# Laryngeal Surgery as It Pertains to the Thoracic Surgeon

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**KEYWORDS**

• Laryngotracheal stenosis • Posterior glottic stenosis • Laryngeal surgery • Intubation injury • Vocal cord paralysis • Endoscopic airway • Tracheal reconstruction

**KEY POINTS**

- Laryngeal function is critical to overall airway functionality.
- Laryngeal videoendoscopy is the mainstay of laryngeal evaluation, additionally aided in some cases by operative examination or laryngeal electromyography.
- Direct operative microlaryngoscopy facilitates management of laryngeal disease via multiple complementary techniques.
- Complex laryngeal or laryngotracheal stenosis often require reconstructive surgical approaches distinct from tracheal or cricotracheal resection.
- Even successful airway reconstruction surgery can generate short- and long-term laryngeal functional impairments.

## BACKGROUND

### *Laryngeal Physiology*

Understanding laryngeal anatomy is crucial for airway surgeons, both in its normal function and role in respiration, as well as its potential for development of respiratory pathology. The larynx is an essential component of the human airway through its role in respiration, phonation, and deglutition. In the healthy larynx, these 3 discrete functional elements converge to promote airway protection via closure, and clearance via cough.[1,2] Achieving these functions requires a complex system of motor and sensory innervation.

All intrinsic muscles of the larynx except 1 are innervated by the recurrent laryngeal nerve (RLN), a branch of the vagus nerve (CN X). This includes the paired thryoarytenoid (TA) and lateral cricoarytenoid muscles (LCA), as well as the unpaired interarytenoid (IA) muscle, all of which contribute to laryngeal adduction. The paired posterior cricoarytenoid muscles (PCA) are also innervated by the RLN, and they manage laryngeal abduction (Fig. 1). The sole muscle with alternative innervation is the cricothyroid (CT), which influences vocal fold lengthening and pitch elevation and is innervated by the external branch of the superior laryngeal nerve (SLN), also a branch from CN X. The internal branch of the SLN provides the majority of sensory innervation to the larynx and serves as the afferent limb for protective reflex pathways including cough and laryngospasm.

Adduction and abduction of the vocal folds also relies on movement of the cricoarytenoid joints (CAJs). The joint allows for a 3-dimensional rocking motion with movement of the vocal folds in the axial, sagittal, and coronal planes. As the PCA muscles contract, the muscular process of the arytenoid (attachment point of PCA) is pulled

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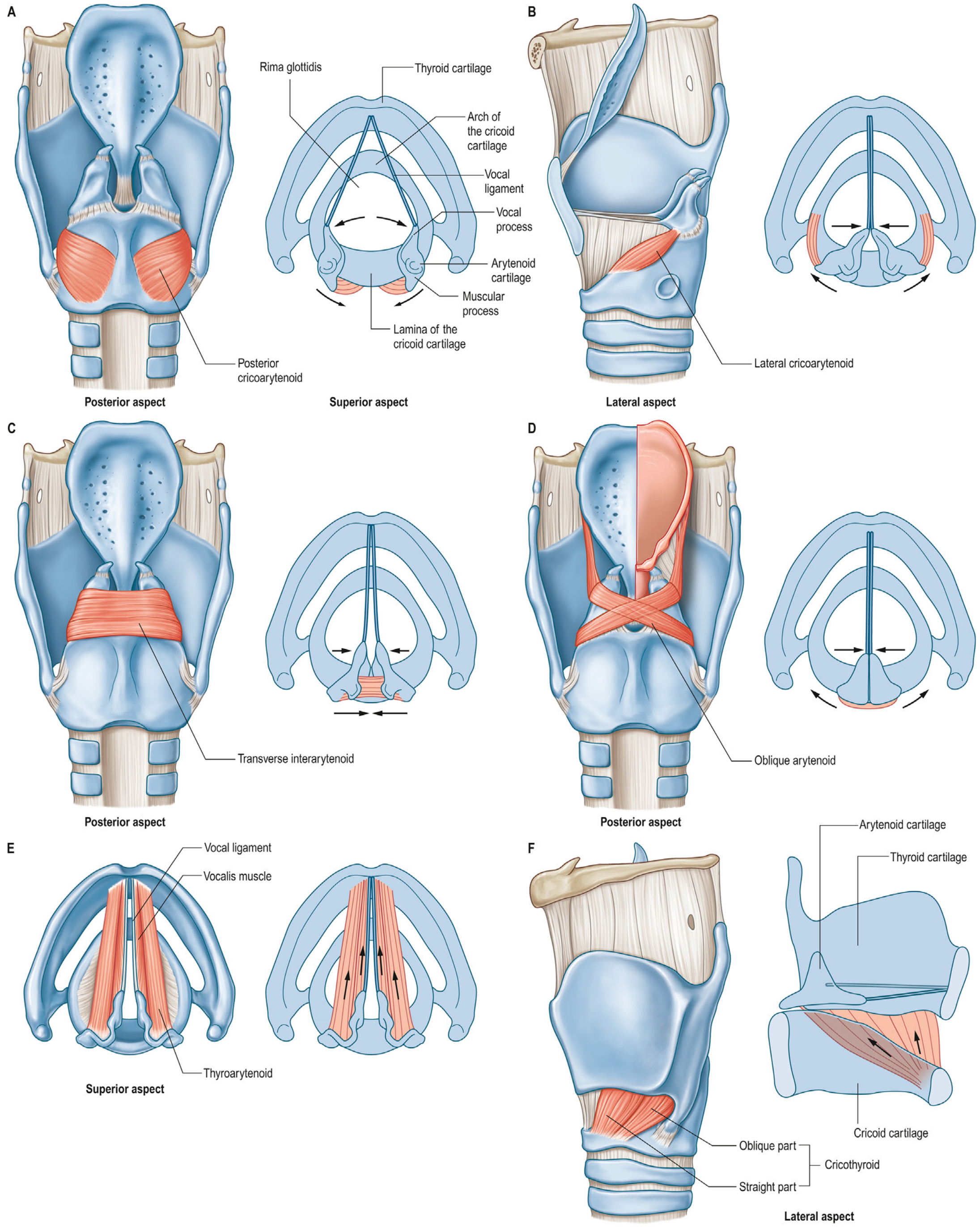


