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Thermal and Electrical Injuries

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Modern burn care has been characterized by substantial increases in survival and improvements in functional outcomes for burn patients over the past 30 years. Twenty-first century optimal burn care consists of a specialized treatment scheme that incorporates early surgical wound closure, critical care management, and rehabilitation efforts. The success of burn treatment as a multidisciplinary model has fostered the organization of burn centers as regional resources for severely injured patients, including individuals with large open wounds.

The review in this article and the Burn Care Guidelines published by the American Burn Association both illustrate the need for Class I evidence to support standards of burn care [1]. In many cases, our practices are based on years of Class II evidence from small clinical trials. Multicenter research collaborations, such as the National Institutes of Health–funded genomics project "Inflammation and the Host Response" (http://www.gluegrant.org), have begun to codify standards of practice that should pave the way for improved future multicenter clinical trials [2,3].

Acute burn care

Burn wound management

Early eschar excision for massive burn injuries has had the greatest impact on burn patient survival by reducing the incidence of wound sepsis, hypercatabolism, numbers of operations, and hospital lengths of stay [4–6]. Wounds that take longer than 3 weeks to epithelialize typically heal with excessive scarring and contractures that produce aesthetic and functional impairment. Clinicians must be able to anticipate the healing potential of

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a fresh wound to weigh the relative risks and benefits of excision and grafting of the burn wound. An accurate estimation of burn depth is paramount to proper wound management. An experienced burn provider usually can identify shallow or full-thickness wounds based on clinical grounds alone. Intermediate dermal injury ("indeterminate" burn) poses the greatest challenge. Unfortunately, several studies indicate that initial evaluation even by an experienced surgeon may be only 50% to 70% accurate as to whether an indeterminate dermal burn will heal within 3 weeks [7–9].

Investigators have searched for an objective adjunct to clinical judgment so that patients with indeterminate burns with poor healing potential also may benefit from early excision. Various techniques attempt to quantify physical changes associated with skin injury, such as the presence of denatured collagen, wound edema, and an altered blood flow pattern [10-13]. The most recent development in this field is noncontact laser Doppler imaging, which records the reflectance shift of moving red blood cells in the dermal capillary plexus to provide a color perfusion map of the wound [14]. Theoretically, reduced dermal blood flow portends a low likelihood of healing and could prompt a clinician to operate sooner. This technique is well tolerated by patients and avoids the artifact of pressure on the wound with the scanning device held at a distance. Noncontact laser Doppler imaging examinations can be repeated serially over the first several days after burn as wound bed perfusion evolves throughout the resuscitation phase. Indeterminate dermal burns may become progressively deeper several days after injury (a process termed "wound conversion") as healing potential is affected by perfusion, edema, and infection [15]. Wound conversion, however, is minimized when a patient receives adequate fluid resuscitation and proper wound management [16]. Although promising, noncontact laser Doppler imaging has not yet demonstrated consistent reproducibility and has been no more reliable than experienced burn surgeons [17-19]. It has not been incorporated into the mainstream of burn care.

Although full-thickness and deep dermal burns are best excised within the first week after injury, more superficial wounds may be treated with topical agents until they heal or have demonstrated that they will not heal within 3 weeks. An ideal dressing should be comfortable for the patient, easy to apply and remove, conform to the wound, be relatively cheap, and require infrequent changes. Biologically, it must provide a moist wound environment, limit growth of micro-organisms with good eschar penetration, have no or minimal systemic effect, and débride devitalized tissue as needed. Currently, such universal dressing does not exist; however, not all wounds require these features. Small shallow burns, for example, do not require dressings with antimicrobial activity. Greasy gauze is appropriate for shallow dermal burns. A recently marketed ointment containing β -glucan (Glucan-Pro, Brennan Medical, St Paul, Minnesota), a carbohydrate derived from oat, may be appropriate for shallow wounds because it is soothing and mitigates itching. β -glucan may have an immunomodulatory effect by

stimulating macrophage activity [20,21]. β-glucan is also available as a dressing (Glucan II, Brennan Medical, St Paul, Minnesota) and is a favored dressing for donor sites at many centers across the United States [22,23].

Biologic dressings may enhance partial-thickness injury healing. Their proposed benefits stem from infrequent dressing reapplication, improved patient comfort, and topical administration of growth factors. A growing list of biologic dressings has been approved by the US Food and Drug Administration, with several more undergoing clinical trial. In larger burns in which cost is a limiting factor and outpatient therapy is not feasible, human cadaver skin and porcine skin remain good choices for temporary biologic dressings. Table 1 lists frequently used biologic and nonbiologic dressings for burn wounds.

Antibiotic activity becomes more relevant in dressings for deeper wounds, because they are more prone to infection. The most common

Dressings	Category	Examples	Appropriate indications
Nonbiologic			
	Petrolatum	Xeroform, Xeroflo, Adaptic, Aquaphor gauze	Partial-thickness burns, skin grafts, donor sites
	Silver	Acticoat, Acticoat-7, Aquacel-Ag, Silvasorb	Partial-thickness burns, skin grafts, donor sites
	Polyurethane	OpSite, Tegaderm	Partial-thickness burns, donor sites
	Foam	Lyofoam	Partial-thickness burns
	Silicone	Mepitel	Partial-thickness burns, skin grafts, donor sites
	Negative pressure therapy	Wound VAC system	Skin grafts
Biosynthetic and biologic	Oat	Glucan II	Partial-thickness burns, skin grafts, donor sites
	Collagen and fibroblasts	Transcyte, Apligraf	Partial-thickness burns
	Collagen, fibroblasts, and keratinocytes	OrCel	Partial-thickness burns
	Allograft (cadaver)	Fresh or cryopreserved	Partial-thickness burns
	Xenograft	Porcine skin, porcine intestinal submucosa (Oasis)	Partial-thickness burns

 Table 1

 Commonly used dressings for burn wounds, skin grafts, and donor sites

topical antimicrobial agent for deeper dermal burns is silver sulfadiazine (Thermazine, King Pharmaceuticals, Bristol, Tennessee). Silver is effective against a broad spectrum of gram-positive and gram-negative organisms, including most types of *Staphylococcus aureus* and *Pseudomonas aeruginosa* [24]. It has been incorporated in commercially available dressings, such as Acticoat (Smith and Nephew, Largo, Florida) and Aquacel-Ag (ConvaTec, Princeton, New Jersey) [25,26]. Both products can be used to cover partial-thickness burns, meshed skin grafts, and donor sites.

Ideally, when a burn wound is excised, the wound bed should be replaced with full-thickness autograft skin; unfortunately, full-thickness skin availability is limited by the number and size of full-thickness donor sites that can be primarily closed [27]. Whenever possible, split-thickness sheet grafts should be applied as sheet grafts to maximize function and aesthetics [28]. The standard practice of expanded meshed split-thickness skin autograft achieves wound closure over larger areas, but its disadvantages include fragile wound beds, suboptimal appearance, reduced pliability, and scarring. In patients with large burns, serial harvesting ("recropping") of donor sites may be necessary with larger body surface injuries; one must wait for donor sites to heal, and subsequent skin grafts are thinner and of lesser quality. In the meantime, fresh or cryopreserved cadaver (allograft) skin can be used as a temporary biologic dressing over the excised burn wound bed. Taken together, the current process of partial-thickness autografting for large burns vields suboptimal results for burn wounds covered with widely expanded skin grafts and the reharvested donor sites. The recognition of current limitations has created an impetus for research on commercially available skin substitutes.

