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A Novel Technique for Clinical Assessment of Laryngeal Nerve Conduction: Normal and Abnormal Results

Lucian Sulica, MD, Bridget Carey, MD, and Ryan C. Branski, PhD

From the Department of Otolaryngology–Head & Neck Surgery (L.S.); the Department of Neurology and Neuroscience (B.C.), Weill Cornell Medical College; and the Department of Otolaryngology (R.C.B.), New York University School of Medicine, New York, New York, U.S.A

Abstract

Objectives/Hypothesis—To describe a novel conduction study of the laryngeal nerves, including normal values and abnormal findings.

Study Design—Prospective nonrandomized.

Methods—Seventeen healthy adult volunteers, as well as three patients with clinically identified laryngeal neuropathy, underwent low-level brief electrical stimulation of the laryngeal mucosa by means of a wire inserted via a transnasal flexible laryngoscope. Bilateral hookwire electrodes recorded the result in the laryngeal adductor muscles.

Results—This study yields an early response ipsilateral to the side of stimulation (LR1), which is uniform and consistent (right 5 13.2 6 0.80 msec; left 5 15.2 6 1.20 msec), and late bilateral responses (ipsilateral LR2 [LR2i] and contralateral LR2 [LR2c]), which exhibit greater variation in latency and morphology (right LR2i 5 50.5 6 3.38 msec; left LR2i 5 52.2 msec; right LR2c 5 50.7 6 4.26; left LR2c 5 50.6 6 4.07). Findings in abnormal patients differ significantly from normal, consistent with the distribution of neuropathy.

Conclusions—We describe a novel, clinically applicable conduction study of laryngeal nerves. Normative electrodiagnostic values and variations of the reflex responses of the laryngeal adductor muscles in response to irritative stimulation of the laryngeal mucosa (Laryngeal Closure Reflex) are proposed. By enabling the determination of electrophysiological parameters of the superior laryngeal and recurrent laryngeal branches of cranial nerve X (CN X), this procedure, which is used as an adjunct to laryngeal electromyography, may provide earlier and more accurate information regarding the extent and grade of nerve injury. Because injury grade relates directly to prognosis, the information derived from this test may have clinical relevance in determining optimal treatment.

Level of Evidence—4.

Send correspondence to Lucian Sulica, MD, Dept. of Otorhinolaryngology, 1305 York Avenue, 5th Floor, New York, NY 10021. lus2005@med.cornell.edu.

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Keywords

Larynx; laryngeal neuropathy; laryngeal paralysis; paresis; vocal fold; vocal fold paralysis; nerve conduction; laryngeal electromyography; electromyography; laryngeal nerve conduction; prognosis; diagnosis

INTRODUCTION

Electromyography (EMG) is a means of studying electrical activity in muscle. As such, it is uniquely suited to diagnose laryngeal neuropathic disease and address several clinical questions that arise commonly during its evaluation. These include distinguishing neuropathic from mechanical immobility, determination of site of lesion, estimation of prognosis, and evaluation of synkinesis and other phenomena of misdirected reinnervation. As the only diagnostic tool that can reveal laryngeal neurologic activity in vivo, EMG has greatly expanded its clinical scope in the decades since the seminal work of Faaborg-Andersen and Buchtal in the late 1950s.^{1,2} Despite widespread acceptance,³ EMG as currently used clinically in the larynx has important limitations. Information is complicated by the potential for synkinetic reinnervation from adductor and abductor fibers commixed in the same nerve, which creates ambiguity in prognostication. In addition, EMG is blind to the afferent nerves.

In most peripheral neuropathies, electrodiagnosis is a two-part undertaking, comprising both EMG and nerve-conduction studies. In the larynx, nerve-conduction techniques are not in routine clinical use, almost certainly because of technical challenges to nerve stimulation imposed by anatomy. The accessible portion of the superior laryngeal nerve is relatively short and lies near other nerves that can create confusing results; and the recurrent nerve is deep and difficult to reach reliably outside of the operating room. Comparison of laryngeal nerve anatomy with that of the facial nerve, routinely assessed by means of nerve conduction testing, reveals the obvious anatomic difficulties presented by the former. Efforts at a noninvasive method of stimulation have included use of surface electrodes, a vibratory stimulator,⁴ and magnetic stimulation.⁵ Isolated reports of needle stimulation techniques have appeared in the literature,^{6,7} but despite apparently promising results the techniques have been incompletely described and have not been widely adopted, especially with respect to determining prognosis.

