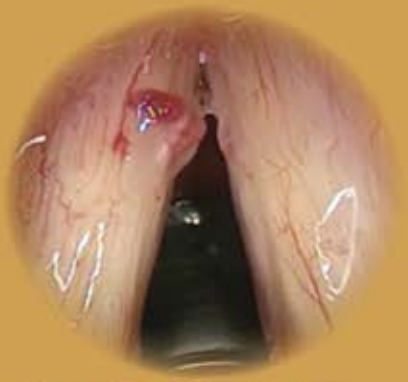

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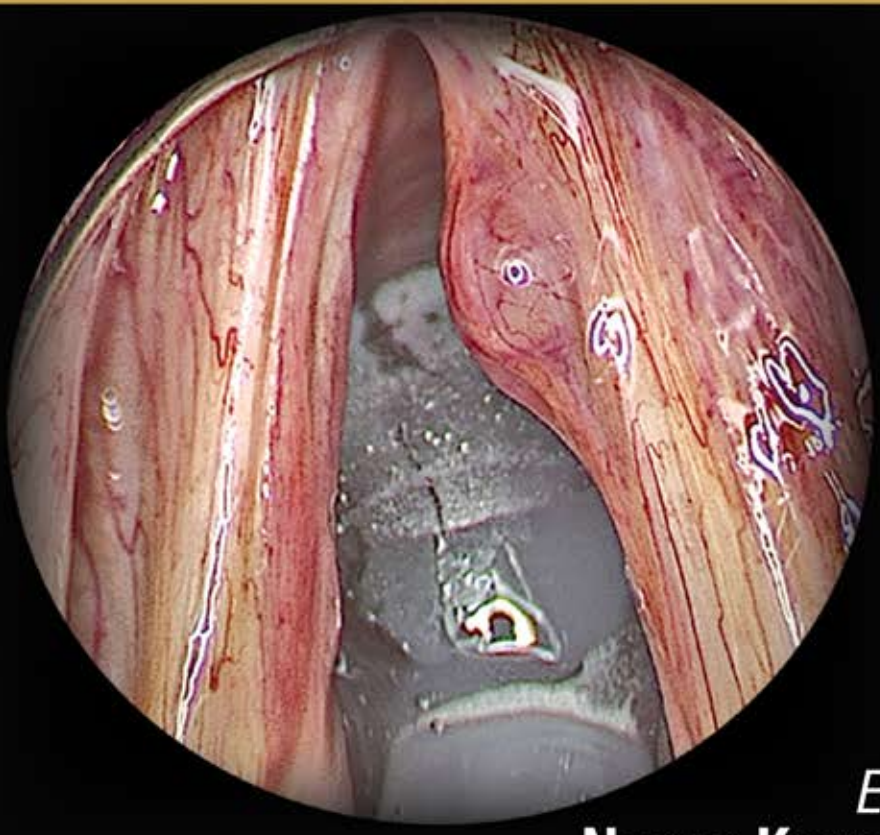

LARYNGOLOGY

Editor-in-Chief
Nupur Kapoor Nerurkar

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Foreword
Peak Woo

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Section 6: Recent Advances

CHAPTER 19

Unilateral Vocal Fold Paralysis and Medialization Laryngoplasty

Hagit Shoffel-Havakuk, Michael M Johns III

INTRODUCTION

The larynx major functions: airway protection, breathing, phonation, cough, and Valsalva all rely on the larynx's dynamic properties. Though the current management of unilateral vocal fold paralysis (VFP) comprises a wide range of effective treatment options, none of those reliably restores vocal fold motion.

This chapter provides a summary of the clinical evaluation and management of unilateral vocal fold immobility, with distinct emphasis on unilateral VFP in adults caused by high vagal or recurrent laryngeal nerve (RLN) injuries. Special considerations including pediatric patients, isolated superior laryngeal nerve (SLN) paralysis, and mechanical fixation are also discussed.

HISTORICAL ASPECTS

Restoration of vocal function by rearrangement of the laryngeal cartilage framework was first introduced at the beginning of the twentieth century. Nevertheless, only after Isshiki et al. published analysis of laryngeal framework surgeries in the 1970s,[1] those procedures became prevalent. Isshiki described four laryngeal framework surgeries that were classified as thyroplasty types I–IV. Type I thyroplasty is a medialization procedure designed to treat glottic insufficiency. In 1978, Isshiki et al. described arytenoid adduction procedure for correction of large posterior glottal gaps.[2] In the following years, several modifications of these procedures were reported, as well as introduction of arytenopexy and cricothyroid subluxation were reported.[3,4] Today, these procedures became the major surgical management for glottal incompetence.

ANATOMY AND PHYSIOLOGY

The innervation to the larynx is long and tortuous, best demonstrated by the left RLN route around the aortic arch. VFP may occur due to general four types of neurologic deficits: (1) central nervous system (CNS), (2) high vagal, (3) isolated RLN, and (4) isolated SLN. Though historically believed that different lesion sites result in a typical vocal fold position, it is currently challenged, as the vocal fold's position depends on additional factors such as degree of paresis, synkinesis, and compensation, along with the patient's laryngeal anatomy.

Since cortical lesions tend to spare the vocal folds, CNS injury causing laryngeal paralysis usually indicates brainstem dysfunction, which generally tends to involve other cranial nerves as well. Injury to the dorsal and ventral nucleus ambiguous would affect only motor innervation without sensory or secretory deficits. As upper motor neuron lesion, the initial flaccid paresis becomes spastic with limited range slow movements. Extracranial high vagal lesions are characterized by motor, sensory, and secretory laryngeal injuries; those lesions precede the branching of the SLN from the vagus trunk. The vocal fold is frequently, but not necessarily in a paramedian position and the vocal process drops anteromedially. Isolated RLN injury resembles high vagal lesions, yet it lacks the sensory, secretory, and cricothyroid muscle dysfunction which is related to the SLN.

ETIOLOGY

While vocal fold immobility might be related to either neurogenic or mechanical disorders, the most probable cause of unilateral vocal fold immobility is neurogenic.

Box 1: Causes of vocal fold paralysis and paresis.

