

# Treatment of Adductor Spasmodic Dysphonia with Selective Laryngeal Adductor Denervation and Reinnervation Surgery

Dinesh K. Chhetri, MD\*, Gerald S. Berke, MD

*Division of Head and Neck Surgery, 62-132 CHS, Head and Neck Surgery,  
UCLA Medical Center, 10833 Le Conte Avenue, Los Angeles, CA 90095, USA*

Spasmodic dysphonia (SD) is a voice disorder characterized by abnormal intermittent spasms of intralaryngeal muscles that result in voice breaks during speech. In the adductor variant of spasmodic dysphonia (ADSD), spasms of the adductor muscles cause strangled voice breaks and a strained-strangled voice quality. In the abductor variant (ABSD), spasms of the posterior cricoarytenoid muscle (PCA) cause breathy voice breaks and a breathy voice quality. Patients with SD typically have no other associated chronic medical problems or handicaps and are highly functioning individuals. The voice breaks lead to a significant difficulty in daily communication. Therefore individuals with SD perceive their voice significantly limits them functionally, physically, and emotionally. Successful treatment of vocal spasms thus leads to a dramatic improvement in the patient's perception of health and social functioning.

The ideal treatment for any disease is a single noninvasive therapy that results in a permanent cure without associated complications. Such ideal treatment does not exist for most medical disorders, and SD is no exception. One main hurdle toward achieving a cure for SD is that the cause and pathophysiology of SD remain unclear. There are no animal models for this disorder. What we know from laryngoscopic and electromyographic exams is that voice breaks in SD are associated with abnormal electrical activity of the laryngeal nerves, resulting in increased muscle movements [1]. SD is thus classified as a focal dystonia that affects the larynx. However, we do

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\* Corresponding author.

*E-mail address:* [dchhetri@mednet.ucla.edu](mailto:dchhetri@mednet.ucla.edu) (D.K. Chhetri).

not know what triggers the onset of SD or how the abnormal neural activities in the recurrent laryngeal nerves (RLN) are generated. Chhetri and colleagues [2] performed a histochemical examination of the lateral cricoarytenoid muscle (LCA) and a morphologic analysis of the adductor branch of the recurrent laryngeal nerve in patients with ADSD and found no obvious neuromuscular abnormalities. Type-II fiber predominance was seen in muscle samples, but this could also be induced by the increased neural activity seen in the pathophysiology of ADSD. Therefore it is likely that the pathophysiologic changes in ADSD are at the level of the central nervous system, and the technological resolution to examine them is unavailable at present.

The voice breaks of ADSD can be understood biomechanically as a mismatch between laryngeal resistance and subglottic pressure, leading to cessation of airflow. The sudden, strong adduction of the vocal folds in ADSD dramatically increases laryngeal resistance that requires matching high subglottic pressure levels for uninterrupted phonation. The lung-thorax system is unable to instantaneously and smoothly generate the increased subglottic pressure required to overcome the sudden increase in laryngeal resistance, and phonatory break ensues until relaxation of the vocal folds leads to decreased resistance and the resumption of airflow. When airflow cessation is not complete but severely reduced, the strained vocal quality is produced.

### **Selective adductor denervation reinnervation surgery**

This article focuses on the surgical management of ADSD by selective laryngeal adductor denervation and reinnervation (SLAD-R). All current treatments for ADSD are designed to reduce the intraglottic adductory force of the larynx. Chemodenervation of the thyroarytenoid (TA) muscles with botulinum toxin (botox) was initiated in the 1980s by Blitzer and colleagues [3] and is now considered the standard of care by many laryngologists. Although this therapy is effective, there are significant disadvantages, including the need for repeated injections, a period of undesirable breathiness after the injection, and the lack of uniform dose-response relationship with this medication. Many patients with ADSD are young and find it difficult to adjust to the potential for lifelong laryngeal injection therapy. However, before the introduction of botulinum toxin, SD was treated mostly with psychotherapy and speech therapy, which were ineffective for this condition. Although Dedo [4] had introduced unilateral recurrent laryngeal nerve section for this disorder in 1976, laryngologists did not embrace this technique because it creates an iatrogenic unilateral vocal cord paralysis and because of studies, for example, by Aronson and Desanto [5], that showed a symptom recurrence rate of up to 64% within 3 years. It was believed that the recurrence of symptoms was the result of a persistent abnormality in the untreated

side and the regrowth of axons into the larynx from the cut nerve stump. Therefore, some otolaryngologists caution against surgery for SD that involves irreversible steps such as the division of the RLN or its branches [6].

