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Laryngeal Manifestations of Cranial Nerve IX/X Compression at the Brainstem

Robert J. Taylor, MD; Stephen R. Lowe, MD; Nic Ellis, BS; Evan Abdullah, BS; Sunil Patel, MD; Lucinda A. Halstead, MD

Objective: We report an association between lower cranial nerve (CN IX/X) vascular compression at the brainstem with laryngeal symptoms utilizing a stepwise algorithm that systematically evaluates and eliminates all other common etiologies. Our experiences with retromastoid craniectomy with lower cranial nerve (LCN) decompression versus non-neurosurgical treatments are detailed.

Study Design: Retrospective chart review at a tertiary care academic medical center with follow-up telephone survey.

Methods: Baseline demographics, clinical characteristics, quality-of-life surveys, and treatment outcomes were recorded for patients with laryngeal symptoms associated with LCN compression at the brainstem.

Results: Forty-nine patients demonstrated LCN compression at the brainstem on imaging and presented with chief complaints of dysphonia (25 of 49, 51%), chronic cough (19 of 49, 39%), dysphoric breathing (3 of 49, 6%), and dysphagia (2 of 49, 4%). Poor initial scores were noted for Voice-Related Quality of Life (V-RQOL), Reflux Symptom Index, and Glottal Closure Index. Twenty-four patients underwent LCN decompression, of which 21 of 24 (88%) reported partial, near-complete, or complete improvement. Major perioperative complications occurred in four of 24 patients (17%). Patients who had undergone decompression were more likely to obtain complete/near-complete symptom resolution (10 of 24 patients, 42%) compared to those undergoing conservative treatments (2 of 25 patients, 8%) \((P = 0.02)\). V-RQOL scores improved more in surgical patients \([mean \ change \ score, 33.0 \ (SD, \ 31.2)]\) than nonsurgical patients \([mean \ change \ score \ 9.6, \ SD \ 20.9] \ (P = 0.03)\) \(mean \ follow-up \ 3.0 \ years, \ SD \ 2.0)\).

Conclusion: Lower cranial nerve compression at the brainstem should be considered when all other etiologies are excluded. Retromastoid craniectomy with LCN decompression demonstrates an acceptable safety profile.

Key Words: Vascular compression, lower cranial nerves, microvascular decompression, cough, vocal fold paralysis, post-viral vagal neuropathy.

Level of Evidence: 4

INTRODUCTION

Cough is a physiologic reflex that protects the airway from inhaled or aspirated material and often serves as a presenting clinical symptom initiating further workup. A chronic cough lasts for more than 8 weeks, is refractory to treatment, and is generally thought to have a different underlying pathophysiology than acute cough. Etiologies of chronic cough can be esophageal, rhinologic, pulmonary, neurologic, neoplastic, or systemic in origin (Table I), with 86% of cases due to postnasal drainage, asthma, or gastroesophageal reflux disease.¹⁻³ Clinical evaluation of a chronic cough requires a full history and physical exam. The otolaryngologist has the advantage of performing in-office laryngeal evaluation of these patients, which in a small percentage may reveal unilateral laryngeal findings necessitating further workup. Because a lesion that incites a chronic cough can occur anywhere along the course of the vagus nerve—cranial nerve X (CN X)—further evaluation with chest and neck computed tomography has been recommended.⁴ If these scans are normal and there are no other focal signs/symptoms, the prevailing practice among otolaryngologists is to attribute the vocal fold weakness and chronic cough to postviral vagal neuropathy (PVVN) even if there is no clear link to a viral illness.⁵

