INTRODUCTION

Burn units have a unique resource set, including surgical and nursing wound care expertise, critical care, and high-level trauma rehabilitation capabilities. Although this resource set evolved for the care of patients with burns, it meets the needs of a large number of nonburn medical and surgical conditions. A brief description of such medical, surgical, and traumatic conditions follows.

Disclosures: None.

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TRAUMATIC CONDITIONS

Burns are severe soft tissue trauma frequently complicated by secondary inflammation, infection, and organ failures. Associated nonburn trauma is frequently seen, depending on injury mechanism. Several nonburn traumatic conditions also fit this description and require the same resource set for successful management.

Blast Injury

Blast injuries are graded complex injuries with 4 characteristics: primary injury to air-filled structures and the central nervous system; secondary injury from flying debris; tertiary injury from personnel impacting stationary objects; and quaternary injury from associated crush, burn, or blunt trauma. Blast injuries of all types can be subtle or delayed in presentation, and missed visceral injuries are especially common. Blast injury is common in war, terrorist attack, and in some high-energy industrial accidents. An understanding of the subtlety of blast injury and the frequency of missed injury is essential in successful management. Blast injury typically presents with a graded soft tissue injury and associated visceral injury from overpressure. The pulmonary and gastrointestinal injuries are particularly lethal.

Management all major soft tissue injuries includes initial hemodynamic stabilization and airway control. This topic is not discussed here. The wounds are managed with initial decompression of any tight compartments to ensure adequate perfusion. Staged debridement of devitalized soft tissue follows, which is complicated by the frequent early difficulty in discerning viable from nonviable tissues. Depending on patient stability, safety and resources of the locale, and particular characteristics of the wounds, definitive closure of wounds and rehabilitation follows. In combat or mass casualty situations, definitive care may have to wait for patient transport or distribution. During these delays, wounds can be managed with a variety of dressings or negative pressure devices. Outcomes from blast injury vary widely depending on the severity of the overall injury and the presence of associated injuries. Amputation and closed head injury are common with high-energy blast injury, but favorable outcomes can be had nonetheless.

Fragmentation Injury

Fragmentation injuries (Fig. 1) are a form of penetrating injury characterized by multiple foreign bodies with variable size, energy, and trajectory. They are commonly seen

Fig. 1. Fragmentation injury is frequently accompanied by occult injury to viscera and soft tissues. Missed injuries are common. Serial reexploration and debridement are important.
as a complication of blast. Fragmentation injury presents in a wide variety of ways depending on the missiles involved (rocks, metal shard, bone, and so forth) and projectile energy. Associated blast injury is common.

After initial hemodynamic and airway control, practical decisions must be made about which wounds to explore and debride and which to dress and observe. However, patients injured with blast-associated fragments, such as from improvised explosive devices or suicide bombs, commonly present with multiple wounds of varying size and unknown trajectory. Those associated with overt hemorrhage are explored immediately. Those associated with large soft tissue defects or likely visceral injury are explored subsequently. If none of these indications are present, some wounds are appropriately dressed and observed. Imaging helps with these often difficult decisions. Surgical exploration of all potential sites of injury is often impossible. Selected exploration is guided by initial and serial examination and by imaging when available. Observation for missed visceral injury is essential. Similar to blast injury, patients with fragmentation injury have outcomes that vary widely depending on the severity of the injuries and the presence of associated injuries.

Crush Injury

Crush injuries (Fig. 2) are graded soft tissue injuries that are associated both with immediate trauma and with secondary ischemia from compartment syndrome and ischemia-reperfusion in the hours following injury. Septic complications are common when necrotic soft tissues become infected in the days following injury. Renal failure from rhabdomyoglobinuria occurs in the days following injury in patients with large burdens of devitalized muscle. Associated bone, visceral, and vascular injuries are also common depending on the unique characteristic of individual injuries. Crush injuries present with a wide variety of severity and pattern, depending on the mechanism of the individual’s injury. Concomitant injuries are common, such as penetrating injury from rebar in building collapses. A major unique feature in presentation variety involves duration of crush, presence of open wounds, presence of bony trauma, rhabdomyoglobinemia, compartment syndrome, and reperfusion injury.

