

# **ELECTRICAL PACING OF THE PARALYZED LARYNX WITH AN IMPLANTABLE DEVICE**

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## **ABSTRACT**

Electrical stimulation of the vocal fold opening muscles, paced with inspiration, offers a physiologic approach to restore ventilation in case of bilateral laryngeal paralysis. This study presents the results of the first clinical trial of laryngeal pacing in the United States. The patient was implanted with an Itrel II stimulator (Medtronic, Inc.), and followed over a 16 month period. Optimum stimulus parameters for vocal fold opening (i.e. abduction) were identified: 1.5 second train of 1 millisecond pulses delivered at 40 hertz and 2-6 volts in amplitude. PCA stimulation with these parameters produced as much as 7.0 mm of dynamic vocal fold opening, which restored normal ventilation without disturbing voice.

## **INTRODUCTION:**

Bilateral vocal fold paralysis (BVFP) is a serious and often life-threatening clinical problem. Surgical techniques such as laser arytenoidectomy can be performed to enlarge the embarrassed airway. However, these procedures sacrifice voice and airway protection to restore ventilation. They also ignore the long-term effects of atrophy on vocal fold mass and position. A new, more physiologic approach termed laryngeal pacing has been studied in animal models.<sup>1-3</sup> It involves electrical stimulation of the posterior cricoarytenoid (PCA) muscle to restore ventilation with inspiration. During noninspiratory phases, the vocal folds passively relax to the midline to allow for normal voicing and airway protection. Recently, the efficacy of laryngeal pacing was directly demonstrated in the human patient. An external pacing circuit and percutaneous needle electrodes were used to deliver stimuli to the abductor muscles.<sup>4</sup>

In view of the feasibility of this treatment using an external device, preliminary trials with an implantable device have been undertaken by Medtronic, Inc. in a phase I multi-institutional FDA study. To date, seven patients have been implanted worldwide with this laryngeal pacemaker. The present report describes the results of the first implantation in the United States.

## **METHODS:**

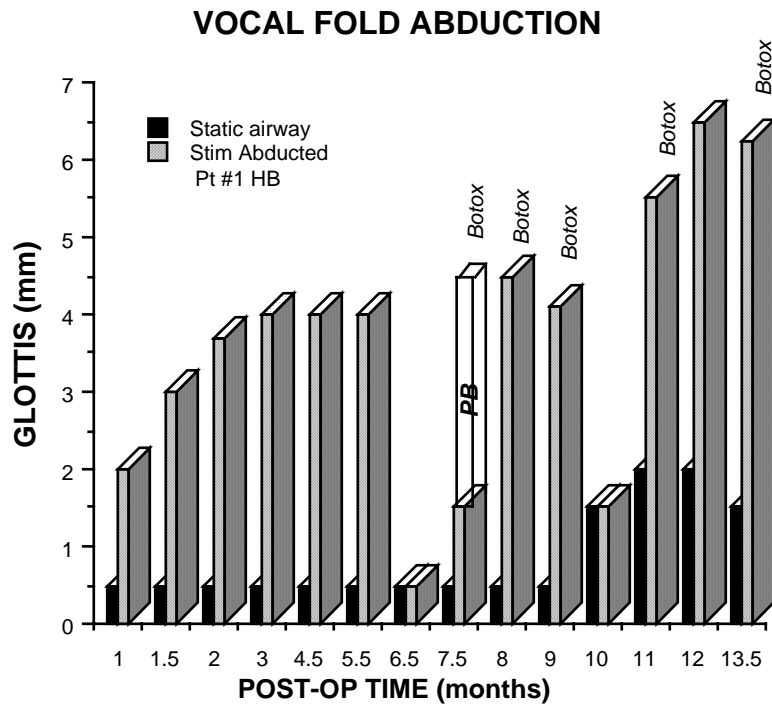
The patient was a 63-year-old female with an eighteen month history of BVFP following thyroidectomy. She was tracheotomized two months later. At the initial visit, she was unable to tolerate closure of the tracheotomy tube for more than a few seconds. The general innervation status of laryngeal muscles was assessed by means of percutaneous needle electromyography. The Itrel II device was then implanted. An incision was made in the neck and the electrode inserted into a subperichondrial pocket between the PCA muscle and its cartilage. The electrode lead was brought subcutaneously to a second incision where the implantable pulse generator (IPG) was positioned. After implantation, the IPG could be programmed through the skin by an external transmitter to change stimulus parameters. In monthly post-operative sessions with the patient, an effective stimulus paradigm was derived, the magnitude of stimulated vocal fold abduction and ventilation measured, and the effect of stimulation on voice production assessed. The primary goal of the study was to determine whether the device could restore ventilation

