

# Determining the Etiology of Mild Vocal Fold Hypomobility

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**Summary:** The prevalence of mild vocal fold hypomobility is unknown. In a study by Heman-Ackah et al, vocal fold hypomobility in a population of singing teachers was found to be associated more frequently with vocal complaints than was the presence of vocal fold masses. The etiology of mild vocal fold hypomobility has not been previously explored. In the present study, a retrospective chart review was performed of 134 patients who presented to a tertiary laryngology referral center over a 6-month period for evaluation of vocal complaints. Of the 134 patients, 61 (46%) were found to have mild vocal fold hypomobility previously undiagnosed by the referring otolaryngologist. Imaging studies and laboratory tests to evaluate for structural, metabolic, and infectious causes of the decreased mobility had been ordered. Forty-nine patients completed the work-up. Of these, 41 out of 49 (84%) were found to have imaging or laboratory findings that could explain the hypomobility. Thyroid abnormalities were found to be associated with vocal fold hypomobility in 21 out of 49 (43%) of those with a complete evaluation. Other causes of vocal fold hypomobility included idiopathic (8 of 49, 16%), viral neuritis (5 of 49, 10%), central nervous system abnormality (4 of 49, 8%), neural tumor (3 of 49, 6%), joint dysfunction (3 of 49, 6%), iatrogenic nerve injury (2 of 49, 4%), myopathy (2 of 49, 4%), and noniatrogenic traumatic nerve injury (1 of 49, 2%). This study shows that unilateral vocal fold hypomobility often is associated with a physiologic process, and a complete investigation to determine the etiology is warranted in all cases.

**Key Words:** Vocal fold hypomobility—Vocal fold paresis—Recurrent laryngeal nerve—Superior laryngeal nerve—Thyroid—Thyroidectomy—Vocal fold paralysis—Laryngeal nerve paresis—Laryngeal nerve paralysis—Vocal fold bowing.

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## INTRODUCTION

Vocal fold hypomobility is a common cause of vocal dysfunction that is often underdiagnosed and undertreated.<sup>2,3</sup> Hypomobility is defined as a decreased mobility of the vocal fold in its adductory, abductory, or tensor functions, as evidenced by sluggish adduction or abduction, vocal fold bowing, limited vocal fold lengthening with voluntary increase in pitch, or axial rotation of the larynx.<sup>2,3</sup> Patients with hypomobility of the vocal fold often present with complaints of vocal fatigue, decreased projection, decreased breath support, effortful phonation, hoarseness, loss of range, and odynophonia.<sup>1-5</sup> In a previous study, Heman-Ackah et al showed that in a population of singing teachers with complaints of "technical difficulties" related to vocal fatigue, trouble singing softly, and difficulty with register transition, all were found to have evidence of vocal fold hypomobility on laryngeal examination.<sup>1</sup>

Vocal fold hypomobility can have one of five main etiologies: psychogenic dysphonia, joint dysfunction, myopathy, neuropathy (central or peripheral), or neuromuscular junction dysfunction. Hyperfunction alone should result in symmetrical muscle forces in the larynx, and asymmetries in mobility should not occur if the underlying neuromuscular system is functioning properly. Psychogenic disorders typically cause symmetric abnormalities in vocal fold mobility and typically reveal normal vocal fold function during involuntary laryngeal maneuvers such as coughing.<sup>6</sup> Joint dysfunction is usually diagnosed by the observance of typical signs on physical examination, computed tomography (CT) scanning, and direct palpation of the joint during direct laryngoscopy.<sup>7-10</sup> When joint dysfunction and psychogenic abnormalities are not apparent, and the neurolaryngeal examination reveals vocal fold hypomobility, the abnormality most probably is one of neuromuscular origin. Myopathic changes generally have a characteristic appearance on magnetic resonance imaging (MRI) and CT scanning, demonstrating abnormalities in signal intensity on T1- and T2-weighted images and areas of hypodensity on CT.<sup>11</sup> Once psychogenic, joint and myopathic changes have been ruled out as possible etiologies, it then logically follows that the vocal fold hypomobility present is likely of neural or neuromuscular

junction origin, although proof of neuropathy or neuromuscular junction abnormality is obtained only with laryngeal electromyography.

