Clinical Aspects of Smoke Inhalation Management

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Inhalational Injury

- In UK more than half of all fire related deaths are either solely attributed to inhalation injury or in combination with burn injury
- Increasing burn size increases your likelihood of inhalational injury

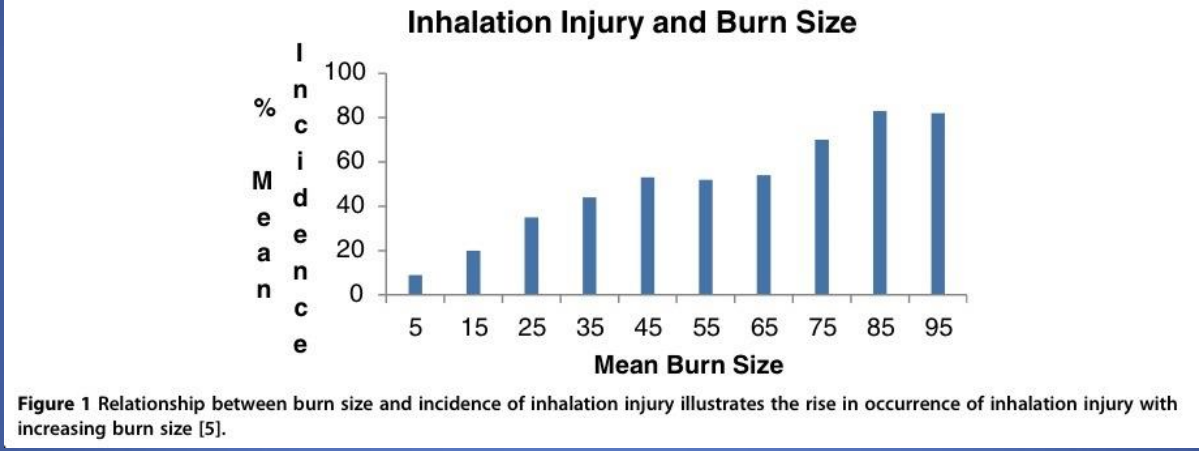
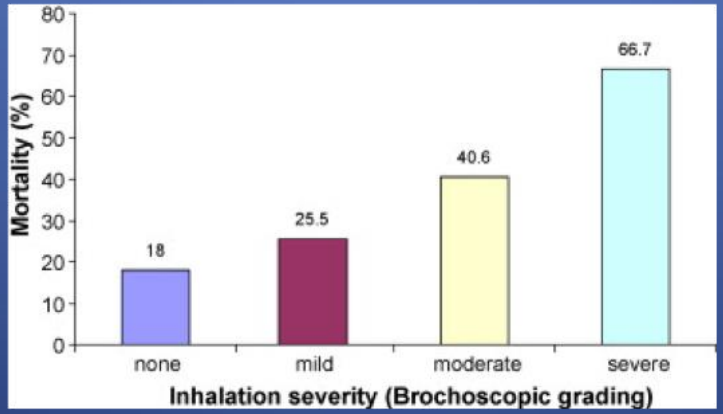


Figure 1 Relationship between burn size and incidence of inhalation injury illustrates the rise in occurrence of inhalation injury with increasing burn size [5].

- Increasing severity of inhalational injury increases your mortality



Inhalational injury in combination with a cutaneous burn

- ↑ fluid resuscitation requirements
- ↑ time on ventilator
- ↑ length of ITU stay
- ↑ incidence of pulmonary complications & longstanding pulmonary dysfunction
- ↑ mortality
  - Revised Baux score (140 generally considered non-survivable)
    - % mortality = age + %TBSA *+ 17 (inhalational injury)*

