Anaesthesia and intensive care for major burns

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Key points
Crystalloid is the fluid of choice for resuscitation, volume calculated by the Parkland formula = 4 ml kg⁻¹ (%burn)⁻¹.

Hypovolaemic shock in the first few hours after a burn injury is never due to the burn alone.

Primary and secondary survey should follow ATLS principles (do not get distracted by the burn).

Blood loss during debridement and grafting can be significant and insidious.

Major burns require a dedicated multidisciplinary team for adequate management.

Major burns are systemic injuries requiring input from multiple specialities. There are ~140,000 cases of new burns per year presenting to emergency departments in England and Wales, with around 10% of these being admitted to hospital. Of these, 4000–5000 cases are complex and require the services of a regional adult burns unit. The majority of the others will be managed by hospitals with a plastic surgery service.

Important considerations in the clinical outcome for these patients are early resuscitation, multidisciplinary team management, early surgical debridement, and prevention of complications. Anaesthesia in this group of patients can be challenging with profound hypermetabolism, pain management issues, alteration of drug pharmacokinetics, potential airway problems, temperature control, and substantial blood loss.

Overall, the mortality rate among hospitalized burn patients in a recent review of European data was 13.9% (4–28.3%). Major risk factors for death are older age, a higher total percentage of burned surface area, inhalation injury [mortality rate 27.6% (7.8–28.3%)], and chronic diseases. There appears to be no sex-related difference in survival after thermal injury. Multi-organ failure and sepsis are the most frequently reported causes of death. The main causes of early death (<48 h) are burn shock and inhalation injury.

Initial assessment and resuscitation

History
The history of a burn injury can give valuable information about the nature and extent of the burn, the likelihood of inhalation injury, the depth of burn, and the probability of other injuries. A patient’s full medical history must be obtained on admission to the emergency department, as this may be the only time that a first-hand history is obtainable.

Primary survey
The initial management of a severely burnt patient is similar to that of any trauma patient. The burn injury must not distract from this sequential assessment, otherwise serious associated injuries may be missed.

Airway with cervical spine control
All burn patients should receive 100% oxygen (O₂) through a non-rebreathing mask on presentation. An assessment must be made as to whether the airway is compromised or is at risk of compromise. Initial compromise of the airway is almost always due to a low Glasgow Coma Score (GCS) and not the burn. Early tracheal intubation should be considered in the presence of any of the following features: stridor, hypoaxemia or hypercapnia, a GCS of <8, deep facial burns, full-thickness neck burns and oropharyngeal oedema. If intubation is required at this early stage, it is usually technically easy as swelling of the airway has not yet occurred. An uncut tracheal tube (size 8.0 mm or above) is used to allow subsequent bronchoscopy. Succinylcholine is safe in the first 24 h after a burn—after this time, its use is contraindicated due to the risk of hyperkalaemia leading to cardiac arrest, thought to be due to release of potassium from extrajunctional acetylcholine receptors. This can persist up to 1 year post-burn.

Inhalation injury
Inhalation injury is defined as the aspiration of superheated gases, steam, hot liquids, or noxious products of incomplete combustion. It is almost never seen in association with flash burns or other forms of brief, albeit high temperature, exposure. True inhalation injury is...
likely to be present if the burn was received in an enclosed space with delayed escape or rescue. Three distinct clinical entities are possible.

**Upper airway thermal injury (above larynx)**

This usually occurs above the glottis, as by the time hot gases reach the larynx, the heat energy has been dissipated. The pharynx and epiglottis may have significant thermal injury which can swell dramatically. Clinical signs include inspiratory stridor, change in voice/hoarseness, and a swollen uvula.

**Lower airway thermal injury (below larynx)**

Inhalation of products of incomplete combustion causes sloughing of the airway’s epithelium, mucus secretion, inflammation, atelectasis, and airway obstruction. Clinical signs include dyspnoea, coughing wheezing, and the production of copious secretions. Findings at bronchoscopy include carbonaceous deposits, oedema, bronchial mucosal erythema, haemorrhage, and ulceration. Bronchial lavage with 1.4% bicarbonate solution has been used to neutralize acidic deposits and remove soot contamination.

