

Key points

- ▶ All patients with facial burns may be suspected of having "difficult-to-control" airways owing to smoke inhalation injury (SII). Many of them either have an incorrect diagnosis, or mild-to-moderate injury with unrecognised aggravating respiratory failure.
- ▶ For a diagnosis of inhalation injury, it is necessary to follow the patient closely for >48 h.
- ▶ Inhalation injury is a condition with different clinical presentations. Clinical follow-up is necessary to improve patient care, to help guide treatment and to provide clues for therapeutic interventions.
- ▶ Notwithstanding intensive care treatment including airway intubation and mechanical ventilation, many patients with severe inhalation injury remain under-treated.



Management of blast and inhalation injury

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Educational aims

- › To discuss the initial approach and assessment of a patient with SII.
- › To help the reader recognise different clinical pictures of inhalation injury.
- › To outline management and discuss treatment.

Summary

"Inhalation injury" describes a variety of insults caused by the aspiration of superheated gases, steam or noxious products of incomplete combustion. Inhalation injury involves the entire respiratory system. Early diagnosis based on history and physical examination, in addition to careful monitoring for respiratory complications, is mandatory. As there is no specific treatment for inhalation injury, management involves providing the necessary degree of support required to compensate for upper airway swelling and impairment in gas exchange. Airway intubation and mechanical ventilation may be required while the endobronchial and alveolar mucosa are regenerating.

Primary blast injury (BI) is caused by immediate pressure variations, which are the product of rapid sequences of compression and decompression. Secondary and tertiary BI include lesions caused when the subject is thrown against rigid structures or is hit by flying objects. Its diagnosis and therapy follows guidelines for emergency care.

▶ A major burn is one of the most devastating physiological and psychological insults known. Severe burns involve a skin injury accompanied by a serious systemic illness with consequences in different distant organs.

Major burns are a serious clinical challenge and have a high mortality rate. Patient age, the percentage of total burned surface area (TBSA) and the presence of multi-organ dysfunction syndrome (MODS) are the main factors influencing outcome. MODS is the leading cause of death (from one-third to two-thirds of deaths in the burn population), and among organs, the lungs are invariably affected (100%), followed in frequency by the gut and kidneys (68%) [1].

In the past 20 years, burn-related mortality has decreased in western countries. This is the result of primary prevention of all causes of burns and the introduction of specific treatment protocols for burn shock. This includes early fluid resuscitation as well as an early diagnosis of suspected smoke inhalation or blast injury. Thereafter, primary prevention also includes an immediate admission to the care of a specialised burns team. A team approach including all caregivers (intensivist, surgeon, nurses, respiratory therapist and psychologist) is mandatory.

Respiratory care, independent of the type of injury is as important as other major components of burn care, such as fluid management, wound coverage and infection control.

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Table 1 Involvement of the respiratory system in burns patients

Early airway involvement (up to 48–72 h)	Late airway involvement (after 48–72 h)
Upper airway injuries	Lower airway injuries
Lower airway injuries	

In the present review, only respiratory involvement (including that caused by smoke inhalation or blast injury) will be tackled. Major burn management, carbon monoxide poisoning and cyanide exposure are beyond the scope of this article.

Respiratory system involvement

Burns can be the result of thermal, chemical, electrical or inhalation injury. Although many organ systems may be affected by a burn, the respiratory system often sustains the most damage. The severity of this insult may range from mild to life-threatening. Injury of the respiratory system in major burns patients may assume many forms. Table 1 summarises the involvement of the respiratory system in burns patients.

Lung injury may result directly from smoke inhalation injury to the lungs or indirectly from inflammatory mediators associated with infection, sepsis, the burn itself or blast injury [2].

It has been estimated that a patient aged >60 years, with >40% TBSA and with inhalation injury, has a >90% probability of death [3].