- Trauma
  - Iatrogenic
    - Cervical
      - *Surgery*: Thyroidectomy, other head and neck surgery, anterior approach to the cervical spine, carotid endarterectomy
      - *Procedures*: e.g. endotracheal intubation, central venous catheterization, forceps delivery
    - *Thoracic*: e.g. repair of thoracic aortic aneurysm, open heart surgery
    - Skull base surgery
  - Noniatrogenic
    - Cervical or chest trauma
- Neoplasia
  - Skull base or brainstem
  - *Cervical*: e.g. thyroid, metastatic lymph nodes, vagal schwannoma
  - *Thoracic*: e.g. thymoma, mediastinal lymphadenopathy, esophageal carcinoma
- Medical disease
  - *Neurological*: e.g. cerebrovascular accident, intracranial neoplasm, Arnold-Chiari malformation, neurofibromatosis, myasthenia gravis, multiple sclerosis, amyotrophic lateral sclerosis, Charcot-Marie-Tooth disease
  - *Cardiovascular*: e.g. Ortner's syndrome (cardiovocal syndrome), aortic aneurysm
  - *Drug toxicity*: e.g. vinca alkaloids, organophosphates
  - Infectious
    - *Viral*: e.g. herpes simplex virus, Epstein-Barr virus, cytomegalovirus, varicella zoster virus, postpolio syndrome
    - *Bacterial*: e.g. Lyme disease, syphilis, botulism
  - *Granulomatous*: e.g. tuberculosis, sarcoidosis
- Idiopathic

The causes of VFP can be categorized by the following four major groups: (1) traumatic (either iatrogenic or noniatrogenic), (2) neoplastic (along the course of the vagus and RLN), (3) general medical diseases (either neurologic or nonneurologic), and (4) idiopathic. Possible causes for VFP are listed in Box 1, rates of leading causes can be found in Table 1. Through the past four decades, the leading causes for unilateral VFP remained unchanged. The most common etiologies are surgical trauma and malignancy, followed by idiopathic causes. In the past, thyroid surgery was considered the most common cause of iatrogenic paralysis; however, this has been changed recently, when nonthyroid surgeries such as anterior cervical approach to the spine and carotid endarterectomies became the most common iatrogenic causes.[5] The most prevalent sites of malignancy that can induce VFP are pulmonary (either by a primary tumor or metastasis) and mediastinal tumors. A significant portion of VFP cases are considered idiopathic. It has been suggested that viral neuritis is responsible for some of those cases, particularly in paresis. This assumption is supported by analogy to other cranial nerves palsies, frequent reports of recent viral illness, and serologic association (e.g. herpes simplex virus, Epstein-Barr virus, and cytomegalovirus).

Table 1: Etiology rates of unilateral vocal fold immobility in adults.

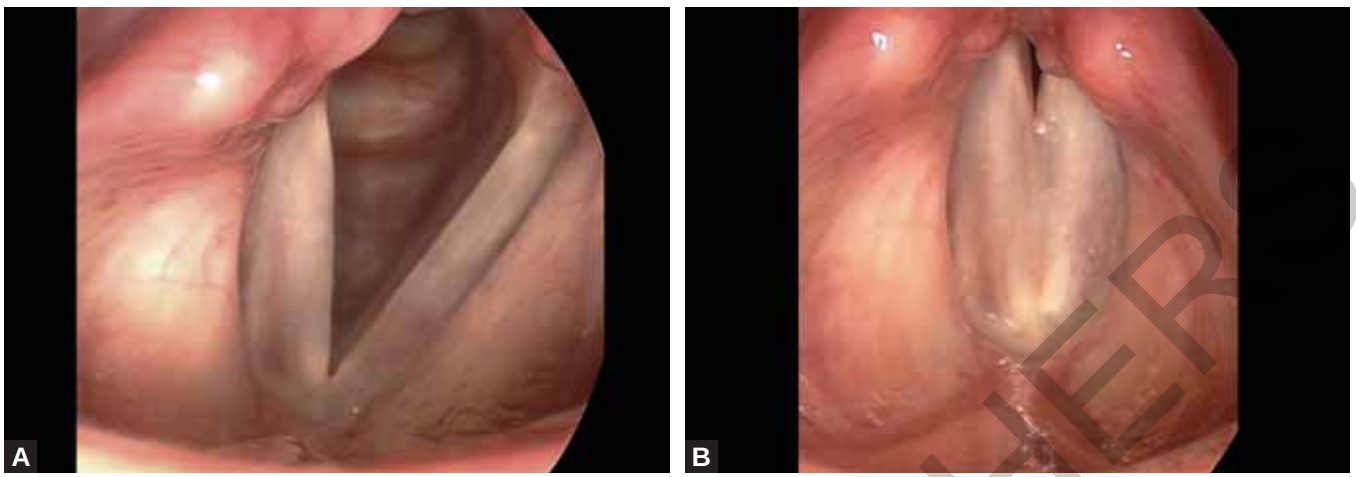
	Maisel (1974)[6]	*Yamada (1984)*[7]	*Terris (1992)*[8]	*Rosenthal (2007)*[9]
Total (N)	127	519	84	363
Surgical trauma	15.7%	22.3%	34.5%	46.3%
▪ Thyroid	7.9%	11.9%	8.3%	15.7%
▪ Nonthyroid	7.9%	10.4%	26.2%	30.6%
Nonsurgical trauma	10.2%	2.3%	1.2%	2.2%
Neoplastic	24.4%	17.7%	40.5%	13.5%
CNS lesions	7.9%	1.2%	2.4%	3.0%
Intubation	3.1%	10.4%	7.1%	4.4%
Idiopathic	21.3%	41.8%	10.7%	17.6%
Others	17.3%	4.2%	3.6%	12.9%

(CNS: Central nervous system).

