

Vocal Fold Paresis: Clinical and Electrophysiologic Features in a Tertiary Laryngology Practice

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Summary: A retrospective chart review was performed at the senior author's voice disorder clinic to report the symptoms, signs, and laryngeal electromyography (LEMG) data of patients presenting with vocal fold paresis (VFP) in a tertiary laryngology academic practice over a 4-year period. Medical records of 739 patients presenting to the clinic with a chief complaint of dysphonia (for 2000–2004) were assessed. History intake forms, stroboscopy images, and LEMG reports were reviewed for all patients with a clinical diagnosis of VFP. Of the 739 patients presenting to the clinic with voice complaints, 195 were initially diagnosed with either vocal fold paralysis or VFP (26.4%). Only 13 out of 739 patients (1.8%) with voice complaints were diagnosed with LEMG-confirmed unilateral or bilateral VFP. The most common findings on stroboscopy were vocal fold bowing (70%), incomplete closure (62%), and increased vibratory amplitude (38%). Seventy percent of the patients had unilateral VFP, predominantly isolated recurrent laryngeal nerve (RLN) disease. Only 9% had unilateral superior laryngeal nerve (SLN) involvement. The most common LEMG abnormality was reduced recruitment of motor units. In our voice center, VFP was a relatively uncommon diagnostic entity. Despite the low prevalence, VFP needs to be considered in all patients who present with dysphonia. Further study is needed to examine the prevalence of "abnormal" LEMG studies in an *asymptomatic control population*, and to determine the utility of LEMG in the evaluation and management of dysphonia. In the same way that stroboscopy has been critically evaluated in the past, there is also a need to determine how commonly LEMG contributes essential data which leads to a change in the patient's management and/or ultimate vocal outcome.

Key Words: Vocal fold paresis–Vocal fold paralysis–Laryngeal electromyography.

INTRODUCTION

Vocal fold paresis (VFP) has been a controversial subject—its incidence described as relatively common and underdiagnosed by some authors,^{1,2} and rare/overdiagnosed by others.

Clinically, the characteristics of VFP have not been fully described and its diagnostic criteria are somewhat ill defined. As with laryngopharyngeal reflux disease (LPR) 20 years ago, VFP has been a lightning rod of controversy. "Believers" and "nonbelievers" have declared themselves over the past several years, as nascent evidence is beginning to accumulate which supports VFP as a real clinical entity. The senior author acknowledges that VFP is a true clinical entity that must be addressed, but is willing to concede that, like LPR, this entity can be overdiagnosed.

The aim of this study is to report the clinical and electrophysiological features of VFP in the senior author's tertiary laryngology practice. A retrospective review of patients presenting complaints, videostroboscopy characteristics, and laryngeal electromyography (LEMG) findings were examined to evaluate for a diagnosis and characterization of VFP.

METHODS

A retrospective review of medical records of 739 patient charts from the senior author's clinic in the period between January

2000 and November 2004 was carried out. Patients who presented to the clinic with voice complaints such as dysphonia, vocal fatigue, and odynophonia were retrieved by current procedural terminology (CPT) code for videostroboscopy (31579), as videostroboscopy was performed on every patient with voice complaints. New patients who were diagnosed with unilateral or bilateral vocal fold paralysis or VFP were then selected based on International Classification of Diseases (ICD)-9 codes (478.31 and 478.33). Patients with complete vocal fold paralysis (immobile vocal fold) were then separated from patients with VFP.

Patients with findings in the history and/or videostroboscopy examination suggestive of VFP were evaluated by LEMG. Common indications for performing LEMG included vocal fatigue/dysphonia and/or changes in the singing voice, especially in cases that did not respond to medical and/or surgical management. Suggestive laryngeal videostroboscopy (LVES) examination findings included increased unilateral amplitude, "chasing wave," rotation of the posterior commissure on high-pitched phonation, or subtle asymmetry in vocal fold movement.

Patients with LEMG findings consistent with unilateral or bilateral VFP were then reviewed. A voice questionnaire and Voice Handicap Index (VHI)-30 was reviewed for each patient to screen for common presenting symptoms. All patients received a physical examination including videostroboscopy with a flexible laryngoscope initially, although 70° rigid laryngoscopies were used in addition, in selective patients. Patients were examined using the "i/-sniff" maneuver to observe full adduction and abduction of the vocal folds. Cases of complete immobility (suggestive of paralysis or fixation) were excluded from this study. The technique of "unloading" as described by Koufman,³ was also used to reveal subtle glottic insufficiency

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that might have otherwise been hidden under compensatory muscle tension patterns.

