Textbook of LARYNGOLOGY

Official Publication of the Association of Phonosurgeons of India

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Nupur Kapoor Nerurkar

Co-Editor
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Foreword
Peak Woo
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Foreword

Peak Woo MD FACS

The Health Sciences Publisher
New Delhi | London | Panama
Dedicated to

My husband Rajeev and my daughters Kanika and Anaaya—the true joy!
My Laryngology colleagues, who have been truly generous in sharing their wisdom.

Nupur Kapoor Nerurkar

Dedicated to

My parents for their inspiration and guidance to pursue my dream
And my wife and my sisters for their encouragement throughout.

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Foreword

Textbook of Laryngology, edited by Dr Nupur Kapoor Nerurkar and Dr Amitabha Roychoudhury, brings forward a textbook that is quite unique in the need for a relevant, state-of-the-art and expert-driven text on laryngology for the laryngologists and general otolaryngologists.

Dramatic advances in laryngology over the last 40 years have made larger textbooks that cover general otolaryngology, largely obsolete for the study of laryngeal disorders. Earlier, the state of the art in diagnosis and management of voice, swallow and airway disorders included subjects and approaches that would be unthinkable and were not addressed in the standard otolaryngology textbooks written for the general otolaryngologists. Today, laryngology is a recognized subspecialty. In the literature, we have seen a steady rise of laryngology papers submitted for review. According to NIH PubMed, in 1976, there were 15 articles on the search topic of vocal fold paralysis, and in 2015, there were 115 citations with the same search word. Such is one example of the proliferation of papers related to laryngeal disorders. From diagnostics to therapeutics, dramatic advances continue to pace the changes that are evolving dramatically in the specialty of laryngology. This is because quality-of-life issues related to voice, swallow, and airway continue to drive the need for expertise care in one of the most treasured of our senses, contributing to high-quality life, i.e. the need for excellence in voice, airway and swallow.

The topics and organization are comprehensive and detailed. From basic science to the future, the topics outlined are of utmost urgency in international forums in laryngology that I have had the privilege to participate in. Topics such as office diagnosis, new lasers, office procedures, new phonosurgery approaches, laryngeal reinnervation and robotic surgery are some examples of the state of the art in laryngology. This carefully edited textbook addresses each topic in detail.

A particular strength in the text is the broad focus on voice, airway and swallow issues related to laryngeal function. From treatment of voice disorders for gender reassignment to surgery for laryngeal-tracheal stenosis, the editors, Dr Nupur Kapoor Nerurkar and Dr Amitabha Roychoudhury have brought together an international group of experts in the specialty of laryngology to address each topic. Many of the experts are the leaders in the inception and the development of the treatment approaches in their chapters. Especially enticing are the chapters written by the experts from Europe, USA and India, making this a truly expert collection of chapters that contributes to the English literature in our specialty.

The editors and the contributing authors have in this collection a compendium of vital information that will be a reference tool for both the practicing laryngologists and those practitioners of otolaryngology who seek to have an up-to-date text on laryngology. For a novice, who is contemplating the subspecialty of laryngology, this textbook will serve as a ‘Bible’ for years to come.

Peak Woo MD FACS
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Preface

“The human voice is the most beautiful instrument of all, but it is the most difficult to play.”

Richard Strauss (German Romantic Composer)

Over the past century, Laryngology has mushroomed to become a major specialty in medicine. Beginning with Manuel Garcia’s discovery of mirror examination of his own larynx, diagnostics have today come a long way indeed.

The human larynx and mechanism of voice production, both in health and disease, have posed great challenges to the clinicians over centuries. The 21st century has witnessed an exponential rise in voice disorders, probably an echo of today’s ever-increasing vocal demands in every walk of life. Despite extensive research by anatomists, physiologists, otolaryngologists and speech pathologists, and continued technological advancements across the globe, many conditions in laryngology remain enigmatic. The specialty of laryngology has further expanded with the incorporation of swallowing and airway disorders into its domain.

Indian laryngology has also grown by leaps and bounds over the past two decades, in tandem with the global scenario. This textbook of laryngology, published under the aegis of ‘The Association of Phonosurgeons of India,’ is intended to be a comprehensive study material for any clinician pursuing practice and research in laryngology. It is also intended to serve as a ready-reckoner for a voice pathologist.