After initial hemodynamic and airway control, practical decisions must be made about which extremities to decompress, which open wounds to explore and debride, and whether treatment of rhabdomyoglobinuria or renal failure is needed. Bony injuries

![Crush injury](image)
should be investigated radiographically and stabilized, generally initially with splinting and/or external fixation. The need for exploration and debridement of open wounds is usually straightforward. The most difficult decisions relate to the need for decompression. Compartment syndromes of extremities can occur early (within a few hours) from direct muscle injury and edema or later (hours to 1 or 2 days) from progressive edema related to reperfusion injury. If ischemia is active in a compartment syndrome, decompression is always beneficial. However, there are some circumstances in which decompression may be harmful. If compartment syndrome has been missed, with edema and ischemia having come and then passed, opening the compartments may cause infection and necessitate amputation of an extremity that might have gone on to develop a Volkmann contracture. This situation is most commonly seen in disaster situations in which late presentation (days) is common after extrication from a collapsed building. In addition, a sensitivity to the common occurrence of rhabdomyoglobinuria is important. If not treated, which is particularly common in those presenting late, renal failure will occur, which is common in disasters involving building collapses and large numbers of casualties. A dialysis capability should be planned for in this clinical setting. Patients with crush injuries have outcomes that vary widely depending on the severity and pattern of the injuries and the presence of associated injuries. Many such patients need amputation and long-term prosthetics and rehabilitation.

**Degloving Injury**

Degloving injuries are a loosely defined class of soft tissue avulsions. The injury mechanisms are varied, as are the specific patterns and severity of individual injuries. Septic complications are common when necrotic soft tissues become infected in the days following injury. Associated bone, visceral, and vascular injuries are common depending on the unique characteristic of individual injuries. Degloving injuries present with a wide variety of severity and pattern, depending on the mechanism of the individual’s injury. Concomitant injuries are common. A major unique feature in presentation involves shearing of physically attached adjacent soft tissues with disruption of small vessels and secondary tissue ischemia. This results in progressive necrosis of adjacent soft tissues that may have appeared viable initially.

After initial hemodynamic and airway control, stabilization of associated fractures, and repair of any associated major vascular injuries, practical decisions must be made about the extent of debridement needed for ischemia of adjacent soft tissue. Because of the shearing nature of most major soft tissue avulsions, small vessel disruption and progressive ischemia of these marginal soft tissues is common, which argues for planned second-look and third-look procedures to be certain that large amounts of nonviable tissue are not left behind after the initial debridement. Once definitive debridement is completed, wound closure must follow. The simplest closure is ideally achieved initially, often using split-thickness skin grafting. Functional and aesthetic reconstruction then follows. When considering free flaps as part of initial closure or reconstruction, the common occurrence of regional partial vascular disruption should be considered.

**Combined Burns and Trauma**

It is common for thermal and mechanical trauma to occur together. This combination is particularly common in high-energy motor vehicle or industrial accidents or trauma associated with explosives. Combined burns and trauma present with a wide variety of severity and pattern, depending on the mechanism of the individual’s injury. A
common occurrence when caring for patients with combined burns and trauma involves adjudicating conflicting management priorities. The key to optimal management is thoughtful identification and balancing of these conflicts (Table 1).

**ENVIRONMENTAL EXPOSURES**

**Electrical Injury**

High-voltage electrical burns (Fig. 3) can be severe, but account for only 3% to 7% of admissions to burn centers. These injuries tend to occur in young working men so there is a high potential for loss of productive lives. There are 3 injury subtypes associated with high-voltage injury: arcing, flame, and true electrical burns. An arc injury is the result of rapid ionization of current in an electrical device. The ionized air may reach 4000°C but exposure is brief. Arc and electrical current may ignite clothing and produce flame injury. True electrical injuries are the result of current coursing through the tissues. The severity of injury after an electrical burn depends on voltage, current, type of current (alternating current [AC] or direct current), path of current, duration of exposure, and whether the tissues are wet or dry.

