53 Vocal Cord Paralysis

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The competencies covered in this chapter are as follows:

EN4.45 Describe the clinical features, investigations, and principles of management of vocal cord palsy.

AN38.3 Describe the anatomical basis of recurrent laryngeal nerve injury.

Introduction

Vocal cord paralysis or vocal fold paralysis (VFP) is due to a reduced or absent movement of one or both vocal folds. A neural or neurological cause is the main etiological factor for a vocal cord paralysis, and it is rarely due to neuromuscular junction disorders. The morbidity due to the paralysis can adversely affect the patient's quality of life.

Vocal cord paralysis can be unilateral or bilateral. It may involve the recurrent laryngeal nerve (RLN) or superior laryngeal nerve (SLN), or both. The glottic airway, phonation, protection of lower airway, and swallowing can get affected depending on whether one or both cords are involved. Males are more commonly affected than females (8:1).

The symptoms depend on whether it is a unilateral or bilateral cord palsy. The other associated symptoms, based on the etiology, include:

- Referred otalgia.
- Cough.
- Hemoptysis.
- Neck lumps.

Unilateral and bilateral cord paralysis symptoms vary due to the functional movement of the cords. Abduction is required for respiration, while adduction is necessary for phonation and prevention of aspiration. There will also be an inability to obtain a positive subglottic pressure which is important for straining (Valsalva maneuver) and phonation.

The subspeciality of laryngology has brought in better diagnostic tools, clinical assessment, and treatment options in the management of unilateral vocal cord palsy.

Pearl

VFP can be a sign of an underlying disease in the neck or thorax and investigations should include these two regions.

Definitions

Vocal cord paralysis is complete absence of movement (immobility) of the vocal cord.

Vocal cord paresis is a condition where there are weak or varying degrees of movement (impaired mobility) of the vocal cord.

Laryngeal Innervation

Phonation is initiated by *Area 4* in the Sylvian fissure of the cerebrum. The vagal nuclei are thus bilaterally innervated. The nucleus of the vagus nerve is in the brainstem (medulla), that is, nucleus ambiguus. It passes through the jugular foramen and travels below in the carotid sheath. Three branches are given off in the neck (**Fig. 53.1**):

- Pharyngeal branch.
- Superior laryngeal nerve.
- Recurrent laryngeal nerve.

The SLN divides into two branches:

- Internal laryngeal nerve.
- External laryngeal nerve.

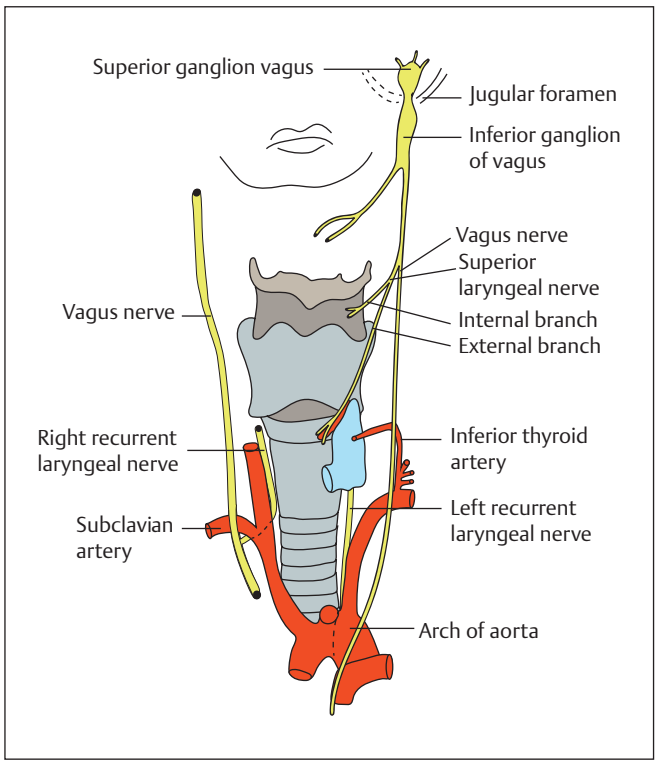

Fig. 53.1 The vagus nerve and its branches.

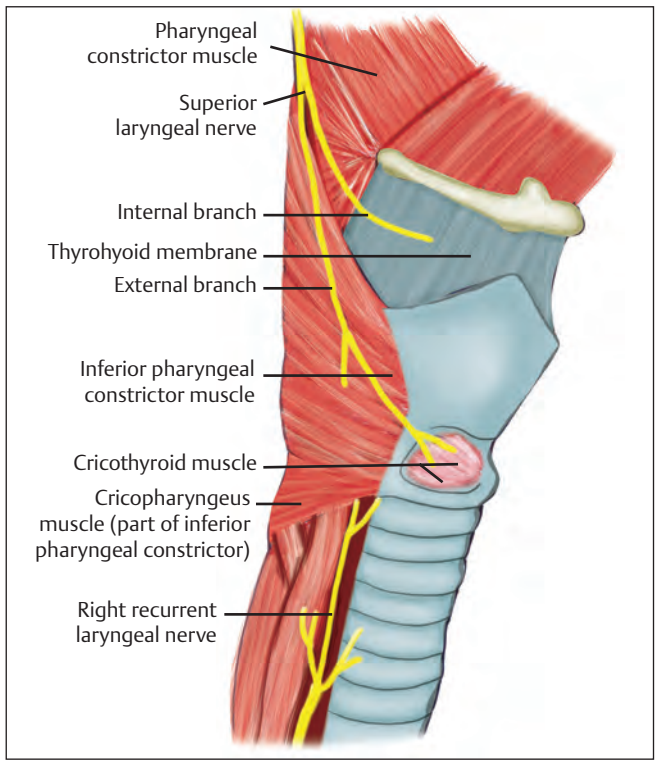

Fig. 53.2 Branches of the superior laryngeal nerve.

The *internal branch of the SLN* is a sensory branch providing sensation to the supraglottis which is above the level of the vocal cords. The *external branch of the SLN* is a motor branch innervating the cricothyroid muscle (**Fig. 53.2**).