PVVN can profoundly impact the psychosocial functioning of patients and generally responds poorly to available treatments.⁶ Such has been noted in the practice of the senior author (L.H.) even after a thorough systematic approach to diagnosis has been performed (Fig. 1). However, prior to assigning the patient a diagnosis of PVVN or an idiopathic etiology of CN X weakness, the senior author (L.H.)—in conjunction with a multidisciplinary team at our institution—has investigated other possible neurologic etiologies with further cranial imaging. This has revealed many cases of lower cranial nerve...
—CN IX and CN X—compression at the brainstem ipsilateral to the side of a unilateral laryngeal finding. This is consistent with previous reports that associate chronic cough, unilateral vocal fold hemiparesis, and dysphoric breathing with compression of CN X by tumors and vascular compression in the neck, chest, along with anatomic shifts secondary to fluctuations in cerebral and cerebrospinal fluid (CSF) volume. Additionally, vascular compression of CN X at the brainstem has been seen in intractable hypertension and hiccups.

When considering treatment options for LCN compression at the brainstem, it is germane to consider other forms of cranial nerve compression at the brainstem. Vascular compression causing trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia are well-described phenomena. These diseases respond favorably to neurosurgical decompression, which provides complete or near-complete resolution of symptoms in the majority of cases with an acceptable risk profile. Symptom improvement for trigeminal neuralgia is often long-lived, persisting in approximately 80% of patients at 5 years and 70% of patients at 10 years. Even greater success rates, up to 85%, for neurosurgical decompressions for hemifacial spasm at 10 years have been observed. Similarly, vascular compression of CN X causing intractable hiccups has been described in several case reports, with neurosurgical decompression demonstrating favorable results.

This has led to the hypothesis that neurosurgical decompression may benefit those patients with symptomatic compression of LCN at the brainstem associated with a laryngeal symptom and ipsilateral laryngeal finding. To test the validity of this hypothesis, the objective of the present study is to provide the first characterization of laryngeal manifestations of LCN compression at the brainstem and to evaluate the efficacy and safety of neurosurgical decompression versus non-neurosurgical treatments.

**MATERIALS AND METHODS**

Institutional review board approval was obtained for this retrospective chart review and follow-up telephone survey. Charts were reviewed for all patients presenting to our tertiary care institution and meeting the following criteria: (1) history of chronic cough; (2) history of unilateral vocal fold paresis; (3) absence of other causes for unilateral vocal fold paresis; (4) presence of anatomic shifts secondary to fluctuations in cerebral and CSF volume.

Table I. Common Etiologies of Chronic Cough.

<table>
<thead>
<tr>
<th>System</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophageal</td>
<td>Gastroesophageal reflux, Zenker's diverticulum, Laryngopharyngeal reflux</td>
</tr>
<tr>
<td>Rhinologic</td>
<td>Rhinitis (allergic and nonallergic), Chronic rhinosinusitis</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Asthma (cough variant), Irritant (smoking/environmental), Bronchiolitis</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Cough hypersensitivity syndrome, Traumatic vagal injury, Behavioral</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Nasal, Laryngeal Bronchogenic Gastric, Skull base, cervical, and thoracic</td>
</tr>
<tr>
<td>Systemic</td>
<td>Congestive heart failure, ACEI therapy, Cystic fibrosis, Sarcoidosis, ANCA</td>
</tr>
</tbody>
</table>

*Nonallergic rhinitis etiologies may be vasomotor, infectious, or irritant in nature.
†Via obstructive sinusitis.
‡Via gastric outlet obstruction related GERD and LPR.
§Via vagal involvement or irritation.
ACEI = Angiotensin converting enzyme inhibitor; ANCA = antineutrophil-cytoplasmic antibody; GERD = gastroesophageal reflux disease; LPR = Laryngopharyngeal reflux.