There are two general classifications of skin substitutes: cultured epidermal grafts and dermal substitutes. Several caveats exist regarding use of skin substitutes for permanent wound coverage. Cultured epithelial autografts have limited use as a stand-alone replacement because they provide a thin and fragile sheet of keratinocytes that frequently sheer and offer little durability [29-31]. Although epithelial allografts may be suitable as biologic dressings, they cannot be used as skin substitutes because they are ultimately rejected by the recipient's immune system. The dermis determines optimal engraftment and graft durability. In vitro autologous dermal regeneration has not been achieved with current available technology. Providing a dermal layer for wounds requires an exogenous matrix template. Integra (Integra LifeSciences Corp., Plainsboro, New Jersey) is a dermal replacement template comprised of an inner matrix layer of bovine collagen and shark glycosaminoglycan, adhered to a silicone outer layer [32-34]. The inner layer forms a scaffold for in situ dermal regeneration while the outer layer contains water vapors and provides a physical barrier to the outside environment. After approximately 2 weeks, the neodermis is sufficiently vascularized to accept a thin partial-thickness autograft (0.06 in thick) [35]. Although Integra is relatively fragile and susceptible to infection, sufficient longitudinal experience in several centers suggests that consistently good results with this product are possible [32,36–38]. An acellular cryopreserved cadaver dermis (AlloDerm, LifeCell Corporation, Branchburg, New Jersey) also has been marketed as a dermal replacement, but clinical endorsement for this product as an acute burn wound replacement remains limited [39,40]. Boyce and colleagues [41] reported on a promising new approach of maturing the epidermal-dermal skin substitute in vitro by culturing autologous keratinocytes on a collagen matrix. The composite skin replacement is applied to the wound 2 to 3 weeks after harvesting autologous skin; in the meantime, the wound bed can be prepared with another layer of dermal substitute. If successful, this strategy could reduce the problem of shearing seen with application of cultured cells directly onto a wound bed and increase the elasticity, pliability, and function of the wound bed.

Fluid resuscitation

Judicious fluid resuscitation is one the greatest challenges in the care of acutely burned victims. Burn injuries over more than 20% of surface area result in increased capillary permeability and edema in burned and nonburned tissues. Vasoactive mediators from injured skin, such as histamine, prostaglandins, and oxygen-free radicals, mediate a massive capillary leak syndrome that typically lasts for 24 hours after injury [42]. Burn shock is characterized by persistent hypovolemia that demands continuous intravenous fluid rate modification over the first 24 to 48 hours of hospitalization. Several formulas developed over the past 50 years to estimate patient fluid needs have been based on body weight and burn surface area. Each formula differs on the amount and type of crystalloid and the necessity for colloid infusion during resuscitation. The most widely used formula in adults is the Parkland (or Baxter) formula [43], which calls for the infusion of 4 mL/kg/% total body surface area (TBSA) burn lactated Ringer's solution for 24 hours. Half of the volume should be administered over the first 8 hours and the other half during the next 16 hours. Throughout this period, the clinician must continuously re-evaluate patient response to resuscitation and titrate the fluids to achieve a mean arterial pressure of more than 60 mm Hg and urine output of more than 30 mL/h. In children, low glycogen stores and maintenance fluid needs should be addressed by augmenting the resuscitation fluid with an isotonic maintenance solution that contains dextrose. Controversy persists among burn specialists over the use and timing of colloids. Animal studies suggest that capillary permeability is maximal within the first 8 to 12 hours and may be exacerbated by colloid administration [42,44]. Centers that routinely use colloids generally administer them later in the resuscitation phase.

Deep burns, inhalation injury, comorbid illnesses, associated injuries, and delay in resuscitation are recognized to increase fluid requirements [45]. Formulas only serve as initial guidelines, and maintenance of urine

output (0.5 mL/kg/h in adults and 1 mL/kg/h in children) is the best surrogate marker of adequate end-organ perfusion. Not satisfied with crude reliance on urine output, many investigators have sought to improve goal-directed therapy during resuscitation. Despite the appeal of invasive hemodynamic monitoring and the natural desire to augment oxygen delivery, a well-designed prospective randomized trial failed to show any advantages to preload-driven resuscitation [46]. Patients who were given preload-driven resuscitation had equally low central filling pressures and intrathoracic blood volumes compared with patients on the Parkland formula. The authors concluded that the additional fluid volume (60% over initial calculations) administered to the "preload" group leaked out of the intravascular space and contributed to peripheral edema.

Although Dr. Baxter repeatedly stressed that most patients could be resuscitated with 3.7 to 4.3 mL/kg/% TBSA burn, recent reports describe average resuscitation volumes significantly exceeding predicted needs, as high as 8 mL/kg/% TBSA [47,48]. This phenomenon has been termed "fluid creep." Proposed explanations for this discrepancy include not reducing fluid rates when urine outputs exceed 0.5 mL/kg/h, relying on invasive monitors to guide resuscitation, and administering larger doses of opioids to control burn pain (termed "opioid creep") [49]. It may be possible that the nature of burn injuries and inhalation injuries has evolved; patients who have been in methamphetamine explosions may exemplify this evolution, because they typically require large resuscitation volumes [50]. Whether higher fluid administration correlates with improved survival is unclear. Compared with the mid-twentieth century, acute renal failure, a common sequela of underresuscitation, is uncommon when resuscitation is initiated early and death because of failed resuscitation is even rarer. Excessive volume resuscitation generates its own complications. Edema may become severe enough in unburned extremities that escharotomies and, occasionally, fasciotomies become necessary [51]. Lung tissue edema may lead to acute respiratory failure [52]. Gut and mesenteric edema manifests as intra-abdominal hypertension; fascial release may be required to treat abdominal compartment syndrome [53,54]. Edema also may become symptomatic in the orbits, as evidenced by elevated intraocular pressures and need for lateral canthotomies [55].

Several strategies to mitigate "fluid creep" are currently being investigated. For instance, hourly urine output measurements have been criticized because hourly intervals are arbitrarily chosen for convenience. A recent animal study suggested that an automated closed-loop system that adjusts fluid administration to continuous urine output measurement may decrease fluctuations based on human interventions [56]. Such systems could be adapted with additional inputs, such as blood pressure or base deficit measurements, to guide resuscitation needs. Considerable interest also exists in antioxidant therapy, because membrane lipid peroxidation and oxygen-free radicals are major components of burn shock physiology [57]. Animal and clinical studies suggest that antioxidants reduce fluid requirements and burn wound edema during resuscitation [58,59]. Early administration of tocopherol and ascorbate in critically ill surgical trauma patients also shortens the duration of mechanical ventilation and decreases the incidence of multiorgan failure [60]. Antioxidant therapy as an adjunct to burn resuscitation mandates large-scale multicenter prospective validation before it should be accepted as standard of care. Another interesting strategy is plasma exchange, which theoretically removes inflammatory mediators circulating in the systemic circulation. Although Warden and colleagues [61] described the use of plasma exchange to salvage patients who were failing resuscitation more than 20 years ago, confirmatory studies to explain its salutary mechanisms or clinical benefits are still lacking.

Inhalation injury and intensive care management

Airway burn injuries can be divided into two types: upper airway thermal injury and lower airway chemical injury. Carbon monoxide poisoning is more accurately categorized as a systemic intoxication with the lung as a portal of entry. Clinicians often group all three into "inhalation injury" because all three insults may coexist, for example, in a patient who has been in a closed-space fire. The diagnosis of an upper airway burn can be made readily by assessment of hoarseness or stridor and examination of the posterior pharynx for edema or mucosal slough. Injuries to the lower airways can be diagnosed by direct visualization (fiberoptic bronchoscopy), suggestion of a ventilation/perfusion mismatch (xenon scan), or radiographic evidence of small airway inflammation and obstruction (CT scan) [62-64]. Xenon scanning is mostly of historical interest, and additional information obtained via CT scan is of questionable clinical value. The transport of patients to the radiology suite with ongoing resuscitation is cumbersome and at times hazardous. Although bronchoscopy confirms a clinical diagnosis of inhalation injury, it rarely alters clinical management.