through the mouth equal to that through the tracheotomy without disturbing voice. If so, mouth ventilation would be deemed sufficient to permit removal of the tracheotomy, i.e. decannulation.

**RESULTS:**

As often observed in patients with BVFP following thyroidectomy, all laryngeal muscles had some reinnervation as shown electromyographically. However, the reinnervation was faulty as indicated by the inappropriate firing patterns of motor units. Both the inadequacy and synkinetic nature of reinnervation likely accounted for the vocal fold immobility.

In post-operative sessions, it was determined that a 1.5 second train of 1 millisecond pulses delivered at a frequency of 40 hertz and amplitude of 2-6 volts effectively produced a dynamic airway. One and a half seconds of stimulated abduction allowed sufficient air exchange with each breath. The device was set to deliver twelve stimuli every minute to match the patient's respiratory rate at a moderate level of activity. (This device in its present form is not synchronized with respiration). The ideal stimulus amplitude was one that evoked maximum vocal fold opening without inducing discomfort or nociception. At this amplitude, the patient could feel the stimulus, which provided a signal to inspire.



**FIGURE 1**

Figure 1 is a histogram of the static and stimulated airway measurements in post-operative sessions. In the initial sessions, a stimulated opening of 2.5 mm was obtained which was significantly larger than the .5 mm static airway. Stimulated abduction increased further to 4 mm with the identification of optimal stimulus parameters. At 6.5 months, the loss in abduction was attributed to a slight shift in electrode position less favorable for PCA activation. It was hypothesized that the shift could have also caused greater recruitment of nearby nerves supplying adductor (closing) muscles, which antagonized glottal opening. In order to nullify the antagonistic response, a short-acting neuromuscular blocking agent, pancuronium bromide (PB), was injected into the ipsilateral thyroarytenoid muscle. Within ten minutes, dynamic abduction

was restored to a level greater than that observed in previous sessions. The success with PB provided a rationale for injection of botulinum toxin (Botox or "B"). As shown in subsequent sessions, the stimulated abduction following Botox treatment was maintained at a higher level. Repeated injections of Botox over the next nine months not only maximized the dynamic airway (7.0 mm), but directly enlarged the passive airway (2.5 mm).

The device malfunction at 10 months was due to the loss of an electrode channel. Since the electrode had only two channels, the stainless steel case was made to serve as the second channel, and the highest level of abduction was regained.

