INTRODUCTION
The treatment of thermal injuries began to change during the latter half of the nineteenth century with the development of the first burn centers and physician scientists dedicated to improving the clinical outcomes through scientific research. Through their work, the LD$_{50}$ (the burn size lethal to 50% of the population) for thermal injuries has risen from 42% total body surface area (TBSA) during the 1940s and 1950s to more than 90% TBSA for young thermally injured patients. This vast improvement in survival is due to simultaneous developments in critical care, advancements in resuscitation, control of infection through early excision, and pharmacologic support of the hypermetabolic response to burns. This article reviews these recent advances in burn care and how they influence modern intensive care of burns.

RESUSCITATION
The foundation behind how clinicians currently replace fluids and electrolytes following thermal injury can be traced to the work of Frank Underhill in the 1930s. Underhill defined the pathophysiology of burn shock, or hypovolemic shock, and suggested a resuscitation formula based on the prevention of hemoconcentration. His...
resuscitation formula laid the groundwork for those used today, which have been established through direct observations, scientific methods, and consensus conferences. This fundamental understanding of burn shock, and the goals of treatment, has significantly reduced early mortality and acute renal failure following thermal injuries.

The basics of any initial resuscitation formula are to provide a balanced salt solution with total volume infused over the first 24 hours proportional to the affected body surface area (second and third degree) and the patient’s body weight. The rate of volume infused is calculated by delivering half of the volume over the first 8 hours and the remainder over the following 16 hours. It cannot be overemphasized that the resuscitative formulas are simply an estimate, and must be adjusted based on the physiologic response of the patient. Urine output has traditionally been designated as the gold standard to guide burn resuscitation, but has many limitations and represents a regional perfusion parameter. An astute physician trends regional perfusion parameters in addition to global perfusion parameters, such as central venous pressure, lactate, base deficit, and central venous oxygen saturations, to guide fluid administration. Other clinicians may apply more direct measurements of preload to avoid hypovolemic shock by using ultrasonography or advanced hemodynamic monitoring. The goal during resuscitation is to avoid overresuscitation or fluid creep. Saffle coined the term fluid creep to describe a trend of increasing fluid volumes administered during the initial 24 hours. Overresuscitation is known to be associated with complications such as compartment syndromes, and more recently Klein and colleagues demonstrated that overresuscitation resulted in increased infectious complications and mortality. The trend of fluid creep may be a result of clinicians attributing the pathophysiology of burn shock to a failure of preload (hypovolemia), and ignoring cardiac and afterload insufficiencies. Recently, a Glue Grant study demonstrated that a thermal injury of only 20% TBSA is associated with maximal stimulation of the inflammatory genome. The stimulation of the inflammatory genome and subsequent inflammatory cascade has been suggested as a cause of decreased vascular tone and cardiac insufficiency following thermal injury. Fortunately, these insufficiencies are generally mild, but a clinician should always use a goal-directed approach to the resuscitation of a thermally injured individual. The goal should be to first maximize preload before administration of pharmacologic support to the cardiovascular system.

Advances in resuscitation have substantially improved survival following thermal injury and investigators continue to explore methods to further reduce mortality and morbidity. One such exploration is the supplementation of resuscitative fluids with albumin. The theory behind this practice is to support the oncotic pressure secondary to the rapid reduction in serum albumin concentrations following thermal injuries. Data suggest, however, that albumin supplementation during resuscitation, or in the subacute period, increases costs without improving clinical outcomes. The burn literature appears to be consistent with the SAFE trial, which suggested that colloid was not superior to crystalloid resuscitation following trauma.

**HYPERMETABOLIC RESPONSE TO THERMAL INJURIES**

Despite the advances in resuscitation, wound coverage, and infection control, thermally injured patients are still at risk for significant morbidity and mortality secondary to complex metabolic changes following the initial injury. These changes are referred to as hypermetabolism, and are characterized by increased body temperature, glycolysis, proteolysis, lipolysis, and prolonged futile substrate cycling. Collectively this
syndrome is manifested by an increased metabolic rate and peripheral protein catabolism. The response was originally described by Sneve in 1905 as exhaustion, and despite his initial observations being more than 100 years old, his recommendations of a nourishing diet and exercise remain two of the key components used today to combat this metabolic disturbance.

