Chapter 12
Management of the Critically Ill Burns Patient

Anthony D. Holley

Abstract Burn injuries in military personnel during combat are well recognized, historically accounting for approximately 5–20 % of conventional warfare military casualties. It is therefore vitally important that military health professionals have an effective strategy for caring for these patients. The initial approach is the same as for all major trauma, with special attention to airway patency given the risk of airway burns. The respiratory assessment and management must address the potential for significant inhalational injury. The pathophysiology of major burns is complex leading to shock, which is predominantly hypovolaemic in origin, but often

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multifactorial. Fluid resuscitation should proceed in a timely and predetermined manner with carefully selected goals/endpoints. The consequences of both under and over resuscitation must be appreciated by the clinician and the need for escharotomy or fasciotomy identified. The mechanism of injury must be considered such that special attention can be provided in the setting of chemical and electrical burns. The surgical principles of deep burn management require early debridement and subsequent surface cover. The debrided areas are ultimately skin grafted, but in the interim maybe covered by a range of biological or synthetic products. The burn patient is at risk of a wide range of complications and therefore requires meticulous intensive care management. This chapter provides a comprehensive strategy for the care of a burns victim.

Keywords Burns • Pathophysiology • Resuscitation • Inhalation • Airway • Assessment • Military • Fluids • Ventilation

Introduction

Burn injuries in military personnel during combat are well recognized. In modern conventional conflicts injuries commonly include burns from explosions or penetrating injuries from small arms fire. Historically burn injuries account for approximately 5–20 % of conventional warfare military casualties [1]. During World War II, 1.5 % casualties resulted from burns injury [2, 3]. In the Korean War, burns accounted for 1 % of all battle casualties increasing to 4.6 % in the Vietnam War. The nature of the conflict has substantial impact on injury patterns [4]. In the Yom Kippur Israeli War, characterised by tank battles, 8.1 % of the casualties' sustained burns. Similarly, in the Falklands War, there was a comparatively high incidence of burn injuries seen (14 %) most of which occurred in the setting of burning ships. Burn injuries constituted 2.5 % of casualties from the recent

Afghanistan War [5]. Despite a significant reduction in the lethality of burn injuries over the last three decades resulting from rapid, focused resuscitation and advances in surgical strategies, burns can be devastating injuries which require substantial resources and protracted treatment [6]. It is reasonable for military health care providers to anticipate an increasing incidence of burns casualties with the changing nature of war. Optimal care of the critically burned patient requires prompt assessment of the patient and evaluation of the burn.

Table 12.1 summarises the potential mechanisms of burn injury.

What Is the Pathophysiology of Burn Injury?

The "Jackson Burn Wound Model" assists in understanding the pathophysiology of a burn wound at a local level. In 1947 Jackson described three zones of a burn wound. The primary injury is the **zone of coagulation** nearest the heat source [7]. This zone has irreversible tissue necrosis at the centre of the burn due to exposure to heat, chemicals or electricity. The extent of this injury depends on duration of exposure and the maximal temperature (or concentration). Immediately adjacent to the central zone of necrosis, is the **zone of stasis** in which there is a reduced dermal perfusion, this tissue although damaged is potentially viable.

The principle goal of burn resuscitation is to increase tissue perfusion to this area and prevent the damage from becoming irreversible [8]. This ischaemic zone may progress to full necrosis unless perfusion is re-established [9, 10]. At the periphery of the burn is a third zone, the **zone of hyperaemia** characterised by a reversible increase in blood flow and inflammation. It is important to conceptualise these three zones of a burn in three dimensions (Fig. 12.1) and so if there is further loss of viable tissue it results in both wound deepening and surface extension [11].

The pathophysiology of large surface area burns (>20 %) is extremely complex and requires a detailed understanding

Table 12.1 Mechanisms of burn injury

Mechanism	Situation
Flame burns	Secondary to burning fuel, vehicles, buildings, or shelters that were ignited by explosives. Often associated with inhalational injury and other concomitant trauma. Tend to be deep dermal or full thickness
Flash injury	High temperatures generated by explosives
Incinerating materials	Napalm or phosphorus munitions
Contact burns	Hot environmental objects
Steam or hot liquid burns	Steam or hot fluids released by damaged machinery
Chemical burns	Agents of warfare or leakage of chemicals used/stored in the immediate environment. Chemical burns tend to be deep, as the corrosive agent continues to cause coagulative necrosis until completely removed. Alkalis tend to penetrate deeper and continue damaging tissue for a longer duration than acids.
Electrical/laser burns	Equipment accidents.
Radiation burns	Exposure to nuclear weapons.

