Inhalation Injury–An Increasing Problem

JOSEPH A. MOYLAN, M.D.,* CHIN-KEUNG CHAN, M.D.†

Inhalation injury is a common complication of thermal accidents occurring in one-third of patients burned. The routine use of fiberoptic bronchoscopy on all patients incurring thermal burns provides an accurate and safe means for diagnosis. Although complications for inhalation injury are common, the mortality can be reduced by early diagnosis and attention to careful fluid resuscitation, aggressive pulmonary therapy and the avoidance of prophylatic steroids.

INHALATION INJURY IS A MAJOR COMPLICATION of fire accidents. This chemical burn of the airways occurs by inhaling the incomplete products of combustion causing surface damage to the larynx, proximal and distal airways and lung parenchyma. Until recently it was thought that the incidence of respiratory tract damage occured in up to 11% of patients with thermal burns[2,12]. However, these estimates which were based on indirect clinical criteria including flame burns involving the face, singed nasal vibrissae and burns sustained in a closed space have been shown to be inaccurate. With the availability of the fiberoptic bronchoscope, direct means of visualizing and evaluating the airways is now possible. A review of a combined series of burn patients was undertaken to define the current incidence of inhalation injury and analyze the complications and outcome of this patient population.

Methods

The study population includes 100 consecutive seriously burned patients treated by one of the authors (JAM) admitted to the University of Wisconsin Burn Center and the Duke University Burn Service. Baseline data on admission included determination of extent and depth of burn, arterial blood gases, blood chemistry survey, hemogram and chest x-ray. Fiberoptic bronchoscopy was performed in all patients following the instillation of topical anesthetic onto the nasal and pharyngeal mucosa and preoxygenation via mask oxygen. Criteria for the diagnosis of inhalation injury by bronchoscopy included edema, erythema or ulceration of the laryngeal or tracheal mucosa and soot in the tracheobronchial tree[5].

From the Departments of Surgery, Duke University Medical Center, Durham, North Carolina and University of Wisconsin, Madison, Wisconsin

Results

Thirty-three of the 100 patients had bronchoscopic evidence of inhalation injury at the time of admission. The burn size of the patients with airway burns varied from 5% to 95% body surface area, with ⅓ of the patients in this group having thermal injuries less than 40% TBS. The ages ranged from seven to 82 years. Ninety-seven per cent of this group with airway damage had facial burns while only 75% were injured in closed space accidents. Chest x-rays were uniformally normal on admission for patients with and without inhalation injury and over 80% of the group with pulmonary damage had normal initial arterial blood oxygenation (Po_2 greater than 70 mm Hg on room air).

The pulmonary complication rate of the patient's with evidence of airway damage was 73% and included partial or complete airway obstruction due to laryngeal edema necessitating nasotracheal intubation in eight patients, pulmonary edema in five, bronchopneumonia in eight and early rapid progressive pulmonary failure with hypoxia and hypercarbia in three individuals. The overall mortality for this inhalation injury group was

* Associate Professor of Surgery, Department of Surgery, Duke University Medical Center, Durham, North Carolina 27710.

† Surgical Fellow, Department of Surgery, University of Wisconsin, Madison, Wisconsin.

Reprint requests: Joseph A. Moylan, M.D., Box 3043, Duke University Medical Center, Durham, North Carolina 27710.

Submitted for publication: June 8, 1977.

0003-4932-78-0700-0034-0060

33% with eight of the 11 expired patients dying as a direct result of the airway burn.

The pulmonary complication rate for patients with no evidence of inhalation injury was 6% and included hematogenous pneumonia following septicemia in two, congestive heart failure in one and a single case of aspiration pneumonia. No patient had upper airway obstruction in this group. The mortality rate was 8% for patient who did not have airway damage.

Twenty-six of the 33 with objective bronchoscopic evidence of inhalation injury were entered into a double blind study evaluating steroids in the treatment of airway burns. Fourteen received steroids and 12 did not under this protocol. Of the remaining seven patients with airway damage, three received steroids by physician's choice. Steroid therapy consisted of methylprednisolone sodium succinate 30 mg/kg as an initial IV bolus, then 30 mg/kg/day in four equal doses per day for 48 hours. The average burn size of the steroid treated airway burn group was 55% body surface area (range 5–95) and the nonsteroid treated inhalation injury group averaged 47% (range 9–80). The mortality rate for the steroid group was 53% (9/17) while the mortality rate for those with inhalation injuries who were not treated with steroids was 13% (2/16). The steroid treated group had almost three times the incidence of infectious complications, as either pneumonia or bacteremias than did those airway injured patients not receiving methylprednisolone (14 versus 5).

Discussion

Inhalation injury remains one the most serious consequences following thermal accidents. While various reports indicate that airway damage occurs in only up to 11% of patients with burns, our experience with direct airway examination shows a three-fold increase in the incidence of inhalation injury. This marked increase represents both better diagnostic capabilities as well as an increase in the actual incidence of the thermal complication. In fact, the so-called "early airborne pneumonia" of burn injury[10] occurring in the third to fifth day postinjury may have been subclinical airway injuries with infection developing in the early resuscitative period. In addition there may be a real increase in inhalation injuries because of more synthetic materials in the environment. Combustion of polyvinyls and other plastic materials produce gases which are extremely noxious to the airway and lung parenchyma[3,9].