Hypomobility of the vocal fold is most often caused by vocal fold paresis, with reports of paresis of the recurrent, superior, or both laryngeal nerves being present in 98% to 100% of patients with documented findings of hypomobility on physical examination.<sup>2,3</sup> Diagnosis of vocal fold hypomobility requires a high index of suspicion, and it is often unrecognized by otolaryngologists. The typical patient with vocal fold paresis will present with what appear to be vague vocal symptoms that are not supported by findings of structural abnormalities in the larynx and that accompany hyperfunctional dysphonic behaviors. Thus, the conditions of many of these patients are often misdiagnosed as muscle tension dysphonia as the primary voice disorder. A review of the literature reveals that many of these patients will have symptoms of vocal fatigue, volume disturbance, range abnormalities, vocal strain, and dysphonia.<sup>1-3</sup> Evaluation of the larynx with stroboscopy or flexible laryngoscopy alone may not reveal any structural abnormalities of the vocal folds or the larynx. All patients should, however, undergo neurolaryngeal evaluation with complete assessment of the function and mobility of the vocal folds in their normal anatomical positions, without the distortion created by pulling the tongue forward, as occurs with intraoral examination of the larynx.<sup>2,3</sup>

Neurolaryngeal evaluation of the larynx involves assessment of the abductor, adductor, and tensor functions of the larynx in isolation as well as during normal phonatory maneuvers such as talking and singing. The neurolaryngeal examination is performed best using flexible laryngoscopy under continuous light. The purpose of the neurolaryngeal evaluation is to assess the mobility of the vocal folds, to elicit subtle movement disorders, to detect paresis and hypomobility, to differentiate neuromuscular dysfunction from joint dysfunction, to diagnose hyperfunctional behaviors, and to evaluate for spasticity. Neurologic assessment of the larynx involves the careful observation of phonatory maneuvers that isolate individual muscle groups of the larynx via repetitive movements of opposing muscle groups. Such maneuvers should eliminate supraglottic hyperfunction as much as possible to allow accurate assessment of vocal fold mobility.<sup>2,3,12,13</sup>

In the study by Dursun et al, paresis of the superior laryngeal nerve, as documented by electromyography, was found to be the etiology of hypomobility in 98% of the patients who had symptoms and evidence of hypomobility in longitudinal tension and vocal fold adduction on examination.<sup>3</sup> A review of the literature using PubMed (1966–present) revealed only two studies that have attempted to define the distribution of etiologies of vocal fold paresis.<sup>2,3</sup> In the study by Dursun et al, viral neuritis was thought to be the etiology of superior laryngeal nerve paresis in 93.6% of the patients; trauma and iatrogenic injury each accounted for 3.2% of the etiologies.<sup>3</sup> Koufman et al reported idiopathic paresis to be the most common etiology (44%), with viral neuritis accounting for 24%, iatrogenic injury 20%, malignancy 6%, multiple sclerosis 4%, and post-chemotherapy peripheral neuropathy 2% of the cases.<sup>2</sup>

There have been several isolated reports in the literature of cases of vocal fold hypomobility and/or paresis caused by other etiologic factors. Vocal fold paresis can occur as a result of neuromuscular junction disorders, such as myasthenia gravis, and can be a symptom of metabolic, infectious, autoimmune, and circulatory disorders, including Lyme disease, neurosyphilis, diabetic neuropathy, ischemic neuropathy, sarcoid neuropathy, among others.<sup>14-17</sup> Vocal fold hypomobility may also occur as a result of injury to or stiffness of the cricoarytenoid or cricothyroid joints. Trauma, rheumatoid arthritis, gout, or other arthritides can cause disorders of these joints.<sup>7-9, 18-22</sup> In addition, vocal fold paresis has been shown to be a possible sequelae of thyroidectomy and a symptom of both benign and malignant thyroid disease.<sup>23-33</sup> Traditionally, vocal fold paresis and paralysis, when associated with diseases of the thyroid gland, have been assumed to occur in the presence of malignancy or as the result of iatrogenic injury during thyroidectomy. Several recent reports in the literature have also shown that paresis and paralysis of the laryngeal nerves can occur from stretch, compression, and inflammation related to benign thyroid disease in nonoperative patients. Recovery of nerve function after thyroidectomy for benign thyroid lesions has been reported to occur in patients who preoperatively had vocal fold immobility. In a series by Rowe-Jones et al, this rate of recovery was found to be as high as 89%.<sup>26</sup>

Other than the report of stroboscopy findings among singing teachers in which mobility was specifically assessed, to our knowledge, there have been no reports in the literature of the prevalence of mild vocal fold hypomobility or paresis in the general population. The purpose of this study is to define the prevalence of mild vocal fold hypomobility among patients with voice complaints and to describe the etiologic diagnoses ascribed to these disorders via a retrospective chart review.

