Vocal fold paresis

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Vocal fold paresis (VFP) is a relatively common and often overlooked condition that can be difficult to diagnose based on the laryngeal examination alone. A retrospective review of the records of 50 consecutive adult patients with VFP was performed. In each case, the diagnosis of VFP was confirmed by laryngeal electromyography. The presenting symptoms were dysphonia (100%), vocal fatigue (76%), diplophonia (40%), and odynophonia (12%), and the findings were unilateral vocal fold hypomobility (50%), unilateral bowing (36%), and bilateral bowing (22%). Laryngoplasty and/or lipoinjection was performed in 54% of the subjects, and significant vocal improvement was achieved in 85%. VFP appears to be underdiagnosed because many VFP patients have compensatory hyperkinetic disorders at presentation. Although the diagnosis of VFP may be suspected based on the patient's symptoms and findings, the diagnostic sine qua non is laryngeal electromyography. In addition, surgical treatment for VFP appears to be safe and effective.
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Refinements in videoendoscopy, acoustical analysis, and laryngeal electromyography (LEMG) now make it possible to diagnose relatively subtle laryngeal neuromuscular disorders. These diagnostics allow the clinician to detect and differentiate fine degrees of glottal hyperfunction and hypofunction. In particular, transnasal fiberoptic laryngoscopy (TFL) has been found to be very effective in assessing laryngeal biomechanics with a variety of different glottal and vocal tasks.[1-3]

In patients with functional, nonorganic, muscle-tension dysphonias, the finding of excessive supraglottic contraction is usually associated with symptoms of dysphonia, effortful phonation, vocal fatigue, and odynophonia.[4,5] Similar abnormal muscle tension patterns (MTPs) and symptoms are seen in patients with organic (pathologic) conditions. For example, compensatory MTPs may be seen when there is excessive vocal fold soft tissue (eg, Reinke's edema, vocal fold polyps, and nodules), deficient or deformed soft tissue (eg, presbylaryngis, vocal fold scarring), or neuromuscular disease (eg, spasmodic dysphonia, parkinsonism). In fact, compensatory glottal behaviors (MTPs) accompany virtually all organic voice disorders.[2] This is because, at the time of presentation, patients with voice disorders have already adopted compensatory vocal behaviors. Thus hyperkinetic glottal behaviors may be *primary* when associated with functional vocal abuse/misuse/overuse syndromes or *secondary* when compensatory for any underlying glottal closure abnormality.[2]

In vocal fold paralysis, the most severe degree of vocal fold paresis (VFP), the most common compensatory MTPs are anteroposterior vocal fold shortening and side-to-side false vocal fold compression. In paralysis, failure to achieve glottal closure on phonation is obvious, but in paresis, the subtlety of the neuromuscular deficit may be overlooked. Thus the diagnosis may be falsely presumed to be a primary functional (muscle tension) dysphonia. In VFP, differentiation from a primary muscle tension dysphonia often requires a sophisticated diagnostic approach; however, regardless of the patient's clinical pattern, the diagnostic sine qua non for VFP remains an abnormal LEMG.

Reported herein are 50 patients with VFP seen at the Center for Voice Disorders during a 5-year period. Simultaneous with diagnostic advances, the surgical treatment of VFP has evolved. In properly selected cases, surgical correction of VFP appears to be very successful. Our approach to the diagnosis and treatment of VFP is presented.

METHODS AND MATERIAL

A retrospective review of the medical records and videotaped laryngeal examinations of 50 consecutive patients with documented VFP seen between 1991 and 1995 was undertaken.

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Table 1. Laryngeal electromyographic findings

Electromyographic findings	n	%
Unilateral findings		
Isolated RLN	17	34
Isolated SLN	5	10
Combined SLN & RLN	8	16
Bilateral findings		
Bilateral RLN	5	10
Bilateral SLN	3	6
Bilateral SLN & RLN (3 or 4 nerves affected)	12	24
TOTAL	50	100

All of the patients were adults; 38 were women, and 12 were men. The mean age of the female subjects was 49.0 years (range 24-76 years), and the mean age of the male subjects was 53.5 years (range 36-66 years).

