Evolving Changes in the Management of Burns and Environmental Injuries

Leopoldo C. Cancio, MD[a], Jonathan B. Lundy, MD[a], Robert L. Sheridan, MD[b,*]

KEYWORDS

• Burns • Inhalation injury • Heat injury • Frostbite • Hypothermia

KEY POINTS

- As in trauma, a formatted initial evaluation of burn patients will minimize missed opportunities for optimal care.
- Fluid resuscitation of burns continues to evolve. Colloid and hourly adjustment play an increasingly important role.
- Critical care of the burn patient has several unique components, particularly pain and anxiety control, environmental control, inhalation injury management, transeschar fluid and electrolyte losses, and nutritional support issues.
- Burn care can be divided into four phases: initial evaluation and resuscitation, initial wound care, definitive wound closure, and rehabilitation and reconstruction.
- Rehabilitation should begin coincident with initial care.
- Injuries due to heat and cold have both systemic and local priorities.
- Freeze-thaw-refreeze should be avoided in frostbite patients. In rare patients with frostbite, there may be a role for thrombolytics.

INTRODUCTION

Burns, soft-tissue wounds, and environmental injuries are common in injured survivors of natural disasters and terrorist incidents. They are also common in those injured

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[a] U.S. Army Institute of Surgical Research, 3698 Chambers Pass, Fort Sam Houston, TX 78234 6315, USA; [b] Department of Surgery, Shriners Hospital for Children, 51 Blossom Street, Boston, MA 02114, USA

* Corresponding author.

E mail address: rsheridan@partners.org

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in combat and peacetime deployed military settings. Burns complicate a significant number of explosion injuries.[1] Effective management is facilitated by preestablished protocols, implementation of which require an understanding of the unique contributions of burns to morbidity and mortality. In general, patient management is divided into four phases: initial evaluation and resuscitation, initial wound management, definitive wound closure, and rehabilitation. The focus of this article is on recent advances in the initial hospital care of patients suffering burns and environmental injuries; long-term issues are only briefly acknowledged.

INITIAL EVALUATION

General Approach

The triage and initial evaluation of the burn patient should focus on identification of life-threatening injuries. During the primary survey, the airway takes first priority. Acute airway loss after thermal injury can be a result of direct damage to, and edema of, any portion of the upper airway—from face to glottis. Stridor, hoarseness, and/or respiratory distress identify a patient with inhalation injury who requires urgent intubation. Airway loss may occur during the initial hours postburn, even in the absence of inhalation injury and especially in patients with total body surface area (TBSA) burned equal to or greater than 40%. This is caused by edema of unburned tissue, for which early elective intubation is encouraged. Intubation may also be required for patients who are obtunded due to hypoxia and/or inhalation of toxic products of combustion (carbon monoxide, cyanide).

Children are at high risk of acute airway loss and tolerate hypoxia poorly. When intubating burned children, a cuffed endotracheal tube is preferable. During burn resuscitation, pulmonary compliance decreases, which can result in an uncontrolled air leak around an uncuffed tube. These same patients often develop massive facial edema, making urgent tube exchange treacherous—a situation which is best avoided from the outset.[2]

The inability to oxygenate after such injuries may be a result of airway obstruction, inhalation injury (see later discussion), or concomitant thoracic trauma. In addition, progressive edema of eschar and subjacent tissue of the chest and abdominal wall may lead to loss of thoracic compliance, elevated peak and plateau pressures, and hypoxia—especially if the patient has sustained circumferential, full-thickness torso burns.

Evaluation of adequate circulation and perfusion should include assessment of peripheral pulses, mentation, level of consciousness, and serum markers of hypoperfusion (base deficit, serum bicarbonate, and lactate). In the absence of concomitant mechanical trauma or a long delay in resuscitation, profound hypotension at initial evaluation is uncommon. During initial evaluation, intravenous access should be obtained and a fluid infusion started. In the absence of hypotension or other evidence of profound hypovolemic shock, no bolus should be given. This is in contradistinction to Advanced Trauma Life Support (ATLS) guidelines for mechanical trauma patients.[3]

Neurologic abnormalities during initial evaluation can result from toxin exposure, head or spine injury, or, less frequently, compression of peripheral nerves as a result of eschar or compartment syndrome. The final component of the primary survey concerns the exposure of the patient for identification of other injuries. If mechanical trauma is suspected, cervical spine precautions should be maintained until injury is ruled out. Facial burns place the patient at risk for corneal injury, so examination using a Woods lamp and fluorescein should be performed. Identification of all burns with mapping of the extent using a technique such as the rule of nines or the

Lund-Browder chart will help determine the severity of burn injury as well as predict expected resuscitative needs. Although obviously superficial burns (**Fig. 1**) and markedly deep burns (**Fig. 2**) are easily identified, many severely burned patients have a mix of superficial partial-thickness, deep partial-thickness, and full-thickness burns not readily distinguished acutely after injury. These wounds should be reexamined daily to assist with determination of depth and future surgical planning to achieve wound closure. Also, circumferential burns to extremities or the torso should be identified to alert the clinician to areas that may be at risk for development of eschar syndrome (see later discussion).

A special note should be made on abuse in thermally injured patients. Cases of abuse can occur in all age groups, but most commonly impact the extremes of age. Patterns of intentional thermal injury include cigarette burns (most common type of abuse-related burn, usually not requiring hospital admission), intentional immersion with scald injury to hands, buttocks, and posterior legs and heels, and iron burns of the hand. Abuse-related burns are most commonly seen in children of 2 years old or younger, who typically also demonstrate signs of neglect such as poor hygiene, malnutrition, and delayed psychological development. Suspicion of a burn related to abuse mandates a thorough investigation of the events surrounding the incident and referral to proper personnel to ensure the safety of the patient.

Transfer Criteria

As early as possible during initial evaluation, a determination should be made as to whether the patient merits referral to a burn center. The American Burn Association (ABA) has established criteria for burn center referral[4]:

- Extent ($\geq$10% TBSA)
- Location (face, hands, feet, genitalia, perineum, joints)
- Depth (any full thickness burns)
- Cause (electric, chemical, inhalation injury)
- Complicating factors (patients with special medical or rehabilitation needs).

When patients have mechanical trauma and burns, initial stabilization may be required in a trauma center, followed by burn center transfer. The key to managing the transfer process is early and frequent communication between the referring hospital and the receiving burn center.

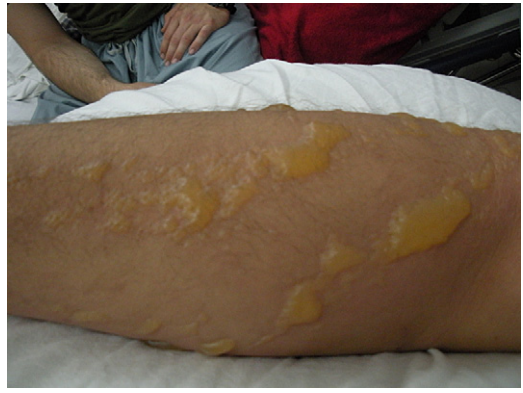

Fig. 1. Superficial thermal injury.

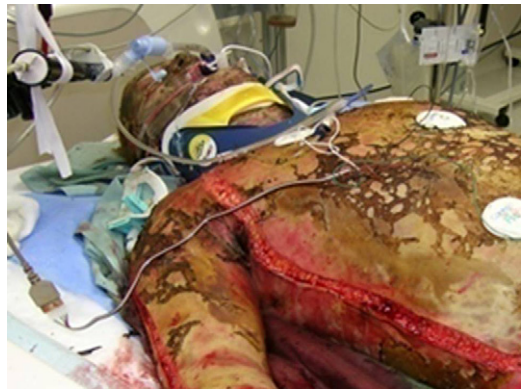

Fig. 2. Full thickness thermal injury.

FLUID RESUSCITATION

Resuscitation Formulas

Thermal injury leads to progressive loss of intravascular volume, edema in burned and unburned tissue, and a decrease in cardiac output and vital organ perfusion. The amount of fluid lost is roughly a function of TBSA. The two classic and most commonly used burn resuscitation formulas are the modified Brooke formula (2 mL/kg/TBSA administered over 24 hours) and the Parkland formula (4 mL/kg/TBSA).[5,6] However, surveys through the ABA, the International Society for Burn Injuries (ISBI), and the European Burn Association (EBA) demonstrated wide variation in resuscitative techniques. The EBA survey revealed that 72% of burn units responding use either the original Parkland formula or some modification thereof.[7] Similarly, the ABA report showed that almost 70% of burn providers preferred the Parkland formula, followed by the Brooke (7%), Galveston (9%), and Warden hypertonic formulas (6%).[8] The complexity of current resuscitation formulas led Chung and colleagues[8] to develop a simplified technique for the initiation of fluid resuscitation (for adult patients only) termed the Institute of Surgical Research (ISR) rule of tens:

- Estimate TBSA burned to nearest 10%.
- Initial fluid rate (in mL/hr) equals TBSA times 10 (for adult patients with weight between 40 and 80 kg).
- In adults weighing more than 80 kg, increase rate by 100 mL/hr for every 10 kg above 80.

