Workshop handouts are prepared as background didactic material to complement a hands-on workshop session. This workshop handout was originally prepared in August 1997 and revised in October 2007. The idea and opinions in this publication are solely those of the author(s) and do not necessarily represent those of the AANEM.
Precise neurological control of delicate laryngeal and pharyngeal structures is required to produce speech. The small size and rapid activation of the neuromuscular components of the larynx place high demands on programmed mechanisms in the central nervous system (CNS). Neurological disorders, both central and peripheral, may impair these fine coordinated motor programs causing dysarthria or dysphonia. Symptoms of voice disruption may present months before declaration of other regional symptoms.

CNS disorders, both pyramidal and extrapyramidal, and peripheral nervous system (PNS) disorders disrupt the coordinated production of speech. Although speech disorders occur commonly and appear in diverse clinical settings, the systematic method of clinical evaluation of voice disorders is seldom taught in medical school curricula. Understanding the unique features of the neurological disorders that effect speech and mastering the unconventional approach to clinical diagnosis and electrodiagnosis of voice disorders rely on a thorough understanding of the regional anatomy and function of the structures of the head and neck.

Central motor control of laryngeal function resides in the lower motor and premotor cortices. These frontal regions project to lower motor neurons in the nucleus ambiguous with complex contributions from the upper brainstem. Vocal cord (VC) adduction occurs with the combined action of the lateral cricoarytenoid (LCA), thyroarytenoid (TA), and interarytenoid (IA) muscles. Opinions differ on the relative role played by each. When the VCs are brought together, air is blown through them, creating a pneumatic vibration and the generation of sound. The intrinsic muscles control the fine tuning of pitch. Higher notes are attained by contraction of the cricothyroid (CT) muscle which pivots the thyroid cartilage forward with respect to the cricoid ring. Since the VCs are tethered to the thyroid cartilage anteriorly, this action creates an additional stretch on the cord.

Vibration of the VCs is produced by movement of air in the process known as phonation. The product of phonation is voice—the musical sound produced by VC vibration, acoustically enhanced and modulated by the upper airway. Phonemes, when combined with dentals, labials, and gutturals, form the building blocks of spoken words. The pitch, volume, and timbre
of the voice impart nonsyntactic qualities to speech that carry information regarding personality, mood, gender, and maturity of the speaker.

Intact laryngeal function also permits unobstructed breathing, blowing, sniffing, laughing, and whispering. Tight closure of the larynx is essential for swallowing and for Valsalva.

**VOICE ANATOMY**

**The Pharynx**

The pharynx is a conical tube formed of muscles and membranes extending from the skull base to the mid-cervical region. Seven openings communicate with the pharynx from the nose, mouth, middle ears, larynx, and esophagus. The pharynx is divided into nasal, oral, and pharyngeal parts, bounded posteriorly by superior, middle, and inferior constrictor muscles (Figure 1).

**Cranial Nerves**

VII: The facial nerve (VII) is almost purely motor. Integrity of seventh nerve function is important for the production of labial sounds for final tuning of sounds as they exit the mouth.

IX: Cranial nerve IX originates in the medulla and exits the skull (with X and XI) through the jugular foramen, supplying the soft palate and oropharynx. Efferent ninth nerve activity is attributed to regions of the tonsils, soft palate, and oropharynx, playing a role in swallowing and in controlling the opening to the nasopharynx for modulation of nasal and guttural sounds. X: The dorsomotor nucleus and nucleus ambiguous supply visceral and somatic efferent contributions to the vagal nerve (X) that empowers most of the muscles of the pharynx, palate, and larynx. The superior laryngeal nerve (SLN) branches from the vagus high in the neck. SLN, by way of the internal laryngeal branch, is the nerve of sensation to the larynx, but it also supplies the CT muscle via its external laryngeal branch. The recurrent laryngeal nerve (RLN) takes a long reflected course from the thorax where it winds forward around the subclavian artery on the right or around the aorta on the left, ascending along the trachea. RLN is the motor nerve of the larynx, supplying all of the intrinsic laryngeal muscles except for the CT.