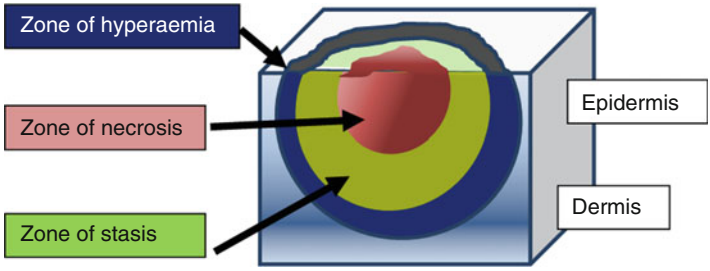


Figure 12.1 Schematic representation of local burn zones

Table 12.2 Factors influencing the degree of "Burn Shock" which may develop following injury

Patient age
Depth of burn
Surface extent of burn
Presence of inhalation injury
Need for escharotomies/fasciotomies
Time delay to resuscitation
Associated injuries

in order to facilitate appropriate resuscitation. Patients are at risk of developing "burn shock" is if they have greater than 20 % of their total body surface area (TBSA) involved with deep or partial thickness burns [12, 13]. The severity of the shock not only depends on the depth and extent of the burn [14], but also on other factors as shown in Table 12.2.

Burn pathophysiology is characterised by a series of predictable phases [14]. The principle insult is the loss of the integument and hence body fluid and temperature homeostasis. The initial phase of injury is characterized by increased capillary permeability and transmembrane cellular changes. The increased capillary permeability results from a massive and sustained cytokine release into the systemic circulation following injury and includes vasoactive substances, interleukins, histamine and prostaglandins [14]. This results in a distributive shock pattern, which is further exacerbated by fluid loss into the interstitium [15]. Increased capillary permeability, mediated by these cytokines, also results in a loss of intravascular proteins and oncotic pressure [16]. Concomitantly the interstitial oncotic pressure may rise, further encouraging volume loss from the intravascular compartment. It is highly likely that this initial "cytokine storm" may have directly negative effects on both myocardial and renal function [14, 17–19]. The cytokine mediated decrease in myocardial contractility leads to a depressed cardiac output which is further exacerbated by impaired intracellular calcium homeostasis. These factors are summarized in Fig. 12.2

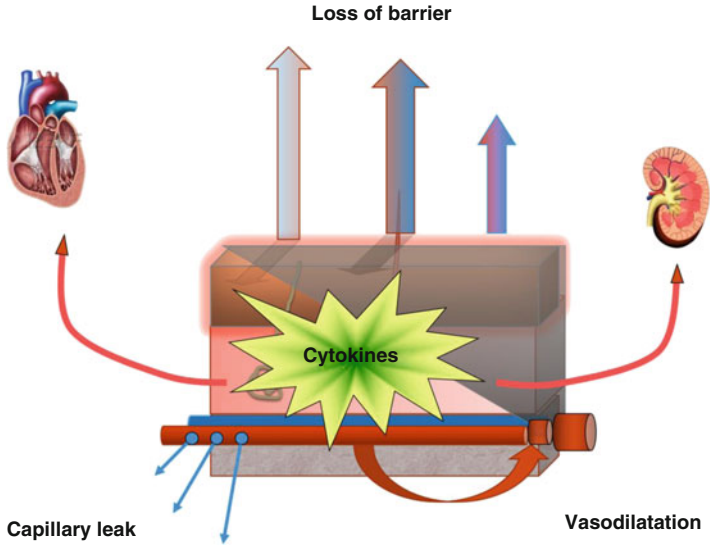


FIGURE 12.2 Burn shock: local and systemic effects of severe burns leading to multifactorial shock

How Should the Patient with Severe Burns Be Assessed?

The assessment of a burns patient is challenging and requires a disciplined and methodical approach. The approach is no different to any other patient with severe multi trauma, but is then followed up by a detailed evaluation of the depth and extent of the burn. See Table 12.3. In order to make an accurate assessment of the burn severity the clinician requires experience, but more importantly diligence. The evolving nature of a burn makes it essential to reassess the wounds frequently within the first 48 h as the exudate and oedema start to resolve.