## METHODS

A retrospective review of the charts of all patients who presented to a tertiary, referral voice center over the course of the 6-month period from July 25, 2001 to January 31, 2002 was performed. All patients had undergone a thorough history and physical examination of the head and neck, followed by a neurolaryngeal and stroboscopy examination for evaluation of voice disorders. None of the patients had a known diagnosis of vocal fold hypomobility prior to presentation. The neurolaryngeal examinations were performed using flexible laryngoscopes (Model ENF P3 and ENF P4; Olympus America Inc., Melville, NY) with video recording. The stroboscopy examinations were performed using the Kay Elemetrics 70° rigid telescopes and the Kay Elemetrics digital stroboscopic system (Model 9100 B; Kay Elemetrics Corp., Lincoln Park, NJ). Our technique for the neurolaryngeal and the stroboscopy evaluations has been described previously; all examinations were performed by the senior author (YDH) using the same protocol.<sup>12</sup> All patients with a documented hypomobility of the vocal fold(s) and whose hypomobility was not temporally related to a surgical procedure in which the nerve was known to have been injured underwent laboratory testing and imaging as part of the routine evaluation to determine the etiology of their vocal fold hypomobility. Laboratory testing included assessment of the parameters listed in Table 1. Imaging included either an initial CT scan or a MRI scan of the neck, extending from the skull base through the mediastinum to evaluate the larynx, the cricothyroid and cricoarytenoid joints, the laryngeal musculature, the thyroid

**TABLE 1.** *Laboratory Evaluation of Vocal Fold Hypomobility*

Thyroid stimulating hormone (TSH)	Fasting glucose
Antimicrosomal and thyroperoxidase (TPO) antibodies	Fasting triglycerides
T <sub>4</sub> (Total thyroxine)	Fasting cholesterol profile (total cholesterol, low-density lipoproteins [LDL], high-density lipoproteins [HDL])
Antithyroglobulin antibodies	Erythrocyte sedimentation rate (ESR)
Fluorescent treponemal antibody absorption test (FTA-abs)	Antinuclear antibodies (ANA)
Lyme Titer	Rheumatoid factor

gland, and the course of the laryngeal nerves. Ultra-sound of the thyroid and ultrasound-guided fine-needle aspiration biopsy of the thyroid were performed as dictated by findings of thyroid abnormalities on CT, **MRI**, or physical examination.

Laryngeal electromyography was reserved for patients in whom laryngeal myasthenia was suspected and in whom improvement in symptoms did not occur with voice therapy alone. Only one patient met these criteria and underwent diagnostic electromyography. Electromyography confirmed bilateral recurrent laryngeal nerve paresis and an absence of a neuromuscular junction disorder. Serial laboratory testing revealed increasing antithyropoxidase antibodies, and thyroid ultrasound confirmed the presence of Hashimoto's thyroiditis in that patient.

Vocal fold hypomobility was defined as sluggishness in vocal fold movement in adduction, abduction, or longitudinal tension. If the hypomobility observed was in both adduction and longitudinal tension, and if abductory motion and integrity of the cricothyroid and cricoarytenoid joints were found to be normal by both examination and imaging, then the vocal fold was described as being hypomobile in the distribution of the superior laryngeal nerve. If the sluggishness observed was in both adduction and abduction, and if longitudinal tension and integrity of the cricothyroid and cricoarytenoid joints were found to be normal by both examination and imaging, then the vocal fold was described as having a hypomobility in the distribution of the recurrent laryngeal nerve. If the hypomobility observed was

in adduction, abduction, and longitudinal tension and if the integrity of the cricothyroid and cricoarytenoid joints were found to be normal by both examination and imaging, then the vocal fold was described as being hypomobile in the distribution of both the recurrent and superior laryngeal nerves.

