The treatment goals are as follows:

- Provide appropriate resuscitation.
- Preserve physical function.
- Promote wound healing and optimal cosmetic results.
- Minimize pain and anxiety.
- Provide adequate nutrition.
- Prevent or aggressively treat complications.

The clinical management of a severely burned patient is complex and frequently challenging. To get a general overview of the important issues, we recommend that clinicians read a recent publication in the "Case Records of the Massachusetts General Hospital" section of the New England Journal of Medicine. The photographs and chronology of patient care provided by the authors provide an excellent presentation of patient management issues, treatment strategies employed in their resolution, and the long-term outcomes of a severely burned patient. Similarly, two recently published articles are of exceptional educational value in their description of burn patient management and the more global issues of public safety and the health care system response to disasters.

The skin is the largest organ of the body, and it performs five major functions. It provides protection from the environment, sensory perception, vitamin production, excretion of water and some wastes, and regulation of body temperature. When the skin is damaged, bacteria are no longer prevented from invading, pain is produced (unless superficial sensory nerves are destroyed), and both fluid and heat are lost through the damaged area.

Extensive skin loss or damage requiring hospitalization can occur by many different mechanisms that produce similar effects, including thermal injury from hot liquids (scalds), flames, or extreme cold (frostbite), and injury from chemicals, radiation (sunburn), electricity, or trauma (abrasion). In addition, patients with extensive exfoliative dermatoses (e.g., Stevens-Johnson syndrome or toxic epidermal necrolysis) are often treated in burn centers. Because the presence of devitalized tissue increases the risk of tetanus, the patient's tetanus immunization status must be ascertained. In an audit of 269 trauma patients, 70 of whom had been burned, 15% of patients were not questioned about their tetanus status in the emergency room and 27% were incorrectly assessed. Treatment with tetanus and diphtheria toxoids and tetanus immune globulin should be initiated according to promulgated guidelines. In contrast to the rarity of tetanus, burn patients are at risk for the common adverse sequela associated with intensive care unit admission, such as venous thromboembolism and sinusitis.

Fire victims can suffer severe injury or death without significant body surface burning. A classic demonstration of this was the 1942 Coconut Grove Nightclub fire, in which 75 of the 114 deaths were caused by smoke inhalation. Carbon monoxide (CO) poisoning is a frequent cause of death. CO binds preferentially to hemoglobin, displacing oxygen and shifting the oxyhemoglobin dissociation curve to the left, resulting in tissue hypoxia. The smoke, which is a function of the burning material, contains toxins other than CO, such as cyanide, acrolein, benzene, and phosgene.
Outcome following thermal injury is determined by a combination of patient and burn factors. The very young, the very old, and those who were previously ill have a poorer prognosis than do healthy, young adults after a similar injury. Scoring systems for injury severity include the Trauma Score, Injury Severity Score, and the Burn Specific Health Scale, which are used to accurately assess the impact of nonfatal burn injury.\(^5^1\) One method for estimating the probability of death after burn injury found that the three risk factors with the strongest predictive value for death were age greater than 60 years, extent of burn, and level of inhalation injury.\(^6^2\) The pathophysiology of inhalation injury is complex and incompletely understood; however, its impact on patient outcome is considerable, accounting for up to 77% of deaths in burn patients who also have inhalation injury.\(^6^3\) Fiberoptic bronchoscopy is the preferred diagnostic strategy, although enthusiasm is growing among burn care clinicians for virtual bronchoscopy, because it is noninvasive and easy to perform in unstable patients.\(^6^4\) Treatment of inhalation injury is mainly supportive (i.e., endotracheal intubation and mechanical ventilation).\(^6^5\) High-frequency percussive ventilation is an innovative strategy that has been shown to benefit patients who do not respond to conventional ventilation.\(^6^6\)

Burn factors that determine patient outcome include depth, extent, and body surface location.\(^6^7\) A list of burn severity criteria is provided in Table 11.1. The important distinction regarding depth of burn is that partial-thickness injuries heal by cell regeneration, but full-thickness injuries, unless very small, require skin grafting. Small full-thickness burns heal by contraction and reepithelialization from progenitor cells at the edges of the wound.

### WOUND ASSESSMENT

Traditionally, the depth of burns has been described according to degrees of injury, as listed in Figure 11.1. As the depth of injury increases, the number representing degree of injury increases. A first-degree burn is very shallow and affects only the epidermis. A second-degree burn involves complete destruction of the epidermis and variable portions of the underlying dermis. When destruction to the dermis is limited to the upper third or less, the burn is called a superficial second-degree burn. Conversely, a deep second-degree burn appears as tissue destruction below the top one third, but not completely through the dermis. A third-degree, or full-thickness, burn reveals destruction of the entire epidermis and dermis. The terms fourth-degree and fifth-degree burn have been used to describe tissue destruction through subcutaneous fat and through muscle, respectively.\(^6^8\)

The typical first-degree burn is easily identified. It is painful and erythematous, and it blanches to pressure. A superficial second-degree burn is painful, forms blisters, and blanches to pressure. A third-degree burn is usually not painful because superficial nerve endings are destroyed; may appear white, leatherlike, or black (charred); and contains thrombosed blood vessels. This dead tissue is called eschar (es’kar). Unfortunately, sometimes even the most experienced clinician cannot differentiate a partial-thickness from a full-thickness injury. In addition, flame injuries typically occur as a mixture of full- and partial-thickness injuries. This classic presentation, as depicted in Figure 11.2, was described as a target or bulls-eye, where the deepest injury is at the center, followed by increasingly superficial injury at increasing distance from the center. Early attempts to improve the accuracy of injury depth assessment included histologic staining, injection of radioactive compounds or dyes such as bromphenol blue, and fiberoptic perfusion fluorometry. These methods were disadvantageous in that they were invasive, cumbersome, labor intensive, and inaccurate. Although it is sensitive to variations in positioning and temperature, the laser Doppler has better than 90% accuracy when compared with histologic analysis of burn wound depth. The laser Doppler documents a reduction in red blood cell velocity in a burn wound, establishing the necessity for surgical

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**TABLE 11.1 Burn Injury Severity Classification**

<table>
<thead>
<tr>
<th>Depth of Burn</th>
<th>Minor Injury</th>
<th>Moderate Injury</th>
<th>Major Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adult</td>
<td>Adult</td>
<td>Adult</td>
</tr>
<tr>
<td></td>
<td>Child</td>
<td>Child</td>
<td>Child</td>
</tr>
<tr>
<td>Partial Thickness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First degree</td>
<td>&lt;0.10</td>
<td>0.50-1.00</td>
<td>&gt;2.00</td>
</tr>
<tr>
<td>Second degree</td>
<td>&lt;0.15</td>
<td>0.10-0.50</td>
<td>0.50-1.00</td>
</tr>
<tr>
<td>Full Thickness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third degree</td>
<td>&lt;0.10</td>
<td>0.20-0.30</td>
<td>&gt;0.30</td>
</tr>
</tbody>
</table>

\(^a\) Irrespective of burn extent, injuries are classified as major when they involve areas of special importance, such as the eyes, ears, hands, feet, or genitals. Injuries are major when burns occur in conjunction with other major trauma (e.g., fractures) or inhalation injury.

