# Vocal Cord Paralysis: Pathophysiology, Etiologies, and Evaluation

Mostafa Alwan[1], Paul M Paddle[2]

## Abstract

Vocal fold paralysis often leads to significant morbidity including dysphonia, dysphagia and aspiration. An appropriate understanding of the pathophysiology, etiologies, and a systematic method of evaluation are essential for discussing prognosis and offering patients appropriate treatments. This article presents a discussion of the most recent literature surrounding vocal cord paralysis, specifically the pathophysiology and different etiologies, and proposes an evaluation strategy involving history, examination and targeted investigations.

**Keywords:** Dysphonia, Laryngology, Otolaryngology, Vocal folds, Vocal fold Paralysis.

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## Introduction

The larynx is a dynamic structure involved in respiration, phonation, and deglutition. The laryngeal muscles are innervated by two branches of the vagus nerve, the recurrent laryngeal nerve (RLN) and the superior laryngeal nerve (SLN). Damage or injury to these nerves may result in vocal fold paresis or paralysis. Vocal cord paresis can be considered separate from vocal cord paralysis, with each having a distinct set of symptoms and examination findings.[1] Vocal fold paresis implies various degrees of vocal cord hypomobility due to neurological injury and may result from weakness of the RLN or SLN or both. Vocal fold paralysis implies complete vocal fold immobility due to neurological injury. This article will discuss vocal cord paralysis. A brief overview of the RLN and SLN anatomy is presented, followed by a discussion of the pathophysiology, etiologies, and evaluation.

[1]Department of Otolaryngology Head & Neck Surgery, Monash Health, Melbourne, Victoria, Australia

[2]Department of Otolaryngology Head & Neck Surgery, Monash Health, Melbourne, Victoria, Australia; Department of Surgery, Faculty of Medicine, Nursing & Health Sciences, Monash University, Melbourne, VIC, Australia; Melbourne Voice Analysis Centre, Victoria Parade, East Melbourne, Australia

**Corresponding Author**: Mostafa Alwan, Department of Otolaryngology Head & Neck Surgery, Monash Health, Melbourne, Victoria, Australia, Phone: +613 9594 6666, e-mail: Research@monashhealth.org

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### Recurrent Laryngeal Nerve

Originating within the nucleus ambiguus in the medulla of the brainstem, the RLN axons travel with the vagus nerve and exit the cranium *via* the jugular foramen, descending down the neck. The RLN leaves the vagus nerve in the chest and loops around the aortic arch on the left and the subclavian artery on the right. Both RLNs travel within or just lateral to the tracheoesophageal groove, entering the larynx posterior to the cricothyroid (CT) joint. The RLN divides into an anterior and posterior branch, with the dividing point being located inside the larynx in two-thirds of cases, and before the entering point in the remainder of cases.[2] The superior branch forms the anastomosis of Galen with the internal branch of the SLN and is likely sensory in nature.[3] Nonrecurrent laryngeal nerves (nonRLN) have an estimated incidence of 5 out of 1000.[1] Previously thought to occur only on the right, recent reviews have demonstrated rare instances of left sided nonRLNs.[4] The nonRLN branches from the vagus nerve at the level of the cricoid cartilage and enters the larynx directly, and is associated with an aberrant retroesophageal right subclavian artery.[4]

The RLN innervates four of the intrinsic laryngeal muscles: the thyroarytenoid (TA), posterior cricoarytenoid (PCA), lateral cricoarytenoid (LCA), and interarytenoid (IA) muscles. Of these muscles, only the IA receives bilateral RLN innervation[5] with recent studies suggesting additional motor contributions from the internal branch of the SLN.[6] The LCA muscle is a strong adductor of the vocal folds. All laryngeal functions requiring glottic closure such as phonation, coughing, straining, and Valsalva's maneuver require a strong contraction of the LCA muscle. Likewise, the PCA muscle is the primary abductor of the vocal folds, with inspiratory vocal fold abduction being the most important function of this muscle.[7] The IA muscle is comprised of three muscles, the transverse arytenoid muscle and two oblique arytenoid muscles.[1] It is theorized that the IA muscle may assist in vocal fold adduction, aiding to close the posterior glottis, however the function is not completely understood.[5,8]

