Vocal Fold Paresis and Paralysis

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Anatomy

Recurrent laryngeal nerve

Anatomy of the larynx and related structures is discussed in detail elsewhere [1]. This article reviews only a few of the relationships that are most important when evaluating vocal fold mobility disorders.

The nuclei of the recurrent laryngeal nerve (RLN) axons lie within the nucleus ambiguus in the medulla of the brainstem. The RLN axons travel with the vagus nerve down the neck until they branch off at the level of the aortic arch on the left and the subclavian artery on the right. On the left, the nerve passes inferior and posterior to the aortic arch and reverses its course to continue ly into the visceral compartment of the neck. The right RLN loops behind the right subclavian artery and ascends superomedially toward the tracheoesophageal groove. Both RLNs travel just lateral to or within the tracheoesophageal groove and enter the larynx posterior to the cricothyroid joint. The positions of the nerves in the neck make them susceptible to iatrogenic injury during surgery. Low in the neck, the course of the right recurrent nerve is more oblique, lateral, and probably more prone to injury than the left RLN [2].

Approximately 5 out of 1000 people have a nonrecurrent laryngeal nerve on the right. A nonrecurrent laryngeal nerve occurs only on the right, except...
in the rare case of situs inversus. It branches from the vagus nerve at the level of the cricoid cartilage and enters the larynx directly, without looping around the subclavian artery. This anomaly occurs in conjunction with a retroesophageal right subclavian artery [2].

The RLN innervates four of the intrinsic muscles of the larynx: the thyroarytenoid (TA), posterior cricoarytenoid (PCA), lateral cricoarytenoid (LCA), and interarytenoid (IA) muscles. Muscle innervation is unilateral except for the IA muscle, which receives contributions from both RLNs [5]. The TA and LCA muscles are vocal fold adductors. Unilateral denervation of these muscles results in an inability to close the glottis with resulting breathy voice and possible aspiration.

The PCA is the main vocal fold abductor. Paralysis of this muscle results in an inability to abduct during inspiration. Denervation of the PCA usually causes the arytenoid cartilage to subluxate anteromedially in unilateral vocal fold paralysis. The denervated PCA no longer counters the anterior pull on the arytenoid cartilage by the vocal ligament [5]. If both PCA muscles are denervated, as in the case of bilateral RLN paralysis, airway obstruction may occur.

The IA muscle is actually three muscles: the transverse arytenoideus muscle and two oblique arytenoideus muscles. The function of the IA muscle is not completely understood; however, it may assist in vocal fold adduction and provide medial compression to close the posterior glottis [5].

**Superior laryngeal nerve**

The laryngeal nerve (SLN) branches from the vagus nerve just inferior to the nodose ganglion, which contains the sensory cell bodies of the SLN. The nerve travels inferiorly along the side of the pharynx, medial to the carotid artery, and splits into two branches at about the level of the hyoid bone. The internal division of the SLN penetrates the thyrohyroid membrane with the laryngeal artery and supplies sensory innervation to the larynx. The external division of the SLN provides motor innervation to the cricothyroid (CT) muscle. The CT muscle changes vocal fold tension by elongating the fold. It is responsible for increasing the fundamental frequency of the voice. The external division of the SLN lies close to the thyroid artery, although its exact relationship to the artery is variable [3,4].

**Vocal fold paresis and paralysis**

Vocal fold paralysis implies vocal fold immobility due to neurologic injury. It does not indicate complete denervation, because nonselective reinnervation of the intrinsic laryngeal muscles may occur immediately after nerve injury without restoration of vocal fold movement. Vocal fold paresis implies vocal fold hypomobility due to neurologic injury and may result from weakness of the RLN, SLN, or both. Nerve injury may be unilateral
or bilateral. Vocal fold paresis may present as dysphonia, loss of the upper register of the voice, hoarseness, breathiness, throat pain, choking episodes, or decreased vocal stamina. Laryngeal findings may be subtle and include asymmetric vocal fold movement, bowing, and rotation of the larynx. Vocal fold paresis may also present as muscle tension dysphonia or in conjunction with benign vocal fold lesions, such as vocal nodules or cysts resulting from compensatory hyperadduction.

Recurrent laryngeal nerve paresis/paralysis

The RLN may be injured by several means, including iatrogenic or non-iatrogenic trauma, neurologic disease, tumor infiltration or compression, infection, collagen-vascular disease, or idiopathic disease. This event may occur with or without concomitant SLN injury, depending on the cause and site of the lesion. The RLN is at risk for injury during many surgical procedures, such as thyroid, anterior cervical spine, and thoracic surgery. The true incidence of vocal fold paralysis remains unknown. The incidence of injury to the recurrent laryngeal nerve from thyroid surgery has been reported as between 0.3% and 13.2%, and from anterior cervical spine surgery as between 2% and 21.6% [6,7]. Mechanisms of iatrogenic injury include intubation, transection, crush, traction, inadvertent ligature placement, and thermal injury.

The laryngologist must always consider unusual causes. Several neurologic diseases may affect vocal fold movement, including multiple sclerosis, amyotrophic lateral sclerosis, syringomyelia, myasthenia gravis, Guillian-Barré, and Parkinson disease [8–15]. Cerebrovascular accidents may result in injury to RLN neurons, but typically other neurons are affected also. Central nervous system (CNS) tumors, such as gliomas, can cause RLN paralysis, and diabetics may develop RLN neuropathy [16–19]. More unusual cases include disorders such as Gerhard syndrome, laryngeal abductor paralysis that may be familial (autosomal dominant, autosomal recessive or X-linked inheritance and with adult onset) or acquired secondary to bulbar lesions or neurodegenerative disease [20].

