Mass Ammonia Inhalation*

Terrence J. Montague, M.D.,** and Arthur R. Macneil, M.D.†

The clinical, roentgenographic and laboratory findings, their relationship to each other and to the subsequent hospital course are reported for 14 victims of gaseous ammonia inhalation. Initial physical examination en-
abled differentiation of a mildly affected from a moder-
ately affected group, but patients in both groups re-
sponded well to conservative medical management.

Ammonia is an extremely noxious gas when in-
haled and exposure to it, usually in the setting of an industrial accident, has long been recognized as a cause of both acute^1^4 and chronic^5^6 respiratory disease. None of the previous case reports, however, provide data which allow correlation of the clinical, roentgenographic, and blood gas findings with the subsequent course. Recently, we have managed 14 patients accidentally exposed to large concentrations of gaseous ammonia. The purpose of this report is to provide a correlative summary of their presentation and hospital course. In particular, analysis of the data reveals that physical examination of the chest in the first 24 hours postexposure allows the best prediction of subsequent hospital course.

CASE REPORTS

While in offshore Canadian waters, 14 foreign male fishermen, ages 18 to 39 years, all well in the immediate past, were suddenly exposed to high concentrations of gaseous ammonia as a result of a leak in their ship’s refrigeration system. The leak occurred while the victims were sleeping, and the exact duration of exposure is uncertain, although it was estimated to range from several seconds to several minutes, some crewmen being exposed longer than others. All of the victims were removed to open deck areas as quickly as possible but no specific medical care was available until they reached the emergency room of our hospital 14 hours postexposure.

The patients’ symptoms on admission varied. All of the patients had developed significant respiratory distress from the time of awakening, and on admission, all continued to complain of some combination of pharyngeal (12) or pleuritic chest pain (9), cough (9) or dyspnea (8). In addition to the respiratory symptoms, six patients had ophthalmologic symptoms, three with photophobia, and one patient had aphonia.

The important physical findings at the time of admission included the following: inflammation of the pharynx (14) and conjunctiva (12) were almost universally present, and corneal burns were evident in two patients. Five patients had a normal chest examination and were judged to have mild respiratory tract involvement (group 1). Tachypnea was present in ten patients. Nine patients had abnormal chest findings manifested as rales, rhonchi, or wheezing and were judged to have moderate involvement of their respiratory tract (group 2). Three of these group 2 patients were smokers and three others had past histories of “asthma” in childhood although none had had clinical attacks for at least ten years prior to admission. Tachycardia (sinus) was present in four patients.

Arterial blood gas values were determined in all patients at the time of admission with the patients breathing room air (Fig 1). The PaO2 values of group I patients ranged from 81 to 91 mm Hg with a mean value of 88. The PaO2 values of group 2 ranged from 45 to 84 mm Hg with a mean value of 64. The PaCO2 values on admission covered a more narrow range from 28 to 39 mm Hg with a mean of 33. Other abnormal laboratory findings included elevated body temperature in two patients (37.5° and 37.7° C) and WBC counts greater than 10,000 cu mm in seven patients (range 11,000 to 17,000/cu mm).

One patient, with rales and rhonchi on chest examination and a PaO2 of 45 mm Hg, demonstrated a small area of opacification in the left lower lobe on his admission chest x-ray film. By 48 hours postadmission, the opacified area had improved to a normal appearance. All of the remaining patients had normal chest x-ray films on admission, regardless of the presence of abnormal physical findings or low PaO2 values.

All patients received single doses of intravenous hydrocortisone sodium succinate (0.5 to 1 g) and nebulized dexamethasone (8 mg), and each received supplemental oxygen therapy in the examining room. They were subsequently transferred to a medical intensive care area for continuing assessment and therapy. Group 1 patients received no further specific therapy, and all became asymptomatic and fit for discharge by the second hospital day. The patients in group 2, also improved during their hospitalization, although more slowly with the mean duration of hospitalization for these nine patients being 6.3 days. Two of these nine patients had significant complications following admission. One patient required a tracheostomy four hours postadmission to relieve stridor secondary to laryngeal edema. Another had persistent airway obstruction and increasing alveolar-arterial P(A-a)O2 gradient for the first 48 hours following admission. In addition, all group 2 patients had a decremental productive cough throughout their hospital course.

Five patients with signs of airway obstruction were treated with intravenous aminophylline and nebulized salbutamol for short periods early in their hospital course; two were still orally receiving theophylline preparations at discharge. Two

*From the Department of Medicine, Victoria General Hospital and Dalhousie University, Halifax, Nova Scotia, Canada.