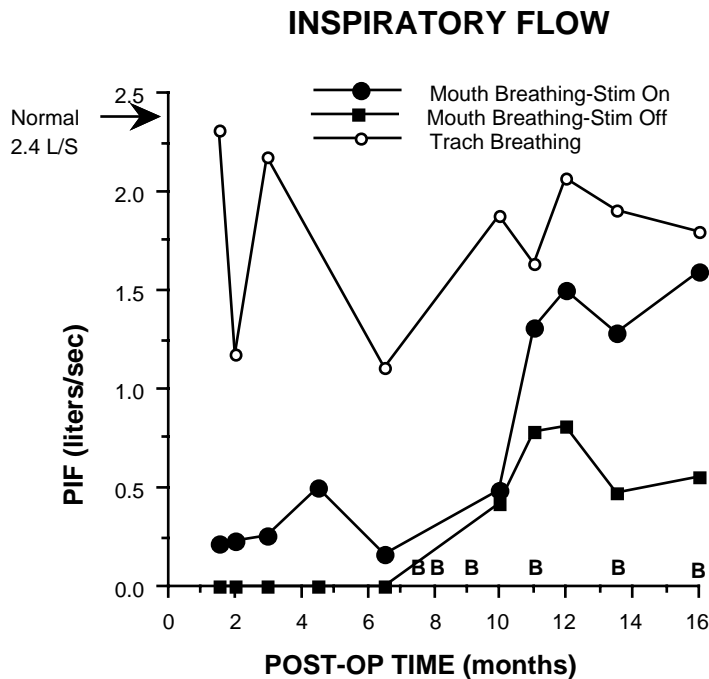


FIGURE 2

Figure 2 summarizes the ventilation data, graphically illustrating the flow rate of inspired air measured at each session. Air flow through the mouth with the device on (filled circles) was always greater than with the device off (filled squares). This finding was not unexpected since the device was observed endoscopically to produce vocal fold abduction. However prior to Botox administration, mouth breathing with the device on was dramatically less than trach breathing (open circles). Air exchange was so limited that the patient could not tolerate trach closure for more than a few minutes. This inadequacy in ventilation was surprising and appeared inconsistent with the relatively large glottal opening viewed endoscopically during stimulation. The explanation lay in the fact that the patient's glottis in the "off" phase was so constricted that air exchange during expiration was severely compromised. Following Botox therapy, the static airway increased sufficiently to allow normal volume exchange during both inspiration and expiration with the device either on or off (not shown). The volume of air that could be passed through the mouth equaled that which could be passed through the tracheotomy. For the first time since her tracheotomy, the patient could sit quietly with her stoma closed for many hours. Furthermore, when the device was activated the patient could engage in more demanding levels of activity such as standing or walking. As shown in figure 2, the airflow during stimulated abduction was sufficient to support this higher level of activity, while airflow through the passive glottis was not (11-16 months) Flow rates with dynamic opening approached 1.6 liters per second characteristic of that through the trach site. With the device on, the patient kept the trach

closed 24 hours a day. Subsequently, two months after completion of the study, the patient was decannulated.

The patient's voice before implantation was hyperfunctional, and rated as moderately rough and strained. Following implantation of the Itrel II, there was no effect of chronic PCA stimulation on voice quality over a six month period of electrical pacing. With subsequent repeated botox injections into both vocal folds, the passive glottis enlarged and the patient's voice quality improved to near normal.

#### DISCUSSION:

The results of this study demonstrate the merit of laryngeal pacing as a new treatment approach. The optimum stimulus parameters for PCA activation were identified. Using this paradigm, electrical pacing restored vocal fold movement with a peak glottal opening as large as 7 mm. Once the passive airway had been enlarged with Botox to permit expiration, ventilation through the mouth was also recovered. An inspiratory airflow of 1.6 liters per second allowed the patient to engage in normal everyday activity, sufficient for her eventual decannulation. The fact that chronic electrical pacing caused no disturbance of voice further validated the physiologic nature of this treatment. The improvement in ventilation with null effect on voice attest to the significant advantage of laryngeal pacing over conventional surgical therapies for BVFP.

#### REFERENCES:

1. Zeale DL, Dedo HH. Control of paralyzed axial muscles by electrical stimulation. *Acta Otolaryngol (Stockh)* 1977;83:514-27.
2. Obert PM, Young KA, Tobey DN. Use of direct posterior cricoarytenoid stimulation in laryngeal paralysis. *Arch Otolaryngol* 1984;110:88-92.4.
3. Sanders I. Electrical stimulation of laryngeal muscle. *Otolaryngol Clin North Am* 1991;24:1253-74.5. Zeale DL, Rainey CL, Nettekville JL, Herzon GD, Ossoff RH. Electrical pacing of the paralyzed human larynx. *Ann Otol, Rhinol, Laryngol* 1996;105(9):689-93.
4. Zeale DL, Rainey CL, Nettekville JL, Herzon GD, Ossoff RH. Electrical pacing of the paralyzed human larynx. *Ann Otol, Rhinol, Laryngol* 1996;105(9):689-93.

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