The origin of the RLN from the vagus is in the superior mediastinum. On the left side the vagus descends posteromedial to the aortic arch, loops around it through the aorticopulmonary window. On the right side, it descends posterior to the subclavian artery, loops around it to ascend upwards into the neck. As it courses upwards it is situated in or adjacent to the tracheoesophageal groove. It enters the larynx posterior to the cricothyroid joint. The RLN supplies all the muscles of the larynx except for the cricothyroid muscle (**Fig. 53.3**).

The RLN supplies the unpaired interarytenoid muscle; therefore, it receives bilateral innervation. This allows adduction of the vocal cord during ipsilateral RLN paralysis. The cricothyroid muscle is a tensor of the vocal cord and does not adduct or abduct the cords.

The final position of the vocal cord is not static and results from a number of forces such as the degree of muscle atrophy, the degree of reinnervation, and the extent of synkinesis (mass movement).

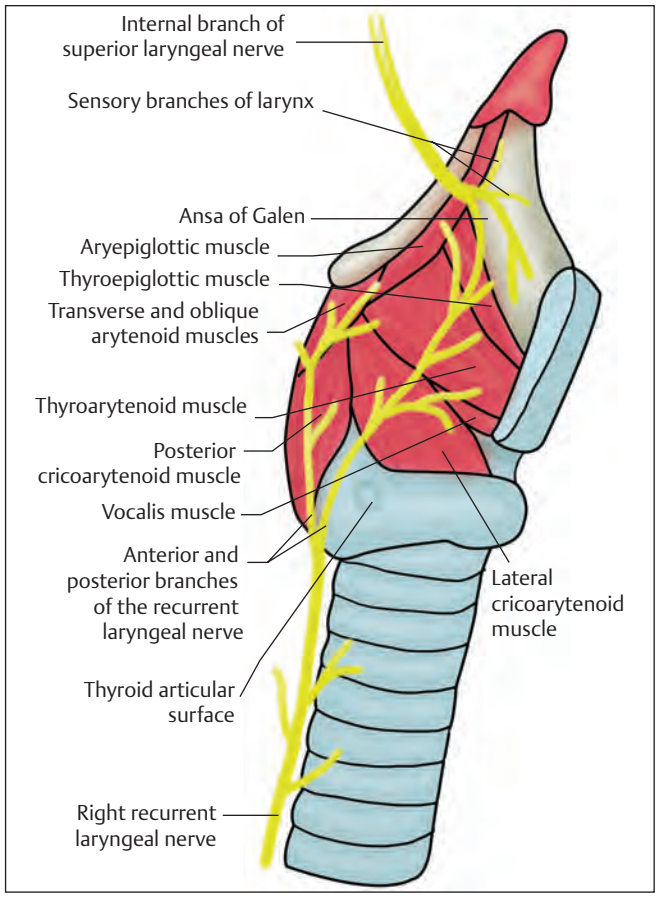

Fig. 53.3 Branches of the recurrent laryngeal nerve.

Muscles Acting on the Vocal Cord

The intrinsic muscles of larynx are those which act on the vocal cord and laryngeal inlet.

Vocal cord adduction is movement of the vocal cords toward the midline. This is carried out by adductor muscles which include:

- Lateral cricoarytenoid.
- Interarytenoid (transverse arytenoid).
- Thyroarytenoid (external part).

Vocal cord abduction (movement of vocal cords away from the midline) is carried out by the sole abductor which is the posterior cricoarytenoid muscle. The cricothyroid and vocalis (internal part of thyroarytenoid) help in tensing or lengthening of the vocal cord and are called tensors.

Pathophysiology

Palsy or paresis of the vocal cord in most cases is due to neural involvement which can affect the motor and sensory nerves. Pathology in the brain and cricoarytenoid joint fixity or dislocation can also affect vocal cord mobility.

The left RLN has a longer course and is more prone to chronic demyelination neuropathy. Injury to the RLN can also cause demyelination, and the rate of recovery depends on the severity of the injury. When Wallerian degeneration occurs due to a severe injury, recovery will depend on the number of viable fibers and the gap between the cut ends of the nerve. The human RLN has a strong tendency for reinnervation. Reinnervation seems to be the rule. Due to this tendency for reinnervation, the position of the vocal cord varies with time and there will be a gradual improvement in the voice. This improvement is also due to the compensatory action by the contralateral vocal cord.

For the vocal cords to vibrate, or for alternate opening and closing, two antagonistic forces are required to work, out of phase with each other, on the vocal cords. The opening force derives from subglottal pressure, whereas the closing force stems from elastic recoil and the Bernoulli effect.

Pearl

One should be able to differentiate between fixity and paralysis of the vocal cord as the pathological processes involved are usually different.

Causes of Vocal Cord Paralysis

The causes of vocal cord paralysis in general are:

- Malignant diseases like bronchial carcinoma, esophageal carcinoma, and thyroid carcinoma.
- Surgical trauma like thyroid surgery, congenital heart surgery, cervical spine surgery, and skull base surgery.
- Nonsurgical trauma like road traffic accident, violence (blunt or penetrating injury to neck or chest), and excessive cuff pressure of endotracheal tube.
- Idiopathic (possibly viral neuritis).
- Central neurological causes like cerebrovascular accidents and multiple sclerosis.
- Inflammatory causes like pulmonary tuberculosis and neck abscess.
- Neuromuscular disorders like myasthenia gravis and Eaton-Lambert syndrome.
- Miscellaneous causes like collagen diseases.

Anatomical Basis of Laryngeal Paralysis

Laryngeal paralysis may be unilateral or bilateral and may involve (1) RLN, (2) SLN, or (3) both recurrent and SLNs (combined or complete paralysis).

The causes for laryngeal paralysis could be at the following levels:

The cause may be central or peripheral, congenital, or acquired.