\(^b\) TBSA, total body surface area.
removal and grafting. Clinicians must remain cognizant of the need for confirmation of initial assessments of burn depth. Reassessment is necessary because of the changing nature of deep partial-thickness injuries, which may become full-thickness wounds because of infection or inadequate resuscitation.

Several systems are used to calculate the relative percentage of total body surface area (TBSA) burned. The rule of nines is a simple system that can be used to estimate the extent of burn in adults. It represents regions of the body surface as 9% or multiples of 9%, for example, the head and arms each represent approximately 9% TBSA, the torso 36%, each leg 18%, and the perineum 1%. For burns with an uneven distribution, the patient’s hand is a useful measuring tool. One side of the patient’s complete hand (i.e., the palm and fingers) is about 1% TBSA. In infants and children, the head represents a larger TBSA than that of adults; therefore, the rule of nines does not hold. A more accurate assessment of TBSA can be made with the Lund-Browder chart. The chart used at the University of Michigan Burn Center is reproduced in Figure 11.3. Favorable assessments of two computerized systems for estimating burn size and calculating fluid requirements [i.e., the Sage II (available at http://www.sagediagram.com) and the 3-D Burn Vision (CD-ROM from Electric Power Research Institute, Concord, CA, 1-800-313-3774)] have been reported.

**WOUND CLOSURE**

Although some clinicians advocate the use of topical creams (i.e., papain-urea or the less painful collagenase) to enzymatically débride full-thickness injuries, the preferred surgical approach over the past decade has been staged eschar excision (débridement) with placement of autologous skin grafts. Split-thickness skin grafts (STSGs) that are approximately 0.06 mm thick are harvested (with the use of a dermatome), expanded by a ratio of 1:1.5 (with the use of a meshing device), and applied to the wound after removal of devitalized tissue and achievement of hemostasis. An important detrimental problem during these operative procedures is coagulopathy, which is produced by hypothermia. To prevent or attenuate hypothermia, the ambient temperature of the operating room is maintained above 37°C, the amount of débridement is limited, and intravenous fluids are warmed to 40°C. Although it may appear hazardous, a pilot
FIGURE 11.3 The University of Michigan Hospital Burn Center's estimation of size of burn by percentage.
study in eight patients demonstrated that intraoperative intravenous fluids could safely be heated to 60°C when administered through a central venous catheter.29

Once adherent and vascularized, the STSG from the patient’s own noninjured skin (autograft or isograft) permanently closes the wound.30 Unfortunately, the amount of noninjured skin available for STSGs often cannot completely cover the open wound. A number of skin substitutes can be used to temporarily cover the open wound while donor site healing and reharvesting of the autograft are awaited. A commonly used biologic skin substitute is cadaver skin (allograft or homograft). Less frequently used biologic skin substitutes include animal skin (xenograft or heterograft), amniotic membrane, and tissue-derived collagen. Synthetic skin substitutes include polyurethane film (e.g., Opsite) and petrolatum-impregnated fine mesh gauze (e.g., scarlet red and Xeroform). Biosynthetic skin substitutes have been developed that are combinations of biologic and synthetic materials, such as Biobrane and TransCyte, which combine collagen with a synthetic membrane.31,32 Skin substitutes adhere to the wound, minimize pain, decrease protein, water, and electrolyte loss, and simulate many important skin functions, such as providing a barrier to bacteria. Although these skin substitutes perform important functions, they eventually must be replaced by autologous skin.

Advances in tissue bioengineering have focused on the development of a true dermal replacement or substitute. One approach is the in vitro production of new skin by culture of autologous epidermis. Since the first successful transplantation of cultured autologous epidermis in 1981, keratinocyte growth techniques have been so improved that current methods allow a several thousand-fold expansion of skin specimens within 3 to 4 weeks.33 However, engraftment of cultured epidermis continues to be inconsistent and should be considered experimental.34 Another strategy provides a dermal matrix attached to a silicon polymer membrane.35 Finally, the availability of a “living skin equivalent,” such as Integra, Apligraf, and Alloderm, is an exciting, albeit costly, strategy for wound healing.32,36,37

Healing of burn wounds often results in scars that impair normal range of motion and are cosmetically unpleasant. Hypertrophic scars are associated with contractures, and they are raised, erythematous, and pruritic. Keloid scars extend beyond the original wound and rarely regress. Recently developed therapeutic strategies include the use of pressure, massage, physical therapy, silicone sheets and gels, glucocorticoids, and surgical procedures.38,39 Another troublesome problem associated with a healing burn wound is pruritus. Attempts to control burn wound itch with the use of moisturizers and antihistamines have been disappointing. Increased success has been achieved by combining the histamine H₁ and H₂ receptor antagonists (H₁ and H₂RAs) cetirizine and cimetidine, through the use of a topical doxepin cream, with adjunct therapy that includes colloidal oatmeal baths, massage therapy, or analgesics.40–43

PAIN MANAGEMENT

Effective pain management in burn patients requires an understanding of physiologic responses to injury and the inter-relationships between anxiety, depression, and pain. The extent of burn is a significant predictor of pain but only during the first week after injury. Pain varies greatly from patient to patient and undergoes wide fluctuations over time in each patient; the greatest pain is usually experienced during therapeutic procedures such as hydrotherapy with wound débridement and dressing changes.44 Patients with high levels of anxiety or depression tend to report greater pain when at rest.