As CO poisoning is frequently associated with inhalation injury, the performance of arterial blood gases to determine the presence of CO is important. Low grade CO poisoning (<15%), was not an unusual finding in this group[4]. While these levels are not particularly dangerous, undiagnosed higher levels may be lethal when associated with actual airway damage. In addition, special attention in preoxygenating patients prior to bronchoscopy should be given to patients with low arterial oxygen saturations.

Direct visualization of the larynx, trachea and bronchus can be accomplished without any morbidity provided that a few principles are observed. Adequate and complete topical anesthesia must be carried out to prevent gagging and possible vomiting. The use of aerosolized carbocaine provides good topical anesthesia for the nasal and pharyngeal mucosa. Our approach is to inspect the upper airway using the fibroptic bronchoscope transnasally, advancing the scope to the level of the epiglottis where the true and false cords, the entire larynx and proximal trachea can be visualized. If any of the criteria for injury are present at this level, 5 cc of lidocaine is injected through the scope directly into the trachea and the scope is advanced through the cords to define the distal extent of the injury in the major airway.

The diagnosis of inhalation injury using the bronchoscopic criteria of tracheobronchial mucosal edema, erythema, ulceration or soot in the airway has proven extremely accurate[1]. Pulmonary complication in patients whose initial bronchoscopy was negative were rare and could be ascribed to causes other than primary airway injury. The value of early diagnosis is important as these patients are at a significantly higher risk for pulmonary complications. They require careful observation as early endotrachial intubation may be necessary as laryngeal obstruction develops. Vigorous pulmonary toilet should be started at the time of admission to clear secretions. Limited and carefully controlled fluid administration is mandatory since minimal over-resuscitation is poorly tolerated.

Inhalation injury can be anatomatically divided into three levels: 1) upper airway injury (burn damage limited to the larynx and vocal cords); 2) major airway injury (burn damage involving the tracheobronchial tree) and 3) parenchymal injury (burn damage involving the terminal bronchi and alveolar space) (Fig. 1). It is important to identify the various levels of injury as the complications are different, especially in those individuals whose airway burn is limited to the upper airway. Using the bronchoscope, upper and major airway burns, which account for 90% of the patients with airway injury, can be diagnosed. Xe^{133} lung scanning is necessary for the early identification of patients with parenchymal burns[7].

Clinically there is a staged progression of the inhalation injury porportional to the extent and severity of the chemical burn. Respiratory distress and insufficiency may develop during the first few hours in patients with the most severe pulmonary injury involving the proximal

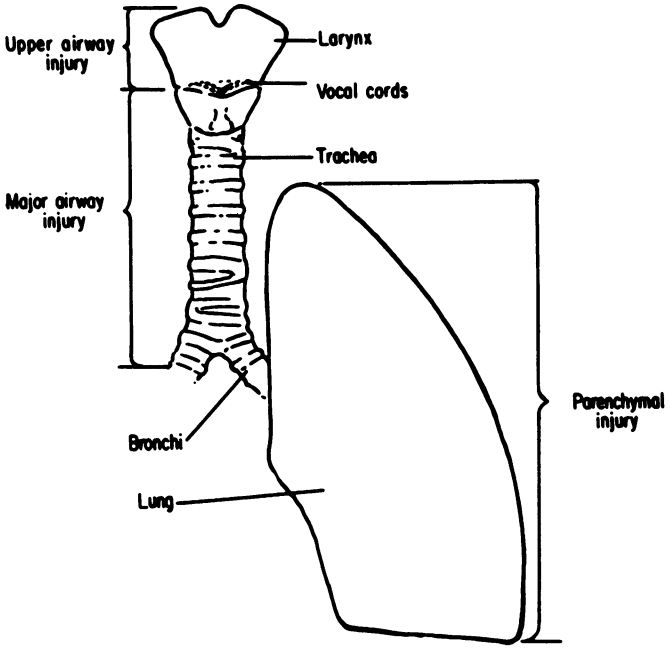

FIG. 1. Classification of inhalation injuries.

and distal airways. This rapid progression results from bronchospasm and/or extensive alveolar damage and is almost universally lethal. The second phase occurring 8–36 hours postinjury is the development of edema resulting in upper airway obstruction or pulmonary edema. It usually becomes clinically evident in those patients with limited cardiopulmonary reserve and in those with over-vigorous fluid replacement. The last stage of inhalation injury, occurring after the third day, is bacterial pneumonia, usually bronchial in nature and associated with profuse bronchorrhea.

The pulmonary complications in this series included acute upper airway obstruction which results from progressive edema primarily of the false cords. This problem becomes manifested during the first 24–36 hours post injury. Attention to the clinical signs of hoarseness and stridor provides indications of impending airway occlusion. Repeat fiberoptic bronchoscopy will confirm developing airway edema. The use of nasotracheal tube rather than a tracheostomy is an important adjunct in the treatment of upper airway obstruction. The airway will require splinting until the edema resides which usually occurs by the fourth day postinjury. The use of a size eight or nine nasotracheal tube will facilitate suctioning in those patients with bronchorrhea. Since the complications of tracheostomy are extremely frequent and serious in this group of patients even when there are no head and neck burns[6], every effort should be made to avoid this procedure.