**Fig. 1.** Laryngeal cartilages, muscles, and mechanisms of movement. (*A*) shows poterior coronal view highlighting paired posterior cricoarytneoid muscles resonsible for aBduction (opening the larynx). (*B*) shows sagital section. (*C*) depicts coronal view of transverse interarytenoid. (*D*) shows oblique arytneoid. (*E*) provides axial view showing thyroarytenoid muscles. (*F*) shows coronal view of cricothyroid muscles. (Susan Standring, Gray's Anatomy International Edition, Chapter 41, 717-734. 2021 Elsevier Ltd.)

posteriorly and caudally. As the arytenoid rocks, the vocal process (attachment point of TA) moves upward and laterally, resulting in abduction of the vocal folds and opening of the laryngeal airway.[3,4]

The LCA, IA, and TA muscles work collectively to close the glottis during speech, cough, and Valsalva. The LCA pulls the muscular process of the arytenoid cartilage anteriorly and caudally, moving the vocal process into a more medial position and thereby closing the glottis.[3] Free motion of the CAJ in 3-dimensions is necessary for complete abduction and adduction (**Fig. 2**).

During normal phonation, the vocal folds are adducted to the point of gentle closure. Pulmonary airflow generates a buildup of subglottic pressure until that pressure overcomes the closure force at the glottis and displaces the vocal fold surfaces laterally. As the air column travels past the vocal folds and they remain adducted, a vertical wave is created, traversing the relatively vertical medial surface of the vocal fold. Precise control of glottic closure and airflow maintains a consistent wave, which is then perceived as sound.[5] The vocal fold surface vibrates due to its structural makeup, with a relatively elastic surface layer (superficial lamina propria), which moves freely over a more fibrous internal layer (vocal ligament).[6]

Laryngeal sensation is essential for control of protective reflexes, as well as normal breathing and voice use. The larynx has sensory receptors that respond to air pressure, airflow, joint motion, and noxious stimuli. The larynx is more richly innervated with sensory receptors than even the lungs, without adjustment for surface area.[7] Sensory information travels via the SLN afferent fibers to the nucleus solitarius, where it is then communicated to various parts of the brain.[8]

### *Laryngeal Airflow and Aerodynamics*

The larynx provides dynamic modification of airflow by variation of resistance via adduction and abduction. The aerodynamics are governed by the physics of fluid dynamics. *Bernoulli's principle* describes that for a moving fluid, if the velocity is increased, the static pressure will decrease. In a specific instance of the same phenomenon, the *Venturi effect* defines that as a fluid moves through a narrowed segment, the fluid velocity increases, and pressure decreases. In this way, air moving through a narrowed airway segment will travel faster and generate negative pressure, which can influence closure or collapse depending on the compliance of the tissue. *Poiseuille's law* determines that resistance is inversely related to the radius of the airway to the fourth power. For example, if the radius of the airway is reduced by a factor of 2, the resistance would increase by 16 times.

In these ways, the constant changes in glottic aperture can modulate velocity, pressure, and resistance. In the axial plane, the glottis is not round but rather roughly triangular. The vocal folds join anteriorly at the anterior commissure, and the membranous vocal fold ends posteriorly at the vocal process of the arytenoid, where the TA muscle inserts. Posterior to the vocal processes, IA mucosa makes up the posterior boundary of the glottic airway. Because of pressure differentials and the wider posterior airway, the large majority of airflow occurs in the posterior glottis.[9]

### *Laryngeal Pathology*

Various pathologies can inhibit airflow at the level of the larynx. Some of the most problematic

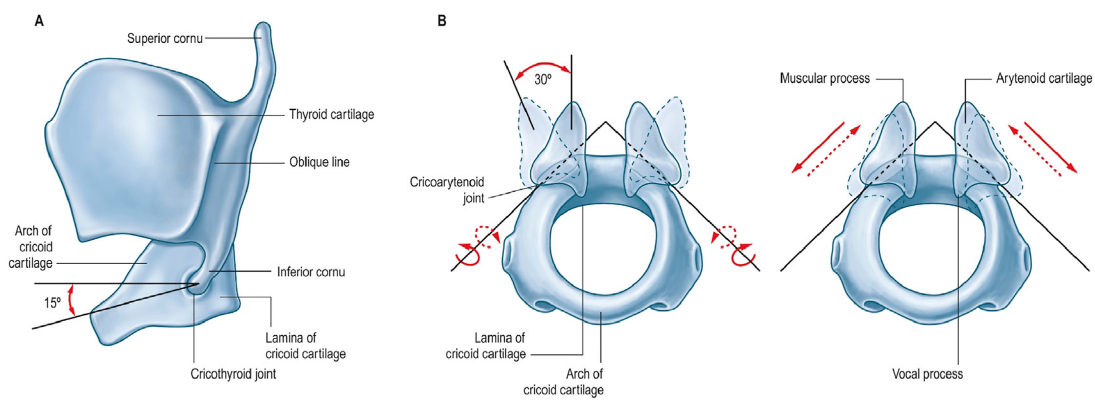


**Fig. 2.** (*A*) Lateral view of the cricothyroid joint showing articulating relationship between thyroid and cricoid cartilage. (*B*) Anterosuperior view of the cricoarytenoid joint, deomonstrating paired of synovial joints between the facets on the lateral parts of the upper border of the lamina of the cricoid cartilage and the bases of the arytenoids. Two movements occur at this joint. The first is rotation of the arytenoid cartilages at right angle to the long axis of the cricoid facet. There is also a gliding movement, by which the arytenoids approach or recede from one another, the direction and slope of their articular surfaces imposing a forward and downward movement on lateral gliding. The movements of gliding and rotation are associated, ie, medial gliding occurs with medial rotation and lateral gliding with lateral rotation, resulting in adduction or abduction of the vocal folds respectively. (Susan Standring, Gray's Anatomy International Edition, Chapter 41, 717-734. 2021 Elsevier Ltd.)