SLAD-R surgery for ADSD was developed to avoid the failures of the Dedo operation. Several *in vivo* canine studies performed at the University of California Los Angeles in the late 1980s established the potential for long-term treatment of spasmodic dysphonia symptoms by selective adductor denervation and reinnervation. Green and Berke [7] measured the intra- and subglottic pressures as the RLN was stimulated from low to high, while keeping the superior laryngeal nerve (SLN) stimulation constant from low to high. They noted that RLN stimulation led to a linear increase in both pressures. Interestingly, SLN stimulation resulted in the attenuation of RLN effects on pressure. The RLN was thus established as the correct target for surgery. Bilateral laryngeal denervation was necessary to effectively treat abnormal signals coming to both vocal cords, and reinnervation was added to prevent the regrowth of axons back into the larynx. It was clear that the nerve section had to be performed distal to the PCA branches to avoid bilateral vocal cord paralysis. However, the effect of bilateral adductor denervation on laryngeal biomechanics was unclear. This was studied by Sercarz and colleagues [8] in a canine model, using photoglottography and electroglottography. After bilateral adductor denervation, high levels of subglottic pressure could not be generated even with high levels of RLN stimulation. More importantly, the geometry of glottic vibration was maintained. In the same study, the laryngeal vibration and vocal-fold histopathology were studied 4 months after surgery in two animals that underwent selective reinnervation of the distal TA nerve stump with the ansa cervicalis nerve and were compared with two animals that did not undergo reinnervation. Non-reinnervated vocal cords were characterized by asymmetric and aperiodic vibration. The vocal-fold bulk and tension were maintained in the reinnervated vocal folds. Thus, the importance of laryngeal reinnervation is to maintain glottal bulk and geometry as well as to prevent reinnervation of the laryngeal muscles by axons from the cut adductor nerve stump.

SLAD-R surgery initially targeted the TA muscle alone. As experience was gained with this operation and intralaryngeal microanatomy was further understood, the LCA branch was also routinely observed coming off the distal adductor branch. The LCA branch was then divided routinely. However, the LCA branch is too short and small for reinnervation, and therefore LCA myotomy was added in the late 1990s to prevent reinnervation of the LCA muscle from the proximal adductor nerve stump. LCA myotomy improved the long-term success of the procedure but also lengthened the period of breathiness after surgery. A few patients (usually male) developed permanent breathiness resulting from incomplete posterior commissure closure. Presently, partial LCA myotomy is performed, and no cases of severe or permanent breathiness have occurred with this modification in technique.

### Preoperative evaluation for SLAD-R surgery

The most important consideration for the long-term success of SLAD-R surgery is that ADSD be accurately diagnosed preoperatively. There are no objective diagnostic tests for ADSD. However, experienced clinicians can diagnose it by its typical voice breaks during connected speech and by normal voice qualities during emotional vocalizations such as laughing, crying, and other instances. Botox therapy can be both diagnostic and therapeutic, and the authors often suggest that patients try botox and experience the “botox lifestyle” before surgery. Patients with concomitant tremor are advised that surgery may control the SD symptoms but not the tremor. The patient should be medically cleared by colleagues in internal medicine to undergo a 3-hour procedure under general anesthesia. The main reasons given by patients for seeking surgery are listed in [Table 1](#).

### Surgical technique

The operation proceeds in steps and is repeated on each side of the neck. An endotracheal tube that allows electromyographic monitoring of the recurrent laryngeal nerves (NIM-Response System, Medtronic Xomed, Inc., Jacksonville, FL) is used for intubation. The bed is turned 90° away from the anesthesiologist so that the surgeon can stand at the head of the bed as needed. A shoulder roll is placed to extend the neck. A 10-mg dose of dexamethasone and prophylactic antibiotics are given intravenously before the skin incision is made.

The skin incision is placed on a skin crease along the inferior border of the thyroid lamina. The incisions should extend bilaterally to the midpoint of the carotid triangle, which is bordered by the sternocleidomastoid and the omohyoid muscles, to obtain adequate exposure for identification of the ansa cervicalis nerves. Subplatysmal flaps are elevated superiorly, approximately to the hyoid bone and inferiorly just below the cricoid cartilage.

Table 1  
Reasons for seeking surgery for ADSD

Reasons for seeking surgical intervention	Percentage of patients
Just wanted to cure SD for good	94
Ups and downs of botox	71
Prolonged voice difficulties after botox	52
Botox stopped working	32
Cost of botox	30
Swallowing difficulties after botox	16
Botox never worked	16
Difficulty with traveling to obtain botox	15

### *Identification of the ansa cervicalis nerves*

The ansa cervicalis nerve is an excellent choice for use in laryngeal reinnervation because of its proximity to the larynx and because it is quite active during phonation. Its anatomy has been reviewed elsewhere [9]. It is a cervical motor nerve formed by the junction of two main nerve roots derived entirely from the ventral cervical rami and innervates the infrahyoid strap muscles. The superior (anterior) root is derived from C1, which joins the hypoglossal nerve for a short distance before branching off from this nerve at the level of the origin of the occipital artery, and descends on the lateral surface of the internal jugular vein. The inferior (posterior) root usually arises from the junction of two primary ventral cervical rami, most commonly C2 and C3, and travels posteriorly and deep to the internal jugular vein. A loop is formed at the point of anastomosis of the superior and inferior roots, usually on the lateral surface of the internal jugular vein.

