Laryngeal Features of External Superior Laryngeal Nerve Denervation: Revisiting a Century-Old Controversy

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A long-standing controversy exists regarding the laryngoscopic features associated with unilateral denervation of the external superior laryngeal nerve (ESLN). Recently, we modeled acute unilateral cricothyroid muscle paralysis by blocking the ipsilateral ESLN with lidocaine hydrochloride, and identified epiglottic petiole deviation to the side of paralysis during high-pitched voice production as a possible diagnostic sign. This study provides preliminary clinical evidence supporting the presence of petiole deviation in cases of ESLN denervation. Epiglottic petiole deviation to the side of weakness was present in electromyographically confirmed cases of unilateral partial or complete ESLN denervation, in isolation or in combination with denervation of other branches of the vagus nerve. In addition, a case of complete ESLN and recurrent laryngeal nerve (RLN) denervation showed return of the petiole to the midline 6 months after surgical reinervation of the ESLN and RLN. Finally, petiole deviation was not present in isolated RLN paralysis — a finding suggesting that the diagnostic sign is uniquely associated with ESLN denervation. We concluded that deviation of the petiole to the side of cricothyroid muscle weakness during high-pitched voice production represents a potential diagnostic sign of unilateral ESLN denervation. Further research is necessary to determine factors that influence the expression and detection of this sign, as well as its diagnostic precision.

**Key Words:** denervation, epiglottic petiole deviation, superior laryngeal nerve.

INTRODUCTION

The external branch of the superior laryngeal nerve (SLN) innervates the cricothyroid (CT) muscle, which is divided into 3 distinct bellies or compartments, including the pars oblique, the pars recta, and a more recently discovered horizontal belly.1 The CT muscle contributes to control of vocal fundamental frequency (F0) and its psychophysical correlate "vocal pitch."2-6 Although the CT muscle undoubtedly contributes to vocal fold shortening — the primary geometric variable for adjustments of F0 — its effects on voice seem to extend well beyond the F0. For instance, in clinical circles, unilateral CT muscle dysfunction due to injury of the external branch of the SLN (ESLN) allegedly produces a wide range of voice and laryngeal effects.8-10 However, their exact nature and degree, as well as the tasks that seemingly provoke these phonatory and laryngeal changes, are not well documented or understood, and have been the source of considerable debate.11

For more than a century, a controversy has existed regarding the laryngeal signs that are presumed to be pathognomonic for unilateral ESLN paralysis. Although myriad descriptions exist of laryngeal behaviors ostensibly associated with unilateral ESLN denervation, no consensus has emerged since Mygind12 in 1906 first reported that the glottis was oblique during phonation in patients with unilateral ESLN paralysis. Indeed, most early descriptions involved rotation of the larynx, presumably due to asymmetric CT muscle dysfunction. This classic view held that ESLN denervation caused the weakened ipsilateral vocal fold to be shortened and to be at a lower level, while the anterior larynx shifted to the side of the intact CT muscle, creating an oblique glottis with the posterior larynx rotated toward the weakened side.13-15 However, as Abelson and Tucker15 reported nearly 30 years ago, there was considerable disagreement and controversy surrounding this view. For instance, although some authorities confirmed that the anterior commissure rotated to the paralyzed side,16 other experts asserted that the posterior larynx rotated as well, but there was no consensus as to which side.17 Still others failed to consistently find an oblique glottis.18 In an attempt to resolve the controversy, Abelson and Tucker15 used local anesthetic to temporarily paralyze the ESLN in 4 volunteers. Laryngoscopy during the block showed a
symmetric larynx at rest, but during phonation the posterior commissure pulled toward the side of temporary paralysis. Also, the affected vocal fold was slightly bowed and appeared shorter. Abelson and Tucker\textsuperscript{15} concluded that ESLN paralysis in humans can be suspected when “an oblique glottic chink is observed during phonatory efforts. Such a finding is caused by rotation of the posterior commissure toward the side of paralysis. The aryepiglottic fold on the side of paralysis is shortened, and the aryepiglottic fold on the opposite side is lengthened.”\textsuperscript{15(p665)} Later, Tanaka et al\textsuperscript{19} confirmed that 9 of 12 patients with SLN paralysis clearly showed rotation of the posterior glottis to the paralyzed side, primarily during pitch elevation.