Historically, pain management practices for burn patients have not been optimal; some centers report that no analgesics or psychotropics were administered to children during wound débridement.45 To the observant clinician, careful evaluation of signs such as heart rate, blood pressure, facial expression, and body movement and position, as well as the quality of an infant’s cries, is sufficient to allow evaluation of pain intensity and to guide the need for and administration of analgesics. In addition, maintaining plasma analgesic concentrations within the range established for good analgesia may be beneficial in centers with rapid access to drug analysis laboratories.46 Currently, a variety of pharmacotherapeutic agents are used in pain management, such as fentanyl, ketamine, propofol, and nitrous oxide. Indeed, a recent study applied the principle of patient-controlled opioid analgesia to the use of propofol for sedation during painful dressing changes.47

The amount of opioid necessary to achieve pain control in burn patients can be substantial, with reports of morphine sulfate self-administered at rates of 108 mg per hour.48 This demonstrates that large doses of morphine sulfate can be administered safely without undue fear of hypoventilation—indeed a dose of 1,650 mg per hour has been reported.49 Another important contribution to the enhanced usage of opioid analgesics is the allaying of unrealistic fears of narcotic addiction in hospitalized patients. The Boston Collaborative Drug Surveillance Program reported only four cases of reasonably well-documented addiction out of 11,882 patients who had received at least one narcotic preparation.50

Rather than conventional analgesic therapy, such as intermittent intravenous morphine injection, patient-controlled analgesia for both adult and pediatric burn patients is gaining popularity.51,52 However, because of situations such as the need for neuromuscular blockade, many patients with acute burns are not suitable candidates for patient-controlled analgesia.53 Another unconventional analgesic strategy that avoids opioid respiratory depression is the use of lidocaine as a continuous intravenous infusion.54

Nonpharmacologic methods or adjuncts to pain management include hypnotherapeutic intervention; distraction therapy, in which video programs of scenic beauty accompanied
by music are used in combination with analgesics; and cognitive-behavioral therapies such as explanation, personal control, altering the meaning of pain, relaxation, imagery, distraction, and self-hypnosis.  

**FLUID RESUSCITATION**

Damaged skin loses the ability to serve as a barrier to percutaneous water loss. Evaporative water loss can be substantial. In contrast to the normal vapor pressure of approximately 3 mm Hg, the vapor pressure of full-thickness burns is about 30 mm Hg. The amount of water loss in milliliters per hour can be estimated with the following formula:

Evaporative loss (mL/h) = (25 + TBSA % burn) × BSA

where BSA is body surface area in square meters and TBSA is total body surface area. In addition, injury to capillaries in the burn wound causes them to leak a protein-rich fluid into the interstitial space, producing edema and blisters. Blood vessels are generally thought of as solid-walled tubes like plumbing, when in fact, they are made up of individual cells. When injured or under the influence of cytokines or inflammatory mediators, these cells swell apart and produce small “holes” in the vessel wall. The problem resolves within 24 hours, but until then, large macromolecules (molecular weights up to 80,000) can leak out of the intravascular space. When the TBSA burned exceeds 25%, a generalized “capillary leak” is produced throughout the body, and fluid exudes from unburned vessels into tissue and organs. The exact pathophysiology of this phenomenon is not clear, but the effects of leukotrienes, prostaglandins, arachidonic acid, and oxygen-derived free radicals have been implicated.

**FLUID REQUIREMENTS**

Treatment or resuscitation of the burn patient who is in shock has been the subject of much interest, research, and controversy. Focused interest was generated in the 1940s because hypovolemic shock was the leading cause of death in burn patients who survived their initial injury. The goal of initial fluid resuscitation is to restore and maintain tissue perfusion while minimizing edema formation. The success of fluid administration is judged primarily by urine production at a rate of 0.5 to 1.0 mL/kg/h. In addition, clinical observation of the adequately resuscitated patient should reveal a pulse rate less than 120 (adults) and a clear sensorium. Use of a physiologic salt solution (crystalloid) such as Lactated Ringer’s is recommended. A number of resuscitation strategies and adjunctive agents have been investigated with varying results; however, one intriguing report on 37 patients demonstrated a statistically significant reduction in resuscitation fluid volume when ascorbic acid was administered as a continuous intravenous infusion during the initial 24-hour period at a dose of 66 mg/kg/h. 

The primary controversy regarding fluid resuscitation of the burn victim involves the necessity of colloid infusion. Colloid is a general descriptive term for nondiffusible, large-molecular-weight molecules that affect osmotic pressure. Available colloid suspensions include fresh frozen plasma, plasma protein fraction, albumin, dextrans, hetastarch, and pentastarch. Clinicians who routinely use colloids suggest that they are physiologic and can reduce nonburned tissue edema. Crystalloid proponents caution that administered colloids can escape from the intravascular space until the capillary leak is sealed. Although no definitive answer is available, it seems reasonable to exclude colloid infusion from resuscitation fluids for the first 12 hours. Representative resuscitation guidelines are listed in Table 11.2.

A second controversy regarding administration of colloid concerns the use of supplemental albumin to prevent or treat hypoalbuminemia in the postresuscitation phase. In two similar studies of pediatric patients, it was demonstrated that albumin administration did not improve pulmonary function, gastrointestinal tract function, wound healing, or out-

| TABLE 11.2 Resuscitation Formulas for Postburn Fluid Requirements During the First 24 Hours Postburn* |
| --- | --- | --- | --- |
| **Formula** | **Crystallloid** | **Colloid** | **Free Water** |
| **Adults** | | | |
| Parkland | Lactated Ringer’s 4 mL/kg/TBSA (%) | None | None |
| 1/2 in first 8 h | | | |
| 1/4 in next 8 h | | | |
| 1/4 in last 8 h | | | |
| Evans | Lactated Ringer’s 1 mL/kg/TBSA | 1 mL/kg/TBSA | 2,000 mL/m² |
| Brooke | Lactated Ringer’s 1.5 mL/kg/TBSA | 1 mL/kg/TBSA | 2,000 mL/m² |
| Modified Brooke | Lactated Ringer’s 2 mL/kg/TBSA | None | None |
| **Children** | | | |
| Graves | Lactated Ringer’s 3 mL/kg/TBSA | None | Maintenance |

*TBSA, total body surface area. Maintenance fluid requirements are 100 mL/kg/day for the first 10 kg body weight, 50 mL/kg/day for the second 10 kg body weight, 20 mL/kg/day for weight in excess of 20 kg.
In addition, a recent meta-analysis of albumin administration in critically ill patients concluded that the risk of death was increased in the albumin-treated group. Fluid requirements after the first 24-hour postburn period are determined in the usual fashion, with consideration of fluids lost through the burn wound and nasogastric suction. The main benefit of published guidelines is to alert the clinician who is unfamiliar with burn care that unusually large volumes of fluids and rates of administration are required for severely injured patients. Almost every author has acknowledged that patient variability prohibits development of a strictly calculated volume of resuscitative fluid and rate of administration.