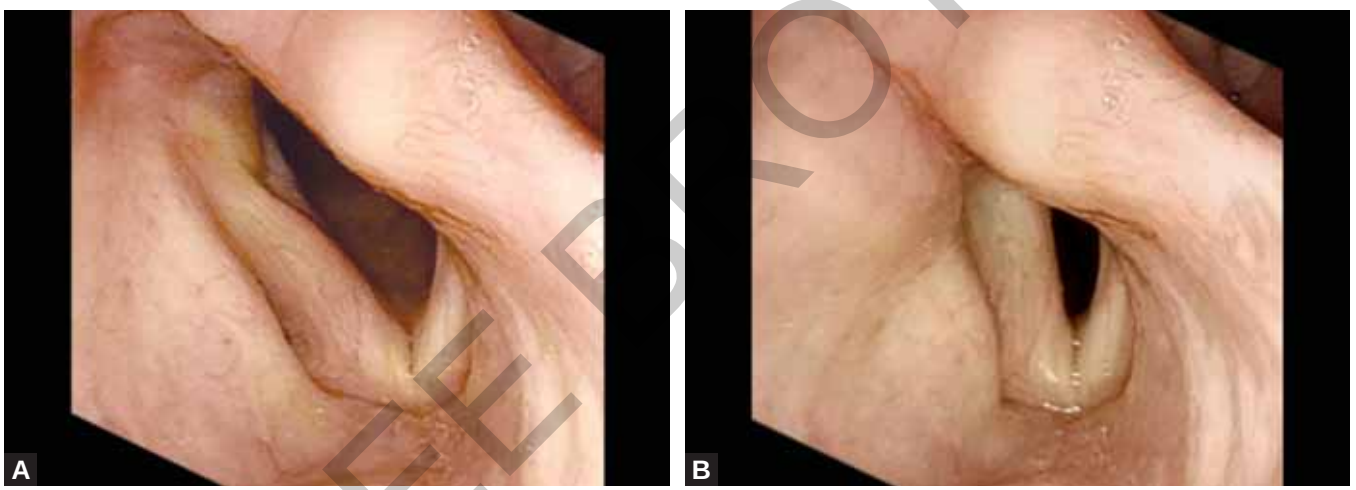
EVALUATION

History

Commonly, patients suffering from unilateral vocal fold immobility may complain of hoarseness with weak and breathy voice, which usually correlates with the degree of glottal gap. Other vocal symptoms may include speech dyspnea, vocal fatigue, increased effort, and difficulty to speak in a noisy environment or over the telephone and general difficulty with voice projection. Additionally, patients may also report dysphagia, particularly for fluids and though aspirations may exacerbate due to weak cough,[10,11] true aspirations are rare in isolated RLN injury, and may suggest "high vagal" injury.[12] Patients may also complain of difficulty to perform a variety of everyday tasks requiring Valsalva maneuver (e.g. lifting heavy weight, defecation). The symptoms and their severity may vary between patients, depending on the degree of paresis/paralysis, site of injury, and patient's compensation. Severity of symptoms varies in different patients and generally depends on the degree of synkinesis and the



Figs. 1A and B: (A) An endoscopic image of open glottis, while breathing, in a patient with right vocal fold paralysis. (B) An endoscopic image of closed glottis, during phonation, in the same patient: demonstrating a relatively favorable paralyzed vocal fold position.



Figs. 2A and B: (A) An endoscopic image of open glottis, while breathing, in a patient with left vocal fold paralysis. (B) An endoscopic image of closed glottis, during phonation, in the same patient: demonstrating an unfavorable paralyzed vocal fold position.

position of the paralyzed vocal fold on both the vertical and horizontal planes.

It is important to ask the patients on associated symptoms, as neurological symptoms may raise suspicion for CNS or neurological disease. It is also important to obtain past surgical and medical history as well as family history, since data may reveal the origin of the paralysis. Positive smoking history along with weight loss may suggest malignancy as a possible cause.

Physical Examination

The voice should be assessed during conversation and speech tasks; it would typically sound breathy, asthenic, and diplophonic. The maximum phonation time can be decreased due to glottal gap. Complete cranial nerves evaluation as well as lungs, head, and neck examination should be performed.[13]

Every patient with suspected vocal fold immobility necessitates examination by either flexible or rigid laryngoscopy (Figs. 1 and 2). Flexible laryngoscopy provides a good view of the palate, pharynx, and larynx; additionally, pharyngeal and laryngeal muscles are in more neutral position than during rigid laryngoscopy, allowing for easier detection of delicate changes in muscle tone and function. The patient should be asked to perform both abduction and adduction; lack of vocal fold motion with phonation or ventilation is required in

order to define vocal fold immobility. In cases of paresis or compensation due to cricothyroid, interarytenoid, and extralaryngeal muscles activity, the decreased motion might be disregarded. This may require repetitive vocal fold motion (/i/-sniff) to reveal the deficit. The patient should also be asked to increase voice pitch, as inability to do so may imply SLN deficit. In order to differentiate neurological disorder from functional disorder, the examiner can ask the patient to cough, sniff, or whistle.

Passive arytenoid movement may be noticed when the mobile arytenoid "bumps" against the paralyzed arytenoid, displacing its muscular process. Some cases may present with vestibular fold hyperadduction that might obscure the view of the paralyzed vocal fold.[14] This finding itself should urge the physician to look for an underlying cause. Pooling secretions in the piriform sinus of the affected side may be observed due to the RLN contribution to the cricopharyngeus muscle. The examiner should look for other possible causes of vocal fold immobility, such as posterior glottis scars or glottic web. A detailed analysis of the glottal configuration and gap (e.g. height mismatch, posterior gap) is required in order to decide on treatment and surgical approach.

In some cases of paresis, the only signs to raise the suspicion might be mild bowing of the affected vocal fold or incomplete glottic closure. In such cases videostroboscopy may yield additional clues, such as prolonged "open phase" of the vibratory cycle, increased vibratory amplitude on the affected side, or asynchronous mucosal wave propagation.

Further Studies

When the cause of the vocal fold immobility is not evident (e.g. immediately following neck surgery), the physician should perform a thorough investigation to find out or rule out etiologies such as malignancy and granulomatous diseases. In such cases, imaging of the vagal nerve route from the brain to the mediastinum is mandatory. Both computerized tomography (CT) scan and magnetic resonance imaging (MRI) of the brain may be appropriate, yet MRI is superior and should be preferred when CNS lesions are suspected. For the mediastinum some may advocate routine chest radiography (CXR), due to its low radiation exposure and as it was found useful in specific cases.[15] However, CXR cannot demonstrate the neck, and CT scan is better in detecting the etiology. There is a general agreement that a contrast CT scan or MRI from the skull base to the upper chest is adequate.

Laryngeal electromyography (LEMG) represents the diagnostic gold standard for VFP and paresis. Nevertheless, due to its cost and invasiveness, LEMG is generally performed later in the evaluation, and if other studies were nondiagnostic. LEMG can distinguish between neuromuscular deficit and mechanical fixation. Useful information from LEMG can be obtained only in the period between 1 month and 6 months after the onset of VFP, as Wallerian degeneration may take as long as 4 weeks depending on the axonal injury location, and since beyond 6 months the use of LEMG is limited due to generally poor prognosis. LEMG is considered the most reliable prognostic indicator for recovery, which is valuable for treatment decision-making. In general, preservation of normal motor unit action potential (MUAP) waveforms, activation during voluntary task, brisk recruitment, and absence of spontaneous activity are indicators of good prognosis. Absence of those findings and the presence of fibrillation potentials indicate less favorable prognosis. Overall, LEMG is a more reliable predictor of poor prognosis.[16] LEMG is further described in Chapter 12.

When there is no suspicion of specific infectious, neurologic, rheumatologic, or granulomatous disease, the yield of blood laboratory studies is very low and should not be performed. Yet, when indicated, studies as Lyme disease titer or acetylcholine receptor antibody levels should be considered.

Direct laryngoscopy for examination and palpation under anesthesia may serve to distinguish between neuromuscular disorder and mechanical disorder, and in cases of mechanical disorder can reveal the underlying cause.

MANAGEMENT

Management of unilateral VFP should address quality of voice and the risk of aspiration. Factors such as prognosis for recovery, degree of impairment, patient's general health, and patient's requirements and desire for recovery influence the timing and type of intervention. Treatment options include observation, voice therapy, or surgical intervention by injection augmentation, laryngeal framework surgery, or reinnervation.