The diagnosis of carbon monoxide poisoning can be measured easily with a serum carboxyhemoglobin level. Administration of 100% oxygen reduces the half-life of carboxyhemoglobin from 4 hours (on 21% O₂) to approximately 45 minutes. In practice, many patients with carbon monoxide poisoning have normalized values upon arrival to the burn center. Proponents of hyperbaric oxygen (HBO) therapy have argued that hyperbaric chamber treatment lessens long-term neurologic sequelae, even with normal pretreatment carbon monoxide levels. Two prospective randomized trials of HBO therapy have yielded conflicting results [65,66]. Scheinkestel and colleagues [65] described sequential chamber treatments over 3 to 6 days, with hyperbaric-treated individuals performing worse on neuropsychological testing compared with normobaric treatment. Conversely, Weaver and colleagues [66] used a treatment algorithm consisting of three HBO treatments within 24 hours of enrollment and reported that cognitive impairments were less frequent at 6 weeks in the HBO group and persisted at 1-year follow-up. The first study specifically excluded burn patients, whereas the second report apparently did not include major burn injuries, as evidenced by few being patients hospitalized (14%) or requiring mechanical ventilation (8%). The presence of a major burn requires careful fluid resuscitation, whereas mechanical ventilation imposes an additional logistical challenge for patients placed in HBO chambers. In our own experience, severely burned victims with concomitant carbon monoxide poisoning experience high complication rates when HBO therapy is attempted [67]. HBO treatment for carbon monoxide poisoning in patients probably should be limited to patients with burn injuries smaller than 15% TBSA.

Patients with lower airway inhalation injury are at risk for developing acute respiratory distress syndrome because of direct airway injury coupled with increased volume resuscitation requirements. Although an optimal ventilation strategy for inhalation injury remains to be defined, many burn centers have adopted the use of lower tidal volumes and reduced airway plateau pressures to treat acute respiratory distress syndrome based on compelling data from the Acute Respiratory Distress Syndrome Network group [68]. Although "prophylactic" use of a lung protective ventilation strategy in inhalation injury is an appealing concept, previous efforts have failed to show clinical benefits in patients at risk for acute respiratory distress syndrome [69]. For the small number of patients who oxygenate poorly on conventional settings, high-frequency oscillatory ventilation can improve oxygenation dramatically while acute respiratory distress syndrome resolves [70,71]. Several pharmacologic means to minimize airway narrowing, prevent airway obstruction, and improve clearance of debris have been shown to have variable success in animal models of lower airway inhalation injury. These strategies include mucus fragmentation (N-acetylcysteine), bronchodilation (B2 agonists, nitric oxide), clot dissolution (antithrombins, tissue plasminogen activator, and heparin), flow turbulence reduction (partial liquid ventilation), and inhibition of inflammation (steroidal and nonsteroidal anti-inflammatory agents) [72-77]. Widespread adoption of any of these agents awaits confirmation with level I evidence.

Prolonged mechanical ventilation often complicates the care of large burns, with or without inhalation injury. The debate over tracheostomy compared with translaryngeal intubation remains unresolved, because there are no prospective studies with appropriate side-by-side comparison [78–81]. For any benefit of tracheostomy to be realized, this procedure should be performed early in the patient's course. Predictors of successful ventilator weaning are often inaccurate, however, and tracheostomy can be a morbid procedure. Outcome comparison is also difficult because all patients with tracheostomy are cross-over from the translaryngeal intubation, and an accurate assessment of long-term tracheal complications can be made only by fiberoptic laryngoscopy on all patients studied. It is likely that individual burn centers will remain entrenched either on the conservative or aggressive side of the tracheostomy debate. Given the absence of Class I evidence, recommendations for airway management must include options rather than standards of clinical care.

Anemia

Hospitalized burn victims become anemic because of hemodilution, relative bone marrow suppression, and frequent laboratory draws. Early eschar excision, currently widely accepted as a standard of burn care in North America, traditionally has been associated with significant operative blood loss [82]. Blood transfusion is a life-saving treatment in some circumstances but has potential drawbacks, such as viral transmission, transfusion reactions, and immunosuppressive effects. "Passenger" leukocytes present in transfused packed red blood cell units are critical components of immune modulation [83]. Transfusion of leukocyte-depleted blood reduces the incidence of infection in postoperative cardiac and noncardiac surgery patients [84,85]; however, the validity of this approach in the injured patient remains to be established. In a multicenter retrospective study on blood use in burn centers, Palmieri and colleagues [86] reported that patients with burns over 20% received on average 14 units of packed red blood cells over the course of their hospitalization, and they suggested that transfusion requirements independently increased the risk of infections and mortality. Methods developed to reduce intraoperative blood loss include use of tourniquets, compression wrappings and elevation for extremities, application of hemostatic agents and epinephrine-soaked pads to excised wounds, and subcutaneous infusion of dilute epinephrine under the eschar and donor sites [82,87]. With accumulating data underscoring the safety of relative anemia (hemoglobin of 7 g/dL) in critically ill patients [88,89], burn centers are gradually accepting lower steady-state hemoglobin levels outside the operating room. The current trend is to adopt a restrictive transfusion policy based on individual patients' demonstrated needs.

The necessity for prophylaxis of deep venous thromboses and pulmonary emboli in burn patients remains unresolved. Although thromboembolic disease was historically viewed as a rare occurrence in burn patients, recent reports document a varying incidence of deep venous thromboses/pulmonary emboli in this patient population proportional to the frequency of duplex ultrasound examinations, whether performed as a serial screening tool or selectively based on symptomatology [90–92]. Compression devices are of unproven value, and their application is poorly tolerated in individuals with lower extremity open wounds. Administration of heparin and related compounds must be weighed against their side-effects. Most notably, heparin-induced thrombocytopenia has emerged as a recognized complication in the burn unit [93,94]. Heparin-induced thrombocytopenia is a severe prothrombotic state that is associated with dreaded complications, such as digit necrosis, limb loss, and even death. The efficacy of alternative anticoagulation agents, such as low molecular weight heparin compounds and pentasaccharides, has not yet been evaluated. Large-scale prospective studies are needed before we are able to define the indications and the most efficacious agents for deep venous thromboses/pulmonary emboli prophylaxis in burn patients.

Modulation of post-burn hypermetabolism

Burn injuries over more than 25% TBSA are associated with a hypermetabolic state that develops over the first 5 days and persists until the wounds are completely healed. Sometimes it lasts up to a year after injury [95]. Protein catabolism is a particularly deleterious feature of this response: the loss of lean body mass is a barrier to rehabilitation for all patients and retards normal growth in burned children. Early surgical wound excision and skin grafting remains the most expeditious way to reduce the inflammatory burden posed by the wound. Routine care in the burn intensive care unit also should include specific daily management strategies to manage hypermetabolism. Maintenance of warm ambient temperatures (33°C) partially reduces the obligatory heat loss created by fever [96]. Nutritional supplementation must be instituted early in the patient's course, ideally during the resuscitation phase and before ileus develops. Enteral feedings initially can be based on estimated needs and subsequently adjusted by indirect calorimetry. The prevention, prompt diagnosis, and treatment of infections represent a daily challenge in burn patients. Control of infection also significantly reduces energy expenditure over a patient's hospitalization. Hyperglycemia is another marker of severe metabolic derangement and has been associated with worse outcomes in burn patients [97,98]. Two recent prospective, randomized evaluations by Van den Berghe and colleagues [99,100] have established that maintenance of euglycemia via continuous insulin infusion is desirable in critically ill patients because it decreases the incidence of infections and reduces mortality.