include vocal fold paralysis and posterior glottic stenosis (PGS). Vocal fold paralysis can be caused by a variety of insults including surgical injury to the RLN, surgical injury to CN X, cerebrovascular disease affecting CN X, neoplastic compression or invasion of the RLN, trauma, and idiopathic/viral etiologies. Unilateral vocal fold paralysis (UVFP) does not often cause symptomatic dyspnea, but it does create interruptions in glottic airflow that can be sensed by the patient.[10,11] Bilateral vocal fold paralysis (BVFP) does often present with symptomatic airway obstruction, even to the point of acute respiratory failure.

BVFP due to bilateral neurologic compromise typically presents as inability to adduct or abduct the vocal folds, as both the adductors and abductors are innervated by the RLN. While presentation may be acute, some patients initially tolerate the limited airway due to the vocal folds being in a lateral, flaccid position, and develop dyspnea when partial reinnervation or synkinetic reinnervation moves the vocal folds to a more midline position.[12] BVFP is readily diagnosed with flexible laryngoscopy (**Fig. 3**).

PGS results most often from iatrogenic trauma to the posterior larynx during prolonged endotracheal intubation. The endotracheal tube passes over the base of tongue, turns caudally, and then transverses the glottis. Due to the natural curvature of the upper airway and gravitational posterior force from the tongue, the tube is pressed into the posterior larynx. This can create pressure injury, especially in the thin mucosa overlying the vocal processes of the arytenoids (**Fig. 4**). The mucosal ulceration can progress to prolonged inflammation, development of granulation tissue, and fibrosis that creates mucosal scarring including ankylosis of the CAJs.[13–16] The PGS can vary in pattern and severity. A classification system by Bogdasarian describes Classes I to IV. Class I includes mucosal scar band with patent aperture posteriorly. Class II is complete mucosal scarring without joint ankylosis. Class III includes complete mucosal scarring and unilateral CAJ ankylosis. Class IV adds ankylosis of both joints[17] (**Table 1**). This system is qualitatively useful but has not been rigorously associated with clinical outcomes.

PGS, especially with joint ankylosis, can cause vocal fold motion impairment. These scenarios are distinct from vocal fold immobility due to paralysis, although they share some identification and management strategies.

**Fig. 3.** Flexible laryngoscopy image demonstrating vocal folds fixed in the midline, in the setting of bilateral vocal fold paralysis. (*Data from* Tibbetts, K.M., Simpson, C.B. Adult Bilateral Vocal Fold Paralysis. Curr Otorhinolaryngol Rep 9, 365–372 (2021). https://doi.org/10.1007/s40136-021-00359-1.)

## *Assessment of Laryngeal Function*

Laryngeal motion abnormalities should be considered with high index of suspicion, especially after surgical dissection near the course of the RLN in thyroidectomy, central neck dissection, open esophageal procedures, upper lobectomy, and cardiac or cardiovascular surgery. While UVFP most often presents with breathy, weak voice, limited cough, and aspiration of liquids, BVFP can present with airway obstruction and stridor. PGS can present similarly to BVFP.

Laryngeal videoendoscopy is used to examine vocal fold anatomy and gross function. This can be accomplished via rigid technique with a 70-degree or 90-degree scope passed transorally. It is more often performed via flexible transnasal technique, after topical anesthesia and decongestion of the nasal cavity. The examiner establishes a real-time view of the larynx and instructs the patient through phonatory and breathing tasks. Vocal fold mobility and RLN function is assessed by motion of the arytenoid complex. If motion is asymmetric or incomplete, the examiner can note the side and the position of the abnormal arytenoid complex. Alternation of nasal sniff and a vocal is useful to assess abduction and adduction. Observation of pitch glide from low to high may reveal external branch SLN injury.[18]

Findings of bilateral vocal fold immobility on laryngoscopy examination can be further investigated, as it may not be possible to differentiate between neurogenic paralysis (BVFP) and complete joint fixation in PGS. Cross-sectional imaging via computed tomography can be useful to evaluate the integrity of the cricoid cartilage and CAJ.

In these cases, operative endoscopy is invaluable for the ability to palpate the CAJ and determine