### Superior Laryngeal Nerve

The SLN branches from the vagus nerve just below the level of the jugular foramen, inferior to the nodose ganglion, which contains the sensory cell bodies of the SLN.[1] The SLN then takes a direct course to the larynx, traveling medial to the carotid artery and divides into two branches at the level of the hyoid bone. The internal branch of the SLN penetrates the thyrohyoid membrane and is sensory to the supraglottic larynx as well as potentially contributing motor fibers to the IA muscle.[6] The external branch of the SLN provides motor innervation of the CT muscle. The CT muscle is primarily

responsible for changing the fundamental frequency of the voice by elongating the vocal fold when contracting.

## Pathophysiology

### *Neuronal Injury*

It is important to differentiate between nerve conduction block and presence of axonal injury for prognostication purposes when possible. Most surgically induced nerve injuries are the result of pressure, crushing or heating intraoperatively, leaving the nerve macroscopically intact.[3] Neuropraxia is the mildest form of injury, in which the Schwann cells are injured but the axon integrity is maintained, often resulting in a conduction block lasting for 6–8 weeks whilst the Schwann cells repair.[3] This appears to also be the case for the RLN.[9] Axonotmesis is when axonal injury is present, leading to possible neuron death or reinnervation with variable functional outcomes.[3]

In the peripheral nervous system (PNS), the myelin sheath is derived from Schwann cells which promote axonal growth and regeneration. Peripheral nerve injuries are therefore more likely to experience regeneration and regrowth than central nervous system injuries.[10,11] Following nerve injury, the distal segment of the injured or severed nerve will remain physiologically active until it degenerates, occurring after approximately 1 week.[12]

**Table 1:** Possible causes of vocal cord paralysis

| *Etiologies* |
|---|
| Surgery |
| Thyroid surgery |
| Carotid endarterectomy |
| Anterior approach to the cervical spine |
| Skull base surgery |
| Thoracic surgery |
| Neoplastic |
| Thyroid |
| Lung |
| Esophageal |
| Mediastinal |
| Endotracheal intubation |
| Viral |
| Herpes simplex virus |
| Epstein-Barr virus |
| Influenza virus |
| Varicella zoster |
| Cytomegalovirus |
| Neurological causes |
| Parkinson's disease |
| Multiple sclerosis |
| Amyotrophic lateral sclerosis |
| Myasthenia gravis |
| Cerebrovascular accidents |
| Central nervous system tumors |
| Miscellaneous |
| Aortic aneurysm |
| Pulmonary artery enlargement |
| Tuberculosis |
| Granulomatosis with polyangiitis |
| Sarcoidosis |
| Pneumoconiosis |
| Systemic lupus erythematosus |
| Idiopathic |

### *Clinical Course of RLN Injury*

Acute unilateral paralysis of one RLN will cause denervation and loss of function to the TA, LCA, PCA, and IA muscles, with the IA being less affected due to additional nervous supply. Denervation of the TA produces a flaccid vocal fold with decreased muscle mass and tension, causing a rounding of the glottic edge due to lateral bowing of the vocal cord.[5] Denervation of the LCA results in a loss of adduction of the vocal fold, which results in an inability to close the glottic opening. PCA acute denervation may result in a partially subluxed arytenoid anteriorly and medially, due to the pull of the vocal ligament and possible pull of the IA, still innervated partially by the contralateral RLN.[5] The summation of these effects, seen in acute complete paralysis of one RLN, is the loss of vocal fold mass, abduction, and adduction resulting in glottic incompetence. Whilst the contralateral vocal fold is able to abduct, this is usually inadequate to produce normal phonatory oscillation bilaterally.[1] The result is an acutely hoarse, breathy and potentially diplophonic voice. This usually lasts for several weeks but may be followed by partial improvement of voice quality as the contralateral vocal cord compensates by increasing movement and moving across the midline.[5] The normal vocal cord can only compensate in the horizontal plane however, and cannot move in the superio-inferior plane if laryngeal nerve paralysis has resulted in height mismatch of the vocal cords. After 4–5 months, nerve regeneration has begun and there may be some clinical signs of improvement.[5]

