Critical care management of severe burns and inhalational injury

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Abstract

Anaesthetists and critical care physicians involved in emergency care provision, must be equipped with the knowledge and skills to accurately assess and initiate treatment in patients with severe burns. This summary aims to review airway management and fluid resuscitation in addition to sedation and analgesic choices. Some of the dogma involved in current aspects of modern burns care will also be questioned.

Keywords Critical care medicine; inhalational injury; resuscitation; severe burns

Royal College of Anaesthetists CPD Matrix: 1A01, 1A02, 1C01, 1C02, 1D02, 1E01, 2A02, 2A05, 2C01, 2C02, 2C05

Learning Objectives

After reading this article, you should be able to:

- List clinical features that indicate the presence of an inhalational injury
- Identify risk factors for and signs of noxious agent involvement in a severe burn
- Outline the key aspects of severe burn pathophysiology including burn shock
- Apply evidence-based practice to commence resuscitation of severe burns patients
- Understand the role of the multi-disciplinary team in burns patient care

Introduction

Throughout one year in England, 116,588 patients attended Emergency Departments with a burn or scald, 12,667 of whom required inpatient treatment.[1,2] A specialized multidisciplinary team with an enhanced understanding of severe burns pathophysiology helps ensure the delivery of safe, high quality care to this specific subgroup.

Gender, age, size and depth of burn are all important factors in prognostication. Between 5% and 35% of burns patients suffer inhalational injury which is associated with significant complications.[3,4]

The need for a co-ordinated approach via a strategically developed burns network is fundamental to reducing morbidity and mortality, whilst promoting high quality specialist care.[5]

Pathophysiology

Irrespective of burn modality, local (Table 1) and systemic effects ensue.

The systemic response begins immediately and is driven by the release of both inflammatory mediators and oxygen radicals. This inflammatory response creates both the hyperdynamic and hypermetabolic phases, driving multi-organ failure.

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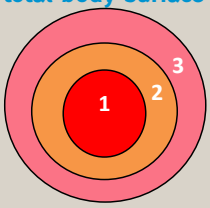
The effects on the cardiovascular system in response to burns are divided into acute and hypermetabolic phases. Hypovolaemia dominates the acute phase, generated by increased capillary permeability resulting in losses of both protein and fluid volume to the extravascular space. The hypermetabolic phase results in hypoproteinaemia causing gross tissue oedema, whilst excess catecholamine synthesis can lead to cardiac dysfunction and acute kidney injury secondary to ischaemia or infarction.[6]

Thermal injury results in multiple gastrointestinal tract complications. Reduced nutritional intake, increased incidence of gastric mucosal ulceration and bowel wall ischaemia may all result in gastrointestinal haemorrhage. Translocation of bacteria across the mucosal wall may also result in sepsis.

There are two aspects to inhalational injury pathophysiology: direct thermal inflammatory response and smoke inhalation. Local cellular inflammation occurs throughout the entire respiratory tract including the parenchyma, resulting in oedema,

Jackson's burn zones and their relevance in calculating total body surface area (TBSA) of any burn



1) Zone of coagulation	Total and irreversible tissue destruction	Include in TBSA calculation
2) Zone of stasis	Damaged tissue suffering reduced perfusion. Salvageable and main target in resuscitative therapy for healing	
3) Zone of hyperaemia	Outermost zone with increase in perfusion and is highly likely to recover from the original insult	Not included in TBSA calculation

Table 1

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irritation and raised airway pressures that can cause challenges during positive pressure ventilation (PPV). Smoke inhalation can cause either direct asphyxiation with O_2 displacement in the alveoli, or systemically via carboxyhaemoglobinaemia.

Assessment and management

The consideration of and transfer to a specialist burns centre is often managed regionally or nationally and local referral guidelines should be consulted.

Airway

C–spine protection must be implemented if there is a suspicion of trauma. Immediate application of supplemental oxygen therapy with high-flow humidified oxygen inhibits secondary injury and provides prompt treatment of smoke inhalation. Risk factors and clinical signs indicating inhalational injury must be recognized early (Table 2).