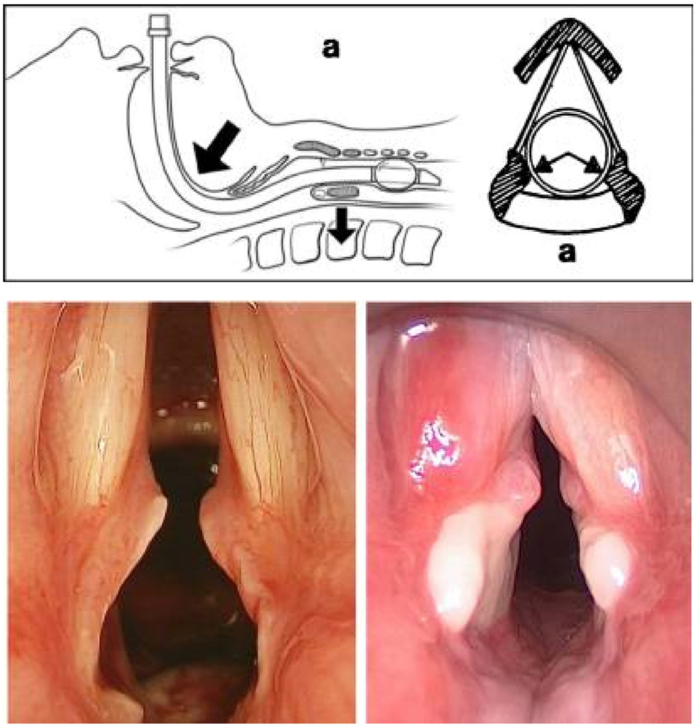


**Fig. 4.** In orotracheal intubation, the weight and pressure of the tongue, as well as the angle of the endotracheal tube exert force on the posterior glottis, including the thin mucosa overlying the vocal processes of the arytenoids. In some cases, this can lead to ischemia, necrosis, mucosal ulceration, and inflammatory remodeling. (*a*) demonstates pressure on the posterior laryngeal mucosa from an endotracheal tube resulting from tongue base pressure (*Data from* Shinn JR, Kimura KS, Campbell BR, Lowery AS, Wootten CT, Garrett CG, Francis DO, Hillel AT, Du L, Casey JD, Ely EW. Incidence and outcomes of acute laryngeal injury after prolonged mechanical ventilation. Critical care medicine. 2019 Dec 1;47(12):1699-706.)

mobility. After initiation of neuromuscular blockade, the patient is placed in suspension with an operating laryngoscope.[19] Under magnified visualization with operating telescope or microscope, the surgeon can palpate the vocal fold and note movement of the CAJ complex. A mobile joint should rock freely of the contralateral side with minimal force. Operative endoscopy also allows for complete visualization of the posterior glottis, which is typically not possible with awake laryngoscopy. With the same approach, the surgeon can visualize the subglottis and trachea to evaluate for any additional levels of injury or abnormality.

**Table 1**
**Staging systems for Posterior Glottic Stenosis**

| Bogdasarian and Olson Staging: Posterior Glottic Laryngeal Stenosis | |
|---|---|
| Stage I | Adhesion of the vocal process |
| Stage II | Scarring of the interarytenoid plane and internal surface of the posterior cricoid lamina |
| Stage III | Unilateral cricoarytenoid joint ankylosis |
| Stage IV | Bilateral cricoarytenoid joint ankylosis |

In the case of iatrogenic PGS, it can be diagnostically useful to obtain a tissue culture during an initial procedure. Mucosal barrier disruption, bacterial displacement, and subsequent inflammatory cytokine cascades have been shown to participate in the pathophysiology of PGS and iatrogenic subglottic stenosis (SGS).[20] Culture directed antibiotics may aid in reduction of the inflammatory response in these cases.

Laryngeal electromyography (EMG) is used in some centers for diagnosis of vocal fold paralysis or paresis. It is performed via transcutaneous placement of needle electrodes in the TA muscles, through the CT space. It can differentiate between structural (PGS or CAJ ankylosis) or neuromuscular etiologies of immobility. It is dependent on the skill and experience of the performing physician but can be useful when taken in context with other clinical findings.[21–24]

## DISCUSSION

### *Techniques for Addressing Isolated Laryngeal Stenosis*

Endoscopic management of laryngeal stenosis begins with operative diagnostic evaluation and palpation of the CAJs, as described earlier. But limited glottic opening can often preclude traditional orotracheal intubation, and the presence of an endotracheal tube can prevent evaluation and operation in the larynx. In the presence of an existing tracheostoma, the airway can be managed with simple tracheal intubation via the stoma. If no stoma has been created and tracheostomy is not indicated, a variety of techniques exist for alternative transglottic ventilation under anesthesia. The surgeon and anesthesiologist can coordinate intermittent intubation with periods of apnea. Transnasal humidified rapid-insufflation ventilatory exchange may extend the apneic interval 10 to 20 min or more.[25] Some patients may tolerate a carefully titrated general anesthesia with spontaneous ventilation, often supported with topical laryngeal anesthesia.[26,27] Laryngeal mask airway can facilitate the passage of flexible instruments including channeled bronchoscope or laryngoscope, but functionality may be less than in the straight-line suspension laryngoscopy approach.[28] Jet ventilation allows for simultaneous ventilation and operative intervention, but especially narrow glottic stenosis may necessitate subglottic jet ventilation with a Hunsaker catheter passed into the trachea. Jet ventilation carries risks including barotrauma, hypoxemia, hypercarbia, and laryngospasm, but it remains safe especially when managed by an experienced operative and anesthesia team.[29,30]

PGS varies in severity, summarized by the Bogdasarian classification system.[17] Regardless of classification and severity, operative management of PGS often begins with simple lysis of scar, either with a laryngoscopic sickle blade or with a $CO_2$ laser. The cut can be extended posterolaterally toward and into the CAJ space.

Vocal fold suture lateralization is typically used for management of BVFP, and utility can be limited in cases of PGS due to scar tethering the posterior glottis and preventing lateralization.

Sutures can be passed either endolaryngeal to extralaryngeal using an endoscopic suturing device (Lichtenberger needle driver)[31] or extralaryngeal to endolaryngeal using a large-gauge hollow-bore needle.[32] In either case, a suture is placed through the thyroid cartilage, with entry point in the larynx just below the vocal process of the arytenoid. Another suture passes through the thyroid cartilage and enters just above the vocal fold at the same location. These sutures are affixed to one another, and the loose extralaryngeal ends are pulled taught. They are tied down and secured either over the thyroid cartilage or even outside the skin, tied over a button for skin protection (**Fig. 5**). This procedure carried potential risks including dysphagia, aspiration, significantly worsened voice, and suture complications, among others.[31–33]

