



Selective laryngeal adductor denervation-reinnervation surgery for spasmodic dysphonia

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SLAD-R;
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Surgical treatment of adductor-type spasmodic dysphonia provides relief from the disabling voice breaks that require repeat botulinum toxin injections. The selective laryngeal adductor denervation-reinnervation surgery is described here. Key points discussed include patient selection, pertinent laryngeal and neural anatomy, and postoperative course.

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Adductor spasmodic dysphonia (ADSD) is a task-specific neural disorder in which inappropriate tension within the intrinsic laryngeal muscles produces audible voice breaks during speech. The etiology of the muscle dysfunction is still unclear. Laryngeal muscle biopsies did show predominantly type II fibers, consistent with remodeling from repeated spasms.¹ Increased somatosensory cortex activation was found on functional magnetic resonance imaging during voice symptoms,² implicating this system in the disease. Delineating primary pathology from secondary changes remains a challenge in this complex disorder.

Despite suggestions that ADSD is a bilateral and central disorder, unilateral and peripheral treatment can dramatically improve the disease signs. The initial surgical treatment involved recurrent laryngeal nerve (RLN) division, eventually modified with measures to reduce synkinetic reinnervation.^{3,4} Short-term enthusiasm dissipated as a high relapse rate emerged, because of either contralateral brain input or regrowth of ipsilateral axons carrying pathologic signals. Botulinum toxin injection to weaken the laryngeal adductor muscles quickly became the treatment of choice after its introduction in the 1980s⁵ and can be effective even when injected unilaterally.⁶ Temporary chemical denervation with botulinum toxin remains the most common treat-

ment, but poses significant drawbacks for some patients. Variable voice quality between injections, inconsistent dose response, and difficulty accessing health care for frequent injections have all been cited by patients as reasons for seeking an alternate solution.⁷

Building on the earlier surgical treatments, selective laryngeal adductor denervation-reinnervation (SLAD-R) surgery was developed to treat the symptoms of adductor-type spasmodic dysphonia.⁸ For a synopsis of *in vivo* studies leading to the SLAD-R technique, the reader is referred to Chhetri and Berke 2006.⁹ SLAD-R introduced two critical advancements to the RLN division technique. First, the terminal nerve branch to the posterior cricoarytenoid (PCA) muscle is spared. Anatomic study reveals that this essential abductor branch of the RLN consistently arises proximal to any adductor branches.¹⁰ As such, it is possible to selectively divide the laryngeal nerve distal to the PCA branch, preserving abductor function while denervating the adductor muscles that mediate voice breaks. Maintaining PCA function allows the procedure to safely be performed bilaterally. Second, the distal laryngeal adductor nerve branch is reinnervated with a branch of the ansa cervicalis (AC) nerve. It is well understood that a severed nerve secretes growth factors and cytokines attracting new axonal growth. Placing a different motor nerve in closest proximity to the distal stump channels nerve regeneration through those fibers, preventing regrowth of the original RLN fibers carrying pathologic signals. Additionally, tone provided by the motor input maintains muscle bulk and prevents voice breathiness.

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Indications and contraindications

Patients with adductor-type spasmodic dysphonia who desire relief of symptoms without repeated botulinum toxin injections are candidates for this surgery. A trial of botulinum toxin injection is very strongly encouraged. This trial presents to patients the less invasive treatment option first. It also introduces patients to the breathy voice that is expected temporarily after surgery and to the degree of voice improvement that can be anticipated. It is expected that the speech fluency improves with surgery, but singing ability is quite variable. Patients who must be able to sing should not undergo SLAD-R. Abductor and mixed spasmodic dysphonia are not amenable to this surgery because the breathy spasms may become more pronounced on adductor denervation.