Some unilateral VFP patients may recover spontaneously, and half of the patients with idiopathic paralysis may regain normal or near normal function within the first year, even without vocal fold motion.[17] Therefore, it is common to practice observation and watchful waiting for a period of 6–12 months prior to decision on definitive surgical treatment. Referral to speech pathology for voice

or swallow therapy may relief symptoms. Additionally, in this early period, symptomatic patients should be offered temporary injection augmentation. After this time period, it is reasonable that the vocal fold has reached stability; therefore, if reassessment shows indication, medialization framework surgery with medialization thyroplasty is the preferred intervention. Nevertheless, symptoms such as significant dysphagia, aspiration, and poor cough should prompt earlier intervention.

SURGICAL TREATMENT OPTIONS

Injection Augmentation

Injection augmentation can improve glottic closure by adding bulk to the paralyzed vocal fold and medializing its free edge. Injection augmentation is usually considered a temporary solution, as most available injection materials absorb over time. Injection can be performed by either transcutaneous or peroral approaches, under topical, local, or general anesthesia. The practice of injection augmentation was popularized due to its minimally invasive nature and as it can be performed at the office. The variety of injection materials and techniques are described in detail in Chapter 10.

Laryngeal Framework Surgery

The ideal framework surgery would simulate the normal vocal fold position during phonation, with concern for the vocal fold axial plane, arytenoid position, vocal fold height, medial edge contour, and vocal fold mass and elasticity.

Medialization Laryngoplasty

Medialization laryngoplasty or type I thyroplasty (by the Isshiki classification) is the most commonly performed laryngeal framework surgery and is indicated to improve dysphonia or aspirations due to glottic insufficiency. It is considered the long-term solution for glottic insufficiency caused by unilateral VFP, vocal fold paresis, or atrophy. In medialization laryngoplasty, the vocal fold is medialized by insertion of an implant into the paraglottic space; it can offer a more favorable phonatory position for the paralyzed vocal fold than what can be achieved by injection augmentation. The procedure is contraindicated when there is concern for airway compromise or when there is an evident local malignancy.

Several implant materials and systems can be employed; these materials include silastic, hydroxylapatite, polytetrafluoroethylene ribbon (Gore-Tex®), and titanium. A medium-grade silastic block can be used to create a hand-carved implant, allowing for individualized implant according to the patient's laryngeal anatomy and the cartilaginous window. Those implants are well controlled and easily fitted into the desired position. Silastic is well tolerated with minimal tissue reactivity over time. Preformed silastic implants are also available (e.g. Montgomery Thyroplasty Implant System). Conversely, some surgeons prefer the use of Gore-Tex® ribbon implants, particularly when there is significant vocal fold bowing or paresis. This implant spares the skills of hand carving and the degree of medialization can be adjusted easily. Self-contained systems of implants and instruments are also available; VoCom (nonporous hydroxyapatite ceramic) allows choosing between five implant sizes and four different shims. The implant can be secured in a horizontal or vertical position and at any position along the anterior to posterior and superior to inferior axes of the window. None of the implants has been proven to be superior to others; therefore, implant selection usually relies on the surgeon preference and previous experience.

Unless contraindicated, decadron should be given preoperatively to avoid edema. Generally, the procedure is performed in the operating room while the patient is sedated. Some anesthesiologists prefer intravenous propofol, which can be reversed quickly when patient cooperation is needed. Local anesthesia and epinephrine is infiltrated to the skin and subcutaneous tissue of the anterior neck and along the thyroid cartilage. Topical anesthesia and decongestion are applied to the more patent nasal cavity, allowing the placement of a monitored flexible laryngoscope and offering the surgeon with visual feedback during the operation. The endoscope should be properly positioned and secured (Fig. 3).

Following proper preparation, a modest incision is made to the neck in a skin crease corresponding to the thyroid cartilage or cricothyroid membrane. Subplatysmal flaps are developed and the strap muscles midline raphae is divided in order to reveal the thyroid cartilage. The external perichondrium on the affected side is incised and a flap is elevated. It is important to expose the inferior border of the thyroid cartilage before creating the cartilaginous window. The window should be placed at the level of the vocal fold. A rectangular window is usually located approximately 5–7 mm posterior to the midline of the thyroid cartilage and 2–3 mm superior to the inferior border of the thyroid ala (Fig. 4A). The final location and size of the window

depend on the chosen implant. After the window had been created, the internal perichondrium can be incised or removed and a careful undermining within the paraglottic space will establish the plane for the implant insertion (Fig. 4B). Satisfactory placement of the implant can be determined by both the patient's voice and via the flexible laryngoscopy monitor (Figs. 5 to 7). The next steps are to secure the implant with a nonabsorbable suture and to perform closure of all layers including the outer perichondrium, strap muscles, platysma, and skin. A drain may be placed as necessary.

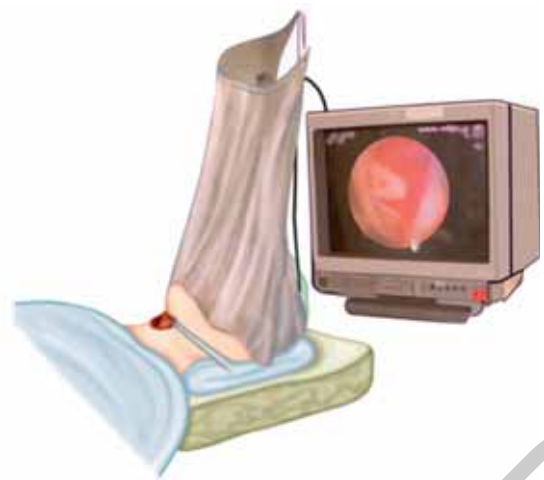
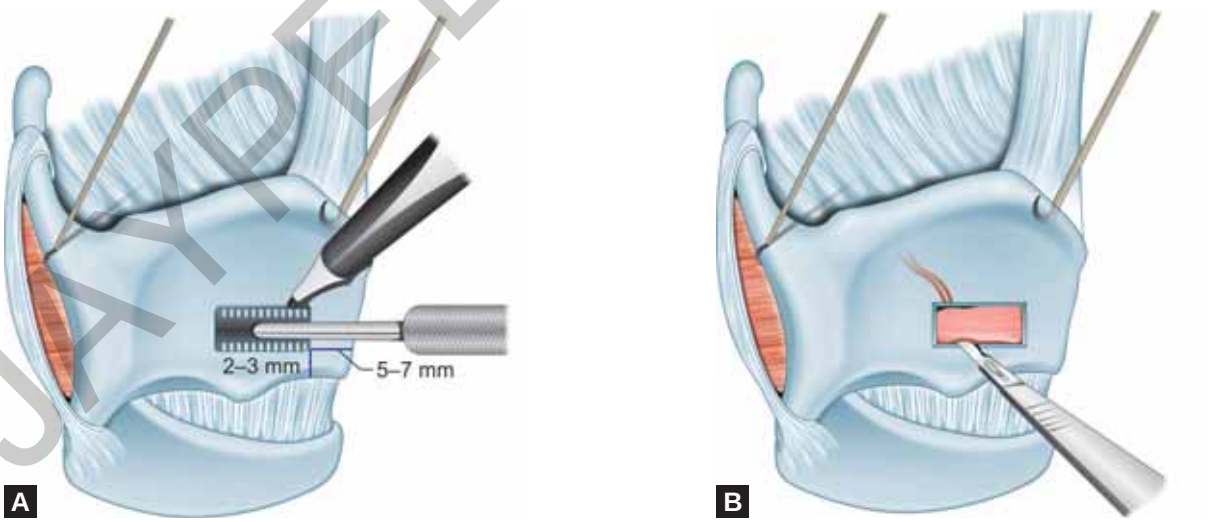


