by the physician, and ordered to stop smoking by the physician. Using a standard approach to evaluation through physiologic and psychologic evaluation, the therapist is able to assist the physician in developing a data base. Intervention and education is individualized and personalized for each smoker. The physically addicted smoker will benefit the most from pharmacologic therapy. It is clear that pharmacologic support alone is not sufficient for most smokers. A comprehensive, behavioral counseling program to prevent relapse must be used to supplement the medication.

The American Association for Respiratory Care is developing additional materials to help hospitals set up intervention programs. Soon to be released is the Nicotine Intervention Kit (NIK). The NIK will include a 10-min video tape of how to organize and offer a bedside intervention program. The business and marketing plan will allow the respiratory care director to fill in the blanks to project workload and costs to comply with the needs of the hospital administrator. The kit also includes camera-ready art for posters to announce the new service. The flyers and brochures for direct marketing to physicians and patients can be reproduced at the local hospital. This kit is available from the AARC at 11030 Ables Lane, Dallas 75229.

Nicotine addiction is a complex problem which will require a comprehensive approach. There are still 50 million smokers in the United States. Many of these smokers are admitted to our hospitals every day. Without a formalized, agreed upon protocol to treat these smokers, we are putting our heads in the sand. We must be as enthusiastic about the treatment of addicted smokers as we are enthusiastic about treating the diseases that result from smoking. The medical disease of nicotine addiction requires a medical approach that: (1) includes all members of the health team; (2) that uses physiologic evaluation with exhaled carbon monoxide and pulmonary function tests, and, in some patients, screening for lung cancer; (3) pharmacologic therapy; and (4) patient education based on counseling skills for relapse prevention. When we have such an organized, systematic program in place to assist smokers, we can, perhaps, say we did our best. On the other hand, perhaps we will take the attitude we do in every other disease, and say, "What else do we need to learn so we can truly assist the patient in smoking cessation?" We never give up on the diabetic patient who is unable to follow dietary advice, or the hypertensive patient who continues to use salt, or the cardiac patient who fails to use his medication regularly. We continue to work with them, to help them. We need to do the same with the addicted smoker.

Louise M. Nett, R.N., R.R.T.
Denver

Clinical Research Division, Presbyterian/St. Luke's Center for Health Sciences Education.
Reprint requests: Ms Nett, Presbyterian/St. Luke's Center for Health Science Education, 1719 East 19th Avenue, Denver 80218

Chemical Warfare and Bronchoscopy

Notwithstanding the 1925 Geneva Protocol, which bans the use of chemical and biological agents in warfare, the recently terminated conflagration in the Middle East was fraught with the specter of victims of these deadly agents.

Chemical and biological agents can be broadly categorized into six groups: incapacitating agents (eg, tear gas, adamsite), blister agents (eg, mustard gas, lewisite), choking agents (eg, phosgene, chlorine, chloropicrin), blood agents (eg, hydrogen cyanide), nerve gases (eg, tabun, sarin, soman, VX, organophosphates), and toxins (eg, anthrax, botulin, ricin, mycotoxins). Even though many of these are contact poisons and cause serious lesions of the exposed skin and mucosa, pulmonary involvement is the most common cause of death. Nerve gases cause death by respiratory arrest.

The recent article by Freitag and associates, which appeared in the November 1991 issue of Chest, describes their experience with the subacute and chronic complications of mustard gas inhalation during the Iran-Iraq war in the 1980s. As noted by the authors, the details of acute response of the respiratory system to inhalation of mustard gas and the initial treatment were difficult to come by, and because of the difficulties involved in follow-up of these soldiers, a truly scientific study was well-nigh impossible. Complicating this is the possibility that the victims may have been exposed to multiple chemical and biological agents in addition to the conventional weapon-related chemicals and gases. Nevertheless, the clinical experience described provides needed insight into the pulmonary complications of mustard gas inhalation.

Nitrogen mustard gas (bis-2-chloroethylsulfide) is an oily, colorless or pale yellow liquid, which is freely soluble in organic solvents, easily vaporized by heat, and quickly spread by the wind. It smells like garlic. Its vesicant action produces blisters of exposed skin and mucous membranes.