We sincerely thank Dr Peak Woo for writing the Foreword of our textbook. We would like to place on record our gratitude to Jaypee Brothers Medical Publishers (P) Ltd. and all our authors, who have worked tirelessly so that this textbook could be born in nine months from the conception of the idea!

Nupur Kapoor Nerurkar
Amitabha Roychoudhury
We would like to acknowledge:

The founders of the Association of Phonosurgeons of India for their vision;
Jaypee Publishers, who first pursued and then supported us wholeheartedly;
Our teachers, who sowed the seeds of knowledge within us; and
Our distinguished contributors, without whom this book would have remained a dream.

Nupur Kapoor Nerurkar
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INTRODUCTION

Bilateral vocal fold paralysis (BVFP) refers to the neurologic causes of bilateral vocal fold immobility (BVFI) and specifically refers to the reduced or absent function of the vagus nerve or its distal branch—the recurrent laryngeal nerve (RLN). BVFI is a broad term that refers to all forms of reduced or absent movement of the vocal folds. Vocal fold immobility may also result from mechanical derangement of the laryngeal structures, such as the cricoarytenoid (CA) joint or posterior glottis stenosis (PGS).

RELEVANT ANATOMY (FIG. 1)

The motor efferent fibers of the vagus nerve originate in nucleus ambiguus which houses the cell bodies of the branchial or special motor efferent fibers, and dorsal nucleus which contains visceral efferent fibers going to the thorax and abdomen, both of which are located in the medulla oblongata. The vagus nerve exits the cranium through the jugular foramen. At the jugular or superior ganglion, sensory fibers to the posterior fossa, external auditory canal, and posterior auricle arise from the vagus nerve.
nerve. When the vagus nerve exits the jugular foramen, it is joined by the cranial portion of the accessory nerve. As the nerve passes through the nodose or inferior ganglion, the superior branches join branches of the glossopharyngeal, hypoglossal, and sympathetic fibers to form the pharyngeal plexus serving pharynx musculature (except for the stylopharyngeus) and palate musculature (except for the tensor veli palatini). As the vagus nerve continues through the cervical region, first between the internal carotid artery and internal jugular vein and then between the common carotid artery and internal jugular vein, it gives off the superior laryngeal nerve. This nerve branches into an internal and an external division. The internal division enters the thyrohyoid membrane and provides sensory innervation to the pharyngeal and laryngeal mucosa above the true vocal fold. The external division travels to innervate the cricothyroid muscle.

The course of the vagus nerve into the thorax differs on the right and left sides. The right vagus nerve travels in front of the subclavian artery into the thorax and abdomen. The right RLN branches off the vagus and travels posteriorly behind the subclavian artery back superiorly into the neck along the tracheoesophageal groove. It enters the larynx at the cricothyroid joint and provides motor innervation to all the laryngeal muscles except the cricothyroid muscle as well as sensory innervation to the laryngeal mucosa below the vocal fold. The left vagus nerve takes a similar course in front of the arch of the aorta continuing into the thorax and abdomen. The left RLN arises behind the aorta near the remnant of the ductus arteriosus and ascends to the larynx in the tracheoesophageal groove. Its longer course makes it more susceptible to injury.

Vocal cord paralysis (VCP) is caused by injury to the vagus nerve from its nuclei intracranially to its peripheral branches. Central lesions such as cerebral agenesis or nucleus ambiguous agenesis or dysgenesis can cause VCP. Any traction or compression injury along the path of the vagus nerve may result in vocal cord dysfunction. A high injury to the vagus nerve can result in affection of swallowing, speech, voice, and airway.

**ETIOLOGIC FACTORS**

The causes of BVCP are:

**Iatrogenic Trauma**

- Surgery—Bilateral injury may be caused by the following:
  - Thyroid surgery: Rosenthal LH et al. have mentioned that thyroidectomy continues to cause the majority (80%) of iatrogenic BVFI and 30% of all bilateral immobility.²
  - Esophageal surgery
  - Neck dissections
  - Cervical spine surgeries
  - Airway surgeries for tracheal reconstruction or anatomies
  - Brainstem surgery.