If the mobility of the cricoarytenoid joint was observed to be limited on examination, then a suspicion for cricoarytenoid joint dysfunction was raised. Joint fixation was then confirmed by CT scanning and intraoperative palpation. Posterior cricoarytenoid joint dislocation as an etiology of hypomobility was diagnosed in those cases in which there was a history of laryngeal trauma, the height of the affected vocal process was higher than that of the normal side, the joint demonstrated limited mobility, and CT scanning and/or intraoperative assessment confirmed the presence of a posterior dislocation. The diagnosis of anterior cricoarytenoid joint dislocation was given to those in which there was a history of laryngeal trauma, the height of the affected vocal process was lower than that of the normal side, the cricoarytenoid joint demonstrated limited mobility, and CT scanning and/or intraoperative assessment confirmed the presence of an anterior cricoarytenoid joint dislocation. Subluxation of the cricothyroid joint as an etiology of vocal fold hypomobility was ascribed to those cases in which there was a history of laryngeal trauma (strangulation), with evidence of an abnormal tilt of the larynx, hypomobility of vocal fold adduction and longitudinal tension, and CT scanning that confirmed the presence of subluxation of the cricothyroid joint.

The charts were reviewed and evaluated for the presence of vocal fold hypomobility, completion of the evaluation, diagnostic findings and etiologies of the hypomobility, and vocal complaints. For the purposes of this paper, those with immobile vocal folds from paralysis or joint fixation were considered separately from those with hypomobile but mobile vocal folds.

## RESULTS

During the 6-month period from July 25, 2001 to January 31, 2002, 134 new patients presented to the tertiary, referral voice center for evaluation of vocal complaints. This included 103 (77%) females and 31 (23%) males. The mean age was 47 years (range,

TABLE 2. *Vocal Complaints (n = 134)*

Vocal Complaint	Vocal Fold Hypomobility		Normal Mobility		Vocal Fold Immobility	
	(n = 61)	(%)	(n=64)	(%)	(n=9)	(%)
Raspiess	36	(59%)	36	(56%)	4	(44%)
Throat Clearing	30	(49%)	37	(58%)	2	(22%)
Vocal Fatigue	29	(48%)	18	(28%)	5	(56%)
Chronic Cough	22	(36%)	17	(27%)	4	(44%)
Heartburn	20	(33%)	12	(19%)	0	(0%)
Decreased Volume	17	(28%)	7	(11%)	4	(44%)
Decreased Range*	17	(28%)*	4	(6%)*	3	(33%)
Globus	16	(26%)	10	(16%)	0	(0%)
Chronic Phlegm	13	(21%)	17	(27%)	3	(33%)
Decreased Breath Support	12	(20%)	10	(16%)	3	(33%)
Sore/Painful Throat	7	(11%)	5	(8%)	0	(0%)
Voice Breaks	7	(11%)	4	(6%)	0	(0%)
Laryngospasm	6	(10%)	8	(13%)	0	(0%)
Voice Strain	6	(10%)	8	(13%)	1	(11%)
Odynophonia	4	(7%)	4	(6%)	0	(0%)
Breathiness	3	(5%)	6	(9%)	0	(0%)

\*Statistically significant at  $p < 0.05$ , chi-square analysis of hypomobility vs. normal mobility. All other complaints,  $p > 0.05$ , chi-square analysis (hypomobility vs. normal mobility and hypomobility vs. immobility).

Other Complaints (prevalence  $< 5\%$ ):

Throat burning, neck/throat tightness, throat irritation, vocal tremor, dry mouth/throat, and otalgia.

17–79 years). Among these patients, 61 (46%) were found to have vocal fold hypomobility as a contributing factor to their voice complaint. Sixty-four (48%) patients had normal vocal fold mobility, and 9 (7%) patients were found to have at least one immobile vocal fold secondary to either joint dysfunction or laryngeal nerve paralysis. Of the 61 patients with hypomobile vocal folds, 49 (80%) completed sufficient work-up to establish an etiology for their hypomobility.

The vocal complaints of these 134 patients are displayed in Table 2. Using chi-square analysis, decreased vocal range was the only vocal complaint that was more common, at a level of statistical significance, in patients with vocal fold hypomobility than in those with normal vocal fold mobility (chi-squared = 9.372,  $p < 0.05$ ). There was no statistically significant difference in vocal complaints in those with vocal fold hypomobility and in those with immobile vocal folds using chi-square analysis ( $p > 0.05$ ). A list of comorbid laryngeal findings in patients with normal and hypomobile vocal folds is presented in Table 3. There was no significant difference in the presence or absence of any of the comorbid pathologies between those with normally

mobile and those with hypomobile vocal folds using chi-square analysis ( $p > 0.05$ ). Vocal fold mass was defined as the presence of a vocal fold polyp, nodule, cyst, pseudocyst, hyperkeratosis, or leukoplakia on the musculomembranous portion of the vocal fold. Within each of these individual categories of lesions, there was no significant difference between those with normal vocal fold mobility and those with hypomobile vocal folds using chi-square analysis ( $p > 0.05$ ).