Table 12.3 General approach to burns resuscitation

Primary survey	Assessment of the airway, breathing, circulation, neurological status with full exposure.
Adjuncts to primary survey	Securing reliable intravenous access. Insertion of urinary catheter
Secondary survey	Detailed clinical examination detecting all injuries
Detailed evaluation of the burn	Depth- very rarely homogeneous so all anatomical areas require evaluation. Extent- TBSA (%)

Assessing Burn Depth

Establishing the depth of a burn is critical to developing a formal resuscitation plan [20, 21] and is important in determining the potential for subsequent wound healing and prognosis. The history and injury pattern often afford the clinician a good indication as to the likely severity of injury. On examination of the burn the extent and speed of capillary refill is useful in helping establish burn depth. The clinician needs to be cognisant that burn wound evolution frequently results in increased depth of burn injury when subsequently reviewed. Depending on the depth of tissue damage, burns are classified as either epidermal, superficial dermal partial thickness, deep dermal partial thickness or full thickness as shown in Table 12.4.

Superficial burns and severe deep burns are readily diagnosed; it is those of intermediate or mixed thickness that are more problematic. It is also important to recognise that most burn wounds, especially those involving larger surface areas, are not homogenous in depth. They are often a composite of areas of different depths and only diligent, frequent clinical examination will establish the true extent of injury [13].

Assessing Burn Area

The establishment of burn surface area is an important component of the initial assessment and is critical in determining resuscitation requirements and the need for transfer to a specialist burns centre [22]. The extent of injury is documented using the percentage of the total body surface area that is affected by the burn. It is important that erythematous areas (superficial burn) are not be included when calculating burn area [7].

Several studies have compared a range of methods of estimating burn surface area. The surface area of a patient's palm (including fingers) is about 1 % total body surface area (Fig. 12.3). The accuracy of this method, however tends to decline when used to estimate larger surface areas. It remains a useful strategy in difficult or austere environments.

The Lund & Browder charts (Fig. 12.4) appear more accurate than either the Wallace Rule of Nines (Fig. 12.5) or palm size in determining the percentage of body surface area involved in a burn [7]. The Rule of Nines is however convenient and easily applied in burn patients with the caveat that it is potentially less accurate in children or obese people.

How Should the Patient with Severe Burns Be Resuscitated?

Initial Management

The presentation of severe burn injuries can be startling even for experienced clinicians and they therefore have the potential to be the ultimate distracting injury. Frequently other injuries may coexist, especially when there is a history of:

- Blast or explosion
- Motor vehicle accident
- High voltage electrical injury

It is therefore vital that the attending clinician conduct an effective primary and secondary survey. The patient must be removed from any sources of heat or ongoing thermal injury. Any clothing that is burned, covered with chemicals, or that is constricting must be removed with due attention to the medical providers own safety. It is also important to recognise that inhalation of the toxic products of combustion and hypoxia may cause a decreased level of consciousness.

Burns that are less than three hours old should be cooled using standard tap water, if available (15–18 °C is adequate) for at least 20 min [23] and then the patient should be carefully dried. The patient should then be covered with a clean

Table 12.4 Classifying burn depth and its impact on management and prognosis

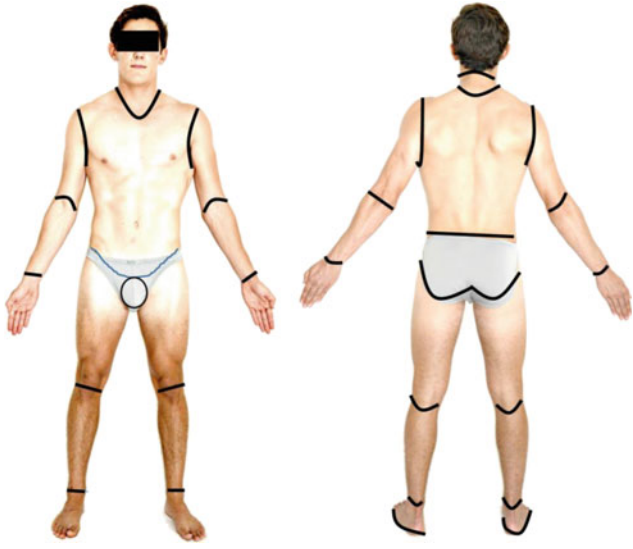
Burn depth	Clinical features	Prognosis
Superficial epidermal burns: Involves the epidermis	Hyperaemic, erythematous and painful. Blister formation and skin desquamation are delayed for few days.	The stratified layers of the epidermis are lost and healing occurs by regeneration of the epidermis from the basal layer. Heals within 7 days without scarring.
Superficial dermal partial thickness burns: Involves the epidermis And Papillary dermis. Skin overlying the blister is dead and separated from the base by inflammatory oedema fluid	Very painful secondary to sensory nerve exposure. Capillary return brisk with preserved vasculature. Blisters characteristic- on the pink papillary dermis is exposed. Desiccation of exposed dermis can increase the depth of tissue loss.	Spontaneous healing by epithelialisation within 14 days.