No guideline exists to instruct on what collective features require a definitive airway. The progressive and potentially fatal complications of an insecure, obstructed or oedematous airway, especially in those requiring transfer, often generates an indoctrinated reaction – intubation. The dogma of endotracheal intubation has to be questioned considering 31% of intubated patients are extubated within the first twenty-four hours of admission at burns centres.[7] Fibreoptic nasendoscopy can help to further stratify the presence of or risk of inhalational injury and help guide decision making on whether to proceed with intubation or opt instead for a period of close observation in a high dependency unit.

Should intubation of the trachea be required, a clear airway plan must be made and communicated to all members of the emergency team. Consideration should be given to awake fibreoptic intubation versus sedation and muscle relaxation. Restricted mouth opening, oedema of the face and upper airways and carbonaceous deposits within the oropharynx mean a 'can't intubate, can't ventilate' scenario must be planned for with equipment prepared in advance.

Suxamethonium use should be limited to within the first 24 hours of injury due to the risk of hyperkalaemia caused by the increase in extrajunctional nicotinic acetylcholine receptors. Increased receptor numbers can also promote a resistance to non-depolarizing agents, requiring higher doses.

The endotracheal tube (ETT) must have a sufficient internal diameter to allow passage of a fibreoptic bronchoscope. The ETT should never be cut short as facial oedema may increase.

Bronchoscopy must be carried out post-intubation in order to fully assess the presence of inhalational injury and/or burns to the lower airway.

Breathing

A lung protective ventilatory (LPV) strategy (Table 3) should be implemented in those who require intubation. Between 40% and 54% of patients requiring mechanical ventilation will develop acute respiratory distress syndrome (ARDS).[4] Rescue interventions including muscle relaxation, prone positioning and extracorporeal membrane oxygenation (ECMO) may be required in cases of refractory hypoxaemia.

Rising plateau pressures and inadequate ventilation should prompt consideration of either circumferential burns of the thorax and abdomen or abdominal compartment syndrome.

The affinity of haemoglobin (Hb) for carbon monoxide (CO) is approximately 200 times that for oxygen (O_2), thus hypoxaemia results from displacement of O_2 from the Hb molecule by CO. A carboxyhaemoglobinaemia (COHb) of >30% requires rapid treatment; increasing the alveolar partial pressure of oxygen by instigating a FiO_2 of 1.0 whilst optimizing ventilation. This reduces the half-life of COHb from four hours to approximately one hour. Hyperbaric oxygen treatment is rarely required.

Circulation

Peripheral venous access with two large-bore catheters via unburnt skin is a priority. An arterial line and central-venous catheter, inserted through unburnt skin, should be considered early. A urinary catheter must be inserted and hourly urometer commenced as urinary output is a useful measure of the adequacy of perfusion.

A balanced crystalloid solution should be used for initial resuscitation; however, the optimal fluid choice continues to be debated. Packed red cells and coagulation products may be required as bleeding from excised wounds can be significant. Hypertonic solutions such as albumin are also showing

Risk factors and clinical signs that indicate inhalational injury

Risk factors	Clinical signs
Exposure to smoke, flames or chemicals and whether these were either industrial or household	Carbonaceous sputum
Duration of time exposed and whether this was in an enclosed space	Evidence of burns to the face or neck
Burning substances such as plastics or fabrics	Oropharyngeal burns (e.g bulla and/or erythema)
Obtunded consciousness at scene	Respiratory embarrassment
At scene fatalities or cardiac arrests	Singed facial or nasal hair
	Added sounds (wheeze or stridor)
	Haemoptysis
	Altered voice
	Odynophagia or dysphagia
	Reduced Glasgow Coma Scale (GCS)

Table 2

Lung protective ventilation strategy criteria determined by the ARDS network

Tidal volume	6 ml/kg (range 4–8 ml/kg titrated against plateau pressures and blood pH) *predicted body weight*
Plateau pressure	≤30 cm H_2O
FiO_2	Wean as low as tolerated to achieve PaO_2 55–80 mmHg (7.3–10.7 kPa)

Table 3

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