![Fig. 1. Diagnostic algorithm for chronic cough, unilateral vocal fold paresis, vocal fold spasm, and dysphoric breathing. CN = cranial nerve; CT = computed tomography; GERD = gastroesophageal reflux disease; IEM = ineffective esophageal motility; MIIPH = multichannel impedance pH probe; PFT = pulmonary function test; PVD = paroxysmal vocal fold dysfunction; RAST = radioallergosorbent; SPT = skin prick testing. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

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academic medical center between January 2005 and February 2017 with laryngeal symptoms associated with a unilateral laryngeal exam finding whose underlying pathology remained unknown after extensive workup of the more common etiologies of cough, paroxysmal vocal fold dysfunction, laryngospasm, and dysphoric breathing. Applicable unilateral exam findings suggestive of laryngeal motor neuropathy included 1) vocal fold paralysis; 2) signs of vocal fold paresis including deficits of vocal fold range of motion, vibratory asymmetries on stroboscopy, unilateral atrophy or hypotonia, and abnormalities of arytenoid motion; and 3) vocal spasm where paresis was suspected but obscured (Fig. 1). Once the etiologies for these laryngeal findings were excluded in the neck and chest, the patients then underwent a high-resolution magnetic resonance imaging (MRI) of the cranial nerve roots to evaluate for LCN compression at the brainstem. The imaging protocol involved a multiplanar multisequence MRI brain with and without intravenous contrast, including volumetric high-resolution heavily T2-weighted sequences and limited post-contrast three-dimensional time-of-flight magnetic resonance angiography. All MRIs were initially reviewed by an attending neuroradiologist and re-reviewed by the same attending neurosurgeon (S.P.) (Fig. 2). If LCN compression at the brainstem was identified ipsilateral to the side of the laryngeal exam finding, patients were referred to our neurosurgery team to discuss the risks and benefits of neurosurgical decompression. Those patients who declined neurosurgical decompression were generally treated with voice therapy and trials of amitriptyline, pregabalin, gabapentin, or vocal fold medialization at the discretion of the senior author (L.H.). The senior author (L.H.) routinely recommended that all neurological and non-neurosurgical patients undergo evaluation and treatment by a certified speech and language pathologist as a first-line therapy if this has not already been performed. A multidimensional treatment program is used at our institution based on the patients’ complaints. This includes vocal hygiene training, cough-suppression strategies, voice therapy, clinical/radiologic swallowing evaluation/treatment, and psychoeducational counseling where appropriate.

Patients who elected LCN decompression underwent a retrosigmoid craniectomy performed by the same attending neurosurgeon (S.P.) (Fig. 3). This involved general anesthesia using endotracheal intubation and vocal fold electromyography (EMG). Patients were positioned in the lateral decubitus position with the head fixed in a head holder. Through a 2-cm retrosigmoid craniectomy, the dura was opened, and cerebellum relaxed with CSF aspiration from the foramen magnum. Microdissection techniques were then used to identify the CN XI and lower cranial nerves, taking care not to place too much lateral traction and stretch on the CN VII/VIII complex. CN IX and CN X were identified and examined from the jugular foramen to the nerve root entry zone in the retro-olivary sulcus and foramen of Luschka. Subsequent surgical technique was dictated by type of pathology. Notably, nerve rootlet sectioning was performed if there was a compressive element that could not be safely dissected away from the rootlet in question and if the same rootlet demonstrated high levels of activity on intraoperative neurostimulation, suggesting that the rootlet was potentially partially responsible for the patient’s symptoms.

Fig. 2. Magnetic resonance imaging demonstrating various sources of lower cranial nerve compression at the brainstem. (A) Note the compression on the IX/X complex root entry zone by an ectatic loop of the anterior inferior cerebellar artery (arrow). (B) Note the close proximity of the right posterior inferior cerebellar artery to the cranial nerve IX/X nerve complex (arrow). (C) Note compression of the IX/X complex by a large, ectatic vertebral artery (arrow).

