Burns injury Part III: Inhalation Injury

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Intended Learning Outcomes

Burns injury Part III: Inhalation Injury

1. Recognise the spectrum of disease that may occur following inhalation of noxious substances
2. Describe the features, assessment and management of upper airway injury.
3. Describe the features, assessment and management of lower airway injury
4. Recognise the potential for and features of ARDS in this population
5. Outline the systemic effects of inhaled toxins and their treatment.

eModule Information

Relevant Competencies from CoBaTrICE

Burns injury Part III: Inhalation Injury

- **2.1** Obtains a history and performs an accurate clinical examination.
- **2.2** Undertakes timely and appropriate investigations.
- **2.6** Obtains and interprets results from blood gas samples
- **2.7** Interprets chest x-rays
- **2.9** Monitors and responds to trends in physiological variables
- **2.10** Integrates clinical findings with laboratory investigations to form a differential diagnosis
- **3.1** Manages the care of the critically ill patient with specific acute medical conditions.
- **3.10** Recognises and manages the patient following intoxication with drugs or environmental toxins.

1. Executive Summary

Inhalation injury can occur when the respiratory tract suffers direct damage from heat or smoke, or when a patient develops systemic pathology following inhalation of toxins. This module covers the assessment and management of upper and lower airway inhalation injury and subsequent complications. It also addresses the clinical consequences of inhaled toxins and their specific management.

2. Types of Inhalation Injury

Although burn injuries most commonly involve the skin, inhalation of noxious substances can produce a spectrum of disease that further complicates the clinical course of patients and significantly increases the risk of death.

2. 1. Definitions

Inhalational injury is an umbrella term that is variously used to describe:

- respiratory tract injury mediated by heat, smoke or other particulates; and
- systemic complications of inhaled toxins that are absorbed into the bloodstream.

It can occur in situations such as fires, chemical spills, or industrial accidents and lead to complications as diverse as upper airway obstruction, respiratory failure, coma and histotoxic hypoxia.

The heterogeneity in cause, effect and treatment represents a barrier to research in this area. However, it has been shown that patients sustaining an inhalation injury in the context of a burn injury require up to 50% more fluid in the resuscitation phase and have a poorer prognosis, to the extent that inhalation injury is now recognised in the modified Baux score as conveying a mortality risk equivalent to a cutaneous burn covering an additional 17% total body surface area (TBSA) (Osler, Glance and Hosmer. 2010).

References

- Osler T, Glance LG, Hosmer DW., Simplified estimates of the probability of death after burn injuries: extending and updating the baux score., 2010, PMID:20038856

2. 2. Clinical manifestations

The clinical manifestations and severity of inhalation injury will depend on the:

- substances inhaled,
- size of the smoke particles,
- solubility of inspired gases and
- duration of exposure.

The latter is a function of the environment in which the injury occurs. Enclosed spaces with poor ventilation provide ideal conditions for incomplete combustion of materials and accumulation of noxious fumes that go on to mediate one or more forms of inhalation injury (Table 1).

Table 1: Characteristics of inhalation injury affecting various anatomical sites.

Type of Injury	Cause	Effect	Diagnosis
Upper airway	Thermal insult	• Hoarseness • Odynophagia • Stridor	• Flexible nasendoscopy • Laryngoscopy
Tracheobronchial	• Chemical irritants • Particulate matter	• Dyspnoea • Cough • Wheeze	• Bronchoscopy
Lung parenchymal	Inflammatory response to noxious stimuli	• Hypoxaemia	• Imaging
Systemic toxicity	• Carbon monoxide • Hydrogen cyanide	• Disturbed consciousness • Haemodynamic lability	• Carboxyhaemoglobi level • Lactic acidosis • Raised venou oxygen saturation

Thermal injury primarily affects the proximal respiratory tract causing tissue oedema and potential upper airway obstruction. Due to the dissipation of heat in the upper airways, thermal injury below the level of the vocal cords is rare but can occur with the inhalation of substances such as superheated steam.

Tissues below the level of the vocal cords are more likely to be damaged by chemical irritants and particulate matter. Soot can cause luminal obstruction, impairing gas flow through the lungs. Chemical irritants can generate both localised and systemic inflammation leading to mucosal hyperaemia and activation of the clotting cascade with fibrin casts further compounding luminal obstruction.

Within the lung parenchyma, the inflammatory response to noxious stimuli leads to increased pulmonary vascular permeability, alveolar oedema, ventilation/perfusion mismatch and hypoxaemia commensurate with acute respiratory distress syndrome (ARDS).

