Utility of CPAP in Gastroesophageal Reflux

To the Editor:

Since 1981, when Sullivan et al suggested continuous positive pressure in the airway by means of a nasal mask (nCPAP) as a therapeutic alternative for the sleep apnea syndrome (SAS), many patients have benefited from this technique. At the present time, there can be no doubt as to the treatment's efficiency for obstructive apneas. The mechanism of action of nCPAP is based on the maintenance of positive pressure in the upper airways during sleep, thus preventing them from collapsing and avoiding apneic episodes, with resultant clinical improvement of the patient. At our SAS research unit we have recently noticed a new application of the nCPAP, which we shall discuss briefly below.

We have observed five patients in whom SAS was associated with severe gastroesophageal reflux (GER) that remained symptomatic in spite of the usual pharmacologic treatment (antacids and anti-H₂). Control of respiratory symptoms associated with SAS was a serious problem owing to the undesirable effects exerted on the upper digestive tract by bronchodilator drugs. In these patients, the use of nCPAP made it possible to control the symptoms of reflux esophagitis, even allowing us to withdraw the anti-reflux medication completely in one case, in addition to eliminating the syndrome's characteristic apneas.

For a long time now, GER has been associated with pulmonary pathologies including pulmonary fibrosis, asthma, bronchitis, aspiration, etc., and the interferences existing between symptomatic treatment of both etiologic entities (anti-H₂ and theophilline, antacids and Gram-negative colonization of the upper airways, theophilline and gastric toxicology). Accordingly, it could be thought fitting to have a common therapy available to benefit patients suffering from GER and pulmonary pathology.

In the case of patients suffering from SAS, the application of nCPAP not only prevents collapse (with the resultant disappearance of apneas), but this increase in pressure also strengthens the organism's natural anti-reflux mechanisms because of retrograde transmission through the esophagus. As a result, the barrier blocking passage of gastric acid to the esophagus is consolidated and an ailment is brought under control.

As we have observed in our patients, nCPAP may constitute an efficient remedy as the only therapy for the treatment of patients suffering from SAS and GER. Further research should confirm nCPAP as a suitable therapeutic alternative in the treatment of patients suffering from GER but not from SAS.

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Hypodermic Needle Aspiration in a Freebase Cocaine Abuser

To the Editor:

Individuals who abuse drugs may develop numerous pulmonary complications including respiratory depression, aspiration pneumonia, and pulmonary edema. Abusers of cocaine, a topical anesthetic, may also develop complications related to anesthetizing

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The white blood cell count was 7,100/mm$^3$ 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 lactobacillae.

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 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

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