all three complications together in preeclampsia and eclampsia provides evidence for the existence of the HELLP syndrome. In 1954, Pritchard and associates reported three cases of eclampsia in women with hemoglobinemia, hemoglobinuria, thrombocytopenia and clotting defects. Two of the women died. In both of those cases, severe epigastric pain and generalized abdominal discomfort were noted on admission to hospital. At autopsy, microscopic examination of the liver showed focal periportal necrosis in one case and diffuse degenerative changes within the parenchymal cells, with subacute inflammation of portal areas in the other.[2]

Killam and colleagues published a report of five cases of HELLP syndrome associated with preeclampsia that were very similar to the one reported here. Three of their patients showed substantially abnormal hematologic and liver function test values but did not have severe hypertension. Initially these patients were mistakenly diagnosed as having hepatitis, hiatal hernia and pyelonephritis, respectively. Despite the difference in presentation, all five patients had evidence of liver dysfunction, depressed platelet counts and intravascular coagulation and hemolysis. The authors concluded that the disorder must be strongly considered in all patients with severe preeclampsia or eclampsia with epigastric pain. They also advised that the poor fetal and maternal prognoses necessitate expeditious delivery.[3]

In conclusion, our report lends support to the existence of a subset of women with preeclampsia and eclampsia who manifest hemolysis, elevated liver enzyme levels and low platelet counts. It is therefore important that physicians caring for obstetric patients be cognizant of the clinical and laboratory findings in the HELLP syndrome in order to give early supportive therapy and delivery.

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Endoscopic Observations of the Pharyngeal Airway During Treatment of Obstructive Sleep Apnea With Nasal Continuous Positive Airway Pressure—A Pneumatic Splint

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SINCE THE FIRST DESCRIPTION OF SLEEP APNEA in Pickwickian patients in 1965,[1] the sleep apnea syndrome has been recognized with increasing frequency in a variety of clinical situations.[2] The cardiopulmonary complications associated with this disorder often lead to consideration of a tracheostomy. The psychological and physical trauma experienced by patients with tracheostomies,[3,4] however, has prompted the development of alternative modes of therapy.

The pathophysiology of the obstructive variety of sleep apnea has been proposed to be a combination of the negative oropharyngeal pressure generated during inspiration and reduced neurogenic tone in the upper airway muscles associated with sleep, especially the rapid-eye-movement period of sleep. The decreased neural activity leads to a relaxation of the upper respiratory muscles, which causes posterior displacement of the tongue and inward movement of the pharyngeal walls. This muscular relaxation, especially if combined with an upper airway anatomic abnormality, such as fatty infiltration,[5] macroglossia[6] or nasal polyps,[7] is often sufficient to cause a physical obstruction to airflow.

ABBREVIATIONS USED IN TEXT
CPAP = continuous positive airway pressure
EMG = electromyography

With this pathophysiology in mind, it can be appreciated that continuous positive airway pressure (CPAP) applied at the nares, as introduced by Sullivan,[8,9] may be useful in the treatment of obstructive sleep apnea. The mechanism of action of CPAP has been eloquently likened to that of a "pneumatic splint" of the upper airway. We offer our observations in one patient on the effects of nasal CPAP on the patency of the nasopharynx and hypopharynx that confirm this concept.

Report of a Case

The patient, a 60-year-old man, while in hospital for the evaluation of new onset of seizures, was referred for evaluation of apneic episodes observed during sleep. He admitted to snoring for many years, and his wife described nocturnal apneic episodes, often of long duration, for many years. The patient complained of frequent sleep interruptions for the past six years, associated with paroxysmal nocturnal dyspnea. These were often so severe as to require him to sleep sitting up in a chair.

The patient was formerly employed as a bus driver, but he often experienced daytime hypersomnolence of such severity that he would fall asleep while parking his bus. He would also fall asleep at the dinner table or during other activities that did not require his attention.

He said he did not have insomnia, nightmares, enuresis, morning headache, depression or sexual dysfunction. There was no history of cardiopulmonary or nasopharyngeal disease. The patient had had hypertension for 18 years and intermittent pedal edema for 6 years and took a thiazide diuretic

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irregularly. He took no other medications. He was a cigarette smoker and consumed alcoholic beverages on social occasions.

