Airway Management and Smoke Inhalation Injury in the Burn Patient

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KEYWORDS

• Smoke inhalation injury • Burns • Burns inhalation
• Carbon monoxide poisoning • Hydrogen cyanide
• High-frequency ventilation

Plastic surgeons frequently provide care to patients who have burn injuries and concomitant smoke inhalation injury (II). About 10% of patients admitted to burn centers have II, which greatly increases their risk for postburn pneumonia and mortality, especially at the midranges of age and burn size.[1–3] This article reviews the essential diagnostic and therapeutic interventions in the treatment of these patients.

An understanding of II and what to do about it has only developed over the last 50 years. Consider the scene at Massachusetts General Hospital on the evening of November 28, 1942, following one of the largest indoor fire disasters in U.S. history, at the Cocoanut Grove nightclub. Of the approximately 1000 occupants, 114 were taken to Massachusetts General Hospital within 2 hours, of whom 39 lived to be admitted:

> *As the patients from the scene of the disaster were crowded into the hospital it became apparent early that they were divided sharply into two groups: the living and the dead or near dead. None in the former group died in the first 12 hours; none in the latter group lived more than a few minutes after arrival.*[4]

It is not entirely clear which process—carbon monoxide poisoning, hypoxia, upper-airway obstruction, or a combination—was responsible for these early deaths:

> *The first clue to the high incidence of pulmonary burns was afforded by the number who died within the first few minutes after reaching the hospital. They were cyanotic, comatose, or restless, and had severe upper respiratory damage…some were cherry-red in color, suggesting carbon monoxide inhalation.*[4]

Of those who were admitted, five developed progressive dyspnea and pulmonary edema over the next several hours that required "radical therapy" (ie, endotracheal intubation, immediate tracheostomy, and delivery of oxygen by tent or transtracheal catheter). In the "final stage" of the injury, they developed diffuse bronchiolitis, mucous plugging, peripheral airway obstruction, and lobular collapse. Uncharacteristically, pneumonia was not observed.[4]

Although it is incomplete from a current-day standpoint with respect to answers, the Cocoanut Grove monograph poses many of the same questions that burn specialists, faced with a patient who has severe II, must address today:

- What are the indications for endotracheal intubation?
- What is the ideal timing for tracheostomy?
- What diagnostic procedures should be performed for patients who are suspected of having II?
- Which method of gentle mechanical ventilation should be used for these patients?
- Are there any special fluid resuscitation requirements?
- Which drugs may improve outcome?

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- How should carbon monoxide and hydrogen cyanide (HCN) poisoning in patients who have II be treated?
- Should patients who have II be transferred to a burn center?
- Are there any life-threatening, long-term sequelae of II?

The pathophysiology of II is complex, but it can be classified into three types, based on anatomic location. The first type includes upper-airway injuries caused primarily by thermal injury to the mouth, oropharynx, and larynx. The second type includes lower airway and parenchymal injuries (eg, tracheal, bronchial, and alveolar injuries) caused by chemical and particulate constituents of smoke. Unless otherwise specified, the term ''inhalation injury'' usually means injuries of this type. The third type includes metabolic asphyxiation, which is the process by which certain smoke constituents (most commonly carbon monoxide or HCN) impair oxygen delivery to, or consumption by, the tissues. All three types of II may coexist in a given patient, whose care may be further complicated by cutaneous burns or mechanical trauma.

AIRWAY MANAGEMENT

The indications for endotracheal intubation in patients who have II include decreased mental status resulting from inhalation of metabolic asphyxiants (see the later discussion in this article) or from other injuries, airway obstruction caused by II or generalized postburn edema, and pulmonary failure resulting from subglottic II. Direct thermal injury to the upper airway (including the larynx, oropharynx, mouth, and tongue) causes edema formation, which may progress to complete airway obstruction within minutes or hours. Orotracheal intubation of such patients after the onset of obstruction is often impossible (**Fig. 1**A), and immediate cricothyroidotomy should then be considered. To avoid that scenario, prophylactic intubation is appropriate.

Patients who have postburn facial and airway edema and those who have symptomatic inhalation injury should be recognized as having potentially difficult airways, and a highly experienced provider should perform the intubation. As with any difficult airway, paralytic agents should be used with caution lest they lead to a ''can't intubate, can't ventilate'' scenario. Instead, the use of short-acting drugs such as fentanyl, midazolam, or propofol may be preferable. Premedication for direct laryngoscopic examination should be performed with an appreciation for the fact that many patients who have II are hypovolemic and may become profoundly hypotensive upon induction of anesthesia. Thus, the author frequently uses intravenous ketamine in doses

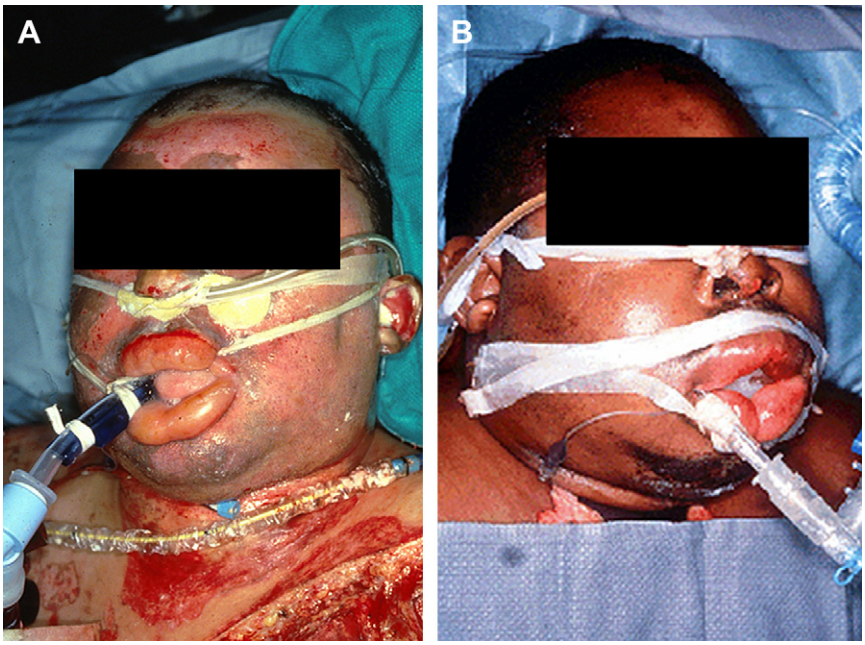

Fig. 1. (*A*) The endotracheal tube must be circumferentially secured around the head and neck of the patient who has significant thermal injury or inhalation injury, using cotton ties or similar methods. Note that care must be taken to protect the corner of the mouth, if possible. (*B*) Adhesive tape will not stick to a burned face.