The patterns of distribution of vocal fold hypomobility in those that completed their evaluations are presented in Table 4. None of the patients with apparent neuromuscular etiologies had joint dysfunction on either physical examination or imaging. Of those with hypomobility attributed to neuromuscular abnormalities, only two had evidence of myopathy on both examination and high-resolution MRI scanning. The patient with unilateral myopathy had edema of the thyroarytenoid muscle after a traumatic intubation, and the patient with bilateral myopathy had laryngeal sarcoidosis. Those patients designated as having hypomobility in the distribution of the superior and/or recurrent laryngeal nerves had corresponding mobility patterns as described in the

**TABLE 3.** Comorbid Findings on Stroboscovideolaryngoscopy (*n* = 134)

Stroboscopic Finding	Vocal Fold Hypomobility		Normal Vocal Fold Mobility		Vocal Fold Immobility	
	( <i>n</i> = 61)	(%)	( <i>n</i> = 64)	(%)	( <i>n</i> = 9)	(%)
Muscle Tension Dysphonia	61	(100%)	64	(100%)	9	(100%)
Reflux Laryngitis	46	(75%)	43	(67%)	5	(56%)
Vocal Fold Mass (polyp, nodule or cyst)	24	(39%)	33	(52%)	1	(11%)
Vocal Fold Varices	13	(21%)	8	(13%)	0	(0%)
Vocal Fold Edema	8	(13%)	12	(19%)	0	(0%)
Pseudosulcus Vocalis	6	(10%)	7	(11%)	1	(11%)
Vocal Fold Tremor	2	(3%)	0	(0%)	0	(0%)
Vocal Fold Scar	1	(2%)	6	(9%)	0	(0%)
Vocal Fold Granuloma	1	(2%)	3	(5%)	0	(0%)
Congenital Anomaly	1	(2%)	1	(2%)	0	(0%)
Cogwheeling	1	(2%)	0	(0%)	0	(0%)
Sarcoidosis	1	(2%)	0	(0%)	0	(0%)
Glottic Ridges	0	(0%)	3	(5%)	0	(0%)
Posterior Glottic Stenosis	0	(0%)	2	(3%)	0	(0%)
Dysphonia Plica Ventricularis	0	(0%)	2	(3%)	0	(0%)
Dysdiadokinesis	0	(0%)	1	(2%)	0	(0%)
Adductor spasms	0	(0%)	1	(2%)	0	(0%)
Vocal Process Ulceration	0	(0%)	1	(2%)	0	(0%)

Chi-square analysis,  $p > 0.05$  for all.

Methods section, normal laryngeal architecture on imaging, and either normal or slightly atrophic laryngeal muscles on imaging, without CT or MRI evidence of myopathy.

The presumed etiologies of the vocal fold hypomobilities observed are shown in Table 5. Thyroid disease was ascribed as the etiology of the vocal fold hypomobility when the disease in the thyroid resulted in expansion of thyroid gland, relative to normal, in the region of the nerve(s) that appeared to be involved in hypomobility on examination. Viral neuritis was ascribed as the apparent etiology of vocal fold hypomobility when the patient gave a history of persistent dysphonia that began immediately after an upper respiratory tract infection and when no other findings were present on imaging or laboratory testing. Nerve tumors and central nervous system lesions were felt to be etiologies of the vocal fold hypomobility when the lesion appeared on the proximal vagus, recurrent laryngeal, or superior laryngeal nerves; in the brainstem, near the region of the motor nucleus of the vagus or the nucleus ambiguus; or in the region of the motor cortex. Iatrogenic etiologies were ascribed as the etiology of the paresis when the operating surgeon reported

stretch injury, significant manipulation, or severing of the involved nerve. A diagnosis of idiopathic vocal fold hypomobility was made when no identifying lesion could be found with history, examination, imaging, or laboratory testing. The patient with traumatic nerve injury, noniatrogenic, reported onset of her dysphonia after having been strangled by her husband several months prior to presentation. She had asymmetrical dilation of the ipsilateral internal jugular vein without evidence of aneurysm or laryngeal framework injury on CT scan, MRI, and magnetic resonance angiography (MRA).