Central causes may be supranuclear, nuclear, or infranuclear (based on the nucleus ambiguus—upper and lower motor neurons synapse here):

- *Supranuclear*: Supranuclear lesions of the cortex leading to isolated paralysis of the vocal cord are extremely rare. It may be due to massive, diffuse bilateral lesions in the brain.
- *Nuclear*: There is involvement of nucleus ambiguus in the medulla. The causes are vascular, neoplastic, motor neurone disease, polio, and syringobulbia. Nuclear lesions have an associated paralysis of other cranial nerves and neural pathways (**Table 53.1**).
- *Infranuclear*:

Table 53.1 Causes of combined paralysis, both recurrent and superior laryngeal nerves

Location of lesion*	Etiology for paralysis
Intracranial	Posterior fossa lesions Tubercular meningitis Cerebrovascular accidents Arnold-Chiari malformation Meningomyelocele Hydrocephalus
Skull base	Fractures Nasopharyngeal carcinoma Skull base paraganglioma Surgical procedures
Neck	Penetrating/surgical trauma Parapharyngeal tumors/abscess Metastatic nodes Lymphomas

*The lesions are high vagal lesions and those involving nucleus ambiguus.

- *High vagal lesions*: Vagus nerve may be involved in the skull base areas, at the level of jugular foramen or parapharyngeal space.
- *Low vagal nerve or RLN* injury.

It is idiopathic in about 12 to 22% of cases. The cause remains obscure.

Anatomical Basis of Recurrent Laryngeal Nerve Paralysis

Recurrent Laryngeal Nerve

The RLN provides motor nerve to all of the intrinsic laryngeal muscles, except cricothyroid, and sensory nerve to the mucosa at and below the level of the vocal cords. The right RLN arises from the vagus at the level of subclavian artery, hooks around it, and then ascends (initially at an angle) between the trachea and esophagus, parallel to the tracheo-esophageal groove.

The left RLN arises from vagus in the mediastinum at the level of arch of aorta, loops around it, and then ascends into the neck in the tracheo-esophageal groove. The left recurrent has a longer course than right and is more prone to paralysis. The ratio of injury is 4:1 in comparison to right side.

The important relations of the RLN in the neck and thorax are outlined in **Table 53.2**. Lesions in these structures can cause paralysis.

The various etiologies for recurrent laryngeal paralysis are mentioned in **Table 53.3**.

Pearl

Although left vocal cord palsy is more common, during thyroidectomy the right RLN is more likely to be injured as it is placed away from the tracheo-esophageal groove.

Pearl

A *non-RLN* arises directly from the vagus and enters the larynx. It may be seen on the right side especially when there is an aberrant right subclavian artery. It occurs in 1% of cases. The nerve may be more prone to injury during thyroid surgery if the surgeon is not aware of this condition.

Systemic disease leading to vocal cord paralysis include the following:

- Diabetes mellitus.
- Rheumatoid arthritis.
- Collagen vascular diseases.
- Viral infections.

Table 53.2 Important relations of the recurrent laryngeal nerve

Right side	Left side
Right subclavian artery	Aortic arch
Apex of right upper lobe of lung	Esophagus
Supraclavicular lymph nodes	Left mainstem bronchus
	Mediastinal lymph nodes
	Left atrium

Table 53.3 Etiology of recurrent laryngeal nerve paralysis

Unilateral—right or left side	Left side	Bilateral
Congenital conditions *Postsurgical trauma*: Post thyroid/parathyroid surgery, tracheostomy, esophageal and lung surgery for carcinoma, partial laryngectomy, neck dissection, removal of pharyngeal pouch, anterior approaches for cervical spine fusion *Nonsurgical neck trauma,* including birth trauma, i.e., use of forceps, blunt or penetrating injury, cuff of endotracheal tube *Malignant disease*: Malignancy of thyroid, cervical esophagus, nasopharynx, larynx *Cervical lymphadenopathy*: Metastatic, tuberculosis, lymphoma *Inflammatory causes*: Pulmonary tuberculosis, parapharyngeal abscess; viral neuritis due to influenza or infectious mononucleosis viruses *Aneurysm* of subclavian artery *Neurological diseases*: Cerebrovascular accident, multiple sclerosis, amyotrophic lateral sclerosis, syringomyelia and Parkinsonism, Guillain-Barre syndrome *Idiopathic*	*Mediastinum*: • Bronchogenic carcinoma • Pulmonary tuberculosis • Aneurysm of aorta • Mediastinal mass **(Fig. 53.4)** • Mediastinal lymphadenopathy **(Fig. 53.5)** • Ortner's syndrome (enlarged left atria) • Intrathoracic surgery—patent ductus arteriosus, tracheo-esophageal repair, cardiac/pulmonary surgery • Sarcoidosis • Syphilitic aortitis	Congenital Post thyroid/parathyroid surgery Malignancy of thyroid or cervical esophagus Cervical lymphadenopathy Guillain-Barre syndrome Idiopathic

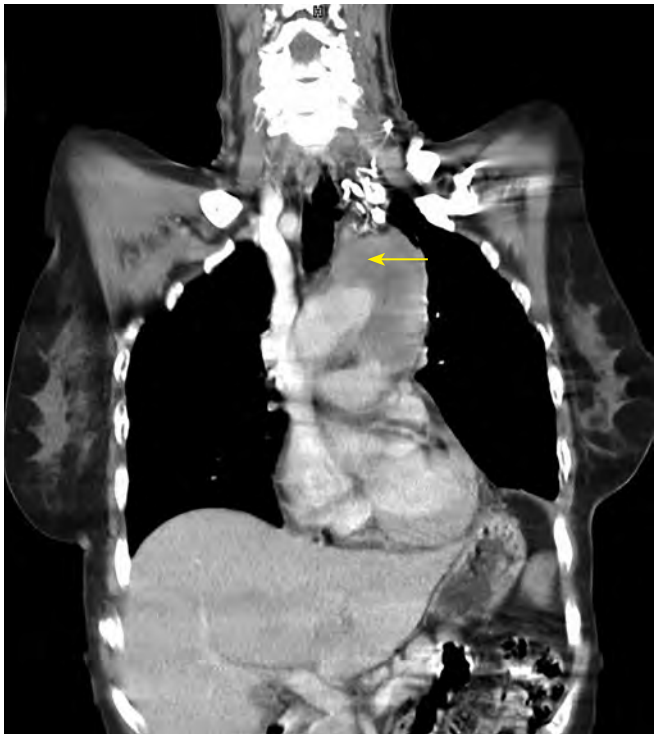

Fig. 53.4 Mediastinal mass causing left recurrent laryngeal nerve palsy.