In severe injuries, release of free hemoglobin from destroyed red cells and myoglobin from damaged muscle (especially following electrical injury) leads to destruction of renal tubules, acute renal failure, and possibly death. Binding of free pigments to the renal tubules can be prevented by establishment of a brisk urine flow with the use of resuscitation fluids and diuretics such as furosemide or mannitol, and alkalization of the urine (pH ≥6.5) with parenteral sodium bicarbonate. It is important to note that burn patients with rhabdomyolysis have estimated fluid requirements of 7 mL per kg/% TBSA—almost twice as large as the Parkland formula estimate.

**PHARMACOKINETIC CONSIDERATIONS IN BURN PATIENTS**

The characteristic biphasic metabolic response to injury of an initial short ebb or shock phase (hypometabolic) followed by a flow phase (hypermetabolic) was described by Cutberson in 1930. A burn injury that exceeds 10% to 15% TBSA causes pathophysiologic alterations in the cardiovascular, gastrointestinal, renal, and hepatic systems. Plasma proteins responsible for drug binding either increase or decrease in concentration, resulting in decreased or increased unbound drug concentration, respectively. Finally, the movement of drugs into and out of the circulation is increased through the burn wound. The pharmacokinetics and pharmacodynamics of many drugs are changed after thermal trauma.

**CARDIOVASCULAR CHANGES**

Cardiac output has been demonstrated to decrease by as much as 50% within 6 hours of severe thermal injury. This reduction in output has been attributed to hypovolemia, increased blood viscosity, increased peripheral vascular resistance, and the presence of a cardiotoxic protein called myocardial depressant factor. Theoretically, intravenous drugs have a slower rate of distribution and elimination during this initial 48-hour period.

Following resuscitation, the hyperdynamic or recovery phase of injury is associated with increases of cardiac output of up to one and one half to three times normal. This may not occur in the patient with preexisting myocardial disease. This increase in tissue perfusion is associated with an increased rate of drug distribution and elimination following intravenous administration.

**GASTROINTESTINAL CONSIDERATIONS**

Acute stress-related mucosal damage (SRMD) of the stomach and duodenum following severe burns is extremely common and is presumably related to increased acid secretion. The first case of acute gastrroduodenal ulcer associated with thermal injury was reported by Swan in 1823. Following the 1842 report on a series of 12 patients by Curling, the syndrome was established and named Curling ulcer. Prophylaxis and treatment of SRMD includes enteral feeding and administration of sucralfate, omeprazole, or H₂ receptor antagonists (H₂RAs).

Cimetidine appears to be unique among the H₂RAs in that after burns occur, it reduces resuscitative fluid requirements and has an increased elimination rate increased clearance. A study in burned children demonstrated a reduced cimetidine pharmacodynamic response, in addition to an altered pharmacokinetic profile. The absorption of orally administered drugs may be increased or decreased, depending on the drug pKa and whether intragastric pH has been modified by antacids or H₂RAs.

**RENNAL FUNCTION**

Initial renal insults following a severe burn injury consist of general hypoxia and reduced perfusion. Following severe injury, liberation of free hemoglobin or myoglobin may result in acute renal failure. These problems can be reversed rapidly with resuscitative efforts and establishment of adequate urine flow. During the postburn hypermetabolic phase, renal blood flow and glomerular filtration rate (GFR) are increased, although tubular secretion may be impaired. This suggests that the elimination of freely filterable drugs such as the aminoglycosides and vancomycin will increase after burn injury. This effect was demonstrated in a study of 20 burn patients, which reported abnormal increases for both GFR and tobramycin elimination in 13 of 20 patients. The need for increased dosage of gentamicin in burn patients has been demonstrated in numerous studies of both adults and children. Similarly, application of the extended-interval dosing strategy for aminoglycosides, that is, the technically incorrect and confusingly named once-daily aminoglycosides, can be problematic. Results are conflicting about increased renal elimination of vancomycin following burn injury, but the need for increased dosing is commonly observed.

**HEPATIC FUNCTION**

The hepatocyte is the most important site for drug metabolism, and, in general, it produces a metabolite that is more water soluble (facilitates urinary excretion) and of greater molecular weight (facilitates biliary secretion) than metabolites produced at other sites. Chemical reactions are classi-
The efficacies of the nondepolarizing neuromuscular blocking agents tubocurarine chloride, metocurine iodide, pancuronium bromide, and atracurium besylate are reduced after the first postburn week, which implies that increased plasma protein binding to AAG is responsible for this. Although increased binding does occur, the relatively small increase cannot explain the sometimes dramatic decrease in response. Investigations of the mechanism for this resistance have ruled out changes in drug clearance or volume of distribution. The decreased potency of these agents may be due to an unidentified substance in the plasma of burn patients.

Drug movement through burn wounds

Destruction of normal barriers to percutaneous absorption occurs with burn injury. Diffusion resistance to water movement through injured skin can be less than one-tenth that of normal skin. Gentamicin is absorbed readily following topical application of a 0.1% cream, and it is absorbed to a smaller extent with a 0.1% ointment. Eschar penetration has also been demonstrated in vitro for mafenide acetate, nitrofurazone, povidone-iodine, silver nitrate, and silver sulfadiazine.

Drug penetration of the burn wound is not unidirectional. Historically, it has been assumed that eschar penetration by systemically administered drugs was prevented by the avascular nature of the wound. However, systemically administered gentamicin and tobramycin both penetrate burn eschar. Drug loss through the burn wound may add substantially to total drug clearance.

INFECTION AND ANTIMICROBIALS

Despite therapeutic advances, infection in the burned patient remains the most important cause of death in those who survive initial resuscitation. Colonization of the burn wound has been demonstrated, even when the patient is cared for in a laminar flow room. Explanations for this phenomenon are that endogenous bacteria translocate from the gastrointestinal tract, bacteria are iatrogenically transmitted, and normal skin flora proliferate. In 15 patients with at least 20% TBSA evaluated within 24 hours of burn injury, the gastrointestinal barrier was compromised, as evidenced by increased absorption of lactulose and mannitol. However, the potential consequences of bacterial translocation continue to be debated.