Among the patients with thyroid disease, 8 out of 21 (38%) had hypomobility in the distribution of the superior laryngeal nerve, 11 out of 21 (52%) had hypomobility in the distribution of the both the superior and recurrent laryngeal nerves, and 2 out of 21 (10%) had hypomobility in the distribution of the recurrent laryngeal nerve only. Among the patients with etiologies other than thyroid disease, 11 out of 23 (48%) had hypomobility in the distribution of the superior laryngeal nerve, 9 out of 23 (39%) had hypomobility in the distribution of both the superior and recurrent laryngeal nerves, and 3 out of 23 (13%) had hypomobility in the distribution of

**TABLE 4.** *Pattern of Distribution of Vocal Fold Hypomobility (n = 49)*

Diagnosis	Unilateral	Bilatera
<i>Neuromuscular</i>		
Superior Laryngeal Nerve Distribution	19(39%)	
Recurrent Laryngeal Nerve Distribution	4(8%)	1 (2%)
Both Recurrent and Superior Laryngeal Nerve Distribution	15(31%)	3 (6%)
>3 Nerve/muscle groups involved		2 (4%)
Myopathy	1(2%)	1 (2%)
<i>Subtotal</i>	39(80%)	7 (14%)
<i>Joint Dysfunction</i>		
Joint Fixation	1(2%)	
Cricoarytenoid Joint Dislocation	1(2%)	
Cricothyroid Joint Subluxation	1(2%)	
<i>Subtotal</i>	3(6%)	
<b>Total</b>	<b>42(86%)</b>	<b>7 (14%)</b>

the recurrent laryngeal nerve only. Using chi-square analysis, there was no significant difference between these two groups ( $p > 0.05$ ).

Among the patients who underwent thyroidectomy, several interesting findings were noted. In no case was the side of hypomobility contralateral to the side of thyroid pathology. In 9 patients, bilateral thyroid disease was associated with only unilateral vocal fold hypomobility. In the other 12 patients, the side of vocal fold hypomobility corresponded to the side of thyroid disease. There were no thyroid malignancies in this series.

Seven patients with thyroid disease underwent hemithyroidectomy or subtotal thyroidectomy for compressive symptoms, abscess, or suspicion for carcinoma by another head and neck surgeon after their initial laryngeal evaluation. In all cases, dysphonia was one of the presenting complaints of the thyroid disease that prompted the referral to the voice center. In none of the cases was dysphonia the primary reason for performing the thyroidectomy. The evaluation before the thyroid procedure demonstrated hypomobility in the distribution of both the recurrent and superior laryngeal nerves on the involved side in five of these patients, hypomobility in the distribution of the recurrent laryngeal nerve only in one patient, and hypomobility in the distribution of the superior laryngeal nerve only in

**TABLE 5.** *Etiologies of Vocal Fold Hypomobility*

Etiology	Number of Patients (n = 49)	% Total
<i>Recurrent and/or Superior Laryngeal Nerve Paresis</i>	44	90%
<b>Thyroid</b>	<b>21</b>	<b>43%</b>
<i>Goiter</i>	11	22%
Multinodular	5	10%
Smooth	3	6%
Single Nodule	3	6%
<i>Single Nodule/Cyst</i>	5	10%
<i>Thyroiditis</i>	5	10%
Hashimoto's	3	6%
Suppurative	1	2%
Subacute	1	2%
<b>Idiopathic</b>	<b>8</b>	<b>16%</b>
<b>Viral Neuritis</b>	<b>5</b>	<b>10%</b>
<b>Central Nervous System</b>	<b>4</b>	<b>8%</b>
Brainstem CVA	1	2%
Cerebral CVA	1	2%
Spinocerebellar Atrophy	1	2%
Parkinson's Disease	1	2%
<b>Laryngeal/Vagal Nerve Tumor</b>	<b>3</b>	<b>6%</b>
Schwannoma	1	2%
Neuroma	1	2%
Neurofibroma	1	2%
<b>Iatrogenic</b>	<b>2</b>	<b>4%</b>
Post Thyroidectomy	1	2%
Post Vertebral Artery Aneurysm Repair	1	2%
<b>Trauma (strangulation)</b>	<b>1</b>	<b>2%</b>
<i>Myopathy</i>	2	4%
<b>Laryngeal Sarcoid</b>	<b>1</b>	<b>2%</b>
<b>Edema</b>	<b>1</b>	<b>2%</b>
<i>Joint Dysfunction</i>	3	6%
<b>CA fixation</b>	<b>1</b>	<b>2%</b>
<b>CA Dislocation</b>	<b>1</b>	<b>2%</b>
<b>CT Joint Subluxation</b>	<b>1</b>	<b>2%</b>

