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The Intensive Care Management of the Adult Burns Patient

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Abstract The severely burned patient represents one of the most complex scenarios to manage in clinical practice. A major burn sets in motion a cascade of events which will result in catastrophic end-organ dysfunction if appropriate treatment is not commenced in a timely manner. Outside of a regional burns center, accurate clinical assessment of a burn is difficult. Clinicians must be judicious in administering treatment as both under- and over-resuscitation can be harmful. Further complicating the picture, the hypermetabolic state induced in the burned patient makes difficult the diagnosis of additional co-morbidities such as sepsis. In this review, we examine the common difficulties encountered in the resuscitation and intensive care of the severely burned patient. Additionally, we review the current and emerging evidence that guides our day-to-day management of extensive burns.

Keywords Burns · Intensive care · Inhalation injury · Parkland formula · Sepsis

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Introduction

Drawing on the expertise of all members of the multidisciplinary ICU team is vital in ensuring the best outcome for a patient with a major burn. The pathological response to a burn is unique and behaves in a distinct manner from other disease processes. As such, severely injured patients are often best served by early referral and transfer to a specialist burns center once initial life saving treatment has been commenced. Published criteria for transfer are widely available, but acceptance of the patient's transfer remains in the hands of the receiving institution. Coupled to this, the patients themselves may be challenging to work with. Burns frequently affect the most vulnerable in society, those at extremes of age, deprived social status, and people with drug and alcohol issues.

Cornerstones of effective burns management include

1. Early and effective airway management. The airway of a patient with inhalation injury is an evolving problem that often becomes increasingly difficult to manage with time. Decisive early control is warranted if concerns of an airway burn exist.
2. Appropriate and guided fluid management. Many formulae exist but none should be followed blindly.
3. Early surgical debridement, fasciotomies, and removal of necrotic tissue are required. Return to theater for repeat debridement is common.
4. Multi-organ support in the intensive care unit by a team who are experienced in caring for this complex subset of patients.
5. Prevention of infection with surveillance and early treatment of established sepsis, which in itself can present diagnostic dilemmas.

6. Nutritional support established via the most appropriate route and guided by dieticians with experience in burns care.
7. Rehabilitation and physical therapy to aid respiratory toilet and improve respiratory function, reduce the risk to pressure areas, reduce contractures in limb burns, and enhance functional outcome once discharged from the intensive care unit.

Evidence shows that integrated burns care as outlined above and discussed in more detail below can significantly improve outcome [1].

Classification of Burns Severity

In order to guide therapy in the burns patient, it is imperative to accurately calculate the percentage of total body surface area (TBSA) affected by the burn and the depth of the burn. The area of skin affected is frequently over-estimated in the emergency department despite multiple useful tools, such as Wallace's "rule of nines," the patient's palmar surface (representing 1 % of body surface area), and Lund and Browder charts. Inaccuracies will impact upon resuscitation fluid volumes and lead to under- or over-resuscitation.

Assessing depth of burn presents a further challenge, as without significant experience it is difficult to distinguish different burn severity. Depth of burn assessment is both a visual and clinical quantification and therefore involves a degree of subjective judgment. The most widely used clinical assessment methods are burn appearance, sensation, and capillary refill. Burns can be classified as superficial (erythema with no blisters); superficial partial thickness (erythema with blisters); deep partial thickness (extends into dermis, yellow or white in appearance); and full thickness (extends through entire dermis, white in color and non-blanching, classically described as painless due to involvement of nerve endings). The depth of burn dictates the likelihood of the injury healing without surgical intervention.

Referral to a specialist burn center depends on accurate initial assessment, with referral criteria being widely available online [2].

Inhalational Injury

Injury to the respiratory tract becomes increasingly common with increasing burn size [3]. The cause of respiratory injury may be primary (direct thermal injury or respiratory irritants) or secondary (systemic inflammatory response or sepsis). Further lung injury may be induced by mechanical ventilation and respiratory failure will be compounded by changes in chest wall compliance from thoracic wall burns.

Direct thermal injury tends to be localized to the upper airway with injury below the glottis being relatively rare. Super-heated steam can cause direct pulmonary injury; however, these injuries are often fatal due to rapidly progressing glottic edema resulting in complete airway obstruction. When inhalational injury is suspected, invasive respiratory support tends to be initiated early due to the risk of worsening airway edema leading to progressively more difficult laryngoscopy and intubation. Where intubation is required in the burns patient, thought should be given to the diameter and length of endotracheal tube inserted. Repeated fiber-optic bronchoscopy may be required, and this is considerably easier and safer in patients with a larger diameter tube. Soft tissue swelling may also evolve dramatically; therefore, endotracheal tubes should not be cut to length prior to insertion.