Transverse vocal cordotomy with or without medial arytenoidectomy remains 1 of the mainstays for treatment of glottis stenosis, whether from BVFP or PGS. Transverse cordotomy is typically performed with the $CO_2$ laser, in a suspension micro laryngoscopy approach. An incision is made approximately perpendicular to the axis of the vocal fold and the TA muscle fibers, to release the muscle from its attachment to the vocal process of the arytenoid. The incision can be carried out as far laterally as the inner perichondrium of the thyroid cartilage. In this way, the natural tension of the TA muscle causes it to release anteriorly, toward its attachment to the thyroid cartilage at the anterior commissure (**Fig. 6**).[34,35] Medial arytenoidectomy is often performed at the same time, which includes removal of the vocal process of the arytenoid, as well as variable amounts of the cartilage body. Despite the destructive nature of these procedures, they typically produce acceptable voice and swallowing outcomes.[36–38]

Additionally, the laryngeal surgery can remove a more significant portion of the arytenoid cartilage, with or without disruption of the mucosal vocal fold. Subtotal endoscopic arytenoidectomy includes an incision of the supraglottic mucosa over the arytenoid cartilage, with preservation of a medial mucosa flap. Once the cartilage is removed with a combination of cold dissection and $CO_2$ laser, the medial mucosal flap can be lateralized with endoscopic suturing,[39] or endolaryngeal/extralaryngeal suture lateralization in a similar technique to vocal fold suture lateralization.[40] Proponents describe superior voice and airway results compared to the simpler transverse cordotomy or medial arytenoidectomy, but the technique can have a significantly steeper learning curve, and there are limitations to application in severe PGS, whereas it can be more favorable in BVFP.[41]

Often combined with 1 or more of the earlier procedures, trans-glottic laryngeal stenting can be used to inhibit re-stenosis after a surgical opening procedure. Several prefabricated stents exist, but many surgeons use readily available materials including endotracheal tubes, Montgomery T tubes, and silastic sheeting to form custom stents. A relatively popular technique among otolaryngologists is to create a modified Montgomery T tube stent by removing the lower half, suturing the top shut to prevent aspiration and better approximate the shape of the glottis, and leaving a horizontal limb exiting the stoma. This can be held in place simply with the horizontal limb preventing superior/inferior movement or via trans-laryngeal suturing.[42] The stent does typically cause transient pain

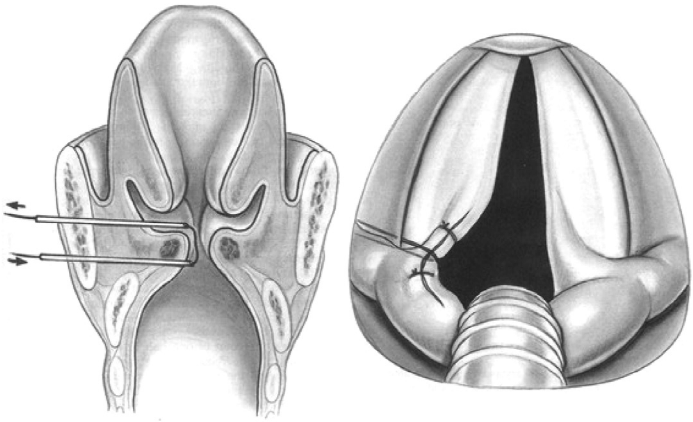

**Fig. 5.** An immobile vocal fold may be lateralized via endo extra-laryngeal suture lateralization to increase the glottic airway aperture. The surgeon passes a suture around the vocal fold and then ties the suture over the skin or over the thyroid cartilage, drawing the posterior vocal fold laterally. (Michael S. Benninger, Neil Bhattacharyya, Marvin P. Fried, Surgical management of bilateral vocal fold paralysis, Operative Techniques in Otolaryngology-Head and Neck Surgery, 9 (4), 1998, 224-229, https://doi.org/10.1016/S1043-1810(98)80008-0.)

and dysphagia, but it is removed after a sufficient period of healing (typically 4–6 w).

Finally, advanced endoscopic techniques can provide cartilaginous framework expansion. Endoscopic posterior cricoid split with cartilage grafting is a technique more commonly employed in pediatric airway surgery where cartilage is reliably soft. The cricoid cartilage is divided in the posterior midline with $CO_2$ laser or in 1 series with an ultrasonic bone aspirator. Either an autograft section of rib cartilage or a cadaveric rib graft is carved to serve as a spreader graft. The cartilage is locked into place, using the natural tension of the cricoid remains in position. The cartilage can be additionally secured with a transglottic T tube stent.[43,44]

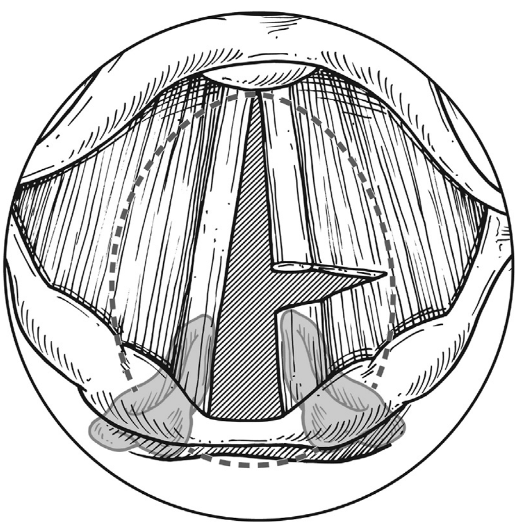

**Fig. 6.** In transverse cordotomy, the surgeon enlarges the glottic aperture by releasing the vocal fold from its attachment to the vocal process of the arytenoid. Via the natural tension of the thyroarytenoid muscle, the vocal fold releases forward, creating a posteriorly enlarged airway. (*Data from* Rayle CA, Fritz MA. Endoscopic management of laryngotracheal and pharyngoesophageal trauma. Operative Techniques in Otolaryngology-Head and Neck Surgery. 2020 Dec 1;31(4):308-16.)