- Contralateral injury after an earlier unrecognized ipsilateral injury may be caused by the following:
  - Completion thyroid surgery
  - Contralateral carotid endarterectomy

- Intubation
  - Compression of the RLNs because of anterior displacement of thyroid cartilage relative to the cricoid cartilage.
  - Hyperextension of the neck that stretches the vagus nerve.
  - Excessive cuff pressure that compresses the RLN as it enters the larynx.³ Secondary to viral infections triggered after local trauma, such as herpes zoster.⁴ In these cases the symptoms are seen immediately after extubation.

- Nasogastric tube compression
  - Sofferman nasogastric tube syndrome.⁵

**External Laryngeal Trauma**

Blunt or penetrating trauma to the neck can cause BVCP if both RLNs are injured.

**Viral Infections**

- Herpes simplex virus⁶
- Varicella zoster⁷
- Epstein-Barr virus⁸
- Influenza virus and cytomegalovirus associated with human immunodeficiency virus
  - These viruses have all been cited to cause RLN paralysis.⁹

**Neoplasms or Diseases within the Neck or Upper Mediastinum**

Tracheal, esophageal, or thyroid malignancies; aortic aneurysm, lymphoma, tuberculosis, sarcoid, silicosis, and mediastinal metastases can involve both the RLNs.
Neurologic Causes
- Arnold-Chiari malformation
- Meningomyelocele
- Hydrocephalus
- Amyotrophic lateral sclerosis: It is a degenerative disease that involves both upper and lower motor neurons.
- Myasthenia gravis: It usually manifests first with bulbar involvement and affects both laryngeal and pharyngeal functions.
- Postpolio syndrome
- Charcot-Marie-Tooth disease: Patients present with bilateral cord palsy with deafness, diaphragmatic weakness, and cerebellopontine weakness.
- Leigh disease: Uncommon neurodegenerative disorder that causes BVCP ophthalmoplegia, nystagmus, ataxia, spasticity, and other neurological deficits.
- Myotonic dystrophy type I: Most common inherited muscle disorder and may present with BVCP.
- Sensory or motor neuropathies.

Closed Head Injuries and Cerebrovascular Accidents
For both the vagi to be affected, the injury has to be in the medulla, which is incompatible with life. Hence, BVCP is not the presenting feature in these patients.

Radiation Injury
Radiation can result in fibrosis and loss of vascularity around nerves in the radiation field. Berger and Bataini discovered 35 incidents of cranial nerve paralysis after radiation to the head and neck. Nine (26%) of these were vagal.

Metabolic Causes
- Hypokalemia
- Hypocalcemia
- Diabetes mellitus: Onset is slow and gradual and will be associated with other neuropathies.

Toxins
- Lead, arsenic, and alcohol intoxication have been linked to cases of vocal fold paralysis through toxicity to the central nervous system.
- Vincristine
- Organophosphates.

Idiopathic
Idiopathic etiologies, by definition, have no obvious causes. Improved imaging, fiberoptic endoscopy, and laboratory determination of viral titers have decreased the incidence of so-called “idiopathic paralysis.”

ETIOLOGY IN CHILDREN
In children, causes of BVCP include central neurologic abnormalities, idiopathic causes, and iatrogenic causes.

Central Neurologic Abnormalities
Central neurologic abnormalities account for most cases of childhood bilateral vocal fold paralysis (BVFP).
- Arnold-Chiari deformity with meningomyelocele and hydrocephalus is the most common abnormality (one-third of cases).
- Hydrocephalus
- Myelomeningocele
- Intracerebral hemorrhage.
These conditions stretch the vagus nerve or damage its nuclei. An MRI can detect these CNS pathologies.

Traumatic
Intubation and birth trauma related to breach or vertex delivery or the use of forceps can lead to compression or stretching of both RLNs in the neck.

Idiopathic Causes
Idiopathic causes are the second most common causes of childhood BVCP and they recover spontaneously in roughly 50% of cases. The spontaneous recovery of VCP most commonly occurs within the first 6 months but has been reported to occur up to 11 years after diagnosis. Delayed maturation in the vagal nuclei has been proposed as likely mechanism to explain the late vocal cord function recovery.