During the recovery phase, a rehabilitation program that includes exercise against resistance builds not only lean body mass but also muscle strength [101,102]. Pharmacologic agents that help preserve and restore lean body mass represent adjuncts in modulating post-burn hypermetabolism. Recombinant growth hormone (administered over 1 year) prospectively evaluated in a double-blind trial in children with severe burns suggested that children on growth hormone gained more lean body mass, height, and bone-mineral content than control subjects [103]. The benefits of growth hormone are not applicable to adults, because hyperglycemia is a common side effect in this group [104]. Oxandrolone, a testosterone analog, is an anabolic steroid with reduced virilizing potential [105,106]. A prospective trial of oxandrolone in children demonstrated improvement in net protein balance after 1 week of administration [107]. In a recently completed randomized, placebo-controlled trial, adults who received oxandrolone had reduced lengths of hospital stay compared with patients on placebo [108]. Although many factors potentially impact length of stay, the study suggests a benefit to oxandrolone. Propranolol, a nonselective beta-blocker, reduces tachycardia, energy expenditure, and substrate cycling and prevents fatty infiltration of the liver [95,109]. In a randomized study of 25 children, Herndon and colleagues [110] demonstrated that propranolol attenuated the effect of hypermetabolism by reversing muscle protein catabolism. Beta-blockade also constitutes an attractive strategy for adults in which tachycardia is undesirable and less well tolerated in patients with pre-existing heart disease. Ongoing trials are indicated to evaluate the efficacy and safety of propranolol in adults.

Electrical injuries

Electrical burns represent a minority of admissions at major burn units but often cause severe morbidity beyond obvious skin injuries. In particular, high-voltage injuries (arbitrarily defined as >1000 V) may lead to temporary dysrhythmias in survivors, be associated with major blunt trauma, and cause deep tissue destruction. Other deficits may manifest themselves in a delayed fashion: two commonly described long-term sequelae are peripheral motor or sensory neuropathy and the appearance of cataracts [111,112]. Most patients with electrical burns are young men injured at work (eg, construction workers, electricians, and linemen). Injury and disability in this demographic group result in major loss of wages and significant medical costs [113,114]. No Class I evidence exists to support standardized management of electrical burns. Available guidelines recommend 24-hour telemetry monitoring for all patients with high-voltage injuries and for patients with low-voltage injuries who have an abnormal initial EKG [115]. Some data, however, suggest that monitoring high-voltage injuries with an initial normal EKG may be superfluous [116]. Deep electrical injuries generate rhabdomyolysis and myoglobinuria. In this setting, fluid resuscitation should be titrated to maintain a urine output of 100 mL/h until the urine clinically appears clear. Acute renal failure from myoglobinuria is rare unless resuscitation is delayed. Several methods have been proposed to enhance renal clearance of myoglobin, including alkalinization of urine and osmotic diuresis with mannitol [117]. These adjunct measures are of unproven value and represent individual centers' practices and will remain so until prospective evidence validates their benefit over simple isotonic crystalloid resuscitation.

Early fasciotomy or surgical débridement of necrotic muscle may be warranted when severe acidosis and myoglobinuria do not rapidly improve with aggressive resuscitation; management in a burn center in which these injuries can be monitored closely by a burn surgeon is optimal. Although most limbs can be salvaged with early diagnosis of compartment syndrome and compartment fasciotomies, major débridement and early amputation occasionally may be necessary [118]. Although routine fasciotomy has been advocated, a review of national trends in management of patients with electrical burns supports selective decompression [119]. Mann and colleagues [113] reported that most patients with high-voltage injuries (70%) did not require emergent operation and no amputations were required in patients who were monitored. Monitoring consists of serial clinical assessments of tissue perfusion and peripheral nerve function in at-risk extremities. The use of technetium scan has not gained wide acceptance for it is overly sensitive in detecting deep tissue damage [120]. Fibrosis is the end of result of limited deep-tissue necrosis, whereas overly aggressive débridement may introduce infection and increase the risk of amputation.

Rehabilitation and reconstruction

With an increasing number of survivors of major burn injuries, successful re-entry into society becomes the next major challenge. A coordinated burn center program that includes surgeons, physiatrists, pediatricians, occupational and physical therapists, vocational rehabilitation specialists, and psychologists is essential to successful rehabilitation. Perhaps because of their resilience and adaptive ability, children recover well even after major burn injury. Sheridan and colleagues [121] reported that most children treated at Shriners Burn Institute (Boston) who survived massive burns ($\geq 70\%$ TBSA) became productive members of society. In their series however, 20% of patients had physical scores below norm; indicating that this subgroup had persistent sequelae. In adults, an important benchmark may be return to work. There is little information reported in the literature on this subject, however. A recent two-center review reported that median time off work approximated 12 weeks, and 90% of patients had regained employment by 2 years [122]. It is noteworthy that only 37% of patients returned to their preinjury job without accommodations. Several factors contributed to this finding: burn size, location of burns, and psychiatric history. A related but seldom reported outcome is impairment. Standard methods to calculate physical impairment are not widely used in burn care because they require either tedious calculations (whole person impairment rating) or initial investment in costly equipment (\$27,000 for the Dexter Evaluation system) [123,124]. Psychological assessment is another important component of impairment rating. Efforts are underway in this arena to develop tools that are appropriate gauges of the quality of life in burn survivors. The ongoing multicenter collaborative Burn Injury Rehabilitation Model System Program funded by the National Institute of Disability and Rehabilitation Research has increased awareness among burn providers and patients about burn survivor needs; despite progress since its inception, much more can be done to improve our patients' return to function.

Reconstructive surgery is essential to the rehabilitation process because it helps restore function and body image. The problems of hypertrophic

scarring and contracture remain enormous challenges for reconstructive burn surgeons. Hypertrophic scars may develop in healed burn areas, grafted sites, and even donor sites. Commonly used preventive strategies aimed at reducing raised scars include pressure therapy, topical silicone gel application, and massage [125–127]. Well-designed prospective studies to support use of these modalities are lacking. Patients with large burns can expect to undergo several scar revisions over their lifetime, because each procedure results in small incremental gains in function and appearance. Current understanding of the pathophysiology of hypertrophic scarring unfortunately remains limited, because many previous studies have studied mature scars and not scars in evolution. A standard animal model for hypertrophic scarring does not yet exist. Recent laboratory efforts have focused on the female red Duroc pig as multiple laboratories attempt to validate similarities in skin healing between this model and humans [128–130].

Access to burn care

The success of modern burn care, characterized by improved survival rates and return to preinjury function, is closely associated with the development of specialized burn centers. The burn center is not just "an area of the hospital" but a system of care that includes a specialized infrastructure, highly trained providers, and treatment algorithms that serve the unique needs of the burn victim. The burn center must be equipped to deliver all aspects of burn care, from initial management and acute surgical wound coverage, through rehabilitation and long-term reconstruction. Akin to other areas of medicine in which a relationship between volume and outcome has been established, the same appears true for burn centers. This process has driven regionalization of burn care in the past two decades, with many low-volume centers closing and seriously injured patients being referred to regional burn centers for definitive care. The American Burn Association has participated actively in this transformation by generating criteria for burn center referral (Box 1). The American Burn Association in association with the American College of Surgeons also has established a burn center verification program for approximately two decades. So far, 43 of the 139 listed burn centers in the United States have been certified by this process, and it is likely that they will continue to serve as centers of excellence for the foreseeable future.

Specialized burn care has created a demand for highly trained providers, including surgeons, nurses, therapists, psychologists, pharmacists, and rehabilitation physiatrists to form a multidisciplinary care team. The ranks of burn surgeons are usually filled with individuals having completed training in general surgery or plastic surgery. Their scope of practice, however, also includes components of pediatric and surgical critical care. Surgeons interested in burn care often seek additional training through burn fellowships. Whereas these individuals only number five to seven per year,

Box 1. American Burn Association burn unit referral criteria

- 1. Partial thickness burns >10% TBSA
- Burns that involve the hands, face, feet, genitalia, perineum, or major joints
- 3. Third-degree (full-thickness) burns in any age group
- 4. Electrical burns, including lightning injury
- 5. Chemical burns
- 6. Inhalation injury
- Burn injury in patients with pre-existing medical disorder that could complicate management or recovery or affect mortality
- 8. Patients with concomitant burn and trauma in which the burn injury poses the greatest risk of morbidity or mortality
- 9. Burned children in hospitals without qualified personnel or equipment for the care of children
- 10. Burn injury in patients who require special social, emotional, or long-term rehabilitative intervention

Adapted from the American Burn Association. Burn unit referral criteria. Available at http://www.ameriburn.org. Accessed June 30, 2006.

many fellowship positions remained unfilled. A similar situation exists for other burn team specialists. Experienced burn therapists are in short supply because proficiency requires many months of on-the-job training, and advertised positions may stay unfilled for indefinite periods of time. Whether this reality will result in a workforce shortage or create additional impetus for regionalization remains an unanswered question.