The carotid artery and internal jugular vein lie immediately beneath the fascial tissue filling the carotid triangle. The fascial tissue is dissected in a superior-to-inferior manner and divided. Dissection proceeds until the internal jugular vein is identified. The superior root of the ansa cervicalis nerve can be identified typically at this time running along the lateral wall of the vein. Occasionally this branch is located more anteriorly under the anterior belly of the omohyoid muscle as it courses inferiorly. Retraction of the sternocleidomastoid muscle laterally and the omohyoid muscle medially often further exposes the ansa cervicalis. The ansa cervicalis nerve is exposed from the level of the superior border of the thyroid lamina and is followed distally until a length needed for easy rotation into the larynx is exposed. Dissection often proceeds under the omohyoid muscle to achieve this length. The nerve is then tagged for later use in reinnervation.

### *Laryngotomy*

The strap muscles are divided in the midline from the hyoid bone to the cricoid cartilage. The thyrohyoid muscle is elevated from the thyroid ala using a freer elevator. The attachment of the thyrohyoid and sternothyroid muscles to the inferior border of the thyroid lamina is divided with bipolar electrocautery and then divided sharply. Attention is given to preserve the external branch of the superior laryngeal nerve, which runs along the superior border of the cricothyroid muscle. When the oblique line is reached, its superior and inferior attachments to the thyroid lamina are divided. The posterior border of the thyroid lamina can then be palpated, and a single pronged hook is placed on the posterior lamina to rotate the larynx medially, thus fully exposing the thyroid lamina.

An inferiorly based rectangular, cartilaginous laryngotomy window is cut into the thyroid lamina (Fig. 1). A sagittal saw is used to make the cartilage cuts, although in younger patients this can be accomplished with a knife blade. The posterior cut is made just anterior to the inferior cornu. The

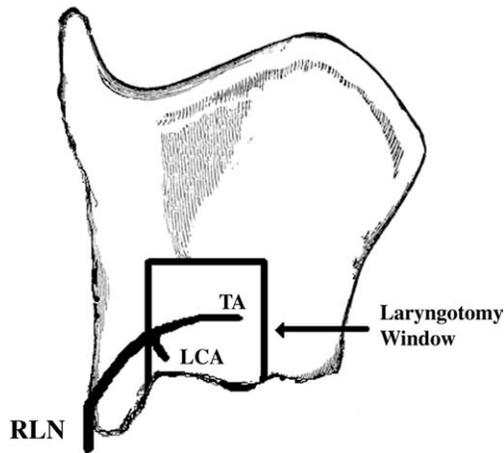


Fig. 1. Normal laryngeal anatomy showing the intralaryngeal course of the adductor branch of the recurrent laryngeal nerve and its relationship to the laryngotomy window.

superior cut is parallel to the inferior border of the thyroid lamina and is placed approximately halfway between the upper and the lower borders of the lamina. The anterior cut is made just anterior to the inferior thyroid tubercle. The cuts can be made through to the inner thyroid perichondrium but not deeper. The cartilaginous window is mobilized with an elevator and rotated inferiorly.

#### *Adductor denervation and lateral cricoarytenoid muscle myotomy*

The intralaryngeal portion of the operation is performed with microinstruments and magnification. The adductor branch runs typically in an oblique course from the posteroinferior corner of the window toward the midbelly of the TA muscle anterosuperiorly (see Fig. 1). Small blood vessels and adipose tissue often surround the nerve along its course to the muscle. Occasionally, the nerve lies deeper in the surgical bed, between the bellies of the TA and the LCA. A 3-0 silk suture is used to tie the nerve close to its insertion into the TA muscle. It is important to leave an adequate distal stump to allow unencumbered neural anastomosis to the ansa cervicalis nerve. The nerve is divided distal to the suture, and the nerve is retracted posteriorly with the suture and freed from its attachments all the way to the posterior border of the laryngotomy window. The branch to the LCA is seen typically during this maneuver and is divided. The suture is then threaded through a French-eye needle, and the nerve is sutured outside the larynx to the posterior lamina (Fig. 2). Partial LCA myotomy is then performed at the midbelly using microscissors. The present authors typically cut less than 50% of the thickness of the LCA muscle.