All study patients underwent the following evaluations: (1) a complete medical history, reflux history,[6] and review of systems; (2) TFL with videostroboscopy; (3) acoustical analysis; and (4) LEMG.[7-9]

In addition to connected speech and sustained vowel samples, we routinely use a variety of vocal tasks during TFL to evaluate the neuromuscular integrity of the larynx. The vocal task used to assess vocal fold mobility is alternating phonation of /i/ with sniffing in rapid succession (ie, /i/, sniff, /i/, sniff, etc). This maneuver produces full adduction and full abduction of the vocal folds. With video recording of the examination, this task can be replayed in slow motion for review. In addition, patients are asked to phonate /i/ using several pitches, including the highest possible pitch. This maneuver is used to look for axial rotation (believed to be a sign of superior laryngeal nerve [SLN] neuropathy).[10]

In addition to the acoustical data, each patient's voice was rated on a 5-point scale by a speech-language pathologist: 1, normal; 2, minimal dysphonia; 3, moderate dysphonia; 4, severe dysphonia; and 5, aphonia.

All patients were followed up for a minimum of 6 months, with a median duration of follow-up of 11 months. Of the 28 patients who had surgical treatment, 6-month postoperative voice assessments were performed in 64% (18/28). (The acoustical data will be presented in a separate report.)

RESULTS

Of the 50 study subjects, 76% (38/50) were female, and 24% (12/50) were male. All 50 patients had dysphonia at presentation. The next most common symptoms were effortful phonation and vocal fatigue, 76% (37/50); diplophonia, 40% (20/50); and odynophonia, 12% (6/50).

The most common laryngeal findings were unilateral hypomobility, 50% (25/50); unilateral vocal fold bowing, 36% (18/50); bilateral bowing, 22% (11/50); bilateral hypomobility, 8% (4/50); and axial rotation, 8% (4/50). In some cases, the bowing and hypomobility were subtle. In many patients with hypomobility, abduction was affected more frequently and more noticeably than adduction. Nearly all of the patients demonstrated abnormal MTPs, with anteroposterior vocal fold shortening and false vocal fold compression almost universally observed.

Of the 50 subjects, LEMG revealed unilateral neuropathic findings in 60% (30/50) and bilateral findings in 40% (20/50). Unsuspected contralateral neuropathy was found in 26% (13/50). Isolated SLN was found in 16% (8/50); isolated recurrent laryngeal nerve (RLN) neuropathy was found in 44% (22/50); and combined (SLN and RLN) neuropathy was found in 40% (20/50). These findings included decreased recruitment, abnormal waveform morphology, and synkinesis. The details of LEMG methodology and interpretation are described elsewhere.[7,9] Table 1 summarizes the LEMG findings.

The presumed cause of the VFP was idiopathic, 44% (22/50); postviral neuropathy, 24% (12/50); iatrogenic, 20% (10/50); malignancy 6% (3/50); multiple sclerosis, 4% (2/50); and postchemotherapy peripheral neuropathy, 2% (1/50). Subjects in the idiopathic group had no precipitating or preceding cause or medical condition. All of the subjects in the postviral group reported that they had had an upper respiratory infection at the onset of their voice problems. Therefore inclusion in this subgroup was based on the patient's history alone. We did not perform any viral antibody or other definitive laboratory investigations.

Of the 10 subjects in the iatrogenic group, 7 had prior endotracheal intubation for surgery not involving the head, neck, or upper chest; 2 had prior thyroid surgery; and 1 had a carotid endarterectomy. All 10 of the patients in this group noted an immediate voice change after surgery. Interestingly, of the 7 patients with postintubation VFP, 4 had SLN neuropathic findings and 4 had bilateral LEMG findings. Of the 12 subjects in the postviral group, 5 had bilateral findings. The LEMG results of the subjects with postintubation and postviral VFP are summarized in Table 2.

Three patients had malignancy as the cause of the VFP; 2 had thyroid carcinoma, and the third had lymphoma involving the superior mediastinum. Finally, multiple sclerosis was diagnosed in 2 patients, and another had a peripheral neuropathy secondary to chemotherapy.