BURN WOUND INFECTION

Methods and materials used in the treatment of burn wound infection have undergone significant changes. Although the importance of bacteria in the burn wound has been recognized, the terminology describing the association between wound bacteria and systemic manifestations of infection is confusing. Moncrief and Teplitz suggested that the term burn wound sepsis be used to describe the events associated with bacterial proliferation to 100,000 colony-
forming units (cfu) per gram of burn wound tissue and subsequent invasion of adjacent nonburned tissue. Unfortunately, this number of bacteria per gram of tissue is not diagnostic of an invasive burn wound infection, and a complex classification scheme ranging from surface contamination to microvascular invasion (I, II, III, IV, V, VIa, VIb, Vlc) has been suggested by Pruitt. 106

Whether bacteria are localized to the burn or are disseminated, a rational method for selecting from available topical antimicrobials is necessary. Similar to the Kirby-Bauer method of determining bacterial susceptibility to systemic agents, Nathan et al first reported on the agar-well diffusion method for determining susceptibilities to topical antimicrobials. 107 Support for this method was supplied by Heggers et al, who demonstrated that the agar-well diffusion test was more reliable than minimum inhibitory concentration determination for predicting bacterial susceptibility. 108

Topical Antimicrobials

Silver Nitrate. The "modern" use of silver nitrate began in the late 1800s with the prevention of ophthalmia neonatorum. Substantial improvement in the treatment of large burns through the use of continuously applied 0.5% silver nitrate solution was reported in 1965. 109 The characteristics that make 0.5% silver nitrate a useful topical antibacterial agent are its safety, water solubility, prolonged antibacterial action, lack of toxicity to viable skin, lack of antigenicity, and ease of preparation. Problems associated with its use include hypochloremia from formation of silver chloride salts, water intoxication because of the hypertonicity of the solution, and hyponatremia or hypokalemia from diffusion into the wet dressings. Other problems consist of a requirement for bulky dressings that restrict joint motion and ambulation, and black staining of everything that comes into contact with the solution.

Silver Sulfadiazine. The use of silver sulfadiazine (SSD) in burns was first reported in both a murine burn model and 16 patients. 110 SSD is unique among the usual topical antibacterial agents in effectively inhibiting Candida albicans. The exact antimicrobial mechanism of action of SSD has not been clearly elucidated, but its effect is attributed to silver inhibition of DNA replication or cell membrane modification. Two studies imply that the sulfadiazine component is not necessary for in vitro bacterial sensitivity. In addition, clinical efficacy may be associated with a reversal of injury-induced suppression of lymphocyte natural killer cell cytotoxicity rather than strict antibacterial effects.

SSD is the topical agent of choice worldwide because of its safety and efficacy. 111-113 Toxicity associated with SSD application is infrequent and is associated predominantly with the propylene glycol component of the cream base. The potential for allergic hypersensitivity is shown by circulating sulfadiazine antibodies (predominantly immunoglobulin [IgG]) in the serum of treated patients. Although SSD-associated leukopenia has been reported, it is probably an artifact of the physiologic response to burn injury of white blood cell margination or diapedesis (movement through vessels) from the intravascular space. 114 Clinicians should continue to apply SSD to patients who develop leukopenia.

Because of its demonstrated efficacy, SSD has been incorporated into a number of biologic and synthetic dressings or skin substitutes, to take advantage of its benefits and eliminate the inconvenience of dressing changes with reaplication of cream. Another method used to improve upon SSD is the addition of other agents such as nitrofurazone, gentamicin, fluoroquinolones, and cerium nitrate. The most successful combination is noted with chlorhexidine; Silvazine (Smith & Nephew, Clayton, Australia), a commercially available combination, has been used in Australia for over a decade.

Mafenide Acetate and Nitrofurazone. Although it causes pain upon application, mafenide acetate is a useful topical antimicrobial for the treatment of subeschar burn wound infection, because of its ability to penetrate the burn wound. Mafenide is often used on burned ears to prevent chondritis. Although the two are closely related chemically, mafenide is not a sulfonamide. The primary metabolite (p-carboxybenzene sulfonamide) is a sulfonamide, and it may cause allergic reactions in patients with sulfonamide hypersensitivity. When applied to large TBSA burns, mafenide can produce systemic metabolic acidosis secondary to carbonic anhydrase inhibition. 115 Another disadvantage of mafenide is its high cost—approximately four times that of SSD. The antimicrobial usefulness of nitrofurazone has been demonstrated since the mid-1940s. 116 Its primary use has been in prophylaxis of infection following skin grafting.

Miscellaneous Topical Agents. Topical nystatin may be useful in limiting candidal growth, even though fungal or yeast colonization in burn wounds has not been linked to increased mortality rates. 117 Although some reduced infection rates have been reported, there is little support for the use of unusual agents such as honey, gentian violet, or acetic acid. The use of polymyxin B and bacitracin ointments is limited to small wounds because of the potential for systemic toxicity when they are used over large areas. Mupirocin may be valuable in the treatment of methicillin-resistant staphylococci in burn wounds, but its use has been inadequately studied. The use of povidone-iodine solutions on burn wounds is considered inappropriate because its antibacterial activity is partially inactivated by wound exudates, and systemic absorption of iodine may cause renal dysfunction.

Systemic Antimicrobials. The use of prophylactic penicillin during the first postburn week was common during the 1950s and 1960s because of a justified concern about infection by Streptococcus pyogenes. This organism produced rapid conversion of partial-thickness to full-thickness wounds and fatalities. However, current laboratory methods for monitoring the burn wound and close clinical monitoring of patients allow the rapid recognition of infection. Recent prospective clinical trials have demonstrated no benefit for
prophylactic penicillin. Indeed, subsequent wound cultures in penicillin-treated patients demonstrate a greater incidence of resistant organisms.

The choice of antibiotic for systemic infection in burn patients should be the same as for other patients. However, because the pathophysiologic changes following burn trauma are dynamic, the dosing of systemic antimicrobials must be individualized when possible. Increased requirements for aminoglycosides and vancomycin have been demonstrated in burn patients (as discussed in the pharmacokinetics section).

**NUTRITIONAL SUPPORT**

The intimate relationship between nutrition and wound healing has been described. The injury-associated hypermetabolic response with altered nutritional requirements, including vitamins and micronutrients, is discussed.