**Assistant Professor, Division of Cardiology.
†Assistant Professor, Division of Respirology.
Manuscript received April 16; revision accepted June 13.
Reprint requests: Dr. Montague, 304 Pavilion, Victoria General Hospital, Halifax, N.S., Canada B3H2XG
patients with fever and productive cough were started on regimens of orally administered penicillin therapy. Sputum cultures from one of these patients grew *Hemophilus influenzae* and *Staphylococcus pyogenes*. Sputum cultures from four other group 2 patients failed to grow any known pathogens.

Long-term follow-up was not possible for any patient in either group. Only one patient (group 1) had pulmonary function testing. The study was performed three days following exposure when the patient was asymptomatic and revealed low normal lung volumes and high normal flow rates. However, comparison of the admission P(A-a)O₂ with determinations at a later point in the hospital course was possible for seven of the group II patients, and the data reveal a trend towards normalization in four of these seven patients (Fig 2).

**DISCUSSION**

Ammonia is an extremely irritating gas on inhalation, capable of producing severe damage to all levels of the respiratory tract, with resultant clinical impairment of respiratory function ranging from mild to fatal.¹⁶ The acute pathologic lesions in fatal cases of ammonia inhalation are laryngotracheitis, pulmonary edema and hemorrhage; and bronchopneumonia;⁴,⁶ and chronic lesions are “those of bronchiectasis and fibrous obliteration of small airways.”⁴ The largest series of cases previously reported was that of Caplin.² He traced the course of 47 patients who inhaled ammonia gas as the result of a bomb explosion in a London brewery cellar. The spectrum of initial respiratory tract involvement in his cases ranged from mild to severe and was directly correlated with the subsequent clinical course. Seven of the 41 patients clinically judged to have severe respiratory involvement eventually died from respiratory complications, either “pulmonary edema” or “bronchopneumonia.” Six of 27 patients with moderate respiratory involvement died with similar complications, but there were no deaths in the mildly affected group of nine patients.

Our patients fell into two clinical groups, roughly comparable to the mildly and moderately affected patients of Caplin’s series.² Group 1 patients all had respiratory tract symptoms but normal chest examination at admission and all recovered very quickly without specific therapy. All of group 2 patients also had respiratory tract complaints at admission, but in addition, all had significant abnormal findings on chest examination. Their subsequent course, although one of gradual improvement, was more prolonged than group 1 patients, and two patients in this more severely affected group had frank clinical deterioration early in their course.

Despite the moderate respiratory tract involvement, clinically, in some of our patients, there was a

---

**Figure 1.** Arterial Po₂ values on admission for both clinical groups.

**Figure 2.** Comparative P(A-a)O₂ gradients for seven patients in group 2. “A” refers to time of admission.
remarkable paucity of chest x-ray film abnormalities. This discrepancy between apparently extensive clinical involvement and normal roentgenographic appearances has been observed before in patients with serious ammonia inhalation. Roentgenographic findings, therefore, appear to offer little help in assessing the immediate status of respiratory tract involvement or in predicting the future clinical course of patients suffering acute ammonia inhalation, at least in patients with mild to moderate respiratory tract injury.

Determination of arterial Po2 values at admission as a measure of gas transfer did show a correlation with the clinical assessment of severity of respiratory tract involvement. Patients with normal chest findings had a higher mean arterial Po2 value, 88 mm Hg, compared to a mean value of 64 mm Hg for the patients with abnormal chest findings. However, arterial Po2 values did not allow complete separation of the patient groups according to their clinical status or future course, as some slight overlap of values between groups was found (Fig 1).

The factor that best allowed prediction of the future course was the presence or absence of abnormal chest findings at admission. Patients with normal examination findings lost their respiratory symptoms and were able to be discharged rapidly within 24 hours. Patients with abnormal examination findings had a more protracted and relatively more complicated course in hospital. This predictive value of early chest findings has important implications for mass casualty situations, such as the one we encountered, since knowledge of the expected clinical course will allow for more efficient staff planning.

Most importantly, perhaps, our experience with these two groups of patients representing a spectrum of mild to moderate respiratory functional impairment demonstrated that all patients improved their clinical state fairly rapidly with conservative medical management. At least until more information becomes available, we feel it is appropriate to manage similarly affected patients in a similar fashion. Patients suffering more extensive inhalation injury than our patients, resulting in more extensive, ie, severe respiratory functional impairment, may require and benefit from additional aggressive therapy. In the past, however, their outlook has been poor. It remains to be determined whether any clinical or other criteria will allow the early differentiation of such severely affected patients from those with less severe impairment of their respiratory function.

REFERENCES
1 Slot GMJ. Ammonia gas burns. Lancet 1938; 2:1356-1357
2 Caplin M. Ammonia gas poisoning. Lancet 1941; 2:95-96
4 Sobonya R. Fatal anhydrous ammonia inhalation. Human Path 1977; 8:293-299