(continued)

Table 12.4 (continued)

Burn depth	Clinical features	Prognosis
Deep dermal partial thickness burns: Extensive destruction of the dermal vascular plexus. Exposed reticular dermis. The dermal nerve endings are also damaged.	These burns tend to be dry, with diminished fluid exudates. Characterised by the early (within hours) development of extensive blisters, which rupture early to expose deep damaged dermis. Exposed reticular dermis is pale in colour due to damage to dermal blood vessels or red secondary to extravasation of red blood cells from damaged vessels. Markedly decreased capillary return, with no or sluggish blanching. Sensation is reduced	Requires grafting in order to heal
Full thickness burns: Epidermis and dermis destroyed. May penetrate more deeply into underlying structures- fat, muscle and bone.	Dense white, waxy or even charred appearance. The sensory nerves in the dermis are destroyed in a full thickness burn, and so sensation is lost. The coagulated dead skin, which has a leathery appearance is referred to as an eschar.	Requires grafting in order to heal

FIGURE 12.3 Palmer surface for estimating burn size



	Birth 1 year	1–4 years	5–9 years	10–14 years	15 years	Adult	Burn size estimate
Head	19	17	13	11	9	7	
Neck	2	2	2	2	2	2	
Anterior trunk	13	13	13	13	13	13	
Posterior trunk	13	13	13	13	13	13	
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5	
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5	
Genitalia	1	1	1	1	1	1	
Right upper arm	4	4	4	4	4	4	
Left upper arm	4	4	4	4	4	4	
Right lower arm	3	3	3	3	3	3	
Left lower arm	3	3	3	3	3	3	
Right hand	2.5	2.5	2.5	2.5	2.5	2.5	
Left hand	2.5	2.5	2.5	2.5	2.5	2.5	
Right thigh	5.5	6.5	8	8.5	9	9.5	
Left thigh	5.5	6.5	8	8.5	9	9.5	
Right leg	5	5	5.5	6	6.5	7	
Left leg	5	5	5.5	6	6.5	7	
Right foot	3.5	3.5	3.5	3.5	3.5	3.5	
Left foot	3.5	3.5	3.5	3.5	3.5	3.5	

Total BSAB _____

FIGURE 12.4 The Lund & Browder Chart

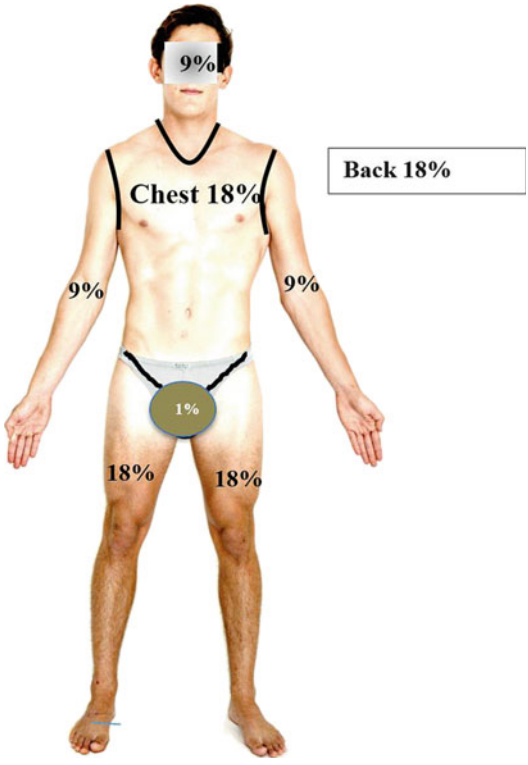


FIGURE 12.5 Wallace Rule of Nines

dry sheet or blanket to prevent hypothermia and all rings and constricting jewellery or garments must be removed.