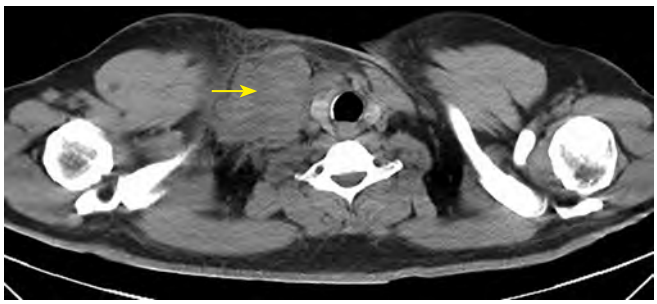

Fig. 53.5 Mediastinal lymphoma leading to right recurrent laryngeal nerve palsy.

- Gout.
- Polyarteritis nodosa.
- Toxic neuritis (lead, zinc, alcohol).
- Granulomatosis with polyangiitis (Wegener's granulomatosis).
- Relapsing polychondritis.
- Drug toxicity (vincristine, organophosphates).
- Radiation to the head and neck region.

Causes for SLN Palsy

- Surgical procedures like thyroidectomy, neck dissection, carotid endarterectomy, cricopharyngeal myotomy, supraglottic laryngectomy, and anterior approach to cervical spine.
- Laryngopharyngeal and thyroid malignancy.
- Neuritis.

Unilateral Vocal Cord Paralysis

Lesion or injury at the level of the RLN is the common cause for unilateral vocal cord paralysis. Lesions at the brainstem and along the path of the vagus nerve can also cause unilateral vocal cord palsy.

Etiology

The causes for unilateral vocal cord palsy are varied (**Table 53.3**). Iatrogenic surgical traumas, as in during thyroidectomy, esophagectomy, and anterior approach to the cervical spine, are some of the common etiological factors.

History

In addition to the onset, duration, and severity, a history of trauma, surgery, systemic illnesses, and recent infections should be documented. Associated symptoms of cough, aspiration (cough on swallowing), dysphagia, odynophagia, and breathing difficulty should be elicited.

Voice Quality

- Patients with unilateral vocal cord palsy may go undetected with minor change in voice without any other symptoms.
- Change in voice is the main symptom. The voice will be *breathy or weak* voice due to imperfect closure of the glottis. The muscle tone and the position of the vocal cord defines the quality of the voice.
- "Paralytic falsetto," a high-pitched voice, that occurs due to contraction of the ipsilateral cricothyroid muscle as a compensatory mechanism, may be present.
- There will be vocal fatigue and an inability to lift, pull, or push objects.

Dysphagia with aspiration can occur when there is vagus nerve involvement at a higher level. The pharyngeal constrictors are affected, and there is loss of sensation in the larynx.

Shortness of breath can occur due to incomplete glottic closure with air leak during phonation.

Pearl

The pharyngeal branch of the vagus, through its innervation, plays an important role in the elevation of the larynx and in regulating the upper esophageal sphincter pressure which is important for swallowing and for protecting the lower airway.

Clinical Examination

Larynx

- Flexible fiberoptic laryngoscopy is done to assess the mobility of the vocal cords. Rigid endoscopy and indirect laryngoscopy involve grasping the tongue which can affect the movements of the larynx or vocal cords.
  - When the patient says "eee" the vocal cords adduct. In vocal cord palsy, the position of the affected vocal cord may be median, paramedian, or cadaveric, depending on the nerves involved. Reinnervation and synkinesis can later affect the vocal cord position (**Fig. 53.6**).
  - Falling forward/anteriorly of the arytenoid on the affected side can occur due to total denervation or incomplete reinnervation of the thyroarytenoid muscle.

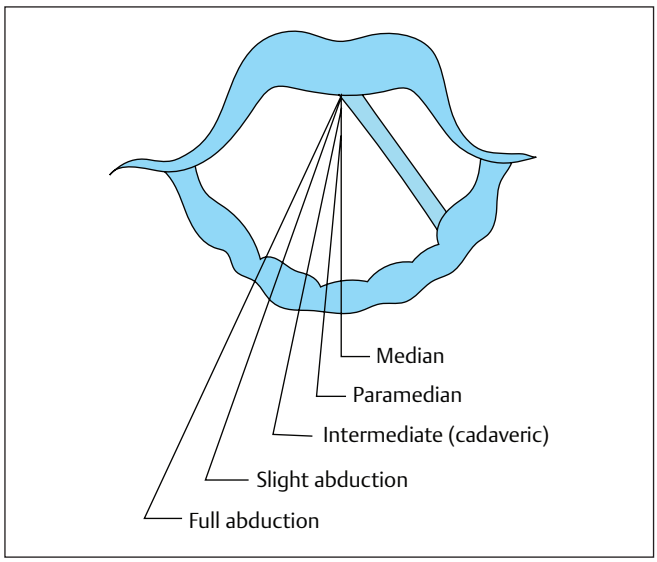

Fig. 53.6 Various positions of the vocal cord during paralysis.

- Maximal phonation time (MPT) gives the degree of air leak (glottic incompetence). The patient is asked to take a deep breath and say "eee" for as long as possible. In a normal individual, MPT is 25 seconds. But with a paralyzed vocal cord, the duration can be reduced to 5 to 10 seconds depending on the degree of glottic air leak.
- Manual compression test: It involves compression of the thyroid alae medially during phonation using the thumb, index, and middle fingers at the level of the vocal cord to assess any changes in voice. The test should be repeated with varying forces at various sites. If significant improvement in voice is noted with the procedure, the patient could have good prognosis following surgery.