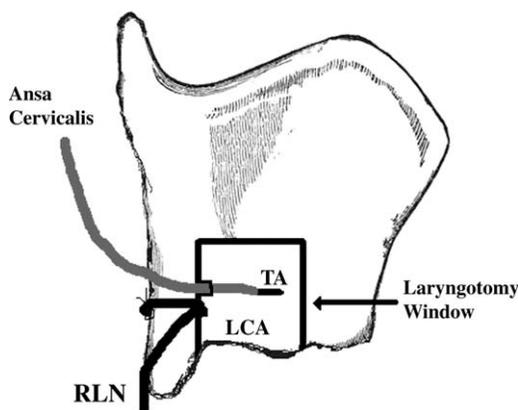


Fig. 2. Neural anatomy after selective adductor denervation and reinnervation. The adductor branches to the TA and the LCA are cut and exteriorized. The ansa cervicalis nerve is anastomosed to the distal adductor nerve stump.

### *Adductor reinnervation*

The previously tagged ansa cervicalis nerve is cut distally, passed under the strap muscles, and brought to the laryngotomy window. The inferior root usually requires division for adequate rotation of the nerve. Typically, the nerve to the sternohyoid or sternothyroid is used. Epineurial anastomosis is performed using 8-0 nonabsorbable sutures between the ansa cervicalis and the distal stump of the adductor nerve branch (see Fig. 2). The laryngotomy window is replaced after a  $2 \times 2$ -mm piece of thyroid cartilage is removed from the posterior border to make an entryway for the ansa cervicalis nerve to enter the larynx. The skin incision is closed over a Penrose drain.

### **Postoperative course and surgical outcome**

Intravenous dexamethasone and antibiotics are continued while the patient recovers in the surgical ward. The Penrose drain is removed the next day. Dysphagia is common during the first postoperative day, with most difficulty experienced with swallowing liquids. A dysphagic diet is given, and the patient is taught to thicken liquids as needed. Most patients' swallowing improves by postoperative day 2, and they are discharged on a medrol dose pack and antibiotics. The voice is typically breathy, similar to the voice after a large dose of bilateral TA injection.

The postoperative laryngoscopic examination should reveal normal abduction and nearly complete adduction at the vocal processes but a large midcord glottal gap. Breathiness lasts between 3 and 6 months, at which time the glottal closure is complete, and reinnervation presumably occurs. Further improvement in vocal strength occurs between 9 and 12 months.

The long-term results of SLAD-R have been very good. The initial surgical update on 21 patients was published in 1999 [10]. Nineteen of 21 patients had absent to mild voice breaks postoperatively. A recent long-term follow-up study of 81 patients at an average follow-up 49 months showed that 83% of the patients had significantly improved Vocal Handicap Index 10 scores, and 91% of patients indicated that their voice after surgery was more fluent than after botox therapy [11].

Patients who have suboptimal results after SLAD-R can be divided into two groups. The first group consists of patients with a recurrence of dystonia, a true failure of the operation. This has occurred in approximately 11% of the present authors' patients. When symptoms recur, they recur in approximately 12 months. These authors have not witnessed any recurrence of symptoms more than 24 months after surgery. Recurrent symptoms do respond to repeat botox injections. The second group consists of patients who have permanent breathiness, a complication of the operation. In the latest follow-up voice evaluation, the rate of moderate breathiness was approximately 14%, with approximately 6% of patients experiencing severe breathiness caused by vocal cord paresis. Severe breathiness appears to be related to the degree of LCA myotomy and resultant posterior glottal chink. Males seem to tolerate posterior glottal chinks much less than women. However, with conservative LCA myotomy, no cases of permanent vocal-fold paresis have occurred.

The voice after SLAD-R surgery is normal in over 75% of patients, with the rest having some degree of dysphonia [11]. The latter group includes some limitation in the upper and lower limits of pitch and loudness. Some patients continue to exhibit abnormal compensatory behaviors, although their voice is fluent. Professional singers will not be able to return to a singing career. To maintain long-term voice quality, excellent vocal hygiene and control of laryngopharyngeal reflux are mandatory. In the present authors' experience, the symptoms of ADSD are generally so disabling and disruptive to patients' daily communication that most find their fluent voice after surgery much more useful and preferable to the "roller coaster" ride with botulinum toxin therapy, despite the vocal limitations described above. SLAD-R is the only surgical treatment for ADSD with demonstrated long-term control of symptoms, and therefore it continues to play an important role in our management of ADSD.

## Summary

SLAD-R is an alternative therapeutic modality in the treatment of ADSD. It provides long-lasting control of symptoms, and patient satisfaction is very high. The majority of patients achieve a fluent voice, with a range of breathiness from none to minimal. Patients should be counseled about the possibility of recurrent symptoms and permanent postoperative breathiness. The latter complication has been minimized by conservative LCA myotomy. A prospective study is underway to further understand this surgery.

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