### *Nerve Regeneration and Synkinesis*

The motor axons of the RLN contain four times as many adductor axons as there are abductor axons, and when nerve injury is followed by regeneration, these axons maintain their adductor and abductor affinity, however may regenerate inappropriately with distal axons of the opposite type.[5] In patients with a RLN injury in whom there is nerve regeneration, laryngeal synkinesis often follows. Laryngeal synkinesis is a form of defective healing where severed nerve sheaths do not accurately regenerate to their preinjury counterpart, causing uncoordinated muscle contraction. Whilst synkinesis occurs as a rule in partial nerve injuries, in complete RLN injuries, it is possible but not certain.[5] If reinnervation occurs, the abductor and adductor muscles will not undergo denervation atrophy, with canine models demonstrating that whilst during the first 3 months after RLN transection there is atrophy of the TA and PCA muscles, after 9 months, the fiber diameters of the denervated laryngeal muscles have approached those of normal muscle.[13] Although synkinetic, the resulting reinnervation may result in a motionless vocal fold, which matches its counterpart in mass and tension, thereby allowing phonation to occur and this is known as favorable synkinesis.[1] In some cases, the resulting nerve regeneration may result in a spasmodic vocal cord, resulting in muscular dyscoordination during phonation and inspiration, producing a dysphonic voice, also known as unfavorable synkinesis.[14]

## Etiologies

### *Introduction*

The vagus and RLN nerves can be injured by multiple means, including vascular insults, viral or bacterial infections, tumor

infiltration or compression, and trauma both iatrogenic and noniatrogenic (Table 1). At present the true incidence of vocal cord paralysis is unknown, and determining an exact incidence is complicated by likely underdiagnosis and patients with asymptomatic vocal cord paralysis.[15]

The RLN is at risk during operations involving the neck, chest, cervical spine and skull base. The mechanisms of neural injury during surgery include thermal damage, compression, vascular compromise, stretching, and transection. Whilst nerve transection is often recognized at the time of surgery, electrocautery injury is frequently not recognized, as stimulation of muscle and nerve fibres can occur within 1 cm of the monopolar cautery tip, and current flow along a dissecting instrument inadvertently contacted by an electrocautery device can also cause nerve damage[16] Whilst primary anastomosis is recommended in the event of nerve transection, exact regeneration remains unlikely, and synkinesis may result even following successful and timely nerve repairs.[15]

### *Surgery*

Iatrogenic surgical injury is one of the leading causes of RLN paralysis, with the most common operations causing damage being thyroid surgery, carotid endarterectomy, anterior approaches to the cervical spine, skull base surgery, and thoracic surgery.[15]

Damage to the RLN following thyroid surgery is a well-known complication, having been recognized for over a century and being a common cause for litigation.[17,18] Thyroid surgery has been reported to be responsible for 4.1–9% of vocal cord paralysis, with the relative incidence decreasing, however this is suspected to be relative as the numbers of skull base and anterior cervical spine operations have increased in recent decades.[15] 0.7% of patients with benign thyroid neoplasms have RLN paralysis at presentation, caused by compression, stretching or inflammation.[19] The incidence of permanent paralysis and temporary paralysis after thyroid surgery is reported at 0.5–2.4% and 2.6–5.9%, respectively,[20,21] with rates being lower in specialist centres.[22] Risk factors for RLN injury during thyroid surgery include cancer surgery, revision surgery, previous radiation, lymph node dissection, and retrosternal extension.[23–25] Additionally, risk to the RLN increases when the nerve is not routinely identified during surgery.[26] Intraoperative monitoring may decrease the rate of RLN damage however it is difficult to draw definitive conclusions, with studies having mixed results or not demonstrating statistical significance in their findings.[27,28]