XI: The spinal accessory nerve (XI) is purely motor to trapezius and sternocleidomastoid (SCM) muscles. It does not play a direct role in speech, but examination of these muscles is important in the differential diagnosis of voice disorders. XII: The hypoglossal nerve is purely motor to the tongue muscles including styloglossus, genioglossus, and hyoglossus, whose function is essential for formation of dental, lingual, and guttural sounds.

**Laryngeal Skeleton**

The laryngeal framework consists of cartilage, bone, and membranes invested by a drapery of mucosal lining. On this framework, small muscles act under neural control to position the laryngeal components in a manner that efficiently subserves the functions of breathing, swallowing, and phonation (Figures 2-4). The thyroid cartilage forms the largest external laryngeal prominence. Viewed anteriorly, the cartilage forms a prow-shaped shield with a central ridge below a V-shaped upper margin. The lateral plates extend back to a posterior border and project superior and inferior horns. The inferior horn is tethered
to the posterior cricoid ring, forming a pivot point essential for modulation of high pitch.

**Laryngeal Muscles**

Laryngeal musculature consists of intrinsic and extrinsic subsets. The extrinsics steady the larynx and perform functions of suspension, elevation, and depression of the larynx. Intrinsic laryngeal muscles determine arytenoid movement and VC tensioning (Figure 5).

Extrinsic laryngeal muscles include the infrahyoid strap muscles, the stylohyoid, digastric, mylohyoid, the pharyngeal constrictors, and other minor pharyngeal muscles. Laryngeal intrinsics include CT, posterior cricoarytenoid (PCA), LCA, IA, and TA muscles. The CT originates from the anterolateral cricoid and inserts on the posterior and lateral margins of the thyroid cartilage. This muscle causes a downward rotation of the thyroid cartilage with respect to the cricoid cartilage, pivoting on the lower thyroid horn. This adds an additional tension on the adducted VCs, elevating pitch. The muscle is phasic in firing. The CT is the only intrinsic laryngeal muscle supplied by the external branch of the SLN.

The TA muscle originates from the inner thyroid cartilage and extends posterolaterally to the anterolateral surface of the arytenoid cartilage. The most medial aspects TA are given the name vocalis. TA contraction rotates the arytenoid cartilage medially and adducts the VCs.

The LCA muscle originates from the lateral cricoid cartilage and inserts onto the anterior surface of the arytenoid cartilage.

Intrinsic muscles of the larynx (A and B) and the movement of the VCs caused by their contraction (C). For (C), dashed lines indicate position of VCs and arytenoids before movement by contraction of the muscles (arrows). Solid lines indicate position after contraction. With CT contraction, 1 and 2 indicate movement of the cartilages.
This muscle is the one most directly responsible for the major force on arytenoid internal rotation and VC adduction.

The PCA muscle occupies a depression on the posterior surface of the cricoid lamina, directing fibers toward the lateral arytenoids. PCA contraction rotates the arytenoids externally and abducts the VCs. The PCA fires during inspiration, very strongly with sniffing (Figure 6).

Laryngeal intrinsic muscles, posterior view. Innervation and blood supply are also depicted.

All of the intrinsic laryngeal muscles are supplied by the RLN with the single exception of the CT. This is a very important point in laryngeal electrodiagnosis. Since the superior and RLNs branch from the vagus, denervation on both territories implies vagal neuropathy above the branching point high in the neck. Skull base lesions (e.g., jugular foramen syndrome) should be suspected. Isolated recurrent laryngeal neuopathies spare CT and usually imply pathology below the larynx or in the upper thorax.