Sometimes it is important to distinguish between a paralyzed vocal cord from one which does not move due to fixity of the cricoarytenoid joint. Fixity prevents the arytenoid from rotating. The main differences between the two are outlined in **Table 53.4.**

In *unilateral RLN palsy*, the vocal cord will be in the paramedian position. There may be a slightly breathy voice. On examination, the vocal cord will be in the paramedian position which leads to a small phonatory gap. Compensation by the opposite vocal cord will occur. Later, mainly the singing voice will be affected (**Figs. 53.7** and **53.8**).

In *unilateral SLN palsy*, due to paralysis of cricothyroid, there may be diplophonia, inability to change the pitch (not able to achieve a high pitch), especially for singers, monotony of voice, and vocal fatigue. Due to anesthesia of larynx above the level of vocal folds, there may be paroxysmal coughing, frequent clearing of the throat, and foreign body sensation.

In *unilateral combined palsy*, there will be severe breathiness of the voice. There will be aspiration. The cough will be ineffective. On examination, the vocal cord will be in the intermediate position and the opposite cord will not be able to compensate. There may be pooling of saliva in the pyriform fossa.

Table 53.4 Difference between vocal cord paralysis and cricoarytenoid joint fixation on endoscopy

Characteristic feature	Vocal cord paralysis	Cricoarytenoid joint fixation
Bowing of vocal cord	Present	Absent
Floppy vocal cord	Present	Absent
Position of cord	Higher	Normal
Tilt of larynx	To paralyzed side	Absent
Flicker of movement on phonation	Present	Absent
Pyriform fossa	Shallow	Normal
Position of cord	Depends on type of palsy	Any position
Arytenoid position	Falls forwards	No movement
Arytenoid mobility on palpation (endoscopy)	Mobile	Fixed

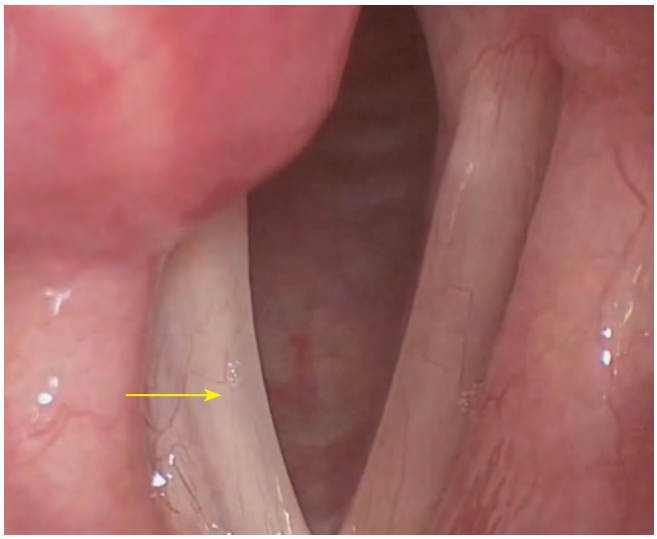

Fig. 53.7 Right vocal cord paralysis (*yellow arrow*). Left vocal cord in full abduction.

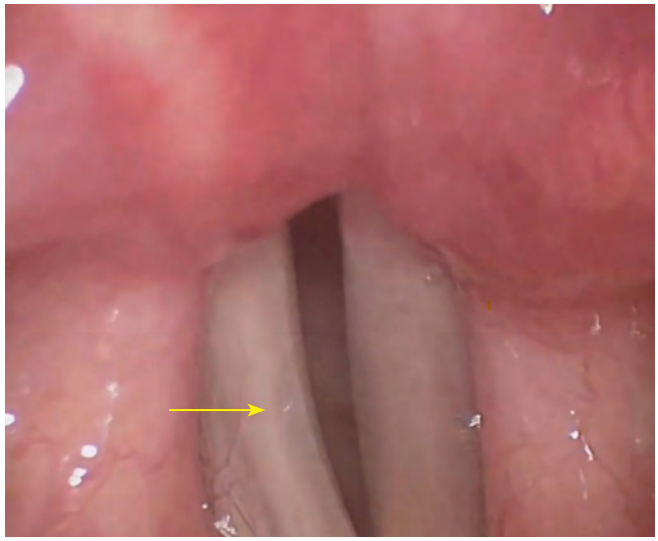

Fig. 53.8 Right vocal cord paralyzed (*yellow arrow*), with left vocal cord in full adduction while phonating. Note the space between the two vocal cords.

Pearl

Patients with high vagal lesions above the level of the pharyngeal nerve will present with nasal regurgitation and dysphagia in addition to change in voice and aspiration.

Neck

Examination for cervical lymphadenopathy, thyroid gland swelling/solitary nodule, and other neck masses should be done.

Cranial Nerves Examination

The function of the cranial nerves, especially IX, X, XI, and XII, should be examined to assess for generalized neuropathy. If the vagus is affected at a higher level, the soft palate will move toward the uninvolved side. The gag reflex should also be noted.

Pearl

Wagner and Grossman theory states that in unilateral RLN paralysis, the vocal fold is in the paramedian position. The adduction action of the cricothyroid pulls the paralyzed cord toward the midline. The vocal fold paralyzed in the intermediate position is due to paralysis of the SLNs and the RLNs.

Investigations

- Endoscopy of larynx with rigid or flexible scopes and stroboscopy are useful for assessing the movement of the vocal cords, and to look for any local pathology.
- Laryngeal electromyography can be prognostic when done in the 6 months of the palsy, as it provides information on the integrity of laryngeal innervation.
- Voice recording for the purpose of acoustic analysis and aerodynamic measurements are other useful tests.
- Preoperative evaluation includes a variety of subjective and objective voice parameters such as maximum phonation time, amplitude perturbation (shimmer), and pitch perturbation (jitter).
- Imaging studies are done when there is no known traumatic cause for the vocal cord palsy. A computed tomography (CT) scan with contrast or magnetic resonance imaging (MRI) of the neck (including skull base) and thorax is done to identify the presence of any lesion. When there is palatal paralysis and vocal cord paralysis, MRI should include the brainstem. MRI of the brain may be required for posterior fossa lesions.
- Esophagoscopy or barium swallow can be done if there is history of dysphagia.