Smoke

Hundreds of toxic components

Variable depending on what's burning

- Superheated gases & steam
- Particulate matter
- Toxins

Products in smoke	Effect
Carbon dioxide	Increased respiratory drive
Carbon monoxide	Tissue hypoxia, organ failure, death
Hydrogen cyanide	Tissue hypoxia, organ failure, death
Oxygen radicals	Mucus membrane damage, alveolar damage
Acrolein or propenal	Irritant necrosing agent, causing airway mucosa death
Aldehydes, formaldehyde, acetaldehyde, butyraldehyde	Necrosing agent to mucosa, denatures protein
Ammonia	Mucus membrane irritant, including airway muscosa
Sulphur dioxide	Mucus membrane irritant
Hydrogen chloride, Phosgene	Necrosing airway mucosa
Aromatic hydrocarbons, eg, benzene	Mucus membrane irritant, systemic toxin
Hydrogen sulphide	Mucus membrane irritant and corrosive

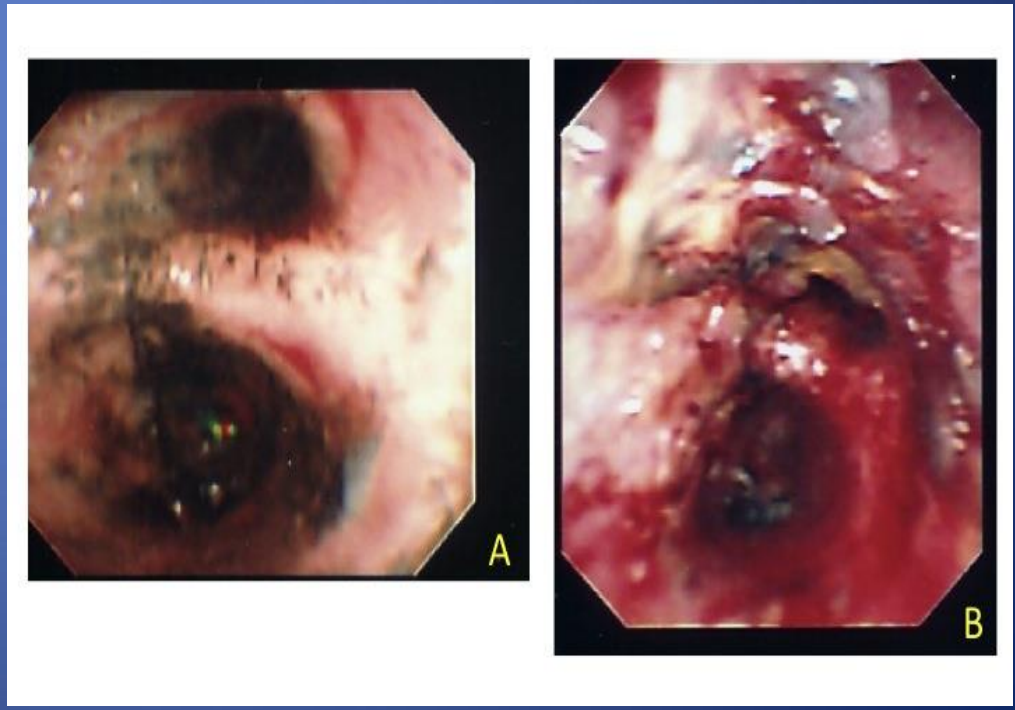
Modes of injury

1. Thermal damage
   - Usually limited to oropharynx
2. Asphyxia
   - Tissue hypoxia from breathing low FiO_2
   - Inhalation of CO +/- cyanide
3. Pulmonary irritation
   - Particulates, inhaled chemicals
   - Inflammatory response (local +/- systemic)

Development of injury

- Hrs to 2-3 days
  - Supraglottic oedema
- Immediate to 48 hrs
  - Intoxication
- Beyond 3- 7 days
  - Atelectasis
  - oedema
  - bronchial casts
  - ARDS +/- pneumonia