Preoperative workup involves assessment of medical suitability for an elective surgery under general anesthesia. Severe chronic lung disease is a relative contraindication because aspiration may occur temporarily during the postoperative period. Advanced age is a relative contraindication, as nerve regeneration is known to be impaired in elderly patients. Laryngeal stroboscopy is performed to rule out other causes of dysphonia. Previous laryngeal surgery, including type II thyroplasty, is a relative contraindication because the resultant scar tissue complicates nerve identification and regeneration. Previous neck surgery, such as neck dissection or anterior cervical spine approach, may preclude SLAD-R if it is suspected that the AC nerve was sacrificed.

Laryngeal tremor commonly coexists with spasmodic dysphonia.¹¹ Tremor must be sought and the degree of its impact on voice quality carefully assessed to appropriately counsel patients considering surgery. The SLAD-R surgery does not eliminate tremor, although anecdotally some patients have found lessened tremor severity after SLAD-R. More widespread dystonias, such as the cervicofacial dystonia of Meige syndrome, must be approached with great caution, as spasms of the strap muscles may be transferred to the larynx via the AC nerve after regeneration.

Surgery is typically performed on both sides of the larynx. For some patients with mild ADSD, performing the operation on only one side of the larynx permits a faster recovery to swallowing and voice function. However, the relapse rate is higher for the unilateral procedure than for bilateral surgery, in concordance with the central neurologic etiology of the disease. Patients electing unilateral surgery are counseled that a staged procedure to treat the opposite side may be required if spasms recur.

Technique

SLAD-R is performed under general anesthesia with a size 5 or 6 endotracheal tube with electromyogram laryngeal nerve monitoring. The neck is extended with a shoulder roll. For bilateral surgery, a 6-cm horizontal midline neck incision is drawn over the lower half of the thyroid cartilage. The incision may be extended slightly if needed in large

necks to access the sternocleidomastoid muscle (SCM) as the lateral border of dissection. For unilateral surgery, the incision exposes the entire ipsilateral thyroid ala and extends to the SCM. Lidocaine with epinephrine is injected along the incision for hemostasis, and preoperative antibiotics and dexamethasone are administered.

The skin and platysma are incised, and subplatysmal flaps are raised. AC nerves are identified before exposing the larynx (Figure 1). Fascia is divided at the anterior SCM border, and the ansa loop is found over the internal jugular vein. The ansa branch to the sternothyroid or sternohyoid muscle is selected and neurolysed far enough to provide adequate rotation to the larynx.¹² Vagus nerve and carotid artery are avoided.

Strap muscles are divided in the midline and retracted laterally to expose the larynx. Thyrohyoid and sternothyroid muscle attachments to the lower border of the thyroid cartilage are divided with cautery, avoiding the cricothyroid muscle and joint and the external branch of the superior laryngeal nerve. A freer elevator or scalpel raises the inferior constrictor muscles from the oblique line on the thyroid cartilage, with the assistance of a hook on the posterior edge of the ala to rotate the larynx. The entire lateral thyroid ala is exposed and pyriform sinus mucosa identified.

A trapdoor laryngotomy window is drawn on the thyroid ala, with the anterior limb at the inferior tubercle, the posterior limb just anterior to the inferior cornu, and the superior limb crossing the oblique line below the vertical midpoint of the cartilage (Figure 2). The window is cut with a sagittal saw and retracted downward. Inner perichondrium, if not elevated with the cartilage, is removed with microscissors under loupe magnification to expose the intrinsic laryngeal muscles.

The RLN is identified in its intralaryngeal course by fine dissection. Exploring the intralaryngeal anatomy in a cadaveric human larynx is a worthy pursuit before performing this step in a patient. The RLN enters the larynx at the cricothyroid joint, gives off a branch coursing posteriorly to the PCA, and then travels superiorly and anteriorly toward the midpoint of the thyroarytenoid (TA) muscle. Identifying the nerve travelling in the appropriate direction and within the cartilage window ensures that it is distal to the PCA branch. Small branches are first given to the interarytenoid (IA) and then the lateral cricoarytenoid (LCA) muscles. The IA nerve branch lies far posterior and is not seen. The LCA nerve branch is very fine and may not be identified within the window. The terminal nerve then supplies the TA muscle (Figure 3). The adductor nerve can be confirmed by applying an electromyogram stimulus at 0.5 mA. The nerve is tied tightly and divided distal to the tie, leaving as much length on the distal stump as possible to allow reanastomosis. The axon can be identified in the distal stump, which is handled gently in preparation for reanastomosis. A French-eye needle is attached to the tie and used to secure the proximal nerve stump out of the larynx by suturing it to perichondrium outside the window. This prevents reinnervation by the pathologic RLN fibers.