Iatrogenic Causes
Cardiovascular surgery (i.e. patent ductus arteriosus ligation and repair of tracheoesophageal fistula) is a common cause. Any mediastinal or cervical procedure, and prolonged intubation may be the causes.
Inflammatory and Infectious Causes

In recent years, with better immunizations and antibiotics, inflammatory and infectious causes of VCP are found rarely.¹

POSITION OF CORDS

The most common (75%) position of vocal cords in BVCP is the paramedian position (glottic chink less than 1.5 mm).²¹ The other positions for the vocal cords are the intermediate (glottic chink between 1.5 mm and 2.5 mm) and the lateral positions (glottic chink more than 2.5 mm).

Synkinesis and Reinnervation

The earlier Wagner Grossman theory stated that in cases of damage to the RLNs only, the cords will lie in the paramedian position as the superior laryngeal nerve, which supplies the cricothyroid muscle, is still intact and this puts the cords in adducted position. According to this theory, a high vagal paralysis causing combined Recurrent and Superior Laryngeal Nerve involvement should result in lateralized position of the cords. But vocal cords are seen to lie in the paramedian position in few of these cases too. The Wagner Grossman theory was disproved by Woodson and Koufman et al.²²,²³ Terms like “paramedian” and “cadaveric” carry no topodiagnostic significance, and they are mere descriptive terminologies. The position of the vocal cord is dependent upon degree of reinnervation and the synkinesis present. Synkinesis causes simultaneous contraction of adductor and abductor fibers producing no net vocal fold motion. The reinnervation that follows nerve injury may be inappropriate, leading to innervation of different sets of muscles. It may also cause nerve regrowth which is appropriate but inadequate (innervating same muscle but to less potential), which may result in decreased force of contraction, loss of motor unit specificity, increased muscle fatigue, and changes in peripheral and central neural organization.²⁴ This gives rise to the variable positions of the vocal cords. This is the reason why the terms like “abductor” or “adductor paralysis” have no physiologic validity.²⁵

CLINICAL PRESENTATION

In 75% cases of BVCP, both the vocal cords are in a paramedian position, leading to a compromised airway. Presentation depends on the severity of obstruction and on the rapidity of development of obstruction. Symptoms may range from dyspnea and mild inspiratory stridor to severe respiratory distress. This distress may develop rapidly over days or weeks, or slowly over months or years, depending on the etiology. Many patients are treated for asthma over years due to their “wheezing”, which, in fact, was stridor. Voice is usually normal.

Presentation in Children

- High-pitched stridor: This is the most common presenting symptom in children with a normal cry.
- Suprasternal and intercostal and subcostal retractions may be present with exacerbations during increased airway demands.
- Cyanotic attacks
- Spells of apnea
- Failure to thrive.

When cords are in the lateral position, the patients present with a breathy voice with history of aspiration (Fig. 2).

DIAGNOSIS

Endoscopic Evaluation for Confirmation of Bilateral Vocal Cord Paralysis

70° Rigid Hopkins Videolaryngoscopy

Cohen SM et al. advocated videolaryngoscopy as an objective method to diagnose bilateral vocal cord palsy and posterior glottic stenosis based on a weighted score.²⁶

Awake Transnasal Fiberoptic Flexible Laryngoscopy

Flexible fiberoptic endoscopy is the gold standard for the diagnosis of pediatric VCP. It can be performed in clinic or at the bedside and small diameter pediatric scopes can be passed even in infants. If true abducting movements are present during inspiration, then it is a normal functioning larynx. Sometimes Bernoulli effect and the absence of complete muscular degeneration results in a paradoxical movement of glottis closure during inspiration and this may give a false impression of adducting movements.²⁷

Examination under General Anesthesia

- When fiberoptic laryngoscopy fails
- To differentiate BVCP and PGS
- To look for associated lesions of upper airways.
This can be done by any one or as a combination of these methods:

- **Fiberoptic laryngoscopy in sedation** is especially useful to look for any supraglottic collapse during active respiration.

- **Laryngobronchoscopy with a 0° nasal endoscope**: A Macintosh laryngoscope is introduced to expose the larynx and a rigid 0° endoscope attached to a camera system is passed though the oral cavity. First the vocal cord movements are assessed prior to giving muscle relaxant to the patient. After the patient is paralyzed, the vocal cords are palpated to assess any underlying lesion. The arytenoids are palpated to look for CA joint fixation or arytenoid dislocation. The subglottis and trachea are also evaluated to look for any pathology.