Expansion of the laryngeal inlet size via posterior cricoid costal cartilage grafting is more typically performed via open approach in adults. In the case of glottic level stenosis, the cricoid or thyroid cartilages can be expanded with the interposition of cartilage grafts in the posterior midline, anterior midline, or both. Again, cartilage is harvested from the rib (cadaveric cartilage graft can also be successfully employed). Laryngofissure describes the opening of the larynx in the anterior midline. Once the larynx has been opened, the posterior midline can be split to allow for insertion of a posterior expansion graft (**Fig. 7**). Additionally, expansion graft can be used to enlarge the subglottic caliber via anterior placement in the inferior thyroid and cricoid incisions. Both anterior and posterior locations can be grafted (depending on need). The procedures typically require endolaryngeal stenting and tracheostomy during the healing period.

### *Techniques for Addressing Laryngeal Stenosis Also Associated with Subglottic or Proximal Tracheal Stenosis*

Management of laryngeal stenosis can be especially challenging when coincident with a second level of stenosis. Laryngeal stenosis can extend in continuity with SGS or be associated with discrete proximal tracheal disease secondary to the endotracheal tube cuff or tracheostomy stomal site injury. Each of these cases requires additional consideration with respect to counseling, airway management, and surgical technique.

Multilevel stenosis can be managed with endoscopic techniques, treating the multiple levels

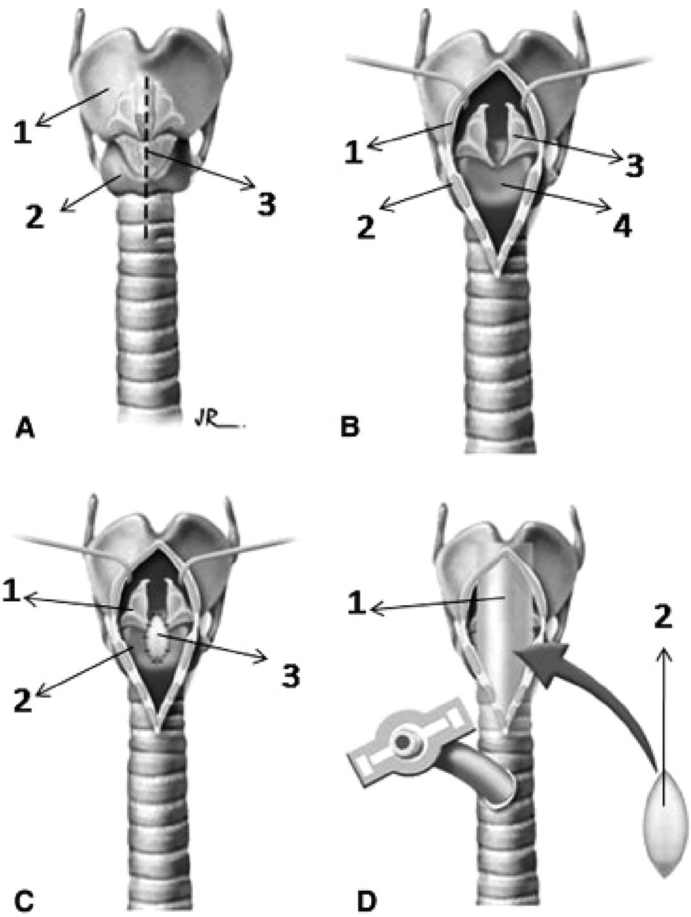


**Fig. 7.** Standard laryngotracheal reconstruction. (*A*) Laryngotracheal anterior exposure: 1, thyroid cartilage; 2, cricoid cartilage; 3, incision site to perform the anterior laryngeal split. (*B*) Airway exposure after anterior laryngeal split: 1, thyroid cartilage split and retracted; 2, cricoid cartilage split and retracted; 3, arytenoids; 4, posterior cricoid plate, exposed and ready to be split. (*C*) Posterior larynx grafting after posterior cricoid split: 1, arytenoids; 2, posterior cricoid plate already opened; 3, rib cartilage graft filling the posterior groove. (*D*) Laryngeal stent and anterior grafting: 1, solid stent inside the airway; 2, rib cartilage graft prepared to fill the anterior defect. (Ricardo Mingarini Terra et al., Laryngeal split and rib cartilage interpositional grafting: Treatment option for glottic/subglottic stenosis in adults, The Journal of Thoracic and Cardiovascular Surgery, 37 (4), 2009, 818-823, https://doi.org/10.1016/j.jtcvs.2008.08.035.)

separately, in a simultaneous or staged approach. In cases of severe subglottic and glottic stenosis, open segmental resection coupled with laryngofissure and posterior cricoid grafting can address both components of physiologic airflow limitation.

The laryngotracheal complex is accessed with a low horizontal cervical incision, with skeletonization of the cartilaginous airway framework. Cross-table ventilation is utilized to maintain endotracheal intubation transcervically. The airway is entered just below the level of structural compromise, and often just below the prior tracheotomy site. In cases of subglottic involvement, the anterior two-thirds of the cricoid are resected, with care to not disturb the region of the CT joint. The endoluminal scar can be directly excised down to the level of the cricoid perichondrium. When laryngeal and specifically posterior glottic expansion are needed, a laryngofissure is performed, and the cricoid is split in the posterior midline. The autologous or cadaveric rib graft is carved into a keystone or I-beam shape and inserted in the posterior cricoid. A flap of trachealis muscle is stretched and secured over the cartilage graft. The laryngofissure is closed, with care taken to reapproximate the anterior commissure of the vocal folds. A suprastomal, transglottic stent facilitates stabilization the distracted larynx and can be left in place during healing. The combination of segmental resection and laryngofissure with posterior cricoid grafting has been termed "extended cricotracheal resection (CTR)" (**Fig. 8**).[45]

### *Management of Recurrent Laryngeal Nerve Injury*

The RLN provides motor innervation to the adductor and abductor laryngeal musculature. The RLN takes a unique route from brainstem to larynx, which makes it vulnerable to injury from various insults. The nerves also have side-specific anatomy. The left RLN travels with CN X downward through the neck, posteromedial to the internal jugular vein, and posteromedial to the common carotid artery, within the carotid space. The left RLN and CNX follow the course