Inhalation of particulate matter and chemical irritants induce microscopic and macroscopic changes to lung tissue. Debris such as carbonaceous particles, fibrin deposits from exuded plasma, neutrophils, and mucus contribute to small airway obstruction [4]. Downstream air trapping and hyperinflation induce further microscopic lung injury and compound lung damage.

Systemic toxins generated during combustion may also contribute to mortality. Carbon monoxide is formed during incomplete combustion and is the leading cause of death due to fire gases [5]. Hydrogen cyanide is also commonly formed during combustion and can contribute to clinical hypoxia despite apparent satisfactory blood oxygen tension.

Early Resuscitation

Pathophysiology

Following a significant thermal injury, changes in capillary permeability permit a large leak of fluid from the circulation. A cascade of secondary mediators induce a microvascular insult resulting in loss of intravascular protein, fluid, and electrolytes to the interstitial tissue. In burns of sufficient surface area, this process manifests as hypovolemic shock, tissue edema, low urine output, and circulatory dysfunction. Replacement of this 'lost' circulating volume is crucial in the management of burns injury.

Intravenous fluid resuscitation becomes necessary to prevent hypovolemic shock once the size of the burn approaches 15–20 % [6] and delaying resuscitation may worsen outcomes [7]. The challenge of the resuscitation phase is to maintain organ perfusion in the face of a rapidly changing cellular and endocrine environment. However,

this resuscitation must be judicious as too much fluid or 'over-resuscitation' can also be harmful [8].

Resuscitation Formulae

To help guide initial fluid resuscitation, a variety of fluid regimens have been suggested [9]. The Parkland formula (see Eq. 1) is simple and one of the most widely used. However, it is worth emphasizing that these are guidelines only and to date no formula has been shown to provide optimum fluid management. Total burn surface area is often hard to estimate for inexperienced physicians, and this is further complicated by alterations in fluid requirement due to age, burn depth, inhalational injury, pre-existing comorbidity, and associated injury [10]. Optimal resuscitation should take place in a critical care environment with continuous monitoring for under- or over-resuscitation by a team of experienced burns staff.

$$\begin{aligned}\text{Total amount of fluid (Ringers Lactate) to be given in 24 h} &= 4 \times \text{Body weight(kg)} \\ &\times \text{percentage of TBSA affected}^{*,\#}\end{aligned} \quad (1)$$

* Half of this to be given in the first 8 h, the remainder over the following 16 h; # This does not include normal maintenance fluid requirement.

Despite guidelines for fluid resuscitation, it is common to over-resuscitate. Collis et al. showed that on average patients received double the amount of fluid as predicted by the Parkland formula prior to assessment by the burn unit [11]. Vasodilatory effects from the use of opioids may also lead clinicians to give additional fluid and further compound the excess fluid administration [12]. Complications from over-resuscitation include burn conversion (superficial to deep), pulmonary edema, peripheral edema requiring fasciectomy, and abdominal compartment syndrome.

Monitoring Resuscitation

Objective resuscitation end-points in burned patients remain elusive. The American Burn Association in their 2008 guideline on burns shock makes a single recommendation to maintain a urine output of 0.5–1 mL/(kg h) [13]; however, recommendations for other physiological indices are absent. Using more sophisticated devices such as invasive cardiac output monitors show that hypovolemia is often present in the initial phases of burns resuscitation [14]. This has the potential to lead clinicians into giving an excessive volume of fluid. To avoid this, protocols have been developed which aim to give a minimum of fluid necessary to maintain a low-normal cardiac index or intrathoracic blood volume [15•]. These have been associated with a lower multiple organ dysfunction score but data are lacking on how this influences mortality [15•, 16]. Measurement of admission blood lactate has been shown to correlate with mortality and severity of burn [17] but its usefulness in guiding fluid administration or as a resuscitation end point in patients with large burns has not been demonstrated.

Crystalloid or Colloid?

The Parkland formula advocates the use of Ringer's lactate. Concerns exist over the use of colloids in the presence of endothelial dysfunction, suggesting that colloids may cross into the interstitial space and draw excess fluid with it. Timing of initiation of colloid therapy remains controversial with some authors suggesting that the majority of extravasation is complete by 12 h and therefore permitting colloid administration after this window [18]. 5 % human albumin solution has been predominantly investigated as the colloid of choice for resuscitation of burns. It has traditionally not been shown to confer a mortality benefit [19, 20], although it may reduce intravenous fluid requirement [21]. A recent meta-analysis, however, has suggested that albumin may decrease mortality, although the evidence is limited [22]. Hypertonic crystalloid has also been postulated as a method to maintain fluid in the plasma compartment, although a Cochrane review in 2004 found that there were insufficient data to demonstrate harm or benefit from its use [23]. Further high-quality evidence is required to determine whether albumin or hypertonic crystalloid is beneficial in patients with major burns.