Absorption of chemicals from the respiratory tract into the bloodstream causes specific and diffuse effects that vary with the toxin involved. Carbon monoxide, commonly produced by the combustion of carbonaceous material in an oxygen poor environment, is the most common toxin implicated. Hydrogen cyanide is another potential contributor that should be considered in patients with cardiovascular and metabolic compromise.

The various type of inhalation injury become manifest in slightly different timeframes (Figure 1).

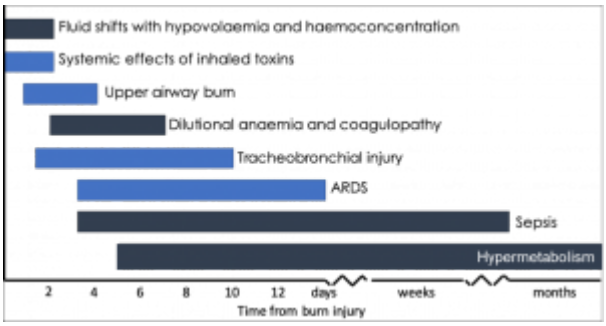

Figure 1: Time from insult to complications in burns patients.

3. Upper airway burn injury and assessment

One of the first priorities in assessing a patient with burns is to determine the risk of upper airway injury. This is usually a thermal injury with the heat from inspired gases being dissipated to tissues by the efficient heat exchange mechanisms in the proximal respiratory tract. Chemical ingestion is a less common trigger (Video 1).

Video 1. Upper airway oedema due to chemical burn

An upper airway burn should be considered if there is soot, singed hair or cutaneous burns in the region of the nose and mouth. Patients with oral burns may report throat pain or tongue swelling and supraglottic involvement causes difficulty swallowing secretions, dysphonia and stridor. Life-threatening airway obstruction may supervene rendering definitive airway management time-critical in such patients.

Upper airway burns can be identified by the presence of erythema, oedema and ulceration on direct inspection of the oral cavity, flexible nasendosopy or laryngoscopy. Flexible nasendscopy is a diagnostic tool that involves inserting a flexible tube with a camera into the airway, usually via the nose. It may help inform decisions around endotracheal intubation when the most appropriate course of action is not immediately apparent. Gas exchange abnormalities are a late sign of airway obstruction and their absence should not be regarded as reassuring.

When intubation is pursued, it is important that the endotracheal tube is left uncut. This allows the tube to be resecured at incrementally greater depths preventing proximal herniation of the tube cuff, as the face, especially if burnt, swells markedly in the first

48 hours of burns critical care. Standard diameter tubes should be used where possible to optimise airway toileting and subsequent bronchoscopic evaluation of the tracheobronchial tree.

There is no specific treatment for upper airway injury. However, good supportive care, sitting the patient up (as much as other injuries allow) and medications targeting gastro-oesophageal reflux generally lead to resolution of airway oedema within days. Occasionally, a tracheostomy may be placed to facilitate healing of facial burns (as oral endotracheal tubes and ties could be detrimental) or when other indications for the procedure apply. A single centre randomised controlled trial showed no benefit of early tracheostomy in the burn-injured population (Saffle, Morris and Edelman. 2002). However, a more recent multidatabase analysis of 9173 burn patients undergoing tracheostomy found that tracheostomy within 10 days decreased ICU and hospital length of stay without increasing discharge to long term acute care or inpatient rehabilitation (Shah et al. 2025).

Prior to extubation, sufficient resolution of upper airway oedema can be confirmed by repeat laryngoscopy or nasendoscopy. Alternatively, a leak test may be performed in which the endotracheal tube cuff is deflated and inspired gas allow to exit via the patient's upper airway. If such a leak is heard or measured by the ventilator, then space must exist around the endotracheal tube and significant airway narrowing is unlikely.

Allied health professionals play a key role in rehabilitating bulbar function in these patients. Long term sequelae may range from no symptoms through mild scarring to severe laryngotracheal stenosis meriting corrective surgery.

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4. Lower airway burn injury and assessment

Damage to the tracheobronchial tree is usually mediated by chemicals and particulate matter in inspired smoke. With inhalation of supraheated steam, there may also be a thermal component to the injury. These mucosal insults incite an inflammatory response with reactive hyperaemia of the airways and bronchorrhoea. Activation of the coagulation cascade generates fibrin casts, which together with airway oedema and carbonaceous deposits predispose to airway obstruction. There is associated dysfunction of mucociliary clearance and significant risk of superadded lower respiratory tract infection. Occasionally, the mucosal injury may be so severe as to cause ulceration and sloughing that can further exacerbate endobronchial obstruction.