Aggressive thyroid malignancies may invade and injure the RLN. Compression by large thyroid goiters, benign neoplasms, and nonthyroid malignancies, such as the classic Pancoast tumor of the left upper lung, may also injure the nerve [21].

Idiopathic vocal fold paralysis is not well understood. Some suspect a viral cause, because many patients report an upper respiratory infection before the onset of vocal symptoms. There are several infectious causes that have been reported to cause RLN paralysis, such as Lyme disease, tertiary syphilis, Epstein-Barr virus, and herpes [22–25]. Other causes of RLN injury reported include systemic lupus erythematosus, patent ductus arteriosus, mediastinal radiation, I-131 therapy, amyloidosis, Charcot-Marie-Tooth, mitochondrial disorders, porphyria, polyarteritis nodosa, silicosis, and familial hypokalemic periodic paralysis [26–36].
The natural history of vocal fold paralysis depends on the mechanism and degree of injury to the nerve. The Sunderland classification system describes different degrees of nerve injury. First-degree injury means neurapraxia. Nerve function should recover completely. Second-degree injury means that Wallerian degeneration has occurred distal to an injured site (axonotmesis). Second-degree injury usually occurs after a crush injury and also results in complete recovery. Because the endoneural sheaths remain intact in a second-degree injury, synkinesis does not occur. Third-degree injury includes endoneural scarring, which can cause misdirected regeneration. Fourth-degree injury involves scarring that may block regenerating axons. Fifth-degree injury signifies complete transection of the nerve [37,38].

Clinically, unilateral RLN injury presents as a breathy voice. Diplopenthia, aspiration, and dysphagia symptoms may also occur. After a few weeks, the contralateral vocal fold may compensate by adducting further to improve vocal quality and aspiration. Should reinnervation occur, typically it may not be detectable initially [39,40]. The clinical course following reinnervation is determined by the degree of reinnervation and synkinesis.

**Synkinesis**

Reinnervation prevents muscle atrophy. Shindo and colleagues [40] demonstrated in a canine model that during the first 3 months after transection of the RLN there is atrophy of the TA and PCA muscles. After 3 months, however, the muscle fiber diameters of the denervated muscle begin to increase. By 9 months, the fiber diameters of the denervated muscles approach those of normal muscle. Spontaneous reinnervation may occur after nerve transection and prevent muscle atrophy. The source of the reinnervation is not known, but may include regenerating fibers from the transected RLN, the SLN, cervical autonomic nerves and nerve branches innervating pharyngeal constrictors [40].

Although reinnervation after a complete RLN transection prevents muscle wasting, typically it does not restore useful movement to the vocal fold because of synkinesis. Synkinesis results from nonselective innervation of adductor and abductor muscles. As a result, muscles that perform opposite functions contract simultaneously, resulting in immobility or hypomobility of the vocal fold [41]. The clinical picture depends on the proportion of adductor and abductor fibers reinnervated.

Crumley [39] describes a classification system for laryngeal synkinesis. In Type I synkinesis, or "favorable synkinesis," there is little or no vocal fold movement. The patient’s airway and voice are fairly normal, however. Types II, III, and IV are considered "unfavorable synkinesis." A spastic vocal fold that may twitch without control characterizes type II. Voice quality is poor. In type III synkinesis there is tonic adduction of the vocal fold. This adduction results in a reasonable voice, but the airway may be
compromised. Type IV synkinesis involves tonic abduction of the vocal fold resulting in a breathy voice and greater risk for aspiration. Type III synkinesis probably results from greater reinnervation of the LCA in comparison with the PCA fibers, whereas in Type IV the opposite likely occurs.

Superior laryngeal nerve paresis/paralysis

Superior laryngeal nerve paresis or paralysis may be caused by various conditions. Iatrogenic trauma during thyroidectomy has been historically accepted as the most frequent cause of SLN paralysis [42,43]. In 1980, Adour and colleagues [44] reported that SLN palsy was part of a combined cranial polyneuropathy attributable to viral infections. Similarly, Dursun and colleagues [45] suggested that viral infections, such as herpetic cold sores and upper respiratory infections, are commonly associated with this condition. A likely explanation is that infections lead to viral neuritis, resulting in SLN damage. To the best of our knowledge, this retrospective review included the largest population of confirmed SLN paresis and paralysis in the medical literature. Of the total sample (n = 126), 118 patients (93.6%) reported a viral infection immediately before the sudden onset of vocal impairment. Some of the patients used aspirin to relieve pain or fever accompanying acute infection. In some patients aspirin ingestion probably contributed to vocal fold hemorrhage, which resulted in further voice dysfunction. Often, several months elapsed after the causative infection before the patients sought medical attention. Before the diagnostic examination, these patients attributed the symptoms of SLN damage to upper respiratory infections. The development of compensatory mechanisms, such as muscle tension dysphonia (MTD), which subjectively improved voice quality, sometimes resulted in further delays in seeking treatment. As expected, singers were more aware of their symptoms than nonsingers, and delay in seeking attention tended to be shorter in this population. This observation was confirmed in a study by Eckley and colleagues [46] that demonstrated that voice range measurement is a useful parameter for analyzing the effects of SLN paresis or paralysis on voice and may also be used for measuring outcome following voice therapy. The effects of SLN paresis on vocal range help explain the sensitivity of professional voice users (especially singers) to the effects of the condition. Laboratory investigations performed during selected patient evaluations often demonstrated increased serum titers indicating herpesvirus types I and II and antibodies to influenza type A and other common viruses. This evidence suggested infection at some undetermined time in the past.