The types and results of treatment are in part reported elsewhere,[11,12] and are summarized in Table 3. We believe that the inclusion of these data is important because the data are encouraging; however, the details

Table 2. Correlation between laryngeal and LEMG findings in the postintubation and postviral subgroups

Subject No./ age (y)/sex	Laryngeal findings (by fiberoptic exam)	Abnormal EMG			
		RCT	LCT	RTA	LTA
Postintubation group					
1/45/F	L paresis & bowing	–	–	–	+
2/37/F	L bowing & rotation	–	–	+	+
3/30/F	Bilateral bowing	+	+	–	+
4/62/F	Supraglottic contraction	–	+	–	–
5/43/M	L paresis & bowing	+	–	–	+
6/43/M	Bilateral bowing	+	–	–	+
7/62/M	R paresis & bowing	–	–	+	–
Postviral group					
8/40/F	L bowing & paresis	–	–	–	+
9/41/F	L bowing & paresis	–	–	–	+
10/50/F	R paresis	+	–	–	–
11/45/F	L bowing	–	–	–	+
12/43/F	L bowing & rotation	–	–	–	+
13/51/F	R bowing & paresis	+	+	–	–
14/51/F	L bowing & paresis	–	+	–	+
15/62/F	Bilateral bowing	+	+	+	–
16/63/F	L bowing & paresis	–	–	+	+
17/50/F	L paresis	–	–	+	+
18/69/F	Bilateral bowing	+	+	–	–
19/61/M	R paresis	–	–	+	–

RCT, Right cricothyroid muscle; *LCT,* left cricothyroid muscle; *RTA,* right thyroarytenoid muscle; *LTA,* left thyroarytenoid muscle; *L,* left; *R,* right; –, normal; +, abnormal.

of technique and outcome of treatment are beyond the scope of this report.

DISCUSSION

During the last few years, we have diagnosed VFP with increasing frequency. This is because we have come to recognize the following: (1) patients with hypokinetic laryngeal conditions often have hyperkinetic findings at presentation (ie, muscle tension dysphonias); (2) the diagnosis of subtle movement disorders, such as VFP, often cannot be made by peroral examination methods that alter and distort laryngeal biomechanics[2,11]; (3) although asymmetrical arytenoid movement and asymmetrical stroboscopic findings are common, by themselves they are not diagnostic of intrinsic neuromuscular disease; (4) LEMG is an essential diagnostic test that provides crucial information about the neuromuscular integrity of the larynx that no other test can provide; and (5) patients with voice disorders often have more than one underlying disorder, and each problem must be identified and corrected if the patient is to have a highly successful outcome.

Hypokinetic Voice Disorders May Present as Hyperkinetic Voice Disorders

Incomplete glottal closure of any cause may present with similar symptoms; it is the TFL findings and laboratory testing results that vary with each cause. Paresis, presbylaryngis, and vocal fold scarring, for example, may all cause similar symptoms, and all may present with hyperkinetic findings. In general, the younger patients are, the more likely they are to compensate adequately.

Case Reports

Case 1. A 24-year-old third-grade teacher had recalcitrant vocal nodules at presentation. She had problems with breathy dysphonia, effortful phonation, vocal fatigue, and odynophonia for 3 years. She dated the onset of her problem to an upper respiratory infection. Examination revealed sluggish mobility of the left vocal fold and bowing of the right vocal fold. On stroboscopy across her limited pitch range, the right vocal fold appeared relatively flaccid. Electroglottography revealed a glottal waveform that was too open. LEMG revealed right SLN and left RLN neuropathies.

Case 2. A 42-year-old businessman had breathy dysphonia of 2 years' duration and a recurrent left vocal process granuloma. During this period, he had been using a proton pump inhibitor to control reflux, and he had the granuloma removed 7 times. At presentation, his only symptoms were chronic dysphonia and vocal fatigue. Examination revealed sluggish mobility of the left vocal fold, and LEMG revealed a left RLN neuropathy.

Table 3. Treatment modalities and results of treatment of 35 subjects

Treatment	Results (%)*		
	Excellent	Good	Poor
Voice therapy alone (n = 8)	3 (38)	4 (50)	1 (13)
Unilateral medialization (n = 13)	6 (40)	4 (27)	3 (20)
Bilateral medialization (n = 10)	4 (40)	5 (50)	1 (10)
Lipoinjection alone (n = 1)	—	1 (100)	—
Medialization & lipoinjection (n = 3)	—	3 (100)	—
TOTAL	13 (37)	17 (49)	5 (14)

Fifteen (30%) of the study subjects did not have any treatment or were lost to follow-up; 8 (16%) had voice therapy alone; and 27 (54%) had surgical treatment.