- **Direct laryngoscopy and suspension microlaryngoscopy**: A false cord retractor is placed at the ventricular bands and opened. In PGS, the glottis space remains narrow, and a stretched band of scar tissue is seen in the posterior commissure (Fig. 3). In BVCP, the airway is restored to normal. The CA joints are palpated by placing an instrument adjacent to the vocal process and pushing it laterally. If the entire posterior commissure moves with lateralization of the vocal process, then this suggests that posterior glottic stenosis may be present.

### General Examination

- Examination of the neck for any masses.
- **Assessment of palatal movements**: Palatal paralysis with VCP may indicate a high vagal lesion.

### Radiologic Evaluation

In cases where a direct relation exists between surgical iatrogenic trauma and vocal cord palsy, no radiologic work-up is necessary. In cases where no cause can be found for the palsy, imaging studies are essential. A CT scan (with contrast) from the base skull to upper chest to study the entire course of the vagus down to the take off point of the RLN is required. Koufman et al. advocates the addition of MRI of the brainstem in cases of high vagal lesions with combined palatal and VCP MRI brain if central neurological disorder is suspected.

### Pulmonary Function Tests

They are helpful in documenting the severity of obstruction and for assessing the post-treatment improvement.

### Laryngeal Electromyography

Laryngeal electromyography (LEMG) measures the integrity of laryngeal innervation with percutaneous needle electrodes. The occurrence of fibrillations and fasciculations in LEMG is more useful as a predictor of poor outcome, suggesting an absence of reinnervation. It is also useful in distinguishing neurogenic from mechanical vocal fold immobility and can offer variable prognostic information when used less than 6 months from the onset of paralysis.
**Neurological Tests**

In certain patients, a more thorough neurological examination is required to assess central disorders or neuromuscular disorders that may result in bilateral vocal cord palsy.

**Laboratory Studies**

Blood investigations may be required only if the history and physical examination is suggestive of a systemic process as the cause of BVCP.30

**TREATMENT**

**No Intervention with Regular Follow-up**

From 4% to 14% of the patients tolerate this condition and do not require any surgical treatment, though some of them may decompensate after few years and surgery becomes inevitable.31 Patients with neurologic conditions (e.g. amyotrophic lateral sclerosis, Parkinsonism, stroke) rarely require surgical intervention because treatment of the underlying condition often improves airway compromise.

**Medical Treatment**

Systemic steroids can reduce edema which will improve the airway for a short term but will not be a definitive solution.

**Intubation**

If urgent stabilization of airway is required, infant should be first intubated, and an MRI should be done. If Arnold-Chiari malformation with hydrocephalous is detected then shunt procedure can decrease the intracranial pressure at the origin of the stretched vagi. Subsequent recovery of vocal cord movements alleviates the need for a tracheotomy.

**Surgery**

The ultimate objective of any procedure for BVCP must be an adequate airway, functional swallow, and functional voice quality.

**Tracheotomy**

For centuries, tracheotomy has been the gold standard for securing the airway. Tracheotomy may serve a dual purpose of creating an airway in patients with stridor, and preventing aspiration in patients with cords in the lateral position. It may be required acutely and may be temporary until a definitive procedure is done for the patient. It provides both airway and voice; however, from a patient perspective, tracheotomy is not commonly preferred because of multiple quality-of-life issues.

**Static Procedures that Enlarge the Laryngeal Airway**

Multiple options are available to the surgeon. But, unfortunately, any surgery that widens the glottic chink further deteriorates the quality of voice.

**Resection of anatomical structures:**

- Endoscopic transverse cordectomy
- Medial arytenoidectomy
- Total arytenoidectomy (external, or endoscopic laser)
  These procedures are now performed endoscopically with the use of CO2 laser. The advantages of using the CO2 laser are the increased precision through the narrow endoscope and improved hemostasis.32

**Endoscopic transverse cordotomy:** Laser cordotomy as devised by Dennis and Kashima is the most popular and effective procedure for the management of bilateral vocal cord palsy.33

- Cordotomy has the advantages of a shorter operative time, better vocal results, and reduced risk of postoperative aspiration.34
- It is quite easy to perform, and the technique is quickly acquired.
- This procedure can also be used as a first-line procedure for definitive management of bilateral abductor vocal cord palsy as an alternative to tracheotomy as supported by studies conducted by Milovanovic et al. and Bernstein and others.35
- It can be proposed even if the patient has a chance of spontaneous recovery.
- Vocal fold tissue is not significantly excised. It only frees the vocal ligament and the vocal muscle from the vocal process of the arytenoids. Tissue retraction enlarges the airway.
- It can be done in children too. In 1999, Friedman described the application of the cordotomy in children from 14 months to 13 years old.36
- When the first procedure is not sufficient, a second one is possible (contralateral cordotomy or on the same side), with a good result after this second procedure.
Unilateral laser cordotomy is the procedure of choice of the author for the treatment of BVCP with cords in adduction.