There were 8 patients in Dursun’s [45] study who had iatrogenic causes, such as thyroid surgery or external trauma. Such findings suggest that iatrogenic trauma can also cause isolated SLN paresis. Extreme care must be taken, particularly during thyroid surgery, to protect the external branch of the SLN in the pole of the thyroid, where it descends close to the thyroid
vessels. Jansson and colleagues [47] performed pre- and postoperative electromyography (EMG) on 20 patients undergoing thyroid surgery. Nine patients had postoperative SLN paresis by EMG. Additionally, 3 patients who had goiters had preoperative SLN paresis, which worsened postoperatively. Some 58% of the SLN pareses were present at 1 year follow-up, although most cases had some nerve recovery. To avoid harming the SLN during thyroid surgery, some authors recommend ligating distal branches of the thyroid artery as close to the thyroid capsule as possible, whereas others suggest identification of the external branch of the SLN to prevent injury [48]. Less common causes have also been reported, including neurologic disorders, anterior approach to the cervical vertebrae during surgery, carotid endarterectomy, nonsurgical trauma, and Reye syndrome [42,47,48].

The clinical manifestations of SLN paresis and paralysis are variable. The variability relates to different degrees of impairment, other associated pathologies, and the voice needs and awareness of each patient. Normally, the CT muscle contracts briskly in falsetto, vocal inflection, and modal phonation to increase tension in the vocal fold [49]. In SLN paresis and paralysis, the loss of this function may lead to lowered pitch, a more monotone voice, and poor vocal performance, especially at higher pitches [42,45]. SLN paresis and paralysis may cause vocal fatigue, hoarseness, impairment of volume, loss of upper range, loss of projection, and breathiness. Vocal fatigue may be caused by the additional effort required to raise vocal pitch and to project, by hyperfunctional compensatory gestures, or by pathologic neuromuscular fatigue in cases of marked paresis. The clinical manifestations of SLN paresis or paralysis, particularly loss of upper range, are more troublesome in singers and professional speakers. These patients often develop MTD to generate a stronger voice. In Dursun’s [45] series, 23.8% of the patients had MTD that seemed to be compensatory. It must be remembered that SLN paresis may be the underlying cause of voice misuse and consequent structural lesions.

Although not a commonly described finding, choking with or without regurgitation and throat clearing may also occur especially if there is associated neuralgia, hypoesthesia, or paresthesia. Anesthesia of the upper laryngeal space suggests injury to the internal branch of the SLN. The absence of anesthesia does not always rule out SLN paresis or paralysis, however, because the external branch may be the only affected portion. Although anesthesia usually is not seen even with complete paralysis of the SLN, subtle decreased ipsilateral gag (hypesthesia) is fairly common.

Indirect laryngoscopy or mirror examination may or may not reveal vocal fold abnormalities. A strong activation of the normal CT muscle must occur to cause laryngeal tilt toward the weak side, another sign of unilateral SLN paresis [50]. Flaccidity of the affected vocal fold may cause irregular vertical movements during respiration, which in turn causes various configurations of the glottis. A bowed vocal fold may be evident in SLN paresis or paralysis. These vocal folds are slightly concave, and glottic closure is
usually incomplete. This finding may be associated with other coexisting conditions, however, such as recurrent laryngeal nerve paresis or paralysis, advanced age, or other neurologic disorders [51].

Several authors have discussed the position of the vocal folds and glottic configuration in SLN paresis and paralysis. The studies reflect different opinions [50,52–54]. Contraction of a normal CT muscle rotates the posterior commissure toward the inactive side, which causes the paralyzed vocal fold to shorten and form an obliquely shaped glottis [49,50,55–57]. A thinned, shortened, and bowed vocal fold and an oblique glottis deviating to the paralyzed side are most consistent signs described in previous reports [42,43,57,58]. These are evident only in some cases of unilateral paralysis, however, and in the authors’ experience the lack of these signs does not rule out paralysis or paresis. Moreover, these findings may be observed if CT contractions are weak or if the patients have MTD, which involves not only the hyperadduction of the normal vocal fold but also anterior–posterior compression of the glottis [54,59]. Vocal fold lag (sluggish motion) during phonation requiring rapidly repeated adduction is the most consistent and easily observed sign of SLN paresis or paralysis.

Evaluation

Evaluation of vocal fold paralysis or paresis begins with a history and physical examination. The history should define the main complaints and symptoms of the patient and likely cause of the hypomobility. One should inquire about previous surgeries, prolonged intubations, and trauma. A complete medical history should be taken, including a thorough neurologic review of systems, smoking and alcohol history, and other questions to evaluate for possible malignancy. Questions pertaining to possible infectious causes should be asked, and a thorough vocal history should be taken to define the patient’s vocal habits and needs.