The large-scale use of air transport for burn patients started during the Vietnam War, during which field burn casualties were flown to the Brooke Army Medical Center in San Antonio, TX. Since that time, transport of burn patients has become more sophisticated, especially with the addition of respirators that were not available in the Vietnam Era. Successful transfer/transport over large distances requires good communication and coordination between referring and receiving facilities and highly trained personnel in the prehospital phase of care. Although our regional burn center covers an area one-fourth the land mass of the United States, outcomes for long-distance transfer patients are equivalent to that of patients directly admitted to the burn center [131].

Regionalization of care also creates two additional challenges: (1) proper patient triage and (2) coordination of transport, sometimes over great distances. It has been long recognized that referring physicians often underor overestimate burn surface area, which leads to inappropriate initial care, increased morbidity and mortality, and unnecessary use of air transport systems. Initial burn triage appears well suited for televideoconference consultation because most injuries can be assessed rapidly by an experienced provider at a remote location. Several burn centers in the United States and abroad have gained experiences with the application of telemedicine to initial burn treatment and patient follow-up [132-134]. Its reported advantages include improved access to tertiary care in rural and medically underserved areas, cost savings with fewer air transports for minor burns, increased patient satisfaction thanks to reduced travel expenses, and more time spent with providers. Cost savings are mainly felt on the patient side, whereas the use of videoconference technology represents a major expenditure for health care systems because of investments in infrastructure, maintenance costs, and communication expenses. Others have reported on the use of e-mail, including pictures for patient data communication [135]. This method has the added benefit of minimal technologic investment. So far, regulations have lagged behind technology with many unresolved issues such as patient confidentiality, licensure and credentialing, malpractice liability for providers, and reimbursement agreements that could offset the cost of telemedicine. Clearly, this area represents a most exciting development in burn care and likely will mature over the next few years.

Burn disaster planning

Mass disasters caused by explosions or structure fires typically result in a large number of burn casualties. The Rhode Island Station nightclub fire on February 20, 2003 resulted in 100 deaths and 215 injured patients, more than 50 of them with serious burns [136]. The terrorist attacks on September 11, 2001 were so lethal that the number of injured survivors was actually small. Still, one third of injured patients in New York City needed treatment for severe burns [137]. One could imagine that had the World Trade Center towers not collapsed, the number of burn casualties would have been much higher. The optimal care for burn victims follows a sequence of rapid and proper field triage, followed by intensive care management, burn excision and wound coverage procedures, and finally rehabilitation. For all these reasons, early access to specialized burn care is of paramount importance.

The triage of casualties at the scene naturally involves the activation of state and local response systems. To augment local capacities, the federal government can deploy disaster medical assistance teams to the scene. Burn specialty teams are specialized disaster medical assistance teams that consist of burn-experienced personnel to provide assistance needed in the initial care of burn victims. Four regional burn specialty teams are currently available for federal deployment. Burn specialty teams were deployed after the World Trade Center attack on September 11 and to support local resources after the Rhode Island nightclub fire. The last layer of this tiered response system involves military support to civil authority via activation of US Army special medical augmentation response teams. Two burn-specialized special medical augmentation response teams are currently based in San Antonio, TX, but so far have never been used for US civilian mass casualties. Because special medical augmentation response teams possess long-range air evacuation capabilities, they could become invaluable in the secondary triage and transfer of victims outside the disaster area.

Recognizing that casualty numbers exceeding 50% of maximum capacity (surge capacity) would quickly exhaust resources of local burn centers, the American Burn Association has advocated for a triage system unique to mass casualty burn events [138]. Primary triage is handled according to state and local activation plans, with burn patients triaged to a burn center within 24 hours of injury. Secondary triage is the coordinated transfer of patients from one burn center to a verified burn center after surge capacity is reached. In the event that casualties overwhelm local and national resources, patients would be triaged according to a survival probability grid that prioritizes treatment for patients with the highest likelihood of survival.

Burn research

A central tenet of any burn center should be its commitment to education and research. The physiologic challenge caused by burn injury may be greater than any other type of insult on the human body. It is a model that lends itself to study and can be replicated in the laboratory. In 2006, the official publication of the American Burn Association was renamed the Journal of Burn Care and Research. This change underscores the need for additional research to validate current practices and test unanswered questions in our field. In the clinical arena, several projects are worthy of mention because they embrace the concept of economy of scale to patient-oriented research. First, the organization of the National Burn Repository has created a large patient database accessible for research. Second, many centers across the United States have organized into a burn multicenter trial group. Their efforts have resulted in noteworthy publications on transfusion practices [85], toxic epidermal necrolysis syndrome treatment [107], and validation of oxandrolone as anabolic agent [86,108,139]. "Inflammation and the Host Response to Injury" is a major National Institute of Health-funded multicenter program that includes trauma and burn patients. This ambitious translational project aims to correlate genomic and proteomic responses to physiologic perturbations observed at the bedside. Finally, the burn injury model system is a multi-institutional project funded by the National Institute of Disability and Rehabilitation Research (http:// bms-dcc.uchsc.edu) to evaluate longitudinal outcomes after major burns. Optimally, current efforts in bench research, translational science, and outcome analyses will generate the necessary Class I evidence to create standards in burn care for the next generation.

References

- Gibran NS. Committee on organization and delivery of burn care, American Burn Association. Practice guidelines for burn care, 2006. J Burn Care Res 2006;27(4):437–8.
- [2] Klein MB, Silver G, Gamelli R, et al. Inflammation and the host response to injury: an overview of the multicenter study of the genomic and proteomic response to burn injury. J Burn Care Res 2006;27(4):448–51.
- [3] Nathens AB, Johnson JL, Minei JP, et al. Inflammation and the host response to injury: a large-scale collaborative project. Patient-oriented research core: standard operating procedures for clinical care. I. Guidelines for mechanical ventilation of the trauma patient. J Trauma 2005;59(3):764–9.
- [4] Janzekovic Z. A new concept in the early excision and immediate grafting of burns. J Trauma 1970;10(12):1103–8.
- [5] Burke JF, Bondoc CC, Quinby WC. Primary burn excision and immediate grafting: a method shortening illness. J Trauma 1974;14(5):389–95.
- [6] Engrav LH, Heimbach DM, Reus JL, et al. Early excision and grafting vs. nonoperative treatment of burns of indeterminate depth: a randomized prospective study. J Trauma 1983;23(11):1001–4.
- [7] Niazi ZB, Essex TJ, Papini R, et al. New laser Doppler scanner, a valuable adjunct in burn depth assessment. Burns 1993;19(6):485–9.
- [8] Hlava P, Moserova J, Konigova R. Validity of clinical assessment of the depth of a thermal injury. Acta Chir Plast 1983;25(4):202–8.
- [9] Yeong EK, Mann R, Goldberg M, et al. Improved accuracy of burn wound assessment using laser Doppler. J Trauma 1996;40(6):956–61 [discussion: 961–2].
- [10] Moserova J, Hlava P, Malinsky J. Scope for ultrasound diagnosis of the depth of thermal damage: preliminary report. Acta Chir Plast 1982;24(4):235–42.
- [11] Kaufman T, Hurwitz DJ, Heggers JP. The india ink injection technique to assess the depth of experimental burn wounds. Burns Incl Therm Inj 1984;10(6):405–8.
- [12] Heimbach DM, Afromowitz MA, Engrav LH, et al. Burn depth estimation: man or machine. J Trauma 1984;24(5):373–8.
- [13] Pape SA, Skouras CA, Byrne PO. An audit of the use of laser Doppler imaging (LDI) in the assessment of burns of intermediate depth. Burns 2001;27(3):233–9.
- [14] Kloppenberg FW, Beerthuizen GI, ten Duis HJ. Perfusion of burn wounds assessed by laser Doppler imaging is related to burn depth and healing time. Burns 2001;27(4): 359–63.
- [15] Pham TN, Gibran NS, Heimbach DM. Evaluation of the burn wound: management decisions. In: Herndon DN, editor. Total burn care. 3rd edition. In press.
- [16] Zawacki BE. Reversal of capillary stasis and prevention of necrosis in burns. Ann Surg 1974;180(1):98–102.
- [17] Kim DE, Phillips TM, Jeng JC, et al. Microvascular assessment of burn depth conversion during varying resuscitation conditions. J Burn Care Rehabil 2001;22(6):406–16.
- [18] Jeng JC, Bridgeman A, Shivnan L, et al. Laser Doppler imaging determines need for excision and grafting in advance of clinical judgment: a prospective blinded trial. Burns 2003; 29(7):665–70.
- [19] Chatterjee JS. A critical evaluation of the clinimetrics of laser Doppler as a method of burn assessment in clinical practice. J Burn Care Res 2006;27(2):123–30.
- [20] Estrada A, Yun CH, Van Kessel A, et al. Immunomodulatory activities of oat beta-glucan in vitro and in vivo. Microbiol Immunol 1997;41(12):991–8.
- [21] Honari S. Topical therapies and antimicrobials in the management of burn wounds. Crit Care Nurs Clin North Am 2004;16(1):1–11.
- [22] Ho WS, Ying SY, Choi PC, et al. A prospective controlled clinical study of skin donor sites treated with a 1–4,2-acetamide-deoxy-B-D-glucan polymer: a preliminary report. Burns 2001;27(7):759–61.