- A - Post-burn day 1
- B - Post-burn day 4



1.**Thermal damage**

- Destroys epithelium, denatures proteins, triggers complement/inflammatory response
- Usually supraglottic
  - Airway reflexes are protective
  - Dry air is a poor conductor so heat dissipates in upper airways
- Exceptions (severe and usually fatal):-
  - Steam
  - volatile gases
  - explosive gases (petrol)
  - aspiration of hot liquids

2. Asphyxia

- Occurs due to three main factors
  - Reduced oxygen
  - Carbon monoxide
  - Cyanide
- Combustion rapidly utilises oxygen
- Ambient concentration of oxygen reduced to 10-13% in an enclosed space

- Fire Triangle

Carbon monoxide

- CO is produced by incomplete combustion and causes hypoxia by decreasing the O2 carrying capacity of the blood.

- Hb binds to CO with an affinity 200 times that of its affinity for O2.

CO concentration ppm	Smoke characteristics	Time to 20% COHb
10,000	Heavy smoke	<5 min
5,000	Moderate smoke	<10 min
2,000	Mild smoke	20 min

- Also CO - left shift in the oxyHb dissociation curve. CO in vitro binds to cytochrome oxidase chain.

- As CO binds to haem molecules it affects myocardial myoglobin, reducing myocardial contractility.

Carbon Monoxide

Carbon monoxide %	Symptoms
0-10%	Reduced exercise tolerance in COPD, decreased threshold for angina
10-20%	Headache, dyspnoea – vigorous exertion
20-30%	Throbbing headache, weakness, difficulty with concentration
30-40%	Severe headache, dizziness, nausea, vomiting, visual disturbance
40-50%	Confusion, syncope
50-60%	Collapse, convulsions
60-70%	Coma, frequently fatal

Hydrogen Cyanide

- Increasing use of polymers means the fires are more likely to generate cyanide.
- Cyanide - plastics, polyurethane, wool, silk, nylon, nitriles, rubber and paper.
- Colourless gas with bitter almond odour. Detectable by 40% of the population. 20 times more toxic than CO and can cause immediate respiratory arrest.
- Binds to ferric ion on cytochrome a3 affecting the electron transport chain. Leads to anaerobic respiration and lactic acidosis.
- Methaemoglobinaemia due to heat denaturing Hb – similar effects to CO.

Diagnosis and treatment of cyanide toxicity

- No timely diagnostic test (half life 1hr)
- Suspect if:
  - Reduced level of consciousness
  - Cardiac dysfunction
  - Significant lactic acidosis, low PCO_2
  - Raised venous O_2 (reduced A-V PO_2)
- Treatment
  - Hydroxycobalamin (70mg/kg) – adult dose 5g
  - Cyanokit™

3. Pulmonary irritation

- Direct tissue injury - acute bronchospasm.
  - particle size
  - solubility in water
  - acid-base status
- High water solubility – Upper airway
  - Ammonia, sulphur dioxide, hydrogen fluoride
- Low water solubility – Distal airway
  - Hydrogen chloride, chlorine, phosgene, oxides of nitrogen
- Inflammatory response

  – activated leukocytes and humoral mediators such as prostanoids and leukotrienes produce free radicals and proteolytic enzymes

- Neutrophil activation, inflammatory response
  - release of NOS → NO
  - Loss of hypoxic pulmonary vasoconstriction
  - VQ mismatch
- Bronchoconstriction- 2° neuropeptide release
- Cast formation - epith cells/ fibrin/inflam cells
- Loss of surfactant and ciliary dysfunction
- Airway Obstruction
  - Overdistension, barotrauma
  - Atelectasis elsewhere

History that Increases Suspicion of Inhalational Injury

- Exposure to smoke in an enclosed space
  - e.g. house or caravan fire
  - vehicles

- Loss of consciousness at the scene
  - patients often improve conscious level with removal to fresh air + O_2

Symptoms and signs

(May be delayed for up to 36 hours post exposure)

- Altered conscious level
- Lacrimation, rhinorrhoea,
- Voice changes, cough, wheeze, tachypnoea, dyspnoea.
- Associated cutaneous or mucosal burn injury
- Soot on face, inside nostrils and mouth
- Associated trauma
- Secondary eg cardiac ischaemia

Not very sensitive!

