Once the blood pressure stabilized on a norepinephrine (Levophed) drip and her respiratory parameters improved with ventilation and suctioning, the laceration was sutured and tourniquet removed, and the patient was transferred to the intensive care unit for continued cardiopulmonary monitoring. The patient was successfully extubated after eight days and discharged home 11 days after her adverse reaction to lidocaine.

Bradford J. Woelke, M.D., Department of Emergency Medicine, and Robert A. Tucker, M.S., R.Ph., Drug Information Specialist, Clinical Pharmacist-Critical Care, Pontiac General Hospital, Pontiac, Michigan

Weight Loss in Chronic Obstructive Lung Disease

To the Editor:

In your editorial (Chest 1982; 82:518) on the etiology of emaciation in patients with advanced chronic obstructive lung disease, one possible cause is not mentioned. This is impairment of intestinal absorption in the presence of severe hypoxemia. Miledge1 showed that xylose absorption was reduced in such patients, and on our recent American Medical Research Expedition to Everest, impaired xylose absorption was found in several subjects living at an altitude of 6300 m where the arterial Po2 was approximately 40 mm Hg. This possible cause of weight loss in patients with severe hypoxemia deserves further study.

John B. West, M.D., Ph.D., Department of Medicine, University of California, San Diego, La Jolla

REFERENCE


To the Editor:

Dr. West’s comments are well chosen. Weight loss in chronic obstructive lung disease should prove preventable when the causative factors are defined. Preventing weight loss should significantly prolong the natural history of this disorder. If chronic hypoxemia turns out to be a major influence on calorie intake, we will have a useful parameter for beginning oxygen therapy. It is my impression that the current use of oxygen does not affect weight loss in these patients.

Richard L. Hughes, M.D., F.C.C.P., Chicago

Noncardiac Pulmonary Edema

To the Editor:

Mountain et al (Chest 1982; 82:371-72) report the development of noncardiac pulmonary edema following the administration of parenteral paraldehyde for treatment of delirium tremens. We agree with the authors that the patient had noncardiac pulmonary edema, but disagree with their assumption that it was due to direct damage to the alveolar-capillary membrane by paraldehyde. The development of noncardiac pulmonary edema may have been due to hypotension—as present in their patient—secondary to the administration of large doses of paraldehyde.

Felix R. DiPinto, M.D., and Prashant K. Rohatgi, M.D., Pulmonary Service VA Medical Center, Washington, D.C.

REFERENCE


To the Editor:

Drs. DiPinto and Rohatgi suggest that hypotension, rather than direct lung injury, was responsible for the noncardiac pulmonary edema we observed following parenteral paraldehyde. Two lines of evidence suggest this notion is mistaken. First, in our patient the respiratory difficulty and hypoxemia clearly antedated the hypotension; prior to the apnea and cyanosis requiring intubation, the minimum systolic blood pressure was 96 mm Hg and generally ranged from 100-110 mm Hg despite previous administration of paraldehyde. Furthermore, other causes of noncardiac pulmonary edema such as sepsis, pulmonary aspiration, disseminated intravascular coagulation, transfusions, and hypoalbuminemia were reasonably excluded. Second, most clinical and experimental evidence indicates a relatively minor role for shock per se in the etiology of permeability pulmonary edema (ie, ARDS).1

In a clinical series from our institution,2 shock alone could be implicated as the etiology in only 2 percent of 100 consecutive cases of ARDS. In a series of trauma patients, Horovitz et al3 found sepsis to be the major risk factor for ARDS, whereas patients without shock actually had a greater incidence of ARDS than patients with shock. Also, animal studies of hemorrhagic shock in sheep4 and baboons5 suggest a minimal role for hypoperfusion in altering pulmonary alveolar-capillary permeability. Consequently, the traditional term “shock lung” is misleading. Hypotension alone appears to be relatively unimportant in the pathogenesis of permeability pulmonary edema, but may potentiate more severe insults, particularly sepsis.

Richard Mountain, M.D., Division of Pulmonary Sciences University of Colorado Health Sciences Center, Denver

REFERENCES


The Effect of Ultrasonically Nebulized Distilled Water on Lung Epithelial Permeability

To the Editor:

We read with interest the editorial by E. S. Lilker (Chest 1982; 82:263-64). He suggests that asthma is a disease of increased permeability of the pulmonary mucous membrane. It is possible to measure directly the permeability of the lung epithelium to large molecules by an isotope technique using the stable chelate "Tc DTPA.1 If an aerosol of this agent is inhaled, the adjusted rate of loss of radioactivity from the lung field as measured by scintillation...
counters, expressed as a half-time, will give an index of lung permeability.