Fig. 3. Diagrammatic representation of neurosurgical lower cranial nerve decompression. Teflon separates the PICA from CN X. AICA = anterior inferior cerebellar artery; PICA = posterior inferior cerebellar artery; SPV = superficial petrosal vein; VA = vertebral artery.
Study participants rated their symptoms using validated quality-of-life instruments routinely administered at their clinic visits. Instruments included the Voice-Related Quality-of-Life Index (V-RQOL), the Reflux Symptom Index (RSI), and the Glottal Closure Index (GCI). Among the neurosurgical decompression cohort, all pertinent operative details and postoperative complications were recorded.

Statistical analysis was performed using GraphPad Prism 7 (GraphPad Software Inc., La Jolla, CA). Descriptive statistics were computed for age, gender, race, symptom duration, V-RQOL/RSI/GCI scores, and follow-up duration. Normality was assessed with D’Agostino-Pearson omnibus normality testing. Means and standard deviations (SDs) were generated for all normally distributed continuous variables, whereas medians and interquartile ranges were calculated for all nonparametric continuous variables. Continuous demographics and clinical characteristics were compared with a Student t test or Mann Whitney U test for parametric and nonparametric data, respectively. Nominal demographics were compared using chi-square or Fisher’s exact test, as appropriate. For those patients with initial and posttreatment V-RQOL/RSI/GCI scores, the change in these metrics was compared between neurosurgical versus non-neurosurgical cohorts using unpaired two sample t testing and Mann-Whitney U testing for parametric and nonparametric data, respectively. Correlations between age/duration of symptoms and quality-of-life scores were performed with Pearson and Spearman correlation coefficients for parametric and nonparametric data, respectively. All comparisons were planned, and thus no statistical correction for multiple comparisons was performed.

RESULTS

Characteristics of Patients with Symptomatic LCN Compression

MRIs were performed on 149 patients with laryngeal symptoms and signs whose diagnosis remained undetermined after comprehensive workup. Lower cranial nerve compression at the brainstem was found in 49 of 149 (33%). Demographics included 33 of 49 (67%) female, 44 of 49 (90%) Caucasian, four of 49 (8%) African American, and one (2%) Hispanic. Chief complaints included dysphonia in 25 of 49 (51%), chronic cough in 19 of 49 (39%), dysphoric breathing in three of 49 (6%), and dysphagia in two of 49 (4%). Mean (SD) V-RQOL, RSI, and GCI scores were found to be 57.5 (26.4), 25.7 (10.3), and 11.8 (5.8), respectively (n = 29). Significant correlations between greater age and duration of symptoms and quality-of-life scores were performed with Pearson and Spearman correlation coefficients for parametric and nonparametric data, respectively. All comparisons were planned, and thus no statistical correction for multiple comparisons was performed.

Comparing the neurosurgical and non-neurosurgical groups, there was no significant difference in their age at presentation, sex, race, symptom duration, chief complaint, or initial quality-of-life scores (Table II).

Operative Characteristics and Postoperative Complications of Neurosurgical Decompression Cohort

