REVIEW ARTICLE

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Fire-Related Inhalation Injury

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Inhalation injury has been recognized as an important clinical problem among fire victims since the disastrous 1942 Cocoanut Grove nightclub fire.[1] Despite the fact that we have had many years' experience with treating injuries related to fires, the complex physiological process of inhalation injury remains poorly understood, diagnostic criteria remain unclear, specific therapeutic interventions remain ineffective, the individual risk of death remains difficult to quantify, and the long-term implications for survivors remain ill defined. Central to these uncertainties is the complex nature of the injuries, which include a varying combination of thermal injury to the upper airway, bronchial and alveolar mucosal irritation and inflammation from topical chemical exposure, systemic effects of absorbed toxins, loss of ciliated epithelium, accrual of endobronchial debris, secondary systemic inflammatory effects on the lung, and subsequent pulmonary and systemic infection.

Incidence, Prevention, and Implications of Inhalation Injury

Data from the National Inpatient Sample and the National Burn Repository suggest that there are roughly 40,000 inpatient admissions for burns in the United States annually; at a conservative estimate, 2000 of these admissions (5%) involve concomitant inhalation injury.[2] Structural fires are most common in developed environments, especially in impoverished communities. During the past decade, a strong emphasis has been placed on the installation of smoke detectors in residential buildings, which seems to have slightly reduced the incidence of burn and inhalation injury resulting from fires in buildings.

In virtually all epidemiologic studies of burns, inhalation injury is an independent predictor of death, particularly in patients with or more of the body-surface area.[3] In a classic study that described a large clinical experience at the U.S. Army Institute of Surgical Research, the predicted mortality among patients with higher when injury was present than when it was not; if developed, was higher.[4]

Pathophysiological Process

Inhalation injury can result from direct and exposures, to these factors, effects of inhaled , accrual of , and . Structural fires generate that contains a large variety of , products of , and debris of widely varying particle sizes. during fires

varies enormously; typically low at floor level, air temperature can be hundreds of degrees Fahrenheit just a few feet above the floor. The effect on individual patients is complex and unpredictable (Fig. 1).

DIRECT LOCAL INJURY

Direct thermal damage is generally confined to the supraglottic airway, except in rare cases of steam inhalation, such as those that involve the inhalation of pressurized steam in engineering spaces. Most injuries that occur below the glottis are caused by aerosolized chemicals and incomplete products of combustion. The type and severity of these injuries are highly unpredictable, depending on the agents released and the particle sizes inhaled; smaller particles travel to a more distal location in the airway before deposition. The local effects include irritation, mucosal slough, bronchospasm, increased bronchial blood flow, surfactant depletion, and inflammation.

SECONDARY INFLAMMATION

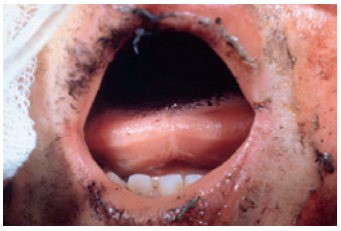
Protean, intense inflammatory responses to inhalation injury may occur, which can generate local reactive oxygen species, attract inflammatory cells, and trigger the release of numerous proinflammatory molecules and cytokines.[5] The local pulmonary effects of the inflammatory responses include bronchospasm and vasospasm, bronchorrhea and alveolar flooding, bronchial exudate and cast formation, and ventilation–perfusion mismatching. The systemic effects lead to a clinically significant increase in the volume of resuscitation fluid required in patients with cutaneous burns who have coincident inhalation injury.[6]

ANOXIA

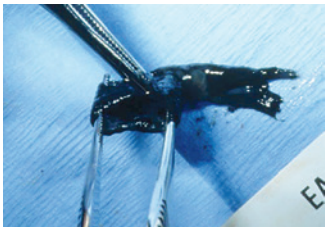
Oxidation of combustibles rapidly consumes available oxygen. Inhalation of oxygen-deficient gas can cause hypoxic brain injury, which is treated like any anoxic brain injury; the neurologic outcome of treatment is variable.

CARBON MONOXIDE EXPOSURE

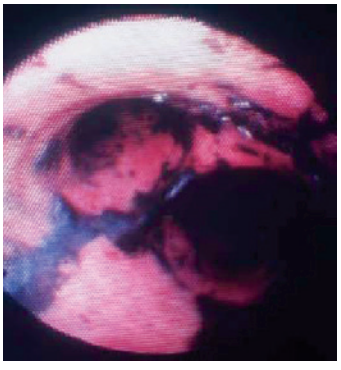
Carbon monoxide, which is released during combustion, is a colorless and odorless gas that is rapidly absorbed after inhalation. Carbon monoxide avidly binds to heme-containing moieties, notably hemoglobin and enzymes of the intramitochondrial cytochrome system; the binding results in reduced oxygen delivery (through the formation of carboxyhemoglobin) and reduced oxygen utilization (through impaired function of the cytochrome cascade). Carboxyhemoglobin levels of 10 to 20% are associated with headache and nausea; levels of 20 to 30%, with muscle weakness and impaired cognition; and levels of 30 to 50%, with cardiac ischemia and unconsciousness. Higher levels are often lethal. Treatment with oxygen during prehospital care may



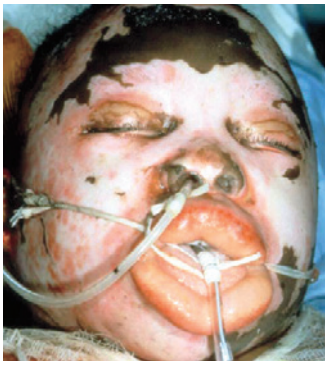
Initial Physical Findings: Direct thermal injury is generally confined to the face and upper airway. Physical findings include facial burns, burned nasal hairs, and soot in the nares and mouth.



Bronchial Cast: Accrued endobronchial debris and exudate can cause obstruction of distal airways contributing to ventilation-perfusion mismatching and secondary infection.



Bronchoscopic View: Aerosolized chemicals and incomplete products of combustion can deposit throughout the subglottic airway and lungs. Severity of injury depends on both the agents and particle sizes inhaled; smaller particles travel more distally. Bronchoscopic findings include mucosal irritation, pallor, ulceration, and carbonaceous debris.



Facial Burn: Anoxia, carbon monoxide effects, cyanide effects, local and systemic inflammation, airway obstruction, and infection contribute to morbidity and mortality in patients with inhalation injury. The effects are more marked in those with large cutaneous burns.

Figure 1. The Pathogenesis of Inhalation Injury.

The physiological process of inhalation injury is complex and involves a variable and often unpredictable degree of direct local thermal and chemical exposure, reactive immune responses, systemic effects of inhaled toxins, accrual of endobronchial debris, and secondary infection. Shown are airway soot observed on physical examination, a positive diagnostic bronchoscopic view, a sloughed endobronchial cast, and a patient with inhalation injury.