another patient. Six of these patients had intraoperative and pathologic findings consistent with inflammation around the laryngeal nerves or significant mass effect in immediate proximity to the recurrent and superior laryngeal nerves. Of those with both recurrent and superior laryngeal nerve symptoms, two had large multinodular goiters producing airway symptoms of compression, one of which had displaced the carotid artery and was found to be exerting pressure on the recurrent laryngeal nerve; one patient had a multinodular goiter with

hemorrhage into a 3-cm nodule that lay on the involved nerves, resulting in stretch; one patient had suppurative thyroiditis with abscess involving the recurrent laryngeal nerve and inflammation of the gland on that side; and one patient had diffuse chronic nodular inflammation of a multinodular goiter. The patient with signs consistent with recurrent laryngeal nerve injury only had a large, compressive multinodular goiter with scarring around the recurrent laryngeal nerve. The one patient who had findings of hypomobility in the distribution of the superior laryngeal nerve only did not have convincing intraoperative findings of nerve compression, inflammation, or stretch, but was found to have a single 1.5-cm nodule, in the mid-portion of a multinodular thyroid gland that was moderately enlarged. All but one of these patients (the patient with diffuse chronic nodular inflammation and hypomobility in the distribution of both the recurrent and superior laryngeal nerves) noted significant improvement in their voices and demonstrated improvement in their vocal fold hypomobilities in the 3 months after their thyroid procedures.

## DISCUSSION

The results of this study show that vocal fold hypomobility is common among those with voice complaints, having a prevalence of 46% in our study population. It should be noted that because the population studied consisted of patients presenting to a tertiary voice referral center, it is likely that this prevalence of vocal fold hypomobility may be skewed and slightly higher than that among patients presenting to general otolaryngologists with voice complaints. A diagnosis of vocal fold hypomobility typically requires a more thorough and detailed neurolaryngeal examination than is usually performed by general otolaryngologists. In this study, vocal fold hypomobility had never been previously diagnosed by any of the referring physicians. Raspiness was the most common vocal complaint and had a similar frequency of presentation among patients with normal vocal fold mobility and those with hypomobile vocal folds. The vocal complaint that was most significantly associated with vocal fold hypomobility was that of decreased range, occurring in 28% of those with hypomobility and only 6% of those

with normal vocal fold mobility. Because pitch is associated with the ability to tense and stretch the vocal folds, which requires the actions of both the thyroarytenoid and the cricothyroid muscles, it reasonably follows that demonstrable abnormalities in the abilities of these muscles to function is associated with perceived difficulties in range and pitch control.

Analysis of the comorbid vocal pathologies revealed a similar prevalence of reflux laryngitis, vocal fold mass, varices, edema, scar, and other vocal fold lesions among those with and without vocal fold hypomobility. The presence of muscle tension dysphonia was ubiquitous among this group of patients with vocal complaints and may account for an equivalent prevalence of phonotrauma, leading to the formation of vocal fold lesions. It has been postulated that muscle tension dysphonia develops in those with vocal fold paresis as a form of compensation for incomplete glottic closure. It was observed in the study by Koufman et al that the presence of a hypokinetic glottis may lead to a hyperkinetic supraglottic compensation, which in turn may lead to organic pathology, such as the formation of lesions on the vibratory margin of the vocal fold.<sup>2</sup> The prevalence of pseudocyst and of other vocal fold masses in the present study was found to be 39% among those with hypomobility and not significantly different from those with normal vocal fold mobility, a finding that is consistent with the observations of others.<sup>1-3</sup>

Laryngeal electromyography is occasionally used to confirm that paresis exists when findings on examination raise such a suspicion.<sup>2,3,34</sup> Laryngeal electromyography is helpful in that it confirms the presence of neuropathy, is able to identify neuromuscular junction abnormalities and myopathies, and is able to identify ongoing degeneration or regeneration when it is performed correctly.<sup>34</sup> The ability to obtain reliable and accurate electromyography results in the larynx requires a significant degree of experience and expertise, specifically in the interpretation of laryngeal data and is heavily dependent on accurate and consistent needle placement, which limits its usefulness and availability in many centers. However, electromyography does not explain the series of events that caused the nerve injury; that is, it does not address the etiology of the paresis. Often, further imaging and laboratory testing is needed to discover the cause of the vocal fold paresis.