With parenchynal as well as major airway damage, pulmonary edema may occur. It is essential to minimize the positive fluid balance necessary to reverse burn shock to prevent pulmonary edema. Excessive fluid administration as evidenced by hourly urine outputs greater than 75 cc may result in increased tissue edema in both the airway and lung parenchyma and should be avoided. Some have suggested the use of central venous or Swan-Ganz catheter to evaluate fluid replacement[8] however these techniques increase the risk of infection and may be misleading due to increased capillary permeability.

Progressive rapid pulmonary insufficiency without frank edema occurred in three patients with inhalation injuries. These patients deteriorated with severe hypoxia and hypercarbia in spite of intubation and volume ventilatory support. While the mechanism is not clear, it probably represents direct capillary-alveolar membrane damage.

Bronchopneumonia occurs commonly with major airway damage because of the loss of protective ciliary action. These patients clear their secretions poorly and have reduced systemic and local immune capabilities. While formerly prophylactic antibiotics have been recommended for this burn complication, they have resulted in the development of pneumonia secondary to resistent bacteria[11] and should not be employed. Daily sputum cultures should be obtained to isolate bacteria early, so that proper antibiotics can be administered if pneumonia develops. The most common infecting organisms are *Staphylococcus aureus* coagulase positive and gram negative bacteria such as Klebsiella Aerobacter[2]. If ventilatory support is necessary, the use of a volume respirator delivering high tidal volumes (15 cc/kg) is recommended. The addition of positive end expiratory pressure (PEEP) can be valuable since respiratory mechanics performed in patients with inhalation injuries demonstrate decreased lung compliance and increase airway resistance.

Vigorous pulmonary physical therapy with coughing and deep breathing exercises is an important aspect of the early care. While these patients frequently have bronchorrhea, the secretions are viscous and inspissated, causing small airway plugs, atelectasis, and predispose to superinfection, making airway humidification important. As the patient begins to expectorate debri and plugs, nasotracheal suction and repeat bronchoscopy may be necessary if routine measures are not satisfactory in maintaining open bronchi. Bronchospasm and wheezing can be treated with systemic or nebulized bronchodilators such as aminophylline.

Of the 33 patients with documented inhalation in-

juries, 17 individuals received systemic steroids for 48 hours and 16 did not. The steroid treatment was associated with an increased mortality rate (53% versus 12%) and significantly more infectious complications such as pneumonia and bacteremia. Since no therapeutic benefit could be demonstrated and a higher mortality and morbidity occurred in the steroid treated group, prophylactic steroids should not be employed.

References

1. Agee, R. N., Long, J. M., Hunt, J. L., et al.: Use of Xenon^{133} in Early Diagnosis of Inhalation Injury. J. Trauma, 16:218, 1976.
2. DiVincenti, F. C., Pruitt, B. A. and Reckler, J. M.: Inhalation Injuries. J. Trauma, 22:109, 1971.
3. Dyer, R. F. and Esch, V. H.: Polyvinyl Chloride Toxicity in Fires. JAMA, 235:393, 1976.
4. Larkin, J. M., Brahos, G. J. and Moylan, J. A.: Treatment of Carbon Monoxide Poisoning: Prognostic Factors. J. Trauma, 16:111, 1976.
5. Moylan, J. A., Abib, K. and Birnbaum, M.: Fibertoptic Bronchoscopy Following Thermal Injury. Surg. Gynecol. Obstet., 140:541, 1975.
6. Moylan, J. A., West, J. T., Nash, G., et al.: Tracheostomy in Thermally Injured Patients, a Review of 5 Years' Experience. Am. Surg., 38:119, 1972.
7. Moylan, J. A., Wilmore, D. W., Mouton, D. E. and Pruitt, B. A., Jr.: Early Diagnosis of Inhalation Injury using 133 Xenon Lung Scan. Ann. Surg., 176:477, 1972.
8. Noe, J. M. and Constable, J. D.: A New Approach to Pulmonary Burns: A Preliminary Report. J. Trauma, 13:1015, 1973.
9. Petajan, J. H., Voorhees, K. J., and Packham, S. C.: Extreme Toxicity from Combustion Products of a Fire Retarded Polyurethane Foam. Science, 187:742, 1975.
10. Pruitt, B. A., Jr., Flemma, R. J., DiVencenti, F. C., et al.: Pulmonary Complications in Burn Patients. J. Thorac. Cardiovasc. Surg., 59:7, 1970.
11. Shook, C. D., MacMillan, B. G. and Altemeier, W. A.: Pulmonary Complications of the Burn Patient. Arch. Surg., 97:215, 1968.
12. Stone, H. H., Martin, J. D. and Claydon, C. T.: Management of the Pulmonary Burn. Am. Surg., 33:616, 1967.