Perioperative data was available for all (n = 24) patients who elected neurosurgical LCN decompression. In 15 of 24 (63%) of the patients who underwent decompression, vascular compression by either the posterior inferior cerebellar or the vertebral artery was identified and relieved by placing a piece of Teflon between the offending vessel and the lower cranial nerve complex (Fig. 3). In seven of 24 patients (29%), the choroid plexus was found to be adherent to and compressing the lower cranial nerve complex, and this was dissected away with microsurgical technique and then cauterized with bipolar electrocautery. Three of these patients with compression by the choroid plexus were also noted to have a cyst within the choroid plexus that was resected with microsurgical technique. One patient (4%) had compression from an arachnoid cyst that was fenestrated, and one patient (4%) was noted to have cerebellar compression of the lower cranial nerves that was treated by resection of the offending portion of the cerebellum. Three (13%) patients underwent sectioning of atrophic lower cranial nerve rootlets.
We observed major operative complications in four of 24 (17%) patients. One patient who had been cleared by our institution’s preoperative anesthesia clinic and had no known coronary history developed a myocardial infarction on postoperative day 1 requiring subsequent coronary artery bypass grafting and postprocedure heparinization. Subsequent intracranial hemorrhage at the patient’s craniectomy site caused transient hydrocephalus required temporary CSF diversion via a ventriculostomy. This was eventually weaned, and the patient was then discharged without further complication and with no neurological deficit except for new-onset left true vocal fold paralysis. Preoperatively, the patient’s vocal fold was paretic. Initially after decompression, the patient’s chronic cough had resolved; however, after the postoperative complications the patient’s voice worsened and the chronic cough returned. Three patients (13%) developed postoperative CSF leaks. Two patients with CSF rhinorrhea required placement of a lumbar drain, which was weaned, and they were subsequently discharged without further complication. The final patient with CSF rhinorrhea developed a lumbar epidural abscess after placement of a lumbar drain and required a laminectomy for abscess evacuation. This patient later was discharged home without further complication and with no neurologic deficit. Of note, one patient whose chief complaint was chronic cough and who initially recovered well after the operation developed recurrence of symptoms and underwent a reoperation 3 years later. A large amount of scarring was discovered at the prior surgical site around the Teflon, which was removed without complication, and the patient recovered without further adverse event. This patient’s chronic cough was resolved at the most recent follow-up visit.

Within-Cohort and Comparative Outcomes of Standard Versus Neurosurgical Groups

Both standard non-neurosurgical and neurosurgical treatments were associated with improvements in symptoms, with greater improvement among the neurosurgical cohort (Fig. 4). Standard non-neurosurgical treatments led to significant improvements when comparing most recent to initial RSI with a mean (95% CI) RSI change score of $-6.8$ ($-11.7$ to $-1.8$, $P = 0.01$). V-RQOL and GCI trended toward statistically significant improved changed scores of $9.6$ ($-1.1$ to $20$, $P = 0.08$) and $-3.5$ ($-7.4$ to $0.4$, $P = 0.08$) points, respectively. Neurosurgical decompression led to improvements in all quality-of-life metrics. V-RQOL, RSI, and GCI improved a mean (95% CI) of $31.7$ ($6.4$ to $56.9$, $P = 0.02$), $-12.9$ ($-23.5$ to $-2.3$, $P = 0.02$), and $-7.9$ ($-14.0$ to $-2.1$, $P = 0.01$). When comparing the neurosurgical decompression change scores to those of the non-neurosurgical cohort, there was a statistically significant greater improvement in V-RQOL among the neurosurgical cohort with a mean difference (95% CI) of $23.4$ ($2.7$–$44.4$, $P = 0.02$). RSI and GCI trended toward but did not meet statistical significance for greater improvements through neurosurgical treatment than standard care with mean differences of (95% CI) of $-6.1$ ($-16.0$ to $3.5$, $P = 0.20$) and $-4.4$ ($-10.9$ to $1.9$, $P = 0.17$). Similar findings supporting a greater degree of improvement among the neurosurgical cohort compared to the non-neurosurgical cohort were observed when comparing the distributions of patients reporting near-complete or complete resolution of symptoms: 10 of 24 (42%) among the neurosurgical cohort versus two of 25 (8%) for the non-neurosurgical cohort (RR 5.2, 95% CI RR 1.5–19.9, $P = 0.008$). Additionally, there was a greater propensity for non-neurosurgical patients to have no significant improvement of their symptoms or worsening of their symptoms over time (15 of 25, 60%) compared to neurological patients (3 of 24, 13%) (RR 4.8, 95% CI RR 1.8–14.3, $P = 0.001$) with a mean follow-up of 3.0 years (SD 2.0 years). Neither cohort demonstrated significant correlations between age or duration of symptoms and degree of quality-of-life improvement with treatment. None (0 of 25) of the patients in the non-neurosurgical cohort demonstrated improvement in laryngeal exam findings, whereas among the neurosurgical cohort nine of 21 (43%) and six of 21 (29%) demonstrated improvement or resolution of laryngeal exam findings, respectively.

