Bilateral Vocal Fold Immobility

Introduction and Background

BVFI is broadly categorized as either paralysis or fixation of the vocal folds. It may be clearly related to iatrogenic surgical injury, such as with total thyroidectomy, or to posterior glottic scarring, such as after prolonged intubation. For the rarer cases in which no history of intubation or surgery is present, it requires imaging of the length of the bilateral recurrent laryngeal nerves and sometimes neurologic referral.

The vocal folds are immobile or fixed near the midline, often obstructing the airway and causing stridor or dyspnea, as opposed to the voice complaints of unilateral vocal fold immobility. When loss of innervation is not present, ankylosis of the cricoarytenoid (CA) joint or posterior glottic scarring physically tethers movement of an innervated vocal fold. The latter is referred to as posterior glottic stenosis (PGS) and the former as bilateral vocal fold paralysis (BVFP). Management is directed at securing a safe airway while preserving voice and swallowing function as much as possible. Patient history is crucial; some cases, in fact, will require little further work-up at all when a clear history of airway trauma or prolonged intubation is present.

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Neurologic Etiologies (Bilateral Vocal Fold *Paralysis*)

BVFP may be the first sign of an underlying systemic neurologic condition. External compression, infarct, or degeneration of the main motor nuclei of the Vagus nerve can cause BVFP. Multiple studies have described BVFI and stridor as presenting signs of conditions such as Multiple System Atrophy (MSA) or Arnold-Chiari Malformation Type 2 in neonates, as well as BVFP resulting from compression by an intracranial mass. In these cases, the nucleus ambiguus is affected by the disease. It is important to note other symptoms that might be present but unnoticed by the patient, including dysphagia, dysarthria, tremors, orthostatic hypotension, neurogenic bladder, fatigue, mood changes, and sleep apnea. In the pediatric population, it is important to note any history of hydrocephalus, repair of meningomyelocele at the base of skull, and dysautonomia.

Neurologic referral is indicated, along with brain MRI with and without contrast. MRI has been suggested to be more sensitive for detection of nerve lesions, but it carries a higher rate of false-positives. While these are rare presentations for neurologic disease, idiopathic BVFP itself is rare and thus such evaluation and referral is reasonable. A thorough neurologic history is also indicated, given that tumors in the central nervous system typically cause a constellation of symptoms, some of which may have gone undetected even by the patient. See Table 1.

Table 1: Causes of BVFP

Processes that physically compromise both nerves:

Iatrogenic:

1. Total thyroidectomy
2. Bilateral carotid endarterectomy
3. Skull base/brainstem surgery

Compressive:

Of bilateral recurrent laryngeal nerves:

1. large thyroid mass
2. paratracheal lymphadenopathy
3. massive mediastinal lymphadenopathy
4. clothesline injury to the neck

Of bilateral vagus nerves:

1. brainstem tumor
2. massive bilateral cervical lymphadenopathy

Systemic neurologic conditions:

1. Shy-Drager (Multiple System Atrophy)
2. Bradbury-Eggleston syndrome (Idiopathic Orthostatic Hypotension)
3. Arnold-Chiari Malformation (congenital cases)
4. Hydrocephalus (congenital cases)

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Posterior Glottic Stenosis (Bilateral Vocal Fold *Fixation*)

PGS cases are more common in patients with a history of prolonged intubation. There is direct correlation between length of intubation and risk of developing a scar causing PGS. Other factors include inflammatory and ischemic conditions. The first group involves disorders such as sarcoidosis and rheumatoid arthritis, in which a chronic state of inflammation leads to a poor healing environment. The second group includes presence of 1) large endotracheal tubes (> 7.5 in males), 2) diabetes, 3) post-radiation changes, and 4) caustic ingestion. The latter promote an environment of localized ischemia of the airway mucosa that leads to tissue necrosis and scar formation.

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History

Figure 1 and Table 2 present a general framework for history-taking in BVFI; they are *not* to be interpreted as a definitive algorithm, but simply a guide to the initial history and differential diagnosis.