Pharmacological Resuscitation

Multiple pharmacological adjuncts have been suggested to reduce the inflammatory response associated with a major burn. Examples include vitamin C, ketanserin, non-steroidal anti-inflammatory drugs, and hydrocortisone, although none of these drugs are in routine use for burn resuscitation. Vitamin C has been shown in animal models to reduce endothelial dysfunction [24], and high-dose vitamin C infusion has been shown to reduce fluid requirements and increase urine output in burned patients [25]. Despite this, a retrospective review of vitamin C treatment in burns was unable to show an improvement in outcome [25], and concerns exist over an increased risk of renal impairment with high-dose vitamin C infusions [26].

Blood Transfusion in Burns

A restrictive strategy (target hemoglobin 7–9 g/dL) toward blood transfusion is increasingly used for critically ill patients, and its adoption in patients with major burns has not been associated with worsening outcomes [27]. A

multicenter retrospective study showed an increase in mortality with increased blood transfusion volumes even after correction for burn severity [28]. However, despite the above evidence, a holistic view of blood transfusion must be taken. For example, it may be clinically prudent to aim for a higher hemoglobin in a patient who is about to undergo an extensive burn excision with the potential for high blood loss.

Respiratory Support

The lung protective ventilation strategies used in patients with ARDS including optimal PEEP, limited tidal volumes, and permissive hypercapnia [29] have been adopted in burns patients requiring invasive ventilation. However, there is limited evidence to suggest the efficacy of these strategies due to the unique pathology in inhalation injury [30].

Fiber-optic bronchoscopy assists in the diagnosis and grading of inhalational injury. In addition, washout and removal of particulate matter at bronchoscopy is likely to improve outcome and reduce the duration of mechanical ventilation [31]. Patients with restricted chest movement secondary to circumferential full thickness burns should be considered for escharotomy to improve chest wall compliance.

Tracheostomy is commonly used for patients with extensive burns in whom prolonged ventilation is likely to be necessary. Although this may facilitate day-to-day management of the burns patient, there remains no evidence for improved outcome or earlier extubation with either early or late tracheostomy [32, 33].

Pharmacological Agents

Pharmacological attempts to assist the clearance of macroscopic airway debris in inhalational injury have been considered. Acetylcysteine aids mucolysis and heparin inactivates thrombin leading to reduced airway cast formation. A regime of heparin, N-acetylcysteine, and β_2 agonist was shown to improve mortality in a retrospective study of 30 patients [34]. A similar benefit was also shown in a pediatric population [35]; however, evidence from a large prospective trial is lacking.

The ability to rapidly measure blood cyanide levels is currently limited, and treatment may be considered in the presence of high blood lactate levels (>7 mmol/L), elevated anion gap acidosis, and reduced arteriovenous oxygen gradient. Hydroxocobalamin is considered one of the safest antidotes for the treatment of cyanide poisoning, although side effects include skin discoloration and transient hypertension.

Hypermetabolism

Major burn injuries are associated with a hypermetabolic physiological state. This is mediated in part by an increase in plasma catecholamines that can persist up to 2 years after the burn injury [36]. Modulation of this response with the use of β-blockers such as propranolol has been shown to reduce muscle catabolism [37] and may be associated with improved outcome [38]; however, evidence from a prospective randomized trial is lacking.

The burn insult causes a catabolic state to develop. This is characterized by decreased protein synthesis and increased protein breakdown with overall negative nitrogen balance. Anabolic steroids such as oxandrolone tend to promote protein synthesis and positive nitrogen balance. Oxandrolone in combination with a high-protein diet has been shown to increase the rate of weight gain during recovery from a burn [39]. A prospective trial of 81 patients with 20–60 % burns also observed a significant reduction in hospital stay when oxandrolone therapy was commenced 5 days post-injury [40].

Sepsis and Diagnostic Issues in Burns

Sepsis occurs in 8–42.5 % of burns patients with a mortality between 28 and 65 %, but the diagnosis of infection requiring treatment can present a challenge [41]. Early treatment of septic episodes is accepted within the ICU community as a cornerstone of reducing mortality and improving outcome, hence rapid diagnosis is vital [42].