The goal of this investigation is to describe a novel technique for the clinical evaluation of laryngeal nerve condition, establish normative values, and describe the results when applied to patients with laryngeal neuropathy.

MATERIALS AND METHODS

This study was approved by the Institutional Review Board of Weill Cornell Medical Center, New York, New York.

The study population consists of two groups. The first is composed of healthy adult volunteers with no complaints of voice, swallowing, or respiratory problems recruited from

the general population. Volunteers were in good health, without symptomatic dysfunction of larynx, and with a normal laryngeal examination by laryngoscopy. The second is composed of patients recruited from among those with vocal fold paralysis or paresis, presenting for clinical care at a university laryngology practice.

Utilizing the standard percutaneous approach used clinically for diagnostic laryngeal electromyography, one hookwire EMG electrode was placed in each thyroarytenoid muscle. Accurate electrode placement was confirmed by volitional activation of these laryngeal adductors (asking the patient to phonate a sustained /i/). Reference electrodes were positioned over the lateral aspect of the thyroid cartilage on each side, and a ground electrode was placed midline over the superior sternum. The evoked potentials recorded from these muscles were displayed on a dual trace oscilloscope after conventional amplification using a two-channel amplifier (Nicolet Viking 11.2). Following electrode placement, the larynx was visualized by means of a flexible distal-chip laryngoscope with a working channel inserted transnasally under topical nasal anesthetic (2% lidocaine with 0.25% phenylephrine administered via atomizer), as is done for routine laryngeal evaluation in the office.

Activation of the sensory territory of the superior laryngeal nerve was achieved by means of unilateral irritative stimulation to the laryngeal mucosa in the form of a low level, brief electrical stimulus (5–10 mA, 0.1 ms). The stimulation protocol was selected based on the standard parameters used for the blink reflex study.⁸ The stimulus was delivered via a wire electrode passed through the working channel of the laryngoscope. Location of the stimulating electrode was observed endoscopically, and the stimulus was not delivered until placement was confirmed by the endoscopist. Stimulation was administered to the mucosa overlying the arytenoid mound, the aryepiglottic fold, or the superior surface of the ventricular fold (Fig. 1). The reflex adduction of bilateral vocal folds evoked by unilateral sensory stimulation (LCR) was recorded by needle electromyography. For each subject, a minimum of four recordings was obtained from stimulation of each side. The minimal latencies of the responses were marked, as agreed upon by two neurologists with fellowship training in clinical neurophysiology and neuromuscular disease. For the abnormal group, a diagnostic laryngeal EMG (LEMG) was performed concurrently. The protocol took approximately 20 to 30 minutes to execute.

RESULTS

Normal Group

Seventeen normal volunteer subjects (8 men, 9 women, ages 20–40) were included in the study. Responses were obtained and recorded following right- and left-sided stimulation of the laryngeal mucosa. This study yields an early response ipsilateral to the side of stimulation (LR1), which is uniform and consistent, and late bilateral responses (ipsilateral LR2 [LR2i] and contralateral LR2 [LR2c]), which exhibit greater variation in latency and morphology (Figs. 2–3).

Mean minimal latencies were calculated for each response (Table I). Proposed preliminary values for the upper limit of normal (ULN) for these latencies were determined by calculating 3 standard deviations (SD) above the mean (Table II).

Abnormal Group

Three subjects with established left vocal fold palsy were examined (Table III).

Subject 1 is a 74-year-old man with idiopathic left vocal fold paralysis (immobility) for 1 year prior to the examination. He recalled no inciting event or illness. Diagnostic LEMG revealed severely prolonged durations, and increased amplitudes of motor unit potentials and severely decreased recruitment with volitional activation on the left thyroarytenoid muscle. There was no spontaneous activity. Study of the left cricothyroid muscle and right cricothyroid and arytenoid muscles were normal. These findings are consistent with severe chronic left recurrent laryngeal nerve dysfunction. Nerve conduction testing resulted in significant delay of the early left-sided response minimal latency (20.8 ms) to left-sided stimulation. This exceeds the calculated ULN for the normal subjects for left LR1 (ie., > 3 SD above the mean). Further, the duration of the early responses is prolonged and variable between sequential responses in comparison to the uniform, triphasic LR1 responses observed in normal subjects. The ipsilateral and contralateral LR2 responses obtained by left-sided stimulation in this subject are within normal limits in comparison with the normal group. However, there is a marked difference between the ipsilateral (left) and contralateral (right) R2 latencies, with the left side being prolonged relative to the right (a finding not observed in normal subjects).

