Pulmonary Problems in the Burn Patient
(A Leading cause of Morbidity and Mortality in the Burn Patient)

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Pulmonary problems are a major source of morbidity and mortality in the burn patient. To help clarify this process, the burn injury can be divided into the following phases. 1) The Resuscitation Phase, 2) The Early Post Resuscitation Phase and the 3) Inflammation, Infection or Hypermetabolic Phase. The pulmonary problems specific to each phase will be discussed.

I) Resuscitation Phase (0–48 hrs)

Abnormalities of ventilation and oxygenation are a common finding in the immediate post burn period. There are four fairly distinct critical disease processes that must be recognized and aggressively managed. The first three are associated with the inhalation injury complex and are presented in the approximate order in which symptoms will develop. The second process is the decreased chest wall compliance caused by a deep chest wall burn.
Smoke Inhalation Injury Complex

• Smoke inhalation injury complex
  - carbon monoxide, cyanide
  - inhalation injury above the glottis with edema
  - inhalation injury below the glottis with a chemical burn
• Impaired chest wall compliance

Smoke Inhalation Injury Complex

Pulmonary insufficiency caused by the inhalation of heat and smoke is the major cause of mortality in the fire−injured person, accounting for more than 50% of fire−related deaths.1−5 The magnitude of the problem has been much better appreciated in recent years. The use of many new synthetics in home furnishings and clothing have resulted in a much more complex form of injury, due to the extremely toxic combustion products of these advances in technology.1−10 A closed space fire can result in a severe hypoxic insult as well as lung damage from the inhalation of the toxic fumes. The exposure time, the concentration of fumes, the elements release, and the degree of concomitant body burn are critical variables. These factors cause a very complex injury with morbidity and mortality risks, especially when combined with a body burn. Improved knowledge of the pathophysiology combined with an aggressive treatment plan has made it possible to improve the outcome.

The causes in Table 1 are presented in the typical order on which they appear with resuscitation.

Fig. 1

Legend: Smoke exposure in a closed space fire
a) Carbon Monoxide Toxicity

Pathophysiology

Carbon monoxide toxicity is one of the leading causes of death in fires. While oxygen is being used during combustion, carbon monoxide is being released, since it is a basic by–product of combustion. Carbon monoxide is rapidly transported across the alveolar membrane and preferentially binds with the hemoglobin molecule in place of oxygen. In addition, carbon monoxide shifts the hemoglobin–oxygen curve to the left, thereby impairing oxygen unloading at the tissue level. The result is a major impairment in oxygen delivery, since 98% of oxygen is carried to the tissues on hemoglobin. With prolonged exposure, carbon monoxide can also saturate the cell, binding to cytochrome oxidase, thereby further impairing mitochondrial function and adenosine triphosphate (ATP) production.

Symptoms and Diagnosis

The magnitude of the carbon monoxide toxicity roughly corresponds with the peak percent of the circulating hemoglobin bound by carbon monoxide (CO Hgb). It is important to remember that the burn victim is typically being treated with oxygen at the scene and during transport. Therefore, the first CO Hgb obtained may be considerably lower than the peak level which would be that at the time of extrication.

Symptoms of carbon monoxide toxicity are usually not present until carboxyhemoglobin (COHgb) exceeds 15%, i.e., 15% of the hemoglobin is bound to carbon monoxide rather than oxygen. Symptoms are those of decreased tissue oxygenation, with initial manifestations being neurologic due to the impairment in cerebral oxygenation. Major myocardial dysfunction can also develop, with evidence of myocardial ischemia or even infarction, especially with pre–existing coronary artery disease. In addition, the neurologic dysfunction caused by carbon monoxide exposure can lead to a progressive and permanent cerebral dysfunction. Frequently, a patient will awaken transiently after severe inhalation injury only to have progressive neurologic deterioration 24 to 48 hours later. Cyanide toxicity presents in a very similar fashion to carbon monoxide, with severe metabolic acidosis and obtundation in severe cases. Diagnosis, however, is more difficult because cyanide levels are not always readily available or very reliable.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Carbon Monoxide Intoxication</th>
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<tbody>
<tr>
<td>CO Hgb</td>
<td>Symptoms</td>
</tr>
<tr>
<td>0–5</td>
<td>Normal value</td>
</tr>
<tr>
<td>15–20</td>
<td>Headache–confusion</td>
</tr>
<tr>
<td>20–40</td>
<td>Disorientation, fatigue, nausea, visual changes</td>
</tr>
<tr>
<td>40–60</td>
<td>Hallucination, combativeness, coma, shock state</td>
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<tr>
<td>60 or above</td>
<td>Cardiopulmonary arrest</td>
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*CO Hgb – carboxyhemoglobin
Treatment of carbon monoxide toxicity consists of the early displacement of carbon monoxide from hemoglobin by administration of 90% to 100% oxygen. The half-life of carboxyhemoglobin in the patient when breathing 20% oxygen is about 120 to 200 mins, whereas the half-life when breathing 90% to 100% high-flow oxygen is 30 mins (i.e., the concentration of carboxyhemoglobin is reduced by –50% every 30 mins if an oxygen concentration of 90% to 100% is used). Oxygen administration is required for all major burns until carbon monoxide toxicity can be excluded or until carboxyhemoglobin levels return to normal.

Endotracheal intubation and use of 90% to 100% oxygen with mechanical ventilatory assistance is indicated for those patients with severely impaired neurologic function and a high carboxyhemoglobin.