- [23] Delatte SJ, Evans J, Hebra A, et al. Effectiveness of beta-glucan collagen for treatment of partial-thickness burns in children. J Pediatr Surg 2001;36(1):113–8.
- [24] Atiyeh BS, Gunn SW, Hayek SN. State of the art in burn treatment. World J Surg 2005; 29(2):131–48.
- [25] Tredget EE, Shankowsky HA, Groeneveld A, et al. A matched-pair, randomized study evaluating the efficacy and safety of Acticoat silver-coated dressing for the treatment of burn wounds. J Burn Care Rehabil 1998;19(6):531–7.
- [26] Caruso DM, Foster KN, Hermans MH, et al. Aquacel Ag in the management of partialthickness burns: results of a clinical trial. J Burn Care Rehabil 2004;25(1):89–97.
- [27] Andreassi A, Bilenchi R, Biagioli M, et al. Classification and pathophysiology of skin grafts. Clin Dermatol 2005;23(4):332–7.
- [28] Archer SB, Henke A, Greenhalgh DG, et al. The use of sheet autografts to cover extensive burns in patients. J Burn Care Rehabil 1998;19(1 Pt 1):33–8.
- [29] Gallico GG III, O'Connor NE, Compton CC, et al. Permanent coverage of large burn wounds with autologous cultured human epithelium. N Engl J Med 1984;311(7):448–51.
- [30] Desai MH, Mlakar JM, McCauley RL, et al. Lack of long-term durability of cultured keratinocyte burn-wound coverage: a case report. J Burn Care Rehabil 1991;12(6):540–5.
- [31] Wood F, Liddiard K, Skinner A, et al. Scar management of cultured epithelial autograft. Burns 1996;22(6):451–4.
- [32] Burke JF, Yannas IV, Quinby WC Jr, et al. Successful use of a physiologically acceptable artificial skin in the treatment of extensive burn injury. Ann Surg 1981;194(4):413–28.
- [33] Yannas IV, Burke JF. Design of an artificial skin. I. Basic design principles. J Biomed Mater Res 1980;14(1):65–81.
- [34] Yannas IV, Burke JF, Gordon PL, et al. Design of an artificial skin. II. Control of chemical composition. J Biomed Mater Res 1980;14(2):107–32.
- [35] Fang P, Engrav LH, Gibran NS, et al. Dermatome setting for autografts to cover INTEGRA. J Burn Care Rehabil 2002;23(5):327–32.
- [36] Heimbach D, Luterman A, Burke J, et al. Artificial dermis for major burns: a multi-center randomized clinical trial. Ann Surg 1988;208(3):313–20.
- [37] Heitland A, Piatkowski A, Noah EM, et al. Update on the use of collagen/glycosaminoglycate skin substitute: six years of experiences with artificial skin in 15 German burn centers. Burns 2004;30(5):471–5.
- [38] Klein MB, Engrav LH, Holmes JH, et al. Management of facial burns with a collagen/glycosaminoglycan skin substitute: prospective experience with 12 consecutive patients with large, deep facial burns. Burns 2005;31(3):257–61.
- [39] Wainwright DJ. Use of an acellular allograft dermal matrix (AlloDerm) in the management of full-thickness burns. Burns 1995;21(4):243–8.
- [40] Gore DC. Utility of acellular allograft dermis in the care of elderly burn patients. J Surg Res 2005;125(1):37–41.
- [41] Boyce ST, Kagan RJ, Meyer NA, et al. The 1999 clinical research award. Cultured skin substitutes combined with Integra Artificial Skin to replace native skin autograft and allograft for the closure of excised full-thickness burns. J Burn Care Rehabil 1999;20(6):453–61.
- [42] Warden GD. Burn shock resuscitation. World J Surg 1992;16(1):16–23.
- [43] Baxter CR, Shires T. Physiological response to crystalloid resuscitation of severe burns. Ann N Y Acad Sci 1968;150(3):874–94.
- [44] Demling RH. The burn edema process: current concepts. J Burn Care Rehabil 2005;26(3): 207–27.
- [45] Shirani KZ, Vaughan GM, Mason AD Jr, et al. Update on current therapeutic approaches in burns. Shock 1996;5(1):4–16.
- [46] Holm C, Mayr M, Tegeler J, et al. A clinical randomized study on the effects of invasive monitoring on burn shock resuscitation. Burns 2004;30(8):798–807.
- [47] Engrav LH, Colescott PL, Kemalyan N, et al. A biopsy of the use of the Baxter formula to resuscitate burns or do we do it like Charlie did it? J Burn Care Rehabil 2000;21(2):91–5.