Thermal and chemical injuries cause coagulative necrosis of the skin and underlying subcutaneous tissue. The major determinants of burns severity are the extension of injury (as % TBSA) and its thickness (partial or full thickness). Burns elicit a time-dependent (from minutes to hours) sustained local and systemic release of inflammatory mediators and changes in hormonal and immunological responses, proportional to their extent and thickness. Circulating and local mediators include cytokines (interleukin (IL)-1, IL-2, IL-6, IL-8, IL-12 and tumour necrosis factor), growth factors, activation products of coagulation and contact phase cascades, complement factors, nitric oxide, platelet activating factor, prostaglandins and leukotrienes. These molecules, present at high concentrations for days, lead to marked vasodilation, generalised increased microvascular permeability and extravascular fluid loss, as well as hypotension if hypovolaemic shock is not properly treated. In

the gut, a few hours after burn injury, an increased mesenteric vascular resistance and decreased gut perfusion can be observed leading to bacterial translocation and endotoxaemia. Hormonal effects include increased levels of cortisol, glucagon and catecholamines, affecting several metabolic functions and causing negative nitrogen and calcium balance, lipolysis, massive peripheral muscle wasting and hepatic fat deposition.

Acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) are likely to occur at any time during the clinical course of the burns patient [4].

DANCEY *et al.* [5] estimated that the incidence of ARDS among mechanically ventilated burn patients is as high as 54%. As a consequence, pulmonary injury is a major source of morbidity and mortality for the burns patient [6].

Smoke inhalation injury

SII is defined as a clinical picture including a variety of insults that are attributable to the inhalation of superheated gases, steam or gases resulting from incomplete combustion of noxious products. It has an incidence of approximately 20% in patients admitted to major burn centres.

A diagnosis of SII may double mortality from that predicted based on age and burn size alone [3].

The mechanism of inhalation injury consists of a combination of [7]:

- direct thermal injury to the upper airway from the inhalation of hot gases
- damage to cellular and oxygen transport mechanisms by inhalation of carbon monoxide and cyanide
- chemical injury to the lower airways caused by inhalation of toxic products of combustion.

The clinically important problems include:

- loss of airway patency secondary to mucosal oedema
- bronchospasm
- intrapulmonary shunting with decreased lung compliance secondary to alveolar flooding and collapse
- pneumonia secondary to loss of ciliary clearance
- bronchiectasis.

Although air temperature in a room containing a fire may exceed 550°C [8], superheated air usually causes thermal injury only to airway structures above the carina. This is because of the combination of efficient heat dissipation in the upper airway, the low heat capacity of air and reflex closure of the larynx. The combustion of most substances may generate toxic materials. Toxins generated by smoke-related products damage both epithelial and capillary endothelial cells of the airway. Table 2 summarises the toxins produced by burning materials [9].

Histological findings in SII show damaged alveolar macrophages producing chemotaxins, which further enhance the inflammatory response. A period of diffuse exudate formation with bronchiolar oedema and an increase in capillary permeability follows inflammatory changes, causing an increase in extravascular lung water (EVLW), which may be worsened by vascular filling during the hypovolaemic state. Respiratory failure is likely to occur within 12–48 h after the smoke exposure, caused by decreased lung compliance, increased airway resistance, increased ventilation perfusion mismatch and increased dead-space ventilation. Early ARDS can lead to death within 48 hours.

Blast injury

When burns are due to an explosion, BI may occur. The potential for a blast to cause lung injury depends on the nature of the explosive and the environment in which the blast occurs.

The environment is important in modifying the effect of blast. The size of the zone of risk depends on the type of explosive, the environment and the size of debris. Reflection from surfaces, such as walls, can enhance the blast effect. Multiple reflections can greatly increase blast wave energy and also produce a sustained or reverberating period of overpressure.

BI can be divided into primary, secondary and tertiary categories.

Primary blast injury

Primary BI is the result of immediate pressure variations caused by rapid sequences of compression and decompression. BI may produce some specific forms of injury if the chest and lung are exposed to high or prolonged overpressure due to the blast wave. Explosions in an enclosed environment are associated with

Table 2 Toxins produced by burning materials

Burning substance	Toxins produced
Rubber and plastic products	May produce sulphur dioxide, nitrogen dioxide, ammonia and chlorine, which form strong acids and alkalis when combined with water in the airways and alveoli
Glues in laminated furniture and wall panelling	May release cyanide gas
Cotton or wool	May produce toxic aldehydes

increased risk of pulmonary BI. Enclosed environments also increase the risk of air and fat embolism and pleural tearing as a result of trauma to the connective tissue between alveolar spaces and pulmonary veins. Lungs are particularly susceptible to damage, owing to their extensive air/lung tissue interfaces. This is also true of other hollow organs such as the gastrointestinal tract (bowel contusion/perforation) and middle ear (tympanic perforation) [10].