Because of the impact of hypermetabolism on morbidity and mortality, physicians and scientists have attempted to modulate this response. Research areas have included nutritional supplementation, environmental ambient temperature, and pharmacologic agents that directly modulate the response. Clinicians rapidly recognized the inability of patients to consume sufficient calories, and recommended continuous nutritional supplementation. Curreri and colleagues published the first formula to estimate the caloric requirements for burn patients: calories/d = 25 (weight in kg) + 40 (TBSA). Supplementation of calories and protein was found to reduce, but not eliminate, the effects of hypermetabolism. Experimentation with supraphysiologic supplementation was found to maintain and, in some cases, increase total body weight. In such cases, however, the increase in total body weight was due to an increase in fat composition and not lean body mass. As a result of hypermetabolism and subsequent impaired substrate utilization, lean body mass continued to decline despite the supraphysiologic supplementation. Today, most clinicians favor continuous enteral nutrition with a high carbohydrate diet consisting of 82% carbohydrate, 3% fat, and 15% protein. This diet has been suggested to stimulate protein synthesis, increase endogenous insulin production, and improve overall lean body mass in comparison with alternative formulas.

Perhaps one of the simplest methods to reduce the hypermetabolic response following thermal injury was suggested by Wilmore and colleagues with respect to environmental ambient temperature. Wilmore demonstrated that ambient temperature had a significant effect on the hypermetabolic response following thermal injury. He suggested that burn-injured patients desire a core body temperature of 38.5°C and that increasing the surrounding ambient temperatures would greatly reduce the hypermetabolic response. In fact, it was shown that elevating the surrounding ambient temperature from 28°C to 33°C could reduce resting energy expenditure from 2.0 to 1.4 in patients with burn injury exceeding 40% TBSA. This simple therapeutic intervention, however, did not completely abolish the effects of hypermetabolism, leading investigators to search for alternative methods to alter this impaired metabolic response.

The final method for directly modulating the hypermetabolic response to thermal injury was administration of pharmacologic agents. Two broad classes of agents have been investigated over the last 30 years: anabolic agents and β-adrenergic antagonists (β-blockers). Anabolic agents have been modestly effective in modulating the hypermetabolic response to thermal injuries. Oxandrolone, a testosterone analogue, has been the most extensively studied. Multiple prospective controlled trials were performed in burn-injured patients, all with positive results. Administration of oxandrolone has been demonstrated to improve net nitrogen balance, increase the speed of donor site healing, decrease weight loss, and shorten overall length of hospital stay. These improvements in muscle metabolism and protein synthesis have been confirmed in both adult and pediatric burn patients. Other hormonal agents, such as human growth factor and insulin, have been investigated and shown to improve protein metabolism; however, their utility in the treatment of hypermetabolism has been limited by their potential side effects. In addition to anabolic agents, β-adrenergic blockade has also been extensively studied and shown to effectively alter the hypermetabolic response to burn injury. The administration of propranolol to acutely burn-injured children was demonstrated to reduce resting energy expenditure
and peripheral lipolysis while enhancing lean body mass by improving intracellular recycling of free amino acids for protein synthesis. Though proven in children, the administration of β-blockade in adults should be performed with caution, as β-blockade was recently found to be detrimental in young healthy adults undergoing surgical intervention.

In summary, the treatment of hypermetabolism is not limited to a single modality. The therapeutic interventions begin with early excision and grafting in an effort to limit the overall caloric needs of the patient and to prevent infectious complications. The ambient thermal environment should be maximized to 33°C to reduce unnecessary caloric expenditure found with lower ambient temperatures. Finally, appropriate, continuous enteral nutrition should be instituted immediately following resuscitation, and should include the addition of agents to modulate the hypermetabolic response.