The physical examination should include a complete head and neck examination, with particular attention to examination of the cranial nerves. The laryngologist should assess the patient’s gag reflex and palatal movement to evaluate vagus nerve function. If the patient has a unilateral high lesion of the vagus nerve, the palate deviates to the intact side. The physician should listen carefully to the patient’s voice, and the larynx should be visualized. A mirror examination should be performed first, followed by laryngoscopy with either a rigid or flexible endoscope, or both. The voice should be evaluated during various phonatory tasks at several frequencies and intensities, as discussed elsewhere [60]. The laryngologist should look for asymmetric movement, vocal fold bowing, horizontal and vertical position of the vocal folds, and tilting of the posterior larynx. The presence of structural lesions and signs of laryngopharyngeal reflux disease can be observed also. Video documentation is important. Even thorough, routine
otolaryngologic examination generally is not sufficient for establishing a diagnosis in these patients, however.

Patients who have vocal fold paralysis deserve comprehensive evaluation. Strobosvideolaryngoscopy and various objective evaluations are extremely helpful in diagnosis, treatment planning, and assessment of treatment efficacy. They are reviewed in other publications [61]. Laryngeal EMG is helpful in confirming clinical impressions, and in detecting abnormalities in other laryngeal nerve–muscle complexes that may be missed because of distortion related to the most severe injury. For example, in a total right recurrent nerve paralysis, a left laryngeal nerve paresis is considerably less obvious than usual. Such information is important in designing optimal therapy, however. We have found laryngeal EMG to be a practical and invaluable component of the voice evaluation, as have other authors [62,63].

Each vocal fold is moved by many intrinsic laryngeal muscles. These muscles permit adduction, abduction, and longitudinal tension of the vocal folds. The laryngeal nerves supply the cricothyroid muscle, which is the primary structure responsible for increasing longitudinal tension. Maintaining stretch of the vocal fold is extremely important for pitch control, volume, and stability during soft singing, especially from the upper mid-range and higher. The recurrent laryngeal nerves innervate all of the other intrinsic muscles of the larynx. Paralysis or paresis may involve one or both vocal folds, although only one vocal fold is involved in the vast majority of cases [64]. When the recurrent laryngeal nerve is paralyzed, the vocal fold appears to stand still, except for slight respiratory motion. The ability to alter longitudinal tension is maintained, however. The vocal processes are therefore usually at the same level, and even the paralyzed side lengthens as pitch is increased. Consequently, if the normal vocal fold can cross the midline far enough to reach the paralyzed vocal fold, compensation is possible and glottic closure and reasonably good phonation can be achieved. The normal vocal fold can only compensate in the horizontal plane, however. It cannot move ly or inferiorly to meet the injured side if laryngeal nerve paralysis is present and has resulted in differences in vocal fold height. Over time, atrophy of the thyroarytenoid may occur, making even horizontal compensation more difficult.

When the superior laryngeal nerve is involved, longitudinal tension is impaired and the vocal fold may be bowed or sagging. Consequently, it typically lies in a lower plane, and compensation is difficult. This finding is especially true if both recurrent and laryngeal nerves are paralyzed, but problems occur even with isolated laryngeal nerve paralysis in the presence of abduction and adduction. Bilateral laryngeal nerve paralysis is often more difficult to diagnose and is probably missed frequently. Patients who have this condition have a “floppy” epiglottis, rendering their larynges difficult to see. Their vocal quality, volume, and pitch range are impaired. It is often particularly helpful to confirm a clinical impression of bilateral laryngeal nerve paralysis through EMG.
Briefly, if vocal fold paralysis seems to occur below the level of the node ganglion, complete evaluation from the skull base through the chest (including the thyroid) is essential. This localization can usually be made reliably in isolated unilateral recurrent laryngeal nerve paralysis. If the paralysis is complete (recurrent and ) or if there are other neurologic findings, intracranial studies should be performed also. Occasionally, central disease (especially multiple sclerosis) can produce unexpected neurologic signs, and if no cause is found after a paralyzed recurrent laryngeal nerve has been thoroughly evaluated, addition of a MRI of the brain and other studies should be considered. Because of the seriousness of missing intracranial lesions, many physicians obtain MRI of the brain and 10th cranial nerve with enhancement in all cases and this practice certainly is not unreasonable.

A few clinical maneuvers are useful for making paresis more apparent. Repeated maneuvers alternating a sniff with the sound /i/ are particularly helpful in unmasking mild PCA paresis. Repeated rapid phonation on /i/ with a complete stop between each phonation frequently causes increased vocal fold lag as the pathologic side fatigues more rapidly than the normal side. Other rapidly alternating tasks are helpful also, including /i/-/hi/-/i/-/ hi/-/i/-/hi/... and /pa/-/ta/-/ka/-/pa/-/ta/ka/-pa/ta/ka/... The vocal fold lag is sometimes easier to see during whistling. Laryngeal posture during this maneuver provides particularly good visibility of rapid vocal fold motions. A glissando maneuver, asking the patient to slide slowly from his or her lowest to highest note and then slide back down, is invaluable for assessing SLN function. The vocal process should be observed under continuous and stroboscopic light. If a laryngeal nerve is injured, longitudinal tension does not increase as effectively on the abnormal side, disparities in vocal fold length are apparent at higher pitches, and the vocal folds may actually scissor slightly with the normal fold being higher.