Fig. 3: The operating room setup: A flexible nasolaryngoscope is suspended above the patient and covered with a sterile sheet. A video monitor is placed behind the patient's head, allowing for vocal folds' visualization throughout the procedure.

Postoperatively, the patients should stay hospitalized overnight to avoid the risk of airway compromise. Voice rest is encouraged for 3–7 days; physical exercise should be refrained.

As medialization narrows the airway, when combined with postoperative edema or hematoma, it may result in airway obstruction. Therefore, overnight observation is recommended and some surgeons would administer the patient an additional dose of steroids postoperatively. In different case series, 0.6–11% of the patients undergoing laryngeal framework surgery required airway intervention such as intubation or tracheostomy during the immediate postoperative period.[18,19] The second principle complication is violation of the laryngeal mucosa, which may result in postoperative infection or implant extrusion. The surgeon should exercise extreme caution while dissecting in the paraglottic space, especially when close to the ventricular mucosa and avoid anterior dissection. When perforation has occurred, presence of endolaryngeal blood can be seen on the monitor; however, this may not be apparent in all cases. Therefore, it is useful to ask the patient for Valsalva maneuver, while the surgical field is irrigated and before the insertion of the implant; presence of bubbles indicates mucosal penetration. Extrusion of the implant is a rare, yet significant complication, as the implant predominantly extrude into the airway rather than



Figs. 4A and B: (A) The window outlines: The cartilaginous window should be located as low as possible, leaving 2–3 millimeters of cartilage below the window. The anterior border of the window is placed 5–7 millimeters back from the anterior midline (5 mm for women and 7 mm for men). The final size of the window itself depends on the chosen implant. (B) After the cartilaginous window has been created, the inner perichondrium is incised. This allows for creating a plane within the paraglottic space for implant insertion.

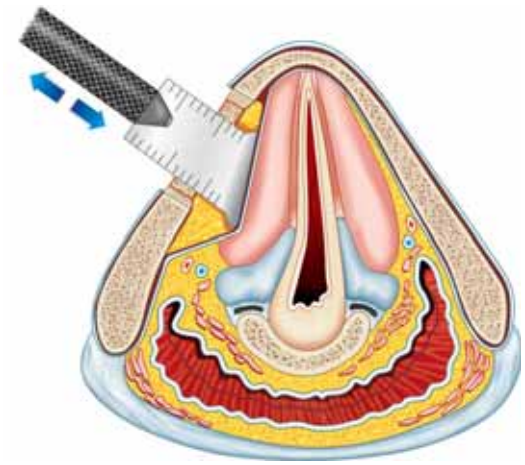
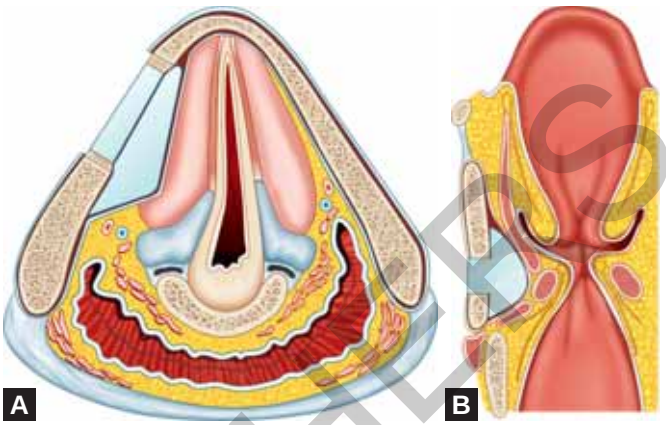


Fig. 5: Measurements for a hand-carved Silastic implant are taken using a depth gauge. The depth gauge is placed within the window to simulate the shape and size of the implant. The patient's voice and the monitor are used for feedback.



Figs. 6A and B: (A) The final position of a hand-curved Silastic implant prosthesis, displacing the thyroarytenoid muscle toward the midline (axial view). (B) The final position of a hand-curved Silastic implant prosthesis (coronal view).

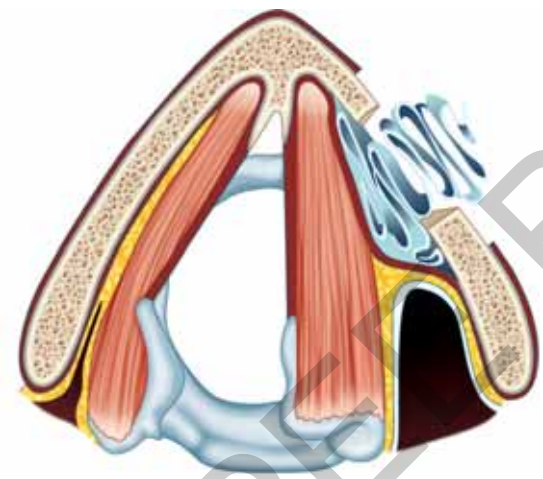


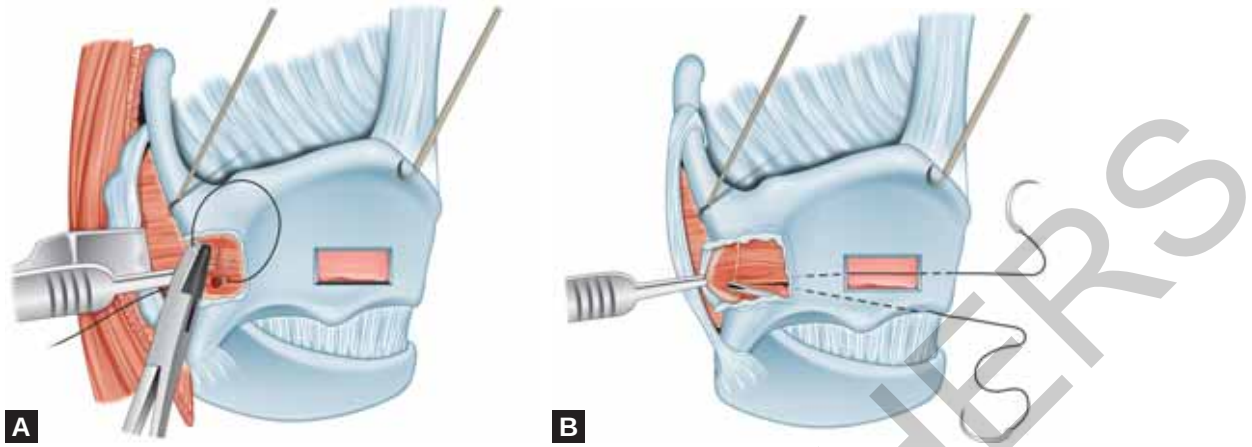
Fig. 7: Gore-Tex® ribbon implant layered through the cartilaginous window, displacing the thyroarytenoid muscle toward the midline (axial view).

transcutaneously. This complication can be prevented by avoiding and identifying mucosal penetrations.