Airway

The patient's airway remains the priority, and this should include protection of the cervical spine if the mechanism of injury is conducive to vertebral injury. The principle airway concern in the burns patient is the risk of obstruction due to subsequent swelling of the oropharynx and soft tissues of the neck. It is now recognised that more patients than actually

Table 12.5 Indications for intubation in burns patients

Stridor is an immediate indication for intubation
Difficulty with phonation is an immediate indication for intubation
Uncooperative/combative/disoriented patient
Significant oral/facial burns
Prophylactic' intubation prior to transfer if history or signs indicate likelihood of inhalation and thus possible airway obstruction
Increasing swelling of head and neck
Unprotected airway

require intubation are intubated, but this situation is preferable to a more conservative approach that may result in delayed unsalvageable airway obstruction [24]. It is important to recognise that airway injury/swelling may not be recognised acutely, but becomes progressively more obvious with time and fluid resuscitation. There are several clinical signs or situations that mandate early intubation as shown in Table 12.5.

A wide range of agents and combinations are available for induction of anaesthesia, including Ketamine, Propofol, Fentanyl and Midazolam, the anaesthetic combination utilised however, needs to account for the significant potential to induce profound hypotension secondary to the loss of sympathetic response in the setting of volume depletion and vasodilatation [25]. The choice of muscle relaxant is similarly extensive, importantly it is safe to use suxamethonium in the first 24 h. The intubating physician must be fully prepared to encounter a difficult airway and be capable and confident of proceeding to a surgical airway [26].

Following successful intubation the endotracheal tube needs to be reliably secured, initially with anaesthetic ties. It is our practice to insert alveolar ridge screws (Fig. 12.6) during the patient's first visit to the operating theatre. This provides a reliable strategy for securing the airway, with the added advantage of avoiding ongoing trauma to facial burns.

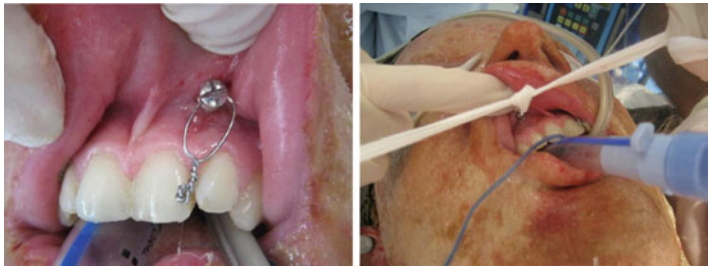


FIGURE 12.6 Alveolar screw providing a secure attachment of the endotracheal tube

TABLE 12.6 Features suggestive of inhalational injury

Facial burns or singed facial/nasal hair is present
Stridor, hoarse voice
Intra- oral oedema
Carbonaceous sputum
Soot or mucosal inflammatory changes in the mouth or nose
The patient was in a confined space at the time of accident

Ventilation

All patients should be evaluated for possible inhalation injury, this includes a detailed review of the history and a reliable clinical examination. Features suggestive of inhalational injury are shown in Table 12.6.

Bronchoscopy

Early bronchoscopy can be valuable in evaluating the extent/presence of inhalation injury [27]. *Masanes* used biopsy histologic findings as the "gold standard," to evaluate the utility of bronchoscopy [28, 29]. Bronchoscopy proved to be sensitive (sensitivity, 0.79) and highly specific (specificity, 0.94) for the

Table 12.7 Bronchoscopic grading of inhalation injury [30]

Grade	Severity	Features on bronchoscopy
Grade 0	No injury	Absence of carbonaceous deposits, erythema, oedema, bronchorrhea, or obstruction.
Grade 1	Mild injury	Minor or patchy areas of erythema, carbonaceous deposits in proximal or distal bronchi.
Grade 2	Moderate injury	Moderate degree of erythema, Carbonaceous deposits, bronchorrhea, with or without compromise of the bronchi.
Grade 3	Severe injury	Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea, bronchial obstruction.
Grade 4 [30]	Massive injury	Evidence of mucosal sloughing, necrosis, endoluminal obliteration.

diagnosis of inhalation injury and was more reliable than the circumstances of the injury or the clinical findings. In this study bronchoscopy-proven inhalation injury was one of the most strongly predictive variables for the onset of ARDS and death. *Endorf* and *Gamelli* [30] developed a grading system for inhalational injury (Table 12.7) that provides an objective and clinically useful approach to evaluating the patient with suspected inhalation.

Ventilatory Strategy

There is no ideal respiratory support strategy for the patient with inhalation injury or indeed with severe burns. A practical approach is to provide a protective lung strategy that delivers tidal volumes of 4–6 ml/kg, avoids plateau pressures greater than 30 cmH_2O and avoids hyperoxia. Limitation of pressure, acceptance of permissive hypercapnia and strategies to man-

age secretions are all important. Patients with smoke inhalation are at significant risk of developing ventilator associated pneumonia or acute respiratory distress syndrome [31].

