Midland Burn Operational Delivery Network guidance on Inhalation Injury in Burn Injured Patients

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Background:-

Inhalation injury can be defined as the aspiration of superheated gas and/or toxic products of incomplete combustion. The type of injury sustained consists of three distinct phases:-

- Thermal
- Chemical
- Toxic.

Inhalation injury can be classified by the anatomical distribution of the injury, (and the phases mentioned above can be typically mapped to these sites):-

- 1. Airway injury above the larynx (Thermal).
- 2. Airway injury below the larynx (Chemical).
- 3. Systemic effects of inhalation injury (Toxic).

In airway injuries above the larynx, the injury is produced by the inhalation of hot gases leading to the same changes as a thermal injury to the skin (oedema=>swelling). This can then lead to obstruction of the patient's airway and hypoxia. It is most likely to occur if the patient was in an enclosed space, trapped in a fire or with steam inhalation. Early recognition, and intubation with an **uncut** endotracheal tube (ETT), is crucial for management of this type of injury. The use of a cuffed ETT is now advocated in both adults and children. Airway swelling may rapidly occur in the first 12-24 hours following an inhalational injury. The most senior practitioner available should perform the intubation. See appendix 1 for further information regarding specific paediatric airway management.

Airway injuries below the larynx arise from the inhalation of products of combustion. Fire causes the oxidation and reduction of compounds containing carbon, sulphur, phosphorous and nitrogen. The resultant various chemical compounds produced will then dissolve in the water of the respiratory mucous and tissue fluids producing different acids & alkalis which then cause a direct chemical injury to the respiratory mucosa. This leads to swelling and impairment of gas exchange. Often patients will demonstrate an increased work of breathing along with increasing oxygen requirements. Treatment is supportive with oxygen, along with intubation to aid ventilation and gas exchange.

Systemic intoxication occurs following the production and inhalation of various toxic compounds found in smoke (e.g. carbon monoxide, hydrogen cyanide), which acutely produce cellular hypoxia, and can cause lung injury by inflammatory cascades.

History and Examination:-

All burn injuries should be approached with a view to excluding an inhalation injury. Toxic exposure (including cyanide and carbon monoxide) should be assumed in fires occurring in an enclosed space. It is paramount to gain as much history as possible surrounding the events and environment of the burn injury (e.g. ignition source, temperature, concentration and solubility of gases involved, enclosed space and duration of exposure). Entrapment and decreased sensory awareness (e.g. drugs or alcohol) will increase likelihood.

Physical features that may be present can be split into strong (high risk) indicators and other indicators. <u>However</u> there is no single diagnostic indicator.

Strong indicators include:-

- Stridor
- Hoarse voice
- Respiratory distress
- High carboxyhaemoglobin
- Sooty sputum
- Burns inside the mouth
- Circumferential or anterior semi-circumferential neck burns

Singed nasal hair *per se* is a poor indicator, since many patients with flash burns to the face will have this present but no actual inhalation injury.

It should be noted that those patients who have been involved with volatile agents/accelerants in an enclosed space will often be cardiovascularly unstable and require significantly more fluid resuscitation than expected.

- 1. Airway injury above the larynx:
 - Maintain the airway (with/without airway adjuncts e.g. oro-pharyngeal airway).
 - High flow oxygen (ideally 100% at the scene) via a non-rebreathing reservoir oxygen mask; 100% O_2 should be continued until carboxyhaemoglobin confirmed as being <6%.
 - Nurse the patient sitting up (to try and reduce swelling).
 - Frequent re-assessment of the airway.
 - Consider securing the airway early with an endotracheal intubation (using an <u>uncut</u> endotracheal tube to allow for swelling), whilst maintaining cervical spine stabilisation if any suspicion of trauma. This will usually be performed by the most senior available anaesthetist and should be done in a controlled timely manner, rather than in extremis. Subsequent IPPV with 30% head up position.
- 2. Airway injury below the larynx:
 - High flow oxygen (ideally 100% at the scene) via a non-rebreathing reservoir oxygen mask; 100% O_2 should be continued until carboxyhaemoglobin confirmed as being <6%.
 - Consider endotracheal intubation if increasing oxygen requirement or work of breathing (worsening alveolar-arterial difference).
 - Intermittent Positive Pressure Ventilation (IPPV) using a "lung protective strategy" if intubation required (Tidal volume 6-8ml/kg ideal body weight; keep plateau pressure below 30 cmH₂0)
 - All patients who are at risk of a direct thermal airway injury, or who have been involved in an enclosed space fire, should have fibre-optic bronchoscopy performed as either a diagnostic tool or therapeutic manoeuvre; but only once they have been intubated. This can be used to document the presence or absence of inhalation injury below the level of the vocal cords. Where there is evidence of soot or particulate contamination, then broncho-alveolar lavage (BAL) should be used to help remove the contamination. This process should be performed and/or repeated within the first 24 hours until clearance is obtained. The use of aliquots of 1.4% Sodium Bicarbonate <u>may</u> be useful to assist in the clearance.
 - All patients with evidence of airway burn or contamination should be commenced on a regime of nebulised salbutamol and 0.9% NaCl, if bronchospasm observed. Consideration should also be given to the use of nebulised heparin (5000 units every 6 hours), and the use of mucolytics such as nebulised N-acetyl cysteine (1g every 6 hours) or enteral Carbocysteine (750mg every 8 hours)
 - N.B. Likely to have significantly <u>increased fluid requirements</u> for the %TBSA (Total burn surface area) burn present.