A slow-motion videostroboscopy review was performed, focusing on incomplete closure of the vocal folds and also on vibratory characteristics, including the presence of increased amplitude and a chasing wave. In addition, alternating low and high frequency phonations were used to evaluate the presence or absence of posterior laryngeal tilting/shifting. LEMG was carried out, evaluating the thyroarytenoid (TA—testing the recurrent laryngeal nerve [RLN]) and cricothyroid (CT—testing the superior laryngeal nerve [SLN]) muscles bilaterally. This needle insertion was performed by the senior author, and LEMG interpretation was provided by a neurologist with extensive clinical experience in electrodiagnostics. The diagnosis of VFP was confirmed by the one or more of the following in either TA or CT muscle: presence of decreased recruitment of motor unit potentials (MUPs), polyphasic MUPs, or fibrillation potentials.

RESULTS

Of the 739 patients presenting to the clinic with voice complaints, 195 were clinically diagnosed with either vocal fold paralysis or VFP (26.4%). Nineteen of these 195 patients were tentatively given a clinical diagnosis of VFP. Of these 19 patients, 14 agreed to undergo LEMG. LEMG confirmed the diagnosis of unilateral or bilateral VFP in 13 of 739 patients (1.8%) with voice complaints (Table 1). One of the 14 patients with suspected VFP had a normal LEMG.

Of the 13 patients diagnosed with LEMG-confirmed VFP, the mean patient age was 52.5 years. Nine of the 13 patients (69.2%) were female with a mean age of 51 years; the remaining five male patients (30.8%) had a mean age of 56 years. One third of the patients presented with a sudden onset of dysphonia, one third with gradual onset, and the remaining one third with episodic recurrent dysphonia. The mean interval between onset of symptoms and the actual LEMG testing was 18 months (range 2–60 months); however, eight of the 14 patients (57%) underwent LEMG within a year of the onset of symptoms.

All VFP patients presented with the initial complaint of hoarseness, with 61.5% complaining of a breathy voice and 46.2% of vocal fatigue. Four out of the five patients who

TABLE 1.
Demographics

Year	Patients Presenting with Voice Complaint	Patients with Initial Diagnosis of Vocal Fold Paralysis (VFP)	Patients with LEMG-confirmed VFP
2000	91	28 (U 19, B 9)	4
2001	60	39 (U 26, B 13)	2
2002	190	12 (U 7, B 5)	3
2003	192	48 (U 34, B 14)	4
2004	206	68 (U 54, B 14)	0
Total	739	195	13

Abbreviations: U, unilateral; B, bilateral.

TABLE 2.
Presenting Complaints for Patients with LEMG-Confirmed VFP

Initial Patient Complaint	Number of Patients (%)
Hoarseness	13/13 (100)
Breathy voice	8/13 (61.5)
Loss of volume	7/13 (53.8)
Voice fatigue	6/13 (46.2)
Frequent throat clearing	4/13 (30.8)
Post-nasal drip	4/13 (30.8)
Globus	3/13 (23.1)
Throat pain	3/13 (23.1)
Loss of quality in high pitch	3/13 (23.1)
Loss of range—high pitch	3/13 (23.1)
Difficulty swallowing	2/13 (15.4)
Cannot sing anymore (singers)	2/13 (15.4)
Loss of vocal stamina (singers)	1/13 (7.8)
Takes longer to warm-up (singers)	1/13 (7.8)

were either singers or music teachers listed loss of quality in the high end of their singing range and/or loss of range as their primary symptom (Table 2).

The incidence of the common presenting complaints is compared to the Koufman et al² study in Table 3. The ubiquitous presentation of dysphonia is congruent with Koufman's findings, although the complaint of vocal fatigue was found in considerably fewer patients in our clinic.

More than 60% of the patients were found to have vocal fold bowing and/or incomplete glottal closure during videostroboscopy. Only one patient was found to have "paresis podule," as previously described by Koufman and Belafsky⁴ (Table 4).

Out of 13 VFP patients undergoing LEMG, results showed decreased recruitment of MUPs for all patients, whereas three patients demonstrated polyphasic MUPs, and two patients had fibrillation potentials. Of those patients, 69% had unilateral VFP, with 46% of patients demonstrating isolated RLN disease. Only 9% had unilateral SLN disease and 15% had combined RLN and SLN pareses. Fifteen percent of the patients had bilateral RLN paresis, 15% had bilateral SLN and RLN pareses where 3–4 nerves were affected, whereas none of the 13 patients had bilateral SLN paresis (Table 5).