(Access Video on Normal neurolaryngeal examination in online version of this article at: http://www.Oto.TheClinics.com.)

Appropriate laboratory studies should be considered to rule out specific causes of vocal fold paresis and paralysis. These may include tests for syphilis, Lyme disease, diabetes, thyroid dysfunction, collagen vascular disease, myasthenia gravis, thyroid neoplasm, and other conditions. In addition to testing gag reflex, more quantitative sensory testing may be helpful.

**Treatment**

Treatment of unilateral vocal fold paralysis is designed to eliminate aspiration and improve the voice. When there is no aspiration, treatment depends on the patient’s need and desire for improved voice quality. It is well recognized that recovery of laryngeal nerve function is common if the
injury was not caused by transection of the nerve. Even when the nerve is transected, some innervation may occur. Consequently, it is best to delay surgical intervention for approximately 1 year, if possible, unless the nerve is known to have been divided or resected. This plan does not mean that treatment should be delayed, however, but only irreversible surgery. The collaboration of an excellent speech-language pathologist is invaluable.

Voice therapy

Objective voice analysis, assessment, and therapy by speech-language pathologists specializing in voice are helpful in virtually all patients who have dysphonia. Voice therapy is invaluable in the management of vocal fold paresis and paralysis. In all cases, the speech-language pathologist can provide detailed preoperative and postoperative assessment. Such assessment is often of diagnostic value. It is also of great help to the surgeon in objectively evaluating the efficacy of treatments. In addition, voice therapy sometimes avoids the need for surgery, saving the patient from exposure to unnecessary surgical risks. Heuer and colleagues [65] studied 19 female patients and 22 male patients who had unilateral recurrent nerve paralysis and found that after excellent voice therapy, 68% of the female patients and 64% of the male patients considered their voices satisfactory and elected not to have surgery. Final outcome satisfaction data were similar for surgical and nonsurgical patients. Even when surgery is eventually required, preoperative voice therapy helps the patient while surgical decisions are pending, provides training for optimal postoperative phonation, and prepares the patient psychologically for surgery with the knowledge that everything possible has been done to avoid unnecessary operative intervention. This strategy results in patient cooperation, motivation, and understanding through educated participation in the voice restoration process. The importance of this factor should not be overlooked in the art of medicine and medicolegal prudence.

In people who have unilateral vocal fold paralysis, initial assessment not only quantitates and documents vocal dysfunction but also explores a wide range of potentially useful compensatory strategies. In addition, the speech-language pathologist identifies spontaneous compensatory behaviors that may be counterproductive. For example, although speech pathology textbooks generally classify and treat vocal fold paralysis as a hypofunctional disorder, undesirable compensatory hyperfunctional behavior is common in these patients [66,67]. This behavior is responsible for most of the voice strain, neck discomfort, and fatigue that may accompany unilateral vocal fold paralysis. Such gestures often can be eliminated even during the first assessment and trial therapy session, increasing vocal ease and endurance. Moreover, if the assessment reveals improved voice with a different pitch, training in safe pitch modification in combination with other techniques may also provide rapid improvement. Indeed, under good guidance, therapy sometimes produces
astonishingly rapid improvements in voice quality despite persistence of the neurologic deficit. In any case, initial assessment is worthwhile to document vocal condition before surgery is considered and to get an estimate of how much the patient’s voice can be improved without surgery.

Most often, initial assessment results in modest but noticeable improvement in voice quality and subjectively important improvement in ease and endurance. Generally, several therapy sessions are needed to optimize vocal function. The speech-language pathologist provides patients with educational information about the workings of phonation, about their specific abnormality, and about vocal hygiene. The importance of and rationale for therapy are also explained. Therapy is directed toward avoidance of hyperfunctional compensation and progressive development of optimal breathing, abdominal support, and intrinsic laryngeal muscle strength and agility. Training includes head and neck muscle relaxation exercises, aerobic conditioning, abdominal and thoracic muscle strength and control exercises, attention to respiration, and various voice exercises that build limb strength through multiple repetitions with light weights. Forced adduction exercises, often recommended in speech pathology texts, such as pushing or pulling on chairs, must be avoided or monitored closely and used with extreme caution. Although such exercises are still in fairly common use, other techniques may be more effective and have less potential for harm. When available, traditional voice therapy combined with a few expert singing lessons may expedite improvement. This plan is analogous to including jogging or running in a rehabilitation program aimed at improving limb strength for walking.

Like surgery, therapy is least successful in combined paralysis. In most patients who have unilateral vocal fold paralysis, therapy results in improvement. In many cases, the improvement is sufficient for the patients’ needs. When the patient has complied with voice therapy, improvements have reached a plateau, and they feel that their voice quality is not satisfactory, surgery may be indicated.

If preoperative voice therapy has been optimal and if surgery has been successful, the postoperative voice therapy course should be short. Nevertheless, the patient is working with a “new voice.” At least a few sessions with a speech-language pathologist generally help the patient apply effective principles learned in preoperative therapy. It is particularly important for the voice therapist and speech-language pathologist to monitor the patient, avoiding development of abusive habits and stressing the importance of vocal hygiene measures. At the conclusion of therapy, objective voice measures should be repeated.