Figure 1: BVFI flowchart

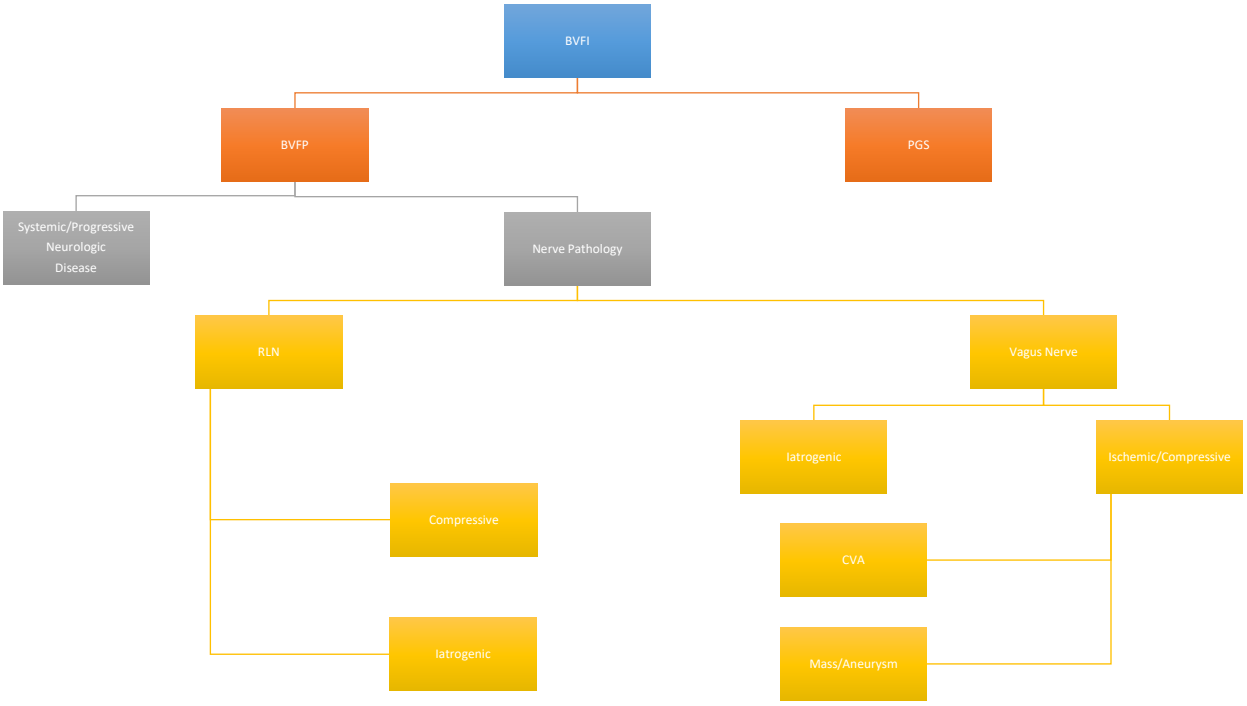

Table 2: Important details from patient history and exam

1. History of recent intubation
    a. Prolonged?

b. Incomplete sedation or significant fighting against the tube?
c. Multiple reintubations?
d. Large tube? (8-0 or greater)
e. Traumatic placement of tube?
f. Site of placement? (in the field, in ER, in OR and by whom)

2. Reasons for poor healing, scarring, or inflammatory tendency:
    a. Poorly controlled DM
    b. Rheumatoid arthritis, polychondritis, or other autoimmune illness
    c. Chronic respiratory tract infection
    d. Severe reflux disease
    e. Hypo- or hyperthyroidism
    f. History of radiation to neck
    g. History of burn, inhalational injury or caustic ingestion
3. Recent surgery? Possibility of iatrogenic nerve injury?
4. Large thyroid or mediastinal masses?
5. History of recent stroke – does it localize to brainstem?
6. History of skull base or high cervical spine surgery?
7. Symptoms suggestive of progressive neurologic disease?
    a. Change in gait, mood, cognition, strength
    b. Change in speech
    c. Numbness or weakness
8. History of clothesline injury to the neck