Pulmonary contusion represents the main anatomical-pathological feature of BI. It includes intralesional and perilesional oedema in the boundary zone. Macroscopic lesions include capillary-alveolar damage, which is responsible for intraparenchymal bleeding, as well as distal vascular thrombosis.

Secondary and tertiary blast injury

In this case, the lesion is caused when the subject is thrown against rigid structures or when the subject is hit by flying objects.

Early upper and lower airway injuries

In early upper airway injuries acute respiratory failure is usually caused by:

- damage above the glottis by superheated, noxious gases and damage below the glottis by chemical agents (SII). Thermal injury to the upper airway may result in massive swelling of the tongue, epiglottis and/or aryepiglottic folds with resultant airway obstruction
- indirect damage of airways due to neck and face oedema
- BI.

Early lower airway injuries are usually due to:

- SII
- traumatic events caused by BI.

Although most pulmonary injuries are related to the size of the cutaneous burn, SII may cause a significant pulmonary injury even without other burns.

Late airway involvement

In patients still breathing spontaneously, decreases in functional residual capacity and lung compliance may be caused by:

- alveolar collapse and atelectasis in the dependent zone of the lung
- alterations of mucociliary transport that inhibits the clearance of bacteria
- surfactant loss
- sputum retention due to impaired cough efficacy (forced immobilisation especially in elderly and heavily overweight patients, chest constriction caused by the burn itself, associated chest or abdominal lesions).

In patients with SII, lesions may progress because of intrapulmonary haemorrhage, resulting in mechanical obstruction of the lower airways and flooding of the alveoli [7].

Because of ulceration and extensive necrosis of the respiratory epithelium, contemporary pulmonary superinfections are very frequent. Patients are predisposed to secondary bacterial invasion and development of a superimposed bacterial pneumonia [11].

The appearance of sepsis is also very common in mild burns (25–30%). ARDS is likely to occur and MODS is still the leading cause of death, with lungs invariably affected [1].

How to approach the patient with respiratory system involvement

A major burns patient is a patient in continuous evolution. A history of exposure to a closed-space fire, the presence of superheated gases or chemical irritants, loss of consciousness, singed nasal or facial hair and a physical examination revealing carbonaceous sputum are all suggestive of inhalation injury.

Patients may have little or no pulmonary dysfunction at initial presentation, as initial chest radiograph is often normal. Blood gases may be normal for the first few hours after injury and thus may not be helpful, especially before fluid resuscitation is complete. Continuous monitoring of respiratory functions is mandatory.

If SII is suspected, fiberoptic bronchoscopy may help in diagnosis, but this should not take the place of clinical judgment. Bronchoscopic findings include carbonaceous endobronchial debris, mucosal pallor, erythema and mucosal ulceration.

Oxygen administration at a fixed percentage, using a Venturi system, enables the physician to calculate the arterial oxygen tension/inhaled oxygen fraction ratio. Early catheterisation of a radial or pedial or even femoral artery (according to the availability of nonburned tissue) allows frequent and less traumatic gas exchange monitoring.

Airway patency

Loss of airway patency caused by mucosal and tissue oedema usually occurs after some time has passed and resuscitation fluid has been started. If the history, initial examination and clinical picture lead the physician to suspect a severe thermal upper airway injury, airway protection with intubation should be considered before mucosal and tissue swelling make intubation very difficult.

Upper airway swelling may increase over time with fluid replacement even when the initial examination suggests the lesion is mild. Airway patency should be monitored continuously to assess the need for airway control and ventilator support. Factors that may predict airway intubation in major burn are listed in box 1 [12].

The likelihood of a difficult intubation may result several hours after patient admission due to mucosal and tissue swelling (figure 1).

As a consequence, many patients are intubated early, owing to the fear of increasing airway oedema, before their admission to a major burns unit. This is certainly a safe option.