Unsatisfactory or suboptimal voice outcome may lead to revision surgery. Common causes include: posterior glottic gap, undermedialization, and malpositioning of the implant.[20-22] The vocal process cannot be medialized effectively by simple medialization laryngoplasty procedures and additionally, it is generally prolapsed and the height mismatch is not corrected. Therefore, in cases of persistent posterior glottic gap, revision medialization alone is not sufficient and should be combined with arytenoid repositioning. Undermedialization is common in prolonged procedures when the tissue becomes edematous and misleads the surgeon to believe the vocal fold was sufficiently medialized. The administration of intravenous decadron preoperatively is also aimed to prevent this surgical error. When vocal fold edema is suspected, the preferred intraoperative voice feedback should be mildly stained, rather than normal. Implant malpositioning may cause unsatisfactory voice outcome; superior malposition is the most common and can result in a rough and diplophonic voice due to overmedialization of the vestibular fold and ventricle. Anterior malposition would result in strained voice due to early anterior contact.

Arytenoid Repositioning

In some patients a medialization procedure alone will not provide adequate treatment, as the vocal process remains lateralized, leaving a posterior gap with height mismatch. Ideally, arytenoid repositioning procedures aim to mimic the arytenoid's physiologic phonatory position; to medially rotate the arytenoid, lower the vocal process, stabilize and medialize the vocal process, and lengthen the membranous vocal fold. Several techniques have been described, yet *arytenoid adduction* is the most commonly used. Generally, arytenoid adduction includes a suture which mimics the thyroarytenoid and lateral-cricoarytenoid (TA-LCA) muscle complex.[23] The arytenoid is approached through the posterior border of the thyroid cartilage lamina, by elevating the piriform sinus mucosa. Additional removal of some posterior thyroid cartilage can assist with



Figs. 8A and B: (A) Arytenoid adduction: A posterior window is created to allow exposure of the arytenoid. Following retraction of the piriform sinus, a suture is placed through the muscular process of the arytenoid. (B) The limbs of the suture are pulled anteriorly and the vocal process is rotated to the midline. The upper limb of the suture is passed through the cartilage.

arytenoid exposure and manipulations. A nonabsorbable suture is placed through the arytenoid's muscular process with gentle tension in the anterolateral direction to mimic the action of the TA-LCA muscle complex (Fig. 8A). Following adequate arytenoid rotation, the suture is secured to the anterior thyroid cartilage (Fig. 8B).

Combined arytenopexy with cricoid subluxation is another alternative technique. Arytenopexy stabilizes the arytenoid while cricothyroid subluxation on the paralyzed side lengthens the vocal fold by increasing the distance from the cricoarytenoid joint to the anterior commissure.

Compared with medialization laryngoplasty alone, arytenoid repositioning procedures are longer, more surgically challenging and pose higher complication rate.[24] The risk for mucosal perforation is increased due to elevation of the pirifom sinus; airway obstruction and edema are more frequent due to the longer procedure and additional manipulations.

Laryngeal Reinnervation

The goal to regenerate the physiologic motion and sensation in the paralyzed larynx cannot be achieved reliably by current reinnervation techniques, as those generally lead to laryngeal synkinesis. Laryngeal synkinesis is characterized by simultaneous activation of adductor and abductor muscles which can result in immobile vocal fold. Nevertheless, patients with immobile vocal fold with evident laryngeal synkinesis per LEMG were found to have better phonatory function compared to those without synkinesis. This phenomenon could be explained by the improved muscle tone, vocal fold bulk, and to some extent, more favorable arytenoid position.

Cases of obvious nerve transection would greatly benefit from primary nerve anastomosis. In cases of intact RLN, the most useful donor nerve is the ansa cervicalis;[25] it matches the size of the RLN, and there is little to no morbidity from transacting the branch to the sternohyoid or homohyoid muscles. Other donor nerves such as the hypoglossal[26] have been described, yet with much higher donor site morbidity. The reinnervation might be performed by, direct end to end neurorrhaphy, neuromuscular pedicle or direct muscle implant. In the neuromuscular pedicle technique, the nerve ending with a small muscle block from the donor site is transferred and implanted in the denervated laryngeal muscle. Laryngeal reinnervation can be combined with medialization procedures.

SPECIAL CONSIDERATIONS

Vocal Fold Paresis

Paresis implies that there is some residual nerve function. The possible causes for vocal fold paresis are comparable to those of paralysis, however with higher rates of idiopathic/viral cases. Moreover, a high index of suspicion must be maintained for neurologic diseases. Similarly to paralysis, when the cause is not obvious from the patient's history, further workup including imaging of the vagal and recurrent laryngeal course should be obtained. Patients may present with effortful, weak and breathy voice, vocal fatigue, odynophonia, difficulty for projection, and problems

with singing voice. Dysphagia-related symptoms are much less common. On examination, there might be a noticeable impaired vocal fold motion, yet in many patients the signs on fiberoptic laryngoscopy can be subtle and difficult to detect. The examiner should ask the patient for repeated "/i/-sniff" and to reevaluate the examination on a slow-motion, looking for any motion asymmetries. The presence of compensatory muscle tension disorders or vestibular fold hyperadduction is also common clues. Other mild signs that may suggest paresis could be bowing of the vocal fold, incomplete glottic closure, and on stroboscopy: prolonged open phase, unilateral increased amplitude, and asynchronous mucosal wave propagation. As in paralysis, the gold standard for diagnosis is LEMG, in challenging cases it can be useful in determining which side is the affected side. Treatment options in vocal fold paresis are similar to those of paralysis; however, the approach in paresis is generally less aggressive.