DISCUSSION

Chronic cough, dysphonia, dysphoric breathing, and dysphagia due to LCN (CN IX and CN X) compression at the brainstem is a newly described phenomenon with no prior characterization in the literature. Given that compression of other cranial nerves gives rise to several well-characterized syndromes such as hemifacial spasm and trigeminal neuralgia, it is reasonable to expect that a similar process could be involved with LCNs that would respond to similar therapeutic modalities such as neurosurgical decompression. The present investigation demonstrates that compression of the LCNs and their rootlets is present in a significant percentage of patients whose diagnosis is uncertain after extensive workup in accordance with the described algorithm (Fig. 1). Thirty-three percent of all patients presenting to our academic medical center with a laryngeal symptom(s) combined with a

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pathologic laryngeal finding on stroboscopy who then underwent MRI showed detectable ipsilateral LCN compression (Fig. 2).

Laryngeal symptoms secondary to LCN compression at the brainstem significantly impact a patient's quality of life. Initial mean V-RQOL of patients with symptomatic LCN compression was 57.5, which is 15 SDs away from the mean for healthy patients on the V-RQOL. It is unclear why advanced age was associated with better scores on initial V-RQOL and GCI, but it potentially may be related to favorable age-related changes in LCN neurophysiology. Further study is warranted to clarify this relationship.

This study demonstrates that neurosurgical decompression improves quality of life for patients with symptomatic LCN compression at the brainstem to a greater degree than non-neurosurgical management. This data corroborates the authors’ experience suggesting that whereas non-neurosurgical interventions may lead to modest symptom improvement, neurosurgical decompression offers a greater likelihood for complete or near-complete symptom resolution. For this reason, after diagnosis of LCN compression at the brain stem we may offer patients a neurosurgical consultation to discuss neurosurgical decompression as a potential next step in treatment without a prior trial of amitriptyline or GABA analogues. We consider non-neurosurgical treatment a second-line therapy for those who decline neurosurgical intervention or are poor candidates for operative intervention due to medical comorbidities. Our finding that LCN decompression led to complete/near-complete symptom resolution in 42% of patients with mean (SD) follow-up of 3.0 (2.0) years suggests that LCN decompression outcomes are not as favorable as trigeminal neuralgia decompression outcomes; in one series, 75% of trigeminal neuralgia decompression patients reported complete symptom relief at 12 months. The etiology of this less favorable symptom improvement among patients with LCN decompression may be multifactorial: 1) the small size of the lower cranial nerves and their rootlets may increase vulnerability to compressive damage; 2) maladaptive compensatory laryngeal changes may persist even after normal nerve function is restored; and 3) the more complex and subtle nature of laryngeal symptoms may lead to delayed initial medical evaluation and require a lengthy workup, thus increasing the duration of nerve compression prior to definitive treatment.

A thorough workup (Fig. 1) must be performed to exclude all other pathologies prior to evaluating for LCN compression at the brainstem and considering neurosurgical decompression. Laryngopharyngeal reflux, asthma, and allergies must be ruled out as potential etiologies to avoid unnecessary expenditures associated with imaging a patient with a low pretest probability of disease and to minimize the risk for a false positive diagnosis that could subject a patient to unnecessary and potentially harmful surgical intervention. The high prevalence (49 of 149, 33%) of symptomatic LCN compression at the brainstem among patients referred for MRI at our institution is secondary to 1) our status as a referral center for patients failing management by other providers, and 2) a rigorous algorithm to exclude other etiologies. The true prevalence of symptomatic LCN compression at the brainstem within the general population is unknown but is likely to be rare. A thorough workup is necessary to minimize the risk of false-positive findings. All (24 of 24, 100%) of our patients undergoing neurosurgical decompression were noted to have anatomical compression of LCN at the brain stem at the time of surgery, corroborating the findings noted on preoperative MRI. Whereas the previously described complication profile of the neurosurgical cohort may be deemed acceptable, it must be stressed that appropriate patient selection and preoperative counseling are imperative due to the potential for serious complications.

