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.

P. D. M. ELLIS, F.R.C.S.
*Senior E.N.T. Registrar*
*The Middlesex Hospital*
*London, England*

REFERENCE

1. Ellis PDM, Pallister WK: Recurrent laryngeal nerve palsy and endotracheal intubation. J Laryngol Otol 89:823–826, 1975

*(Accepted for publication January 18, 1977.)*

Anesthesiology
46:375, 1977

## 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.

RICHARD J. STILZ, M.D.
*Resident in Anesthesiology*

HAROLD CARRON, M.D.
*Professor of Anesthesiology*
*University of Virginia Medical Center*
*Charlottesville, VA 22903*

REFERENCES

1. Abram SE: Increased sympathetic tone associated with transcutaneous electrical stimulation. ANESTHESIOLOGY 45: 575–577, 1976
2. Drucker WR, Hubay CA, Holden WD, et al: Pathogenesis of post-traumatic sympathetic dystrophy. Am J Surg 97: 454, 1959

*(Accepted for publication January 17, 1977.)*

Anesthesiology
46:375–376, 1977

## 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,

one of the end products of halothane, has a prolonged serum half-life of 12 to 14 days. Serum bromide concentrations were therefore re-examined with the use of a new highly specific and sensitive method of measurement: x-ray fluorescence spectrometry.[2] Ten healthy, non-operating-room volunteers, five anesthetists who had not used halothane for at least two months, and five anesthetists who had used this anesthetic daily without scavenging devices for the previous two months or more were studied. In the latter individuals, blood samples were drawn at least 12 hours after they had left the operating room. We found no significant difference in the serum bromide concentrations between healthy volunteers and anesthetists who had not been exposed to halothane. The values averaged 49.2 ± 9.1 μM and 43.3 ± 7.6 μM, respectively. In contrast, serum bromide values were significantly higher, 242.6 ± 72.5 μM ($P = 0.001$) in anesthetists chronically exposed to halothane.

The most likely explanation for our finding of much higher serum bromide values in exposed anesthetists than previously reported is our use of a more sensitive method for measuring serum bromide concentrations.

Whether the higher bromide concentrations chronically achieved among anesthetists are responsible for side effects remains to be established.

PHILIPPE DUVALDESTIN, M.D.
*Department of Anesthesia*
*University of Paris VII*
*Hôpital Bichat*
*75018, Paris, France*

Anesthesiology
46:376, 1977

## REFERENCES

1. Johnstone RE, Andrews R, Brummund W: Bromide concentrations of anesthetists. ANESTHESIOLOGY 43:128, 1975
2. Barbier Y, Girard C, Vandroux JC, et al: Mesure de l'espace de diffusion du brome chez l'homme par fluorescence X, Radio-aktive Isotope in Klinik und Forschung, Vol. 11. Edited by R Hoefer. München–Berlin–Wien, Urban and Schwarzenberg, 1975, pp 231–236

*(Accepted for publication January 18, 1977.)*

# More on Baricity

*To the Editor:*—I feel compelled to call attention to an apparent error in Dr. Rosenberg's article which states that the concentration of "light dibucaine" is "0.66 per cent."[1] Although the proprietary solution of dibucaine commonly used for hypobaric spinal anesthesia and referred to as (Howard) Jones' solution is known to have a higher density at body temperature than tetracaine hydrochloride diluted to 0.1 per cent with distilled water (0.9967 *vs.* 0.9943 g/ml, 37 C),[2,3] this is *not* because the concentration of dibucaine in Jones' solution is *greater* than that of tetracaine hydrochloride in 0.1 per cent solution. Indeed, the concentration of dibucaine in Jones' solution is only 0.066 per cent (*not* 0.66 per cent). It is a 1:1,500 mixture containing dibucaine, 0.66 mg/ml of 0.5 per cent sodium chloride solution.

Furthermore, it would appear from Dr. Rosenberg's data for tetracaine that the baricity of Jones' solution, though known to be greater than that of 0.1 per cent tetracaine in distilled water, might more closely approximate that of 0.33 per cent tetracaine in distilled water. However, such a comparison cannot be made with accuracy without converting the density values to a common temperature.

It is unfortunate that Dr. Rosenberg combined graphically the density values for various concentrations of tetracaine determined by him at 23–25 C with "CFS Density Limits 37 C" found by others who established the densities of a number of spinal anesthetic solutions at body temperature.[2,3] It would have been more helpful if Dr. Rosenberg had standardized his data to those already published by others so that his baricity values could be compared with those obtained by others at normal body temperature and so that the data would relate more closely to the clinical portion of his interesting study.

CHARLES M. LANDMESSER, M.D.
*Department of Anesthesiology*
*Albany Medical Center*
*Albany, NY 12208*

## REFERENCES

1. Rosenberg H: Density of tetracaine–water mixtures and the effectiveness of 0.33 per cent tetracaine in hypobaric spinal anesthesia. ANESTHESIOLOGY 45:682–684, 1976
2. Davis H, King WR: Densities of common spinal anesthetic solutions at body temperature. ANESTHESIOLOGY 13: 184–188, 1952
3. Davis H, King WR: Densities of cerebrospinal fluid in human beings. ANESTHESIOLOGY 15:666–672, 1954

*(Accepted for publication January 18, 1977.)*