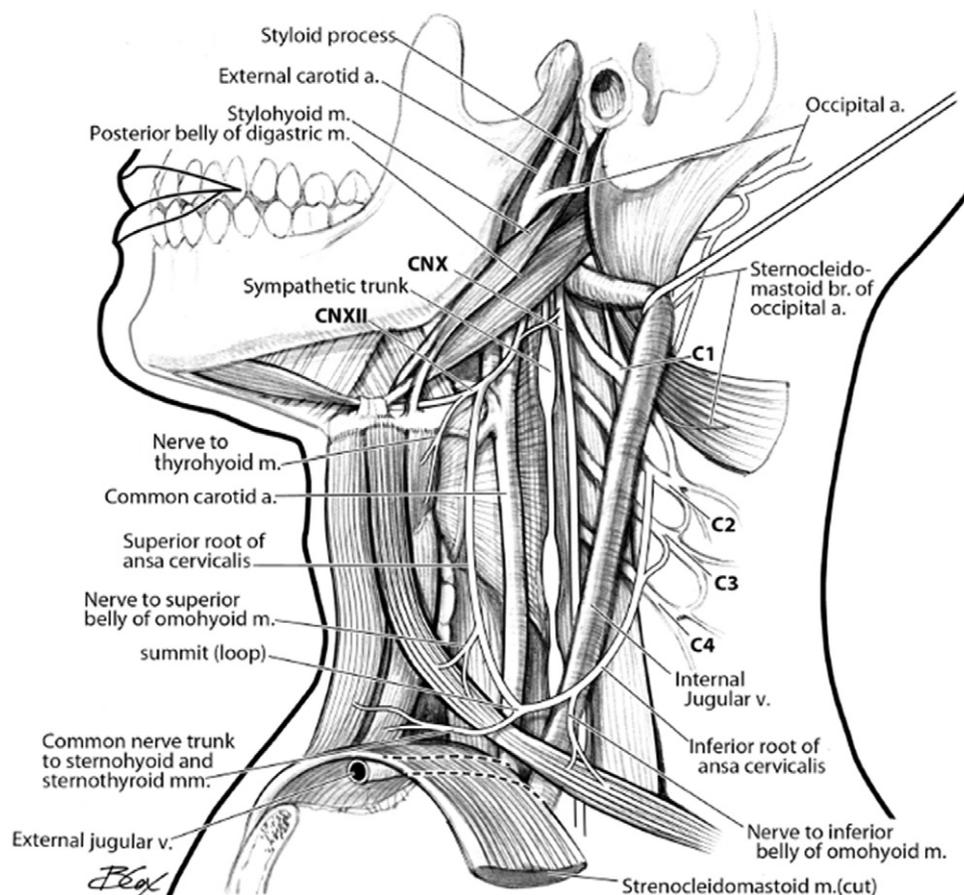


Figure 1 Anatomy of the left ansa cervicalis nerve. (Adapted with permission from Chhetri and Berke.¹²)

LCA muscle fibers are seen immediately medial to the divided nerve. About half of these fibers are divided with microscissors to reduce the adductory force they may generate. The LCA nerve branch is typically not reanastomosed with AC fibers because it is very fine and variable in its branching position. Therefore, the myotomy serves to prevent spasm recurrence in case of RLN reinnervation of the LCA muscle. Excessive myotomy is avoided because it can produce prolonged breathiness, especially in males.