**VOICE DISORDERS**

**Central**

Many central disorders affect voice either primarily or secondarily. These include stuttering, essential tremor, cerebellar dis-
Peripheral—Neuropathies

Recurrent Laryngeal Nerve

Neuropathies of the RLN paralyze the intrinsic laryngeal muscles, most importantly those that participate in control of arytenoid rotation. Unilateral lesions cause hoarseness of voice; bilateral lesions result in severe dysphonia. Diplopodia (doubly resonant pitch), voice fatigue, and shortness of breath with exercise are accompanying symptoms. Surgical injury, such as in carotid arterectomy, are obvious causes, but idiopathic cases are common and are thought to be post-viral or immune-mediated neuropathies. Laryngoscopy is a diagnostic standard, but laryngeal needle EMG is often needed to demonstrate nerve injury, as opposed to mechanical or structural cause for the reduced VC movement.

Superior Laryngeal Nerve

The CT muscles, supplied by the external branch of the SLN, rotate the thyroid cartilage and cricoid ring toward one another anteriorly, tensing the VC to produce high pitch. Lesions of the SLN cause nonspecific voice complaint, such as vocal fatigue, diplopodia, and lack of high pitch. Accurate diagnosis can be made only with careful needle EMG demonstrating denervation isolated to the CT, sparing the other intrinsic laryngeal muscles. Most cases of isolated SLN neuropathy are post-viral or idiopathic.

Combined Neuropathies

Proximal neuropathies affecting both the SLN and the RLN cause worse vocal dysfunction as well as reduced sensation in the throat, choking, aspiration, and weakness in swallowing. A careful bedside examination should focus on adjacent cranial nerves. A reduced gag reflex, palate deviation, and wasting of the SCM and trapezius muscles, when found in a dysphonic patient make up the jugular foramen syndrome. Needle EMG of the relevant musculature helps to confirm the physical findings, but skull base imaging to look for a glomus tumor or cervical magnetic resonance imaging with attention to the retropharyngeal region is the next step.

Peripheral—Myopathies

Myopathies

Hoarseness of voice is a common symptom in the inflammatory myopathies, especially inclusion body myositis. Laryngeal and pharyngeal weakness may directly affect phonation or may contribute to laryngeal injury through dysphagia with aspiration. Dystrophies, congenital myopathies, and periodic paralysis may rarely cause direct laryngeal involvement.

Neuromuscular Junction Disorders

Dysphonia in myasthenia gravis (MG) commonly accompanies dysphagia and other bulbar symptoms and implies generalized disease. Isolated vocal symptoms occur only rarely. Pharyngeal and laryngeal symptoms in MG should turn the physician’s attention first to assuring integrity of the airway and breathing. Electrodiagnosis in such cases follows the conventional approach of repetitive nerve stimulation and single fiber EMG, making sure to test proximal muscles.

Amyotrophic Lateral Sclerosis

Motor neuron disease in the form of progressive bulbar palsy commonly presents with dysphonia. Unfortunately, diagnosis is often delayed by successive ear, nose, and throat evaluations and elaborate testing without a thought to the possibility of neuronal disease. Even in experienced hands, the diagnosis is tough to prove when mostly central neurons have dropped out; in that case, even careful needle EMG of tongue, face, and neck muscles shows only a reduced pattern of recruitment.

ELECTRODIAGNOSIS OF LARYNGEAL DISORDERS

Technical Aspects

Walker lists several challenges unique to the performance of laryngeal needle EMG:11

- The laryngeal muscles are neither visible nor palpable.
- Landmarks for needle insertion are the cartilaginous structures of the larynx, identifiable only by palpation.
- Laryngeal muscles are small and thin, and at times voluntary relaxation of laryngeal muscles (like other axial muscles) is difficult.
- Activation of laryngeal muscles is by phonatory maneuvers—the forces of contraction cannot be directly assessed.
- Laryngeal needle EMG is uncomfortable for the patient. Careful technique is needed to avoid coughing, choking, or retching spasms.