If the patient is interested in optimizing voice quality, it is reasonable to continue therapy as long as it continues to produce voice improvement. This judgment is usually made jointly by the patient, speech-language pathologist, and laryngologist. In most patients who have had good preoperative
voice therapy, this juncture or goal is reached within 1 to 3 months after surgery.

Bilateral vocal fold paralysis creates much greater problems; this is true for bilateral recurrent, bilateral, or bilateral combined nerve paralysis, or combinations thereof. There is still no satisfactory treatment of bilateral recurrent nerve paralysis. Frequently, this condition leaves the patient in the uncomfortable position of choosing between good voice and tracheotomy, or a good airway and bad voice. Therapy may provide some help to these patients, but it is rarely definitive. It is hoped that laryngeal pacing can provide a solution to these problems, as discussed later in this article. If so, there is an important role of the voice therapist following pacemaker implantation.

**Surgical therapy**

The two main surgical options for patients who have unilateral vocal fold paralysis are medialization and reinnervation. The most common and important techniques for surgical management of patients who have vocal fold paresis and paralysis are discussed in the article on voice surgery by Sataloff and colleagues elsewhere in this issue [68]. In this article we have included only a brief overview of some of these procedures and have highlighted discussions of techniques of reinnervation, gene therapy, and laryngeal pacing that are not discussed comprehensively elsewhere in this issue. Medialization procedures include injection laryngoplasty and laryngeal framework surgery. Several materials have been injected to medialize the vocal fold and improve glottic competence. These include polytetrafluoroethylene (commonly known as Teflon, E.I. DuPont Nemours and Company, Wilmington, Delaware), absorbable gelatin powder (Gelfoam, Pharmacia, Kalamazoo, Michigan), fat, collagen, dermal collagen, hydroxylapitate, and others. Teflon used to be the most popular choice; however, it has few (if any) indications today. The senior author (RTS) has not used Teflon since 1988. Teflon is permanent and leads to a chronic granulomatous inflammatory response [69]. Teflon can also migrate and may even spread to other parts of the body [70]. Teflon granulomas are difficult to remove and often result in a poor vocal outcome [61].

Gelfoam is used as a temporary measure, typically when future return of vocal fold function is possible but the patient needs or wants immediate symptomatic improvement. Gelfoam is absorbed within 3 months. If vocal fold function has not returned by then, the surgeon must decide whether reinjection or a more permanent procedure is warranted.

Fat is resorbed partially within 3 to 4 months [70], but improvement may be permanent. Autologous fat is harvested easily using liposuction or by direct excision and generally allows the vocal fold to maintain normal vibratory qualities.

Allogeneic, autologous, and bovine collagen have been used to medialize paralyzed vocal folds [69,71]. Collagen incorporates into host tissue [69].
Some report collagen lasting as long as 3 years. Collagen may be injected into the vocal ligament. It softens scar tissue and can improve the vibratory qualities of the vocal fold.

**Medialization**

Type I thyroplasty was popularized by Isshiki and colleagues [72]. Arytenoid adduction surgery was designed by Isshiki and colleagues [73] also to improve closure of the posterior glottis. Some laryngologists believe that after a long duration of vocal fold paralysis, the cricoarytenoid joint scars and becomes fixed. In this case, the ankylosis must be addressed for a medialization procedure to be effective [74]. Several animal and cadaver studies suggest that the cricoarytenoid joint remains normal for as long as 17 years after RLN injury, however [75,76].

**Reinnervation**

Several reinnervation procedures for the paralyzed vocal fold have been described using the ansa cervicalis [74], phrenic nerve [77,78], preganglionic sympathetic neurons [79], hypoglossal nerve [80], and nerve–muscle pedicles [74,77–82]. The main purpose of reinnervation procedures is to prevent denervation atrophy of laryngeal muscles. Crumley [74] reports improved vocal quality and restoration of the mucosal wave after reinnervation using the ansa cervicalis. The ansa cervicalis provides weak tonic innervation to the intrinsic laryngeal muscles. Reinnervation of the TA muscle restores tension resulting in a more normal mucosal wave. Reinnervation of the PCA and LA muscles stabilizes the arytenoids and prevents inferior displacement of the vocal process, which may occur in some patients. Crumley [74] reports additionally that the ansa cervicalis–RLN anastomosis is particularly useful in cases of synkinesis after nerve injury resulting in jerky movements of the vocal folds. Although there is still synkinesis after ansa–RLN anastomosis, the weak tonic innervation supplied by the ansa produces a vocal fold that is less spastic.

Attempts to design reinnervation techniques that might avoid synkinesis and restore movement to the paralyzed vocal fold have been reported [83,84]. Hogikyan and colleagues [85] examined muscle-nerve-muscle neurotization in the cat. In this technique, the paralyzed thyroarytenoid muscle is reinnervated by way of axons that sprout from the contralateral, innervated TA muscle through an interposed nerve graft. The authors demonstrated histologic and EMG evidence of this specific reinnervation pathway in more than half the cats used. Actual return of vocal fold adduction was demonstrated in one cat. This technique of motion-specific reinnervation is promising for restoration of physiologic movement after vocal fold paralysis.
Tucker [81] has reported improvement in voice quality and restoration of adduction of the unilateral paralyzed vocal fold after nerve–muscle pedicle transfer. This technique involves implanting a piece of strap muscle innervated by nerve terminals from the ansa cervicalis into one of the denervated laryngeal muscles, usually the LCA or TA [82,86]. Tucker [86] also reports better vocal quality in patients who have unilateral vocal fold paralysis when they are treated with nerve–muscle pedicle and medialization, than when treated with medialization alone.