Hyperbaric oxygen (2 to 3 atm) produces an even more rapid displacement and is most useful in cases of prolonged exposure, when carbon monoxide is also present in the mitochondria, since the carbon monoxide is more difficult to displace from the cytochrome system. The drawback of hyperbaric oxygen use is the inability to “get to the patient” during this crucial period of hemodynamic and pulmonary instability. Hyperbaric oxygen is best used in cases in which the patient has severe neurologic compromise with high carboxyhemoglobin (i.e., >50%), but no major burns or severe pulmonary injury and the patient is not responding to high-flow oxygen with clearance of symptoms. However, the vast majority of cases can be successfully managed by simply using 100% oxygen.

### Table 2. Treatment of Carbon Monoxide and Cyanide Toxicity

<table>
<thead>
<tr>
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<th>Carbon Monoxide</th>
<th>Cyanide</th>
</tr>
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<tbody>
<tr>
<td>Awake</td>
<td>High flow by mask oxygen (FiO2 100%) until carboxyhemoglobin &lt; 10%</td>
<td>Intubate 100% oxygen via positive pressure ventilation Hyperbaria used if patient not responding to 100% oxygen (specific indications remain undefined)</td>
</tr>
<tr>
<td>Obtunded</td>
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In general, for cyanide poisoning, cardiopulmonary support is usually sufficient treatment, since the liver, via the enzyme rhodenase, will clear the cyanide from circulation. Sodium nitrite is used (300 mg iv over 5 to 10 mins) in severe cases, especially in those patients in which the diagnosis is made by blood concentrations. Methemoglobin is produced by the nitrite, which, in turn, binds the cyanide. However, methemoglobin does not transport oxygen and a tissue hypoxia can develop, which is similar to the original cyanide effect. Ordinarily, thiosulfate is also given, which, in turn, binds the cyanide to form thiocyanate. One must be reasonably sure of the diagnosis of cyanide toxicity before giving sodium nitrite.

b) Upper Airway Injury from Smoke Exposure
(Obstruction from tissue edema)

Pathophysiology

Direct heat injury caused by the inhalation of air heated to a temperature (150° C or higher) ordinarily results in burns to the face, oropharynx, and upper airway (above the vocal cords). Even superheated air is rapidly cooled before reaching the lower respiratory tract because of the tremendous heat–exchanging efficiency of the oropharynx and nasopharynx. Heat and chemicals in smoke produces an immediate injury to the airway mucosa, resulting in edema, erythema, and ulceration. Although these mucosal changes may be anatomically present shortly after the burn, physiologic alterations will not be present until the edema is sufficient to produce clinical evidence of impaired upper airway patency. This may not occur for 12 to 18 hours. The presence of a body burn magnifies the injury to airways in direct proportion to the size and depth of the skin burn. The massive fluid requirements necessary to treat the skin burn is in part responsible, as are mediators released from the burned skin. Oxidants in smoke and from inflammatory cells are an important cause of injury. Another compounding injury is any face or neck burn that will produce marked anatomic distortion and, in the case of the deep neck burn, external compression on the larynx. A particularly dangerous injury is the third degree facial burn in which minimal external edema is present. The lack of external edema is due to the non–elastic third degree burn, which does not allow expansion. Intraoral edema in this case is usually massive but unrecognized unless looked for. A more superficial burn causes massive external edema but may produce much less mucosal edema and airway compromise. The effect of deep face burns on airway maintenance are presented. The local edema usually resolves in 4 to 5 days.
**Upper Airways (Heat and Chemical)**

- Facial burn
  - rapid edema formation impairing access to oropharynx and disturbing anatomy
- Oral burn
  - rapid swelling of tongue and mucosa impeding airway patency
- Supraglottic edema –
  - progression to obstruction
- Cord and infraglottic edema
  - progression to obstruction

Note marked facial and oropharyngeal distortion caused by the resulting tissue edema.

**Symptoms:**
Symptoms of obstruction, namely, stridor, dyspnea, increased work of breathing, and eventually cyanosis, do not develop until a critical narrowing of the airway is present. Upper airway noise indicative of increased turbulent airflow often precedes obstruction. It is difficult to distinguish noise from a narrowed airway from that caused by increased oral and nasal secretions due to smoke irritation. The airway edema and the external burn edema process have a parallel time course so that by the time symptoms of airway edema develop, external and internal anatomic distortion will be extensive.
Note erythema and edema of supraglottic tissue and cords. Progression of edema can lead to obstruction.

**Diagnosis:**
A history must be obtained regarding the nature of the burn, the presence of smoke and the patients initial neurologic status. Inspection of the oropharynx looking for soot or evidence of a heat or chemical injury should be done with every burn victim. A number of techniques have been used to assess further the degree of injury and to determine the need for early endotrachéal intubation. Direct laryngoscopy is a valuable method to determine whether an injury is present. Typically erythema and edema will be found. Repeat exams will be needed if an injury is present and intubation is not performed, as this process often progresses over the next 24 hours. Fiberoptic bronchoscopy is also very useful and can be done very safely.\(^{22,23}\)

**Treatment**
A very important judgement decision must be made in the initial assessment as to whether the injured airway can be maintained safely without an endotracheal tube. When in doubt or if progressive edema is likely, it is safer to intubate. Three major categories of patients at risk for airway compromise.\(^{20-24}\)

**Heat and smoke injury plus extensive face, neck burns.**
This group invariably requires early intubation.

**Deep facial burns but no smoke injury.**
These patients have difficulty controlling secretions as edema evolves and external edema can compress the airway. Early intubation is a safe approach as anatomical distortion of the face makes intubation at a later period very difficult.

**Heat and smoke injury; no facial burn**
If there is no evidence of severe edema, this group can be carefully observed. The lack of a facial and mouth distortion makes it feasible to intubate later.