for 12 to 13 days before tracheostomy was performed. He, therefore, could have suffered a similar injury.

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REFERENCE

To the Editor:

We wish to thank Dr. Wilson for suggesting that the bilateral vocal cord paralysis we reported in a patient with Guillain Barré syndrome may have resulted from trauma secondary to intubation with a cuff-inflated endotracheal tube. Although this explanation could have contributed to the paralysis, we believe it was primarily the result of the Guillain Barré syndrome because:
1. Prior to intubation the patient's cough reflex was absent and he could speak in only a whisper, suggesting the paralysis had already begun.
2. Laryngoscopy during intubation revealed partially abducted cords with minimal movement.
3. The cuff of the endotracheal tube (size 9 foam cuffed Bivona) was not manually inflated and the cuff's port was left open to the atmosphere.
4. The intubation was easy and atraumatic.
5. Two otolaryngologists examined the patient and dismissed trauma as a cause for the paralysis.
6. The paralysis persisted for more than two years which is markedly different from the four-eight weeks required to regain normal function in the group of patients studied by Cox and Welborn. In summary, the pre-intubation onset of paralysis, the apparent lack of trauma to the cords or trachea, and the duration of the paralysis suggests that our patient's vocal cord paralysis was a result of the Guillain Barré syndrome and not from damage to the recurrent laryngeal nerve.

The patient mentioned by Dr. Wilson is interesting because the duration of her bilateral vocal cord paralysis is uncommon in cases of recurrent laryngeal nerve damage. Our experience is that edematous and poor moving (partially paralyzed) vocal cords are sometimes seen in anesthetized patients intubated with an air-filled endotracheal tube. We find this type of vocal cord malfunction always resolves within eight weeks, as also reported by Cox and Welborn.

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REFERENCE

Chlorothiazide Relieves Asthmatic Wheezing

To the Editor:

In the article by Ziment (Chest 1983; 83:392-395), the possible adverse side effects of diuretics in the treatment of asthma was mentioned. His article as well as others, dealing with the treatment of asthmatic patients, failed to mention or suggest any beneficial effect of diuretics on the asthmatic condition. I would like to testify to the cessation of wheezing after being placed on diuretic medication.

I have been an asthmatic for most of my 47 years. I have lived in Georgia for the past seven years and was constantly bothered by periodic (sometimes daily) bouts of wheezing and reduced expiratory capability which required use of an isoproterenol inhaler for relief. About nine months ago, I started taking 125 mg (4 tablet) of chlorothiazide once a day for my recently discovered mild hypertension (140/90). I have not wheezed since. I am still subject to bronchiolar constriction caused presumably by certain allergens occasionally in my environment, resulting in the minor sensation of some tightness in my chest, but not once has this feeling justified the necessity of the use of the isoproterenol inhaler.

According to the pharmacologic description of chlorothiazide (both in the PDR and basic textbooks), it causes persistent small reduction in extracellular water and plasma volume. While I am tempted to conclude that I do not wheeze because of bronchiolar interstitial fluid reduction, there may be other factors involved. However, it is interesting to note that children, during an acute, severe asthmatic attack, had dramatic increases in antidiuretic hormone in their plasma. Also, during induced allergic bronchoconstriction in guinea pigs, the airway epithelial permeability increases. Therefore, it is conceivable that with bronchoconstriction, excess interstitial fluid may be transported across the epithelium into the lumen contributing to wheezing.

Regardless of the mechanism, I wish to share this beneficial effect of chlorothiazide with physicians, so that others may perhaps enjoy the definite relief I have experienced.

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To the Editor:

Dr. Odend’hal’s observation on the apparent beneficial effects of chlorothiazide in asthma cannot be taken as evidence that diuretic therapy will bring relief to any other patient with asthma. Obviously, he would have to present much more evidence from controlled investigation before his correlation can be accepted. Nevertheless, the concept he raises does merit further consideration.

Clinically, there appears to be no recognition that the use of diuretics is of value in asthma, unless there is underlying left heart failure ("cardiac asthma") or fluid overload (as in "renal asthma"). Although fluid exchanges across the tracheobronchial membrane are of importance in asthma, there is no evidence of a significant increase in interstitial fluid occurring during an exacerbation of bronchospasm. Furthermore, if such fluid accumulation did occur, it would probably be equally as inaccessible to removal by diuretic therapy as is the edema fluid in the skin or other tissues in inflammatory or allergic disorders.