**METABOLIC RESPONSE TO TRAUMA**

Hypermetabolism following trauma was initially explained as a physiologic response to increased heat loss. The rationale was that burned skin allows increased water loss that lowers the skin/wound temperature when the water evaporates. However, the precise relationship between evaporative water loss and postburn hypermetabolism is unclear, because conflicting results have been reported from similar investigations.

Similarly, it has been assumed that increased thermogenesis was necessary to compensate for heat loss in a cold environment, because damaged skin cannot respond with decreased perspiration and cutaneous vasomotor. However, postburn hypermetabolism is not attenuated, even when the environmental temperature is increased to above thermal neutrality. A resetting of the hypothalamic thermal regulatory setpoint is suggested by a study comparing burn patients with normal controls, in which burn patients selected a significantly higher environmental temperature when placed in a metabolic chamber.

Metabolic rate may be reduced following relief of pain, although the degree of reduction is not well defined. Historically, pain management of hospitalized patients with opioids has been suboptimal because of unnecessary fears of addiction. Morphine requirements of burn patients can be substantial, exceeding 60 mg per hour before tolerance develops.

Other contributors to postburn hypermetabolism are prostaglandins, interleukins, components of the complement cascade, and the catechol neurohumoral milieu of elevated serum cortisol, growth hormone, catecholamines, and glucagon levels. Initial insulin secretion inhibition is usually followed by normal or supranormal plasma insulin levels. Despite this insulin recovery, hyperglycemia persists, secondary to insulin resistance at the tissue insulin receptor.

Fuel stores that are mobilized to sustain postburn hypermetabolism include hepatic and muscle glycogen; visceral, plasma, and muscle protein; and fat. Because the major metabolic source of adenosine triphosphate (ATP) provided to the burn wound is anaerobic glycolysis, the obligatory glucose requirement is increased. Production of glucose from glycolysis is relatively short lived because stores only approximate 100 to 200 g and endogenous glucose production exceeds 400 g per day. Significant endogenous glucose is provided by efficient recycling of pyruvate and lactate via the Cori cycle and the glucose-alanine cycle. Catabolism of muscle protein and direct oxidation of amino acids provide approximately 15% to 20% of the total caloric expenditure in the fasting injured patient. The body adapts by using fat as its main energy source and can mobilize abundant energy from the typical fat stores of approximately 160,000 kilocalories.

Pharmacologic interventions that have been attempted to ameliorate the catabolic state following burn injury include anabolic steroids (testosterone and oxandrolone), insulin, insulinlike growth factor, and human growth hormone. A growing body of evidence supports the use of oxandrolone, with both short-term and long-term benefits demonstrated. Although a number of studies have demonstrated clinical benefits from adjunctive growth hormone treatment, there is little enthusiasm for its use because it is expensive and must be administered parenterally, and there is some evidence that growth hormone administration is associated with premature mortality.

The specific cause of postburn hypermetabolism is not clear, but it appears to be multifactorial. Complete arrest of postburn hypermetabolism is not currently possible. A reasonable approach is to provide the patient with a warm environment, adequate pain relief, early enteral nutrition, and aggressive wound coverage. In addition, an attempt should be made to minimize endogenous protein catabolism by providing exogenous protein and nonprotein calories.

**METHOD OF NUTRIENT ADMINISTRATION**

Patients with less than 20% TBSA burns can usually be maintained on a normal diet, unless there is an associated condition such as severe preburn malnutrition or an injury that prevents mastication. Patients with larger burns are often unwilling or unable to consume enough high-protein and caloric-dense food to fulfill requirements. For these patients, nutritional requirements can be met by insertion of a small-bore nasoenteric feeding tube and administration of commercially available enteral feeding formulations such as Osmolite-HN, TwoCal HN, Traumacal, and Replete. Enteral nutrition is preferred to parenteral nutrition because it is more physiologic and less costly, and it avoids the complications associated with parenteral nutrition, such as catheter-related sepsis.

In contrast to historical recommendations that focused on parenteral nutrition, current guidelines call for early enteral feeding. Even in severely burned patients with absent...
bowel sounds, feeding into the small intestine through a nasoenteric tube is still possible, because postburn ileus is confined primarily to the stomach.\textsuperscript{134} In these severely injured patients, a nasogastric tube is inserted and connected to suction for 2 to 3 days until gastric function returns. Experimental evidence favoring early enteral feeding demonstrated a reduction in catabolism and the hypermetabolic response in a guinea pig burn model. Another beneficial effect of early enteral feeding is the maintenance of gut mucosal mass. Improved gut wall integrity may prevent the increased intestinal permeability that allows translocation of enteric bacteria.

**MACRONUTRIENT NEEDS**

The metabolic demands of patients with severe burns exceed those of any other hospitalized patient. Postburn energy expenditure increases with increasing burn size. However, there is an upper limit to required calories. This upper limit is approximately twice the calculated basal energy expenditure (BEE), according to the Harris-Benedict equation. Numerous formulas have been developed to calculate a burn patient’s daily energy requirement, and representative formulas are listed in Table 11.3. Unfortunately, an investigation into the bias and precision of 46 methods published from 1953 to 2000 demonstrated that none were precise.\textsuperscript{132} Although mathematical calculation to estimate energy requirements is convenient, determination of the patient’s specific caloric needs is desirable.\textsuperscript{133} A complex metabolic chamber is necessary to specifically measure energy expenditure, but a reasonably accurate estimation can be performed at the bedside with the use of indirect calorimetry.\textsuperscript{134} Metabolic carts measure the respiratory gas exchange of oxygen (VO\textsubscript{2}) and carbon dioxide (CO\textsubscript{2}), thereby indirectly measuring energy expenditure (via the reverse Fick equation). Refer to Chapters 29 and 30.

### Carbohydrate Requirements

Energy liberated by oxidation of enterally administered carbohydrate is approximately 4 kilocalories per gram. The carbohydrate commonly administered parenterally is hydrous dextrose, which liberates 3.4 kilocalories per gram when completely oxidized. The optimal amount of administered carbohydrate will minimize gluconeogenesis without exceeding energy requirements and being stored as triglycerides.