For example, in a 70 kg man with a 50% TBSA burn, the initial fluid resuscitation volume would be 500 mL/hr. Alternatively, in a 100 kg man with a similar 50% TBSA burn, the initial resuscitation volume would be 700 mL/hr. Using a computerized validation tool, these investigators showed that, in 88% of simulated patients, the initial resuscitative fluid rate using the ISR rule of tens fell between initial rates predicted by either the modified Brooke or Parkland formulas.[9]

Multiple studies have documented actual delivered fluid volumes far in excess of target volumes predicted by resuscitation formulas, a phenomenon termed fluid creep.[10] Several hypotheses have been proposed to explain this long-term trend, to include increased use of opioids.[11] It is unclear whether choice of resuscitation formula contributes to fluid creep because there are no randomized controlled trials of the Parkland versus Brooke formulas. However, Chung and colleagues[12] recently

reported that, when combat casualties were started on the modified Brooke formula, they actually received 3.8 mL/kg/%TBSA. When started on the Parkland formula, they actually received 5.9 mL/kg/%TBSA. Patients initially begun on the Parkland formula more often surpassed input of 250 mL/kg over 24 hours, a level associated with increased risk of abdominal compartment syndrome (ACS). However, in this study, this overshoot in fluid resuscitation did not result in different outcomes between the groups.

Monitoring

The various formulas only provide a starting point. Fluid input must be titrated hourly based on patient response. Attention to this detail improves outcomes. In combat casualties, Ennis and colleagues[13] showed that compliance with a paper flow sheet for documentation of hourly fluid input and output improved a combined endpoint of mortality and ACS. Urine output remains the indicator most providers use (95%) to titrate resuscitative fluids.[8] In adults, the goal for urine output is 30 to 50 mL/h (alternatively, 0.5–1.0 mL/kg/h); in children it is 1 to 2 mL/kg/h.[5] This is achieved by increasing or decreasing the fluid infusion rate by 20% to 30% every 1 to 2 hours.

The modern era provides an array of techniques for monitoring intravascular volume or organ perfusion. Such technologies were used by 23% of providers in addition to urine output to guide resuscitation in an ABA survey. These included the pulmonary artery catheter (8%), base deficit (7%), lactate (5%), lithium indicator dilution (5%), transpulmonary thermodilution (3%), and hematocrit (1%). Caution should be used in interpreting these results because overenthusiastic attempts to normalize intravascular volume or, worse, achieve a supranormal cardiac output during the first 24 hours postburn place the patient at risk of overresuscitation and compartment syndromes.

Salinas and colleagues[14] recently reported the development of a computerized decision support program that is currently used for resuscitation of all severely burned patients at the US Army Burn Center. The main function of the program is to provide a recommendation each hour for the lactated Ringer's infusion rate based on the trend in the urine output over the past 3 hours, the time postburn, and the patient's burn size. Compared with historical controls, use of this program resulted in a reduction in crystalloid volumes infused during the first 24 and 48 hours, and the urine output was more frequently within the target range. A prospective study is planned.

Fluid of Choice

The most commonly used resuscitative fluid is lactated Ringer's (91% of those surveyed). Almost half of burn providers supplement crystalloid resuscitation with some type of colloid, typically starting 12 to 24 hours postburn.[8] This timing reflects that, during the initial 8 to 12 hours postburn, the microvasculature is incapable of sieving proteins. Use of colloid before hour 8 to 12 hours postburn may be ineffective or, worse, enhance edema formation.

Albumin (5% in normal saline) is the most commonly used colloid. The modified Brooke formula provides the following dose calculation for 5% albumin to be given over 24 hours:

- 0% to 29% TBSA: no albumin is normally given
- 30% to 49% TBSA: 0.3 mL/kg/TBSA
- 50% to 69% TBSA: 0.4 mL/kg/TBSA
- 70% to 100% TBSA: 0.5 mL/kg/TBSA.

The crystalloid infusion rate is then titrated as before, anticipating that it will be possible to decrease it. Fresh frozen plasma has also been used for burn shock. In

one study, this practice resulted in fewer instances of elevated intraabdominal pressure.[15]

Adjuncts to Resuscitation

Preclinical data indicate that high-dose intravenous vitamin C reduces lipid peroxidation in the postburn period, ameliorates the increase in postburn vascular permeability, decreases resuscitative volume requirements, and reduces edema associated with thermal injury.[16] Tanaka and colleague's[17] single-center, prospective study in 37 patients admitted with burns greater than 30% TBSA revealed a significant reduction in resuscitative volume, weight gain, wound edema, and pulmonary dysfunction. The dose of vitamin C used in this study was 66 mg/kg/h, begun as rapidly as possible after injury. Although promising, these single-center results need further verification.

Therapeutic plasma exchange (TPE) has resurfaced as an adjunct for patients with refractory burn shock. TPE involves removal of blood from the patient via a large-bore intravenous catheter and separation of components. Plasma is collected and the remaining components are returned to the patient. The efficacy of TPE in inflammatory states is thought to be due to removal of large molecular weight proteins such as cytokines.[18,19]

Decompression

In burn patients, transvascular fluid flux during the first 48 hours postburn causes not only shock but also massive edema formation. Thus, the counterpart to fluid resuscitation in these patients is a decompressive strategy designed to minimize the effects of edema.[20] Circumferential or near-circumferential full-thickness burns involving the torso or extremities can result in a leather-like, noncompliant constrictive band. Progressive edema formation beneath the eschar then compresses underlying structures to include nerves, vessels, muscle, or lungs. This process is termed eschar syndrome. In the chest, it decreases thoracic compliance and may present as increased airway pressure, decreased tidal volume, respiratory acidosis, hypoxia, and, ultimately, cardiac arrest. Thoracic eschar syndrome is treated emergently at the bedside with escharotomy. Bilateral incisions are made through the eschar into underlying viable fat, from the midclavicular line, downwards along the anterior axillary line, and across the midline in the epigastric region (**Fig. 2**). An immediate improvement in compliance should be obvious. An analogous problem occurs in the extremities and is treated with extremity escharotomy (see later discussion).

With massive fluid resuscitation (eg, >250 mL/kg), ACS may develop. ACS requiring decompressive laparotomy is a highly lethal complication in this patient population. Every effort should be taken to anticipate and avoid it. The incidence of ACS in a review at the US Army Burn Center was 1%, with a mortality of 90% (18/20).[21] Latenser and colleagues[22] described a 9-patient pilot study of the use of percutaneous drainage for the treatment of intraabdominal hypertension (bladder pressure >25 mm Hg) in burns. They found that catheter drainage resulted in successful amelioration of the process and prevented progression in five patients. In a recent survey of burn physicians on the subject of ACS, 34% of respondents advocated percutaneous catheter decompression before decompressive laparotomy for ACS.[23]

Vulnerable Organs

Initial care of burn patients is focused, appropriately, on sustaining life. Nevertheless, failure to attend to certain burn-specific vulnerable organs throughout the

resuscitation and intensive care course may result in lasting injury. These vulnerable organs include the extremities (the hands especially) and the eyes.

Several factors combine to place burn patients at risk for permanent extremity injury or loss. The most obvious risk is that of the extremity eschar syndrome, which develops during the first 48 hours postburn. In circumferential deep burns of an extremity, edema formation in the soft tissue beneath the inelastic burned skin (eschar) elevates internal pressure within the limb, constricting venous outflow and ultimately arterial inflow. Elevation of the burned extremities reduces the transvascular pressure experienced by the microvasculature during a period of increased permeability, and is essential to decreasing the risk of this syndrome.

Extremity eschar syndrome may be manifested by distal cyanosis (if the fingertips are unburned), numbness, tingling, and other signs and symptoms of vascular compromise. The progressive diminution or loss of distal pulses, which should be monitored hourly by Doppler flowmetry, is the classic indication for escharotomy. In the right clinical setting (ie, circumferential full thickness burns of an extremity) an experienced surgeon may perform escharotomy before a change in peripheral pulses. Escharotomy is commonly performed at the bedside under semisterile conditions using a scalpel and or electrocautery to incise the eschar along the midmedial and midlateral joint lines. Care must be taken to incise all circumferential eschar, to achieve good hemostasis, to incise all the way through the eschar but to stay out of the viable tissue beneath it, and to document pulse restoration (**Fig. 3**).