Smoke Inhalation

Assess as per EMSB / ATLS protocols

(Swansea algorithm)

Indications for Admission

- History of enclosed-space exposure for more than 10 minutes
- Carbonaceous sputum
- Arterial pO2 < 8kPa
- COHb > 15%
- Bronchospasm
- Painful swallowing, voice changes
- Central facial burns

Treatment - Patients NOT Requiring Intubation

- General supportive therapy
- Oxygen – high flow humidified
  - Until COHb <5% and O_2 sats maintained >96%
- Chest physiotherapy
- Patients with wheeze or sputum retention
  - nebulised salbutamol 2.5 - 5mg / 4 hourly
- Fluids - Avoid fluid overload – aim for UO of 0.5mls/kg

Indications for intubation

1. Maintain airway
   - impending airway obstruction
   - Stridor, voice changes
   - obtunded consciousness GCS <12, symptoms of CO/CN poisoning
   - Significant burns to the face and neck
2. Facilitate ventilation
   - respiratory distress requiring ventilatory support
3. Shock

Investigations

- ABG and lactate
- COHb
- CXR
  - Not useful diagnostically

- Bronchoscopy
  - Our gold standard

Treatment - Patients Requiring Intubation and Ventilatory Support

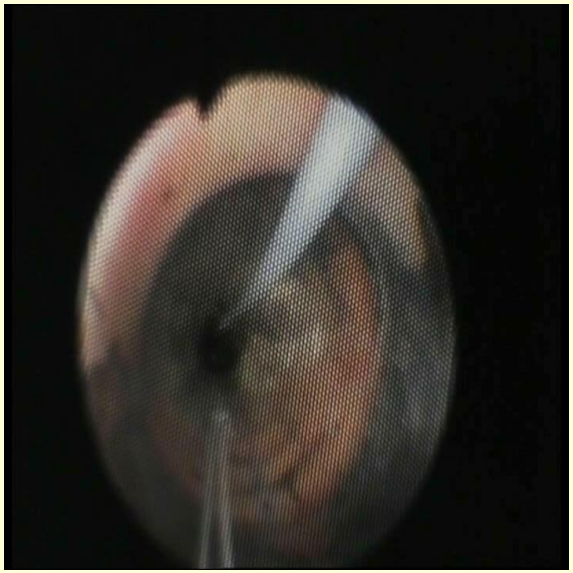
100% O_2 until COHb <5%

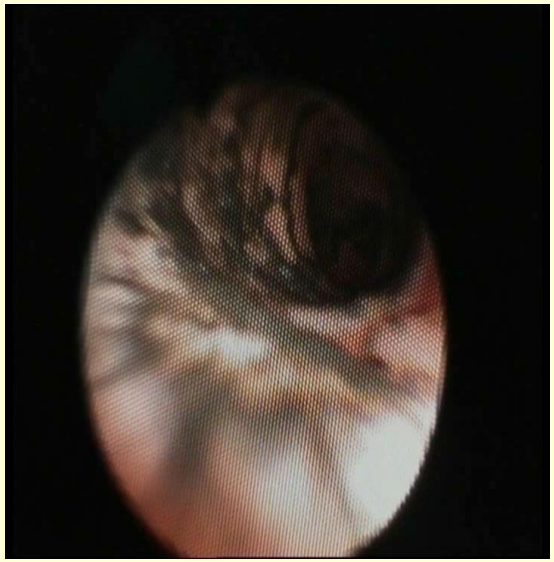
Full warm air humidification

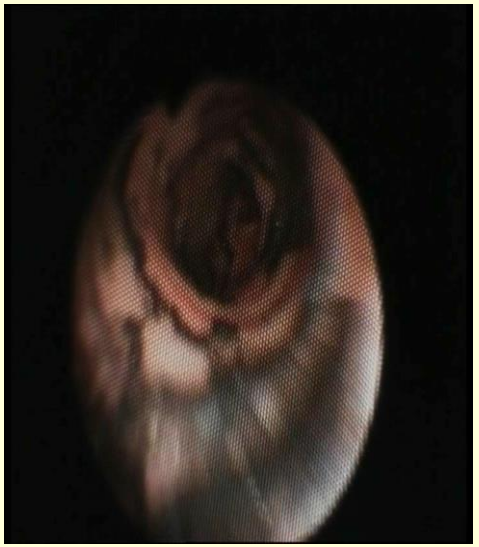
- Lung protective ventilation. PEEP >5 mmHg
- Aim for 'normal' pO_2 and pCO_2 initially
- Assess first sputum sample for evidence of soot or pre-existing infection – send to lab for culture
- Regular physiotherapy

Bronchoscopy

Perform formal fibreoptic bronchoscopy (with video/photographic record if possible) as soon as feasible within 12-24 hours of injury to confirm diagnosis and assess severity