**Injury as a result of noxious gases**

Carbon monoxide (CO) poisoning should be suspected in any unconscious patient. The clinical symptoms of CO poisoning include nausea and vomiting, headache, hypotension, convulsions, and coma. The amount of carboxyhaemoglobin (HbCO) formed depends on inspired CO concentration and duration of exposure, and helps to diagnose inhalation injuries but correlates poorly with severity of toxicity and outcome. The oxyhaemoglobin (HbO₂) dissociation curve is shifted to the left, and the cellular cytochrome oxidase system is inhibited, causing tissue hypoxia and metabolic acidosis. Pulse oximetry cannot differentiate between HbO₂ and HbCO and will overestimate the true oxygen saturation. Arterial blood gas analysis using a co-oximeter is required.

Treatment is with 100% oxygen which will decrease the elimination half-life of CO from 4 h to under 1 h—it is reduced further to under 30 min with hyperbaric O₂ at 3 atm but this is rarely practical. It should be considered in pregnant or comatose patients, those with HbCO levels >40%, or patients failing to respond to conventional therapy. Patients with HbCO levels >25–30% should be ventilated.

Cyanide poisoning should be suspected in burn patients with an unexplained and persistent lactic acidosis, despite adequate fluid resuscitation.

**Breathing**

Breathing, chest movement, and tracheal position should be assessed clinically. There are several ways that a burn injury can compromise respiration.

**Mechanical restriction of breathing**

Deep dermal or full-thickness circumferential burns of the chest may severely restrict chest wall movement and relieving escharotomy may be necessary to allow adequate ventilation. These are rarely, if ever, needed before admission to a burn service.

**Blast injury**

Penetrating injuries can cause tension pneumothoraces, and the blast itself can cause lung contusions and alveolar trauma which may lead to adult respiratory distress syndrome.

**Circulation**

Two large-bore i.v. cannulae should be inserted through the unburnt skin if possible and baseline bloods sent. Traditional sites for i.v. access may be unavailable and unusual peripheral venous sites or central venous access is required (usual indications for central venous line insertion also apply). The groins are usually spared so femoral venous cannulation is often possible. Burns are not the cause of immediate hypovolaemia. If there are features of hypovolaemic shock, the patient is almost certainly bleeding from other injuries. Warmed Hartmann’s solution should be started and titrated to cardiovascular signs, but if stable can be run slowly until the burn calculation is made (see the Exposure and estimation and Fluids sections).

**Disability**

A brief assessment of the conscious level should be made using the GCS score and pupils examined.

**Exposure and estimation**

Expose and ensure all jewellery and watches are removed from burnt limbs. The patient should be examined (including the back—including possible sites or central venous access is required (usual indications for central venous line insertion also apply). The groins are usually spared so femoral venous cannulation is often possible. Burns are classified by total body surface area (TBSA) and depth. A standard Lund–Browder chart is readily available in most emergency departments for a quick assessment of BSA burnt. If this is not available, the ‘Rule of Nines’ is fairly accurate in adult patients (Fig. 1).

**Fluids**

I.V. fluid resuscitation is required in adults if the burn involves more than 15% BSA or 10% with smoke inhalation. The Parkland formula is the most widely used resuscitation guideline and is 4 ml kg⁻¹ (%burn)⁻¹ which predicts the fluid requirement for the first 24 h after the burn injury. Starting from the time of burn injury (not time of presentation), half of the fluid is given in the first 8 h and the remaining half is given over the next 16 h. The fluid of choice is Hartmann’s solution. Any fluid already given should be deducted from the calculated requirement. A urinary catheter
should be inserted and hourly urine output is a used as a guide to resuscitation. In adults, at least 0.5 ml kg$^{-2}$ h$^{-1}$ should be passed.

Example of calculation of fluid resuscitation in burns

A 32-yr-old man weighing 80 kg with a 30% flame burn was admitted at 23:00 h. His burn occurred at 22:00 h. He has already received 1000 ml of crystalloid from the emergency services.