Features of unilateral RLN palsy on flexible endoscopy:

- Immobile cord in the paramedian position.
- Falling forwards of the arytenoid.
- Bowing of the affected cord.
- Affected cord at a lower level when compared to the normal cord (during phonation).
- The ventricle may appear prominent/roomy.
- Compensation by the opposite cord (by shortening to allow the vocal processes to be opposite each other) helps close most of the glottis chink, except posteriorly.
- The ipsilateral pyriform fossa may appear shallow.

Features of unilateral SLN palsy on flexible endoscopy:
- Askewed position of glottis.
- Shortening of cord with loss of tension or bowing.
- Cord may be at a lower level.
- Wavy, asymmetrical cord seen during phonation at high pitch.
- Posterior commissure points toward the paralyzed side.

Treatment

Four treatment options can be considered in unilateral vocal cord paralysis:
- *Observation* with regular follow-up for 6 to 9 months to assess for recovery or compensation.
- *Treat the cause* when feasible. Steroids may be helpful especially in idiopathic cases and in suspected viral etiology.
- *Voice therapy* or vocal cord strengthening exercises. Swallowing therapy especially in combined palsy.
- *Surgery*: Although the aim is to improve the voice, in combined palsy, the more important indication is to protect the lower airway and prevent aspiration.
  - Temporary: Medialization laryngoplasty, i.e., augmentation of the vocal fold with a filler substance like gelfoam, fat, collagen derivatives, or teflon. This can be done under local anesthesia transcutaneously with fiberoptic endoscopy guidance, or under general anesthesia using an operating microscope.
  - Permanent:
    - *Medialization thyroplasty*: This type I thyroplasty can be done using silastic, titanium, or Gore-Tex. It is done early in patients with aspiration. The silastic and titanium can be removed to reverse the procedure.
    - *Arytenoid adduction* procedure can be done along with medialization thyroplasty for better posterior glottic chink closure.
    - *Reinnervation* procedures, like end-to-end anastomosis, hypoglossal-to-RLN anastomosis, and ansa hypoglossi-to-RLN anastomosis, may help obtain neural continuity.
    - *Cricopharyngeal myotomy* may help facilitate swallowing and reduce aspiration in combined palsy.

The period of 6 to 9 months is recommended before any permanent surgical intervention; the exception being the presence of aspiration which requires early intervention.

Bilateral Vocal Cord Paralysis

Etiology

Bilateral vocal cord paralysis occurs in about 6% of cases. The common cause for bilateral vocal cord paralysis is thyroidectomy. Malignancy of the laryngopharynx, esophagus, and thyroid, thyroiditis, viral infection, trauma to the larynx (whiplash, post intubation), neurological conditions, and idiopathic causes are other etiological factors. The neurological causes include stroke, encephalitis, syringobulbia, progressive bulbar palsy, multiple sclerosis, and Arnold-Chiari malformation in children.

Clinical Presentation

Bilateral RLN (abductor) palsy has a dramatic presentation with mild to severe stridor or breathing difficulty on exertion. The presentation can be early or can present in the following days or weeks after the incident. The glottic airway varies from 1 to 3 mm. The voice will be good. There may be a history of a surgery like thyroidectomy. Rheumatoid arthritis and prior intubation history should be sought (**Fig. 53.9**).

Bilateral SLN palsy is not common and is usually part of a combined palsy. There will be some aspiration. A high pitch of voice cannot be achieved. On examination, the epiglottis will overhang the laryngeal inlet obscuring the view of the anterior glottis.

Bilateral combined palsy presents with severe breathiness to aphonia. There will be severe aspiration due to an incompetent larynx. On examination, both the cords will be in the intermediate position or cadaveric position.

Pearl

A voice which is normal in the morning and tires as the day goes on occurs in unilateral abductor paralysis. Stridor occurs in bilateral abductor paralysis whereas aspiration implies bilateral adductor paralysis.

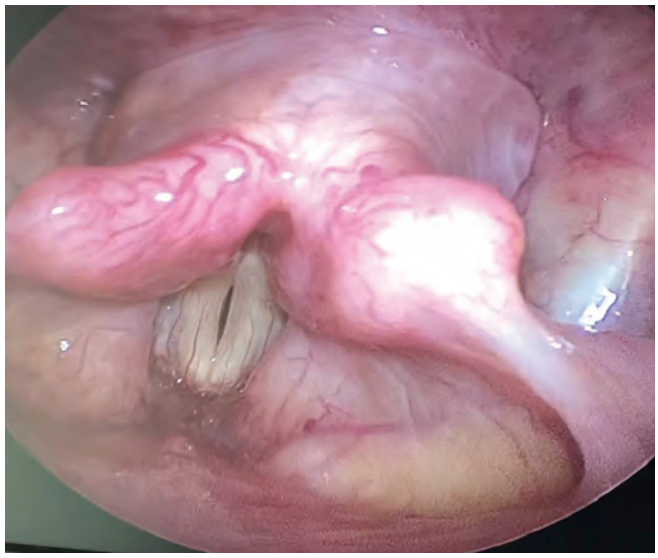

Fig. 53.9 Bilateral vocal cord palsy. Note the reduced airway between the vocal cords. This patient will be in stridor.

Clinical Examination

In addition to examination of the larynx and neck, a neurological examination (including examination of the cranial nerves) should be done.

Investigations

- When there is a history of surgical trauma, other investigations to evaluate for the cause is not necessary.
- MRI of the brain would be useful in children to rule out hydrocephalus and Arnold-Chiari malformation.
- Laryngoscopy (flexible) is done to assess the mobility of the vocal cords. A rigid, direct laryngoscopy done under anesthesia without an endotracheal tube can help in assessing cricoarytenoid joint fixation and interarytenoid scarring.