We have applied this technique to normal subjects to investigate the effect of cold air, distilled water fog and normal saline fog on permeability. Nine nonsmoking nonasthmatic subjects were studied on four occasions. An initial control value for permeability was obtained; they were then challenged with cold air, distilled water fog and normal saline fog with at least five days between challenges. For the cold air challenge, subjects hyperventilated for three minutes from an adapted deep freeze. For the two fog challenges, subjects breathed tidally to a total of 80 liters from a DeVilbiss 66 ultrasonic nebulizer on a setting of 10 (maximum). No significant change in FEV$_1$ occurred for any subject for any challenge. Permeability was measured immediately after each challenge and compared to the control value by paired $t$ test. Following distilled water fog, the clearance halftime fell by a mean of 22.4 min ($p<.05$). For cold air a decrease of 8.4 min (NS) and normal saline a decrease of 0.7 min (NS) was observed.

We conclude that an increase in permeability to large molecules without concomitant bronchoconstriction occurs in response to distilled water aerosol inhalation in normal subjects. Whether similar, perhaps larger, changes occur in asthmatic patients or whether their difference in response is due to other mechanisms requires further study.


REFERENCE


**Recurrent Spontaneous Pneumomediastinum**

**The First Reported Case**

*To the Editor:*

Although first reported by Hamman in 1939,¹ spontaneous pneumomediastinum (SP) is still considered a rare entity. In recent years, as awareness of SP has grown, the syndrome seems to be much more prevalent than previously believed.²³ We have encountered 14 cases of SP over a four-year period,² all of which occurred in young and healthy individuals. One of the patients in this series suffered from a recurrent episode of SP, a phenomenon that has not been reported to date.

A 19-year-old soldier was admitted for severe retrosternal pain that occurred after running 5 kilometers. His past history was unremarkable, and he was accustomed to participating in intensive athletic activity. On admission, subcutaneous emphysema was palpated and Hamman's sign (mediastinal crunch) was heard. Chest x-ray film revealed mediastinal and cervical emphysema. The patient was hospitalized, and after four days of bed rest the mediastinal air resolved completely. Four months later, he underwent resection of a benign thymus tumor under general anesthesia. Chest x-ray examination on that occasion was within normal limits. Fourteen months following the first episode of SP, the patient was readmitted with a second episode of retrosternal pain that again occurred after running. The diagnosis of recurrent SP was confirmed by chest x-ray film. He was submitted to bed rest at home and a repeated chest roentgenogram performed a week later showed no residual mediastinal air.

Although only about 300 cases of SP have been reported in the literature, we believe there have been more. Regarding the incidence of 1:7,115 admissions reported by Munsell² and the incidence of 1:12,850 admissions found in our series, it can be calculated that a few thousand cases should occur every year. We have observed that SP is second to spontaneous pneumothorax as a cause for admissions in young (under 30 years old), healthy individuals experiencing sudden chest pain or shortness of breath.

Most authors accept Macklin and Macklin's theory⁴ of alveolar hyperpressure with rupture of marginal alveoli and dissection of air along interstitial planes to the mediastinum as the pathogenic basis for SP. This is usually caused by a sudden elevation in bronchiolar pressure, a mechanism shared by many cases of spontaneous pneumothorax. Although both situations affect a similar age group and have almost the same preference to males, spontaneous pneumothorax is notorious for its high recurrence rate.⁷ It is therefore believed that the majority of patients suffering from spontaneous pneumothorax have some underlying pulmonary parenchymal pathology. Judging from the lack of recurrences of SP, one can assume that lung architecture is preserved in the latter condition. It is possible that episodes of recurrent SP have occurred, but have not been reported or have passed unrecognized, and thus, spontaneous pneumothorax and SP may be two presentations of the pulmonary air leak differing only in anatomic location of the alveolar rupture. Nevertheless, evidence so far favors the acceptance of SP as a separate entity, and as such, a recurrent episode is exceptional.

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REFERENCES


**Needle in a Haystack**

*To the Editor:*

This is to report an unusual and heretofore undocumented complication from fiberoptic bronchoscopy. Separation of the wire cytology brush (Olympus B.C. -SC) from its attachment to the wire guide recently occurred during routine diagnostic bronchoscopy. The 3 cm tip of the brush/wire was lost in the parenchyma of the right upper lobe beyond the view of the bronchoscopist approximately 8 cm from the hilum near a suspected tumor. The wire was retrieved successfully without complication by use of forceps (Olympus FB15C) with the aid of the C-arm fluoroscope (G.E. TH9412E) within a 45 minute period. The manufacturer of the instrument and the