Although the experiment of Abelson and Tucker,\textsuperscript{15} combined with the clinical report of Tanaka et al,\textsuperscript{19} seemed to briefly quiet the controversy, more recent findings have seriously questioned the value of larynx rotation as a salient laryngoscopic sign of ESLN paralysis. Several researchers have identified a number of additional laryngostroboscopic characteristics that seemingly occur more frequently. For instance, in a population of 126 patients with SLN paresis and paralysis, Dursun et al\textsuperscript{9} reported vocal fold lag, asymmetry, and height disparity (scissoring) as the most distinct findings of SLN dysfunction, as well as decreased amplitude and mucosal wave of the affected fold. Interestingly, however, Dursun et al\textsuperscript{9} did not identify larynx rotation as a salient feature of SLN paralysis. More recent studies have also failed to identify larynx rotation as a key feature of SLN injury. Rather, sluggishness of the ipsilateral vocal fold during repetitive adduction and abduction tasks and decreased longitudinal tension have been offered as the sine qua non of SLN injury. For example, Eckley et al\textsuperscript{20} reported on a group of 56 adults with SLN paresis or paralysis diagnosed from laryngeal electromyographic (LEMG) findings. The authors described 3 severity levels of paresis with associated laryngeal findings. Mild SLN denervation showed a slight sluggishness of the vocal fold on the weakened side and usually a mild deficit in the ability to increase longitudinal tension during pitch elevation. In moderate SLN denervation, the sluggishness and deficit in pitch elevation were obvious, and severe SLN denervation manifested as almost no motion of the CT muscle on the affected side, but with tone still present. There was no reporting of rotation of the larynx as a distinct feature of SLN paresis or paralysis, however.

Like Eckley et al,\textsuperscript{20} Heman-Ackah and Batory\textsuperscript{21} emphasized sluggishness of the affected vocal fold as evidence of SLN injury. The authors declared that if the integrity of the CT and cricoarytenoid joints is shown to be normal, hypomobility in the distribution of the SLN is manifested by sluggishness in adduction and longitudinal tension, observed especially during rapid repetition of “ee” and “hee” (ie, repetitive phonatory tasks [RPTs]). Rubin et al\textsuperscript{22} echoed the claims of Heman-Ackah and Batory\textsuperscript{21} that vocal fold lag (sluggishness), especially during fatiguing RPTs, was a hallmark of SLN injury, explaining that paretic nerves fatigue more quickly than normal nerves. Rubin et al warned that SLN paresis can be misinterpreted as recurrent laryngeal nerve (RLN) paresis, because “vocal fold lag from SLN paresis can present as sluggish abduction as well as adduction.”\textsuperscript{22(p685)} They argued that RPTs were useful diagnostic tests for identifying SLN paresis.

In an apparent reversal, however, Heman-Ackah and Barr\textsuperscript{10} later stated that in cases in which mild hypomobility is observed on physical examination, “no muscle pattern of hypomobility can identify the paretic nerve or nerves accurately.”\textsuperscript{10(p277)} This retreat from their previous conclusions presumably reflects the authors’ attempts to explain their finding of poor agreement between the actual LEMG evidence, which identified which side of the larynx was affected and/or which nerves (ie, RLN, SLN, or both) were affected on that side, and the clinician reports of the sidedness of hypomobility observed during laryngoscopy. According to the authors, there was frequent disagreement between the LEMG findings and the laterality of the hypomobility observed clinically. In an attempt to explain the poor agreement observed between physical examination and LEMG data, Heman-Ackah and Barr\textsuperscript{10} suggested that compensation for weakness may explain the disparate results. They reasoned that “paresis of the laryngeal nerves results in asymmetrical muscle forces in the larynx, and depending upon the relative compensation from the unaffected muscles and the degree of pull from the affected muscle, the pattern of hypomobility observed may not necessarily coincide with the expected mobility pattern.”\textsuperscript{10(p279)} Therefore, this finding not only cast doubt on the diagnostic precision of vocal fold lag as a reliable marker of SLN injury, but it raised the possibility that asymmetries observed in the larynx may actually reflect secondary attempts to compensate for unilateral weakness, rather than the pure effects of the primary disorder (ie, SLN denervation). To date, no studies have examined clinical manifestations of acute SLN injury to determine whether some of the heterogeneity in the reported findings may reflect merely individual differences in compensatory adjustments or the specific vocal tasks used to evaluate patients.