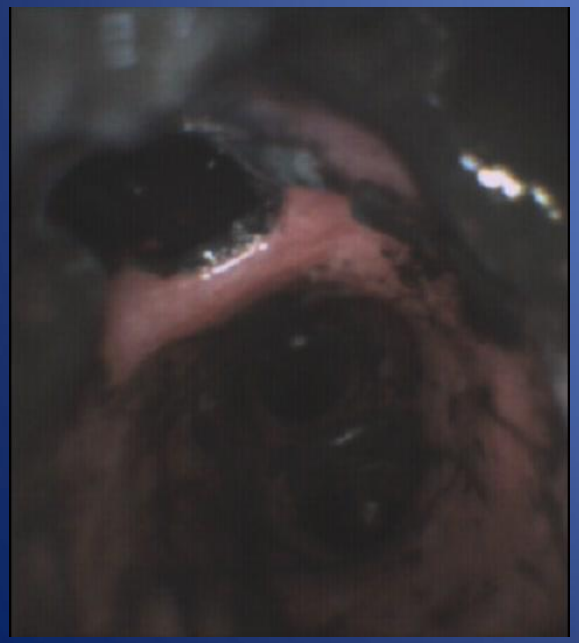


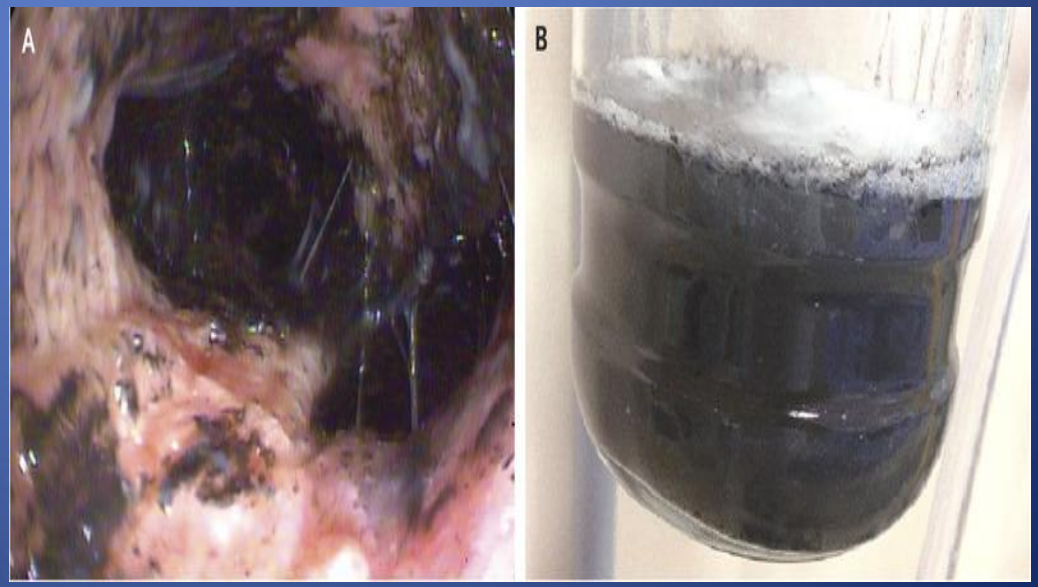


Bronchial lavage

1.26%/1.4% sodium bicarbonate or 0.9% Saline

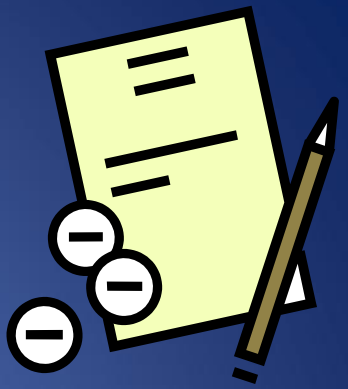
Lavage bronchi with 5-10ml aliquots of dilute $NaHCO_3$ or N Saline solution at bronchoscopy (initially up to 100ml) and administer at hourly intervals (if tolerated) combined with bagging and suction as tolerated. Physiotherapy input if possible.





- Fluids as per burns resus guidelines.
  - aim U/O of 0.5mls/kg only
- Avoid fluid overload and consider early use of vasoconstrictors as appropriate
- Start early enteral feeding via NGT

Drugs – Prescribe as standard



- **Analgesia and sedation** (as per unit protocol)

- **Antipyretics**
  - Regular paracetamol and consider NSAIDs

- **Bronchodilators and inhalational agents**
  - Consider Salbutamol 5mg/ Ipratroprium 4 hourly by nebuliser
  - Consider N-acetyl cysteine 2ml PRN QDS if sputum tenacious