Non-neurosurgical treatment is an acceptable option for those patients with LCN compression at the brain stem wishing to avoid neurosurgery. Pharmacologic trials of amitriptyline, pregabalin, or gabapentin utilizing standard dosing regimens are discussed with patients. Evidence suggests that these agents inhibit the neurogenic cough reflex and can be associated with clinically significant symptom improvement. All require careful titration and monitoring for adverse effects, including anticholinergic effects of amitriptyline and sedation and/or fatigue in the GABA analogues. For patients with vocal fold atrophy and paresis/paralysis, injection medialization is offered (Fig. 5).

The role of laryngeal electromyography (LEMG) in these patients is unclear. Whereas LEMG is utilized in the practice of the senior author (L.H.) and was performed on a number of these patients, we express concern regarding the reliability of qualitative LEMG findings in patients with vocal fold paresis, as has been observed elsewhere. The issues of 1) guarding that lead to reduced recruitment and 2) the small sampling area of the LEMG needle significantly impair the reliability of LEMG interpretation in paresis. These issues, combined with patient intolerance of and refusal to undergo the procedure, limit our ability to make any definitive conclusions at this time about LEMG in these patients. Thus, as has been observed in the greater laryngology community, our primary modality for diagnosing paresis in these patients has been stroboscopy. Further

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**Fig. 5.** Algorithm for non-neurosurgical management of patients with laryngeal manifestations of lower cranial nerve (LCN) IX/X compression at the brainstem. Amitriptyline is our first-line pharmacologic therapy. Patients are counseled about its anticholinergic effects. GABA analogues—pregabalin and gabapentin—are our second-line pharmacologic therapy, and patients are counseled about the dizziness and somnolence associated with them. Speech and language pathologist evaluation includes vocal hygiene training, cough suppression strategies, voice therapy, clinical/radiologic swallowing evaluation/treatment, and psychoeducational counseling. Injection medialization or medialization laryngoplasty are offered to patients with significant vocal fold atrophy. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]
study is needed to understand the role of LEMG in these patients and to develop optimal protocols for reliable interpretation.

This study is limited by its nonrandomized retrospective design and small sample size. Complete data was available to characterize all patients’ chief complaints, demographics, operative findings, and postoperative complications. However, initial quality-of-life surveys were available for only 29 of 49 (55%) patients. Although it has been the practice of the senior author (L.H.) to give V-RQOL, RSI, and GCI surveys to all patients at each visit, the retrospective nature of this study makes it susceptible to incomplete data. Although it is unclear to what degree a placebo effect may contribute to differential outcomes among patients treated with non-neurosurgical compared to neurosurgical interventions, the difference in postoperative laryngeal exam findings observed in the neurosurgical cohort—nine of 21 (43%) and six of 21 (29%) demonstrating an improvement or resolution in their exam findings—suggests a specific treatment effect. This is consistent with prior meta-analyses demonstrating no significant placebo effect on objective outcomes.34,35 Additionally, this study’s nonrandomized design creates a selection bias and limits our ability to directly compare the neurosurgical and non-neurosurgical cohorts, a common difficulty among surgical investigations. This limitation is mitigated by the lack of differences noted in the two cohorts’ demographics and initial clinical characteristics (Table II). Given these limitations, the current study thus serves as preliminary evidence characterizing symptomatic LCN compression at the brainstem and its response to treatment, which merits further prospective multi-institutional investigation.

CONCLUSION

Lower cranial nerve compression at the brainstem should be investigated when all other etiologies are meticulously excluded. Neurosurgical LCN decompression demonstrates acceptable results but requires careful patient selection and preoperative counseling about potential complications.

BIBLIOGRAPHY