Standard strategies to mitigate this risk should be employed and include: elevation of the head of the bed to 30–45°, frequent position changes and fastidious oral care. Antibiotic prophylaxis has no role and may increase infection rates. There may be a role for extracorporeal membrane oxygenation as an extreme rescue therapy, however there is currently insufficient evidence to make firm recommendations. Prone ventilation can be logistically challenging, but is effective and practical in the hypoxic patient [32].

The efficacy of aerosolised heparin in the adult burn and inhalation injury population remains unclear, however a number of centres continue to use regular nebulised heparin in order to decrease the tenacity of secretions and provide an anti-inflammatory effect [33].

The burn patient may have mechanical factors that impede respiration or precipitate respiratory failure. Deep circumferential burns of the chest or abdomen can restrict chest expansion sufficiently to compromise ventilation and require escharotomy as demonstrated in Fig. 12.7.

What Is an Appropriate Fluid Resuscitation Strategy Following Severe Burn Injury?

While conducting the primary survey reliable intravenous access must be established. Intravenous cannulas may be placed through the burned area if there is no other viable option, it is then necessary to suture the cannula in place to prevent dislodgement. Intraosseous access is an excellent short term alternative while more reliable access is achieved.

The extent of the burn is assessed with respect to percentage body surface area involved and depth. This allows for utilisation of one of the many formulae available (Table 12.8) to guide the initial resuscitation [34, 35].

Figure 12.7 Escharotomy for circumferential chest burn compromising ventilation

Table 12.8 Examples of burn resuscitation formulae

Formula	Strategy
Brooke	First 24 h Lactated Ringers at 1.5 ml/kg/% TBSA burn and colloid at 0.5 ml/kg/% TBSA burn.
Modified Brooke	First 24 h: Lactated Ringers at 2 ml/kg/% TBSA burn. Half in the first 8 h and half in the remaining 16 h.
Parkland	First 24 h Lactated Ringers at 4 ml/kg/%TBSA. Half in first 8 h and half in the remaining 16 h.
Rule of tens	%TBSA × 10 = Initial Fluid Rate mL/h For every 10 kg > 80 kg increase rate by 100 mL/h

The Parkland formula remains the most commonly used formula in the United Kingdom, Ireland, United States and Canada where clinicians report using this formula in 78 % of all burns cases [36].

It is important that maintenance fluids should be added over and above the Parklands formula for children weighing less than 30 kg.

The initial fluid rate selected is not as important as the resuscitation itself, which must be closely monitored and adjusted by a diligent clinician [21]. For burns > 20 % TBSA we would recommend the following:

1. Insert a central venous catheter
2. Insert a urinary (Foley) catheter
3. Use advanced haemodynamic monitoring (e.g. echocardiography to guide resuscitation)
4. Monitor intra-abdominal (bladder) pressure every four hours during the initial resuscitation

The principle aim of fluid resuscitation following a severe burn is to ensure appropriate end-organ tissue perfusion and minimise extension of the injury in the zone of stasis. This task can be extremely difficult to efficiently achieve because of the complex pathophysiology involved in a burn injury. Resuscitation should focus on optimizing global flow and perfusion as outlined in Chap. 6.

Most resuscitation formulae call for the use of isotonic crystalloids. However, there have been proponents of hypertonic solutions and colloids [3, 37]. Studies have demonstrated that colloids provide little clinical benefit when given in the first 24 h post burn and may have detrimental effects on pulmonary function and potentially worsen tissue oedema. The term "fluid creep" has been coined [38], suggesting that some patients are over-resuscitated with resultant complications. Over-resuscitation can result in "resuscitation morbidity", a term

used to describe complications secondary to excess fluid delivery including pulmonary oedema and orbital/extremity/abdominal compartment syndromes [39–41]. All of these are associated with increased morbidity and mortality.

Signs of extremity compartment syndrome need to be actively reviewed on an ongoing basis throughout the resuscitation. They may include:

(a) Severe pain at rest
(b) 'Tight' muscle compartments in limbs
(c) Increased pain on passive extension of digits
(d) Decreased distal sensation
(e) Decreased distal perfusion

Early multi-compartment fasciotomy, such as shown in Fig. 12.8 may be limb saving in this setting. The eschar of a burn wound consists of leathery dead skin producing a non-elastic exoskeleton which impedes perfusion. In general, escharotomies are required for circumferential full-thickness extremity burns in which distal perfusion has been compromised or as mentioned for chest burns in which the eschar impedes respiration.