**Bilateral vocal fold paralysis**

Although voice quality is typically good in the presence of bilateral vocal fold paralysis (BVFP), airway patency is jeopardized by the paramedian position of the vocal folds. Tracheotomy may be required acutely, followed by surgery to improve the size of the glottic airway. Surgical techniques are designed to lateralize one or both vocal folds to improve airway patency and assist with decannulation. Voice quality is impaired when the paralyzed vocal fold is lateralized. The most important of these techniques are reviewed elsewhere [68].

Cordotomy and arytenoidectomy with or without suture lateralization of the vocal fold are the most commonly performed lateralization procedures to treat bilateral vocal fold paralysis [87]. These procedures are typically performed endoscopically with use of the CO2 laser. The advantages of using the CO2 laser include arguably increased precision through the narrow endoscope and improved hemostasis requiring less need for tissue manipulation [88]. Potential complications include granuloma formation, scar, chondritis, and endotracheal tube fire. Patients should be put on antireflux medication preoperatively to reduce the risk for scar and granuloma formation [88,89].

Good results have been reported using the above techniques. Efforts continue to improve lateralization techniques. Cummings and colleagues [90] have developed a polyethylene device with a double-helix screw that engages and lateralizes the vocal fold. The authors have reported in animals, promising potential advantages of this new device including more control of the lateralization process and adjustability to fine-tune voice and airway results.

Several reinnervation procedures to the PCA muscle have been described [77,79,81]. Given its inspiratory activity, the phrenic nerve is an obvious candidate for anastomosis. Despite animal models, however, there has been no reported clinical success with such a technique [91]. Tucker [81] has reported airway improvement and return of abductor function after nerve–muscle pedicle transfer. Such success has not been universal, however [92].

The use of botulinum toxin injection in the treatment of bilateral paralysis has been explored in animal models [93,94]. Injection of toxin into the cricothyroid muscle results in decreased tension in the vocal fold and subsequent lateralization with airway improvement. The author (RTS) has also
used botulinum toxin injections in the adductor muscles (TA and LCA) for bilateral severe paresis to eliminate synkinesis and permit unopposed action of the PCA to abduct the vocal folds.

When both vocal folds are paralyzed in the cadaveric position, as from a high vagal lesion, the airway may be fine, but voice and swallowing may be impaired. In this setting, unilateral or bilateral medialization procedures may be useful.

**Laryngeal pacing**

Functional electrical stimulation (FES) of the larynx, or laryngeal pacing, continues to be explored as a potential therapeutic option for unilateral and bilateral paralysis [91,94–102]. FES systems have been used to restore motor function to patients who have spinal cord injury, to control heart rhythms in cardiac disease, and to restore sensory function (cochlear implant, for example) [91].

Unlike cardiac pacemakers, laryngeal pacers require an efferent and an afferent limb. An afferent limb is needed to provide information to enable effective timing of muscle contracture [95]. For example, in the setting of unilateral vocal fold paralysis (UVFP), if the paralyzed side is stimulated to adduct when the innervated side is abducted, this does not result in improvement of glottic competence or voice. In the setting of BVFP, firing of the phrenic nerve, a change in intrathoracic pressure, or chest wall expansion can provide the afferent input signaling inspiration [91,96]. This activity results in stimulation of the PCA muscles to abduct the vocal folds. In the setting of UVFP, the contralateral TA or LCA muscles are the best candidates for afferent input [97].

The efferent limb of the system may be connected to a nerve, either the vagus or RLN if it is still intact [91], to the nerve of a nerve–muscle pedicle [95], or to the denervated muscles themselves [91,98,99]. After an RLN transection, axons may fail to regrow through a neurorrhaphy or other reinnervation procedure. By placing the electrodes in the denervated muscles themselves, the system would bypass this potential pitfall. In addition, functioning of the system would not rely on regeneration of axons.

Several animal studies have been performed to explore the ideal parameter settings for laryngeal pacemakers. These parameters differ depending on where electrodes are placed and what muscles are being stimulated [91,100]. In the canine PCA muscle, the optimal stimulation frequency is between 60 and 90 Hz and the optimal pulse duration is 2.0 milliseconds. Stimulation intensities up to 6 V are tolerated without tissue damage. In a model of canine UVFP, maximal adduction was achieved with stimulation intensities from 3 to 7 V, pulse duration of 0.5 milliseconds, and frequencies from 84 to 100 Hz [97]. In human patients who had vagal nerve stimulators placed for intractable seizures, abduction was noted at 20 Hz, whereas 40 Hz was required for adduction. Pulse duration of 3 milliseconds and stimulation
intensities of 3 mA were used for all patients [101]. Some implantations of laryngeal pacers have been done in patients who have bilateral vocal fold paralysis. More than one half of the patients have been decannulated. Patients must turn on the device manually and train themselves to breath synchronously with the device. In the future, pressure-sensing devices may be added to stimulate abduction with inspiration [102].

