Polyvinyl Chloride Pulmonary Disease

To the Editor:

The article by Cordasco et al (Chest, 1981; 78:828-34) and the accompanying commentary by Lills (78:828-28) provide an excellent review of the pulmonary manifestations of polyvinyl chloride (PVC) exposure. Pulmonologists must be aware of the constantly expanding number of potentially dangerous industrial, and often ubiquitous, environmental hazards. The authors have thus provided an important service by their studies.

We would like to describe our experience with a patient who exhibited many of the findings reviewed in the above papers and in addition point out an additional source of PVC pulmonary injury.

CASE REPORT

A 30-year-old fireman presented to Montefiore Hospital and Medical Center in 1977, two years after he was exposed to heavy concentrations of PVC fumes emanating from burning cable insulation. At that time, he was admitted for one day because of shortness of breath (SOB) and severe hypoxemia. Since that episode he complained of cough with purulent sputum, inability to exercise, chest pain, wheezing, and a 10 lb weight loss. The patient was a nonsmoker with a negative prior pulmonary history and was an active long distance runner. Physical examination revealed a few scattered rales. A chest roentgenogram revealed increased interstitial markings at the bases, and pulmonary function studies revealed normal results of spirometry, and a minimal reduction in diffusing capacity (DLco). Arterial blood gases were normal. His SOB continued and a transbronchial lung biopsy was normal. Histologic sections revealed interstitial fibrosis with scattered areas of more extensive scarring. The patient continued to deteriorate with further decreases in his exercise tolerance, continued cough and SOB and had to retire from the fire department.

The products of combustion of polyvinyl chloride are numerous, the most important of which are chlorine, phosgene and hydrochloric acid. We believe that the pulmonary lesion was caused by the patient's inhalation of these agents along with the thermal degradation products of PVC. Although acute pulmonary damage from PVC combustion has been described,1 there are few data on the occurrence of chronic pulmonary parenchymal lesions, and this area needs further study.

The widespread use of PVC in wrapping material, electrical insulation, furniture coverings, and in many plastic products, make it a potential hazard of enormous consequence.

Kenneth L. Pinsker, M.D., F.C.C.P.; Stanley Fell, M.D., F.C.C.P.; and Stephan L. Kamholz, M.D., F.C.C.P., Departments of Pulmonary Medicine and Thoracic Surgery, Montefiore Hospital and Medical Center, Albert Einstein College of Medicine, Bronx

REFERENCE

1 Dyer RV, Esch VH. Polyvinyl chloride toxicity in fires: hydrogen chloride toxicity in fire fighters. JAMA 1976; 235:393-97

A Syndrome of Sleep, Stridor, and Panic

To the Editor:

Recently, we have encountered four patients with what appears to be rare episodes of stridor that develop only during sleep. The four had virtually identical complaints.

Our patients are men (ages 41, 49, 55, 55 years). In each case, the patient awakens from sleep in a panic and is unable to breathe in. He struggles to breathe and in order to break through the obstruction, makes prodigious inspiratory efforts which may be silent or associated with stridulous sounds. When the obstruction is finally overcome (within seconds or up to a minute) a loud stridulous or crowing sound is made. Once effective breathing begins, the obstruction seems to disappear. In two of the patients, the voice remained slightly hoarse after the episodes.

The episodes appear to occur at random. In three of the patients, an episode occurred about once every two or three months. In the other patient, episodes occurred once per month or sometimes more frequently. None of the patients had clinical features suggesting sleep apnea.

All four patients had polygraphic sleep studies which were entirely normal. Two had endoscopic evaluation of the upper airway within hours of an episode. No abnormality was found.

That the problem involves the upper airway is suggested by the stridulous inspiratory sounds and in two, hoarseness. We suspect that glottic closure or spasm is responsible, perhaps caused by irritation of the larynx by mouth or stomach contents. The reason we were unable to document these episodes during sleep was related to their random and rare occurrence.

The disorder appears benign, and the patients have had the problem for several years. In spite of the fact that the patients know that the obstruction is temporary, during each event the patient is in a panic state.

Meir H. Kryger, M.D.; John C. Acres, M.D.; and Laurence Brownell, M.D., Department of Respiratory Medicine, St. Boniface General Hospital, Winnipeg, Canada

Reprint requests: Dr. Kryger, 2C, St. Boniface General Hospital, 409 Tache Avenue, Winnipeg, Manitoba R2H 2A6, Canada

Cotton-Picker Froust

To the Editor:

The April, 1981 Chest supplement on byssinosis covers the subject comprehensively and includes introductory historical references. The naming of the condition, however, is unattributed.

I believe the word "byssinosis" was first used by Dr. Adrien Froust, father of Marcel, in 1877 in his paper "Affections pulmonaires succedant à l'inhalation des poussières de coton—byssinosis." With roots in Greek and Latin, the French words bysse and byssus denote linen or similar material of high quality.

L. Fred Ayvazian, M.D., F.C.C.P. Chief, Pulmonary Section, Veterans Administration Hospital, East Orange, NJ