The intended AC nerve branch is cut low in the neck, preserving the superior ansa hypoglossi root. Cutting the omohyoid branch increases the rotational length. If multiple cervical rootlets are identified joining the inferior ansa root, one may be sacrificed to ease rotation to the larynx. The free nerve end is tunneled under the strap muscles to reach the laryngeal trapdoor. The ansa is anastomosed to the distal laryngeal adductor nerve branch without tension using 3 epineurial sutures of 8-0 nylon. The anastomosis can be performed with loupe magnification, although some surgeons prefer using a microscope.

A rongeur is used to remove a small corner of the trapdoor to allow passage of the ansa nerve into the larynx. The trapdoor is then closed and secured with prolene suture through the perichondrium (Figure 4). A Penrose drain is placed in the lateral neck compartment and beneath the straps, which are closed in the midline. Platysma and skin are closed and a pressure bandage placed.

Postoperative course and complications

Patients are admitted to the hospital ward until adequate oral intake is achieved, usually 1 or 2 nights. The drain is removed the day after surgery. Intravenous steroids are continued for 1 or 2 days postoperatively to minimize dysphagia and airway edema. Acid reflux is aggressively controlled.

Postoperative dysphagia is expected and is because of multiple factors, including edema from larynx rotation, strap muscle impairment from division of AC nerve, elevation of inferior constrictor muscles, and impaired glottic closure reflex. All patients are given a dysphagia diet with thickened liquids until their swallowing is clinically improved, typically within a few days. Permanent dysphagia or aspiration has not occurred in our patients.

The voice is free of spasms and voice breaks immediately postoperatively. The voice is very breathy postoperatively, consistent with a large glottal gap. Some adduction of the vocal processes occurs from IA action (because this nerve is preserved) and sometimes from residual LCA muscle if that nerve branch is not divided. The breathiness improves 4 to 6 months postoperatively as the regenerating nerve axons reach their target muscles. Rarely a breathy voice may persist. This can occur because of excessive LCA myotomy producing a posterior glottal gap or from failure of nerve regeneration with resultant TA atrophy.

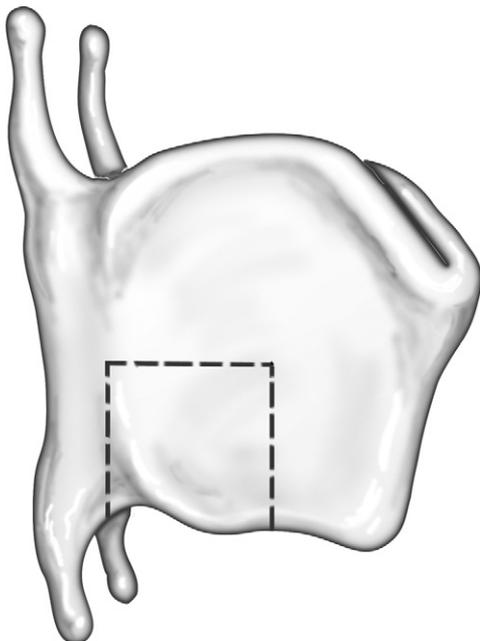


Figure 2 Lateral oblique view of right thyroid cartilage. Hashed lines show the trapdoor laryngotomy window, crossing the oblique line after removing inferior constrictor muscle attachments. Posterior limb is just anterior to the inferior cornu, anterior limb passes through the inferior tubercle, and superior limb is below the vertical midpoint of the cartilage.

Voice breaks may recur if the AC regeneration is impaired, allowing RLN reinnervation. To prevent this, we limit patients' age to <70 years and meticulously approximate epineurium

