the airway. We have recently seen a patient who illustrates this complication.

A 30-year-old black man presented to our emergency room with pleuritic left chest pain, a cough producing yellow sputum, chills and night sweats. He had recorded fevers of 38.3°C and lost eight pounds during a two-week period. The patient had a nine-year history of drug abuse, and frequently smoked cocaine alkaloid (freebase).

Upon examination, he was alert, oriented and afebrile with normal respiratory rate and blood pressure. The remainder of the examination revealed palpable lymph nodes in the left supravacuicular, right axillary and both femoral regions, and needle marks on the forearms.

The white blood cell count was 7,100/mm³ with 55 percent neutrophils, 35 percent lymphocytes, 6 percent eosinophils and 1 percent metamyelocytes. Hematocrit, serum chemistries and coagulation profile were normal. Sputum smear revealed no bacteria, AFB or fungi. A chest radiograph demonstrated a parenchymal infiltrate and a 2 cm metallic, pin-like object in the left upper lobe.

Rigid bronchoscopy revealed edema and erythema of the left upper lobe orifice and a 25 gauge hypodermic needle lodged in the bronchial lumen (Fig 1), which we removed. Secretions from within the needle hub grew Streptococcus viridans and lactobacilli.

We believe that freebase cocaine inhalation predisposed our patient to foreign body aspiration by topical anesthetization of his airways. Freebase cocaine is inhaled by mixing cocaine hydrochloride with baking soda and a flammable solvent, and smoking the mixture in a special pipe with wire screens. Our patient, using a hypodermic needle, punched holes in aluminum foil creating the screen through which he smoked freebase cocaine. He may have left the needle dangling in the foil and inhaled it into an anesthetized airway. Similarly, Bezmalinovic and associates recently reported a patient who suffered severe upper airway burns after using freebase cocaine. They hypothesized that decreased sensation allowed their patient to inhale smoke hot enough to severely burn his upper airway. We suggest that whenever a history of cocaine inhalation is obtained from a patient with an infiltrate on chest radiograph, the possibility of foreign body aspiration should be considered.

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Sleep Apnea Investigations

To the Editor:

The interesting article by Levy et al (Chest 1989; 95:95-99) examines the effect of negative pressure ventilation on sleep and respiration in five normal subjects. The conclusions (as stated in the discussion and the abstract) are that sleep quality can be impaired and that sleep apneas can be induced by this form of treatment. The authors have, however, clearly shown by their results that there is no significant difference in sleep efficiency, percentage of time spent in each sleep stage, or the total number of apneas per hour between the control and treatment nights. The number of apneas plays hypopneas per hour did increase during negative pressure ventilation, but the significance of this is doubtful for two reasons. First, the hypopneas are difficult to define satisfactorily, largely because measurement of air flow using thermocouples is at best only semiquantitative. Secondly, the frequency of apneas plus hypopneas only increased slightly; in this number they would be unlikely to cause any symptoms or significant physiologic changes. Far from showing that negative pressure ventilation increased the frequency of apneas and impairs sleep quality, the results of this study have shown that these complications are not a significant problem in normal subjects.

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To the Editor:

We are pleased that our study stimulated the interest of Dr. Shneerson. Although the total number of apneas did not increase with negative pressure ventilation (NPV), the number of obstructive, and obstructive plus mixed apneas did increase significantly. The number of central apneas did not increase, and this is reflected in the total number of apneas. This is not surprising since, by definition, a breath was always provided by the mechanical ventilation; therefore, central apneas could not occur with NPV.

As pointed out by Dr. Shneerson, the indices used in our study to assess sleep quality were not statistically different between the two study nights. Presumably this reflects the small number of subjects studied. Although no differences were noted in sleep architecture for the group data, whereas all subjects reached all sleep stages on control nights, only four reached stage IV sleep, and three reached REM sleep with NPV. It is conceivable that patients with impaired arousal responses might have even greater disruption of sleep.