**Technique**
- The larynx is suspended and a CO\(_2\) laser system is attached to the microscope with a 400-mm lens.
- If the patient is tracheotomized, the patient is ventilated with a laser-resistant tracheotomy tube. If there is no prior tracheotomy, this procedure can be done with intermittent apnea technique or with a small laser-resistant endotracheal (ET) tube.
- Saline-soaked cottonoids are placed in the subglottis to protect the ET tube and cuff.
- With CO\(_2\) laser scanning system in the super pulse or ultra pulse mode, a 2–3 mm long incision line is set to operate in a continuous mode giving a power of 10–12 W.
- Attachment of the vocal fold to the tip of the vocal process is severed by vaporizing the tissue. Once the entire vocal fold is separated from the vocal process, the cordotomy is extended into the false vocal fold tissue (Figs. 4 to 6).
- The lateral extent of the cordotomy site should be flush with the lateral subglottic wall. The residual vocal fold will retract anteriorly and appear shortened.
- Frequently, a branch of the superior laryngeal artery causes troublesome bleeding. Suction and bipolar laryngeal cautery are effective in stopping the bleeding.

**Medial arytenoidectomy:**
*Endoscopic laser medial arytenoidectomy (ELMA)*: It was first described by Crumley in 1993.\(^1\) The medial part of the arytenoid body is resected with the preservation of its lateral, posterior, and inferior aspects and the vocal process. The area of resection should not extend to the posterior arytenoid tissue and should spare adjacent mucosa in the intra-arytenoid area (Fig. 7). This method allows the posterior third of the glottis to be widened while the membranous (phonatory) glottis is preserved.

**Total arytenoidectomy:** It was initially performed by the laryngofissure, transthyroid, or posterolateral approaches. Endoscopic surgical techniques were introduced at the

**Fig. 4:** Diagrammatic representation of transverse cordotomy. (TVC: True vocal cord; FVC: False vocal cord; Ary: Arytenoid).

**Figs. 5A and B:** Endoscopic transverse laser cordotomy. (A) Initial laser incision on the left vocal cord marked by red arrow; and (B) Vocal cord at the end of surgery with a wide posterior glottis.
beginning of the second half of the twentieth century and the most widely accepted one was the Thornell’s technique. It was carried out under a temporary tracheotomy.38

*Endoscopic laser arytenoidectomy:* The CO2 laser total arytenoidectomy first described by Ossoff et al. in 1983 consisted of complete ablation of the arytenoid cartilage, including the muscular process.39 The operative defect thus obtained should be flush with the walls of the cricoid ring, both posteriorly and laterally. Tissue removal posteriorly should not remove any interarytenoid mucosa.

**Contraindications for resective procedures:** Recovery of laryngeal functions can occur as long as 12 months after injury. In adults, any definitive procedure to address VCP must not be undertaken while a possibility for recovery exists. Patient should be assessed by direct videolaryngoscopy and bronchoscopy to look for any tracheotomy-related complications at regular intervals. In addition, laryngeal electromyography can be used to evaluate normal action potentials (normal nerve), the absence of potentials (nonfunctioning nerve), defibrillating potentials (worsening nerve), or polyphasic potentials (regenerating nerve).

In children, spontaneous recovery has been reported in the vocal cord function in 56% of the patients who had bilateral VCP.17 Berkowitz had a 66% rate of spontaneous recovery for cases of congenital idiopathic bilateral VCP, with tracheotomy tube decannulation after 5-7 years.40 Hence, it is reasonable to wait until child reaches 2-3 years of age before any surgery is envisaged. Optimum time for surgical intervention should be discussed on case-to-case basis. The least invasive and damaging procedure should be selected to avoid irreversible sequelae that would compromise the late recovery of vocal cord movements.