Isolated Superior Laryngeal Nerve Paralysis

The SLN may be injured independently of the RLN, following neck surgery (thyroid or nonthyroid), or it might be idiopathic. Symptoms include throat clearing, globus sensation, and cough owing to sensory loss; when sensory loss is significant or bilateral it may even cause aspirations. Vocal symptom due to the loss of cricothyroid muscle function might be noticed only in voice professionals, who would complain of inability to reach high registers. Other vocal symptoms include voice fatigue, diplophonia, and abnormal singing voice. There is also an associated cricopharyngeal muscle dysfunction causing dysphagia.[27] Signs during physical examination would be secretions pooling in the ipsilateral piriform sinus and rotation of the arytenoids and posterior commissure toward the affected side during phonation. Gold standard diagnosis is by LEMG of the cricothyroid muscle. Cases of symptomatic SLN paralysis are generally treated with voice therapy aimed at building cricothyroid muscle strength and stability. Though several surgical treatment options have been tried (e.g. Isshiki type 4 thyroplasty, reinnervation), their use for the treatment of SLN paralysis is uncommon and the outcome is not predictable.

Mechanical Vocal Fold Fixation

The differential diagnosis of an immobile vocal fold includes neurogenic versus mechanical fixation. Mechanical vocal fold fixation is much less common cause for unilateral vocal fold immobility. Nevertheless, mechanical fixation is more commonly associated with bilateral vocal fold immobility by means of posterior glottic stenosis, or bilateral cricoarytenoid joint fixation, and is generally related to severe scarring and contraction. Posterior glottic stenosis is further described in Chapter 35.

Unilateral mechanical vocal fold fixation is very rare and may be related to cricoarytenoid joint fixation due to trauma or rheumatologic disease (e.g. rheumatoid arthritis); or less commonly to cricoarytenoid joint dislocation which is generally related to severe external trauma. Cricoarytenoid joint abnormalities can be differentiated from VFP by either LEMG or palpation during direct laryngoscopy under general anesthesia. It is important to distinguish mechanical fixation from neurologic disorders as physiological vocal fold motion may be restored following the release of fixation.

Pediatric Patients

Vocal fold immobility in pediatric patients differs from the adult population in etiology. In young children and neonates congenital central nervous system disorders, as Arnold-Chiari malformation type II, are the leading cause for bilateral VFP, and iatrogenic injury is the leading cause for unilateral paralysis. Cardiovascular anomalies and surgeries may be responsible for left VFP. Birth trauma is a common cause for paralysis in neonates. As opposed to adults, unilateral vocal fold immobility in children often causes stridor, other common symptoms might be a weak cry, feeding difficulties, and aspirations. Evaluation for etiology includes complete neurologic assessment, MRI of the brain, neck, and mediastinum. Fiberoptic laryngoscopy followed by rigid direct laryngoscopy and examination of the upper airway under anesthesia are recommended, as a great portion of those patients may have an associated upper airway anomaly. Management of VFP in children differs from that in adults, since children have much higher spontaneous recovery rate, which can occur even several years after the onset.[28] Moreover, children compensate well, using the contralateral vocal fold, and only few will have permanent dysphonia. Therefore, most practitioners would prefer to delay intervention in those patients. Nevertheless, in patients with considerable disability, all surgical procedures described for adults can be applied also in pediatric patients; temporary injection, medialization laryngoplasty, arytenoid repositioning, and reinnervation. Importantly, children pose higher risk for airway complications.

Revision Surgery

Possible reasons for suboptimal results which may require a revision surgery are listed above. Many cases are due to implant malpositioning; however, a substantial portion of patients will require additional arytenoid repositioning. When planning the location of the new cartilaginous window, the location of the original window should not be taken into consideration. The same measurements as in primary surgery should be used to establish the new window location, even if there is some overlap between the windows, creating an unusually shaped window. Most implants can be removed easily through the new window. Following the removal of the previous implant and before the insertion of the new implant, the fibrous capsule which was formed around and deep to the implant should be incised.

NEW HORIZONS

Future investigation in VFP treatment is twofold. Since the ideal implant position in medialization laryngoplasty is yet to be determined, the physiological phonatory position of the vocal fold is a subject for ongoing studies. Future development of safe, durable, and easy to use injectable material might produce an attractive alternative for medialization procedures. On the other hand, the restoration of vocal fold motion still poses a challenge, though recent studies on laryngeal pacing have demonstrated promising results.[29-31]

PEARLS

- Vocal fold's position depends on the site of injury, degree of paresis, synkinesis, compensation, and the patient's laryngeal anatomy.
- In the past, thyroid surgery was the most common cause of iatrogenic paralysis; however, currently nonthyroid surgeries, such as anterior cervical approach to the spine and carotid endarterectomies are the most common iatrogenic cause.
- Laryngeal electromyography represents the diagnostic gold standard for VFP and paresis. Useful information from LEMG can be obtained only in the period between 1 month and 6 months after onset.
- Some unilateral VFP patients may recover spontaneously, others may regain normal function within the first year even without vocal fold motion. Therefore, it is common to practice observation for a period of 6–12 months.
- Common causes for suboptimal surgical outcome of medialization laryngoplasty: persistent posterior glottic gap, undermedialization and malpositioning of the implant.
- In some patients medialization procedure alone will not provide adequate correction, due to posterior gap or height mismatch, those patients require additional arytenoid repositioning.
- The suture in arytenoid adduction mimics the TA-LCA muscle complex.
- Laryngeal reinnervation techniques improve phonatory function by enhanced muscle tone and vocal fold bulk and to some extent more favorable arytenoid position.
- Mild signs that may suggest vocal fold paresis: bowing, incomplete closure, prolonged open phase, unilateral increased amplitude, and asynchronous mucosal wave propagation.
- Signs for isolated SLN paralysis would be: secretions pooling in the ipsilateral piriform sinus and rotation of the arytenoids toward the affected side during phonation.
- Mechanical vocal fold fixation can be differentiated from VFP by either LEMG or palpation during direct laryngoscopy under general anesthesia.
- Children have much higher spontaneous recovery rate and they tend to compensate well, therefore only few will have permanent dysphonia.

CONCLUSION

Vocal fold paralysis can be due to general four types of neurologic deficits: (1) CNS, (2) high vagal, (3) isolated RLN, and (4) isolated SLN. When the cause of the vocal fold immobility is not evident, the physician should investigate for etiologies as malignancy and granulomatous diseases. Imaging of the vagal nerve route is mandatory. Factors as prognosis for recovery, degree of impairment, patient's general health, and patient's requirements and desire for recovery influence the timing and type of intervention. The treatment options include observation, voice therapy and surgical intervention by injection augmentation, laryngeal framework surgery, or reinnervation. Medialization laryngoplasty is considered the long-term solution for glottic insufficiency caused by unilateral VFP, vocal fold paresis, or atrophy. Several implant materials were confirmed to be safe and have excellent voice outcome: Silastic, hydroxylapatite, polytetrafluoroethylene ribbon, and titanium. Medialization laryngoplasty may be combined with arytenoid repositioning or reinnervation procedure.