Treatment

For Bilateral Abductor Palsy

The aim is to achieve a safe and stable airway, preserve the voice quality, and allow for safe swallowing without aspiration.

Immediate Airway Management

Secure the airway by endotracheal intubation. If bilateral vocal cord paralysis still persists a tracheostomy is done.

Reversible/Temporary Procedures

- *Tracheostomy* is the commonest emergency procedure that is done to secure the airway. The patient should be assessed over a period of 6 to 12 months for spontaneous recovery.
- *Suture lateralization* involves using a nonabsorbable suture from the skin of the neck to around vocal process and then back out from the neck where it is secured over a button externally with adequate pressure to pull the posterior part of the ipsilateral cord laterally.
- *Botox injection* into the thyroarytenoid–lateral cricoarytenoid muscle complex may help in improving the airway for 2 to 4 months.

Permanent Procedures

- *Posterior transverse cordotomy or cordectomy*, using laser or coblation, is done in front of the vocal process to create posterior glottis space which improves airway but does not significantly affect the voice (**Figs. 53.10** and **53.11**).
- *Arytenoidectomy* is done either alone or with other procedures like cordotomy. There is a risk of aspiration after the procedure.
- *Arytenoidopexy* uses suture to displace the arytenoid and cord laterally.
- *Laryngeal pacing* involves placing an implant under the skin which gives electrical stimulation to the posterior cricoarytenoid muscle during inspiration. The inspiration is detected by an electrode placed on the diaphragm.

For Bilateral Combined Palsy

The aim of the treatment is to protect the lower airway.

The various positions of the vocal cord and the symptoms experienced by the patient in different types of palsies are summarized in **Tables 53.5** and **53.6.**

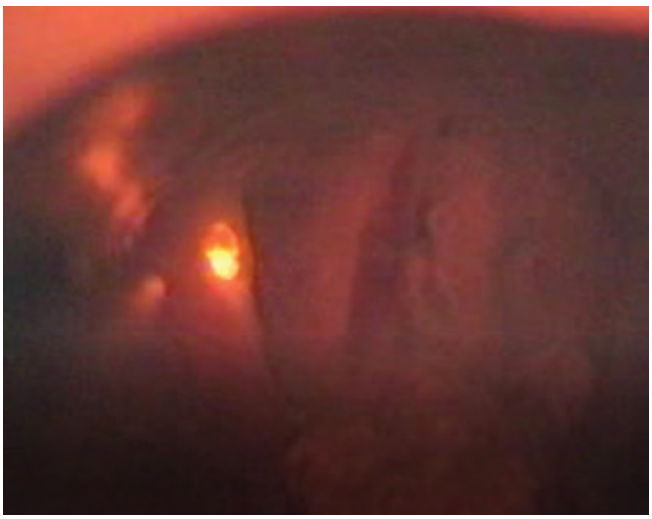

Fig. 53.10 The white spot on the posterior part of the right vocal cord indicates the position of the beam of the carbon dioxide laser during laser cordotomy. This cuts and vaporizes the vocal cord in order to create space for breathing in bilateral vocal cord paralysis.

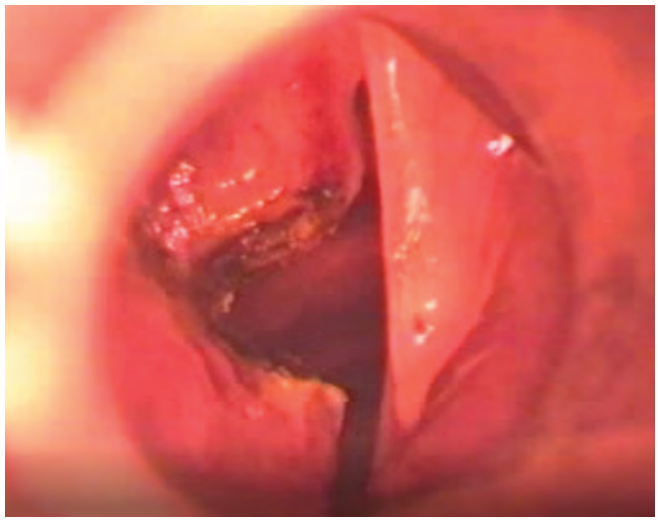

Fig. 53.11 Laser cordotomy performed. Note the space obtained after the posterior part of the right vocal cord has been vaporized.

Table 53.5 Characteristic feature of various types of palsies

Characteristic feature	Unilateral RLN palsy	Bilateral RLN palsy	Unilateral SLN palsy	Bilateral SLN palsy
Position of cords	Paramedian	Midline	Apparent shortening with asymmetric length	Apparent shortening
Voice	Breathy/normal	Normal/strained due to stridor	Diplophonia; cannot achieve high pitch	Loss of vocal range
Airway	Adequate	Compromised	Adequate	Adequate
Aspiration	Absent	Absent	Possible	High risk
Treatment	Medialization	Lateralization		

Abbreviations: RLN, recurrent laryngeal nerve; SLN, superior laryngeal nerve.

Table 53.6 Characteristics of combined nerve palsies

Characteristic feature	Unilateral combined palsy	Bilateral combined palsy
Position of cords	Intermediate on affected side	Intermediate or cadaveric position on both sides
Voice	Severe breathiness	Aphonia
Airway	Persistently open at glottic level; no compensation from opposite cord	Persistently open at glottic level; incompetent larynx
Aspiration	Present	Severe
Treatment	Medialization of cord +/− cricopharyngeal myotomy; voice and swallowing therapy	Protection of lower airway

Pearl

Involvement of the external branch of SLN will lead to inability to change the pitch of the voice, especially high pitch, and affects singers.

Specific Neurological Conditions Affecting the Vocal Cords

Wallenberg Syndrome or Lateral Medullary Syndrome

It is the infarct of the lateral medulla due to occlusion of the posterior inferior cerebellar artery. In addition to dysphagia, dysarthria, impaired pain, and temperature sensation in the ipsilateral face and contralateral trunk and extremities, there will be unilateral vocal cord paralysis.