On physical examination, the patient was obese (165 cm, 134 kg), and had normal vital signs except for a blood pressure of 160/90 mm of mercury. The nasopharynx showed no evidence of septal deviation or polyps. The soft palate, uvula and tonsils were of normal size. The remainder of the examination elicited no abnormalities.

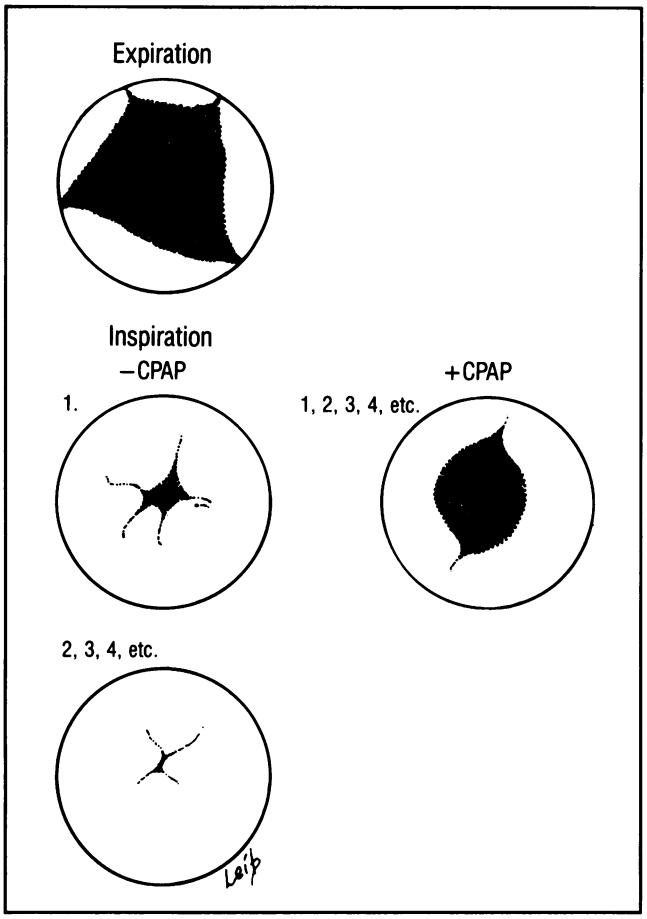

Figure 1.—Diagrammatic representation of the pharyngeal airway during sleep seen through a fiber-optic bronchoscope. During respiration without continuous positive airway pressure (−CPAP), inspiration is accompanied by occlusion of the airway after one breath. With CPAP (+CPAP), the airway remains patent throughout.

Laboratory findings of interest included normal hematocrit and thyroid function test values. Arterial blood gas determinations with the patient breathing room air showed a partial oxygen pressure of 66 mm of mercury, a partial carbon dioxide pressure of 52 mm of mercury and pH of 7.37. A chest roentgenogram and electrocardiogram were normal.

After informed consent, the patient was studied with the nasal CPAP mask in place (a pediatric CPAP mask, Vital Signs). This was fitted with a Portex T-piece Adaptor to allow passage of the fiber-optic bronchoscope (Olympus BF-4B2) and delivery of CPAP. After the patient's right nostril was anesthetized with 2% viscous lidocaine hydrochloride solution, the bronchoscope was introduced through the nose and into the pharynx to a point just above the posterior tongue.

The patient was then allowed to fall asleep with the nasal CPAP system and bronchoscope in place. He was simultaneously monitored for thoracoabdominal movements by impedance pneumography, arterial oxygen saturation by ear oximetry (Hewlett-Packard 47201A) and nasopharyngeal airflow by pressure fluctuations in the CPAP system.

Serial episodes of obstructive apnea with desaturation developed within minutes after the patient fell asleep. The pharyngeal airway was seen to collapse, as described by others.[8,10,11] The sequence of events observed is illustrated in Figure 1, with increasing circumferential narrowing of the lumen with sequential inspirations, and then complete obstruction followed by inspiratory tugging of this structure.