If the hand is burned, and if limb escharotomies do not restore pulsatile Doppler flow to the palmar arch and digital arteries, then additional hand and finger escharotomies may be required. Dorsal hand escharotomies are performed over the location of the dorsal interossei (between the metacarpals). Finger escharotomies are performed on the radial aspect of the thumb and on the ulnar aspect of the other digits, using care to stay between the extensor mechanism and the neurovascular bundle.

Extremity eschar syndrome must be distinguished from extremity compartment syndrome. The authors use the latter term to refer to the process whereby pressure

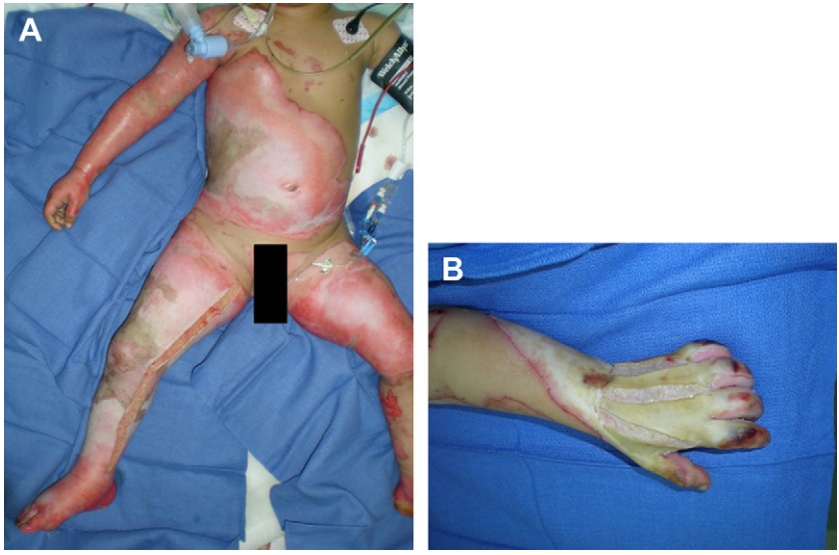

Fig. 3. (*A, B*) Escharotomies.

within the investing fascia of an extremity causes vascular compromise and neuromuscular damage. Common causes of compartment syndrome include vascular injury and repair, crush injury, or fracture. In a burn patient, compartment syndrome may also result from a delay in escharotomy, leading to ischemia-reperfusion injury; from direct muscle injury (eg, from high-voltage electricity or blast injury); or from massive fluid resuscitation and anasarca. Regardless of the cause, recognize that the treatment of eschar syndrome is escharotomy, whereas the treatment of compartment syndrome is fasciotomy. Performing a prophylactic fasciotomy on a patient who requires only an escharotomy exposes uninjured muscle to microbial contamination. Equally, failure to diagnose compartment syndrome in a burn patient places the limb at risk. When diagnosis is delayed, compartment syndrome may present as sepsis with dead, infected muscle anywhere from approximately 12 days to 2 months after injury.

Following successful resuscitation, burned hands remain at high risk until successful wound closure and rehabilitation have been achieved. This is a function of depth of injury. Ninety-seven percent of patients with superficial hand burns had normal function at discharge compared with 81% of those who required surgery for deep dermal or full thickness injuries. Furthermore, only 9% of patients with injury to the extensor mechanism, joint capsule, or bone had a normal functional outcome.[24]

The above concepts are well-recognized problems in burn care. Less well documented are postburn peripheral nerve injuries. These may be manifested by weakness, numbness, and/or tingling. In a prospective study, symptomatic patients underwent nerve conduction studies, and peripheral neuropathy was diagnosed in 10%. The most commonly involved nerve was the median sensory nerve, followed by the ulnar sensory nerve. All patients but one had sensory and motor involvement of at least two nerves.[25] Risk factors for neuropathy in another study included ICU days, history of alcohol abuse, age, and electric injury. Attention to eschar and compartment syndromes, careful positioning and splinting, avoidance of tight dressings, and detailed neurologic examination are keys to prevention and early detection of peripheral neuropathy in burn patients.

It seems likely that burn patients, like ICU patients generally, are at risk for critical illness polyneuropathy (CIP) and critical illness myopathy (CIM). CIP is a distal axonal sensory-motor polyneuropathy affecting the limbs and phrenic nerve. CIM is a primary myopathy not secondary to denervation. CIP and CIM often coexist. Both may present as extremity weakness, difficulty weaning from the ventilator, and months to years of disability.[26] The pathophysiology of these syndromes is not fully understood.

Thermally injured patients are particularly vulnerable to ocular injury throughout their ICU course. In one study, one-quarter of patients with facial burns, TBSA greater than 20%, and/or inhalation injury had ocular complications. Patients receiving mechanical ventilation, with wound infections, and with decreased Glasgow Coma Scale score were at particular risk.[27] Accordingly, all patients with periorbital burns should undergo Wood's lamp examination on admission to rule out corneal abrasions. Positive or doubtful results merit immediate ophthalmologic consultation. Failure to treat corneal abrasion aggressively may lead to corneal ulceration, perforation, and blindness. Amniotic membrane transplantation is one technique available to treat significant corneal injury. For most patients, aggressive treatment with topical antibiotics and daily follow-up by the ophthalmologist is effective.

Like abdominal and extremity compartment syndromes, orbital compartment syndrome (OCS) is increasingly recognized in thermally injured patients who receive large fluid resuscitations. If untreated, OCS can cause blindness. Based on retrospective data, the intraocular pressure (IOP) should be measured daily using a portable

tonometer for the first 2 to 3 days postburn, particularly in patients whose 24-hour fluid resuscitation volume exceeds 5.5 mL/kg/% burn. When the IOP is found to be elevated (ie, above 30 mm Hg), orbital release by lateral canthotomy and cantholysis should be considered.[28]

Over time, deeply burned eyelids may scar and contract open, leading to extrinsic ectropion, conjunctivitis, and exposure of the corneas. When this occurs, secondary keratitis again places the corneas at risk. One approach to this problem is tarsorrhaphy. Because this procedure does not correct the underlying scarring process, tarsorrhaphy often fails, damaging the tarsal plates in the process. For this reason, many investigators consider tarsorrhaphy to be contraindicated in this setting.[29] Instead, release of deep eyelid burns should be considered when the patient can no longer protect the corneas. Moisture goggles help protect the corneas until this operation can be performed.

CRITICAL CARE OF THE BURN PATIENT

The Burn ICU

Both the environment of care and a team approach are exceedingly important for successful outcomes in burns. Three characteristics make the burn ICU environment different from other ICUs: infection control, temperature control, and hydrotherapy. Burn centers were the original research institutes for infection control. Individual isolation rooms; rigorous hand washing; personal protective gear such as gowns, gloves, masks, hats, and shoe covers; microbial surveillance; and antibiotic stewardship constituted the infection-control bundle enacted at the US Army Burn Center ICU in 1983, which was associated with eradication of pandemic multiple-drug resistant organisms. Other units have reduced cross-contamination with bacteria-controlled nursing units (BCNUs), which further isolate the patient within a laminar airflow chamber with plastic walls.[30] The importance of housekeeping and the quality of the physical plant in preventing infection cannot be ignored.

Another essential feature of the burn center environment of care is temperature control. Because one of the functions of the skin is to act as a barrier against heat loss, and because injury redirects blood flow to the wound surface, patients with extensive burns are at risk of hypothermia. Even when hypothermia is not overtly recognized as a decrease in body temperature, a normal room temperature increases a burn patient's metabolic rate through the process of nonshivering thermogenesis. This adds to the patient's already hypermetabolic, hypercatabolic state. In the operating room (OR), burn patients are at high risk of hypothermia for several reasons:

- Exposure of multiple wounds
- Significant blood loss and fluid requirements
- Impairment of peripheral vasoconstriction by anesthetic agents.

The main solution to the hypothermia problem is to elevate the room temperature to suit the patient's needs rather than the providers' comfort. This means an ICU room temperature of 85°F and an OR temperature of 90° to 95°F.

