A NEW APPROACH TO TREATMENT OF SPASTIC DYSPHONIA

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ABSTRACT

A case of successful treatment of spastic dysphonia is described. The procedure used was suggested by Dr. Herbert Dedo, and involved surgical sectioning of one recurrent laryngeal nerve causing paralysis of one vocal cord in paramedian position. The result in this case was similar to results reported by Dedo: complete elimination of spastic dysphonia.

INTRODUCTION

Spastic dysphonia characterized by strained, choked voice with aphonic breaks and extreme tension of the phonatory system, is usually described as highly resistant to any form of therapy. (Luchsinger and Arnold, 1965; Cooper, 1973; Boone, 1971). While many authors suggest psychotherapeutic treatment (Cooper, 1973; Luchsinger and Arnold, 1965; Moncur and Brackett, 1974), Boone (1971) states that few, if any spastic dysphonic patients have regained normal phonation through psychotherapy. He states that the only success he has had with spastic dysphonia has been in teaching the patient a relaxed phonatory response such as a sigh, then having him use this relaxed phonation in a hierarchy of contexts. Even with this approach, however, he describes progress as erratic and limited, with no one patient experiencing a permanent restoration of normal voice. Current therapy methods have proven ineffective in the treatment of spastic dysphonia.

Dedo (1976) recently described a surgical treatment for spastic dysphonia in which the recurrent laryngeal nerve is sectioned to cause paralysis of one vocal cord in paramedian position. In all of the 26 cases that he reported, the operation resulted in improved voice. In 24 of the cases, the strained vocal quality and associated struggle behaviors were eliminated completely. Some of the patients achieved a clear voice immediately, others initially spoke in a breathy voice which was improved after several speech therapy sessions.

The following case replicates those described by Dedo, and is presented here to inform speech pathologists of this new approach to treatment of spastic dysphonia.

CASE REPORT

Background

A 45 year old male was admitted to the Montreal General Hospital in December, 1975 for investigation of dysphonia of ten years duration. He reported that his voice had been normal until 1965, when a benign tumor was removed from his mandible and he was subsequently readmitted for surgical drainage of infection. Two months later, he began to experience difficulty speaking. Since the extreme effort required for speaking caused headaches and dizziness, he avoided speech as much as possible. Attempts to improve his

voice with hypnosis, speech therapy, acupuncture, and psychotherapy were uniformly unsuccessful.

The patient's voice on assessment was strained and choked, with frequent aphonic moments during which lip tremors, facial grimaces and flushing were observed. He was able to whisper and to speak in the falsetto register without difficulty.

Under indirect laryngoscopy the vocal cords appeared normal, and approached the midline symmetrically. Neurological testing revealed no abnormalities. The patient showed no characteristics that might suggest a psychogenic basic for his voice disorder. Mild depression and an unwillingness to talk seemed to be consequences of his disorder rather than causative factors. The fact that hypnosis and psychotherapy were unsuccessful further supports the view that the disorder was not psychogenic in this case.

The patient was discharged with a diagnosis of spastic dysphonia of unknown cause, and arrangements were made for him to attend speech therapy.

Speech Therapy

The patient attended four speech therapy sessions weekly for a period of four months, and reported that he practiced for two hours daily at home. Therapy was directed at teaching him to phonate without adducting the vocal cords completely, so that spasms would not occur. Within the first month of therapy, the patient progressed from breathy sighs, to breathy phonation on one, two, and three syllable words and phrases. He began to use the breathy voice intermittently in conversation, and although spasms still occured, they were milder and the patient reported that he did not experience headaches nor dizziness while using this voice. He renewed social contacts, began using the telephone, and showed satisfaction with his progress. Frequently, however, he was unable to produce the breathy voice. During the next three months of therapy, no further improvement was noted. The patient continued to experience occasions when he was unable to produce the breathy voice.

Surgical Treatment

In May, 1976, an article appeared in the Medical Post (de Vries, 1976) describing Dedo's experimental surgical treatment for spastic dysphonia. The otolaryngologist and the speech pathologist mentioned the procedure to the patient, who showed immediate interest.

Paralysis of the left vocal cord was first effected temporarily, by injection of Xylocaine into the patient's left recurrent laryngeal nerve. For a half hour following the injection, the left cord was fixed in paramedian position, and the patient spoke in a soft, breathy voice, with no spasms. As the effect of the Xylocaine wore off there was gradual reappearance of the spastic dysphonia. Tape recordings were made before and after the injection.

The patient, the speech pathologist and the otolaryngologist agreed that there was sufficient vocal improvement to warrant permanent sectioning of the recurrent laryngeal nerve.

Surgery was carried out under general anaesthesia. An incision was made along the anterior border of the left sternocleidomastoid from the level of the thyroid notch to the sternoclavicular joint. The underlying muscles were retracted and the recurrent laryngeal nerve was identified as it was passing from the tracheo-esophageal groove into the larynx.

The recurrent laryngeal nerve was stimulated and movement of the ipsilateral cord was confirmed by direct laryngoscopy. An eight millimeter section of the nerve was removed.

RESULTS

Immediately upon waking up after surgery, the patient began to speak. His voice was completely free of spasms, and was slightly breathy but less so than during the trial procedure. Under indirect laryngoscopy the left cord was seen to be fixed in paramedian position. The right cord adducted during phonation, leaving a one or two millimeter gap between the cords.

The patient showed great satisfaction, both with the sound of his voice and with the ease of speaking, but mentioned that he hoped to be able to speak more loudly after the surgical wound healed.

To help increase loudness, speech therapy was resumed one week after surgery. Pushing exercises were used to bring the mobile right vocal cord into closer approximation with the paralyzed cord. The patient produced a clear, loud voice while pushing, and then attempted to imitate this voice without pushing. He initially tended to raise his pitch when he increased loudness, but corrected this when it was pointed out to him. After four weeks his voice was louder, with less of the breathy quality than was present after surgery.

There have been no signs of recurrence of the spastic dysphonia, nor of conversion symptoms. The patient states that he feels 'liberated', and is able to speak continuously without fatigue.

DISCUSSION

The patient described in this report commented that he could have bought a Cadillac with the money he had spent on hypnosis, acupuncture and psychotherapy, even though he found it difficult to believe that his disorder was psychological in origin. At the time of onset, he had assumed that it was related to a persistent infection he had suffered two months before. His impression is interesting, in view of the suggestion made by Dedo (1976) that spastic dysphonia may be caused by a neurotropic viral infection which disrupts the proprioceptive control of the vocal cords, making them adduct too tightly. The surgery used by Dedo fixed one vocal cord slightly away from the midline, thus eliminating the excessive adduction. This appears to be the first treatment which has totally eliminated spastic dysphonia.

The success of this procedure in the cases reported does not in any way confirm Dedo's impression that spastic dysphonia is caused by viral infection. Further research is necessary to test the many theories concerning the disorder.

Dedo's suggested etiology and treatment for spastic do warrant further study. Research in the field of speech pathology may be directed at specifying the role of voice therapy pre- and post-operative for the patients undergoing this procedure.

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