Carotid endarterectomy has a reported RLN paralysis incidence of 2–6%.[29] Suspected mechanisms of injury include traction injuries, clamp injuries, and interruption of neurovascular supply to the nerve.[29] RLN fibres are located on the medial aspect of the vagus nerve at the level of the carotid bulb, and are therefore at risk when skeletonizing the artery.[30] Clamping of the artery may disrupt the vasa nervorum leading to RLN injury, and compression from vascular loops placed inadvertently around the vagus nerve also contribute to RLN paralysis.[31]

Recurrent laryngeal nerve injury from anterior approaches to the cervical spine is the most common complication of these procedures, quoted at 2–6%.[32,33] Injuries are thought to be related to stretching of the nerve whilst retracting between the carotid sheath and the larynx, or compression from the endotracheal tube cuff in conjunction with the surgery.[33] The right RLN approaches the larynx at a sharper angle relative to the sagittal plane than the left, and is therefore more vulnerable to injury,[34] with injury rates reflecting this.[32] RLN injury from these operations appear to recover in time however, with one study demonstrating that 80% of patients with dysphonia after anterior approaches to the cervical spine experiencing a full recovery within 1 year.[32]

Skull base surgery often involves manipulation of cranial nerves, which are segmentally demyelinated and therefore more susceptible to injury.[35] Whilst proximal vagal nerve injuries produce vocal cord paralysis, they also result in loss of laryngeal and hypopharyngeal sensation and loss of palatal function, increasing the risk of aspiration and oropharyngeal dysphagia.[15] Vagal neuropathy from skull base paraganglioma excision has a reported incidence of 50–100%, with several cases reporting intraoperative preservation of the vagus nerve still experiencing postoperative ipsilateral vocal cord paralysis.[35,36]

As the left recurrent laryngeal nerve passes under the aortic arch lateral to ligamentum arteriosum, it is susceptible to compression from aortic dilatation, left atrial dilatation secondary to mitral valve stenosis or progressive heart failure, also known as Ortner syndrome.[37] The left RLN is also at risk of injury in thoracic, open heart, and esophageal surgery. Vocal cord dysfunction following left pneumonectomy or lobectomy has been cited at 31% in a 2001 study of 99 patients, and was associated with a 19% mortality.[38] More recent studies have suggested that continuous intraoperative neuromonitoring may be used to predict postoperative vocal cord function and may be helpful to prevent RLN during video-assisted thoracoscopic surgery lobectomy.[39]

### *Neoplastic Etiologies*

The most common nonlaryngeal neoplasms causing RLN paralysis are thyroid, lung, oesophageal and mediastinal.[15] Thyroid malignancies may invade the RLN causing paralysis and benign thyroid neoplasms and thyroid goiters may cause compression induced RLN injury. Nonthyroid neoplasms along the course of the RLN may cause vocal cord paralysis, including neurogenic, oesophageal, lung, and mediastinal masses. Whilst paragangliomas do not frequently cause RLN paralysis, the surgery for these tumours may cause postoperative paralysis.[40]

Recurrent laryngeal nerve paralysis caused by malignant neoplasm carries an unfavorable prognosis regarding return of function, with one series examining 98 patients reporting no functional recovery in RLN paralysis caused by malignant neoplasm, compared to a 31% recovery rate in cases attributed to surgical injury.[41]

### *Endotracheal Intubation*

The exact mechanism of endotracheal intubation-induced RLN paralysis remains unknown. The internal branch of the RLN is at risk of compression between the arytenoid cartilage, the thyroid cartilage and the inflated cuff of the endotracheal tube.[42] However there have also been theories that RLN paralysis following intubation may be caused by opportunistic viral infections secondary to local trauma.[43] When vocal cord paralysis is suspected following endotracheal intubation, arytenoid cartilage dislocation must be excluded.[15] A review of 210 patients found the incidence of temporary and permanent RLN paralysis following short-term intubation to be 1.4% and 0.5%, respectively.[44]

