

Functional Reinnervation of Vocal Folds After Selective Laryngeal Adductor Denervation-Reinnervation Surgery for Spasmodic Dysphonia

Adam S. DeConde, Jennifer L. Long, Bob B. Armin, and Gerald S. Berke, *Los Angeles, California*

Summary: Selective laryngeal adductor denervation-reinnervation surgery (SLAD-R) offers a viable surgical alternative for patients with adductor spasmodic dysphonia refractory to botulinum toxin injections. SLAD-R selectively denervates the symptomatic thyroarytenoid muscle by dividing the distal adductor branch of the recurrent laryngeal nerve (RLN), and preventing reinnervation, by the proximal RLN and maintaining vocal fold bulk and tone by reinnervating the distal RLN with the ansa cervicalis. We present a patient who had previously undergone successful SLAD-R but presented 10 years postoperatively with a new regional dystonia involving his strap muscles translocated to his reinnervated larynx by his previous ansa-RLN neurorraphy. The patient's symptomatic vocal fold adduction resolved completely on division of the ansa-RLN neurorraphy confirming successful selective functional reinnervation of vocal fold adductors by the ansa cervicalis.

Key Words: Spasmodic dysphonia—Functional reinnervation—Selective laryngeal adductor denervation-reinnervation surgery—SLAD-R.

INTRODUCTION

Adductor spasmodic dysphonia (ADSD) is a focal laryngeal dystonia in which spasmodic contractions of the intrinsic muscles of the larynx cause vocal tension and voice breaks. The current standard of therapy for ADSD is chemodenervation of the thyroarytenoid muscle with botulinum toxin.¹ Inherent limitations of botulinum toxin therapy such as temporary relief of symptoms, an unpredictable therapeutic window, breathy downtime, and the risk of developing antibodies² can be overcome by selective laryngeal adductor denervation-reinnervation surgery (SLAD-R).³ SLAD-R achieves a selective denervation of the thyroarytenoid muscle by identifying and dividing the adductor branch of the recurrent laryngeal nerve (RLN) distal to the take-off of the branch to the posterior cricoarytenoid muscle thereby preserving abduction. Denervation of the adductors interrupts the pathologic signals causing the laryngeal dystonia, whereas reinnervation with the ansa cervicalis nerve prevents reinnervation by the dysfunctional RLN.⁴ We present a case of demonstrated dynamic reinnervation of adductor muscles after SLAD-R surgery for spasmodic dysphonia. The University of California at Los Angeles's Institutional Review Board exempted this report.

CASE REPORT

A 40-year-old male presented with ADSD to the laryngology clinic after failure of botulinum toxin injections, and underwent

bilateral SLAD-R surgery. The patient's laryngeal dystonia resolved postoperatively and was quiescent for 9 years when the patient noted worsening tension in his voice temporally associated with muscle spasm of his anterior neck. Voice exam was notable for intermittent voice breaks and a strained quality. Physical examination demonstrated palpable strap muscle spasms concurrently with the voice breaks. Laryngostroboscopy showed normal vocal fold bulk and mobility. A course of botulinum toxin injections was attempted but was unsatisfactory to the patient.

The patient elected to undergo re-exploration of his neck with lysis of his ansa cervicalis reinnervation. The operation revealed bilaterally intact neurorraphies between the superior root of the ansa cervicalis and the distal RLNs. Electrical stimulation of the superior roots of the ansa cervicalis produced laryngeal adductor activity as evidenced by an electromyography nerve monitor and by palpation of the arytenoid cartilages. The neurorraphies were sectioned and postoperatively the patient's laryngeal dystonia immediately resolved.

The patient was seen at 9-months postoperatively when he remained free of tension in his voice and the anterior neck.

DISCUSSION

Our patient presented with simultaneous strap muscle and laryngeal dystonia 9 years after SLAD-R. The patient's long period of relief from spasms confirms the successful adductor denervation from his original SLAD-R surgery. His voice quality during that period was strong, implying successful reinnervation by the ansa cervicalis nerve. Surgical exploration confirmed functional reinnervation as ansa cervicalis stimulation led to vocal fold adduction. His delayed recurrence can be attributed to the not uncommon finding of a focal laryngeal dystonia progressing to a more regional dystonia including other cranial nerves.⁵ In this case, the patient's ansa cervicalis dystonia was transmitted to the larynx *via* the neurorraphy. The immediate and lasting cessation of voice spasms after

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From the Division of Head and Neck Surgery, University of California, Los Angeles, California.

Address correspondence and reprint requests to Adam S. DeConde, 62-132 CHS, UCLA Medical Center, Los Angeles, CA 90095-1624. E-mail: adeconde@mednet.ucla.edu

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lysing the ansa cervicalis verified that this transposed nerve was responsible rather than reinnervation by the original RLN.

This case provides conclusive evidence of successful laryngeal adductor reinnervation by the ansa cervicalis nerve after SLAD-R surgery for spasmodic dysphonia. It also presents the unusual transmission of ansa cervicalis dystonia to the larynx. It highlights that functional laryngeal reinnervation does occur when applied to single muscle groups, avoiding synkinesis.

CONCLUSION

We present a case of demonstrated dynamic reinnervation in SLAD-R surgery for spasmodic dysphonia. In this case, the disease recurred through aberrant activity of the translocated ansa cervicalis nerves.

REFERENCES

1. Blitzer A, Brin MF, Stewart CF. Botulinum toxin management of spasmodic dysphonia (laryngeal dystonia): a 12-year experience in more than 900 patients. *Laryngoscope*. 1998;108:1435–1441.
2. Zuber M, Sebald M, Bathien N, de Recondo J, Rondot P. Botulinum antibodies in dystonic patients treated with type A botulinum toxin: frequency and significance. *Neurology*. 1993;43:1715–1718.
3. Chhetri DK, Mendelsohn AH, Blumin JH, Berke GS. Long-term follow-up results of selective laryngeal adductor denervation reinnervation surgery for adductor spasmodic dysphonia. *Laryngoscope*. 2006;116:635–642.
4. Berke GS, Blackwell KE, Gerratt BR, et al. Selective laryngeal adductor denervation-reinnervation: a new surgical treatment for adductor spasmodic dysphonia. *Ann Otol Rhinol Laryngol*. 1999;108:227–231.
5. Marsden CD, Sheehy MP. Spastic dysphonia, Meige disease, and torsion dystonia. *Neurology*. 1982;32:1202–1203.