Escharotomies are easily be performed at the bedside with either a scalpel or the use of electrocautery following recognised landmarks as shown in Fig. 12.9. It is important that the incision extends only through the eschar and not through the muscle fascia. Adequate release is apparent by separation of the eschar and improved distal perfusion.

In those situations, where it appears that excess crystalloid has been given, for example patients who exceed the Parkland formula calculation by more than 1.5 times or 6 ml/kg/%TBSA, some suggest "colloid rescue" be employed [42]. The colloid rescue formula advocates 1/3 of the Parkland volume be given as given as albumin and two thirds be given as Lactated Ringer's solution. This formula has been shown by some studies to decrease fluid requirements without any associated increase in mortality or renal failure. The use of hypertonic saline in burn patients has been limited by the

Figure 12.8 Upper limb Fasciotomy

concern of hypernatraemia, renal failure and increased mortality [43]. Therefore at the current time it cannot be recommended.

How Should Patients with Electrical Burns Be Managed?

Electrical injuries are classified as low (<1000 V) or high (>1000 V) voltage injuries. Low voltage injuries are usually the result of domestic (240 V single phase AC) or industrial (415 V 3 phase AC) accidents. High voltage electrical injuries usually result from contact with overhead powerlines and other sources of high voltage electrical currents [44].

Electrical burn severity is determined by:

- Voltage
- Current
- Type of current

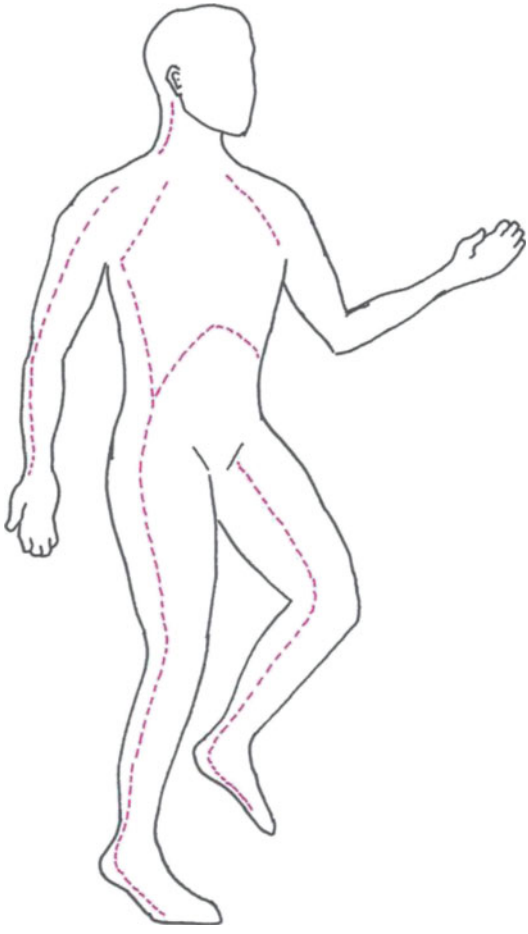


FIGURE 12.9 Diagram demonstrating surface anatomy for escharotomy incisions

- Presence of water
- Duration of contact
- Resistance at contact points

Low voltage injuries are associated with localized areas of tissue destruction, while high voltage injuries tend to be char-

acterized by deep and extensive tissue damage and are frequently associated with other injuries.

Three general patterns of injury are described:

1. 'True' electrical injury caused by current flow
2. Electrical arc injury caused by arc of current from the source to an object
3. Flame injury secondary to the ignition of clothes/garments

The management of these injuries is similar to that of standard burns injury, except for the need to isolate power at the scene and the need to recognise potential associated injuries. Importantly these patients are at high risk of myocardial injury and dysrhythmias. Daily determination of creatinine kinase levels and myoglobin is useful in assessing for occult rhabdomyolysis. Electrical burns may be deceptive with fluid resuscitation requirements that are often greater than that predicted by the area of the cutaneous burn. The extent of the burn and associated muscle damage may not be fully appreciated resulting in fluid loss which is not accounted for by the standard burns formula. It is extremely important to be vigilant for the development of rhabdomyolysis, myoglobinuria, and potential acute kidney injury. Continual clinical review to identify early limb compartment syndrome is also critical.

What Are the Principles of Burn Wound Management?