### *Viral Etiologies*

Several viruses have been cited as causing RLN paralysis, however proving viral etiology remains difficult, as positive biochemical testing does not prove neural involvement nor causation. To demonstrate a direct relation between viral infection and vocal cord paralysis, a neural specimen is required and these are not routinely

performed. It is therefore difficult to accurately report the incidence of viral induced vocal cord paralysis, however Herpes simplex virus,[45–47] Epstein-Barr virus,[48,49] influenza virus,[50–52] varicella zoster,[53–55] and cytomegalovirus[56,57] have all been cited in case reports. The mechanism of neural injury by viruses is not completely understood. It is theorized that either direct viral invasion or immune response to viral infection causes neural edema and subsequent myelin sheath damage.[48] Neural involvement from Epstein-Barr virus and influenza virus may recover,[49,50] however RLN damage from herpes simplex virus has been reported to be permanent.[46]

### *Other Etiologies*

The clinician must always bear in mind unusual causes for vocal cord paralysis should the more common causes prove to be not responsible. Several neurological causes may affect vocal cord movement including Parkinson's disease, multiple sclerosis, amyotrophic lateral sclerosis, Guillain-Barré syndrome, and myasthenia gravis.[58-63] Whilst cerebrovascular accidents and central nervous system tumours may result in vocal cord paralysis, typically other neurons are also affected, causing additional symptoms.[64]

Additionally, there are reported cases of left RLN paralysis caused by pathologies which may compress the RLN as it passes through the chest. Aortic aneurysms and pulmonary artery enlargement secondary to pulmonary hypertension have been reported to directly compress the RLN.[65] Processes which may result in mediastinal lymphadenopathy can likewise compress the RLN, including tuberculosis, granulomatosis with polyangiitis, sarcoidosis, pneumoconiosis, and systemic lupus erythematosus.[66,67]

Idiopathic etiologies have no cause by definition. However, the true incidence of idiopathic RLN paralysis is related to the thoroughness of the investigative evaluation and therefore the true rate remains unknown. As technological advances are made in imaging, fiberoptics and viral titres, the rate of RLN paralysis deemed to be idiopathic has decreased.[15] It is interesting to note that the incidence of "idiopathic vocal cord paralysis" varies between different nations, with English literature supporting a rate of 10–27% whilst Japanese literature reports a rate of 25.9–41.3%. Spontaneous recovery in idiopathic vocal cord paralysis has been reported in up to 24% of cases, with the suggestion that these were potentially caused by undiagnosed viral illness.[68]

## Evaluation

### *History*

The patient with a suspected vocal cord paralysis requires a systematic and comprehensive evaluation beginning with a history and physical examination. The purpose of the history should be to accurately define the patient's symptoms, the impact of these symptoms on their quality of life, and to ascertain the likely cause of their vocal cord hypomobility or paralysis.[1] A recommended approach is to consider the symptoms of the palsy itself, the symptoms of vocal compensation, and to establish symptoms to potential favorable or unfavorable synkinesis.

Symptoms of vocal cord palsy can be broadly categorized into the impact on voice, impact on breathing or airway, and impact on patient swallowing ability.[69] Patients with vocal cord paralysis may complain of symptoms regarding their projection, pitch and voice quality and each of these must be elucidated. Patients may complain of a reduction in the subjective loudness or projection of their voice, whereby patients cannot yell or their voice "goes away" when attempting to project (or becomes more abnormal). Likewise, patients may express concern regarding an alteration in their normal vocal pitch, with a reduction in pitch range, or more often a higher baseline vocal frequency, known as paralytic falsetto.[69] Breath-wasting phenomena may also occur, owing to reduced ability to properly adduct the vocal cords, resulting in a glottic gap, or an inability to maintain glottic closure with sufficient force, leading to a "rough voice" or breathlessness during speech, classically presenting as worsening dysphonia (breathy voice) throughout the day, with greater levels of fatigue at the end of a day of talking.[14] Airway concerns regarding vocal cord paralysis may present themselves as difficulty with heaving lifting or straining (due to an impaired Valsalva maneuver secondary to weak glottic closure), a baseline breathy voice, and rarely stridor or respiratory obstruction due to vocal cord prolapse into the glottic opening.[15] Complaints of voice changes with associated cough, globus sensation, and/or choking following oral intake may be suggestive of aspiration.[14] Subclassification and further exploration of these symptoms may yield valuable information and help to differentiate between isolated RLN palsy and high vagal lesions. Patients who notice little to no swallowing symptoms, or those who only have difficulty with thin liquids are more commonly found to have isolated RLN palsy, conversely, patients with more severe dysphagia for both solids and liquids, and who have ancillary symptoms such as nasal regurgitation of oral intake (suggesting pharyngeal and hypopharyngeal paralysis) are more likely to have a high vagal lesion.[69]

