Functional reinnervation of vocal cords after selective laryngeal adductor denervation-reinnervation surgery for spasmodic dysphonia

Adam S. DeConde MD1; Jennifer L. Long MD, PhD1; Bob B. Armin MD1; Gerald S. Berke MD1
Division of Head and Neck Surgery, University of California, Los Angeles, CA

ABSTRACT

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 treatment for ADSD is chemodenervation of the thyroarytenoid muscle with botulinum toxin. Inherent limitations of botulinum toxin therapy such as temporary relief of symptoms, an unpredictable therapeutic window, breathy downtime, and the risk of developing antibodies can be overcome by selective laryngeal adductor denervation-reinnervation surgery (SLAD-R). SLAD-R achieves a selective denervation of the thyroarytenoid muscle. Denervation of the adductor muscles interrupts the aberrant nerve fibers causing the laryngeal dystonia, while reinnervation with the ansa cervicalis nerve may prevent reoccurrence of the dystonia.

INTRODUCTION

Objective: 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 treatment for ADSD is chemodenervation of the thyroarytenoid muscle with botulinum toxin. Inherent limitations of botulinum toxin therapy such as temporary relief of symptoms, an unpredictable therapeutic window, breathy downtime, and the risk of developing antibodies can be overcome by selective laryngeal adductor denervation-reinnervation surgery (SLAD-R). SLAD-R achieves a selective denervation of the thyroarytenoid muscle. Denervation of the adductor muscles interrupts the aberrant nerve fibers causing the laryngeal dystonia, while reinnervation with the ansa cervicalis nerve may prevent reoccurrence of the dystonia.

Methods: 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.

CASE PRESENTATION

A 46-year-old male presented with ADSD to the laryngology clinic after failure of chemodenervation with botulinum toxin injections in the sternothyroid muscle. The patient underwent a bilateral SLAD-R surgery ten years ago after failure of botulinum toxin injections. The patient’s voice quality during that period was strong, implying successful reinnervation by the ansa cervicalis nerve. Surgical exploration confirmed functional reinnervation as the area presented for the neuronophy.

SURGICAL STEPS

1) Harvesting the Ana Cervicalis

- The area is found over the internal jugular vein. The area is dissected with the use of fine artery clips and is dissected to the level of the internal jugular vein. The area is dissected to the level of the internal jugular vein.

2) Exposure of Intralaryngeal RLN Anatomy

- A trapdoor laryngotomy is performed (Fig 2).

3) Denervation of Adductors

- The adductor nerve is always distal to the PCA, and can be confirmed using laryngostroboscopy. The distal recurrent laryngeal nerve is denervated and elevated to the tie, leaving as much length on the distal stump as possible to allow re-anastomosis. (Fig 7)

4) Reinnervation of the TA muscle with the ana cervicalis

- The adductor nerve is harvested during the endoscopic arytenoidectomy to the level of the cricoid cartilage. The neurorrhaphies are sectioned and post-operatively the patient’s dystonias immediately resolved.

Conclusion: We present a case of demonstrated functional activity of nerves used for reinnervation in SLAD-R surgery for spasmodic dysphonia. Unfortunately in this case, the disease recurred through the aberrant activity of these translocated nerves.

REFERENCES