Thermal injury removes the protective barrier function of the skin and therefore dressings are required to protect the body against evaporative heat loss and environmental microbes. Initially a clean sheet or plastic wrap is useful. Superficial and superficial partial-thickness burns classically will heal without surgical excision and grafting. Regular dressing changes and wound baths remove necrotic debris and enhance heal-

Table 12.9 Examples of burn wound care products

Product	Indication
Adherent dressings e.g., Opsite or Duoderm	Superficial burns
Bacitracin, Neomycin, Polymyxin B	Superficial burns Facial burns Burns close to mucosal surfaces
Multiday dressings e.g. Mepilex and Acticoat	Superficial burns and partial thickness burns. Also used post debridement
Silver sulpurdiazine (SSD)	Deep dermal and full thickness burns (away from face and mucosal surfaces).

ing that should be established by two weeks. Superficial wounds require a dressing that provides a moist environment to optimise epithelialization and this is easily achieved with the application of ointments or lotions. With partial-thickness and full-thickness wounds, however, it is necessary to include agents that protect against microbial colonization. It is important to acknowledge that systemic antibiotic prophylaxis has absolutely no role in the management of acute burn wounds and use of prophylactic antibiotics in burn patients is associated with increased infection risk [45]. A comparison of burn wound dressings is shown in Table 12.9.

When managing deep and full thickness burns it is necessary for the patient to be under the care of a surgeon. Early wound excision and closure reduce the incidence of infection and improve survival. It has been increasingly recognised that early removal of dead and severely damaged tissue controls the dramatic systemic inflammatory response that is characteristic of deep burns. Early excision of deep burn wounds also appears to decrease hypertrophic scarring.

The standard modern approach is early staged excision commencing as early as post burn day two. Subsequent operations are conducted at intervals of 2–3 days until the entire

eschar is removed and full wound coverage is achieved. The debrided wounds are temporarily covered with biologic dressings or cadaveric allograft until further autogenous donor sites are regenerated [46, 47]. Donor sites usually epithelialise within about 2 weeks allowing new skin to be reharvested.

What Are the Other General Principles of Burn Injury Management in the Intensive Care Unit?

It is important to recognise that this is a highly labour and resource intensive process with large burns often remaining in intensive care for weeks to months. These patients are at substantial risk of hypothermia, anaemia, deep vein thrombosis [48–50] stress ulceration, nutritional deprivation, decubitus and corneal ulceration and so require diligent critical care [34].

Burn victims have multiple factors which may contribute to deep venous thrombosis and pulmonary embolism risk. Patients with greater than 40 % TBSA burn appear to be at highest venous thromboembolic (VTE) risk (2.4 %). The combination of increased TBSA burn and ICU admission predicts those patients at highest risk. Patients with these risk factors probably benefit from early, aggressive VTE prophylaxis [51].

Effective nutritional support is required in patients with severe burn injury, which is characterised by a dramatic elevation in plasma catecholamines, cortisol, and inflammatory mediators resulting in a prolonged and pronounced hypermetabolic response. The risk of inadequate provision of nutrition is rapid whole-body catabolism, muscle wasting, and severe cachexia. Furthermore, nutritional support is an essential component of burn care to prevent ileus and stress ulceration. The American Burn Association practice [15] guidelines recommend the commencement of enteral feeding as soon as practical. Adults are able to maintain their body weight after a significant burn injury only with adequate and continuous

Table 12.10 Summary of key points

Burn injury requires a standard trauma approach to avoid missing associated injuries
The burn needs to be carefully evaluated with respect to depth and percentage surface area.
Burn formulae are simply a guide
Endpoints/Goals need to be frequently reviewed to avoid over/under resuscitation
Compartment syndromes need to be excluded
Prophylactic antibiotics are contraindicated.
Early and sequential debridement is indicated for deep and full thickness burns

nutrition of 25 kcal per kilogram body weight per day and an additional 40 kcal per percent total body surface area burn per day. Aggressive protein delivery, providing approximately 20 % of calories from protein, has been associated with improved mortality and morbidity. However, it is important to recognise that the provision of excess calories and/or protein is not only ineffective and but likely to increase complications such as hyperglycaemia and overfeeding syndrome [52–55].

Summary

The thermally injured patient presents significant challenges for the clinical team that requires a coordinated response to resuscitation and vigilance for a wide range of complications. The patient is initially evaluated as for all major trauma. The surface area and depth of burn involved then dictate the initial resuscitation, which is continually modified based on the clinical response. Early surgical involvement is critical and allows for expedient debridement and subsequent coverage. The critical care physician requires a diligent and methodical approach to these complex patients. Key points in the management of severe burns are shown in Table 12.10

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