In an attempt to shed light on this controversy, Roy et al\textsuperscript{23} selectively blocked the ESLN using li-
doxycaine hydrochloride in order to identify the salient laryngeal features associated with acute, unilateral CT muscle dysfunction. Ten vocally normal men underwent lidocaine block of the right ESLN with LEMG verification. Flexible videolaryngostroboscopic recordings of participants performing a wide variety of vocal tasks were acquired before and during the block. Eleven blinded, expert judges rated randomized recordings from before and during the block of 10 vocal tasks using standardized flexible videolaryngostroboscopic rating protocols. Contrary to clinical reports, the investigators reported no evidence of hypomobility or sluggishness of the ipsilateral vocal fold, nor a consistent pattern of axial rotation of the larynx. Instead, the analysis revealed deviation of the petiole of the epiglottis to the side of CT muscle weakness in 60% of participants during an upward glissando maneuver produced at normal volume. This finding had not been reported previously as a manifestation of unilateral CT muscle paralysis. Several questions remained, however, regarding the validity of using lidocaine-induced paralysis to model selective ESLN damage only. One limitation of the model involved the possibility that lidocaine may have diffused into and/or infiltrated surrounding ipsilateral extrinsic laryngeal muscles (ie, sternothyroid, sternohyoid, thyrohyoid) or nerves. Thus, by extension, epiglottic deviation might have reflected the cumulative effects of ESLN dysfunction, as well as any regional extrinsic muscle dysfunction. A second limitation of lidocaine-induced paralysis is that it may not have replicated the mechanism of injury and repair seen clinically in the postacute and chronic stages of neural recovery. There may be functional differences between lidocaine-induced CT muscle paralysis and what occurs clinically when the nerve is damaged via trauma (ie, nerve sectioning, stretching, or compression) or, alternatively, via infectious processes, especially during the postacute recovery phase. Finally, the lidocaine-induced CT muscle paralysis model attempted to simulate the effects of acute paralysis, and it is possible that such a model differs substantially from what would be observed clinically in cases of chronic denervation, wherein adaptive or maladaptive compensatory muscle activation patterns can exist. Thus, Roy et al cautioned that clinical studies were necessary to evaluate the presence of epiglottic petiole deviation not only as a possible diagnostic marker of acute, complete, unilateral ESLN denervation, but also in cases of chronic partial or complete denervation, and/or in combination with other neuropathies, including those of the RLN.

In this regard, our report presents evidence from several clinical cases that illustrate epiglottic peti-ole deviation to the side of weakness (during high-pitched voice production) in the presence of confirmed ESLN denervation. Clinical cases are presented to illustrate either isolated unilateral partial or complete ESLN denervation, or denervation of the ESLN in combination with the other branches of the vagus nerve. In addition, a case of complete RLN and ESLN denervation is presented that shows a return of the petiole to the midline 6 months after surgical reinnervation of the ipsilateral ESLN and RLN. Finally, a case of isolated RLN paralysis is presented to suggest that petiole deviation is not necessarily a consequence of any asymmetric intrinsic laryngeal muscle forces, but rather, seems to be uniquely associated with ESLN involvement. These cases of chronic partial or complete ESLN denervation at various stages of reinnervation seem to provide preliminary support for the finding of epiglottic petiole deviation derived from the lidocaine-induced model of acute CT muscle paralysis.