- [48] Friedrich JB, Sullivan SR, Engrav LH, et al. Is supra-Baxter resuscitation in burn patients a new phenomenon? Burns 2004;30(5):464–6.
- [49] Sullivan SR, Friedrich JB, Engrav LH, et al. "Opioid creep" is real and may be the cause of "fluid creep". Burns 2004;30(6):583–90.
- [50] Warner P, Connolly JP, Gibran NS, et al. The methamphetamine burn patient. J Burn Care Rehabil 2003;24(5):275–8.
- [51] Sheridan RL, Tompkins RG, McManus WF, et al. Intracompartmental sepsis in burn patients. J Trauma 1994;36(3):301–5.
- [52] Pruitt BA Jr. Protection from excessive resuscitation: pushing the pendulum back. J Trauma 2000;49(3):567–8.
- [53] Greenhalgh DG, Warden GD. The importance of intra-abdominal pressure measurements in burned children. J Trauma 1994;36(5):685–90.
- [54] Hobson KG, Young KM, Ciraulo A, et al. Release of abdominal compartment syndrome improves survival in patients with burn injury. J Trauma 2002;53(6):1129–33 [discussion: 1133–4].
- [55] Sullivan SR, Ahmadi AJ, Singh CN, et al. Elevated orbital pressure: another untoward effect of massive resuscitation after burn injury. J Trauma 2006;60(1):72–6.
- [56] Hoskins SL, Elgio GI, Lu J, et al. Closed-loop resuscitation of burn shock. J Burn Care Res 2006;27(3):377–85.
- [57] Horton JW. Free radicals and lipid peroxidation mediated injury in burn trauma: the role of antioxidant therapy. Toxicology 2003;189(1–2):75–88.
- [58] Matsuda T, Tanaka H, Williams S, et al. Reduced fluid volume requirement for resuscitation of third-degree burns with high-dose vitamin C. J Burn Care Rehabil 1991;12(6): 525–32.
- [59] Tanaka H, Matsuda T, Miyagantani Y, et al. Reduction of resuscitation fluid volumes in severely burned patients using ascorbic acid administration: a randomized, prospective study. Arch Surg 2000;135(3):326–31.
- [60] Nathens AB, Neff MJ, Jurkovich GJ, et al. Randomized, prospective trial of antioxidant supplementation in critically ill surgical patients. Ann Surg 2002;236(6):814–22.
- [61] Warden GD, Stratta RJ, Saffle JR, et al. Plasma exchange therapy in patients failing to resuscitate from burn shock. J Trauma 1983;23(10):945–51.
- [62] Masanes MJ, Legendre C, Lioret N, et al. Using bronchoscopy and biopsy to diagnose early inhalation injury: macroscopic and histologic findings. Chest 1995;107(5):1365–9.
- [63] Moylan JA Jr, Wilmore DW, Mouton DE, et al. Early diagnosis of inhalation injury using 133 xenon lung scan. Ann Surg 1972;176(4):477–84.
- [64] Park MS, Cancio LC, Batchinsky AI, et al. Assessment of severity of ovine smoke inhalation injury by analysis of computed tomographic scans. J Trauma 2003;55(3):417–27 [discussion: 427–9].
- [65] Scheinkestel CD, Bailey M, Myles PS, et al. Hyperbaric or normobaric oxygen for acute carbon monoxide poisoning: a randomised controlled clinical trial. Med J Aust 1999; 170(5):203–10.
- [66] Weaver LK, Hopkins RO, Chan KJ, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002;347(14):1057–67.
- [67] Grube BJ, Marvin JA, Heimbach DM. Therapeutic hyperbaric oxygen: help or hindrance in burn patients with carbon monoxide poisoning? J Burn Care Rehabil 1988;9(3):249–52.
- [68] Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 2000;342(18):1301–8.
- [69] Stewart TE, Meade MO, Cook DJ, et al. Evaluation of a ventilation strategy to prevent barotrauma in patients at high risk for acute respiratory distress syndrome: Pressureand Volume-Limited Ventilation Strategy Group. N Engl J Med 1998;338(6):355–61.
- [70] Cioffi WG, Graves TA, McManus WF, et al. High-frequency percussive ventilation in patients with inhalation injury. J Trauma 1989;29(3):350–4.

- [71] Cioffi WG, deLemos RA, Coalson JJ, et al. Decreased pulmonary damage in primates with inhalation injury treated with high-frequency ventilation. Ann Surg 1993;218(3):328–35 [discussion: 335–7].
- [72] Suter PM, Domenighetti G, Schaller MD, et al. N-acetylcysteine enhances recovery from acute lung injury in man: a randomized, double-blind, placebo-controlled clinical study. Chest 1994;105(1):190–4.
- [73] Palmieri TL, Enkhbaatar P, Bayliss R, et al. Continuous nebulized albuterol attenuates acute lung injury in an ovine model of combined burn and smoke inhalation. Crit Care Med 2006;34(6):1719–24.
- [74] Enkhbaatar P, Murakami K, Shimoda K, et al. The inducible nitric oxide synthase inhibitor BBS-2 prevents acute lung injury in sheep after burn and smoke inhalation injury. Am J Respir Crit Care Med 2003;167(7):1021–6.
- [75] Enkhbaatar P, Murakami K, Shimoda K, et al. Ketorolac attenuates cardiopulmonary derangements in sheep with combined burn and smoke inhalation injury. Clin Sci (Lond) 2003;105(5):621–8.
- [76] Murakami K, Enkhbaatar P, Shimoda K, et al. High-dose heparin fails to improve acute lung injury following smoke inhalation in sheep. Clin Sci (Lond) 2003;104(4): 349–56.
- [77] Nieman GF, Clark WR, Hakim T. Methylprednisolone does not protect the lung from inhalation injury. Burns 1991;17(5):384–90.
- [78] Barret JP, Desai MH, Herndon DN. Effects of tracheostomies on infection and airway complications in pediatric burn patients. Burns 2000;26(2):190–3.
- [79] Palmieri TL, Jackson W, Greenhalgh DG. Benefits of early tracheostomy in severely burned children. Crit Care Med 2002;30(4):922–4.
- [80] Hunt JL, Purdue GF, Gunning T. Is tracheostomy warranted in the burn patient? Indications and complications. J Burn Care Rehabil 1986;7(6):492–5.
- [81] Kadilak PR, Vanasse S, Sheridan RL. Favorable short- and long-term outcomes of prolonged translaryngeal intubation in critically ill children. J Burn Care Rehabil 2004; 25(3):262–5.
- [82] Gomez M, Logsetty S, Fish JS. Reduced blood loss during burn surgery. J Burn Care Rehabil 2001;22(2):111–7.
- [83] Bordin JO, Heddle NM, Blajchman MA. Biologic effects of leukocytes present in transfused cellular blood products. Blood 1994;84(6):1703–21.
- [84] Bilgin YM, van de Watering LM, Eijsman L, et al. Double-blind, randomized controlled trial on the effect of leukocyte-depleted erythrocyte transfusions in cardiac valve surgery. Circulation 2004;109(22):2755–60.
- [85] Jensen LS, Andersen AJ, Christiansen PM, et al. Postoperative infection and natural killer cell function following blood transfusion in patients undergoing elective colorectal surgery. Br J Surg 1992;79(6):513–6.
- [86] Palmieri TL, Caruso DM, Foster KN, et al. Effect of blood transfusion on outcome after major burn injury: a multicenter study. Crit Care Med 2006;34(6):1602–7.
- [87] Kahalley L, Dimick AR, Gillespie RW. Methods to diminish intraoperative blood loss. J Burn Care Rehabil 1991;12(2):160–1.
- [88] Hebert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care: Transfusion Requirements in Critical Care Investigators, Canadian Critical Care Trials Group. N Engl J Med 1999;340(6):409–17.
- [89] Palmieri TL, Greenhalgh DG. Blood transfusion in burns: what do we do? J Burn Care Rehabil 2004;25(1):71–5.
- [90] Wibbenmeyer LA, Hoballah JJ, Amelon MJ, et al. The prevalence of venous thromboembolism of the lower extremity among thermally injured patients determined by duplex sonography. J Trauma 2003;55(6):1162–7.
- [91] Wahl WL, Brandt MM, Ahrns K, et al. The utility of D-dimer levels in screening for thromboembolic complications in burn patients. J Burn Care Rehabil 2002;23(6):439–43.