### Table 1

<table>
<thead>
<tr>
<th>Issue</th>
<th>Problem</th>
<th>Resolution</th>
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<tbody>
<tr>
<td>Intracranial pressure</td>
<td>Fluid resuscitation for burn can exacerbate cerebral edema</td>
<td>Tightly control resuscitation</td>
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<tr>
<td>monitoring</td>
<td>Infection more likely through or near burn</td>
<td>Place only when absolutely needed (no examination), try to place through unburned scalp, remove as soon as possible, prophylactic antibiotics indicated</td>
</tr>
<tr>
<td>Chest tubes</td>
<td>Chest tubes through burn are more prone to infection and can cause empyema</td>
<td>Try to place through unburned skin, remove as soon as possible, consider prophylactic antibiotics based on wound flora</td>
</tr>
<tr>
<td>Pain control in rib fractures</td>
<td>Epidural catheters have higher risk of infection in or near burn</td>
<td>Place only when absolutely needed, rely on parenteral narcotics, try to place through unburned skin, remove as soon as possible, prophylactic antibiotics indicated</td>
</tr>
<tr>
<td>Diagnosis of intra-abdominal</td>
<td>Overlying burn and pain medications and hyperdynamic state may mask intra- abdominal injury</td>
<td>Liberal use of CT in stable patients with appropriate mechanism, FAST preferred to DPL to look for intraperitoneal blood because it is noninvasive</td>
</tr>
<tr>
<td>injury</td>
<td></td>
<td>Careful monitoring, serial FAST and/or CT, operate for severe injuries</td>
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<tr>
<td>Nonoperative management</td>
<td>Observation for nonoperative management difficult with overlying burn and ongoing burn resuscitation</td>
<td>Early external fixation and simultaneous grafting may be optimal</td>
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<tr>
<td>of intra-abdominal injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fractured and burned</td>
<td>Internal fixation through burn has higher risk of infection</td>
<td></td>
</tr>
<tr>
<td>extremity</td>
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</table>

Abbreviations: CT, computed tomography; DPL, diagnostic peritoneal lavage; FAST, focused assessment with sonography for trauma.
contact, resistance at the point of contact, and individual susceptibility. Low-voltage electrical injury is classified as less than 1000 V and can damage tissue at the contact site. High voltage, characterized as more than 1000 V, leads to damage that extends into the underlying tissues. AC may cause tetanic muscle contraction ventricular fibrillation. Muscle injury may lead to myoglobinuria and renal failure if not addressed.

Patients with major electrical burns should have an individualized fluid resuscitation to produce a target urine output of 0.5 mL/kg/h (1 mL/kg/h in children <20 kg). Deep tissue injury makes formula-based resuscitation generally inadequate and the surface burn is only a portion of the injury. If the urine is pigmented the urine output target should be increased to twice the typical targets (1–2 mL/kg/h) until the urine color normalizes. Alkalinizing the urine with sodium bicarbonate is also somewhat protective for the kidneys.

Compartment syndromes are a potential complication and patients should be monitored and decompressed. After initial decompression, wound management of high-voltage injuries often requires staged debridement, because the extent of myonecrosis is often difficult to ascertain initially. Because there is ongoing capillary thrombosis, myonecrosis can extend over time. Wound closure often requires creative use of grafts and local or distant flaps. High-voltage electrical burns can be severe injuries that result in multiple amputations. Returning to work can be difficult. Each patient presents a different set of needs, mandating an individualized approach to each. In most cases, satisfactory functional outcomes can be achieved.

Chemical Injury

Chemical burns constitute less than 3% of burn center admissions. Most occur in the workplace, but some are secondary to assault, typically to the face. Injury severity depends on quantity of chemical, concentration, duration of contact, penetration, and mechanism of action.6 Mechanism of action is divided into 6 categories: oxidation, reduction, corrosion, protoplasmic poisoning, blistering, and desiccation.