A third essential feature is a dedicated tank or shower facility for hydrotherapy. Eighty-three percent of North American burn centers report such a capability. Because hydrotherapy tanks are a potential locus for transmission of nosocomial organisms, they are less common today. Instead, patients can be showered on a special shower cart. Either way, hydrotherapy is widely used to facilitate wound care.[31]

The most important aspect of burn ICU care, however, is not the physical plant but the team approach to care. Just as burns are among the most lethal and disfiguring

injuries known to humans, the coordinated efforts of multiple disciplines are required to achieve functional survival. These disciplines include surgery, nursing, rehabilitation, respiratory therapy, nutrition care, psychology, and social work. Multidisciplinary team rounds should be conducted daily. Success is a function not of any one individual, but of an effective burn team. The surgeon's key role is to lead this team by developing and communicating an overall management strategy based on a complete head-to-toe evaluation of the burn patient on admission to the ICU and frequently thereafter.

Pain and Anxiety

Burns and burn treatment is painful. Pain generates anticipatory anxiety that amplifies the experience of pain. Pain makes it difficult for even experienced personnel to perform thorough wound care and rehabilitation. Poorly controlled pain and anxiety may contribute to long-term psychological sequelae. The cornerstone of initial pain management is frequent, small doses of an intravenous narcotic. Opiates, benzodiazepines, and ketamine are useful in procedural sedation but must be carefully monitored. Nonpharmacologic approaches to management of pain and anxiety are an important part of burn care. Available techniques include hypnosis, immersive virtual reality, and music therapy. Anxiety should be specifically managed and benzodiazepines are the pharmacologic tools most frequently used. Dexmedetomidine may play an important role in these procedures in coming years.[32]

Pulmonary

Inhalation injury consists of three processes that can coexist in any single patient: upper airway injury, subglottic injury, and chemical asphyxiant inhalation. Injury to the upper airway—lips, tongue, pharynx, and larynx—causes edema and may cause precipitous airway obstruction during the first 48 hours postburn. Patients presenting with symptomatic inhalation injury should be prophylactically intubated, particularly before interhospital transfer. The same is true of patients with TBSA of approximately 40% or more, even in the absence of inhalation injury, because edema during resuscitation can cause airway obstruction.

Once the patient has been intubated, continuous attention to the security and patency of the endotracheal tube is critical. Adhesive tape does not stick to a burned face and should not be used. Security is ensured by tying the tube using umbilical tape (cotton ties) circumferentially around the head. As the face swells during resuscitation, repositioning of the ties may be needed to ensure that the endotracheal tube remains at the correct distance from the carina. Likewise, the face should be protected from and inspected for lesions that can be caused by the ties. The tube can alternatively be wired to the upper incisors using an arch bar.[29] Patency is achieved by frequent suctioning and by the use of nebulized heparin (5000 units in 5 mL NS every 4 hours, or 10,000 units every 6 hours) to prevent the formation of endoluminal clots and casts in patients with inhalation injury. Early detection of endotracheal tube obstruction is facilitated by end-tidal carbon dioxide monitoring. Fiberoptic bronchoscopy is performed on admission to diagnose inhalation injury and is repeated as needed to perform pulmonary toilet.

Early tracheostomy (within a few days of injury) may facilitate pulmonary toilet in patients with severe inhalation injury and copious cast formation. The larger, shorter, easily replaceable airway provided by the tracheostomy may be lifesaving. In other patients, tracheostomy is often performed after 21 days of endotracheal intubation. It is still unclear, however, whether tracheostomy or prolonged translaryngeal intubation is preferable in burn patients.[33] In one retrospective study, translaryngeal

endotracheal intubation was safe and effective in thermally injured children for up to 3 months.[34] Increasingly, chronic pathologic conditions following prolonged intubation or tracheostomy in burn patients have been documented. These problems include dysphagia; dysphonia; granulation tissue; vocal cord paresis, fixation, or fusion; arytenoid dislocation; and bronchial, tracheal, or subglottic stenosis.[35]

The second form of inhalation injury is injury to the subglottic airways and pulmonary parenchyma, mediated by toxic gases and particulate matter. Distinct from other causes of acute lung injury, smoke inhalation injury attacks the small airways more so than the alveolar-capillary membrane.[36] Major mechanisms active in inhalation injury include oxidative[37] and nitrosative stress[38]; activation of coagulation and inhibition of fibrinolysis[39,40]; increased bronchial blood flow[41]; hypersecretion of mucus[42]; bronchiolar obstruction[43]; and ventilation-perfusion mismatch. Therapies directed at maintaining small airway patency are, therefore, key to the treatment of patients with this injury. Examples include inhaled heparin (see previous discussion) and high-frequency percussive ventilation (Volumetric Diffusive Respiration [VDR], Percussionaire, Sandpoint, Idaho, USA). A randomized controlled trial of VDR versus low-tidal-volume ventilation according to the ARDSnet algorithm—in burn patients with or without inhalation injury—demonstrated a higher rescue need (ie, transition to another mode due to failure to meet ventilation and oxygenation goals) for the ARDSnet group.[44] Inhaled beta agonists, such as albuterol, are routinely given to patients with inhalation injury to prevent bronchoconstriction. A recent preclinical study demonstrated that inhaled epinephrine improves pulmonary function after inhalation injury by reducing airway blood flow (and, in turn, airway edema, mucus secretion, inflammation, and ventilation-perfusion mismatch).[45]

The third form of inhalation injury is systemic toxicity caused by the absorption into the blood of the chemical asphyxiants present in smoke. Carbon monoxide (CO) binds to hemoglobin more avidly than does oxygen, forming carboxyhemoglobin (COHb). This has two deleterious consequences for oxygen delivery: the hemoglobin binding sites occupied by CO are not available to carry oxygen, causing a relative anemia; and CO binding alters the hemoglobin such that the oxygen dissociation curve is shifted toward the left. CO also binds to the terminal cytochrome oxidase on the mitochondrial electron transport chain.[46] This causes impaired cellular respiration and oxidative stress. Oxidative stress and inflammation also result from several other pathways.[47] Symptoms of CO toxicity range from mild (eg, headache, nausea) to severe (eg, coma, myocardial infarction, death). Treatment of CO poisoning consists of 100% oxygen until the COHb is less than 5%.[48] Because 100% oxygen decreases the half life of COHb from approximately4 hours to approximately1 hour, use of hyperbaric oxygen (HBO) to further accelerate CO elimination–particularly in unstable burn patients–may be impractical. However, a randomized controlled trial demonstrated that HBO (within 24 hrs of end of exposure) prevented delayed neurologic sequelae of CO,[49] likely by mechanisms other than hemoglobin binding.

Cyanide (CN) also binds to the terminal cytochrome oxidase, albeit at a different site than CO. By interfering with the cell's ability to use oxygen, CN produces rapid cardiovascular collapse and unconsciousness.[50] The prevalence of CN poisoning in patients with smoke inhalation injury is debated; the diagnosis is made difficult by the lack of a rapid assay, but it can be suspected in patients with lactic acidosis on initial presentation out of proportion to the burn size. Several antidotes are available for CN poisoning. Amyl and sodium nitrite convert hemoglobin to methemoglobin, which chelates CN. This treatment is risky, however, because methemoglobin is incapable of carrying oxygen and causes vasodilatation. Recently, high-dose hydroxocobalamin (Cyanokit) has become available for the treatment of CN poisoning; it also chelates

CN without the side effects of the nitrites.[50] Finally, sodium thiosulfate serves as a sulfur donor for hepatic rhodanase, which converts CN to thiocyanate. It has a slower onset of action than the other drugs.

Renal, Fluid, and Electrolyte Issues After Resuscitation

Successful resuscitation of a patient with burn shock is signaled by a sustained decrease in the fluid infusion rate required to maintain a urine output in the target range to maintenance levels, and usually occurs by 48 hours postburn. Other indicators of successful resuscitation include hemodynamic stability, resolution of lactic acidosis, and normalization of the base deficit. Patients can then be expected to offload the large amounts of resuscitation fluids during the ensuing 10 days or so. Assessment of volume status and of the adequacy of end-organ perfusion can be challenging in these patients, whose burn-induced hypermetabolic state and systemic inflammatory response syndrome drives an elevated heart rate, increased cardiac output, and decreased systemic vascular resistance even in the absence of any infection or other complications. When volume status is in doubt, measurement of the base deficit, lactate, central venous pressure, central venous saturation of oxygen, stroke-volume variability and, most important,of the response to a therapeutic intervention, such as a bolus of fluid, are diagnostically useful.

