

Correspondence

Anesthesiology
46:374, 1977

Unilateral Vocal-cord Paralysis

To the Editor:—Any description of a vocal-cord paralysis that follows endotracheal anesthesia is appreciated in that it draws attention to the poorly understood clinical phenomenon of idiopathic post-anesthesia vocal-cord paralysis. Beyond this service, however, Dr. Minuck's report¹ inflates rather than extends the literature. Worse, the commentary is superficial and the conclusions misleading.

Dr. Minuck, like most authors detailing cases of unilateral vocal-cord paralysis in the immediate postoperative period, indicts the endotracheal tube on the basis of circumstantial evidence. In fact, the *raison d'être* for his report is ostensibly to reconsider this lesion in light of information recently published by Ellis and Pallister, who suggest a possible anatomic mechanism for indirect trauma to the recurrent laryngeal nerve from an endotracheal tube cuff. These investigators showed by cadaver dissection that when an endotracheal tube was passed and the cuff inflated within the larynx, it compressed an anterior branch of the nerve between the cuff and the thyroid lamina.²

In a paper that stresses the etiologic role of an improperly positioned or defective endotracheal tube cuff, it seems incredible that Dr. Minuck does not specify the type of endotracheal tube employed (high-compliance cuff? red rubber?). Were the tube of the disposable, low-pressure cuff variety, the author's suggestion that nerve injury might be secondary to "irregular inflation of a cuff [defective] in manufacture or following many sterilizations" makes little sense. Granting for the sake of argument that the tube had a damaged cuff that inflated asymmetrically, exerted high pressures, and was located high in the larynx, one must question the author's awareness of the following facts: 1) many cases of post-endotracheal-intubation vocal-cord paralysis involve both cords.³ 2) in cases of unilateral paralysis, the left vocal cord is affected approximately twice as frequently as the right.⁴ 3) males are affected approximately seven times more fre-

quently than females.⁴ 4) a number of cases of unilateral and bilateral paralysis following the use of non-cuffed tubes in pediatric patients have been reported.⁵ 5) there have been isolated reports of vocal-cord paralysis following mask⁶ and spinal anesthesia (Mass L: Anesthesiol Rev 3:18-21, 1976).

Obviously, variations in the quality and positioning of endotracheal tube cuffs cannot account for all cases of idiopathic postanesthesia vocal-cord paralysis. I respectfully but completely disagree with Dr. Minuck's assertion that "prevention of this apparently rare complication lies in a) eliminating the use of endotracheal tubes with [defective] cuffs, b) desisting from the practice of deliberately placing the cuff within the larynx, and c) filling the cuff with a sample of the inspired mixture of gases, regular deflation of the cuff, or alternatively, routine use of a simple pressure-relief valve."

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To the Editor:—I was most interested to read Dr. Minuck's article (*ANESTHESIOLOGY* 45:448-449, 1976), in which he considered an hypothesis proposed earlier by us,¹ namely, damage to the recurrent laryngeal nerve by a cuff inflated within the

larynx as a cause for unilateral vocal-cord paralysis following endotracheal intubation. As a laryngologist I must emphasize that I see patients who have no symptom referable to the larynx but who nonetheless on mirror examination have a paralyzed

vocal cord. If such patients should undergo endotracheal intubation, it is likely that any inflammation or edema would precipitate hoarseness, and a laryngologist would diagnose a recurrent laryngeal-nerve palsy. It is important to recognize that an asymptomatic laryngeal-nerve palsy can predate an endotracheal intubation as well as being caused by it. Examination of the vocal cords at the time of endotracheal intubation may be helpful in distinguishing between these two.

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Iatrogenic Reflex Sympathetic Dystrophy?

To the Editor:—In a recent report by Abram,¹ a patient with severe burning pain and hyperesthesia of the hand was treated with transcutaneous nerve stimulation for three months. Initially there was complete analgesia lasting one to two and a half days after use of the stimulator, but later pain in the arm and shoulder appeared, along with hyperesthesia of the right hand. These symptoms were treated effectively with stellate ganglion blocks. Because decreased temperatures in the affected hand were found 40 minutes after the device had been used and because the arm and shoulder pain recurred with its continuous use, Dr. Abram concluded that transcutaneous nerve stimulation can cause increased sympathetic tone and frequent use may aggravate a reflex sympathetic dystrophy.

There has been no previous report of increase in sympathetic tone, or the *de-novo* appearance of reflex sympathetic dystrophy with stimulation, nor has increased sympathetic tone been implicated in later failures of transcutaneous nerve stimulation. We have treated several patients with reflex sympathetic dystrophy at the University of Virginia Pain Clinic using transcutaneous nerve stimulation, and have consistently observed decreases in sympathetic tone as measured by thermistors, plethysmography and thermography. Currently a study is under way in a series of patients with reflex sympathetic dystrophy to determine the long-term effects of transcutaneous nerve stimulation. Moreover, there has been no instance of reflex sympathetic dystrophy in patients treated with transcutaneous nerve stimulation in our clinic for a variety of other disease processes.

There are several possible explanations for this disparity. In the case reported by Dr. Abram, it is

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Serum Bromide Concentrations in Anesthetists

To the Editor:—It has been observed that anesthetists who administer halothane daily to patients do not show significant increases in serum bromide

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probable that reflex sympathetic dystrophy was present before the initiation of transcutaneous nerve stimulation, as manifested by the severe burning pain and hyperesthesia, the most characteristic symptoms of reflex sympathetic dystrophy. Progression of the process including spread of the pain into the arm and shoulder has been well established previously.² It is conceivable that this represents the natural course of the disease process in this patient. Also, it is possible that there was another disease process occurring in this 75-year old woman, which modified her symptoms; possibilities include bursitis, arteritis, or collagen vascular disease.

Before transcutaneous nerve stimulation is implicated as the cause of reflex sympathetic dystrophy, the more likely possibilities of natural progression of the disease and other contributing diseases should be considered.

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concentrations.¹ This is somewhat surprising, since anesthetists who inhale halothane chronically are known to metabolize it higher rates and bromide,