Although most common chemical injuries are local only, hydrofluoric acid can cause both local tissue injury and systemic effects.7 Damage is caused by both release of the hydrogen ion and penetration of the fluoride ion (F⁻)10, which binds Ca²⁺ and
Mg++ and thus interferes with essential cellular functions. Sizable exposures can lead to significant hypocalcemia and hypomagnesemia, with their associated systemic effects. Initial presentation with dilute exposures can be insidious.

The initial management of any chemical exposure is to eliminate the agent quickly without contaminating direct care providers. Neutralizing agents are generally not advised. Wounds that initially appear superficial may progress to full thickness, mandating serial examination. Treatment of eye exposure is by extensive irrigation. The initial care of hydrofluoric acid burns is irrigation. Because fluoride anion binds calcium most wounds are treated with a 10% calcium gluconate gel that is applied topically over the burn site. On rare occasions, injection of calcium gluconate into the subcutaneous tissues beneath the burn is indicated. Very rarely, proximal arterial infusion or Bier block with calcium gluconate is useful. The insidious nature of some chemical injuries allows them to progress for prolonged periods so that an initially superficial-appearing wound ultimately requires surgery. This progression occurs with burns from alkali and hydrofluoric acid, which tend to penetrate the tissues for prolonged periods.

**Radiation Injury**

The incidence of radiation burns is very low. The most common radiation injuries result from radiation therapy for malignancies, but most are superficial. Radiation burns caused by large exposures may lead to immediate signs of injury, starting with erythema followed by blisters. These severe exposures may progress rapidly to full-thickness skin loss. More commonly, people are exposed to lower doses that develop erythema over hours to days, followed by gradual blister formation. They may then progress to full-thickness injuries that fail to heal over prolonged periods. The initial care of radiation burns is similar to that of any other thermal injury. Rare patients have systemic radiation effects. Because radiation causes damage to DNA, there are often long-term problems with prolonged breakdown of the local tissue. These wounds often develop chronic skin changes and there may be persistent pain issues. There are also theoretic risks for malignancies in these tissues.

**Tar Injury**

Tar injuries usually occur during road and roofing work. The burn usually occurs as a spill, splash, or from slipping an extremity into a container of hot tar. Splatter marks are commonly seen with splashes or spills. Tar sticks to the skin and holds the heat against the tissues for prolonged periods, generally resulting in deep burns. Initial management mandates immediate cooling. It is a mistake to try to peel the burn from the skin because it injures the underlying tissue. Tar dissolves with lipophilic solvents or any oil-based ointment. After removing the tar, management is no different than for any other thermal burn. Because of the temperature and viscous nature of tar, these injuries are generally full-thickness injuries.

**Cold Injury**

Cold injuries (Fig. 4) often occur in homeless, elderly, or intoxicated people who have reduced ability to respond to the cold. They also occur during expeditions into cold areas such as for mountain climbers, skiers, or military personnel in cold regions. Cold injury can be described in 3 stages (Table 2). Frostbite tends to occur in the distal extremities or exposed areas of the face. At first, the wound may present with blisters that do not appear to be deep. There is often progression of the wounds over days to weeks because of injury to the microvasculature. Blisters often progress to full-thickness loss, and toes and fingers may mummify over prolonged periods.
These patients often present with hypothermia caused by the prolonged exposure to the cold.

A freeze-thaw-refreeze sequence results in worse injury than a longer duration of freeze, so involved parts should not be warmed if there is significant risks for refreezing during transport. After arrival to hospital, all patients should be evaluated for hypothermia and treated appropriately. The local areas should be rewarmed in water at 37°C to 40°C. In some cases, if ischemic frozen parts do not reperfuse after thaw, angiography, thrombolysis, and anticoagulation may be warranted.11 Wound management is general conservative, allowing ischemic tissues to demarcate before excision. Like high-voltage injuries, wound closure often requires creative use of grafts and local or distant flaps.