Frequent (eg, twice daily) measurement of serum electrolytes, including calcium, magnesium, and phosphate, is important during the management of critically ill burn patients. The most striking feature of fluid and electrolyte balance in these patients after resuscitation is evaporative water loss, which will result in hypernatremia if untreated. Such water loss is proportional to the open wound size. Water losses during a 24-hour period can be estimated as 1 mL/kg/(open burn size,%). This provides an estimate of water requirements. Water intake can be provided enterally (as a component of tube feedings or as additional water flushes), and/or intravenously (as 5% dextrose in water [D5W] or D5W in half-normal saline). Water intake is then adjusted based on daily or twice daily measurements of the serum sodium level. With wound healing, the open wound size gradually decreases and with it the daily water requirement. Burn patients are also at risk for hyponatremia, particularly if allowed to drink freely. Hypophosphatemia requiring intravenous replacement is common in patients with major thermal injury, particularly during days 3 to 5 postburn. Potassium and magnesium levels should be checked daily and replaced as needed.

Acute kidney injury (AKI) classified according to Acute Kidney Injury Network (AKIN) or Risk, Injury, Failure, Loss, End-Stage (RIFLE) criteria, is fairly common in burn patients. Using the RIFLE criteria, AKI occurred in 27% of 304 patients with burns equal to or greater than 10% TBSA.[51] Risk factors for AKI in this study included inhalation injury and sepsis. Failure (RIFLE, F) was an independent predictor of mortality. To address this problem, Chung and colleagues[52] implemented a continuous renal replacement therapy program for adult burn patients at the US Army Burn Center. This program is managed by intensivists and operated by specially trained ICU nurses. Patients undergo continuous venovenous hemofiltration (CVVH) if they meet AKIN 2 (with shock) or AKIN 3 criteria. Compared with historical controls, CVVH decreased mortality and pressor requirements. A multicenter study of CVVH for this patient population is underway.

Gastrointestinal

Abdominal complications occurred in 7% of patients admitted to one burn center and are more likely with increasing burn size.[21] ACS is a feared intraabdominal catastrophe, the avoidance of which is a principal goal during the initial fluid resuscitation

of burn patients; however, it can also occur at other times during the ICU course, for example following massive perioperative resuscitation.[21]

Another cause of intraabdominal catastrophe in thermally injured patients is nonocclusive mesenteric ischemia (NOMI). This highly lethal complication (mortality approximately 70%) presents as necrosis of variable portions of the small and/or large bowel.[21] The onset of NOMI is usually later in the course than ACS, although some ACS patients also have NOMI at laparotomy. The cause of NOMI in burn patients is unknown. Retrospective efforts to relate it to pressor use, massive burn wound excision, and/or hyperosmolar enteral feeding have been inconclusive. Diagnosis is often made by CT scan, but diagnostic peritoneal lavage can be used in the unstable patient.

Whereas stress gastroduodenal ulceration in burn patients (Curling's ulcer) is now uncommon, it is important to recall that thermal injury causes a dose-related ulcer diathesis with evidence of mucosal injury (in untreated patients) within 12 hours of injury. In the era before antacids, H_2 blockers, and proton-pump inhibitors, this led to laparotomy for control of hemorrhage or perforation in a significant number of patients, highlighting the importance of prevention in this patient population.

Impaired bowel function is both a marker of critical illness and a frequent consequence of narcotic administration in burn patients. Critically ill burn patients are also at risk of pancreatitis. Forty percent of patients with burns greater than 20% TBSA had hyperamylasemia and or hyperlipasemia in one review, of whom 82% had symptoms of pancreatitis.[53] The burn patient with elevated cholestatic enzymes and evidence of infection merits evaluation for acute cholecystitis, which may be successfully treated with percutaneous cholecystostomy in many cases.

Nutrition and Metabolism

No population has greater nutritional requirements than thermally injured patients, whose metabolic rate may increase to over twice normal. As a rule of thumb, patients cannot meet their nutritional requirements orally if their total burn size is equal to or greater than 30%, and enteral tube feeding should be initiated as soon as hemodynamic stability is achieved. In a review of critically ill burn patients enrolled in the Inflammation and the Host Response to Injury (Glue Grant) study, enteral feeding was begun within 24 hours of burn in 80% of patients, with no increase in complications and a shorter ICU stay.[54] Whenever possible, the enteral route is preferred, but some centers cautiously use partial parenteral nutrition to make up the difference in patients unable to tolerate full enteral nutrition.[55] Malnutrition is a real risk: 61% of children, with burn size equal to or greater than 20% and chronically open wounds transferred to a burn center between 3 and 24 weeks postburn, were classified as malnourished.[56]

A variety of formulas are in use to estimate the caloric requirements of burn patients. These requirements are proportional to the burn size and decrease over time as healing occurs. Because of differences in healing rate, infections, or other factors, this formula becomes inaccurate at a month postburn, and we perform metabolic cart studies (indirect calorimetry) to measure the resting energy expenditure (REE). Even at discharge and despite successful wound closure, the measured energy expenditure remains elevated in patients with large burns.[57] This phenomenon persists for up to 3 years postburn and is accompanied by elevations in cortisol, catecholamines, cytokines, and acute phase proteins.[58]

Nitrogen requirements are likewise elevated. Skeletal muscle breakdown in patients with large burns provides a pool of amino acids that the rest of the body uses for wound healing, acute phase protein synthesis, and gluconeogenesis.[59] It stands to

reason, therefore, that enteral protein intake should be augmented in these patients. A high-protein enteral feeding formula was shown to improve outcomes in thermally injured children, including decreased bacteremia and increased survival. Therefore, high-protein nutrition is routinely provided to burn patients (ie, 1.5–2.0 g/kg/d for adults and 2.5–3.0 g/kg/d for children). Also, a lower nonprotein calorie-to-nitrogen formula should be used (ie, 100 kcal/g or lower). Visceral protein levels including prealbumin, retinol-binding protein, and transferrin correlate weakly with nitrogen balance. We measure urine urea nitrogen levels and estimate nitrogen balance weekly. Even when a positive nitrogen balance is maintained, proteolysis continues throughout the hospital stay. With wound closure, proteolysis decreases.[59] However, total protein turnover is increased compared with normal children even at discharge, reflecting the persistent hypermetabolic state.[60]

Glutamine, a conditionally essential amino acid, is greatly decreased in burn patients. This may contribute to immune failure and intestinal mucosal atrophy. Intravenous glutamine supplementation has been associated with decreased bacteremia, improved measures of nutrition, and decreased measures of inflammation. Pending larger studies, we provide glutamine supplementation to critically ill burn patients.

Trace elements such as copper, selenium, and zinc are depleted in burn patients.[61] This may impair wound healing and, because selenium is a component of glutathione peroxidase, may degrade the intracellular antioxidant system.[62] We routinely replace these elements in critically ill patients and await outcome results of this treatment strategy.

Stress-induced hyperglycemia is common in thermally injured patients. A tight glucose-control strategy improved outcomes in burned children.[63] There are multiple therapeutic effects of insulin beyond glucose control, including a reduction in inflammation,[64] an improvement in wound healing,[65] and maintenance of lean body mass (see later discussion). The ideal glucose target in critically ill patients is controversial.[66] We currently use intravenous insulin to achieve a target glucose level of 100 to 150 mg/dL and have found a computerized decision support system useful in dosing continuous insulin infusions.[67]

Patients with major thermal injury are catabolic for the duration of their hospital stay and beyond, resulting in significant loss of lean body mass despite adequate replacement of calories, nitrogen, and other nutrients. This phenomenon has been addressed in various ways. Unfortunately, feeding more calories than 1.2 times REE leads to fat deposition instead of than lean body mass accretion.[68] An anabolic steroid, oxandrolone, maintained net protein balance and lean body mass in severely burned children, with increased gene expression for functional muscle proteins.[69] Oral propranolol (targeted to achieve a 20% decrease in the heart rate) decreased REE while increasing net muscle protein balance in burned children.[70] Intraarterial insulin infusion resulting in extremity hyperinsulinemia caused an increase in muscle protein synthesis but did not affect proteolysis.[71] Based on these data, we routinely administer oxandrolone and propranolol to our patients with severe thermal injury, but studies of propranolol efficacy in adult burn patients are still needed. The desire to achieve the anabolic effects seen with higher doses of insulin without the risk of hypoglycemia has pointed researchers to metformin, fenofibrate, and related agents.[72,73]

BURN-SPECIFIC MANAGEMENT

Wound Care: Topical Antimicrobials

Initial wound care should be performed as soon as the patient is hemodynamically stable. Most burn centers have dedicated wound care rooms for this purpose. The

OR is also appropriate. Use of analgesics and anesthetics must be carefully managed because patients with total burn size equal to or greater than 20% are likely to be hypovolemic during this period. The principles of wound care include total exposure of the patient, aggressive debridement of all nonviable tissue, and thorough cleansing with a surgical skin antiseptic. The solution of choice for this is chlorhexidine gluconate (except for infants and on the face).