Diagnosis of Sepsis

For the general ICU population, systemic inflammatory response syndrome (SIRS) plus evidence of infection is used to diagnose sepsis; however, things are not so simple in burns [43]. The vast majority of burned patients (>95 %) meet the SIRS criteria as a result of the primary insult of the burn, despite the absence of infection [44]. Baseline temperature is reset to 38.5 °C, with tachycardia and tachypnea persisting in patients with extensive burns. This creates the need for different diagnostic criteria [45]. The American Burns Association (ABA) issued a consensus statement in 2007 outlining diagnostic criteria specifically for sepsis in burns victims (see Table 1)

Additional markers of infection may be helpful in the burned patient. There are multiple biological markers that have been investigated including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and procalcitonin (PCT). None are truly sensitive or specific for infection in burns. Lavrentieva et al. showed raised levels of PCT to be a useful early indicator of bacterial infection, whereas

Table 1 The ABA Guidelines for the diagnosis of Sepsis

Patient should have documented evidence of infection either by positive cultures, or tissue biopsy, or have a clinical response to antimicrobials
Plus at least 3 of the following criteria:
(1) Temperature >39 or <36.5 °C
(2) Progressive tachycardia, HR >110 bpm
(3) Progressive tachypnea (RR > 25, or MV > 12 L/min if on mechanical ventilation)
(4) Thrombocytopenia <100,000/μl (3 days after resuscitation)
(5) Hyperglycemia (in patients without pre-existing diabetes)
(a) Glucose >200 mg/dL untreated
(b) Insulin resistant
(6) Inability to continue enteral feeding >24 h due to
(a) Abdominal distention
(b) High residual volume
(c) Severe diarrhea (>2.5 L/day)

CRP, ESR, and fever were non-predictive of infection [46, 47]. Multiple studies in non-burns patients have correlated PCT level and severity of sepsis; however, uncertainty exists regarding cut-off values of significance and PCT should be viewed as an adjunct to clinical assessment [48]. In burns patients, PCT should be viewed with even higher skepticism. Seoane et al. showed that PCT was non-discriminative in patient with SIRS vs. sepsis and septic shock, whereas other studies have found PCT to be prognostic and advocate the use of daily PCT to monitor effectiveness of antibiotic therapy [49, 50].

Prevention of Infection

Infection control measures within the burns ICU are of paramount importance. Patients have reduced immune efficiency, prolonged hospital stays, invasive monitoring devices, recurrent surgical procedures, and lack the innate protection offered by an intact layer of skin rendering them highly susceptible to infection. Additional risk factors include patient age, total body surface area, and depth of burn and the presence of inhalation injury [51]. The importance of robust basic infection control measures cannot be overstated.

Central venous catheter (CVC) related blood stream infection (CLRBSI) is of particular risk in the burns patient. CVC lines are often necessary to allow monitoring and organ support in the ICU. Invasive devices should be placed through unburned skin where possible to reduce the risk of infection. In the general ICU, it is accepted that routine, timed line changes do not reduce the risk of line infection, yet it remains standard level of care in multiple burns ICUs [52]. Units have demonstrated that the use of a 'care bundle' approach to central line care along with scheduled line changes (every 3 day rewiring and every 6 day at a new site) has reduced line-related infection in the burns ICU [53••]. An historic study has shown that increasing the interval of line change from 3 to 4 days in the burns patient increased the risk of CLRBSI from 4 to 12 % [54]. In a recent survey of ICUs in the US, 61 % perform scheduled CVC change at 3–7 days [55].

Antimicrobial Resistance

Pathogens causing infection in the burns ICU are markedly different from those which cause infection in the general ICU or the burns ward. In a recent study, the most common burns ICU pathogens were *Acinetobacter baumannii* (34 %), *Pseudomonas aeruginosa* (17 %), *Staphylococcus aureus* (12 %), and *Klebsiella pneumoniae* (10 %) [56•]. This complicates antibiotic selection in the burns ICU as these pathogens may be resistant to multiple common antimicrobials. It should also be remembered that large burns are a risk factor for fungal colonization, and therefore, antifungals should be considered. In general, other than to cover surgical procedures, prophylactic antibiotics are not indicated in adult burns because they may increase antibiotic resistance [57•].

Conclusion

Survival and length of stay are improving for patients with severe burns. There are multiple factors underlying this positive change, most important is the recognition that burns are best managed in regional centers by a multi-professional team who frequently encounter major burn injury. Improvements in the wider 'general' critical care environment have also aided the improvement in mortality.

Fluid resuscitation will continue to benefit from ongoing research. Current evidence seems to advocate a more restrictive strategy with vigilance for over-resuscitation. It seems likely that the use of colloid for burn resuscitation will increase with some units incorporating albumin as rescue therapy in their fluid administration protocols.

Modifying the physiological response to the burn with pharmacological agents may provide further improvements in mortality; however, to date, there are a lack of data from large trials to confirm their safety in clinical practice. Oxandrolone, propranolol, and vitamin C have shown promise in smaller studies.

Sepsis is frequently encountered in burns patients but is a diagnostic challenge in the context of the hypermetabolic state due to the burn injury. Specific criteria such as those from the ABA can help guide antimicrobial treatment. Ensuring adherence to robust infection control practices is also vital in preventing the emergence of organisms which are resistant to antimicrobial therapy.

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Compliance with Ethics Guidelines

Conflict of Interest Michael D. Spiro and Mark W. Lambert declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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