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Letter to the Editor

Laryngeal Manifestations of Speech Dysfluency: A Topical Anesthesia Treatment Approach

Dear Editor:

Stuttering is a developmental disorder that usually begins in childhood. Its cause is unclear in most cases. Specific focus on the role of the larynx during stuttering has demonstrated cocontraction and spasmodic bursts of antagonistic (abductor/adductor) laryngeal muscles and excessive supraglottal activity. Similar findings have been demonstrated in patients with spasmodic dysphonia. Consequently, experimental use of Botox injections into the vocal folds of stutterers with hyperkinetic laryngeal signs have been shown to result in fewer dysfluencies and overall improvements in speech rate and intelligibility.[1] The purposes of this case study were to describe the aberrant phonation subsystem features exhibited by a patient with an adventitious dysfluency disorder, and the unique laryngeal treatment technique used to resolve this condition.

SUBJECT

The patient is a 38-year-old man with no previous speech, language, or hearing difficulties. Four weeks after a motor vehicle accident, he developed severe speech dysfluency, which persisted for 4 months despite traditional speech therapy. All consultations yielded the diagnosis of psychogenic stuttering, prompting referral to our office. Detailed analyses in our speech physiology laboratory revealed that the patient was dysfluent on approximately 50% of all words uttered, with an associated slow speaking rate of 69 syllables per minute (less than half normal). Experimental use of a transcervical electrolarynx (EL) promptly resulted in 100% speech fluency and a normal speaking rate. Laryngeal articulatory biomechanics, through videoendoscopic examination, revealed unremarkable anatomy but intermittent supraglottal sphincteric contractions, shimmying of the aryepiglottic folds, and dyssynchronous true vocal fold movement patterns. When aided by use of the EL, these pathophysiological features subsided.

CLINICAL DIAGNOSIS AND TREATMENT

All test results suggested that the dysfluency disorder might, at least in part, be attributable to the observed aberrant laryngeal behaviors. We recently reported success in treating patients with muscle tension dysphonias (MTD), who struggled with similar laryngeal signs, using topical anesthesia of the larynx.[2] This experience prompted experimental application of the same technique with the current patient. A transcricothyroid membrane injection of 4 cc 4% topical lidocaine was administered using a 5-cc syringe attached to a 1.5-in 21-g needle. The injection induced a reflexive cough that distributed the anesthetic solution over the subglottal, glottal, and supraglottal mucosal mechanoreceptors. Immediately postinjection a 15-minute series of easy voice onset exercises were used.

RESULTS

Within 15 minutes postinjection the patient was dysfluent less than 15% of the time and speaking rate was 82 syllables per minute. Without further intervention, dysfluency dropped to less than 10% and speaking rate increased to 127 syllables per minute at the 1-week follow-up. At 1-, 6-, and 12-month follow-up examinations both speech fluency and speaking rate were within normal limits, without evidence of relapse.

DISCUSSION

The larynx contains numerous mechano- and stretch receptors located within its mucosal linings, intrinsic muscles, and joints that provide continuous sensory feedback to the lower brainstem through the internal and recurrent laryngeal nerves. These communications form a sensorimotor laryngeal feedback loop that influences prephonatory vocal fold "set-points" such as vocal fold position, length, and muscular tension. Both neurologic and psychologic traumatic events may potentially disrupt the physiological preparedness of this complex system. It has been well documented that stuttering may be partially attributable to excessive tension and cocontraction of laryngeal muscle antagonists.[1] Such pathophysiology has also been observed in patients with MTD whose voice symptoms dramatically improved after topical laryngeal anesthesia.[2] For the current patient, this treatment technique resulted in quick and substantial speech fluency gains.

The mechanism of action of topical lidocaine in these cases remains unclear, although it is possible that this treatment served to disrupt the sensory arm of the aforementioned feedback loop. The probable physiological effect was a sensory "trick," which facilitated automatic rebalancing of prephonatory set points by anesthetically stifling the hyperfunctional larynx. Whether developmental stutterers or others with adventitious fluency disorders would respond as favorably to this treatment regimen requires further investigation. We suggest that dysfluent patients who are refractory to speech therapy might also benefit from appraisal of laryngeal anatomy and physiology. If a potential trigger is identified during these examinations, the lidocaine technique may be a reasonable

treatment consideration. It is an easy in-office technique, which takes less than 5 minutes to perform. Other than vigorous coughing and mild discomfort during the injection, no complications have been encountered. Presently, we are using nebulized lidocaine as an alternative to the injection approach. Results thus far have been encouraging, although this technique mandates an allotment of at least 20 minutes per patient for complete evaporation of the lidocaine canister.

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