The method of topical care developed at the US Army Burn Center is twice-daily cleansing with chlorhexidine gluconate (including removal of all previously applied creams), followed by application of an opaque layer of mafenide acetate (Sulfamylon) cream in the morning, and of silver sulfadiazine in the evening. This method maximizes the advantages and minimizes the disadvantages of either drug used alone. 0.5% silver nitrate solution (aqueous) may be used instead of burn creams. Patients are dressed in 8-ply gauze dressings and the solution is applied to the dressings once every 2 hours.[74]

Several dressings now exist that provide alternatives to the original antimicrobial agents. Careful patient selection and understanding of the products are essential for effective use. Biobrane (Mylan, Canonsburg, PA, USA) is a bilaminar artificial material composed of an inner collagen-impregnated polyethylene mesh (dermal equivalent) and a perforated silicone layer (epidermal equivalent). We use it in patients with new ($\leq$48 hours), clean, superficial partial thickness burns such as scalds. After the Biobrane is applied, it is kept covered with gauze for 24 hours and then inspected. Accumulation of pus under the material indicates need for immediate removal and conversion to topical antimicrobials. Otherwise, it is left in place until the wound is healed. In the right patients, this material reduces hospital days, healing times, and pain. It should be noted that Biobrane contains no antimicrobial agent. Instead, any method of wound closure, if successful, acts per se to decrease bacterial burden on a wound surface.

The other major class of new dressings provides silver in a variety of slow-release formats (eg, Silverlon [Argentum Medical, Geneva, IL, USA] and Acticoat [Smith & nephew, St Petersburg, FL, USA]). An advantage of such dressings is that they can be left in place for up to 7 days with sustained release of silver cations onto the wound surface. However, safe use for such a period of time without wound inspection presumes a low risk of an adverse outcome from an infection, should one occur. Thus, we prefer to use silver dressings mainly in patients with deeper burns (who thus are not candidates for Biobrane), but whose wounds are clean and of limited size. Newer silver dressings have been engineered to enhance the local wound healing environment. One such dressing is Mepilex Ag (Molnlycke Health Care, Norcross, GA, USA), a soft silicone foam dressing that is changed every 3 to 5 days initially and then every 5 to 7 days. Compared with silver sulfadiazine, this product was associated with less pain, fewer dressing changes, and no difference in healing.[75] A Cochrane review identified a paucity of data on the treatment of burns with silver preparations, indicating the need for more well-designed studies.[76]

Wound Care: Surgery

Rapid and lasting wound closure is the main effort in the care of burn patients. The time to closure of full-thickness burns was an independent predictor of survival in a 1983 study in which standardized mortality rates differed by a factor of two among burn centers.[77] In children with burn size equal to or greater than 40%, delays in excision and grafting of the burn wound were associated with longer length of stay, delayed wound closure, and increased rates of invasive wound infection and sepsis.[78] In the authors' experience, patients with larger burns are at greater risk of failure to take skin grafts and to heal (so-called wound failure). The explanation for this is no doubt multifactorial.

In view of this, the authors' goal for patients with major thermal injuries is to excise all areas of full thickness burn (and those areas of deep partial thickness burn judged too deep to heal within 21 days of injury) as soon as hemodynamic stability can be achieved, normally within 2 to 3 days of injury. For patients with TBSA less than 50%, donor sites are available such that the excised burns can be closed with the patient's own skin (autograft). For patients with larger burns, cadaver allograft is used to make up the difference until the donor sites have healed and can be reharvested. Allograft can be used to cover excised wound beds, or it can be applied over widely meshed (eg, 3:1 or 4:1) autograft in a sandwich technique. Close postoperative attention to the graft sites by an experienced surgeon is essential to gauge the success of surgery and to preemptively address areas of wound infection.

Given the importance of wound closure, several technologies have emerged to provide more skin for the patient with massive wounds. None of these is a panacea and they should only be used in burn centers with a multidisciplinary commitment to their safe use. Cultured autologous epithelium consists of keratinocytes grown in tissue culture from small skin biopsies. We perform such cultures for patients with massive burns ($\geq$80% TBSA) and thus limited donor sites. We typically achieve closure of a limited (5%–10%) portion of the body, anticipating that many patients will experience delayed loss of the cells even if initial engraftment is achieved.

Integra (Integra LifeSciences, Plainsboro, NJ, USA) is a bilaminar dermal regeneration matrix. The dermal layer is made of a matrix of cross-linked collagen and glycosaminoglycan. The temporary epidermal layer is made of silicone. About 14 to 21 days after application to the wound, the dermal layer engrafts, the patient is taken back to the OR, the outer layer is peeled off, and a thin autograft (0.04–0.08 in) is grafted onto the dermal layer. Ryan and colleagues[79] documented a decreased length of stay in high-risk burn patients, possibly related to more rapid wound closure. As of yet, a mortality benefit for Integra has not been demonstrated. In a postapproval multicenter study, the infection rate was 16% (13% superficial and 3% invasive)[80]; this risk should be considered when using Integra.

StrataGraft (Stratatech Corporation, Madison, WI, USA) was developed as a substitute for cadaver allograft. It consists of an epidermal layer of keratinocytes from a single human donor, grown on a collagen matrix embedded with fibroblasts from a second human donor. ReCell (Avita Medical, Northridge, CA, USA) is a technology whereby a 2 cm by 2 cm autograft is processed in the OR to yield a suspension of noncultured autologous epithelial cells. This cell suspension is then immediately sprayed onto the wound.[81] Clinical trials of these products are underway.

Infection

Immune failure, open wounds, and invasive devices place burn patients at high risk of infection. Infections are a leading cause of death in burn patients, but the location of these infections and the causative organisms have gradually shifted. In a recent autopsy series, cause of death included infection in 61%. Common organisms involved were true fungi, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae*. Of lesser importance were gram-positive organisms, to include methicillin-resistant *Staphylococcus aureus*, and the multiple-drug resistant but relatively indolent *Acinetobacter baumanni* complex. The location of these infections was predominantly wound and lung.[82] Even when infection has been eliminated, systemic inflammation—induced by tissue injury and multiple infectious episodes—may cause death by multisystem organ failure.

Before the discovery of effective topical antimicrobial agents, patients commonly died of invasive gram-negative burn wound infection.[83] Currently, invasive infection by true fungi (eg, *Aspergillus, Fusarium,* and *Mucor*) is more common than bacterial

invasion. Diagnosis of invasive burn wound infection is made by inspection of the wound and by histopathology. In a patient with clinical evidence of sepsis, changes in the color of the wound (tinctorial changes) may suggest infection and merit biopsy. Patients with smaller burns are at risk of burn wound cellulitis, which is manifested by erythema spreading more than a centimeter from the wound margin. *Streptococcus* is the most common causative organism but, because some of these patients are infected with methicillin-resistant *S aureus*, we often initiate treatment with intravenous vancomycin.

Intubated burn patients are at high risk for pneumonia. The presence of inhalation injury and the severity thereof increase the risk of pneumonia. Bundles to prevent ventilator-associated pneumonia are appropriate for these patients.[33] Animal studies confirmed that smoke inhalation injury predisposes to pneumonia by several synergistic mechanisms. Small airway damage and obstruction lead to distal atelectasis, colonization, and infection. Damage to the mucociliary apparatus and deleterious effects on immune function interfere with host response. Thus, it is unclear whether pneumonia in intubated patients with inhalation injury should be termed ventilator-associated pneumonia or inhalation-injury-associated pneumonia. High-frequency percussive VDR was associated with a decrease in pneumonia in patients with inhalation injury. One likely mechanism for this benefit is enhanced clearance of material from the distal airways. No therapy, however, is superior to weaning and extubation, which requires a multidisciplinary approach to daily sedation breaks, spontaneous breathing trials, and physical therapy of intubated patients.

Because burns in excess of about 20% TBSA induce a systemic inflammatory response syndrome—manifested by fever, tachycardia, increased cardiac output, elevated white blood cell count, decreased peripheral vascular resistance—the diagnosis of infection in these patients may be particularly difficult.[84,85] A high index of suspicion and attention to nontraditional indicators of sepsis is required. A consensus conference sponsored by the ABA proposed that the following indicators be used as triggers for a search for infection[86]:

- Temperature (>39° or <36.5°C)
- Progressive tachycardia
- Progressive tachypnea
- Thrombocytopenia
- Hyperglycemia (or insulin resistance)
- Enteral feeding intolerance (distension, high residuals, or diarrhea).