Box 1. Factors increasing the likelihood of airway intubation

- Absence of airway protection
- Presence of SII or BI
- Stridor
- Face, neck or upper airway injury
- Total surface area involved in burn damage >50–60%
- Ingestion of hot liquid in children

In burns patients with >15% TBSA, alteration of hepatic and renal functions may modify the pharmacokinetics and pharmacodynamics of many drugs. Significantly, a major change in the activity of muscle relaxants occurs, which can be specific to this pathology. Succinylcholine is contraindicated during recovery from a burn trauma because of a possible hyperkalaemic response, directly related to the dose, the post-burn delay and the area of burned body surface. The kalaemic response and the related cardiac complications remain unpredictable in the administration of succinylcholine, which is consequently contraindicated from the fifth day until ≥ 2 years after the injury.

Conversely, the action of nondepolarising muscle relaxants is characterised by a resistance, correlated with both the post-traumatic delay and the extent of the burned area. This starts on about the seventh day after injury, peaks between days 15–40 and can persist for up to 2 years after thermal injury [14].

The use of muscle relaxants should thus be avoided both because of the modified pharmacokinetics and because their use will not prevent a "cannot intubate, cannot ventilate" scenario caused by a possible airway oedema. Direct laryngoscopy with a rigid blade, without the use of paralysis, should be performed. This allows for the mechanical displacement of oedematous tissues and, with oral suctioning, gives the best possible view of the airway. Intubation of conscious patients with very small use of sedatives such as ketamine or opioids allows intubation while maintaining spontaneous ventilation [7].

The American Society of Anesthesiologists guidelines for difficult airway intubation may also be followed [13].

If long-term intubation is anticipated, a nasal endotracheal tube may be preferable for patient comfort, stability, mouth care and the potential ability to communicate by lip movement [7]. A longer endotracheal tube may be needed due to ongoing tissue and mucosal swelling. Although oral intubation may reduce sinusitis, oral endotracheal tubes are uncomfortable for the patient and make oral care difficult. In addition, oral tubes are difficult to secure, especially in patients with facial burns. A nasal endotracheal tube [7] may be more comfortable for the patient and easier to secure.

If intubation is predicted to be, or proves to be, impossible, or when long-term intubation is anticipated, a tracheostomy may be considered. As during airway intubation, a longer tracheal tube may be needed due to progressive tissue swelling.



Figure 1
Swelling may make intubation increasingly difficult.



Figure 2
Securing a tube using a strap.

The role of tracheostomy is still debated [15]. In the authors' institution and others, tracheostomy is the preferred method for patients predicted to need mechanical ventilation for >2–3 weeks [7].

Unplanned extubation in the general intensive care unit setting is hazardous but does not usually cause death or permanent neurological injury. However, in a critical care burns setting in the presence of massive face, neck and airway oedema, reintubation after unplanned extubation can be very difficult, if not impossible. As mentioned above, nasal endotracheal tubes [7] are easier to secure. If tape does not adhere well to the skin because of a facial burn, a strap can be passed through a taped tube (figure 2).

Acute endotracheal tube obstruction may occur at anytime, caused by wads of debris or even casts of small airways lodging in the endotrachea in patients with SII. This problem can be minimised by aggressive pulmonary hygiene including tracheal suctioning and fiberoptic bronchoscopy. Active airway humidification is mandatory in mechanically ventilated burns patients.

Bronchial lavage using small quantities (3–5 mL) of diluted sodium bicarbonate instilled directly into the artificial airway has been used to minimise obstruction. When acute endotracheal tube obstruction occurs, it is managed by prompt recognition and an attempt to clear the tube with a relatively stiff and large suction catheter. The use of an airway tube exchanger is mandatory especially if facial oedema predicts a difficult reintubation.

Clinical management

Clinical management of inhalation injury is supportive only. Prophylactic antibiotics and steroids are of little value.

Airway resistance may increase in patients with inhalation injury as the result of both injury-induced bronchial oedema and bronchospasm. Wheezing occurs as a result of bronchial swelling, bronchospasm and irritant receptor stimulation. In some patients, intense bronchospasm from aerosolised chemical irritant agents may occur during the first 24–48 h. This can be managed with inhaled bronchodilators (β -agonists administered with a spacer) in most patients. Some more severe patients may require intravenous bronchodilators such as low-dose epinephrine infusions, or even parenteral steroids.

