar JM, et al. Allograft is superior to topical antimicrobial therapy in the treatment of partial-thickness scald burns in children. J Burn Care Rehabil 1997; 18(4):338–341.
52. Lal SO, Barrow RE, Heggers JP, et al. Biobrane, a synthetic skin substitute, improves wound healing without increased risk of infection. Surgical Infection Society, April 2000 (Abstract 33).
53. Barret JP, Dziewulski P, Ramzy PI, et al. Biobrane versus 1% silver sulfadiazine in second-degree pediatric burns. Plast Reconstr Surg 2000; 105(1):62–65.
54. Naoum JJ. The use of homograft compared to topical antimicrobial therapy in the treatment of second-degree burns of more than 40% total body surface area. American College of Surgeons Committee on Trauma, October 2000 (Abstract).
55. Schecter N, Allen DA, Hanson K. Status of pediatric pain control: a comparison of hospital analgesic usage in children and adults. Pediatrics 1986; 77:11–15.
56. Stevens B, Hunsberger M, Browne G. Pain in children: theoretical research and practice dilemmas. J Pediatr Nurs 1987; 2:154–166.
57. Staddard FJ. Coping with pain: a developmental approach to treatment of burned children. Am J Psychiatry 1982; 139:736–740.
58. Benjamin D, Herndon D. Successful prevention programs for gas hot water heater burn injuries. J Burn Care Rehabil 2000; 21(1 part 2):S152.
59. Benjamin DA, Tompkins RG, Warden GD, et al. Increasing incidence in sleepwear related burns. J Burn Care Rehabil 2001; 21(2): S67.
60. Fleisher G, Ludwig S, eds. Textbook of pediatric emergency medicine, 2nd edn. Baltimore: Williams & Wilkins; 1988:268.
61. Eichelberger MR, ed. Pediatric trauma: prevention, acute care and rehabilitation. St Louis: Mosby Year Book; 1993:572.