The respiratory complications are proportional to the quantity of mustard gas inhaled; these can be divided into acute, subacute, and chronic phases. In the acute stage, upper airway obstruction from mucosal edema and respiratory failure from pulmonary edema and hemorrhage are the main features. Contributing factors include physical trauma to the chest from projectiles, thermal injury to the tracheobron-
chial mucosa, soot deposition in the airways, and carbon monoxide poisoning. The subacute stage, which may last from hours to several days, is manifested by necrosis of the tracheobronchial mucosa, hemorrhagic tracheobronchitis, persistent pulmonary edema and hemorrhage, and secondary infection. Weeks to months later, the chronic stage results, with development of scarring and stenoses of the tracheobronchial tree, bronchiectasis, formation of granulation tissue, recurrent infections, bronchiolitis obliterans, and progressive respiratory insufficiency. The increased incidence of lung cancer as a complication of mustard gas injury is well established.10

Initial treatment of victims exposed to these chemicals should include immediate removal of the victim from the contaminated location, stabilization of general and hemodynamic status, maintenance of the airway, oxygenation, and other appropriate supportive therapeutic measures. The role of systemic corticosteroid therapy as prophylaxis against subacute and chronic pulmonary complications is debatable.

Freitag and associates6 have demonstrated the invaluable role of bronchoscopy in the diagnosis and treatment of this special group of patients. In the acute stage, bronchoscopy should help in assessing the extent of damage to the upper airway mucosa and in keeping the tracheobronchial tree patent by facilitating removal of the charred and necrotic mucosa. It is unlikely that bronchoalveolar lavage will benefit the patient during the acute stage. The importance of using the bronchoscope to obtain materials for culture and to remove thick, purulent secretions is obvious. As observed by Freitag and colleagues,4 repetitive, multiple, and not infrequently life-threatening stenoses and strictures and granulation of the tracheobronchial tree seem to be the recurrent theme in the subacute and chronic stages. Again, repeat bronchoscopic examinations (averaging seven per patient6) seem necessary to evaluate and treat the tracheobronchial complications. While the progression and recurrence of tracheobronchial stenoses is relentless, bronchoscopic dilatation, stent placement, and Nd:YAG laser therapy seem to lessen the respiratory distress and prolong the life of these unfortunate victims. Endobronchial brachytherapy was attempted out of desperation in a difficult case, but its role in treating this type of airway problem is uncertain. Although balloon dilatation via fiberoptic bronchoscope was employed in some patients described by Freitag and associates6 and others11,12 in the management of tracheobronchial stenoses, it is important to stress that the rigid bronchoscope is far more versatile in managing stenotic lesions of the airways, particularly in stent placement.13-15 Granted that repeated bronchoscopy has the potential to further irritate the already damaged tracheobronchial mucosa, it is unlikely that, if carefully performed, it will increase the occurrence of airway strictures or other pulmonary complications.

It was fortuitous that no chemical weapon was unleashed in the recent Gulf war. The recent revelations by the media that research in chemical warfare was actively pursued in Iraq during the Gulf war is again a stark reminder to physicians of the devastating effects of these agents on the respiratory system. Nevertheless, the pulmonary specialist can take some solace from the knowledge that the versatile bronchoscope has emerged as a powerful weapon to fight many of the pulmonary impairments inflicted by deployment of chemical warfare.

Udaya B. S. Prakash, M.D., F.C.C.P.
Rochester, Minnesota

Professor of Medicine, Mayo Medical School, and Director of Bronchoscopy, Mayo Clinic, Rochester, Minn.
Reprint requests: Dr. Prakash, East 18, Mayo Clinic, Rochester, Minnesota 55905

REFERENCES

1 Chemical and biological weapons in the 1980s [editorial]. Lancet 1984; 2:141-43
3 Watts TE, Craig P. Colloquia on radiation, arms race, chemical and biological warfare. Med War 1960; 6:12-9
5 Orient JM. Chemical and biological warfare: should defenses be researched and deployed? JAMA 1989; 262:644-48
9 Winternitz W. Chronic lesions of the respiratory tract initiated by inhalation of irritant gases. JAMA 1919; 73:588
11 Cohen MD, Weber TR, Rao CC. Balloon dilatation of tracheal and bronchial stenosis. AJR 1984; 142:477-78
14 Dumen JF. A dedicated tracheobronchial stent. Chest 1990; 97:328-32