The use of glucose for energy by burned patients has limits. When glucose is oxidized to liberate energy, equimolar concentrations of oxygen are consumed and carbon dioxide produced (respiratory quotient [RQ] = 1). The normal, fed RQ is approximately 0.84, and it rises when the rate of administered glucose exceeds the maximum rate of utilization. When glucose is converted into fat, more than eight times as much carbon dioxide is released for each mole of oxygen (RQ > 1). Excretion of this extra carbon dioxide could be difficult for a burn patient with an associated inhalation injury. An additional negative aspect of lipogenesis is that it is an energy-consuming process. An elegant study of intravenous glucose, performed with isotopic lipogenesis, demonstrated that the maximum rate of oxidation is approximately 5 mg/kg/min. At faster rates of glucose administration, the RQ rapidly increased above 1.0, suggesting lipogenesis. For a 70-kg patient, this maximum rate of glucose utilization translates into 2 liters of 25% dextrose—containing total parenteral nutrition solution (500 g) per day.\textsuperscript{135}

### Fat Requirements

Fat is an efficient provider of energy at 9 kilocalories per gram, but it is vital only for supplying essential fatty acids to prevent essential fatty acid deficiency syndrome. The amount of fat necessary in burn patients is not known, but fat should provide a minimum 20% of total calories. Fat is an essential component of cell membranes, functions as a carrier for fat-soluble vitamins, and is important for wound healing.

Patients with severe thermal injury may have reduced lipolytic capacity, especially following parenteral administration of fat emulsion. It appears that parenteral administration of long-chain triglycerides is associated with hepatomegaly, impaired clotting, and decreased resistance to infection. Another advantage of enteral administration is that medium-chain triglycerides are absorbed without the need for bile, and at the cellular level, these are transported into mitochondria without the need for carnitine.

Because of constraints on the rate of carbohydrate administration, fat must usually be provided in substantial quantities as an energy source. Although it is not often important

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**TABLE 11.3 Various Formulas Used to Estimate Energy Requirements in Burn Patients**

<table>
<thead>
<tr>
<th>Adults</th>
<th>Various Formulas Used to Estimate Energy Requirements in Burn Patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Harris-Benedict equation (estimates basal energy expenditure [BEE])</td>
<td></td>
</tr>
<tr>
<td>Male: BEE (kcal) = 66 + (13.7 x W) + (5 x H) - (6.8 x A)</td>
<td></td>
</tr>
<tr>
<td>Female: BEE (kcal) = 655 + (9.6 x W) + (1.7 x H) - (4.7 x A)</td>
<td></td>
</tr>
<tr>
<td>2. Burke and Wolfe</td>
<td>Kilocalories per day = 2 x BEE</td>
</tr>
<tr>
<td>3. Curreri</td>
<td>Kilocalories per day = 25 x W + (40 x TBSA)</td>
</tr>
<tr>
<td>4. Davies and Liljedahl</td>
<td>Kilocalories per day = 20 x W + (70 x TBSA)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Children</th>
<th>Various Formulas Used to Estimate Energy Requirements in Burn Patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Wolfe</td>
<td>Kilocalories per day = 2 x BEE</td>
</tr>
<tr>
<td>2. Curreri Junior</td>
<td>Kilocalories per day = (0-1 years) BEE + (15 x TBSA)</td>
</tr>
<tr>
<td></td>
<td>(1-3 years) BEE + (25 x TBSA)</td>
</tr>
<tr>
<td></td>
<td>(3-15 years) BEE + (40 x TBSA)</td>
</tr>
</tbody>
</table>

*W, weight in kilograms; H, height in centimeters; A, age in years; and TBSA, total body surface area burned [%].
clinically, fat has a specific advantage over glucose in patients with pulmonary dysfunction, for whom reduced carbon dioxide production for an equivalent amount of oxygen consumed is useful. The optimal fatty acid chain length and exact dietary fat requirements for burn patients remain to be determined.

**Protein Requirements.** Protein loss across burn wounds is considerable and is greatest during the first 3 postburn days. Although early protein loss across full-thickness burns is greater than that in partial-thickness burns, the rates become approximately the same after postburn day 3. The rate of protein loss is reduced by application of either antimicrobial creams or skin substitutes. With the use of average protein loss during the first postburn week (0.5 mg/cm²/h), a formula that estimates daily protein loss (grams) across the burn wound can be devised: $1.2 \times \text{body surface area (m²)} \times \text{TBSA burned (‰)}$. Protein loss across the burn wound during the second postburn week occurs at approximately half this rate.

The recommended daily allowance of protein for healthy adults is 0.8 g per kg. The optimal amount of protein required by burn patients to prevent catabolism of protein stores and promote wound healing is not well defined. The importance of protein sparing attained by providing energy must be considered, but some clinicians advocate a high-protein diet aimed at achieving a 100:1 nonprotein calorie-to-nitrogen ratio, in contrast to the standard 150:1 ratio. In clinical practice, approximately 1.5 to 2 g/kg/day (according to lean body weight) of protein is provided initially. Nitrogen balance studies then determine the adequacy of this regimen. Although the nitrogen balance calculation appears simple:

$$\text{Nitrogen balance} = \frac{N\ (\text{in}) - N\ (\text{out})}{11-2}$$

there is potential error in assessment of both $N\ (\text{in})$ and $N\ (\text{out})$. $N\ (\text{in})$ is the number of grams of nitrogen ingested or infused; it is common practice to multiply the number of grams of protein or amino acid by 0.16 to estimate grams of nitrogen. This calculation assumes that the protein is made up of 16% nitrogen, but the percentage of nitrogen available in parenteral amino acid products varies from 11.1% to 16.9%. The $N\ (\text{out})$ is calculated by adding the urinary urea nitrogen (UUN) from a 24-hour urine collection to an estimate of nitrogen excretion other than that measured as urine urea. This estimate comprises nonurea urinary nitrogen (ammonia, uric acid, creatinine) and nonurinary nitrogen loss (fecal and skin). A commonly used estimate for non-UUN losses is 4 g per day. One group advocates the measurement of total urinary nitrogen rather than use of an inaccurate estimate. As described previously, significant quantities of protein (nitrogen) are lost through open burn wounds and must be included when an estimate is used.