Airflow at 60 liters per minute was delivered through the CPAP system without change in the obstructive apneas. A positive airway pressure of 2.5 cm of water was then introduced using a CPAP valve (Vital Signs). This produced immediate opening of the hypopharynx (Figure 1), associated with the return of airflow (Figure 2). When the CPAP valve was removed, there was an immediate recurrence of obstruction at the same level. This sequence of events was observed repeatedly.

Discussion

In this patient we have described the sequence of events observed with obstructive sleep apnea and the anatomic changes accompanying its treatment with nasal CPAP. To

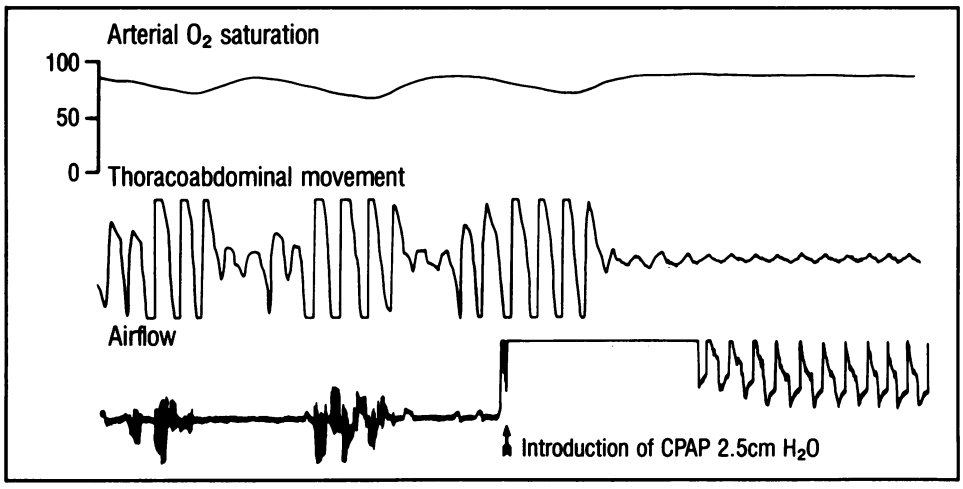

Figure 2.—Arterial O_2 saturation, thoracoabdominal impedance and airflow at the mouth and nose were recorded during sleep. The first half of the tracing shows two episodes of obstructive apnea accompanied by desaturation. Continuous positive airway pressure (CPAP) of 2.5 cm H_2O is introduced, with relief of the obstruction and return of regular respirations.

understand the mechanism by which nasal CPAP overcomes this form of sleep apnea, it is first appropriate to review the pathophysiology of the obstruction.

Most observers have shown that obstruction occurs at the level of the nasopharynx and hypopharynx, with or without a contribution from the tongue.[10-13] Hill and co-workers[12] have noted that in normal persons there is electromyographic (EMG) evidence of repetitive firing of the pharyngeal abductors and adductors that is synchronous with the respiratory cycle during both sleep and wakefulness. This same basic pattern of EMG activity was observed in patients with obstructive sleep apnea, except at the times of obstruction. During the apnea, the pharyngeal abductors and adductors were shown to be either greatly hypotonic or atonic. Just before cessation of the apnea, there was an increase in EMG activity in these muscles. Sauerland and Harper[13] have placed a greater emphasis on the change in EMG activity of the genioglossus muscle in the pathogenesis of obstructive sleep apnea. They noted that obstruction occurred at a time when the oropharyngeal pressures and the EMG activity of the genioglossus muscle were most reduced.

It has therefore been postulated that obstruction to airflow results from an imbalance between the negative oropharyngeal pressure during inspiration, which tends to collapse the upper airway, and the tone of the pharyngeal abductors and adductors and the genioglossus muscle, which tends to maintain airway patency. Nasal CPAP, when delivered as previously described, serves to counteract the negative inspiratory forces by introducing positive pressure into the oropharynx and thus compensates for the hypotonicity of the upper airway muscles.

In conclusion, our observations clearly show that the obstruction to airflow in obstructive sleep apnea occurred at the level of the pharynx in this patient and that this obstruction can be successfully overcome with the use of nasal CPAP, which acts as a "pneumatic splint."

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