Patients with vocal cord paralysis, especially after a period of time, may acquire compensatory mechanisms to overcome their reduced vocalization. Unfortunately, as is often the case with compensatory mechanisms, these may lead to problems in their own right. Patients may attempt to compensate for their vocal changes by increasing pharyngeal squeeze to facilitate vocalization, however with time this may leading to muscle tension dysphonia and strain or soreness in the neck with voice use, or odynophonia.[5] Ventricular dysphonia, also known as dysphonia plica ventricularis is defined as phonation using false vocal fold vibration rather than true vocal cord vibration, and is most commonly associated with severe muscle tension dysphonia or may occasionally be seen as a compensatory mechanism for true vocal fold paralysis.[70] Because of the structural and vibratory characteristics of the false cords, patients may complain of a low-pitch voice (owing to the increased mass of the false cords compared to the true cords). Additional typical symptoms of ventricular dysphonia include a harsh, rattling, rumbling or cracking voice, because the false cords are not as sophisticated in their vibratory patters as the true vocal cords.[70] Less commonly, patients report somatic symptoms such as effortful phonation, unproductive throat-clearing and pain or globus sensation.[70]

The progression, stability, or improvement of patients' dysphonia may hint at the presence of synkinesis (either favorable or unfavorable) or lack thereof. Careful attention should be paid to the timeline of patient symptoms, with initial dysphonia that slowly improves over subsequent weeks and months (however usually not returning to normal) indicating favorable synkinesis,[71] and ongoing deterioration either in voice quality or regarding aspiration episodes developing weeks after the onset of dysphonia suggesting the presence of unfavorable synkinesis.[71] Similarly, it is important to note that some patients may experience complete spontaneous recovery of their voice, implying partial RLN injury which has successfully recovered, or an otherwise self-limiting cause such as

viral infection or idiopathic RLN paralysis, which may completely recover in up to 24% of cases.[15,68]

Self-assessments are often used to quantify the severity of voice-related symptoms, and can be used to monitor treatment response.[72] Patient-reported outcome measures are questionnaires which patients complete and assess the subjective experience of their symptoms in both quality and severity. They are a reliable way to measure a patient's sense of symptom improvement postintervention[72] with recent studies demonstrating a reliable improvement in the Voice Handicap Index in a cohort of patients with unilateral vocal fold paralysis who underwent arytenoid adduction.[73] Similarly, a recent study examined the voice characteristics of patients with known vocal fold paralysis specifically through the Voice Symptom Scale and found that limitation score and voice roughness score were reliably elevated in these patients compared to patients without a vocal cord palsy.[74]

In addition to a vocal history, the clinician should inquire about past medical history, including past infections, previous surgeries, prolonged intubation, and trauma. Special attention should be made to timing between the aforementioned and onset of perceived voice changes, as this may suggest possible causal relationship or mechanism involved. A complete medical history should be taken, with particular attention to neurological symptoms, smoking and alcohol history, and questions pertaining to possible malignancy.[1]