The branched-chain amino acids (BCAAs) leucine, isoleucine, and valine are unique in that skeletal muscle can oxidize them directly for energy. In contrast, the other amino acids are metabolized almost wholly by the liver. Under ordinary circumstances, only 6% to 7% of daily energy expenditure is provided through BCAA oxidation by skeletal muscle. The administration of supplemental BCAAs, especially leucine, to burn patients should theoretically reduce protein catabolism in skeletal muscle and increase protein synthesis. However, conclusive evidence of beneficial effects for BCAA-enriched solutions in burn patients has not been demonstrated, and further studies are needed. Another strategy for improving outcome in burn patients is the administration of beneficial nutrients, such as n-3 polyunsaturated fatty acids, arginine, glutamine, and nucleotides, in an attempt to reduce inflammation or enhance immunity. The hypothesis is that lipids high in linoleic acid (an omega-6 fatty acid in safflower or soybean oil) are potentially proinflammatory because linoleic acid gives rise to arachidonic acid, interleukins-1 and -6, and tumor necrosis factor-α. A number of clinical trials have been performed on several types of critically ill patients with the use of commercially available formulas such as Impact, Perative, and Crucial. Unfortunately, the results are conflicting, the benefits of these expensive diets have yet to be definitively demonstrated in burn patients, and much controversy exists. Irrespective of these scientific limitations, some groups of experts advocate the use of these specialty diets. The best evidence for a beneficial effect from these nutritional supplements appears to involve glutamine.

**MICRONUTRIENT NEEDS**

In contrast to the extensive information that has been obtained about macronutrient requirements, little information is available about the micronutrient needs of burn patients. Evidence suggests that micronutrient needs are increased following burns, although the exact amounts have not been defined. The branched-chain amino acids (BCAAs) leucine, isoleucine, and valine are unique in that skeletal muscle can oxidize them directly for energy. In contrast, the other amino acids are metabolized almost wholly by the liver. Under ordinary circumstances, only 6% to 7% of daily energy expenditure is provided through BCAA oxidation by skeletal muscle. The administration of supplemental BCAAs, especially leucine, to burn patients should theoretically reduce protein catabolism in skeletal muscle and increase protein synthesis. However, conclusive evidence of beneficial effects for BCAA-enriched solutions in burn patients has not been demonstrated, and further studies are needed. Another strategy for improving outcome in burn patients is the administration of beneficial nutrients, such as n-3 polyunsaturated fatty acids, arginine, glutamine, and nucleotides, in an attempt to reduce inflammation or enhance immunity. The hypothesis is that lipids high in linoleic acid (an omega-6 fatty acid in safflower or soybean oil) are potentially proinflammatory because linoleic acid gives rise to arachidonic acid, interleukins-1 and -6, and tumor necrosis factor-α. A number of clinical trials have been performed on several types of critically ill patients with the use of commercially available formulas such as Impact, Perative, and Crucial. Unfortunately, the results are conflicting, the benefits of these expensive diets have yet to be definitively demonstrated in burn patients, and much controversy exists. Irrespective of these scientific limitations, some groups of experts advocate the use of these specialty diets. The best evidence for a beneficial effect from these nutritional supplements appears to involve glutamine.

**Vitamins.** At a minimum, burn patients should receive vitamin supplements based on the recommended dietary allowances (RDA) for enteral administration, or the American Medical Association (AMA) Nutrition Advisory Group recommendations for parenteral administration. With the exception of vitamin D, in the absence of preexisting deficiency, little information indicates that increased amounts of fat-soluble vitamins should be administered. Vitamin C is often supplemented to $5 \times \text{RDA}$ because it has little inherent toxic potential and it plays an important role in collagen deposition and wound healing. Because of their role
as cofactors in metabolism and the potential for increased losses through the wound and urine, the B vitamin group is supplemented to $2 \times \text{RDA}$.

**Trace Elements.** In the acute-phase reaction to trauma, plasma concentrations of zinc, iron, and copper are markedly decreased. Similar to vitamin C, zinc is thought to promote wound healing, and it is supplemented to $2 \times \text{RDA}$. Aggressive iron supplementation must be undertaken with some caution because of the potential for increased bacterial growth due to plasma unbound iron. Deficiency syndromes of copper, selenium, chromium, iodine, manganese, and molybdenum occur in patients on long-term total parenteral nutrition, but no cases of deficiency appear to have been reported as a direct result of burn trauma. These trace elements are administered according to RDA or AMA guidelines.

**CONCLUSION**

The complex clinical management and rehabilitation of a severely burned patient require the efforts of a multidisciplinary team, including surgeons, nurses, a pharmacist, a dietitian, a psychotherapist, a physical therapist, an occupational therapist, a respiratory therapist, and a social worker. A large TBSA full-thickness burn requires surgical excision and split-thickness skin grafting. Fluid requirements during the initial postburn period are large, and guidelines for fluid resuscitation have been devised by experienced clinicians. The pharmacist must be aware that the postburn hyperdynamic and hypermetabolic phase produces multiple pharmacokinetic and pharmacodynamic changes. The nutritional requirements of burn patients can be substantial, with energy needs often approaching twice those of other hospitalized patients. A number of methods are available for estimating energy requirements through mathematical calculation, but determination of the patient’s specific caloric needs by indirect calorimetry is desirable.

- The amount of dietary protein required by burn patients to promote wound healing, replace losses, and prevent catabolism of protein stores is not well defined. Usually, intravenous amino acids or enteral protein at 1.5 to 2 g/kg/day is provided, and nitrogen balance studies are performed.
- Current guidelines call for the preferential use of enteral (rather than parenteral) nutrition.
- Prevention and treatment of infection in the burn patient are of paramount importance, because infection is the most common cause of death among patients who survive initial resuscitation. Treatment approaches for systemic infection in burn patients are similar to methods used for other patients, with dose individualization determined by antimicrobial serum concentration monitoring when possible. Microbial growth in the burn wound can be substantial following colonization by endogenous or exogenous organisms. The availability of topical antimicrobial agents has dramatically improved the control of burn wound infections.

**KEY POINTS**

- The complex clinical management and rehabilitation of a severely burned patient requires the efforts of a multidisciplinary team, including surgeons, nurses, a pharmacist, a dietitian, a physical therapist, an occupational therapist, a respiratory therapist, and a social worker.
- A large TBSA full-thickness burn requires surgical excision and split-thickness skin grafting.
- Fluid requirements during the initial postburn period are surprisingly large, and guidelines for fluid resuscitation have been devised by experienced clinicians.
- The pharmacist must be aware that the postburn hyperdynamic and hypermetabolic phase produces multiple pharmacokinetic and pharmacodynamic changes.
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**SUGGESTED READINGS**

REFERENCES