Prospective validation of these indicators is needed.[85] In the presence of clinical evidence of sepsis, we usually initiate broad-spectrum antibiotics while aggressively searching for a source. On the other hand, because burn patients have protracted ICU lengths of stay (1–2 days per percent burn, on average) and multiple bouts of infection, indiscriminant use of antibiotics only exerts pressure for the development of multiple-drug resistant organisms, without improving outcome: making judicious use of antibiotics imperative.

Rehabilitation

Although rehabilitation has been an integral part of the care of burn patients for decades, the scientific understanding of its impact on pathophysiology and on outcomes is in its infancy. The magnitude of the burn rehabilitation problem is indicated by a meta-analysis that showed a return-to-work rate of only 72% in previously employed burn patients with a mean burn size of 18%.[87] Similarly, 67% of surviving combat casualties returned to duty, whereas the remaining 33% were medically

discharged.[88] Rehabilitation is increasingly recognized not as a phase of care that transpires after wound healing has been completed but as an integral part of all phases of care—from resuscitation, through ICU care, to reintegration. The priorities and the time investment by the therapy team change during these phases, but the hospital course of a burn patient is simply too long to postpone rehabilitation. Furthermore, the authors propose not only that rehabilitation be integrated into ICU care, but also that it be re-envisioned as a way to change ICU outcomes such as ventilator days.

Burn patients should be evaluated by a burn therapist within 24 hours of admission.[89] Early priorities in rehabilitation are to preserve fine motor function and gross motor strength, to facilitate wound healing, and to counteract scar contracture formation. During resuscitation, the therapist evaluates the patient for functional deficits and elevates the upper extremities to decrease edema formation. While in the ICU, the therapist works with the other team members to combat the deleterious effects of prolonged bed rest, CIP-CIM, and mechanical ventilation. The feasibility of physical therapy despite endotracheal intubation to include sitting, standing, and ambulation, has recently been demonstrated in a prospective trial in medical ICUs.[90] In burn patients unable to transition rapidly to standing, devices such as the tilt table and the Moveo exercise platform (Chattanooga, Inc, Vista, CA, USA) are commonly used.

As healing progresses, contracture formation threatens the patient's mobility, particularly across the joints. Range-of-motion exercises, splinting, and positioning are used to counteract this process. Following excision and grafting of the burn wound, splinting is performed to maximize graft take, followed by gradual mobilization beginning about 5 days postburn. As the patient transitions from the ICU to the ward, he or she becomes able to participate in physical and occupational therapy in a clinic setting and relearns the activities of daily living.

OUTCOMES

A systematic approach to burn care by multidisciplinary teams has resulted in significant improvements in survival and functional recovery from burn injuries. Overall mortality relates to the age of the patient, the size of burn, and the number of underlying comorbidities. The National Burn Repository (2007) indicates a 4.35% mortality rate for women and a 4.72% mortality rate for men.[91] In multiple studies, rural locales that refer patients to a burn center,[92] urban areas with significant burn resources,[93] and international burn care centers[94] all demonstrate significant improvements in burn outcomes over time. As survival increases, emphasis has been appropriately placed on measures of functional outcome.[95]

INJURIES DUE TO HEAT AND COLD

Injuries due to heat and cold can be primary presenting problems or can complicate the management of patients suffering blunt or penetrating injuries. The later is particularly common in patients injured in disaster scenarios or combat operations where prompt access to medical care is commonly impossible. Local and systemic manifestations of heat and cold exposure can occur together or separately. The systemic manifestations can be quite subtle and dangerous and should be considered in all casualties in whom a delay in initial care has occurred.

Heat Injury

The local effect of heat is thermal injury (see previous discussion). The systemic effects constitute a spectrum of conditions called heat illness. Heat illness is a graded

elevation of core body temperature due to a failure of normal thermoregulation.[96] It is different from fever, which is a centrally regulated response. Body temperature is tightly controlled in mammals because so many metabolic reactions are susceptible to variations in temperature. Mammals have two major categories of cooling mechanisms: autonomic and behavioral. Humans evolved in a hot environment and are hairless to improve heat dissipation. Core temperature is controlled by the anterior hypothalamus via the autonomic nervous system. Major autonomic cooling mechanisms are conductive (cutaneous vasodilation) and evaporative (sweating). Conductive cooling becomes less effective as ambient temperature rises. Evaporative cooling becomes less effective as ambient humidity rises. Behavioral cooling strategies include seeking shade, reducing activity, and removing clothing.

Causes and consequences of hyperthermia

There are three primary causes of systemic hyperthermia: exertional, nonexertional, and iatrogenic. Exertional heat illness occurs in soldiers and other athletes whose activity-related heat generation exceeds their autonomic cooling capabilities. Behavioral cooling methods are often suppressed to accomplish a task. The consequences can be severe, and use of the Heat Stress Index and planning for heat management is an important component of mission success.[97] Nonexertional hyperthermia results from conditions that impair normal autonomic or behavioral thermoregulation. Principal causes are psychiatric or neurologic problems, obesity, hyperthyroidism, or behavioral limits seen in those at the extremes of age. Iatrogenic causes of systemic hyperthermia include neuroleptic malignant syndrome, malignant hyperthermia, and the use of anticholinergic drugs. For management of nonenvironmental hyperthermia, the reader is referred to many available references.[98]

Hyperthermia is deleterious for several reasons. Initially, O_2 consumption and CO_2 production are increased with greater demand placed on ventilation. Above 42°C (108°F), oxidative phosphorylation is impaired in mitochondria, interfering with cellular oxygen use. At higher temperatures, many enzyme systems cease to function; cell membranes become incompetent, and multiorgan failure develops, eventually leading to death.

Clinical presentation of hyperthermia

Heat illness occurs when behaviors, generally exercise in hot environments, exceeds autonomic cooling mechanisms and behavioral cooling mechanisms are voluntarily suppressed. Initial symptoms are mild and constitutional but can progress to cardiovascular failure, multiple organ failure, and death if not addressed.[99,100]

The initial symptoms of heat illness are called heat cramps. The symptoms consist of sweating, fatigue, and cramping of major torso and/or extremity muscle groups. With continued exercise or systemic heating, symptoms progress to heat exhaustion that is characterized by heavy sweating, pallor, muscle cramps, weakness, headache, vomiting, and often fainting. Core temperature is by definition less than 40.5°C (105°F). With continued exercise or systemic heating, symptoms progress to heat stroke. In addition to the findings of heat exhaustion, symptoms of heat stroke include confusion, coma, seizures, respiratory failure, and cardiovascular collapse. Core temperature is, by definition, greater than 40.5°C (105°F).

Physical and laboratory findings also present a spectrum. Early heat illness has little in the way of hard physical abnormalities. As the disease progresses through heat exhaustion, heavy sweating, and high temperature (under 105°F or 40.5°C) are noted. This is followed by cutaneous vasodilation, tachypnea, tachycardia, and an altered sensorium. Heat stoke is signaled by temperatures in excess of 105°F (40.5°C) and cessation of sweating. Neurologic dysfunction ranges from confusion through

seizures and coma. Rhabdomyolysis occurs. Noncardiogenic pulmonary edema and hypotension lead to cardiovascular collapse. Disseminated intravascular coagulation and renal and hepatic failure follow with corresponding laboratory findings.

Management of hyperthermia

The spectrum of response is guided by the severity of the individual's condition. Heat cramps and exhaustion are managed with hydration, rest, and external cooling via clothing removal, shower, sponge bath, and enhanced evaporative cooling by fanning.[101] Heat stroke, in contrast, is a medical emergency with a mortality of up to 20% even with prompt treatment. Management principles include standard airway management, if needed, and restoration of circulating volume.[102] A rapid but controlled core temperature reduction to an initial target of 39°C (102.2°F) is appropriate. A combination of evaporative and conductive cooling is used by unclothing the patient and spraying him or her with tepid water adjacent to a fan. Core temperature should be monitored via rectal or esophageal probe. In general, tepid water is preferable to ice water because the latter may cause cutaneous vasoconstriction and thus reduce the rate of heat dissipation. Intravascular volume is usually low and should be promptly replenished.