  - Consider nebulised steroids
  - Consider nebulised heparin

Specific Therapies

Antidotes

- If suspected cyanide poisoning not responding to standard supportive therapy consider specific antidotes.
- Hydroxycobalamin (Cyanokit) 70mg/kg up to 5g iv over 15 minutes, Additional doses up to 10g can be given.

Hyperbaric oxygen

- Remains controversial and currently not recommended as standard treatment.

CVVHDF

- If severe persistent metabolic acidosis

Complications of Smoke Inhalation

Early

- Pulmonary oedema (4-9%)
- Atelectasis (1-5%)
- Pneumonia (3-23%)
- ARDS

Later

- Bronchiectasis
- Subglottic stenosis

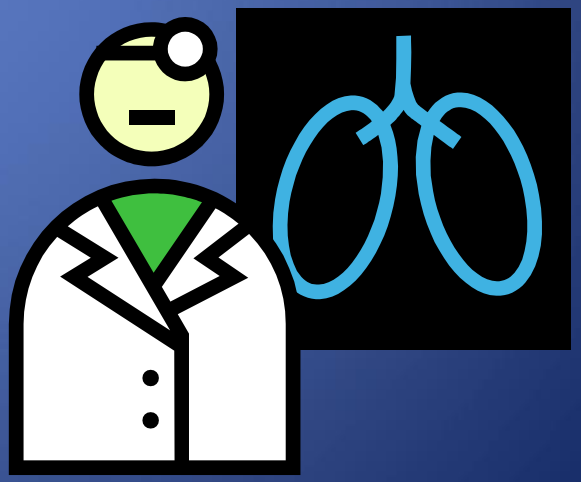
Complications of Smoke Inhalation

Long term

- Restrictive impairment of respiratory function
- Patients more likely to develop reactive airway disease

Follow up

Refer to respiratory physician



Questions and Comments

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