We certainly agree that the number of apneas and hypopneas observed with NPV in our normal subjects would be unlikely to be of clinical consequence. However, it should be pointed out that these subjects presumably had normal arousal responses as well as normal ventilatory function. The importance of the phenomenon of induction of obstructive apneas with NPV would be expected to be greater in patients with impaired arousal responses, such as those with sleep apnea syndrome. Furthermore, apneas may be of considerable concern in patients who are on the steep portion of
the oxyhemoglobin dissociation curve or in patients with low expiratory reserve volume due to obesity or chest wall deformities since derangements of pulmonary mechanics and awake arterial oxygen tension are of major importance in establishing the severity of nocturnal hypoxemia in patients with sleep apnea syndrome.

Clearly, it would be of major importance to determine the impact of apnea induction with NPV in patients for whom the treatment is prescribed during sleep.

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Behcet’s Disease with Half and Half Nail and Pulmonary Artery Aneurysm

To the Editor:

Half-and-half nail, also known as Lindsay nail, is defined as a red to brown discoloration occupying 20 to 50 percent of the distal fingernails on both hands.1 We report a patient who had Behcet’s disease and half-and-half nail.

A 31-year-old man presented with bilateral pleuritic chest pain, hemoptysis, fever and mild attacks of dyspnea. His medical history revealed that he had recurrent oral and genital ulcerations for the past 16 years. Behcet’s disease was diagnosed in another hospital three years ago, and he has been irregularly taking varying doses of colchicine since.

Medical examination was remarkable for subcutaneous thrombosed veins on the anterior surface of his right forearm and multiple elevated hypopigmented macular lesions on his left forearm. Half-and-half nails were also noticed (Fig 1). It was learned from the patient that the color change in his fingernails was present before the administration of colchicine therapy and there were other individuals in his family with similar nail appearance.

Laboratory values were all within normal limits. Mycologic research on the nails was negative. Pulmonary digital subtraction angiography (DSA) revealed an aneurysm in the left main pulmonary artery, including its lower lobar branch. Obstruction of some of the peripheral pulmonary artery branches were also noted.

This patient’s history and clinical picture is compatible with the diagnosis of Behcet’s disease in its complete form.2 The clinical picture of pulmonary vascular involvement usually consists of fever, chest pain, hemoptysis and recurrent opacities on the chest x-ray film.3 Our patient presented with these symptoms and signs. Obliteration of the small branches of the pulmonary arteries, in addition to an aneurysm, was demonstrated.

We were unable to find any reported case of half-and-half nail associated with Behcet’s disease in the literature. A brownish-red band covers 20 to 50 percent of the nail beds in half-and-half nails.4 There may be also increased melanin in the nail plates, but this was absent in this case. It is usually diagnosed in patients with renal disease and uremia, and infrequently in healthy persons.5,6 We concluded that this patient with Behcet’s disease has half-and-half nails; an additional renal or hepatic disease was excluded in the face of clinical and laboratory findings.

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Skunk Lung

To the Editor:

We recently evaluated a 33-year-old man who was in excellent health until he developed symptoms of breathlessness and wheeze after being "skunked". The patient was camping and awoke to find a skunk in his immediate vicinity. Upon confrontation, the skunk discharged, and the patient immediately became ill. He noted nausea but denied immediate respiratory symptoms or symptoms of aspiration. After a few hours of distress, he returned to sleep; but the following morning, he awoke with a sense of chest tightness which progressed over two days and became associated with significant dyspnea on exertion. This led to his initial medical evaluation at which time wheeze and a reduced peak flow rate of 200 were noted. Examination was otherwise unremarkable, and a chest x-ray film was normal. Without specific therapy, his symptoms stabilized and gradually improved. One week after his initial skunk exposure, he was free of symptoms and physical examination revealed no wheezing even on forced expiration and a peak flow rate of 550 was measured.

We believe the patient suffered an episode of bronchial hyperreactivity following the exposure to an extremely noxious, proteinaceous substance. The material discharged from the anal glands of a skunk has not been widely studied but has been noted to cause severe eye and skin reactions in addition to its effect on the olfactory system. We have been unable to find any reports of similar respiratory problems from skunk exposure. However, bronchial hyperreactivity can occur in animal handlers. Laboratory exposure to rats, mice, guinea pigs, and rabbits has been reported to cause asthma. The major source of allergens was found to be in the

Figure 1