Subject 2 is a 27-year-old man with Marfan's syndrome with left vocal fold paralysis (immobility) following a repair of a thoracic aortic aneurysm 7 months prior to examination. Diagnostic LEMG showed severely decreased recruitment with volitional activation in the left thyroarytenoid muscle. Morphology of the few remaining motor units was normal. There was no spontaneous activity. This is consistent with moderate chronic left recurrent laryngeal nerve dysfunction. In nerve conduction testing, left-sided stimulation yields a LR1 latency that is within normal limits compared to the normative data set. However, the morphology of the early responses exhibits greater variability than observed in normal subjects. In this subject, left-sided (ipsilateral) late responses (LR2) were disorganized and indistinct, possibly reflecting temporal dispersion, and preventing agreement in latency measurement between the evaluating neurologists. They are marked as absent in Table III, although some response is probably present. Of note, this is consistent with the morphologic disarray demonstrated in LR1. The right-sided (contralateral) late responses are within normal limits in comparison with the normal group.

Subject 3 is a 41-year-old woman with an idiopathic right vocal fold paresis (hypomobility) following upper respiratory infection 8 months prior to testing. The stroboscopy examination was atypical in that the arytenoid mobility was near normal, but the membranous vocal fold was severely atrophic. Diagnostic LEMG revealed abnormal spontaneous activity (fibrillation potentials and positive sharp waves) in the right cricothyroid and thyroarytenoid muscles. Increased motor unit potential amplitudes were observed in the right cricothyroid muscle.

Volitional activation is absent in the right thyroarytenoid muscle and moderately decreased in the right cricothyroid muscle. In the left thyroarytenoid muscle, a mildly decreased interference pattern is observed with volitional activation. Study of the left cricothyroid is normal. This is consistent with severe right recurrent laryngeal nerve dysfunction, moderate right superior laryngeal nerve dysfunction, and mild left recurrent laryngeal nerve dysfunction. In nerve conduction testing, left-sided stimulation yielded an early response (LR1) within normal limits compared to normative data, as well as ipsilateral (left) late response (ipsilateral LR2) that was also within normal limits. With left-sided stimulation, contralateral (right) LR2 responses are also obtained. These responses are notably longer than the mean value for the normal subject series, but within 3 SD of ULN, therefore within our proposed definition for normal range. To right-sided stimulation, neither the ipsilateral (right) early (LR1) nor the ipsilateral late (LR2) responses were obtained. Contralateral (left) late responses (contralateral LR2) were obtained. The minimal latency of these responses is within 3 SD of ULN, and thus within our proposed parameters for normal range, although again we note a response latency above the mean for normal subjects. This is consistent with the right superior laryngeal nerve dysfunction on the diagnostic LEMG.

DISCUSSION

Reflexes involving cranial nerve-brainstem pathways have long been identified and are well-described.⁹⁻¹¹ The corneal/blink reflex, gag reflex, and oculocephalic reflex have important clinical relevance as part of the neurologic examination. A nerve conduction study of the blink reflex (Blink Reflex Study) was described by Kimura in 1969.¹² This electrodiagnostic study provides quantifiable parameters allowing for evaluation and assessment of the peripheral afferent and efferent limbs of the blink reflex, as well as information regarding relay of this reflex through the central nervous system. Today, the Blink Reflex Study is performed frequently, along with nerve conduction studies and electromyography, as a routine component of a clinical electrodiagnostic examination. It can provide essential information regarding localization and degree of neuropathic injury involving cranial nerves V and VII, as well as provide information regarding relay through the brainstem.

Determination of the electrophysiological parameters of the nerves subserving the larynx in a similar manner may have similar clinical value for diagnosis and prognosis in patients with laryngeal neuropathic dysfunction. The laryngeal closure reflex (LCR) ensures protection of the airway via closure of bilateral vocal folds in response to irritative stimulation of laryngeal mucosa. The afferent and efferent limbs of this reflex are subserved by different branches of CN X; the afferent limb by sensory fibers of the superior laryngeal nerve (SLN) and the efferent limb by motor fibers of the recurrent laryngeal nerve (RLN). As with the Blink Reflex Study, we hypothesized that deviations from normative values can provide precise localization and information regarding the extent of neurological injury to the larynx, lending greater insight into the mechanisms of laryngeal dysfunction than available via current diagnostic tools.