(i) Total fluid requirement for first 24 h

4 ml × (30% TBSA) × (80 kg) = 9600 ml in 24 h

(ii) Half to be given in first 8 h, half over the next 16 h

Will receive 4800 ml during 0–8 h and 4800 ml during 8–24 h

(iii) Subtract any fluid already received and calculate hourly infusion rate for first 8 h

4800 (for first 8 h) – 1000 (fluid already received) = 3800 ml

Burn occurred at 22:00 h, so 8 h point is 06:00 h. It is now 23:00 h, so need 3800 ml over next 7 h.

3800/7 = 543 ml h$^{-1}$ from 23:00 to 06:00 h

(iv) Calculate hourly infusion rate for next 16 h

Divide figure in (ii) by 16 to give fluid infusion rate. Needs 4800 ml over 16 h:

4800/16 = 300 ml h$^{-1}$ from 06:00 to 22:00 h the next day.

Table 1 British Burns Association referral criteria for patients with burn injuries

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<thead>
<tr>
<th>Complex burn injuries include any of the following</th>
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<td>Extremes of age</td>
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<tr>
<td>Site involved (dermal or full-thickness loss)</td>
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<tr>
<td>Inhalation injury</td>
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<tr>
<td>Mechanism of injury</td>
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<tr>
<td>Size of skin injury with dermal or full-thickness loss</td>
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<tr>
<td>Pre-existing co-morbidities</td>
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<td>Associated injuries</td>
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Analgesia

Full-thickness burns are painless; however, a mixed picture is common and the patient should receive i.v. morphine titrated against response.

While all hospitals will be involved in the initial resuscitation of burn victims, complex burn injuries should be referred to and managed in a burns unit (Table 1). If you are not sure whether a burn should be referred, discuss the case with your local burns unit.

Anaesthesia and surgery for burns

Inpatient burn injury care should be provided only by specialists trained in burn care in a burns unit. This is a reflection on the team approach to burn injuries, the resources, and infrastructure necessary to provide both critical care and the long-term management of the patient in terms of the planning and timing of surgical procedures.

Recent trends in patient care have focused on early excision and wound coverage, aiming to remove the full-thickness injury and get biological closure. This potentially reduces the risk of wound infection and the development of sepsis. The risk of this approach is the physiological insult of surgery to a patient who may well be deteriorating rapidly from their initial injuries. The most important and difficult clinical decisions are often made by the team at this stage.

Escharotomy and decompressive therapies

These are necessary when a number of factors come into play. The patient will have a full-thickness circumferential burn resulting in an ‘eschar’ (from French eschare meaning scar or scab) which acts as a non-compliant tourniquet. When there is an additional insult from the accumulation of extracellular fluid as a result of
Anaesthesia for wound debridement and grafting

The conduct of anaesthesia will be predicted by the TBSA, degree of systemic insult, and planned area for debridement and grafting. There is a trend towards earlier intervention and burn excision with the application of skin substitutes if donor skin is limited or further harvesting is not appropriate.

Monitoring can be a challenge in major cases when access to chest (ECG), arms (arterial pressure), and digits (pulse oximetry) can be limited. Skin staples or subcutaneous needles attached to crocodile clips can be used for ECG monitoring. Alternative sites for pulse oximetry such as the nose, lips, or tongue may be necessary. An arterial line is essential for major excisions and a cardiac output monitor can be useful. Blood loss can be significant and occasionally insidious. In major deep burns, we plan for 50–100 ml blood loss per % area excised, depending on the time post-burn (max 7–16 days) and the presence of infection. This can often be compounded by thrombocytopenia and prolonged coagulation time which requires close liaison with haematology and blood bank.

The patient will be hypermetabolic with a systemic inflammatory response (SIRS)-type cardiovascular system and often on vasoactive substances. This, combined with anaesthesia and significant blood loss, can make interpretation of haemodynamic changes quite taxing.