In mechanically ventilated patients, bronchodilators are best delivered directly into the airway through the inspiratory limb ventilator circuit *via* nebuliser or metered-dose inhaler with the use of a spacer. Fluid resuscitation must be adequate because both under- and over-resuscitation are detrimental to pulmonary function [2, 7]. Inhalation injury may increase fluid requirements for the initial management of burn shock.

Approximately half of patients with inhalation injury can be expected to develop pulmonary infection, either pneumonia or purulent tracheobronchitis [7]. Pulmonary infection may complicate the clinical course of a patient because of the presence of an artificial airway with superimposed tracheal soiling and bacterial colonisation in a patient with compromised host defences.

Blast injury alone or associated inhalation injury may change the therapeutic approach.

It has been suggested that the onset of response to blast lung injury may be delayed for 24–48 h after exposure. The classical clinical features range from dyspnoea and dry cough to

frothy blood-stained sputum and frank haemoptysis. Exposure to blast may indirectly produce haemodynamic involvement such as bradycardia and hypotension. Treatment changes according to the lesions caused by blast injury.

The early goal-directed therapy approach using fluid, vasoactive agents and blood, directed by monitoring central venous oxygen saturation, might be important in minimising tissue and organ insults. Early surgical treatment should follow the principles of damage control.

Prophylaxis against infection in blast victims is mandatory. When the early phase of treatment has passed, the aim of critical care is to prevent or effectively manage MODS.

Ventilatory strategies

In the presence of an imbalance between ventilatory pump performance and its impedance (elastic and resistive load; increased arterial carbon dioxide tension ($P_a\text{CO}_2$)) and/or a severe ventilation/perfusion mismatching or alteration in diffusion (reduced oxygen), therapy (antibiotics, O_2 supply, *etc.*) is no longer safe and reliable. Therefore ventilatory support must be started.

There is a role for noninvasive positive pressure ventilation (NPPV) in burns patients, because they often need prolonged mechanical ventilation. Mild-to-moderate respiratory distress attributable to primary injury, or after extubation, can be treated by NPPV. A nasal, full-face or helmet mask may be used depending on patient acceptance, facial contour and burn pattern [16].

In major burns, the use of NPPV is conditional on the absence of:

- loss of consciousness
- loss of airway reflexes
- face or neck burns especially in the case of early oedema of these structures in the early phase of major burn management
- fractures to the base of the skull
- traumatic lesions of the facial mass
- recent surgery of the gastroesophagus and/or trachea
- $P_a\text{CO}_2 > 50$ mmHg (6.6 kPa) or $\text{pH} < 7.35$ if the patient is not affected by chronic respiratory or renal insufficiency.

The patient must be assessed frequently by the team to ensure a proper fit and acceptance of the interface, in order to avoid gastric insufflation and skin breakdown.

More critical patients with inhalation injury or ARDS may have decreased lung compliance as well as reduced chest wall compliance; an extensive burn of the trunk may produce a non-compliant chest wall with the need for escharotomies.

Surfactant is also often depleted in patients with SII, leading to alveolar closure. Ensuring adequate patient sedation and ventilator synchrony with adequate sedation and analgesia is mandatory.

The use of positive end-expiratory pressure (PEEP) maintains alveolar patency and increased functional residual capacity [17].

The prevention of alveolar collapse is important because reopening the alveoli requires much higher airway pressure. An open ventilation strategy, which maintains the airway through sufficient levels of PEEP while limiting airway plateau pressure (<32–35 cmH₂O) and avoiding alveolar overdistension, will help prevent ventilation-induced lung injury [7, 18].

As long as there is no contraindication, such as associated head injury, a gradual increase in hypercapnia with mild-to-moderate respiratory acidosis is acceptable to prevent ventilation-induced lung injury [18].

Prone positioning can be also considered. The authors use early prone positioning whenever the patient condition allows, as soon as a lack of airway aeration is detected in the dependent lung region [19].

In patients with severe bronchospasm and increased airway resistance, ventilatory parameters must be set as for a patient with status asthmaticus.

In patients with BI, mechanical ventilation is often complicated by the high risk of barotraumas or pneumothorax. In these patients, many authors recommend prophylactic chest drains. In those patients for whom mechanical ventilation is not sufficient, the use of innovative adjuncts, such as nitric oxide, or extracorporeal support may be considered.