In seven of the 14 patients who underwent LEMG (50%), the information obtained from the LEMG changed the management of the patient. In four patients, the LEMG information was used to guide voice therapy. In one patient (because of confirmation

TABLE 3.
Initial Presentation Compared with Koufman et al²

Initial Complaint	Simpson (N = 13) (%)	Koufman et al (N = 50) (%)
Dysphonia	100	100
Vocal fatigue	46.2	76
Diplophonia	N/A	40
Odynophonia	23.1	12

TABLE 4.
Stroboscovideolaryngoscopy Findings

Laryngeal Findings	Number of Patients (%)
Vocal fold bowing	9/13 (70)
Incomplete glottal closure	8/13 (62)
Increased amplitude	5/13 (38)
Vocal fold hypomobility	3/13 (23)
Vocal fold "shortening"	2/13 (15)
Posterior rotation with high-frequency phonation	2/13 (15)
Paresis podule/pseudocyst ⁴	1/13 (7.8)

of diagnosis), the surgical management was changed (bilateral laryngoplasty instead of unilateral), and in another, the LEMG results obtained early in the disease course allowed observation. Another patient was referred to a neurologist after LEMG results were obtained, eventually leading to a diagnosis of Evan's disease. Finally, one patient was treated successfully with Gabapentin after an SLN neuropathy was noted.

DISCUSSION

As VFP still seems to represent a controversial entity, we consider our point of view regarding VFP to be a "centrist," or moderate stance. We acknowledge that VFP is indeed, a true clinical entity, but perhaps is not a common *clinically relevant* entity in patients presenting with dysphonia.

According to the analysis of dysphonic patients presenting to our voice clinic, only 1.8% had confirmed VFP. It is likely that a number of patients had "suspected paresis" based on history and/or LVES findings, but declined to undergo an LEMG, generally because of the lack of interest in undergoing further testing or treatment for their condition. In these cases, additional LEMG data may have changed the overall incidence of VFP in our study, but would not likely have contributed a meaningful change in the patient's outcome.

Other factors which may have contributed to a falsely low estimation of VFP include (1) patients whose condition improved before an LEMG was performed or were lost to follow-up and not included, (2) investigator bias, as patients were selected for

LEMG based on subjective findings on videostroboscopy, and (3) patient refusal to undergo the LEMG study because of apprehension of the procedure.

More research is needed to correlate positive LEMG findings with their clinical significance. We do not know the *clinical* relevance of the finding of "reduced recruitment" of MUPs on LEMG. Reduced recruitment of motor units is a subjective finding that cannot be adequately quantified.

Perhaps a better way to look at VFP would be to examine the utility of LEMG in the treatment of patients with dysphonia; in the same way that videostroboscopy has been evaluated.^{5,6} In other words, in what percentage of patients does LEMG alter the diagnosis and treatment of our patients, and in what percentage of patients does this result in a *meaningful difference in the patient's ultimate outcome*? Our small sample of patients revealed that 50% of patients had a change in the management because of LEMG findings. The number could be lower if all patients had LEMG performed (which is an impractical and unnecessary protocol), as subtle degrees of paresis might not have any effect on the ultimate management of the patient. Evidence-based studies are obviously needed to define the role of LEMG in patients with dysphonia.

CONCLUSION

VFP is an uncommon clinical entity in our practice, yet does play a role in a small number of patients who present with dysphonia. Patients present commonly with hoarseness, loss of volume, vocal fatigue, and loss of their upper vocal register. Females in their 40–50 years of age predominate versus their older male counterparts. Suggestive findings on videostroboscopy include incomplete closure, asymmetry of vocal fold abduction/adduction, and increased vibratory amplitude on the affected side. LEMG is used to confirm the diagnosis, and most commonly reveals unilateral RLN disease. LEMG findings are generally limited to reduced recruitment of MUPs on the affected side. VFP should be considered in the differential diagnosis of patients presenting with dysphonia. Further research is needed to determine the clinical significance of abnormal LEMG findings and to establish the diagnostic utility of LEMG in the evaluation of the dysphonic patient.

TABLE 5.
LEMG Findings Compared with Koufman et al²

Electromyographic Findings	Simpson (%)	Koufman et al (%)
Unilateral		
Isolated RLN neuropathy	6/13 (46)	17/50 (34)
Isolated SLN neuropathy	1/13 (8)	5/50 (10)
Combined RLN/SLN neuropathy	2/13 (15)	8/50 (16)
Bilateral		
RLN neuropathy	2/13 (15)	5/50 (10)
SLN neuropathy	0/13 (0)	3/50 (6)
SLN/RLN neuropathy (3–4 nerves affected)	2/13 (15)	12/50 (24)

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