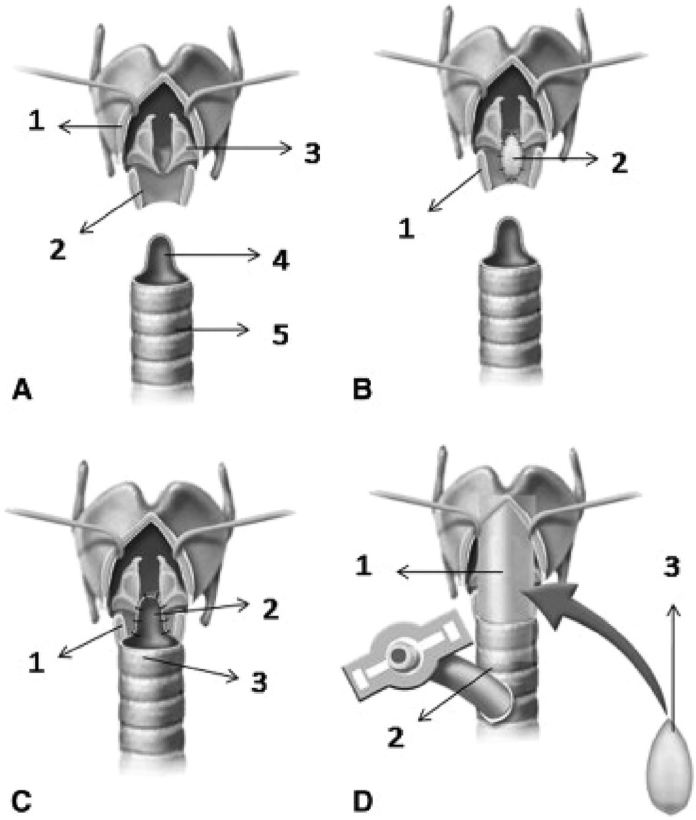


**Fig. 8.** Laryngotracheal reconstruction associated with cricotracheal resection. (*A*) Larynx apart from the trachea, anterior laryngeal split and cricotracheal resection already performed: 1, thyroid cartilage split and retracted; 2, posterior cricoid (anterior arch already resected); 3, arytenoid cartilage; 4, tracheal posterior wall flap; 5, remaining healthy trachea. (*B*) Cartilage grafting after posterior cricoid split: 1, posterior cricoid already split; 2, rib cartilage graft filling the posterior cricoid plate groove. (*C*) Posterolateral cricotracheal anastomosis hiding the posterior cartilage graft: 1, posterior cricoid cartilage; 2, posterior tracheal wall flap sutured to posterior cricoid remnant mucosa; 3, healthy trachea, posterolateral anastomosis with the larynx already completed. (*D*) Laryngeal stent and anterior grafting: 1, laryngeal stent; 2, trachea, tracheostomy already performed; 3, rib cartilage graft prepared to fill the anterior defect. (Ricardo Mingarini Terra et al., Laryngeal split and rib cartilage interpositional grafting: Treatment option for glottic/subglottic stenosis in adults, The Journal of Thoracic and Cardiovascular Surgery, 37 (4), 2009, 818-823, https://doi.org/10.1016/j.jtcvs.2008.08.035.)

of the carotid into the mediastinum and cross-anterior to the aortic arch. The RLN then loops 180° under the aorta and travels superiorly in the tracheoesophageal groove. This position results in a course intimate with the thoracic esophagus and the parathyroid and thyroid glands. The nerve enters the larynx at the CT joint. The right RLN similarly travels into the chest with the carotid artery and CN X before looping around the subclavian artery. It then travels along the upper lobe pleura and toward the tracheoesophageal groove. It does not travel along the groove until a point just inferior to the CT joint. Its course is therefore more oblique in the neck. In less 1% of humans, the right RLN is nonrecurrent and branches directly from CN X at the level of the thyroid gland or higher. This typically associated with a retroesophageal course of the right subclavian artery.[46–48]

A variety of insults can lead to nerve dysfunction including vascular interruptions, infectious/inflammatory disease, and compression by neoplastic lesions and trauma. But surgery remains a leading and common source of RLN injury. This risk exists during surgery for the skull base, carotid artery, heart and great vessels, bilateral lungs, esophagus, parathyroid, thyroid, and cervical spine via anterior approach. The nerve can be damaged via stretch, compression, thermal injury, or direct insult. Injuries are typically not recognized at the time of surgery.[46,49] The RLN function may be monitored during surgery with use of a specialized endotracheal tube with surface electrodes placed between the vocal folds.[50] This practice has become increasingly common in thyroid and parathyroid surgery but is not frequently employed in other disciplines. Intraoperative knowledge of nerve function in thyroid and parathyroid surgery can influence decision-making in completing bilateral surgery, especially when RLN signal is lost on 1 side intraoperatively. Knowledge of RLN anatomy, as well as these frequent sites of injury is critical in limiting iatrogenic RLN trauma. In cases of known or suspected RLN injury, a patient should be referred to a laryngologist (if available) or otolaryngologist for diagnosis and management.