### *Physical Examination*

A complete examination of the head and neck should be performed next, with particular attention to the cranial nerves, especially the vagus nerve. Palpation for neck masses may reveal a thyroid mass or cervical lymphadenopathy. An absent gag reflex or a deviated palate may reveal a high unilateral vagus nerve lesion.[75] The clinician must also listen to the patient's voice and follow this with direct visualization of the larynx. Historically a mirror examination was performed first, but this has been largely replaced with either rigid or flexible endoscopic examination.[72] Findings from direct visualization can be divided into laryngeal findings and extra-laryngeal findings. When specifically assessing for laryngeal changes, it is important to appreciate both the motor and sensory findings that may be present. Motor findings are the most commonly examined, and pertain to vocal cord abnormalities as seen endoscopically during rest, with volitional movement, and with repeated movements. During quiet breathing with the endoscope in appropriate position, the larynx can be examined for resting position of the vocal cord (midline, paramedian, or intermediate), position and direction of the vocal processes, symmetry of vocal fold contour (with particular attention to presence of vocal fold atrophy, ventricle capaciousness and fullness of the conus elasticus), synechia in the posterior glottis, and scarring along the cricoarytenoid joints.[69] After the clinician is pleased with the assessment of the resting vocal cords, the patient should be instructed to phonate. During phonation the mobility of the membranous part of the vocal cords should be assessed, with particular attention to degree and symmetry of adduction and abduction, completeness of glottic closure or presence of glottic gap, the level of match between the vocal processes and flaccidity of vocal fold structures. Particular maneuvers are useful for aiding in the assessment of vocal cord paralysis, including asking the patient to perform sound /i/ for assessing adduction or gently inhaling to assess abduction, and a glissando maneuver (asking the patient to slide from their lowest to highest frequency and then back down) is useful for assessing SLN function.[1] Abduction in particular is perhaps the most useful maneuver assessing the presence of vocal cord paralysis or paresis.[14] Lastly, where the vocal cord paralysis is not complete, repetitive phonation (alternating a sniff with the sound /i/) may reveal fatigability of the affected side.[1] Markedly reduced or absent sensation during examination may indicate SLN involvement, when this is suspected a functional endoscopic evaluation of swallowing should be performed, as patients with unilateral vocal cord palsy have higher rates of pooling, penetration, and aspiration compared to patients without vocal cord palsy.[69,76] Furthermore, recent studies have demonstrated that the degree of sensory deficit as measured by calibrated air pulses correlates with the degree of dysphagia and aspiration.[76]

Extralaryngeal findings may also support the diagnosis of vocal cord paralysis and establish the functional status of the pharynx and hypopharynx. Unilateral secretions pooling in the pyriform sinus or a dilated pyriform sinus may indicate pharyngeal weakness ipsilaterally. Likewise, with unilateral pharyngeal paralysis, the posterior midline raphe is pulled toward the contralateral side with patient phonation.

When recurrent laryngeal nerve paralysis is present, the ipsilateral vocal cord appears to be immobile, with the exception of slight respiratory movement likely due IA and supraglottic muscle fibres as well as elastic and aerodynamic forces on the paralyzed vocal cord.[77] The contralateral vocal cord must be assessed for degree of movement, to ascertain whether it is able to cross the midline and allow for reasonable glottic closure and facilitate some phonation, as well as ensuring a safe and patent airway after any medialization is performed on the paralyzed side.[77]

If the SLN is intact, the CT muscle is still innervated, and with this the ability to alter longitudinal tension maintained. Therefore, the vocal processes will be at the same level, with the paralyzed vocal cord lengthening as pitch is increased.[1] If the SLN is injured, then the ipsilateral vocal fold may appear bowed, and typically lies in a lower plane due to reduced CT muscle tone. Compensation in this setting is more difficult, and the result is often a more impaired voice, with greater effects on vocal quality, volume, and pitch control.[77] This is especially true in cases where both the RLN and SLN are paralyzed, however problems may still occur in isolated SLN injury where adduction and abduction are preserved.[1]