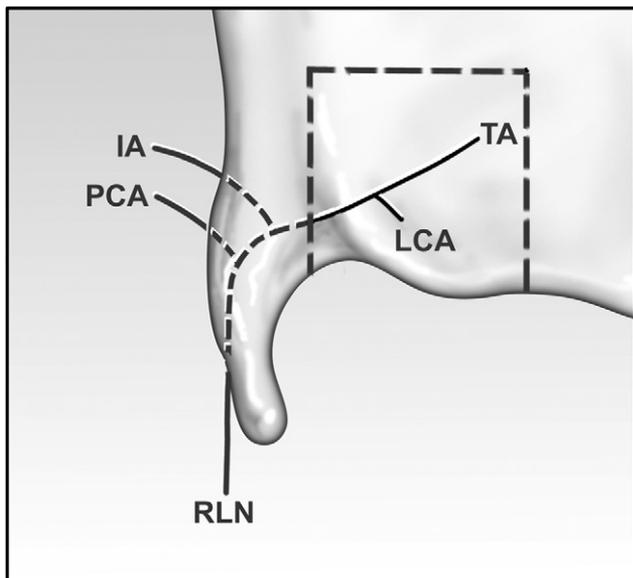


Figure 3 Intralaryngeal course of the right recurrent laryngeal nerve (RLN). Thyroid cartilage laryngotomy window is drawn with long hashed lines. Short dashed lines represent nerves behind cartilage. Characteristic oblique anterosuperior course of the adductor nerve within the window is shown, destined for thyroarytenoid muscle (TA) fibers. Other branches to the posterior cricoarytenoid (PCA) and interarytenoid (IA) muscles are not encountered during the dissection. Lateral cricoarytenoid (LCA) branch may be seen, although it is short and fine.

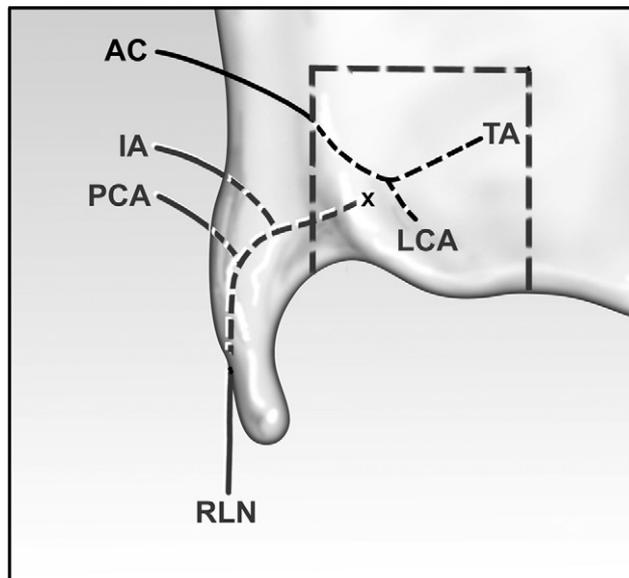


Figure 4 Postoperative arrangement of laryngeal nerves, after closure of laryngotomy trapdoor (outlined with long hashed lines). Ansa cervicalis (AC) enters through the cartilage window and anastomoses the distal stump of the adductor branch serving thyroarytenoid muscle (TA) and lateral cricoarytenoid (LCA) muscles. The proximal adductor branch from the recurrent laryngeal nerve (RLN) is divided and secured out of the larynx. The branches to posterior cricoarytenoid (PCA) and interarytenoid (IA) are unaltered.

during the anastomosis. In one patient with more generalized dystonia, the AC nerve mediated delayed recurrence of voice breaks with concurrent strap muscle spasm. Either of these conditions causing recurrent dysphonia can be treated with botulinum toxin injection.¹³ Should patients desire a longer-lasting solution, TA myotomy reduces the aberrant adductory force, thereby reducing the intrusion of the voice breaks. In the case of generalized spasms in the regenerated AC distribution, severing the ansa input to the larynx eliminated voice breaks, although the strap muscle spasms continued.

Conclusions

SLAD-R is a safe and effective treatment for the voice symptoms of ADSD. Success depends on proper patient selection by making an accurate diagnosis. Critical surgical steps include AC nerve identification, adequate laryngeal rotation for laryngotomy window placement, adductor nerve branch identification, LCA myotomy, and TA reinnervation with the AC nerve. All patients are counseled to expect short-term postoperative dysphagia and several months of breathy voice. A smooth and strong voice is expected after nerve regeneration occurs. Long-term patient satisfaction is excellent.¹⁴

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