Symptoms of lower airway injury include dyspnoea, wheeze and cough, often with carbonaceous secretions. Chest imaging may be normal initially, although focal consolidation and diffuse bilateral infiltrates may be seen as complications ensue (Gill and Martin. 2015).

References

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4. 1. Diagnostic bronchoscopy

Fibreoptic bronchoscopy is considered the gold standard for diagnosing and assessing the severity of tracheobronchial injury in burns patients.

The Abbreviated Injury Score (AIS) for gradation of inhalation injury can be used to assess severity of injury based on bronchoscopic appearances (Table 2 and Figure 2). The grading of inhalation injury has been demonstrated to predict outcomes, although this has not been consistent, possibly due to interobserver variation in grading However, there is a strong association between the presence of any inhalation injury at bronchoscopy and increased duration of invasive mechanical ventilation, ICU length of stay and mortality.

Table 2: AIS gradation of inhalation injury

Grade	Features
Grade 0: No injury	Absence of carbonaceous deposits, erythema, oedema, bronchorrhoea or obstruction
Grade 1: Mild injury	Minor or patchy areas of erythema, carbonaceous deposits in proximal or distal bronchi (any or combination)

Grade 2: Moderate injury	Moderate degree of erythema, carbonaceous deposits, bronchorrhoea with or without compromise of the bronchi (any or combination)
Grade 3: Severe injury	Severe inflammation with friability, copious carbonaceous deposits, bronchorrhoea, bronchial obstruction (any or combination)
Grade 4: Massive injury	Evidence of mucosal sloughing, necrosis, endoluminal obliteration (any or combination)

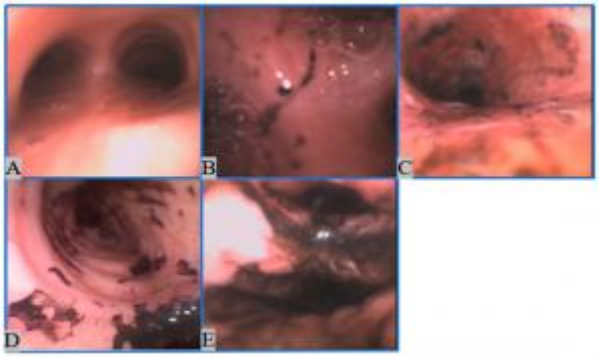

Figure 2: Examples of grading of inhalation injury using the abbreviated injury score (AIS). (A)—no injury; (B)—mild injury; (C)—moderate injury; (D)—severe injury; (E)—massive injury. Reproduced from Li et al under the terms of the Creative Commons Attribution Non Commercial License 4.0.

Video 2: Bronchoscopic footage of massive (grade 4) tracheobronchial injury with circumferential mucosal sloughing extending down the trachea and main bronchi.

In Text References

(Li et al. 2022)

References

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4. 2. Bronchial toilet

While the diagnostic role and prognostic impact of bronchoscopy in identifying tracheobronchial injury is well supported, there is minimal evidence for intervention in the form of bronchoalveolar lavage. Some centres advocate repeated lavage with saline or sodium bicarbonate until the airways are clear of soot. However, there is theoretical risk of removing surfactant and predisposing to atelectasis with this approach. In the absence of a clear outcome benefit, it is our practice to reserve bronchoalveolar lavage for those patients with proximal airway obstruction and consequent gas exchange abnormalities. Beyond this scenario, bronchial toilet is routinely aided by regular suctioning, chest physiotherapy and mobilisation. There is no role for prophylactic antibiotics or steroids.

4. 3. Nebulised therapy

The role of nebulised therapy in lower airway inhalation injury is not yet clear, although several types of agent have been trialled and advocated by expert panels (Milton-Jones et al. 2023).

N-acetylcysteine (NAC) is a mucolytic that may convey additional benefit in neutralising reactive oxygen species. It is an attractive option in inhalation injury where the secretion load is increased and the mucociliary escalator impaired. However, when nebulised, NAC has the potential to irritate the airways and cause bronchoconstriction. Co-administration of a bronchodilator is therefore advocated.

Bronchodilators target airway reactivity in inhalational injury. Beta-adrenergic agonists (e.g. salbutamol) are generally used and have been shown in animal models to improve pulmonary functioning (Palmieri et al. 2006). However, pending human trials, our practice is to restrict their use to potentially reversible airflow obstruction. The routine use of such drugs should be avoided in patients with ARDS, in which these agents have been linked to harm (Gates et al. 2013).