Technique

Insertion of a monopolar electromyographic needle or teflon coated blocking needle is easily accomplished in the vocalis, lateral cricoarytenoid, and cricothyroid muscles using the method described by Hiroto and colleagues.7,9 It should be noted that the intrinsic laryngeal musculature cannot be directly
palpated, so that laryngeal cartilage landmarks must be used. Anatomic landmarks guide the direction of the needle insertion. The appearance of insertional activity informs the electromyographer that the electrode has entered muscle. Ultimately, proper placement of the needle electrode is confirmed by recognizing the activation pattern of the muscle. Familiarity with the anatomy can be aided with the use of models, or better still, examination of a cadaver larynx. Examination of a cadaver larynx is an excellent way of learning landmarks and proper direction of the needle electrode (Figure 7). Landmarks can be palpated and the needle inserted. One can section the larynx along the mid sagittal plane and observe whether the insertion was directed appropriately into a vocal fold adductor. It is more difficult in the cadaver larynx to keep the electrode in a submucous location than in actual patients.

To prevent coughing during needle insertion in patients, the needle should be directed so that it remains in a submucous location and does not enter the subglottic space. Injection of a local anesthetic agent into the subglottic space through the cricothyroid membrane (to prevent coughing) is utilized by techniques that do not keep the EMG needle submucous, but this is not necessary if the subglottic space is not entered. Requesting the patient to softly vocalize "E" during insertion, adducts the vocal fold making it easier to avoid penetrating the subglottic space. If the subglottic space is inadvertently entered, coughing will usually ensue, requiring removal of the needle electrode and reinser tion. Another technique for botulinum toxin injection uses an oral approach using a needle at the end of a curved tube directed by indirect laryngoscopy without EMG guidance. This technique is not suitable for electromyographic assessment because the electrode cannot be maintained long enough within the muscle. The submucous percutaneous technique for a full electromyographic assessment is described below. Of course, injection of botulinum toxin (without an attempt at electromyographic assessment) requires only the measures necessary to assure proper electrode placement.

The ground electrode is placed over the forehead, and reference electrode on the chin. Filters of 10Hz-10KHz, and a sweep speed of 10 msec per division are utilized. The patient is coached to perform three different types of vocalization. These included a high-pitched "E", the musical scale, and a vocal click-abrupt relaxation of the adducted vocal folds. The patient lay supine, with slight cervical extension. The thyroid and cricoid cartilages are outlined as landmarks. A 37mm (26-gauge) monopolar electrode is used. The fine (28 gauge) needles are too flexible to direct easily. Longer electrodes (50mm) cannot at times be directed superiorly adequately when sampling the adductors because of lack of room over the sternum.

The cricothyroid muscle is sampled by inserting the electrode a few millimeters to the side of midline at the level of the superior border of the cricoid cartilage, aiming superiorly and laterally (Figure 8). The electrode is directed toward the inferior border of the thyroid cartilage, while the patient vocalizes softly. Care is taken to stay superficial to the cricothyroid membrane: monitoring must be performed to define sharp motor unit activity, which indicates when the electrode is properly located. To distinguish cricothyroid from strap muscles (i.e., sternohyoid and sternothyroid), the patient is instructed to relax without vocalizing and to contract these muscles by lifting the head; motor unit activity appears distant if the electrode is properly placed in the cricothyroid muscle. Care must be taken to avoid the Valsalva maneuver because this can lead to cocontraction of the cricothyroid muscle. Once the cricothyroid muscle has been pierced, relaxation is obtained to observe for spontaneous activity. The muscle can be activated by having the subject perform a musical scale. The number of motor units increases with increasing vocal pitch, with full recruitment normally obtained at the top of the vocal register.