Myasthenia Gravis

Due to reduced availability of acetylcholine at the myoneural junction, there will be weakness during sustained muscular effort. There will be a breathy voice with reduced loudness. The symptoms worsen with sustained speaking. There may be associated hypernasality with nasal regurgitation, dysphagia, and articulation defects.

The various multiple cranial nerve syndromes involving the vagus nerve are outlined in **Table 53.7.**

Pearl

Functional aphonia is the inability to phonate which usually occurs after a specific event. On flexible endoscopy, the cords do not adduct during phonation but do so when the patient is asked to cough. Previously called hysterical aphonia.

Laryngeal Framework Surgery or Thyroplasty

Laryngeal framework surgery can change the position, shape, and tension of the vocal cord. From the functional view point this type of surgery may be classified into four categories:

- *Thyroplasty type I* for medialization of the vocal cords (**Fig. 53.12**).
- *Thyroplasty type II* for lateralization of the vocal cords.
- *Thyroplasty type III* for relaxation of the vocal cords.
- *Thyroplasty type IV* for lengthening or tensing of the vocal cords.

Table 53.7 Vagus nerve involvement in syndromes

Syndrome	Site of disease	Nerves involved
Collet-Sicard	Posterior cranial fossa	IX, X, XI, XII
Hughlings-Jackson	Posterior cranial fossa	X, XI, XII
Vernet	Jugular foramen	IX, X, XI
Tapia	Parapharyngeal space	X, XII
Villaret	Parapharyngeal space	IX, X, XI, sympathetic chain
Klinkert	Superior mediastinum	X (RLN), phrenic
Pancoast	Apex of lung	X, sympathetic chain, phrenic
Garcin	Skull base	Usually, 7 or more cranial nerves; can involve all 12 cranial nerves

Abbreviation: RLN, recurrent laryngeal nerve.

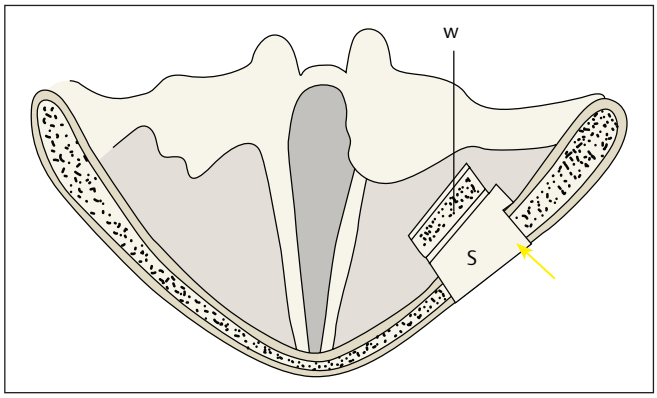

Fig. 53.12 Diagram depicting type 1 thyroplasty. A block of silastic, wedge shaped (*yellow arrow*), is inserted into the left thyroid cartilage pushing the left vocal cord medially in left recurrent laryngeal nerve palsy.

Points to Ponder

- Unilateral SLN palsy is not common.
- In bilateral RLN (abductor) palsy, the vocal cords are in median position. Voice may be good but the glottic airway is compromised, and there will be stridor, especially on exertion, which requires early intervention.
- In bilateral SLN palsy, there is loss of supraglottic sensation leading to aspiration.
- In unilateral combined palsy, the voice is breathy and there is aspiration. Early surgical intervention is done to protect the lower airway.
- In bilateral combined palsy, the vocal cords are in intermediate position leading to complete aphonia and severe aspiration.

Case-Based Questions

1. A 42-year-old woman had undergone a hemithyroidectomy. Postoperatively, there was a change in voice. There was no aspiration.

a. What would be the position of the cord if the recurrent laryngeal nerve was involved?

b. What would be the position of the cord if the recurrent and superior laryngeal nerves were involved?

c. What would be the treatment of choice if the change in voice was mild?

d. What would be the treatment of choice if there was a breathy voice with aspiration?

Answers

a. In paramedian position.

b. In intermediate position or cadaveric position.

c. Wait and watch with regular follow-up. Compensation by the opposite cord will usually occur. If paralysis is persistent, a medialization thyroplasty can be performed after 6 to 9 months.

d. Medialization thyroplasty with or without cricopharyngeal myotomy needs to be done early to protect the lower airway.

2. A 4-month-old infant was brought to the emergency ward with noisy breathing. A flexible endoscopy done in the operating room showed bilateral abductor palsy.

a. What further investigation will be required?

b. What is the position of the vocal cord?

c. In a 36-year-old woman after total thyroidectomy, with a similar presentation, what would be the treatment options?

Answers

a. A magnetic resonance imaging (MRI) of the brain is required to rule out Arnold-Chiari malformation or hydrocephalus.

b. The cords will be in the median position. Mild edema will precipitate stridor.

c. Wait and watch. If there is stridor, tracheostomy is done. Later, in order to improve the posterior airway, a laser posterior cordotomy, arytenoidectomy, or suture lateralization can be performed.

Frequently Asked Questions

1. What are the causes of unilateral recurrent laryngeal nerve palsy?
2. How does bilateral abductor palsy present?
3. What is the emergency management of bilateral abductor palsy?
4. What is the aim of treatment of unilateral combined palsy?
5. What are the various positions of the vocal cord in lesions of the recurrent laryngeal nerve and superior laryngeal nerve?

Endnote

Vagus means "wandering" in Latin. The vagus nerve is the wandering nerve. It wanders from the head, into the neck and thorax, and enters the abdomen.

Sushruta in Sushruta Samhita (6th century BC) documented what is considered the first reference to the "control of voice in relation to the wind-pipe." Galen (2nd century AD) described a nerve from the brain that courses down toward the heart and winds around in a reversed course toward the larynx. He named it "reversivi."

Frank Lahey (1938) popularized the dissection of the recurrent laryngeal nerve in surgery of the thyroid gland.