CASE REPORTS

Case 1: Right ESLN Paresis. A 70-year-old man reported an 8-month history of voice difficulties following right transcervical removal of a C4-5 vertebral schwannoma. The patient’s Voice Handicap Index (VHI) score was 35, indicating a mildly handicapping voice disturbance. He also self-assessed his voice problem as mild. He presented with a mild to moderate dysphonia, and his voice was rated by a laryngologist and a speech-language pathologist as G2 R2 B1 A1 S1 on the GRBAS scale (grade, roughness, breathiness, asthenia, and strain). During flexible laryngoscopy, the vocal folds initially abducted and adducted normally. However, after RPTs designed to fatigue the laryngeal mechanism (rapid repetition of the syllable “hee” for approximately 15 seconds), the right vocal fold began to lag during abduction, with slightly reduced mobility as compared to the left. During vocal fold abduction, the petiole was also observed to remain in the midline (Fig 1A). During stroboscopy, phonation produced at normal pitch and loudness was associated with only touch closure of the true vocal folds, with the open phase dominating the vibratory cycle. The petiole was observed to be in the midline (Fig 1B); however, there was no evidence of a reduced mucosal wave, reduced amplitude of vibration, or phase asymmetry. During phonation elicited at highest pitch and during upward glissando, there was obvious deviation of the petiole of the epiglottis to the right side (Fig 1C). After clinical examination, the patient underwent LEMG. Concentric-needle LEMG of the CT and thyroarytenoid (TA) muscles revealed normal electrodiagnostic findings for the right TA muscle. However, there was electrodiagnostic evidence of
reduced recruitment and complex-appearing motor units in the right CT muscle, indicating denervation and partial reinnervation consistent with axonal injury to the right SLN only. This finding not only provides support for petiole deviation as a marker of isolated ESLN denervation, but provides some evidence to support the contention of Rubin et al\textsuperscript{22} that vocal fold lag during fatiguing RPTs may reflect SLN denervation.

Case 2: Right ESLN and RLN Paresis. A 40-year-old man reported a 4-month history of globus sensation, vocal fatigue, and increased physical effort associated with voice production, especially after extended voice use. The patient reported a VHI score of 16, indicating a mildly handicapping voice disorder. He self-assessed his voice disturbance as mild. At the time of examination, his voice was judged by both a laryngologist and a speech-language pathologist to be perceptually normal (ie, G0 R0 B0 A0 S0). Videolaryngostroboscopy showed a normal range and speed of abductive and adductory movements of the vocal folds. During abduction, the petiole was positioned in the midline, and there were two small vocal process granulomas (right greater than left; Fig 2A). During phonation at normal pitch and loudness, the right arytenoid cartilage hooded over the right vocal fold, with scissoring of the arytenoid cartilages. The petiole remained in a central position (Fig 2B). Vibratory characteristics were judged to be within normal limits. During pitch excursion into the uppermost region of the pitch range during upward glissando, the petiole of the epiglottis was observed to deviate substantially to the right (Fig 2C). Concentric-needle LEMG completed after the clinical examination showed no abnormal spontaneous activity, but showed increased complexity and amplitude of fast-firing motor units in the right TA muscle and right CT muscle consistent with previous injury to the right RLN and SLN with subsequent partial reinnervation.

Case 3: Brain Stem Arteriovenous Malformations and High Left Vagal Injury. The patient was an 18-year-old man with a history of arteriovenous malformations of the brain stem beginning at 2 years
of age, for which he underwent craniotomy with subsequent severe dysphagia, left vocal fold paralysis, and mild hypernasality. According to the patient, the voice difficulties, vocal fold paralysis, and dysphagia resolved after approximately 6 months. At 16 years of age, however, he experienced rebleeding of the arteriovenous malformation, with worsening of hypernasality and dysphagia. At the time of examination, he reported a VHI score of 34, indicating a mildly handicapping voice disturbance, and he self-assessed his voice disturbance as moderate. Clinical examination revealed moderate hypernasality and mild dysphonia. Inspection of the oral cavity showed that the palate drooped on the left side at rest, and during phonation the palate was observed to elevate asymmetrically to the right side. Flexible videolaryngostroboscopy confirmed incomplete velopharyngeal closure with deviation of the soft palate to the right during sustained high-pressure oral consonant productions, leaving a left velopharyngeal gap. During quiet breathing and voice produced at normal pitch and loudness, as well as during upward glissando, the petiole of the epiglottis was lateralized to the left of the anterior commissure (Fig 3A-C). The patient underwent LEMG, which confirmed complete denervation of the left CT and TA muscles with fibrillation potentials and no recruitable motor units. There was no electrodiagnostic evidence of reinnervation. The patient subsequently underwent laryngeal reinnervation surgery with left RLN and left SLN-ansa cervicalis reinnervation with injection of Cymetra (LifeCell Corp, Branchburg, New Jersey) approximately 7 months after the onset of symptoms. At 2.5 months after reinnervation surgery, the patient returned for vocal fold injection of hyaluronic acid, as the effects of the Cymetra injection had abated. At 6 months after reinnervation surgery, the patient returned for reevaluation. The patient judged his voice to be 80% of normal, and it was perceptually assessed by the laryngologist and speech-language pathologist as G1 R1 B1 A1 S0. Inspection of the larynx with rigid videostroboscopy documented an improved medial position of the left vocal fold with the arytenoid cartilage stabilized and not hooded over, and the epiglottic petiole now approximated the midline during abduction (Fig 4D), during voice produced at normal pitch and loudness (Fig 4E), and during high-pitched voice production (Fig 4F). This case suggests that reinnervation of the ESLN potentially restores tone to the ipsilateral CT muscle, thereby reversing the
effects of the asymmetric muscle forces likely responsible for petiole deviation. Furthermore, this case suggests that chronic, complete unilateral denervation of the ESLN (with no evidence of reinnervation) may produce petiole deviation at rest and during other phonatory tasks, rather than just during high-pitched voice productions.