The vocalis (thyroarytenoid) muscle is approached by inserting the electrode through the skin just superior to the cricoid cartilage in the midline and angling superiorly at approximately 60°. This approach allows for easy penetration of the cricoarytenoid junction and provides a direct route to the vocalis muscle.
degrees and laterally approximately 15 degrees, piercing the cricothyroid membrane and proceeding deep to the thyroid cartilage while the subject performs a high-pitched "E" (Figures 9, 10). Vocalization of the "E" adducts the vocal cord and facilitates electrode entrance into the vocalis muscle. The electrode should remain in the submucosa to avoid stimulation of the cough reflex. The muscle is recruited submaximally by vocalization and maximally by performing the vocal click. Full recruitment is not observed in the cricothyroid with a vocal click, or in the vocalis at the top of the vocal register. Once relaxation is obtained, spontaneous activity can be observed. This must be observed (in all of the intrinsic laryngeal muscles) in the presence of minimal motor unit activity occurring in a phasic respiratory pattern. A swallow results in a brief period of inhibition of this activity, although elevation of the larynx can result in disruption of the electrode position. The lateral cricoarytenoid muscle is sampled in a manner similar to the vocalis, except that the electrode is angled approximately 40 degrees superiorly, and 40 degrees laterally (Figures 9a, b). The same vocal maneuvers are used to activate the lateral cricoarytenoid as were used for the vocalis muscle. Vocal maneuvers cannot distinguish vocalis from lateral cricoarytenoid activity. Anatomically, both of these muscles are vocal fold adductors and are not separated by fascia.

The PCA muscle is difficult to sample electromyographically because this muscle lies largely behind the cricoid and arytenoid cartilages. The needle can be advanced from the posterior edge of the thyroid lamina while the other hand gently rotates the thyroid cartilage to the other side. The needle is then advanced through the inferior constrictor muscle to the cricoid cartilage and adjusted until motor units are identified that are maximally activated by a sniff. Alternately, the subglottic space can be anesthetized with 0.5 cc of 1% lidocaine placed through the cricothyroid membrane. A stiff blocking needle can then be inserted through the cricothyroid membrane, just over the cricoid cartilage and advanced in this plane through the subglottic space until it hits the rostrum of the cricoid cartilage, which is impaled. When the electrode emerges through the cartilage, motor units are observed recruited maximally by a sniff. Experiments with a specially fabricated stiff curved needle showed that the PCA could be entered in this fashion, but the fabrication proved to be impractical for routine use. Figure 10 shows the anatomic relationship of the PCA to the laryngeal cartilages.

Electromyography is performed by first observing the muscle at rest, monitoring for spontaneous activity, and observing motor unit morphology and recruitment. Laryngeal muscle motor units are similar in their motor unit action potential parameters to sphincter or facial musculature. Recruitment differs from
facial musculature in that there is often one or two units observed that fire at rest in a respiratory pattern with activation and firing rate maximal at the end of inspiration. These units must not be confused with fibrillations. This activity is usually inhibited briefly after a swallow. Unfortunately, a voluntary swallow will often disrupt needle position. Motor unit action potential parameters and firing rates are assessed at submaximal levels of voluntary recruitment. I have assessed maximal motor unit recruitment using a zero-to four scale. Zero represents no observable motor units, 1 an average of a single motor unit recruited at each needle site within the muscle, 2 two motor units, 3 more than 2 motor units but less than a full interference pattern, and 4 a full interference pattern. Recruitment is also evaluated during the three types of vocalization (high "E", vocal click, and a scale). Normal motor unit action potential parameters are provided in Table 1. It is difficult to maintain electrode position in the PCA to perform this examination, but the CT, TA, and LCA are easily examined.

**ELECTRODIAGNOSTIC FINDINGS IN DISORDERS OF THE LARYNX**

Laryngeal needle EMG should be preceded by a carefully conducted neurological examination and, in most cases, by fiberoptic laryngoscopy in the hands of a trained physician. Conventional electrodiagnostic testing with attention to muscles of the extremities, trunk, neck, tongue, and face may give a quicker and more accurate answer, especially with muscle or neuromuscular junction disorders.