While study of this (or any) reflex arc does not provide direct examination of sensory or motor nerves or nerve conduction, localization to afferent (sensory) or efferent (motor) limb can be deduced by analyzing the pattern of abnormalities on a bilateral study. For example,

in the case of a right sensory deficit (that is, dysfunction of right superior laryngeal nerve/afferent limb), stimulation of the right (affected) side would be expected to yield a delay or absence of all responses, while stimulation of the left (unaffected) side would result in normal responses throughout. If a right motor deficit was present (that is, dysfunction of right recurrent laryngeal nerve/efferent limb), a different pattern of abnormalities would be expected. Stimulation of the right/affected side would result in delayed or absent ipsilateral (right) LR1 and LR2 responses, with a normal contralateral (left) LR2 response. Stimulation of the left (unaffected) side would yield normal ipsilateral (left) LR1 and LR2 responses, with delayed or absent contra-lateral (right) LR2 responses.

The presence of an early, consistent unilateral response ipsilateral to the side of stimulation and more variable late bilateral responses is analogous to the results seen in the Blink Reflex Study. Extrapolating from the neuroanatomical understanding of the Blink Reflex Study, we postulate that the early ipsilateral LR1 response in this study represents a disynaptic connection between the nucleus solitarius and the ipsilateral nucleus ambiguus in the medulla, whereas the late bilateral LR2 responses are mediated by a polysynaptic pathway between the activated sensory nucleus (pre-summably the solitary nucleus, although some general afferent vagal sensory fibers are understood to synapse in the spinal trigeminal nucleus) and the ipsilateral and contralateral nuclei ambiguus^{12–15} (Fig. 4).

The recurrent laryngeal nerve has a unique anatomical feature of significant disparity in the length of the right and left nerves. In humans the average difference in length between the right and left recurrent laryngeal nerves is approximately 11 cm (for comparison, right to left differences average 0.8 cm in rats, 13 cm in dogs, 30 cm in giraffes).¹⁶ This is reflected in our data, in that a significant side-to-side difference in LR1 latencies is observed in our study ($P < 0.001$), with longer left LR1 latencies compared with right LR2 latencies, consistent with the longer anatomical pathway that the left-sided response must travel. Significant lateralized differences were not observed for the LR2 responses, perhaps as a result of polysynaptic relays within the brainstem that may mitigate this difference. It is also possible that the variation of these responses will require that a larger population is studied before a pattern emerges.

In each case, the responses obtained in the three abnormal subjects clearly depart from the normative data. Interestingly, they differ from the normative data in different ways. This is perhaps not surprising given that the abnormal subjects did not have identical mechanisms of nerve injury, nor did they have the same degree of neuropathic injury, as established by diagnostic LEMG. The results in abnormal subjects also suggest that we may be setting the upper limit of normal too high at three SD from the mean. For example, the latency of ipsilateral LR2 from subject 1 falls into the normal range, but is clearly prolonged (Table III). The three SD limit is determined arbitrarily without an underlying physiologic rationale. Further study may establish clearer patterns of abnormality correspondent with degree of injury or pathophysiological basis of injury, as well as more clearly defined limits of normal, which we are not able to appreciate given the limited sample size included here.

The larynx is home to one of the most dense and diverse networks of sensory fibers in the human body, comprising chemoreceptors sensitive to pH and various ion salts and their

concentrations, and mechanoreceptors sensitive to muscle position and contraction, superficial and deep touch, airflow, and intraluminal pressure.¹⁷ The current technique is limited to galvanic stimulation; and it remains unclear to what extent this reflects the function of the broader array of laryngeal receptors and whether, for example, it accurately mirrors clinically important dysfunction in aspiration. However, the technique does permit the measurement of nerve condition, heretofore not possible in the awake, unsedated patient.

The current electrodiagnostic assessment of laryngeal neuropathy is limited to electromyography. While capable of demonstrating motor neuropathy, and of establishing whether the nerve is in continuity in cases of trauma, electromyography has proved disappointing in prognostication. A recent meta-analysis of 10 series of patients assessed with laryngeal electromyography series has shown that over 40% (88 of 207) of patients with findings predictive of recovery did not in fact recover.¹⁸ This indifferent predictive value is probably a consequence of the admixture of adductor and abductor fibers in the recurrent nerve trunk, which creates the possibility of inappropriate and even counterproductive reinnervation. In the analogous case of facial neuropathy, electromyography alone has proven similarly unsatisfactory, yet nerve excitability,¹⁹ latency,²⁰ and amplitude of the muscle action potential²¹ have all been shown to add prognostic information. Prognostic information in laryngeal neuropathy is of obvious importance in selecting patients for early definitive treatment and sparing them unnecessary and repeated temporary procedures.

