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