Diagnosis of an RLN injury is typically make with laryngeal videoendoscopy, which can be guided by typical presenting symptoms including weak, breathy voice, limited cough, and dysphagia to liquids. Serial laryngoscopy can often observe partial or complete neural recovery, which subsequently confirms that the initial injury was partial. Laryngeal electromyography (LEMG) can also be useful in select cases to provide prognostic information, and specifically to diagnose total nerve injury. In the case of total injury, all voluntary motor activity will cease. After about 3 w, LEMG will demonstrate fibrillation potentials and positive sharp waves. In contrast, a partial nerve injury (with variable potential for recovery) will generate polyphasic action potentials.[51]

### Neural Injury Rehabilitation

Unilateral RLN paralysis is routinely managed by otolaryngologists with medialization laryngoplasty, whether by temporary injection or more permanent type 1 thyroplasty.[52] In cases of partial injury, temporary recovery can take place over the course of up to 12 mo.[53,54] In the acute and subacute setting, while awaiting that recovery, the otolaryngologist may offer injection medialization with products including carboxymethylcellulose, hyaluronic acid, calcium hydroxyapatite, or fat autograft. In some cases, these injections can be performed under local anesthesia either transcervically or transorally.[55] Type 1 thyroplasty entails insertion of an implant in the paraglottic space via an open cervical approach, after creation of a window through the inferolateral thyroid cartilage. Gore-Tex strips and carved silastic are common implant materials.[52,56–58]

Especially in cases of known complete transection, direct neurorrhaphy, cable grafting, or reinnervation may provide symptomatic benefit and improve the chances to regain tone in the denervated hemilarynx. Nerve repair does not typically restore dynamic adduction and abduction. In successful cases, nerve repair can maintain muscular tone and influence the vocal fold to rest in a midline or paramedian position. One series of known RLN transections in thyroplasty showed positive and similar outcomes from direct neurorrhaphy, cable grafting, and CN X to RLN anastomosis.[59] Similar results have been shows with ansa cervicalis to RLN grafting.[60] Reinnervation shows superior results in younger patients, especially pediatric patients.[61,62] Long-term voice results have inverse correlation with time from injury to reinnervation.[62]

BVFP is additionally challenging with respect to nerve repair and reinnervation. Since the RLN is responsible for both adductor and abductor muscles, reinnervation can result in synkinesis and does not typically restore dynamic function. Selective reinnervation is required for separate control of adductors and abductors. This can be achieved with select reinnervation of the PCA (sole abductor), either by neuromuscular pedicle transfer, selective neural micro anastomosis, or direct nerve implantation to the PCA.[63–65] Encouraging results have been seen in clinical trial settings with implantable PCA muscle stimulators or "laryngeal pacers".[66–69] Stem cell transfer to the PCAs may also have promise in restoring vocal fold abduction.[70] With any technique, reanimation of the PCA abductor function requires mobility of the CAJ.

### Management of Voice After Cricotracheal Resection

CTR with laryngotracheal anastomosis typically produces excellent respiratory outcomes in correctly selected patients. Although the surgical technique does not directly alter the vocal folds, it often causes negative changes in voice. After CTR, patients demonstrate reduced vocal pitch and reduced dynamic range.[71] This is principally attributed to disruption or resection of the CT muscle, which is responsible for lengthening and dynamically tightening the vocal folds.[72] In case series, the prevalence of at least moderate dysphonia after CTR was greater than 80%.[73] Voice therapy has documented benefit for management of postoperative voice changes, with specific success in reducing tightness, limiting pain with voicing, and improving loudness.[74] Minor alterations in technique have been described to avoid resection of the CT muscles, including partial or complete preservation of the anterior cricoid ring.[75,76]

### Perioperative Management of Laryngeal Edema After Cricotracheal Resection

CTR has multiple risks and known complications, ranging from minor wound healing complications to anastomosis dehiscence and acute airway compromise. Laryngeal edema is a common adverse effect in the immediate postoperative period. Edema can result from direct manipulation, as well as from disruption of the venolymphatic drainage pathways in the larynx and subglottis. Intravenous corticosteroids may be administered perioperatively as a single dose or consecutive doses every 8 h[77]; but some centers recommend against the routine use of corticosteroids with concern for inhibition of wound healing,[78] and no evidence-based consensus exists. If granulation tissue develops at the level of the anastomosis, corticosteroids can used for managed by inhalation, topical application through a tracheostomy, intralesional injection, or by intravenous route.[79]

Acute laryngeal edema can also be managed with inhaled racemic epinephrine, typically dosed every 3 to 4 h as needed.[80] Whether inhaled oronasally or via tracheostomy, continuous cool mist humification can aid in reducing viscosity of sputum, reduction in cough, and improved comfort.[81] Routine use of proton pump inhibitors may reduce laryngotracheal exposure to reflux during recovery, but effect on healing and comfort is unknown.

## SUMMARY

Effective airway surgery requires a working knowledge of laryngeal physiology and pathology. This often entails cooperative work between the thoracic surgeon and otolaryngologist, especially in complex cases.

The larynx is an essential component of the human airway through its role in respiration, phonation, and deglutition. Unilateral and BVFP can each result in significant disruption of all 3 dynamic components of laryngeal function. Laryngeal stenosis, particularly PGS and coincident laryngotracheal stenosis, requires complex operative management for restoration of upper airway patency.

Through interdisciplinary collaboration and ongoing education, thoracic surgeons and otolaryngologists can jointly improve outcomes for patients with complex airway diseases. Recent and upcoming advances hold promise for further innovations in laryngeal surgery, enhancing the collective ability to care for patients with complex airway disease.

## CLINICS CARE POINTS

- The larynx serves vital functions in airway protection, and disruption of laryngeal function can lead to respiratory compromise.
- Great care should be taken to preserve the RLN, inclusive of its course in the bilateral mediastinum, around the aortic arch, within the left intrathoracic and cervical tracheoesophageal groove, around the right subclavian artery, and along its more oblique right cervical course.
- Laryngeal neuromuscular function can be easily assessed via transnasal videolaryngoscopy, and in come clinical setting, laryngeal electromyography can provide additional prognostic information.
- Isolated laryngeal stenosis can often be managed with direct laryngoscopy-based techniques including vocal cordotomy, arytenoidectomy, with or without laryngeal stenting.
- More complex cases of laryngeal and laryngotracheal stenosis may require open laryngotracheal reconstruction, with possible inclusion of cartilage grafting.
- CTR often leads to post-operative voice changes, which can possibly be prevented via variations in technique and can be managed post-operatively with the help of voice therapy administered by trained speech-language pathologists.

## DISCLOSURE

None.

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