In addition to prognostic considerations, electromyography is blind to the afferent nerves. Laryngeal sensory neuropathy has been hypothesized to be critically important in swallowing safety, as well as a host of other dysfunctional phenomena believed to be related to sensation, including cough, laryngospasm, and paradoxical vocal fold motion. The only means of assessment of sensation is currently the administration of a puff of air via the working channel of a laryngoscope, which is in practice poorly reproducible and nonquantitative, yielding only the presence or absence of the laryngeal closure reflex as an observation. By incorporating the afferent arm of the laryngeal closure reflex, the method presented in this article may yield improved and more consistent data regarding this aspect of laryngeal neural function.

Given the likely heterogeneity of degrees and distribution of laryngeal neuropathy, a much larger group of abnormal subjects will require study to further explore the potential and clinical relevance of the technique. In addition, the technique is more time-consuming than EMG (20–30 minutes per study) and technically demanding. The principle technical limitation in our experience has been maintaining placement of bilateral thyroarytenoid electrodes while the laryngoscope is inserted and the stimulating electrode deployed, although some facility is gained with repetition. Some patients with chronic laryngeal irritative conditions do not tolerate the stimulating electrode without gagging. In addition to providing only an indirect assessment of nerve conduction as discussed above, the technique does not provide a direct measure of motor amplitude, analogous to facial nerve compound motor action potential.

CONCLUSION

This technique yields clear, quantifiable data regarding neurologic integrity of laryngeal function, including both afferent and efferent function, previously not routinely obtainable in the office setting. Data from affected patients clearly differs from normal values, although the characterization of these abnormal findings awaits further study.

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Figure 1.

The endoscopic view of the procedure in a normal patient. At left, the stimulating wire is positioned to stimulate the left-sided laryngeal mucosa. The wire extends well past the red insulation in the foreground. At right, the laryngeal closure reflex is triggered.

Figure 2.

A schematic of laryngeal innervation pathways as examined by this technique, and the early ipsilateral LR1 and late bilateral LR2 for left-sided stimulation.

Figure 3.

Study results from a normal subject. The top register records tracings from four individual stimulations to each side of the larynx. The bottom register superimposes these. The left tracing corresponds to stimulus on the left, and the right with stimulus on the right. Ipsilateral response is recorded on the top, and contralateral response on the bottom. The first vertical black line on the tracing is the LR1. The latency from stimulus is recorded as “Ipsi R1-Lat” in the table in the upper left of the figure. The second black line is the LR2 (recorded as “Ipsi R2-Lat” and “Contra R2-Lat” in the table in the upper left of the figure). The difference between ipsilateral and contralateral LR2 is shown at right in the table in the upper left of the figure.

Figure 4.

Neural pathways for laryngeal reflex responses. The LR1 (left) is a ipsilateral disynaptic response. The LR2 (right) is a bilateral response mediated via a hypothesized polysynaptic relay represented by the starred structure above.

Table 1

Mean Minimal Latencies and Standard Deviations in Normal Subjects.

Stimulation Side	LR1 Latency Ipsilateral	LR2 Latency Ipsilateral	LR2 Latency Contralateral
Right	13.2 ± 0.80 ms	50.5 ± 3.38 ms	50.7 ± 4.26 ms
Left	15.2 ± 1.20 ms	52.2 ± 2.97 ms	50.6 ± 4.07 ms

Table 2

Upper Limit of Normal (ULN) = 3 SD Above Mean.

Stimulation Side	LR1 Latency Ipsilateral	LR2 Latency Ipsilateral	LR2 Latency Contralateral
Right	15.6 ms	60.6 ms	63.5 ms
Left	18.8 ms	61.1 ms	62.8 ms

Table 3

Patients with Laryngeal Neuropathy.

	Stimulation Side	LR1 Latency Ipsilateral	LR2 Latency Ipsilateral	LR2 Latency Contralateral
Subject 1	Left	20.8 ms	60.2 ms	54.1 ms
Subject 2	Left	14.6 ms	Indistinct 1	54.6 ms
Subject 3	Right	Absent	Absent	54.0 m

The ipsilateral LR2 in subject 2, while present, is indistinct and likely due to marked temporal dispersion. The interpreting neurologists could reach no consensus regarding latency.