In the critically ill patient with hyperthermia, sedation and/or anticonvulsants may be needed.[103] Hypoglycemia may occur and should be promptly corrected. One-third of these patients may develop rhabdomyolysis. In the presence of urine pigment from rhabdomyolysis, urine output of 2 to 3 mL/kg/h should be targeted. There may be a limited role for alkalinization of the urine and/or mannitol. Electrolyte abnormalities may occur, including hyperkalemia, hypocalcemia, and hyperphosphatemia. Coagulopathy may develop and, in rare cases, patients may require blood product transfusion. Noncardiogenic pulmonary edema should be managed with positive pressure support. Renal failure may occur and should be managed as in nonhyperthermia patients. Cerebral and intracompartmental edema may develop in severe cases and should also be managed as in the nonhyperthermia patient. It is important to exclude these complications in critically ill hyperthermia patients.

Cold Injury

Cold injury has both local and systemic manifestations that are seen in similar circumstances, including wilderness experience and military deployment. Both have similar exacerbating factors, including tobacco, alcohol, drugs, diabetes, and neuropathies. Prevention strategies include training and situational awareness.[104] Both local and systemic cold injury are described by a confusing archaic nomenclature and are described here as stages 1 to 3 (**Table 1**).

Hypothermia

Systemic cold injury, or hypothermia, presents with a range of symptoms from violent shivering and piloerection through confusion to paradoxic behaviors, arrhythmias, organ failures, and death. It is generally useful to describe this spectrum in stages associated with specific temperatures but, in reality, one stage blends gradually into another. Paradoxic behaviors are seen in later stages of hypothermia and are thought to originate with cold-induced hypothalamic dysfunction resulting in a sensation of extreme heat. This leads to the classic paradoxic behaviors of undressing and burrowing into snow that are seen in about half of those who die of hypothermia.

Hypothermia is best prevented through training, use of proper equipment, and situational awareness. Treatment requires prompt systemic rewarming using external or internal means.[105] External rewarming involves the use of blankets, hot-air covers,

Table 1
Stages of local and systemic cold injury

Cold Injury Type	Stage One	Stage Two	Stage Three
Hypothermia (Systemic Cold Injury)	35 37°C (95 98.6°F) Strong shivering and piloerection Poor fine motor coordination, hands become numb Shallow breathing, fatigue, nausea, visual disturbance	33 35°C (91 94.9°F) Violent shivering that then stops, pallor, distal cyanosis Poor gross motor coordination, stumbling Confusion, alertness maintained	Below 32°C (89.6°F) Shivering stops, reduced level of consciousness progressing to stupor Paradoxic behaviors, terminal burrowing and undressing Bradyarrhythmias and tachyarrhythmias, reduced respiration Cold diuresis, organ failures, death
Frostbite (Local Cold Injury)	Burning and numbness Pallor warms to erythema	Insensate Pallor warms to blistering Perfusion after warming	Insensate Frozen warms to hemorrhagic blisters Variable perfusion or necrosis after warming

and/or warm-water immersion. Internal methods primarily involve administration of warm intravenous fluids and warm humidified air. More invasive techniques include nasogastric, peritoneal, or pleural warm-water lavage. Venovenous perfusion and cardiopulmonary bypass have been reported as effective but are rarely used in clinical practice. Providers should be aware that dehydration is extremely common in hypothermic patients because of the situations in which the injury occurs as well as the profound tubular dysfunction and diuresis that follows renal cooling. Patients in later stages of hypothermia are prone to arrhythmias; care should be taken when moving and repositioning patients and with placement of upper body central venous catheters. Finally, profoundly hypothermic patients can seem clinically dead, so resuscitation should be continued until they have been rewarmed.

Frostbite

Local cold injury, or frostbite, also presents with a range of symptoms from burning and numbness, through loss of sensation and hemorrhagic blistering, to small vessel thrombosis and necrosis after rewarming (see **Table 1**). Treatment requires prompt local rewarming using passive and/or active means. Passive rewarming involves external covers and the use of the patient's body heat (eg, placing a hand in an axilla) or the body heat of another person. Friction should not be used because this aggravates local tissue injury. Active rewarming generally involves application of, or immersion in, warm (40°C/104°F) water. Care must be taken not to burn the insensate part. Refreezing of frozen parts must be avoided because this has been shown to exacerbate tissue loss. Rewarming of frozen parts should be carefully considered in the field and only done when it is clear that refreezing will not occur during evacuation.

There are a large number of anecdotally reported and poorly supported adjuncts, including nonsteroidal antiinflammatory drugs, hyperbaric oxygen, dextran, Coumadin, heparin, vasodilators, calcium-channel blockers, alpha-blockers, pentoxifylline, aspirin, vitamin C, and surgical sympathectomy. None of these are established as standard of care. Imaging with Tc-99 or PET scanning has been reported but has not been shown to improve outcome. Fasciotomy is generally not helpful because

Table 2
Thrombolytic screening checklist: patients are potential candidates if all queries are answered "yes"

Query	"Yes" or "No" Answer
Does the patient demonstrate stable gas exchange and hemodynamics?	
Is flow absent flow after rewarming (No capillary refill, no Doppler signals)?	
Is the cold ischemia (frozen) time <24 h?	
Is the warm ischemia time <12 24 h?	

the tissue necrosis is caused by small vessel thrombosis instead of intracompartmental edema.

In rare patients, acute thrombolytic therapy may be useful. In stage 3 frostbite, in which involved parts are frozen, small vessel thrombosis may follow rewarming and can lead to nonperfusion and necrosis. The pathophysiology is related to endothelial cell disruption from freezing with secondary thrombosis of smaller vessels. The experience has been mixed, with patients treated who show no perfusion immediately after rewarming being potential candidates. Ideal patients have little warm ischemia time. A thrombolytic treatment screening tool has been reported (**Table 2**).[106] Patients should demonstrate stable gas exchange and hemodynamics, have no detectable perfusion after rewarming, have a cold ischemia time fewer than 24 hours, and a warm ischemia time fewer than 24 hours. Potential candidates are taken to

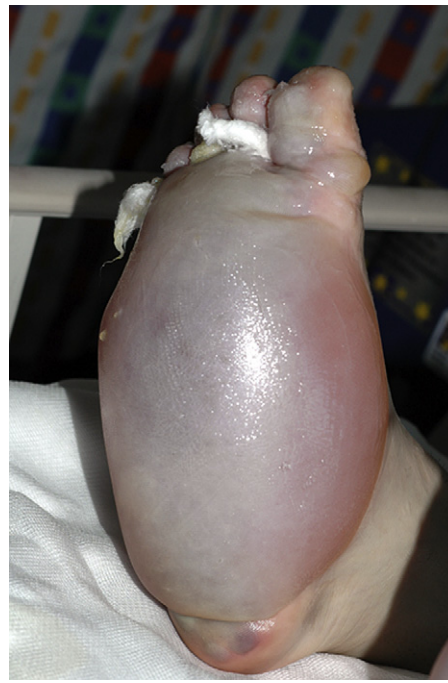

Fig. 4. Frostbite. After rewarming, initial wound care is generally conservative, with minimal debridement unless infection occurs. Blisters are allowed to collapse when possible.

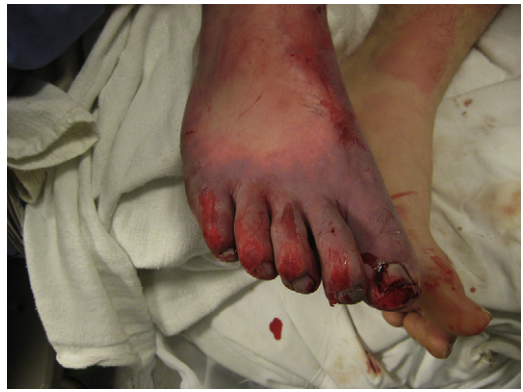

Fig. 5. Frostbite. When necrosis is clearly demarcated, excision and closure is performed.

angiography for a diagnostic study with intraarterial vasodilators. If there is no flow in the affected part, intraarterial tissue plasminogen activator (tPA) is given. Angiography is repeated in 24 hours; tPA is stopped for restoration of flow, bleeding complications, or absence of flow at 72 hours. Empiric prophylactic anticoagulation is given for 1 month.

After rewarming, initial wound care is generally conservative, with minimal debridement unless infection occurs (**Fig. 4**). When necrosis is clearly demarcated, excision and closure is performed.[107] This may require a creative combination of grafts and flaps depending on the individual wound (**Fig. 5**). Long-term challenges are generally related to the wounds, but some patients will develop neuropathic pain and sensory and motor dysfunction depending on ischemia time. Many of these symptoms will improve with time.

SUMMARY

Burns, soft-tissue, and environmental injuries are common in survivors of natural disasters, terrorist incidents, and combat. Many other patients will suffer from such exposures in addition to their primary injury as a consequence of delays inherent in dangerous and chaotic environments. Effective and organized initial care will enhance survival and optimize long-term outcome.

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