Case 5: Isolated Complete Right RLN Denervation

A lingering question remains regarding the significance and interpretation of epiglottic petiole deviation. Is it possible that any asymmetric muscle forces will produce epiglottic petiole deviation, and that such a finding should not necessarily be ascribed exclusively to ESLN denervation? In other words, is epiglottic petiole deviation unique to unilateral ESLN denervation, or is it observed in cases of isolated RLN paralysis wherein asymmetric muscle forces likely responsible for petiole deviation. Furthermore, this case suggests that chronic, complete unilateral denervation of the ESLN (with no evidence of reinnervation) may produce petiole deviation at rest and during other phonatory tasks, rather than just during high-pitched voice productions.

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cle forces also exist? To explore this question, we looked at a case of isolated unilateral RLN denervation. The patient was a 40-year-old man who reported altered voice quality following a right anterior approach cervical laminectomy for C7 radiculopathy approximately 1 year earlier. On examination, the voice was breathy and of decreased volume. Concentric-needle LEMG of the right TA and CT muscles was completed. The right TA muscle showed profuse abnormal spontaneous activity but no recruitable motor units. The CT muscle motor units appeared normal. There was electrodagnostic evidence of complete denervation of the right TA muscle consistent with right RLN denervation. The function of the right SLN was considered normal. During videostroboscopy the petiole was observed to be positioned in the midline across all tasks, including quiet breathing (Fig 5A), voice produced at normal pitch and loudness (Fig 5B), and voice produced during highest pitch (Fig 5C). This case provides preliminary evidence that epiglottic petiole deviation, if present, appears to be uniquely associated with asymmetric CT muscle dysfunction, rather than general asymmetric muscle forces within the larynx.

CONCLUSIONS

These clinical cases of chronic partial or complete ESLN denervation, at various stages of reinnervation, seem to provide preliminary support for the findings derived from the lidocaine-induced model of acute CT paralysis. Epiglottic petiole deviation to the side of CT muscle weakness during high-pitched voice production represents a potentially valuable diagnostic sign of unilateral ESLN denervation. However, further research is necessary to establish the diagnostic precision of this sign, as well as the factors that influence sign detection and expression. Larger clinical studies are needed to determine whether petiole deviation routinely occurs in vocally normal individuals, in those with isolated RLN paralysis, and/or in those with other types of voice disorders not associated with ESLN or RLN denervation. In addition, factors that may potentially influence the presence, detection, and interpretation of this laryngoscopic sign or feature need to be better understood. For instance, anatomic variation (prominent versus flat petiole), camera angle, flexible versus rigid endoscopy, the patient’s ability or talent in performing upward glissando, the degree of petiole deviation, the mobility of the CT joint, and the presence and amount of supraglottic compression are a few factors that may influence the degree of petiole deviation, and the examiner’s ability to accurately detect and interpret it. In summary, rigorous evaluation of diagnostic test performance (using conventional estimates of sensitivity, specificity, and positive and negative likelihood ratios) is critical to establishing the diagnostic worth of epiglottic petiole deviation as a potential marker of unilateral ESLN denervation.

REFERENCES

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