**ACUTE KIDNEY INJURY AND FAILURE**

Despite advances in resuscitation, acute renal failure remains a major complication following thermal injury and is associated with a high mortality rate. The incidence of renal failure in the burn population ranges from 0.5% to 30% with a projected mortality rate greater than 50%, and renal replacement therapy (RRT) only marginally affects overall mortality. The best treatment option for renal failure, therefore, remains prevention. One of the greatest dilemmas regarding renal failure over the years has been the lack of a true consensus with respect to its definition and classification. Although most clinicians agree that acute renal failure is a rapid and prolonged decline in renal function, some 30 different definitions have been cited in the literature. Recently, the International Acute Dialysis Quality Initiative group attempted to standardize the definition of acute renal insufficiency by developing the RIFLE criteria (Table 1). The RIFLE criteria define 3 different grades of acute renal injury (risk, injury, failure) based on glomerular filtration rate, urine output, and 2 clinical outcome parameters (loss and end-stage kidney disease). The RIFLE classification system has been validated in multiple clinical scenarios including thermal injury, and it is expected that this consensus definition and classification will aid in future studies.

Acute kidney injury (AKI) related to thermal injury is most likely to occur at two distinct time points: early, during resuscitation; or late, secondary to septic episodes. Historically, early AKI was primarily a result of hypovolemia or being under resuscitation. At present, however, data suggest that early acute renal injury is multifactorial. In fact most patients with AKI after a major burn receive more than 4 mL/kg/%TBSA; therefore, hypovolemia is less likely to be the primary cause of kidney injury in these cases. Cardiac insufficiency,

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Abbreviations: ARF, acute renal failure; ESRD, end-stage renal disease; GFR, glomerular filtration rate; S.Cr., serum creatinine.
inflammatory mediators and cytokines, release of denatured proteins, and nephrotoxic
agents have all been suggested to contribute to the development of early AKI.39

Early AKI depends on the degree and duration of shock following thermal injury;
therefore, prevention of AKI requires early and aggressive fluid resuscitation and pres-
ervation of normal renal perfusion. This precept was demonstrated by several
investigators showing that the timing and adequacy of resuscitation was critically
important in the prevention of AKI.40,41 Modern-day burn surgeons, therefore, monitor
global parameters of perfusion (lactate, base deficit, central venous saturation), as
these parameters are more appropriate than urine output alone in reflecting the degree
and recovery from a hypoperfused state, or a state of shock. Despite a physician’s
best attempt to monitor the response to resuscitation, obligatory intercompartmental
fluid shifts will occur and may influence renal perfusion. Individuals demonstrating
greater than anticipated fluid requirements should be monitored for the development
of intra-abdominal hypertension and/or compartment syndrome. If detected, each
condition should be appropriately addressed to maintain normal renal perfusion.42

Although appropriate fluid administration is critical following a major thermal injury,
clinicians should not automatically attribute all hypoperfused states to a lack of pre-
load (ie, hypovolemia). Although a clinician must first ensure that preload is adequate,
increasing evidence has suggested that cardiac dysfunction, induced by the release of
direct myocardial suppressants (including tumor necrosis factor), is now known to
occur following major thermal injuries.43–48 Coupled with the potential of vasodilata-
tion secondary to a burn-related systemic inflammatory response, a goal-directed
approach is suggested as the best way to preserve normal renal perfusion.

Although a goal-directed approach should maintain normal renal perfusion, a burn-
related injury could be complicated by circulating denatured proteins. Rhabdomyoly-
ysis and free hemoglobin have both been suggested to contribute to the development
of AKI.49 Rhabdomyolysis may result from direct thermal damage or a compartment
syndrome. Commonly associated with electrical injuries (>600 V), rhabdomyolysis re-
sults in blockage of the renal tubules, constriction of afferent arterioles, and generation
of oxygen free radicals.50 The degree of injury is related to preexisting renal insuffi-
ciency, the amount of circulating iron-containing substances, and the overall state
of hydration.51 Fortunately, the prognosis is favorable for renal recovery if the source
is identified early and appropriate hydration is initiated. Isotonic crystalloids are the
fluid of choice, with bicarbonate supplementation reserved for those individuals
demonstrating an acidic urinary pH despite adequate resuscitation. Nevertheless,
one study of more than 2000 trauma patients in the intensive care unit (ICU) demon-
strated no differences in the rates of renal failure, dialysis, or mortality in those patients
who received bicarbonate or mannitol. Furthermore, renal failure occurred only in
patients with creatine kinase levels greater than 5000 U/L.52