REFERENCES

1. Isshiki N, Morita H, Okamura H, et al. Thyroplasty as a new phonosurgical technique. Acta Otolaryngol. 1974;78:451-7.
2. Isshiki N, Tanabe M, Sawada M. Arytenoid adduction for unilateral vocal cord paralysis. Arch Otolaryngol. 1978;104:555-8.

3. Zeitels SM, Hochman I, Hillman RE. Adduction arytenopexy: a new procedure for paralytic dysphonia with implications for implant medialization. Ann Otol Rhinol Laryngol Suppl. 1998;173:2-24.
4. Zeitels SM, Hillman RE, Desloge RB, et al. Cricothyroid subluxation: a new innovation for enhancing the voice with laryngoplastic phonosurgery. Ann Otol Rhinol Laryngol. 1999;108:1126-31.
5. Merati AL, Shemirani N, Smith TL, et al. Changing trends in the nature of vocal fold motion impairment. Am J Otolaryngol. 2006;27:106-8.
6. Maisel RH, Ogura JH. Evaluation and treatment of vocal cord paralysis. Laryngoscope. 1974;84:302-16.
7. Yamada M, Hirano M, Ohkubo H. Recurrent laryngeal nerve paralysis. A 10-year review of 564 patients. Auris Nasus Larynx. 1983;10 (Suppl):S1-15.
8. Terris DJ, Arnstein DP, Nguyen HH. Contemporary evaluation of unilateral vocal cord paralysis. Otolaryngol Head Neck Surg. 1992;107:84-90.
9. Rosenthal LH, Benninger MS, Deeb RH. Vocal fold immobility: a longitudinal analysis of etiology over 20 years. Laryngoscope. 2007;117:1864-70.
10. Bhattacharyya N, Kotz T, Shapiro J. Dysphagia and aspiration with unilateral vocal cord immobility: incidence, characterization, and response to surgical treatment. Ann Otol Rhinol Laryngol. 2002;111:672-9.
11. Heitmiller RF, Tseng E, Jones B. Prevalence of aspiration and laryngeal penetration in patients with unilateral vocal fold motion impairment. Dysphagia. 2000;15:184-7.
12. Flint PW, Purcell LL, Cummings CW. Pathophysiology and indications for medialization thyroplasty in patients with dysphagia and aspiration. Otolaryngol Head Neck Surg. 1997;116:349-54.
13. Merati AL, Halum SL, Smith TL. Diagnostic testing for vocal fold paralysis: survey of practice and evidence-based medicine review. Laryngoscope. 2006;116:1539-52.
14. Belafsky PC, Postma GN, Reulbach TR, et al. Muscle tension dysphonia as a sign of underlying glottal insufficiency. Otolaryngol Head Neck Surg. 2002;127:448-51.
15. Altman JS, Benninger MS. The evaluation of unilateral vocal fold immobility: is chest X-ray enough? J Voice. 1997;11:364-7.
16. Gupta SR, Bastian RW. Use of laryngeal electromyography in prediction of recovery after vocal cord paralysis. Muscle Nerve. 1993;16:977-8.
17. Willatt DJ, Stell PM. The prognosis and management of idiopathic vocal cord paralysis. Clin Otolaryngol Allied Sci. 1989;14:247-50.
18. Weinman EC, Maragos NE. Airway compromise in thyroplasty surgery. Laryngoscope. 2000;110:1082-5.
19. Rosen CA. Complications of phonosurgery: results of a national survey. Laryngoscope. 1998;108:1697-703.
20. Anderson TD, Spiegel JR, Sataloff RT. Thyroplasty revisions: frequency and predictive factors. J Voice. 2003;17:442-8.
21. Woo P, Pearl AW, Hsiung MW, et al. Failed medialization laryngoplasty: management by revision surgery. Otolaryngol Head Neck Surg. 2001;124:615-21.
22. Cohen JT, Bates DD, Postma GN. Revision Gore-Tex medialization laryngoplasty. Otolaryngol Head Neck Surg. 2004;131:236-40.
23. Woodson GE, Picerno R, Yeung D, et al. Arytenoid adduction: controlling vertical position. Ann Otol Rhinol Laryngol. 2000;109:360-4.
24. Abraham MT, Gonen M, Kraus DH. Complications of type I thyroplasty and arytenoid adduction. Laryngoscope. 2001;111:1322-9.
25. Crumley RL. Update: ansa cervicalis to recurrent laryngeal nerve anastomosis for unilateral laryngeal paralysis. Laryngoscope. 1991;101:384-7.
26. Paniello RC, Lee P, Dahm JD. Hypoglossal nerve transfer for laryngeal reinnervation: a preliminary study. Ann Otol Rhinol Laryngol. 1999;108:239-44.
27. Halum SL, Shemirani NL, Merati AL, et al. Electromyography findings of the cricopharyngeus in association with ipsilateral pharyngeal and laryngeal muscles. Ann Otol Rhinol Laryngol. 2006;115:312-6.
28. Daya H, Hosni A, Bejar-Solar I, et al. Pediatric vocal fold paralysis: a long-term retrospective study. Arch Otolaryngol Head Neck Surg. 2000;126:21-5.
29. Mueller AH, Hagen R, Foerster G, et al. Laryngeal pacing via an implantable stimulator for the rehabilitation of subjects suffering from bilateral vocal fold paralysis: a prospective first-in-human study. Laryngoscope. 2016;126:1810-6.
30. Faenger B, Arnold D, Schumann NP, et al. Method to test the long-term stability of functional electrical stimulation via multichannel electrodes (e.g., applicable for laryngeal pacing) and to define best points for stimulation: in vivo animal analysis. Eur Arch Otorhinolaryngol. 2016 [Epub ahead of print].
31. Foerster G, Arnold D, Bischoff S, et al. Pre-clinical evaluation of a minimally invasive laryngeal pacemaker system in mini-pig. Eur Arch Otorhinolaryngol. 2016;273:151-8.

Textbook of
LARYNGOLOGY

Official Publication of the Association of Phonosurgeons of India

The Association of Phonosurgeons of India (APSI) is a relatively young association, which was founded in 2003. Besides conducting Annual conferences and regional meetings, the Association accredits Laryngology Fellowship Programs for young ENT Surgeons who aspire to specialize in this increasingly popular sub-specialty. Keeping this in mind the APSI decided to release an official textbook on Laryngology, which was conceptualized primarily for the young and budding Laryngologists in India.

However, having reached its completion, we now feel this textbook will serve as an excellent reference guide in the field of Laryngology in the global arena. We have contributions from acclaimed Laryngologists, both National and International, who have a wealth of knowledge and practical experience in their respective domains.

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We hope this textbook is able to achieve its two primary aims: simplifying the complex subject of Laryngology and enabling one to deal with various difficult laryngology scenarios with confidence.

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