**MEDICAL AND INFECTIOUS PROBLEMS**

**Soft Tissue Infections**

Patients with serious skin and soft tissue infections benefit from the critical care, wound management, and rehabilitation expertise available in burn programs.12 The decision to explore an infected area, often presenting as a severe cellulitis, can be difficult before the development of overt soft tissue gas and bullae or frank systemic toxicity. When in doubt, imaging and/or direct exploration of involved muscle compartments can facilitate early excision of necrotic infected tissues and improve survival and limb salvage.13

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**Table 2**

<table>
<thead>
<tr>
<th>Stages of Frostbite</th>
<th>Clinical Features</th>
<th>Response to Rewarming</th>
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<tbody>
<tr>
<td>Stage 1</td>
<td>Burning and numbness, pallor</td>
<td>Erythema and discomfort</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Insensate, pallid</td>
<td>Blistering and pain with restored perfusion</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Insensate, pallid, may be frozen and hard</td>
<td>Hemorrhagic blisters, variable pain and variable perfusion, potential tissue necrosis</td>
</tr>
</tbody>
</table>
Staphylococcal Scalded Skin Syndrome

Staphylococcal scalded skin syndrome (SSSS) is a reaction to a staphylococcal exotoxin that causes a separation at the granular layer of the epidermis.\textsuperscript{14} It is most commonly seen in infants and very young children. The exotoxin can be released from areas of colonization; frank staphylococcal infection is not required. The superficial wounds generally heal quickly if superinfection and desiccation can be prevented through bland wound care. Mucous membrane and conjunctival involvement are not seen. This finding is important and helps to differentiate SSSS from toxic epidermal necrolysis. Elimination of any focal infection is an important component of therapy. Empiric antistaphylococcal antibiotics are justified.

Purpura Fulminans

Purpura fulminans (PF) is less often seen in the era of routine meningococcal vaccination.\textsuperscript{15} It is a complication of bacteremia (most commonly with meningococcus) in which large areas of skin and soft tissue undergo necrosis secondary to microvascular thrombosis, which is thought to be related to transient sepsis-induced protein C deficiency. These patients are usually children or young adults and they present with sepsis-induced hypotension and organ failure. Patients may present with early signs of meningeal inflammation and a rash. The condition progresses rapidly, with deterioration to hypotensive shock and multiple organ failure in hours. Prognosis is poor without prompt antibiotic treatment. The role of early anticoagulants, protein C concentrate, vasodilators, and thrombolytic agents is unclear.\textsuperscript{16} Once hemodynamic stability is attained, necrotic tissue should be excised and wounds closed, which may require amputation and creative use of grafts and flaps.

Toxic Epidermal Necrolysis

Toxic epidermal necrolysis is a diffuse slough at the dermal-epidermal junction involving variable areas of skin, mucous membranes, eyes, and hollow viscera (Fig. 5). It is often associated with a prior drug administration or viral illness.\textsuperscript{17} Differentiation from SSSS or a variety of other drug eruptions can sometimes be challenging. In these cases skin biopsy can be useful.\textsuperscript{18} Treatment involves prevention of wound desiccation and superinfection with topical antimicrobial agents and selective use of wound membranes. Ophthalmologic care is critical to minimize long-term eye morbidity. Patients with severe

Fig. 5. Toxic epidermal necrolysis results in a diffuse separation at the epidermal-dermal junction of the skin, eyes, and hollow viscera.
oropharyngeal involvement often require intubation for airway protection and enteral tube feeding for nutritional support. Septic complications can follow involvement of the gut and genitourinary tract. Survivors generally have an excellent prognosis, although skin, nail, and eye morbidity is common.19

**Epidermolysis Bullosa**

Epidermolysis bullosa (EB) is an inherited skin disorder caused by defective anchoring of the epidermis to the dermis.20 There are cases of autoimmune acquired EB with similar manifestations.21 There is a broad range of severity, with a large number of subtypes. However, most patients can be classified by examination and skin biopsy as either a simplex, junctional, or dystrophic variant, with increasing severity of disease. This disorder is a multisystem lifelong condition that requires compassionate long-term care. Family support is particularly important.22 Peer support has been especially helpful through such networks as the Dystrophic Epidermolysis Bullosa Research Association (www.debra.org).

**REFERENCES**