The pathophysiology of late AKI is unlike that of early AKI, and remains a formidable
problem within the burn intensive care unit (BICU). Sepsis, or septic shock, accounts
for 87% of the cases of acute renal dysfunction identified in the BICU.29,53 The path-
ophysiology of late AKI is multifactorial, but primarily relates to the systemic inflamma-
tory response that accompanies a septic event. The sepsis-induced inflammatory
response is characterized by a generalized vasodilatation and a hypercoagulable
state, which ultimately culminate in AKI via a reduction in renal perfusion: globally
via vasodilatation, and locally by the formation of microthrombi in the glomeruli.39
Treatment is similar to that of early AKI, with a goal-directed approach modified to
include early treatment of the infectious process. Source control must be established
and appropriate antibiotic selection is critical, keeping in mind that many antibiotics
can be nephrotoxic.
The early diagnosis of acute renal insufficiency is difficult because of the lack of early biomarkers. Most clinicians rely on urine output or elevations in serum creatinine to detect renal insufficiency, but both have their clinical limitations. A clinician must maintain an environment favorable for normal renal function by avoiding hypoperfusion and nephrotoxic substances. If renal insufficiency is clinically apparent, a physician should classify the etiology based on microscopic and biochemical analysis. The goal is to differentiate primary renal dysfunction from a prerenal state, which then may influence therapeutic options. This goal can be achieved by calculating the fractional excretion of urea, which is more specific and sensitive than the fractional excretion of sodium.54

If renal function cannot be stabilized by goal-directed therapy, RRT should be instituted. Unfortunately, the optimal timing and type of RRT following thermal injury has yet to be determined. A recent consensus conference regarding RRT in critically ill patients suggested that RRT should be instituted before the development of significant metabolic derangements or life-threatening events.55 Despite advances in intensive care and RRT, mortality rates for thermally injured individuals requiring RRT approaches 80%.

**HYPERGLYCEMIA**

Hyperglycemia and glucose intolerance are frequent manifestations of the metabolic response to injury, and have been recognized for more than 50 years. Both oral and intravenous glucose tolerance tests following serious burns and other forms of injury have demonstrated impaired glucose metabolism. During the stress response to injury, both the sympathoadrenal and hypothalamic-pituitary-adrenal axis stimulate a hyperglycemic state through the release of catecholamines and glucocorticoids.56 Counterregulatory hormones also seem to play a role by promoting hepatic and muscle glycogenolysis, gluconeogenesis, and peripheral lipolysis.56,57 The net effect is an exaggerated increase in net glucose production that is critical to the uncoupling of the normally tightly controlled serum glucose levels.58 The postinjury glucose production rate has been measured to be two standard deviations above normal values.59 This increase in glucose production contributes to stress-induced hyperglycemia, but hepatic and peripheral insulin resistance also plays a role in this phenomenon. A wide range of studies have been conducted on the insulin resistance of peripheral muscle and the manifestation of insulin resistance in the liver.60,61 In terms of unsuppressed hepatic glucose production, especially in the stressed physiologic condition, the hormonal modulators of this phenomenon need to be further explored. Previous studies have demonstrated that in healthy subjects, hepatic glucose production can be inhibited by exogenous glucose infusion at rates less than 2 mg/kg/min.62 In severely burned patients, however, even 5 mg/kg/min of exogenous glucose supply was unable to inhibit this process; therefore, unsuppressed glucose production from the liver plays a major role in causing the detrimental hyperglycemia.