Nebulised heparin has perhaps shown most promise as a treatment for lower airway inhalation injury. It conceivably exerts beneficial effects in reducing the inflammatory response and fibrin cast formation. Previous studies have utilised 5000 to 10,000 units every 4 hours alternating with NAC and bronchodilators (Lan et al. 2020). While these studies have shown potential benefit of nebulised therapy in terms of duration of mechanical ventilation and mortality, they have methodological flaws in that none are randomised and most are retrospective analyses utilising historical controls over a period where burn outcomes are improving with time.

The HEPBURN trial sought to provide better quality evidence via a prospective international randomised controlled trial. However, it was stopped prematurely due to inadequate recruitment and highlighted drug delivery issues with saturation of filters and concern regarding bleeding risk (Glas et al. 2020). Recently, a single centre, blinded RCT involving 88 patients showed more days alive and free of ventilation at 28 days (but no difference in mortality) in patients receiving 5000 units nebulised heparin 4 hourly in addition to N-acetylcysteine and albuterol. However, further collaborative research is awaited.

In Text References

(Hakim et al. 2025)

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5. ARDS in patients with burn injury

5. 1. Causes of ARDS

Acute Respiratory Distress Syndrome (ARDS) is characterised by non-cardiogenic pulmonary oedema and lung inflammation driven by the profound inflammatory response initiated by a burn injury.

Direct lung injury leading to ARDS can occur due to thermal injury or, more commonly, due to inhalation of aerosolised irritants. These trigger inflammation and damage within the lungs and subsequent widespread release of proinflammatory molecules, worsening the systemic inflammatory response experienced by burn injured patients with inhalation injury (Albright et al. 2012).

ARDS can also occur with burn injury in the absence of inhalation injury, when systemic inflammation and aggressive fluid resuscitation lead to alveolar oedema. A similar clinical pattern is seen with other causes of ARDS such as sepsis, trauma and pancreatitis.

As with any cause of ARDS, severity is categorised based on the degree of respiratory dysfunction measured by the ratio of the partial pressure of oxygen in arterial blood to the inspired oxygen concentration (P:F ratio) (ARDS Definition Task et al. 2012).

References

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5. 2. General principles of management

The management of ARDS is largely supportive, and most approaches have been translated from the non-burn population. The standard approach to lung protective mechanical ventilation (LPV) pioneered by the ARDSnet group includes limitation of volutrauma by targeting patient-specific tidal volumes, and barotrauma by limiting airway plateau pressures (ARDS_Network. 2000). Unique features of burn patients

may affect the application of LPV, with poor chest compliance often experienced by patients with circumferential chest wall injuries or dressings leading to high airway pressures. Furthermore, the burn-associated hypermetabolism can lead to significant hypercapnia in the context of low tidal volume ventilation and adjustment to the respiratory rate and/ or tidal volumes may be required.

Prone positioning has evidence for improved survival in patients with moderate/ severe ARDS (Guérin et al. 2013) however, evidence is lacking for use in burns patients. While it is likely to improve gas exchange and lead to more favourable respiratory dynamics, there are specific logistical and safety concerns for patients with burn injury. Position changes increase the risk of airway dislodgement, which could be potentially life-threatening in patients with upper airway burns and consequent swelling. Furthermore, the impact of position changes on recently grafted skin can lead to higher failure rates. Careful consideration of abdominal compression during prone positioning needs to be made for all patients but should be particularly considered in the burn injury cohort who have a higher risk of intra-abdominal hypertension (due to high volume fluid resuscitation) and are more vulnerable to nutritional deficiencies if position changes lead to impaired gastric emptying.

Neuromuscular blocking agents are sometimes used in patients with severe ARDS to enhance gas exchange and reduce plateau pressure. There is no specific evidence for or against their use in burn-injured patients, however, consideration should be given in patients with a high minute volume due to hypermetabolism. These patients will often need high respiratory rates with potentially increased tidal volumes to mitigate respiratory acidosis from hypercapnia.

For patients with refractory hypoxia, extra-corporeal membrane oxygenation (ECMO) may have a role as a rescue technique. Similar outcomes for burn-injured patients with ARDS on ECMO compared to non-burn patients has been demonstrated (Nosanov et al. 2017). However, consideration should be made to the requirement for anticoagulation and whether further operative intervention is planned.