These patients often require multiple anaesthetics/operations for dressing changes and grafting and pain management is difficult. There is no evidence for improved outcome using TIVA vs inhalation anaesthesia and the technique used is an individual choice. With multiple anaesthetics, it is important to review the patient response to the previous event and adapt accordingly with the assumption that pain will be an escalating issue. Regional anaesthesia has a part to play but can be limited due to the large areas required to block and the risk of infection. There needs to be a multimodal approach to analgesia in these patients with consideration given to the early use of neuropathic pain agents, such as pregabalin, and opioids and psychology input. Acute perioperative pain may need benefit from agents such as ketamine in addition to the usual analgesic ladder. The time taken for application of major dressings is another consideration, particularly with regard to temperature homeostasis.

There are many preoperative anaesthetic considerations for burn wound excision/grafting (Table 2).

Critical care management

In the first 24–48 h after resuscitation in patients with major burns, the hormonal response and inflammatory mediators cause hypermetabolism, immunsuppression, and SIRS.

Fluid resuscitation and electrolyte management

The fluid resuscitation aims to restore tissue perfusion avoiding end-organ ischaemia, preserving viable tissue and minimize tissue oedema. The Parkland formula is a guide and fluid resuscitation should be titrated against clinical response, invasive monitoring,
and urine output (>0.5 ml kg\(^{-1}\) h\(^{-1}\) in adults). Invasive monitoring is necessary in the severely burnt patient to help guide both volume replacement and the use of ionotropes. The term ‘fluid creep’ describes the excessive volumes of fluid used for resuscitation which has occurred in some burn patients with complications (Table 3).\(^5\) Hypokalaemia, hypophosphataemia, hypocalcaemia, and hypomagnesaemia are common and should be treated. There is a phenomenon known as ‘burn shock’ which describes a combination of hypovolaemic, distributive, and cardiogenic shock which is refractory to massive i.v. resuscitation. Experimental studies have looked at the use of hypertonic saline, high-dose ascorbic acid, or plasma exchange in this setting.

**Mechanical ventilation**

Usual lung protection strategies apply, and routine ventilator-associated pneumonia prevention strategies should be implemented. Prophylactic corticosteroids or antibiotics have no role.\(^6\) In patients with inhalation injury, early tracheal intubation, aggressive pulmonary toilet, bronchodilator therapy, and bronchoscopic lavage are all important.

**Hypothermia**

Patients with large burns reset their baseline temperature to 38.5°C. This means that a patient with a core temperature of 37°C is relatively hypothermic. Strategies to prevent hypothermia include a warmed room (ambient temperature 28–32°C), warmed inspired air, warming blankets, and warmed fluids. This is also important to minimize the increase in basal metabolic rate caused by heat and evaporative water loss.

**Metabolism and nutrition**

The increase in basal metabolic rate is proportional to the size of the burn and the presence of infection, peaking at 7–10 days. This hypermetabolism can persist for up to 2 yr post-injury. Early enteral nutrition has been shown to improve survival in burn patients. Meeting nutritional requirements (high protein, high carbohydrate feeds) is crucial to prevent protein breakdown, decreased wound healing, immune suppression, and an increase in infective complications.

**Infection**

This is a significant cause of mortality in major burns (Table 3). Diagnosis of sepsis in burn patients is difficult as signs of SIRS induced by the burn are all normal findings, so regular microbiological surveillance is essential. Burn-specific criteria for sepsis include (three of the following with documented infection):\(^7\)

- Temperature >39 and <36.5°C
- Tachycardia >110 beats min\(^{-1}\) or >2 SD standard value for age
- Progressive tachypnoea (spontaneous ventilation: respiratory rate >25 or mechanically ventilated)
- Hyperglycaemia (plasma glucose >12.8 mmol litre\(^{-1}\) in the absence of diabetes mellitus)
- Thrombocytopenia (will not apply until 3 days after initial resuscitation; platelet count <100 000 µl\(^{-1}\))
- Inability to continue enteral feeding >24 h

Protecting patients from infection is by primary excision and skin grafting, asepsis with regular dressing changes, and patient isolation.

**Psychological care/rehabilitation**

A multidisciplinary team of physiotherapists, psychologists, nurses, councillors, and occupational therapists are vital to aid rehabilitation and reduce long-term impairment.

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**Declaration of interest**

None declared.

**References**