### *Stroboscopy*

Stroboscopy is a method of endoscopic examination, which can be performed *via* flexible or rigid endoscope that uses pulsed light to better capture vocal cord mucosal oscillation, or mucosal wave. The light pulses incongruently with the glottal cycle, generating a series of still images during different points of the glottal cycle that are then interpreted into a fluid and continuous sequence in the examiner's eye.[78] Stroboscopy is the best diagnostic instrument for the evaluation of most cases of dysphonia and offers valuable information about mucosal pliability.[72] Stroboscopy can provide useful information on the glottic cycle regarding regularity, amplitude, mucosal wave, phase symmetry, vertical level, and glottic closure patterns.[72] If the video recording aspect of stroboscopy is utilized, the examination may be further enhanced by slowing down the replay time, and freeze-framing, allowing more subtle examination findings to be detected, such as sulci and mucosal pliability alterations. Additionally, video archiving allows for more accurate comparison of examinations across time as well as postintervention. In cases of suspected vocal cord paralysis, stroboscopy allows for better viewing of subtle

movements of the vocal processes and body of the vocal folds, aiding to differentiate from severe vocal fold paresis and true paralysis, as well as concurrently enabling the clinician to better assess the contralateral vocal fold for subtle mucosal or movement pathologies.[77] Stroboscopy is an especially potent tool in the arsenal of the laryngologist whenever the findings of continuous light examination do not explain the severity of the patient's symptoms, when hoarseness persists post-treatment of its assumed cause, and for cases of unexpected hoarseness postmicrolaryngoscopy.[79–81]

### *Diagnostic Imaging*

In cases of complete vocal cord paralysis, a complete diagnostic imaging evaluation should be performed including a CT from the skull base through the chest, tracing the path of the vagus and recurrent laryngeal nerves. Additionally, an MRI brain is recommended to exclude intracranial pathologies such as multiple sclerosis or mass lesions, especially when no cause is identified through initial evaluation and a CT scan, or when vagal symptoms are present.[69] A recent prospective study of 53 patient with vocal cord paralysis demonstrated cancer as the cause after CT evaluation in 27 (51%) with 15 patients (28%) having vocal cord paralysis as their primary presenting complaint.[82]

### *Laryngeal Electromyography*

Laryngeal electromyography (LEMG) is considered the gold standard investigation to diagnose and evaluate the type and degree of neurological injury in vocal cord paralysis.[83] LEMG utilizes needle electrodes to record electrical activity from muscle fibres, including motor unit recruitment, configuration and detections of fibrillation or synkinesis, to estimate the severity of injury and prognosticate the likelihood of recovery, thereby providing additional information for clinicians to discuss management options with patients.[84] In vocal cord paralysis due to lower motor neuron injury (e.g., surgical injury), initially there is complete electrical silence, followed by the appearance of positive sharp waves or fibrillations within 2–3 weeks, indicating denervation and axonal loss.[85] Reinnervation is characterizedd by the recording of larger motor unit activity with characteristic high-amplitude, long-duration, and polyphasic responses.[85] Reinnervation findings may occur after several months and are a positive prognostic indicator, especially in young patients.[86] Recent studies have incorporated quantitative data analysis examining presence synkinesis and characteristics of reinnervation with promising results, with one study of 23 patients with acute vocal cord paralysis demonstrating a 100% positive predictive value and 89.5% negative predictive value in a 6-month return of function prognostication.[84]

Laryngeal electromyography is also well suited to differentiate between vocal cord paralysis due to RLN injury and an apparent vocal cord paralysis due to other conditions such as myopathy, upper motor neuropathy, or CT joint hypomobility.[85] In myopathic processes, there is rapid recruitment of motor units, with low voltage and short duration, in keeping with the context of a weak and short muscle contraction.[85] Likewise, in upper motor neuron disorders, the amplitude and duration of the motor unit potential is normal, but there is reduced motor unit recruitment and a reduced motor unit firing rate. In patients with isolated CT joint hypomobility, a normal LEMG result would be expected. In the setting of vocal cord paralysis, it can be difficult to appreciate contralateral vocal cord paresis, especially if subtle, with LEMG offering a more quantitative tool for analyzing the contralateral cord and recording the findings for further monitoring and prognostication.[14,85]

One of the limitations of LEMG is the qualitative nature of the skill of the electromyographer doing the assessment. This is a particular concern in the larynx where muscle bundles are small, overlap, and are adjacent to other muscles with different functions.[77] And whilst several studies have reported the utility of LEMG in providing valuable information for predicting the likelihood of recovery, LEMG findings alone may not change clinician's approach to management in patients with an acute vocal cord paralysis.[84–86]

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