Critical care management of inhalational injury and severe burns

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Abstract

Inhalational injury and severe burns are common. Most will present to district general hospitals and thus an understanding of how to assess and manage these patients is vital for all practising anaesthetists with on-call commitments. In this article we emphasize the requirement for a highly functioning multidisciplinary approach with excellent communication both within and between treating teams. We will challenge the dogmatic approach to airway management and discuss the increasing problem of fluid creep. The key areas of burns assessment, sedation/analgesia and nutrition management in these patients is also discussed.

Keywords Burns; inhalational injury; resuscitation

Royal College of Anaesthetists CPD matrix: 1C01, 1C02, 2A02, 2C01

Learning objectives

After reading this article, you should be able to:

- recognize the features and management of airway burns and smoke inhalation
- explain the pathophysiological response to a burn injury
- classify the type of burn and estimate total depth and body surface area
- discuss the multidisciplinary and systemic approach required of burns management

Epidemiology

Approximately 140,000 patients with burns present per annum to emergency departments in England and Wales. About 4000 −5000 of these will require the specialist services of a burns centre.[1] Severe burns requiring critical care are associated with significant physical and psychosocial morbidity, and mortality. The management of these complex cases demands a multidisciplinary approach with a highly performing and motivated team.

Mortality depends on a number of factors including percentage full-thickness burn, age and gender of the patient together with other physiological variables (APACHE II score). Large studies from Europe[2] and Australia[3] show the mortality rate to be in the order of 10−14% for severe burns requiring ICU admission. Mortality is usually due to multiorgan failure secondary to sepsis.

The Australian Burns Evaluation and Mortality Study (BEAMS) risk of death model has recently been developed and has been shown to be a valid and reliable tool by which to predict mortality (www.beamsurvival.com). Although the BEAM score should not be used to predict individual mortality it can be used to compare outcome of different units, to monitor progress of a given unit over time and to monitor the effect of any management change within a given unit.[3]

Improvements in mortality over recent years has been attributed to centralization of specialist burn services, rapid burn excision and grafting, the multidisciplinary approach to critical care and topical antimicrobials.

Pathophysiology of burns

Whether a burn is thermal, chemical or electrical, a local and a systemic response can ensue. Burns involving more than 20% of body surface area will have a significant systemic response.

Local response

The local response to a burn results in three zones, as described by Jackson in 1947[4] (Figure 1):

- central zone of coagulation which is unsalvageable
- zone of stasis − this zone is characterized by tissue hypoperfusion and is salvageable with treatment optimization of tissue perfusion
- zone of hyperaemia − this zone has increased perfusion and will recover provided sepsis or prolonged hypoperfusion does not occur.

Systemic response

Cytokines will be released from damaged cells leading to a generalized inflammatory response. Both burnt and unburnt tissue will develop increased capillary permeability. Proteinaceous fluid will pass into the interstitial space and this will contribute to tissue oedema and hypoperfusion.

Cardiovascular

Myocardial depression can occur secondary to the influence of cytokines. Fluid loss also contributes to tissue hypoperfusion.

Respiratory

Inhalation of smoke and noxious substances can cause localized inflammation to the airway. More severe burns can result in a generalized inflammatory response leading to acute respiratory distress syndrome (ARDS).

Metabolic

The metabolic response can increase threefold. Hypermetabolism is caused by supra-normal levels of circulating catabolic stress hormones (cortisol, adrenalin, and glucagon). This leads to increased gluconeogenesis, accelerated osteoclastic activity, lipolysis and decreased protein synthesis. Splanchnic vasoconstriction can

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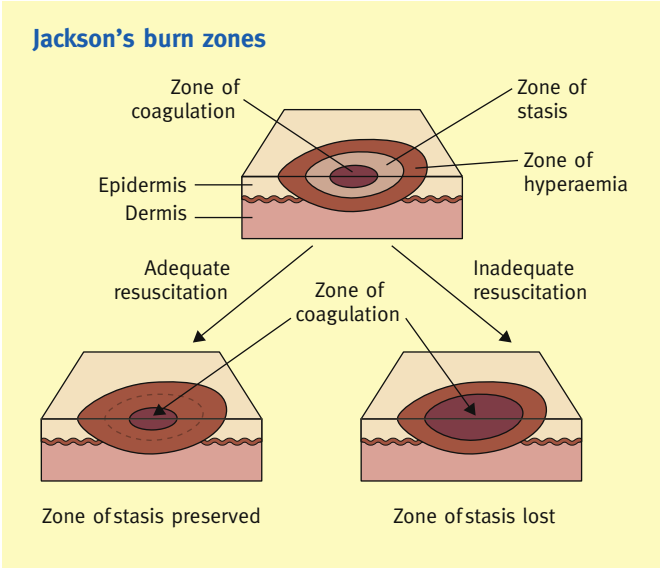