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6. Systemic Effects of Inhaled Toxins

Inhaling toxic substances generated by combustion may have direct systemic effects. The two agents of most relevance are carbon monoxide and hydrogen cyanide. However, volatile organic compounds and polycyclic aromatic hydrocarbons may also exert effects beyond the respiratory tract, including carcinogenesis.

6. 1. Carbon Monoxide

Carbon monoxide is a colourless, odourless gas produced by incomplete combustion of carbonaceous fuels. Poisoning with this gas may accompany burn injuries, particularly those sustained in enclosed spaces and with prolonged extrication due to entrapment or coma. Carbon monoxide exposure may also arise deliberately in suicide attempts utilising vehicle exhaust fumes and accidentally with faulty heaters.

Carbon monoxide has high affinity for haemoglobin, displacing oxygen from its binding sites and thereby reducing tissue oxygen delivery and causing cellular damage as a consequence of anaemic hypoxia.

Features of carbon monoxide toxicity are contingent on the extent to which it displaces oxygen from haemoglobin (Table 3). This, in turn, depends on the carbon monoxide concentration in inspired gas and the duration of exposure.

Table 3: Features of carbon monoxide toxicity

COHb Level	Features
<10%	Asymptomatic
10-20%	Non-specific symptoms
20-40%	Headache Nausea / vomiting Confusion

40-60%	Seizures Coma Dysrhythmias Respiratory distress
>60%	Cardiorespiratory collapse

Carbon monoxide toxicity increases with minute ventilation, metabolic rate and anaemia. Smokers have increased carboxyhaemoglobin (COHb) levels at baseline (around 3 to 10%) and require less exposure before developing clinical features of toxicity.

In the presence of COHb, the patient's measured oxygen saturations(SpO_2) will reflect the sum of COHb and oxyhaemoglobin, and thus, may considerably overestimate oxyhaemoglobin. The pulse oximeter cannot therefore be relied upon to determine blood oxygenation in patients with recent carbon monoxide exposure. Where the clinical history is suggestive of carbon monoxide exposure, COHb level should be measured by co-oximetry. It is worth noting that COHb levels change rapidly. Hence, the COHb level at presentation may be falsely reassuring, particularly if there have been delays in measurement and oxygen therapy in the interim.

Management of carbon monoxide poisoning involves removing the patient from the source and administering high concentration oxygen. This raises the arterial oxygen tension and displaces carbon monoxide from the haemoglobin molecule, whereupon it can be eliminated unchanged via the lungs, Administration of 100% oxygen reduces the half-life of carbon monoxide from 4 - 5 hours to 40 - 80 minutes. High flow oxygen administration should be continued until the measured COHb level is <5%. In severe cases, hyperbaric oxygen therapy may be considered to reduce the risk of long-term neurological sequelae. At two atmospheres, the half-life of carbon monoxide is further reduced to 23 minutes. However, this is impractical for the majority of critically injured burns patients and not without complications.

The foetus is at particular risk for toxicity as foetal haemoglobin has greater affinity for carbon monoxide. Also, the half-life of carbon monoxide is prolonged and arterial oxygen tension greatly reduced in the foetal circulation. Hyperbaric oxygen may have a particular role to play in this scenario as it allows increased delivery of oxygen to the tissues independent of haemoglobin, as well as promoting displacement and elimination of carbon monoxide.

6. 2. Cyanide

Cyanide can be inhaled as a component of smoke when plastics and other synthetic polymers burn. It binds the ferric ion of cytochrome oxidase, leading to histotoxic hypoxia with cells unable to utilise oxygen. Anaerobic metabolism ensues and a lactic acidosis is generated. Cyanide is also thought to exert a direct neurotoxic effect mediated by NMDA release and resulting in seizures and altered mental status.

Victims of fires may therefore present with cardiovascular or neurological disturbance at least partially driven by cyanide toxicity. Measurement of cyanide levels is possible, but results are seldom available within clinically useful timeframes. So, the diagnosis must be considered, and treatment instituted on clinical grounds.

Cyanide toxicity is suggested by a history (or evidence) of smoke inhalation and lactic acidosis that is poorly responsive to fluid resuscitation or associated with high venous oxygen saturations (reflecting poor oxygen extraction by the tissues).

Management requires a specific antidote to convert the cyanide ion to a non-toxic metabolite. Most centres now use intravenous hydroxycobalamin at a dose of 5g (70mg/kg for children) over 15-30 minutes for this purpose. The dose can be repeated if needed. Hydroxycobalamin has few side effects other than benign orange-red discolouration of the skin and body fluids lasting around 48 hours.