Weaning, extubation and tracheal decannulation

The time spent on ventilation is often increased in the burns patient because of the staged excisions and grafting procedures that cause

weaning delay [7]. Ventilator weaning in general should follow the evidence-based guidelines developed through literature review.

The patient must be awake and alert enough to guard their airway. Upper airway oedema must be resolved to the degree that there is an audible air leak around the endotracheal tube (with cuff deflated) at an inflating pressure of ~20–30 cmH₂O. Steroids may be used in selected patients to reduce airway oedema. A spontaneous breathing trial should always be undertaken before extubation or decannulation.

Patients with mild stridor after extubation may respond to inhaled racemic epinephrine, which reduces airway swelling via vasoconstriction, NPPV [20] or to inhalation of a mixture of helium and oxygen (≥65% helium). Helium, because of its low density, reduces resistance and patient fatigue but does not reduce airway swelling.

Patients requiring prolonged intubation or tracheostomy have a low but important incidence of subglottic stenosis.

After the initial acute phase, the authors ventilate tracheostomised patients leaving leaks around the tracheal tube by reducing the pressure of the cuff of the tracheal cannula.

The whole concept of noninvasiveness is tied not only to the way that the respiratory prosthesis is applied but, above all, to the possibility of keeping glottic functioning intact, thus avoiding the complications associated with conventional upper airway intubation (the presence of a completely cuffed tracheal tube).

Pulmonary function may not return to normal for several months [21].

Conclusions

Airway and respiratory issues remain important sources of morbidity and mortality in burns patients. Respiratory failure is caused as often by sepsis as it is by inhalation injury. More rarely it is caused by BI. While initial care can be carried out by an emergency physician, only experienced teams should undertake the prolonged treatment of these patients. The best outcomes are achieved with optimal care by a well-trained team providing up-to-date, evidence-based care. Of particular importance is balancing the medication needs, airway care and mechanical ventilation needs.

Educational questions

1. Upon which of the following is the definition of smoke inhalation injury based?
 - a) The presence of airway remodelling.
 - b) The occurrence of at least one respiratory infection.
 - c) The amount of inhaled superheated gases.
 - d) The degree of airway hyperresponsiveness.
 - e) None of the above.
2. Which of the following clinical pictures of severe inhalation injury is common?
 - a) Upper airway bleeding.
 - b) Upper and lower airway oedema.
 - c) Hypertension.
 - d) Anaemia.
 - e) Snoring.
3. The likelihood of difficult airway intubation in a patient with inhalation injury is:
 - a) Extremely rare.
 - b) Associated with greater arterial blood gas derangement.
 - c) Always possible when oedema is present.
 - d) Associated with gastric reflux.
 - e) As possible as in other pathologies.
4. Which of the following factors is a known risk factor for inhalation injury?
 - a) A history of loss of consciousness.
 - b) Singed nasal or facial hair.
 - c) A history of presence of superheated gases or chemical irritants.
 - d) A history of exposure to a closed-space fire.
 - e) All of the above.
5. A primary blast injury may be caused by:
 - a) Abnormal airway anatomy.
 - b) Superheated gases.
 - c) Inhaled extra-fine aerosol of chemical agents.
 - d) Immediate pressure variations caused by rapid sequences of compression and decompression.
 - e) All of the above.

Suggested answers

1. c

SII is defined as a clinical picture including a variety of insults that are attributable to the inhalation of superheated gases, steam or gases coming from incomplete combustion of noxious products. It has an incidence of ~20% in patients admitted to major burn centres.

2. b

The clinically important problems of SII include loss of airway patency secondary to mucosal oedema, bronchospasm and intrapulmonary shunting. A period of diffuse exudate formation with bronchiolar oedema and increased capillary permeability follows inflammatory changes.

3. c

The likelihood of a difficult intubation may result several hours after patient admission due to mucosal and tissue swelling. Although fiberoptic intubation may be used to facilitate airway intubation if airway oedema has already developed, this technique may be not helpful.

4. e

Inhalation injury is defined as a clinical picture including a variety of insults and factors.

5. d

BI may produce some specific forms of injury if the chest and lung are exposed to high or prolonged overpressure due to the blast wave.

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