Figure 1 Jackson's burn zones and the effects of adequate and inadequate resuscitation. Reproduced with permission from BMJ Publishing Group Ltd.[4]

Risk factors for inhalational injury

Suspicious features from history	Clinical signs
• Fire in a closed space • Burning wool, silk, polyurethrane • Prolonged entrapment • Loss of consciousness • Death of another victim/CPR at the scene • Past history: smoking, asthma, chronic obstructive pulmonary disease	• Respiratory distress/ tachypnoea • Persistent cough, stridor, wheeze • Hoarseness, odynophagia, change in voice, sore throat • Haemoptysis • Facial/neck burns • Singed nasal hair • Carbonaceous sputum • Blistering of oropharynx • Mental obtundation: drowsiness, lethargy, muscle weakness, headache

Table 1

compromise gastrointestinal mucosal perfusion leading to sloughing and translocation of bacteria causing sepsis.

Immunological

Both cell-mediated and humoral immunity are downregulated leading to an increased susceptibility to infection.

Assessment and resuscitation of the severely burned patient

Airway

Like all trauma resuscitation scenarios airway assessment (with C-spine control) and management is the initial top priority. High-flow humidified oxygen should be administered on admission.

The history of the circumstances giving rise to the burn injury and clinical examination will give some idea as to the risk of an airway burn. The clinical features which may indicate an airway burn are well known (Table 1), but it is important to understand that these clinical indicators have both a low sensitivity and specificity for the presence of an airway burn.

Understandably, intubation is often undertaken as a precautionary measure to facilitate transfer to a burns unit. Data from burn centres, however, consistently show that only a minority of patients require intubation. The UK National Burn Injury Database shows that between 2003 and the end of 2012, 17% of the 1029 patients admitted acutely to burns ICUs underwent extubation on the same day as admission. About 49% were extubated within 1 day. It has been argued that significant numbers of acutely burned patients are undergoing intubation and ventilation unnecessarily, thus exposing these patients to the morbidity and mortality associated with this procedure.[5]

Data suggest that a significant number of patients with mild to moderate facial burns could be managed in a high-dependency area with fibreoptic techniques such as nasendoscopy on admission and at pre-defined intervals to assess for increasing airway oedema. An inhalational injury score based on fibreoptic evaluation of the upper airway and tracheobronchial tree has recently been proposed.[6] An improvement in clinical decision making may occur as a result of providing an objective score based on mucosal appearance.

The key to successful airway management is early involvement of the anaesthetic team, regular re-assessment and good communication with the burns centres. This may well facilitate the transfer of the patient, un-intubated, after a period of observation in the base hospital.

If the patient does require early intubation then a rapid sequence induction would be the technique of choice. Suxamethonium can be used within the first 24 hours after the burn. After 24 hours suxamethonium should be avoided as there is an increased risk of hyperkalaemia due to the proliferation of extrajunctional nicotinic acetylcholine receptors. The endotracheal tube should never be cut and should be large enough to allow passage of a fibreoptic bronchoscope.

Breathing

Inhalation of noxious substances such as carbon monoxide (CO) and cyanide (CN) can rapidly be lethal.

Haemoglobin has a much greater affinity for CO (×240) than oxygen, and so CO will displace oxygen from its binding site on the haemoglobin molecule to form carboxyhaemoglobin (HbCO). The absorbance spectrum of HbCO is similar to HbO_2 and so pulse oximetry will mistakenly show adequate saturations on the plethysmograph. The detection of HbCO requires point of care arterial blood gas analysis with a co-oximeter. HbCO levels of up to 15% can be normal in smokers. Patients with COHb levels of 25–30% should be mechanically ventilated. 100% oxygen forms the mainstay of treatment. This will reduce the half-life of HbCO from 4 hours to 1 hour. The indications for hyperbaric oxygen therapy include COHb poisoning in pregnancy and coma/seizures but in practice this is rarely pragmatic.

Like CO, CN exerts its pathological effects by preventing mitochondrial respiration via inhibition of cytochrome oxidase