Gene therapy

Gene therapy may offer future treatment options for recurrent laryngeal nerve injury. Several growth factors have been identified that promote neuronal survival and sprouting. Delivery of genes encoding such growth factors into host tissue may protect against neuronal degeneration and stimulate regeneration after nerve injury. Shiotani and colleagues [103] delivered the gene for IGF-I in a nonviral vector to the rat thyroarytenoid muscle after RLN transection. Rats who received the gene demonstrated greater reinnervation and less muscle atrophy than rats who did not receive the treatment.

Viral vectors carrying gene products can be delivered to the CNS by retrograde transport after peripheral injection into nerve or muscle. Rubin and colleagues [104] demonstrated that delivery of viral vectors to the CNS is possible through the recurrent laryngeal nerve. This technique could be useful in the treatment of neurodegenerative diseases, such as amyotrophic lateral sclerosis, or for RLN injury with a partially intact nerve.

Vocal fold paralysis in children

Vocal fold paralysis represents 10% of congenital anomalies of the larynx, second only to laryngomalacia [105,106]. It is also the second most common cause of neonatal stridor [105,107].

The most common cause of pediatric vocal fold paralysis is controversial. CNS anomalies are the most common causes of bilateral vocal fold paralysis [105]. Of these, Arnold-Chiari malformation is the most common [105,108]. This anomaly involves herniation of the cerebellum and brainstem because of an abnormally small posterior fossa and results in either unilateral or bilateral paralysis. Some controversy exists as to whether the reason for paralysis is increased intracranial pressure secondary to hydrocephalus or pressure on the vagus nerve exerted by the herniating central nervous system tissue [105,109]. Other causes of vocal fold paralysis in children include birth trauma,iatrogenic injury, blunt trauma, mediastinal masses, cardiac anomalies, and other neoplasms [105,108,110].

Stridor is the most common sign of unilateral vocal fold paralysis (UVFP) or bilateral vocal fold paralysis (BVFP) in children. UVFP also may present as a breathy cry, feeding difficulties, and aspiration. Bilateral
vocal fold paralysis typically presents with airway obstruction and aspiration. Evaluation for vocal fold paralysis includes a complete history, careful listening to the airway and child’s cry, full head and neck examination with particular attention to the neurologic exam, fiberoptic examination of the airway, direct laryngoscopy and bronchoscopy to assess cricoarytenoid joint function and to look for other anomalies, and MRI from the brain and skull base through the mediastinum. EMG is used at some centers [111].

Recovery rates for pediatric vocal fold paralysis have been reported from 16% to 64%. Function may return after 6 weeks to 5 years [105]. Children who have UVFP can be observed in most cases, although occasionally a tracheostomy may be warranted. Positioning maneuvers can be performed to try to prevent aspiration. Type I thyroplasty has been performed in some cases [112]. BVFP typically requires urgent airway management and tracheotomy. Lateralization procedures, such as arytenoidectomy or cordotomy, may be performed if bilateral paralysis does not recover [105,107]. Many otolaryngologists recommend waiting at least 12 months before surgery, but this too is controversial [107]. EMG may provide prognostic information [105].

Arytenoid dislocation/subluxation

Arytenoid dislocation or subluxation, although frequently unrecognized, is not a rare entity. Arytenoid dislocation is frequently mistaken for vocal fold paralysis. It is extremely important for the otolaryngologist to be aware of this condition, because it can usually be treated successfully if diagnosed promptly. When missed or misdiagnosed as vocal fold paralysis, surgical repair becomes more difficult, although not impossible as previously believed [113–118]. Understanding the complex anatomy and embryology of the arytenoid helps clarify the condition [119].

Traditionally, arytenoid dislocation has been suspected by history and absence of the jostle phenomenon present in many cases of unilateral vocal fold paralysis. Often it is not diagnosed until direct laryngoscopy reveals impaired passive mobility of the vocal fold. Preoperative differentiation between vocal fold paralysis and arytenoid dislocation should be possible in virtually all cases. If not considered specifically, however, it is often missed. Disparity in height between the vocal processes is much easier to see in slow motion under stroboscopic light at various pitches. In posterior dislocations, the vocal process is higher on the abnormal side. In anterior dislocations, the vocal process is lower on the involved side. In either case, the injured vocal fold may move sluggishly or may be immobile. Rarely, abduction and adduction may appear almost normal under continuous light. Video documentation of the preoperative and postoperative appearance can prove particularly helpful in cases of arytenoid dislocation, because many of these patients are involved in litigation related to their injuries.

The most valuable tests are the stroboscopic examination to visualize differences in vocal process height; CT scan, which may image the arytenoid
dislocation and reveal clouding or obliteration of the cricoarytenoid joint space; and laryngeal EMG to differentiate an immobile dislocated arytenoid joint from vocal fold paralysis. Airflow analysis is also helpful in documenting changes.

Although surgical reduction of a dislocated arytenoid may rarely be impossible even in early cases, it is worth attempting in all patients before treatment with vocal fold injection, adduction/rotation, or other surgery. Even many months after injury it has been possible to move the arytenoid enough to bring the vocal process back to normal height and allow good approximation with the mobile vocal fold.

Summary

Vocal fold paralysis and paresis remain incompletely understood phenomena. Although evaluation techniques continue to improve, we still diagnose many cases as idiopathic. Although current surgical techniques enable us to improve voice, swallowing, and airway, we have not been able to restore useful movement consistently to the paralyzed vocal fold. With the development of new diagnostic and surgical techniques, we will continue to improve our understanding and treatment of the paralyzed or paretic vocal fold.

References