In 1979, The group led by Burke and Wolfe of the Boston Shriner’s Hospital investigated and reported on the importance of adequate glucose supply for the severely injured burn patient.58,63 These observations were later confirmed by the same group in the pediatric burn population.64 Since 2001, physicians have become increasingly aware of the concept that even short-term elevations of serum glucose can be detrimental to an ICU patient. Van den Berghe and colleagues55 used a continuous insulin infusion to maintain normoglycemia in a population of surgical ICU patients. Their intensive control of blood sugar was defined as a level between 80 and 110 mg/dL versus a conventional control of blood sugar less than 215 mg/dL. The investigators
demonstrated a statistically significant lower morbidity and mortality in the group under intensive control of blood sugar. This finding has been supported by further investigations demonstrating that elevated blood sugars were associated with an abnormal immune function, increased infection rate, and abnormal hemodynamics. The sequelae appear to be secondary to the elevated glucose levels, and not due to the direct lack of insulin. The deleterious effects of elevated blood sugar have also been demonstrated in the burn-injured population. Gore and colleagues suggested that in the pediatric population, bacteremia/fungemia, reduced skin graft take, and mortality were all directly related to hyperglycemia. Although it is becoming apparent that hyperglycemia worsens outcomes in the ICU population through multiple mechanisms (Fig. 1), the intensive insulin therapy to control blood sugar levels is not without complications. The original study by Van den Berghe and colleagues revealed a 6-fold higher incidence of hypoglycemia in the intensive group (5.1% vs 0.8%) in comparison with the conservative group. The concern for hypoglycemia within the ICU has resulted in the creation of an entire industry interested in glycemic monitoring. Although the data are highly suggestive of improved morbidity and mortality with tight glycemic control (80–110 mg/dL) within the ICU patient population, the implementation of such protocols has been less than accommodating in both workload and prevention of hypoglycemic events. In summary, clinicians should maintain glycemic levels of less than 180 mg/dL in the intensive care setting while preventing even moderate hypoglycemia (<80 mg/dL).

**POSTBURN VENTILATION**

Over the past decade, significant advances had been made in the support of intensive care patients suffering from respiratory failure. This aspect is of particular importance to the thermally injured patient because the risk of inhalation injury is proportional to the TBSA affected by thermal injury and the association of increased mortality. Inhalation injury is a nonspecific term referring to direct injury to the pulmonary system (airway passages or lung parenchyma) or systemic toxicity secondary to substances absorbed. Before the widespread acceptance of the Acute Respiratory Distress Syndrome Network (ARDSnet) recommendations, inhalation injury was reported to increase the risk of burn-related mortality by as much as 30%. It has been classified into 4 separate categories: upper airway injury, lower airway injury, parenchymal injury,

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**Fig. 1.** Carbon monoxide nomogram.
and systemic toxicity. The extent of injury depends on the ignition source, heat energy transferred, and the concentration and solubility of toxic gases generated. Unfortunately, at present there is no consensus on the diagnostic criteria for inhalation injury.

Proper diagnosis and care of a patient with potential inhalation injury begins with a high clinical index of suspicion. Any patient with soot or thermal injuries to the face, or a history of prolonged exposure to toxic gases in an enclosed environment, should be presumed to have an inhalation injury. A systematic approach to the evaluation of the aerodigestive tract and bronchial tree should then be performed.

Upper airway injury should be excluded by direct or indirect visualization. Most commonly, upper airway obstruction is secondary to systemic anasarca, which typically progresses over the first 24 hours and is related to resuscitation of larger burns (>20%) and the accompanying capillary leak. Progression of systemic edema may result in increasing dyspnea, and should alert the clinician that an airway needs to be established and secured. Securing the upper airway with an endotracheal tube should be performed early to avoid emergent intubation, as this has been demonstrated to be associated with contamination of the respiratory tract with pathologic microorganisms.