*Retailoring and displacing the existing structures, with minimal tissue removal/resection:* Endoscopic vocal fold lateralization: Described first by Ejnell, it was based on extralaryngeal suture technique and was applied only in patients with long standing (up to 30 years) BVCP as a measure for definitive airway enlargement.41 Lichtenberger proposed endo-extralaryngeal suture technique (1979) and first implemented it as a single temporary measure for laterofixation.42 It is indicated for early, symptomatic BVCP (first 2 months) with uncertain prognosis for recovery. It is contraindicated in cases of recent trauma to the posterior glottis from indwelling ET tube.

**Procedure (Fig. 8):**

- Suspension laryngoscopy is performed.
- The neck skin on the side of the proposed suture lateralization is prepared and draped.
- The Lichtenberger needle carrier is loaded with a 2.0 or 3.0 Prolene suture. Under microscopic visualization, the needle is positioned below the posterior vocal fold at a point just anterior to the vocal process. The needle is then pushed through the larynx until the tip of the needle appears externally through the skin of the neck. The needle is grasped, and the suture is advanced through the skin and temporarily secured with a clamp.
The other end of the same Prolene suture is then threaded through a free needle, and with the help of the needle carrier, it is pushed through the larynx externally at a level slightly superior to the true vocal fold. The needle is again grasped through the skin of the neck. A similar suture is taken just anterior to the previous one.

Traction is now placed on the two sutures to create lateralization of the posterior vocal fold.

A 2 cm horizontal incision is made in the neck and the sutures are then pulled deep to the skin incision. The two ends of the suture are then tied over a button on the sternohyoid muscle.

A 95% success rate has been reported with this method. Rovo et al. proved the reliability and the reversibility of the temporary endo-extralaryngeal lateralization, when performed early after the onset of BVFP.43

This technique can be combined with submucosal laser resection of thyroarytenoid and/or partial arytenoidectomy. It will then be a permanent lateralization procedure.

This approach has several problems, including the need for definitive treatment, failure due to the suture-cutting through the tissues, inadequate widening due to splintering of the arytenoids cartilage, granulations and formation of scar tissue, and poorer voice quality that can be expected after arytenoidectomy.

Medialization thyroplasty: When both vocal folds are paralyzed in the cadaveric position, the airway may be fine, but voice and swallowing may be impaired and patients have aspiration. In this setting, unilateral or bilateral medialization procedures may be useful.43 It can be done by temporary injectable implants or by external type I thyroplasty with silastic or titanium implants. The thyroplasty can be reversed if there is recovery of function.45

Reinnervations of the laryngeal musculature: Several reinnervation procedures to the posterior cricoarytenoid (PCA) muscle have been described. In the intervention developed by Tucker, ansa hypoglossi with a small part of the omohyoid or sternohyoid muscle is used for implantation into the PCA muscle. Tucker reported a 50% decannulation rate in 9 of 18 tracheotomized children who sustained BVCP.46,47 However, due to technical difficulties and inconsistent results, these techniques are not routinely used in clinical practice. Further research is required before implementing this procedure in daily practice.

Laryngeal pacing: Functional electrical stimulation of the larynx, or laryngeal pacing, continues to be explored as a potential therapeutic option for bilateral paralysis.48 The afferent input is provided by the phrenic nerve, which is stimulated by inspiration. The efferent limb of the system may be connected to the denervated muscles themselves. This activity should then result in stimulation of the PCA muscles to abduct the vocal folds.
Botulinum toxin: Botulinum toxin injections in the adductor muscles (thyroarytenoid and lateral cricoarytenoid) can eliminate synkinesis and permit unopposed action of the PCA to abduct the vocal folds. The results are very inconsistent and temporary.

**CONCLUSION**

Over time, a large number of surgical procedures have been developed. But still there is no single surgical technique that is perfect. This is due to the complex neuromuscular apparatus of the larynx. It simultaneously provides closure for phonation and protection of the lower airways; and opening for breathing. This cannot be substituted by static remodeling of anatomical structures. The only physiological approaches to BVCP can be the reinnervation procedure, but they have proved to be successful in the hands of few surgeons only and are currently not applicable in clinical routine. All in all, bilateral vocal cord palsy is an enigma that needs to be understood, assessed, and tailored to suit the individual needs.

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