- [92] Fecher AM, O'Mara MS, Goldfarb IW, et al. Analysis of deep vein thrombosis in burn patients. Burns 2004;30(6):591–3.
- [93] Horner BM, Myers SR. Don't miss HIT (heparin induced thrombocytopenia). Burns 2004; 30(1):88–90.
- [94] Scott JR, Klein MB, Gernsheimer T, et-al. Heparin-induced thrombocytopenia in burns: a retrospective review. J Burn Care Res, in press.
- [95] Herndon DN, Tompkins RG. Support of the metabolic response to burn injury. Lancet 2004;363(9424):1895–902.
- [96] Wilmore DW, Mason AD Jr, Johnson DW, et al. Effect of ambient temperature on heat production and heat loss in burn patients. J Appl Physiol 1975;38(4):593–7.
- [97] Gore DC, Chinkes D, Heggers J, et al. Association of hyperglycemia with increased mortality after severe burn injury. J Trauma 2001;51(3):540–4.
- [98] Pham TN, Warren AJ, Phan HH, et al. Impact of tight glycemic control in severely burned children. J Trauma 2005;59(5):1148–54.
- [99] Van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001;345(19):1359–67.
- [100] Van den Berghe G, Wilmer A, Hermans G, et al. Intensive insulin therapy in the medical ICU. N Engl J Med 2006;354(5):449–61.
- [101] Suman OE, Spies RJ, Celis MM, et al. Effects of a 12-wk resistance exercise program on skeletal muscle strength in children with burn injuries. J Appl Physiol 2001;91(3):1168–75.
- [102] Suman OE, Thomas SJ, Wilkins JP, et al. Effect of exogenous growth hormone and exercise on lean mass and muscle function in children with burns. J Appl Physiol 2003;94(6):2273–81.
- [103] Klein GL, Wolf SE, Langman CB, et al. Effects of therapy with recombinant human growth hormone on insulin-like growth factor system components and serum levels of biochemical markers of bone formation in children after severe burn injury. J Clin Endocrinol Metab 1998;83(1):21–4.
- [104] Takala J, Ruokonen E, Webster, et al. Increased mortality associated with growth hormone treatment in critically ill adults. N Engl J Med 1999;341(11):785–92.
- [105] Demling RH, DeSanti L. The rate of restoration of body weight after burn injury, using the anabolic agent oxandrolone, is not age dependent. Burns 2001;27(1):46–51.
- [106] Demling RH, DeSanti L. Oxandrolone induced lean mass gain during recovery from severe burns is maintained after discontinuation of the anabolic steroid. Burns 2003;29(8):793–7.
- [107] Hart DW, Wolf SE, Ramzy PI, et al. Anabolic effects of oxandrolone after severe burn. Ann Surg 2001;233(4):556–64.
- [108] Wolf SE, Edelman LS, Kemalyan N, et al. Effects of oxandrolone on outcome measures in the severely burned: a multicenter prospective randomized double-blind trial. J Burn Care Res 2006;27(2):131–9 [discussion: 140–1].
- [109] Wolfe RR, Herndon DN, Jahoor F, et al. Effect of severe burn injury on substrate cycling by glucose and fatty acids. N Engl J Med 1987;317(7):403–8.
- [110] Herndon DN, Hart DW, Wolf SE, et al. Reversal of catabolism by beta-blockade after severe burns. N Engl J Med 2001;345(17):1223–9.
- [111] Saffle JR, Crandall A, Warden GD. Cataracts: a long-term complication of electrical injury. J Trauma 1985;25(1):17–21.
- [112] Luce EA. Electrical burns. Clin Plast Surg 2000;27(1):133-43.
- [113] Mann R, Gibran N, Engrav L, et al. Is immediate decompression of high voltage electrical injuries to the upper extremity always necessary? J Trauma 1996;40(4):584–7 [discussion: 587–9].
- [114] Arnoldo BD, Purdue GF, Kowalske K, et al. Electrical injuries: a 20-year review. J Burn Care Rehabil 2004;25(6):479–84.
- [115] Arnoldo B, Klein M, Gibran NS. Practice guidelines for the management of electrical injuries. J Burn Care Res 2006;27(4):439–47.
- [116] Purdue GF, Hunt JL. Electrocardiographic monitoring after electrical injury: necessity or luxury. J Trauma 1986;26(2):166–7.

- [117] Yowler CJ, Fratianne RB. Current status of burn resuscitation. Clin Plast Surg 2000;27(1): 1–10.
- [118] Yowler CJ, Mozingo DW, Ryan JB, et al. Factors contributing to delayed extremity amputation in burn patients. J Trauma 1998;45(3):522–6.
- [119] Engrav LH, Gottlieb JR, Walkinshaw MD, et al. Outcome and treatment of electrical injury with immediate median and ulnar nerve palsy at the wrist: a retrospective review and a survey of members of the American Burn Association. Ann Plast Surg 1990;25(3):166–8.
- [120] Hunt J, Lewis S, Parkey R, et al. The use of Technetium-99m stannous pyrophosphate scintigraphy to identify muscle damage in acute electric burns. J Trauma 1979;19(6):409–13.
- [121] Sheridan RL, Hinson MI, Liang MH, et al. Long-term outcome of children surviving massive burns. JAMA 2000;283(1):69–73.
- [122] Brych SB, Engrav LH, Rivara FP, et al. Time off work and return to work rates after burns: systematic review of the literature and a large two-center series. J Burn Care Rehabil 2001; 22(6):401–5.
- [123] Costa BA, Engrav LH, Holavanahalli R, et al. Impairment after burns: a two-center, prospective report. Burns 2003;29(7):671–5.
- [124] Moore ML, Engrav LH, Vedder NB, et al. Dexter: a tool to facilitate impairment ratings. J Burn Care Rehabil 2001;22(6):397–400.
- [125] Chang P, Laubenthal KN, Lewis RW II, et al. Prospective, randomized study of the efficacy of pressure garment therapy in patients with burns. J Burn Care Rehabil 1995;16(5):473–5.
- [126] Patino O, Novick C, Merlo A, et al. Massage in hypertrophic scars. J Burn Care Rehabil 1999;20(3):268–71 [discussion: 267].
- [127] So K, Umraw N, Scott J, et al. Effects of enhanced patient education on compliance with silicone gel sheeting and burn scar outcome: a randomized prospective study. J Burn Care Rehabil 2003;24(6):411–7 [discussion: 410].
- [128] Zhu KQ, Engrav LH, Gibran NS, et al. The female, red Duroc pig as an animal model of hypertrophic scarring and the potential role of the cones of skin. Burns 2003;29(7):649–64.
- [129] Zhu KQ, Engrav LH, Tamura RN, et al. Further similarities between cutaneous scarring in the female, red Duroc pig and human hypertrophic scarring. Burns 2004;30(6):518–30.
- [130] Gallant CL, Olson ME, Hart DA. Molecular, histologic, and gross phenotype of skin wound healing in red Duroc pigs reveals an abnormal healing phenotype of hypercontracted, hyperpigmented scarring. Wound Repair Regen 2004;12(3):305–19.
- [131] Klein MB, Nathens AB, Emerson D, et al. An analysis of the long-distance transport of burn patients to a regional burn center. J Burn Care Res 2006;32(8):940–5.
- [132] Saffle JR, Edelman L, Morris SE. Regional air transport of burn patients: a case for telemedicine? J Trauma 2004;57(1):57–64 [discussion: 64].
- [133] Nguyen LT, Massman NJ, Franzen BJ, et al. Telemedicine follow-up of burns: lessons learned from the first thousand visits. J Burn Care Rehabil 2004;25(6):485–90.
- [134] Massman NJ, Dodge JD, Fortman KK, et al. Burns follow-up: an innovative application of telemedicine. J Telemed Telecare 1999;5(Suppl 1):S52–4.
- [135] Smith AC, Youngberry K, Mill J, et al. A review of three years experience using email and videoconferencing for the delivery of post-acute burns care to children in Queensland. Burns 2004;30(3):248–52.
- [136] Harrington DT, Biffl WL, Cioffi WG. The station nightclub fire. J Burn Care Rehabil 2005; 26(2):141–3.
- [137] Yurt RW, Bessey PQ, Bauer GJ, et al. A regional burn center's response to a disaster: September 11, 2001, and the days beyond. J Burn Care Rehabil 2005;26(2):117–24.
- [138] ABA Board of Trustees; Committee on organization and delivery of burn care. Disaster management and the ABA Plan. J Burn Care Rehabil 2005;26(2):102–6.
- [139] Palmieri TL, Greenhalgh DG, Saffle JR, et al. A multicenter review of toxic epidermal necrolysis treated in US burn centers at the end of the twentieth century. J Burn Care Rehabil 2002;23(2):87–96.