Intraoperative Diagnosis and Findings

A trip to the operating room (OR) is often indicated due to symptoms of dyspnea and stridor. A safe airway can be secured via tracheostomy or by enlarging the posterior glottis through cordotomy, medial arytenoidectomy, total arytenoidectomy, suture lateralization, or a combination. Evaluation of the interarytenoid space and CA joint palpation (as described by Simpson and Rosen) can assist in differentiating bilateral paralysis from joint or scar fixation. Similarly, when a patient with severe stridor pre-operatively becomes relatively quiet with sedation and/or paralysis, a BVFP may be more likely; if the vocal folds are not fixed by scar or CA joint fixation, the positive pressure applied during induction may easily open the glottis and eliminate the stridor. Special attention should also be placed on the evaluation of the subglottis and trachea to rule out secondary levels of airway stenosis, particularly when scar or an inflammatory process is identified as the cause of the BVFI; the same intubation that caused a posterior glottic scar band or CA joint fixation could also have caused a subglottic or tracheal stenosis.

Risk of creating new dysphagia or dysphonia must be discussed pre-operatively.

Any improvement in airway is often at the cost of worsened voice quality and often swallowing function as well. Pre-operative pharyngeal swallowing assessment such as a modified barium swallow (MBS) or functional endoscopic evaluation of swallowing (FEES) might be considered

in older patients, those with any risk for pre-existing swallowing dysfunction, or those who would poorly tolerate post-operative dysphagia or aspiration due to pulmonary status.

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Imaging

In cases in which BVFI is iatrogenic or clearly related to PGS, imaging is seldom necessary. But in cases of BVFP without an obvious cause, imaging of the nerve path from brainstem to entry into larynx is indicated. The RLN is typically imaged with contrast CT and the proximal portion of the vagus nerve with a brainstem MRI, with and without contrast. While it could be argued that only the RLN path and not the vagal nerve path is necessary when palate movement is normal, BVFP absent the possibility of iatrogenic injury is often elusive enough that the entire tract is included upfront, under the assumption that the patient is likely to go on to eventual neurology referral and brain MRI anyway.

Studies showing the diagnostic utility of imaging studies for cases of BVFI are sparse at best. Therefore, most decision-making is extrapolated from unilateral vocal fold immobility (UVFI) studies. Many of these studies have studied CT as the standard screening tool for UVFI, with rates of positive findings ranging from 15 – 62%, depending on the composition of the study population. In cases of PGS, CT is less helpful but may show abnormal soft tissue obliterating the glottic airway, medialized vocal folds or arytenoid cartilages, and/or malignancy involving the CA joints or paraglottic space. Since most PGS patients have been intubated at some time, CT may be most helpful in these cases for revealing subglottic stenosis and/or tracheal stenosis, and to this end, fine cuts (1-2 mm) should be requested upfront. Ultrasound has been inadequately evaluated but may have some utility in pediatric populations.

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Laryngeal EMG

Laryngeal EMG (LEMG) may prove helpful in the work-up of BVFI. When operative assessment does not provide clear evidence of either joint fixation or presence of posterior glottic scar, an EMG that shows normal innervation can definitively rule out BVFP. More importantly, however, in cases of suspected or confirmed paralysis, it can provide prognostic information to guide patient expectations and timing of operative interventions. LEMG provides objective information about the state of the motor unit: normal innervation, absence of innervation, reinnervation, or synkinesis, using specific characteristics in the electrical signals obtained.

The timing is important. Testing too early or late is not useful. It takes at least 4 and possibly 12 weeks for nerve damage to reliably be seen in LEMG. Low correlation with outcomes is seen after 6 months. Patients with BVFI often have a marginal airway if tracheostomy has not been performed; thus the manipulation of laryngeal structures during the test contraindicates LEMG in many of these patients.

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