- 3. Systemic intoxication:
 - This should follow an ABC supportive approach with high-flow oxygen via a non-rebreathing reservoir mask. If suspecting carbon monoxide (CO) poisoning then do not rely on SaO₂ readings, since they may appear artificial high (carboxyhaemoglobin and oxyhaemoglobin have similar degrees of absorption for the wavelengths of light used in oxygen saturation monitors). Get an early carboxyhaemoglobin (COHb) level to help guide duration of oxygen therapy. There is currently insufficient evidence to support the use of hyperbaric oxygen therapy in carbon monoxide poisoning. PaO₂ readings from arterial blood gases are also unreliable as they do not reflect availability of oxygen intra-cellularly. Where there is a risk of CO poisoning then 100% O₂ should continue until COHb <6%, regardless of SaO₂ or PaO₂. This should also occur, even in cases of associated COPD and MI.
 - If suspecting cyanide (HCN) poisoning, then again a supportive ABC approach with high-flow oxygen should be used, and empirical treatment commenced with an antidote. There is no quick laboratory test currently available for measuring cyanide levels, thus making the diagnosis fraught with difficulty. It is thankfully very rare. Available antidotes are hydroxycobalamin (Cyanokit[®]), sodium thiosulphate or sodium nitrite (Nithiodote[®]). Consider antidote if: clinical history of PVC combustion in an enclosed space with severe lactataemia (lactate >10 mmol/L) despite adequate resuscitation, with concurrent metabolic acidosis and/or reduced gradient between PaO₂ and PvO₂.

References:-

- Emergency Management of Severe Burns (EMSB) Course Manual, Australia and New Zealand Burns Association (ANZBA) & British Burn Association (BBA) 15th edition, 2012. <u>http://www.britishburnassociation.org/emsb</u>
- 2. Mlcak RP, Suman OE, Herndon DN. Respiratory management of inhalation injury *Burns* (2007);**33**:2–13
- 3. Toon MH, Maybauer MO, Greenwood JE, Maybauer DM, Fraser JF. Management of acute smoke inhalation injury *Crit Care Resusc* 2010;**12**: 53–61
- 4. Ipaktchi K, Arbabi S, Advances in burn critical care *Crit Care Med* 2006; **34[Suppl.]**: S239–S244
- 5. Dixon B, Santamaria JD, Campbell DJ. A phase 1 trial of nebulised heparin in acute lung injury *Critical Care* 2008, **12**:R64 (doi:10.1186/cc6894)
- 6. British National Formulary for children August 2014. <u>http://www.bnf.org/bnf/index.htm</u>

Appendix 1

Management of Paediatric Airway (from BCH PICU guidelines):

Resuscitation Airway Interventions

In those with severe airway oedema or symptoms of ensuing airway obstruction at

presentation, CALL on-call ENT surgeon, Senior Anaesthetist

Ensure both difficult intubation and surgical airway kit available (in BCH the latter can be located in ED difficult airway drawer and PICU difficult airway trolley)

Inhalational induction of anaesthesia

Intubate ORALLY first prior to changing to a NASAL tube where possible.

Avoid Suxamethonium if >6 hours post-burn exposure

SITE A CUFFED ET TUBE IF AVAILABLE

DO NOT cut the ET tube as facial swelling may increase over 12-72 hours

ARDS and ventilation difficulties are common and can be better managed with a cuffed ETT

Airway Securing Options

Standard adhesive tapes ('Melbourne Strapping') may be used if facial burns NOT present. Cotton Ties may be used for initial tube stabilisation when facial burns are present, however when securing the airway for medium term ventilation (>6-12h) discuss ET tube fixation with the ENT and Burns team. Fixation methods include:

- 1. Nasal intubation (Preferred)
- 2. Inter-dental wiring
- 3. Fixation to maxilla or mandible
- 4. Fixation to tubing looped around the palate or nasal septum
- 5. Fixation to dental plates (really for very young children or those with no teeth)
- 6. Other clever devices fixed to the teeth (discuss with ENT and Burns surgeon)
- 7. Tracheostomy