Rodriguez and Simpson identify five uses for laryngeal needle EMG in evaluation and management of dysphonia:

- To aid in the diagnosis of neurogenic lesions affecting the SLN or the RLN.
- To assist in the prognostication of neurogenic lesions.
- To provide information about the degree and extent of nerve damage.
- To guide the surgeon by indication where voluntary control and neurogenic damage are maximal.
- To guide the injection of botulinum toxin in the management of spasmodic dysphonia. Several patterns of dynamic needle EMG characterize disorders of the CNS, so the applicability of these techniques is being extended. In laryngeal dystonia, adductor tone is increased at rest. Strong discharges precede attempted phonation and die out late, reflecting the strained and strangled voice patterns of this disorder. Needle EMG guidance now is the standard for injection of botulinum toxin, a remarkably effective treatment for laryngeal dystonia. Tremor, usually at 4 to 5 Hz can also be nicely displayed be TA needle EMG, but the technique adds no substance to simple bedside voice analysis.

VC paresis is often first detected by laryngoscopy. Distinguishing mechanical and connective tissue causes from neurogenic etiologies is seldom possible by laryngoscopy, so the diagnosis of dysphonia due to laryngeal paresis is greatly en-

---

**TABLE 1. Normal Quantitative Motor Unit Analysis**

(‘Monopolar Electrodes) by selection of individual motor units from seven subjects

<table>
<thead>
<tr>
<th></th>
<th>Amplitude (µV)</th>
<th>Duration (ms)</th>
<th>Phases</th>
<th>Turns</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean SD</td>
<td>mean SD</td>
<td>mean SD</td>
<td>mean SD</td>
</tr>
<tr>
<td>Thyroarytenoid</td>
<td>426</td>
<td>194</td>
<td>3.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Cricothyroid</td>
<td>500</td>
<td>224</td>
<td>4.4</td>
<td>1.6</td>
</tr>
</tbody>
</table>

**Normal Quantitative Motor Unit Analysis from Decompositional Analysis (Monopolar Electrodes) from four subjects**

<table>
<thead>
<tr>
<th></th>
<th>Amplitude (µV)</th>
<th>Duration (ms)</th>
<th>Phases</th>
<th>Turns</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean SD</td>
<td>mean SD</td>
<td>mean SD</td>
<td>mean SD</td>
</tr>
<tr>
<td>Thyroarytenoid</td>
<td>487</td>
<td>191</td>
<td>5.4</td>
<td>2.0</td>
</tr>
<tr>
<td>Cricothyroid</td>
<td>635</td>
<td>322</td>
<td>4.6</td>
<td>0.6</td>
</tr>
</tbody>
</table>

---

**Figure 10.** Lateral view of the larynx showing the relationship between the posterior cricoarytenoid (PCA) and laryngeal cartilages. The curved needle is shown in the PCA but the requirement for special fabrication makes this an impractical clinical method.
hanced by the use of needle EMG. Mononeuropathies of the laryngeal nerves can be documented by needle EMG of the CT and TA of the affected side and by demonstrating normal activity on the unaffected side and in non-laryngeal muscles. When abnormalities of the laryngeal intrinsic muscles are accompanied by ipsilateral denervation in the trapezius and SCM, a jugular foramen lesion is very likely. Laryngeal denervation may also accompany syndromes affecting multiple cranial nerves such as meningeal carcinomatosis or cylindromatosis. Polyradiculoneuritides and polyganglionopathies (e.g., cytomegalovirus) can present with neurogenic dysphonia.

**SUMMARY**

Since speech is our most common method of thought expression and plays so great a part in social interactions, the disorders of voice are very disabling. Neurogenic voice disorders appear rarely in electrodiagnostic practice. Attention is deserved because of the importance of accurately demonstrating localized or regional neurologic dysfunction. Use of conventional techniques in electrodiagnostic medicine can be simply applied, with practice and patience, to the small but most important structure of the larynx.

**REFERENCES**

ELECTRODIAGNOSTIC LARYNGEAL STUDIES

Michael R. Swenson, MD
Arthur A. Rodriguez, MD, MS