If fluid retention were of significance in asthmatic exacerbations, one would expect that women patients would note an increase in symptoms prior to menstruation. Further, many women take diuretics to treat the premenstrual tension syndrome, and asthmatic
patients would surely note that this treatment would relieve their bronchospasm if indeed the phenomenon that Dr. Odendahl postulates actually occurs. There appears to be no literature on this, and my own experience, as well as that of several authorities whom I have consulted, does not support such a clinical correlation.

I therefore conclude that there is insufficient evidence to suggest that diuretics can influence the usual types of asthma that we see. However, if a subset of asthmatic patients does appear to manifest a beneficial response to diuretic administration, it would be important to identify these individuals who could then be evaluated so as to determine if there is a cause-and-effect relationship.

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Home Care for Ventilator-dependent Individuals

To the Editor:

We basically agree with the views expressed in the recent editorial concerning the need for improvements in home care delivery systems for ventilator-dependent individuals. The case histories presented by Hughes dramatically emphasize that ventilator-dependent persons cannot be discharged home by institutions and health care professionals with little experience in this area. At the present time, the problems illustrated by Dr. Hughes' patients can only be avoided by hospitals and home care systems which can marshall the resources to develop programs focused primarily upon the care of these individuals. The approach used by the University Hospital at Boston University Medical Center has been successful in large part because of the organization of a coordinated out-patient and in-patient multidisciplinary team under the daily co-direction of a pulmonary physician and a respiratory nurse-specialist; the major responsibility of each team member is for the care of ventilator-dependent patients. As implied by Goldberg, a regional approach toward home care for ventilator-dependent persons may be an effective method of assuring optimal home care with the limited resources imposed by hospital cost restraints.

As with any new medical innovation, further information conveyed through published reports documenting both benefits and problems, monographs outlining specific approaches, and seminars to train health professionals, should allow expansion of home care to additional institutions in the future. Because of the divergent approaches and relative lack of published data concerning the home care of ventilator-dependent persons, the American Thoracic Society is considering a workshop to make specific recommendations concerning such issues as the appropriateness of home care, criteria for hospital discharge, patient and family teaching, equipment and personnel necessary in the home, psychosocial issues of patients and families, and the costs of home care. Until additional information is available concerning the efficacy of home care, discharge of ventilator-dependent patients should be considered only by institutions with comprehensive, carefully designed programs.

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Osler and Sarcoïdosis

To the Editor:

In a recent delightful historical report (Chest 1984; 86:263), Sharma and James discussed the case of Hannah W., who was seen by William Osler in 1894. This 11-year-old child presented bilateral gland enlargement, generalized lymphadenopathy, and an enlarged spleen. These signs gradually improved, and when Hannah died three years later of cavitary tuberculosis, there was no trace of the previous findings at autopsy. Osler considered her a case of chronic parotitis. Now, 90 years later, Sharma and James have re-examined the case and concluded that Hannah's illness was actually a case of sarcoidosis. They bemoan the fact that Osler described a "noble disorder" but was robbed by Nature of the opportunity to describe a new disease.

I agree with the authors that it was truly a case of sarcoidosis, but I must remind them that Osler was not "robbed." It was Sir Jonathan Hutchinson (1829-1913), a surgeon, who first described the disease in 1869, fully 25 years earlier, and again published his findings in 1875. He published two more cases in 1898 and, as a tribute to the female patient whose name was Mortimer, entitled the paper "Cases of Mortimer's Malady." The 1981 edition of Dorland's Medical Dictionary calls our noble disease "Hutchinson-Boeck sarcoidosis."

It may be of interest to know that this underrecognized surgeon, clinician, and pathologist made many other findings, and his name is associated with Hutchinson's facies, mask, patch, unequal pupils in cerebral hemorrhage, teeth, triad, and varicella gangrenosa. In addition, he wrote a ten-volume Archives of Surgery and with Hastings Gilford described progeria.

Also prior to Osler's case, Besnier in 1889 and Tenneson in 1892 described other facets of the disease. Boeck described his first such case in 1899, and in 1916 described additional cases; he called the disease "multiple benign sarcoid of the skin." The history is well described by Longcope and Freiman in their monograph on sarcoidosis.

As a matter of fact, although Osler's textbook of medicine ran through eight editions prior to his death in 1919, not one of them mentioned the syndrome of sarcoidosis. Even the tenth edition, published with McCrae in 1925, failed to record it.

I am grateful to Drs. Ronald J. Knudson, Benjamin Burrows, and Charles Mittman for helping me distinguish between Sir Jonathan Hutchinson and John Hutchinson who developed a spirometer in 1844.

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