Injuries to the subglottic airways or lung parenchyma are almost exclusively secondary to inhaled toxins and particles and the subsequent inflammatory response. The one exception is a steam-related airway injury. Inhaled steam has a significantly higher heat-carrying capacity than that of dry air, and therefore can overwhelm the protective mechanisms of the upper airway and result in direct thermal injury. The diagnosis of this inflammatory response can be established by bronchoscopy, because hyperemia of the bronchial airways is the most consistent clinical finding of an inhalation injury. Unfortunately, the degree of inhalation injury seen on bronchoscopy does not always predict the extent of pulmonary insufficiency. It has been suggested that the arterial serum carbon monoxide levels may be helpful in determining the extent of exposure to inhaled toxins or particles, but this has not been shown to be a precise marker. All patients, therefore, should be treated as though they have suffered a significant inhalation exposure or injury. For intubated patients, the ARDSnet protocol should be instituted immediately on arrival at the emergency department. The practice of low tidal volume ventilation, 5 to 7 mL/kg, prevention of barotrauma, and maintenance of plateau pressures less than 30 mm Hg has significantly influenced the rate of acute respiratory distress syndrome (ARDS). In the trauma literature there has been a 50% reduction in the incidence of ARDS since the publication of ARDSnet recommendations, despite similar injury severity scores. Similar findings are being suggested in the burn population after the adoption of these new ventilator concepts.

The administration of a β-agonist should be limited to symptomatic patients, as routine use has been associated with increased mortality. Among intubated burn patients, the goal is to prevent additional injury to the pulmonary system via artificial respiration or unnecessary pharmacologic agents. Fortunately, the long-term effects of inhalation injury following thermal injury seem to be well tolerated, and despite demonstrating signs of a restrictive pattern of respiration, most patients compensate well, especially in the pediatric population.

As mentioned previously, inhalation injury may also lead to the absorption of toxins whose effects are systemic and therefore may influence multiple organ systems. The two most well-known agents are carbon monoxide and hydrogen cyanide. Any individual exposed to the gases produced from the combustion of modern materials is at risk for significant poisoning of either carbon monoxide or hydrogen cyanide. Clinicians must have a high index of suspicion to appropriately diagnose and treat these potentially lethal exposures.
Carbon monoxide is a colorless and odorless gas formed as a result of incomplete combustion. It has a high affinity for hemoglobin and displaces oxygen, resulting in systemic hypoxia. The predominant toxic effect is related to the formation of carboxyhemoglobin, but carbon monoxide also binds intracellular cytochromes and other metalloproteins, which explains its 2-compartment elimination. The clinical signs of carbon monoxide exposure are directly related to the percentage of carboxyhemoglobin (COHb) in the blood (see Fig. 1). The diagnosis is established by measuring the arterial COHb, but care should be taken when determining the actual amount of exposure. Delay in laboratory analysis and the administration of supplemental oxygen may falsely lower the true value related to the exposure. A nomogram has been developed that allows for calculation of the “actual” COHb level at the time of the inhalation event. Regardless, all patients should be treated with high-flow oxygen until symptoms resolve and COHb levels return to normal. Some investigators have advocated for hyperbaric oxygen for carbon monoxide poisoning, but the risks and benefits of this therapy must be weighed, especially following a major thermal injury to a patient.

Hydrogen cyanide is the gaseous state of cyanide, produced from the combustion of nitrogen and carbon-containing materials. The gaseous state is colorless but has the odor of bitter almonds. Hydrogen cyanide produces its toxic effects by causing a reversible inhibition of cytochrome c oxidase. This intracellular interaction inhibits cellular oxygenation, resulting in tissue anoxia. The diagnosis of hydrogen cyanide toxicity is clinical, because laboratory analysis for cyanide levels is not routinely available at most hospitals. The diagnosis may be suggested by laboratory analysis demonstrating a gap metabolic acidosis following an inhalation injury. Serum lactate levels greater than 10 mmol/L and/or an elevated mixed venous oxygen saturation may also indicate cyanide poisoning. The treatment of cyanide poisoning has been simplified since the introduction of hydroxocobalamin. A precursor of vitamin B₁₂, it has been found to be safe but may falsely alter some laboratory values. Clinicians should familiarize themselves with the alterations of standard laboratories measured within the ICU.