**Excellent,* normal voice (ie, class I); *Good,* near-normal voice, (ie, mild dysphonia, class II); *Poor,* moderate dysphonia (ie, class III).

Table 4. Summary of the criteria for performing LEMG to rule out VFP

Symptoms
- Breathy dysphonia, diplophonia, effortful phonation, vocal fatigue, odynophonia, loss of the high range of the voice

Laryngeal findings
- Asymmetrical vocal fold (arytenoid) movement, especially hypomobile abduction
- Unilateral vocal fold bowing or bilateral bowing in a patient aged <50 y
- Asymmetrical stroboscopy with relative flaccidity of one vocal fold across the pitch range
- Axial rotation on high-pitched phonation
- Pitch-locked, false vocal fold phonation (plica ventricularis)

Voice laboratory findings
- Abnormal electroglottography (too open)
- Loss of the high range of the voice
- Recalcitrant muscle tension dysphonia (eg, persistent vocal nodules) in a patient who appears to be complying with voice therapy

Hyperkinetic conditions that may be associated with underlying paresis
- Contact ulcers/granulomas
- Localized or unilateral Reinke's edema
- Vocal nodules
- Free-edge leukoplakia, especially in a nonsmoker
- Recalcitrant muscle tension dysphonia

Case 3. One year after having undergone a bilateral salpingo-oophorectomy under general endotracheal anesthesia, an otherwise healthy 30-year-old woman presented with severe breathy dysphonia and diplophonia. She dated the onset of her voice problem to the day of her surgery. In addition to the dysphonia, she reported effortful phonation, vocal fatigue, and loss of the high range of the voice. Examination revealed severe bowing of the vocal folds and sluggish mobility of the left vocal fold. LEMG findings confirmed "old" bilateral SLN neuropathies and a left RLN neuropathy. Subsequently, bilateral medialization laryngoplasty was performed with a dramatic and long-lasting restoration of voice.

All of these examples demonstrate a *hyperkinetic* compensation for an underlying *hypokinetic* glottis. In some individuals, this compensatory muscle tension dysphonia may lead to organic pathology such as nodules or granulomas.

We currently have a much higher index of suspicion for VFP than previously. Table 4 summarizes our criteria for performing LEMG to rule out VFP. It is important to note that for most VFP patients, many of the criteria are met. By itself, however, unexplained diplophonia (double-tone) is a reason to perform LEMG. This is because diplophonia implies differential tension or mass between the two vocal folds (ie, diplophonia implies paresis unless proved otherwise).

LEMG Is an Essential Diagnostic Test

Despite extensive experience and a high index of suspicion for VFP, we find that they still require LEMG for diagnosis, particularly when attempting to differentiate paresis from arytenoid fixation.[7,9] In addition, at presentation patients with glottal closure problems such as paresis may have vocal nodules, vocal process granulomas, or other lesions that result from hyperkinetic compensation, so that LEMG becomes essential as a test of neuromuscular integrity.

We do not suggest that all patients with hyperkinetic lesions undergo LEMG. LEMG is indicated for patients with symptoms and findings that suggest an underlying hypofunctional state. One of the most important diagnostic markers is the patient's response to voice therapy. Patients with primary muscle tension dysphonias usually show improvement with voice therapy—softening of glottal attack, good breath support, and relaxation of laryngeal and neck muscles—whereas patients with VFP do not. In the hands of an experienced voice therapist, if the muscle tension dysphonia patients do not improve, we recommend that LEMG be performed as part of the patient's evaluation.

CONCLUSIONS

1. VFP is more common than previously realized, and it may be disguised as a hyperkinetic voice disorder.
2. Red flags for VFP are diplophonia, effortful phonation, asymmetrical or unilateral vocal fold bowing, and vocal fold hypomobility.
3. Suspicion of VFP, based on symptoms and fiberoptic laryngoscopy, must be confirmed by LEMG, which is the diagnostic sine qua non for VFP.
4. Surgical treatment of VFP (laryngoplasty and/or

injection augmentation) appears to be safe and effective.

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