Dursun et al showed that with a thorough neuro-laryngeal examination in the hands of an experienced laryngologist, the diagnosis of superior laryngeal nerve paresis is confirmed in 98% of the patients with characteristic findings on neurolaryngeal examination.<sup>3</sup> During the current study period, laryngeal electromyography was reserved for those patients in whom laryngeal myasthenia was suspected and in whom improvement of symptoms did not occur with voice therapy. Only one patient met these criteria. Laryngeal electromyography confirmed the bilateral recurrent laryngeal nerve paresis that had been observed on neurolaryngeal examination and excluded neuromuscular junction dysfunction as the underlying etiology.

In this study, those patients who had vocal fold hypomobility and who had no evidence of joint dysfunction, myopathy, or psychogenic disorder were classified as having vocal fold hypomobility in the distribution of the recurrent and/or superior laryngeal nerves, depending on the pattern of distribution of their hypomobilities. A diagnosis of paresis was not given to any of the patients; however, etiologic findings consistent with nerve injury were sought with imaging, laboratory, operative, and pathologic evaluations. Among patients with vocal complaints who had findings on physical examination of vocal fold hypomobility, a physiologic cause could be identified in 84%. The most common cause of vocal fold hypomobility appeared to be neuropathy in the distribution of the superior and recurrent laryngeal nerves, with hypomobility in the distribution of the superior laryngeal nerve being more frequent than that in the distribution of the recurrent laryngeal nerve (80% vs. 51%, respectively).

Among those with apparent neuropathy, benign thyroid disease in the region of the laryngeal nerves appeared to be the etiologic cause, occurring in association with and without any other identifiable pathology in 43% of the cases of vocal fold hypomobility. In 1993, Rowe-Jones et al reviewed the charts of 2321 patients who had undergone thyroidectomy for benign thyroid disease.<sup>26</sup> All of the patients had had preoperative laryngoscopy. Twenty-two of these patients were found to have had a preoperative vocal fold palsy, and 89% of these had return of function of their vocal folds after thyroidectomy for benign goiter, as confirmed histologically.

Other case reports have demonstrated reversible recurrent laryngeal nerve paresis as a symptom of small and multinodular goiters, which demonstrates that benign thyroid disease can involve the laryngeal nerves and that even minimally enlarged or nodular thyroid glands can result in significant laryngeal nerve dysfunction.<sup>27,28,32,33</sup> Nodular and cystic lesions of the thyroid occur in response to inflammation or goiterous growth, and stretching or inflammation of the laryngeal nerves in the vicinity of such lesions can result in a localized neuropathy and vocal fold paresis. In the present study, when thyroidectomy was performed for benign goiter or nodular or cystic masses in the thyroid, perineural inflammation or stretch was found intraoperatively, thus confirming the causal relationship between the benign thyroid disease and the observed laryngeal neuropathy. In addition, in all cases in which vocal fold hypomobility was found on examination and benign thyroid disease was deemed the etiologic factor, the thyroid disease always occurred on the same side as the vocal fold hypomobility or was bilateral.

A limitation of this study is that laryngeal electromyography was not performed in all cases to confirm the presence of neuropathy. Nonetheless, the results are convincing that a thorough evaluation and search for the etiology of vocal fold hypomobility should be performed as an adjunct to the physical examination. Even if one excludes the cases of benign thyroid disease that did not have intraoperative or laryngeal electromyographic confirmation of nerve involvement, the other 27 cases in which a lesion was found involving the neural pathway, the joint, or the muscle indicates that in at least 55% of the cases of vocal fold hypomobility, an etiology can be found with thorough evaluation. Further investigation into the role of benign thyroid disease as an etiologic factor in vocal fold hypomobility is warranted.

## CONCLUSIONS

The results of this study show that vocal fold hypomobility is a common finding among patients with vocal complaints, and a thorough neurolaryngeal examination is warranted in all cases. In those in whom vocal fold hypomobility is found on neurolaryngeal examination, a complete evaluation that allows the diagnosis of neuropathic, myopathic, arthritic, metabolic, infectious, autoimmune,

and structural etiologies of vocal fold hypomobility is warranted. In most patients, an etiology for the vocal fold hypomobility can be found. In some cases, hypomobility is at least partially reversible with treatment of the underlying disorder and may preclude the performance of medialization procedures that may later fail if the underlying etiologic process progresses. Treatment of vocal fold hypomobility should focus on alleviating the underlying cause as well as on rehabilitating the voice disorder.

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