Lastly, as part of any discussion on postburn ventilation, the topic of tracheostomy should be addressed. Similar to that of general trauma or other critically ill patients, the debate revolves around late versus early tracheostomy, and more recently that of open versus percutaneous tracheostomy. In summary, there are no level 1 data to clearly establish early versus late or percutaneous versus open tracheostomy as a standard of care in burn patients. Similarly, in critically ill patients no clear benefit of early tracheostomy has been proved. With respect to percutaneous versus open tracheostomy, outcomes are similar, although studies have shown that percutaneous tracheostomy can be performed in less time, and has a lower rate of surgical-site infection and a lower rate of clinically significant postoperative bleeding. The most recent data on burn patients have demonstrated early percutaneous tracheostomy to be safe and effective when performed with bronchoscopic guidance.

INFECTION CONTROL

Infections, primarily wound infections, have always complicated the management of thermal injuries. Since the time of Dupuytren, physicians have understood the concept that burn wounds should be treated with debridement followed by definite closure. Whereas the concept of removing all nonviable tissue was established, the timing of this tissue removal varied significantly over the years. With early burn wound excision and closure, however, surgeons began noting improvements in length of hospitalization, functional outcome, and survival. These improved outcomes were thought to
be attributed to maintaining lower colony counts at the wound surface, thus preventing invasive wound infections and sepsis. Today, early burn wound excision (first 72 hours) is the standard of practice for all full-thickness thermal injuries.

As previously mentioned, proper management of the burn wound is critical in decreasing the incidence of burn wound infections and sepsis. However, despite the best management, thermally injured individuals are at risk for standard infectious processes within the ICU. In fact, the incidence of central-line infection in BICUs is approximately double that seen in other ICUs. This higher incidence of central-line infections occurs despite the rotation of insertion sites on a regular basis and the use of antibiotic-coated central venous catheters. Owing to the risk of infectious events, an astute clinician must constantly monitor a thermally injured patient for evidence of infection. However, as a result of the hypermetabolic response to burn injury, common markers of infection, such as elevated core temperatures, are generally unreliable in the burn population. A clinician must combine known unique markers of infection in a burn patient (enteral feeding intolerance) with other known markers of infection (elevations in acute-phase reactants) to properly diagnose and treat infectious insults. All infectious events should be treated aggressively with de-escalation of antimicrobial therapy based on culture and sensitivity data.

The modern treatment of thermal injuries requires a multidisciplinary approach to infection control. Strict infection-control practices originally suggested by Simes and Colebrook form the foundation of care in most burn centers. The prompt removal of full-thickness thermal injuries coupled with proven topical antimicrobial therapy such as mafenide acetate, silver nitrate, silver sulfadiazine, and 0.5% Dakin solution have greatly improved infection complications. Systemic antimicrobial and antifungal therapy is of therapeutic benefit in the treatment of a thermally injured individual, but should be reserved for documented invasive or systemic infections. Despite the improvement in infection-control measures, sepsis leading to multiorgan failure is still lends a significant risk of mortality to the thermally injured patient, especially with the emergence of multiple drug-resistant bacteria.

SUMMARY

As a result of advances in resuscitation, control of infection, and support of hypermetabolism, thermally injured patients now have an excellent chance of survival. The latest data would suggest that the LD₅₀ for burns based on TBSA affected is currently 90%. To achieve these outstanding outcomes, a multidisciplinary approach must be used within the BICU. The multidisciplinary team must appreciate the interrelationships between various components of burn care, such as the role of overresuscitation and its influence on infection control, and the influence of sepsis on the hypermetabolic state. Fortunately, there exist physicians, scientists, and professionals within each clinical support service who continue to explore these relationships in an effort to improve care.

REFERENCES


