REVIEW ARTICLE

Neurolaryngology

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KEYWORDS
Neurolaryngology; Laryngeal electromyography; Laryngeal physiology

Abstract The neuroanatomy of voice and speech is complex. An intricate neural network is responsible for ensuring the main functions of the larynx: airway protection, cough and Valsalva production, and providing voice. Coordination of these roles is very susceptible to disruption by neurological disorders. Neurological disorders that affect laryngeal function include Parkinson’s disease, stroke, amyotrophic lateral sclerosis, multiple sclerosis, dystonia and essential tremor. A thorough neurological evaluation should be routine for any patient presenting with voice complaints suggestive of neurogenic cause. Endoscopic visualisation of the larynx using a dynamic voice assessment with a flexible laryngoscope is a crucial part of the evaluation and ancillary tests are sometimes performed. Otolaryngologic evaluation is important in the diagnosis and treatment of neurological disorders that affect laryngeal function.

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PALABRAS CLAVE
Neurolaringología; Electromiografía laringea; Fisiología laringea

Resumen La neuroanatomía de la voz y el habla es compleja. Una intrincada red neural se responsabiliza de que se ejecuten las principales funciones de la laringe: la protección de la vía aérea, la producción de la tos y el Valsalva y la fonación. La coordinación de esos roles es muy susceptible a verse afectada por enfermedades neurológicas, tales como la enfermedad de Parkinson, los accidentes cerebrovasculares, la esclerosis lateral amiotrófica, la esclerosis múltiple, la distonía y el temblor. Una cuidadosa evaluación neurológica debe ser llevada a cabo en todo paciente que presente síntomas vocales que orienten a una causa neurológica. La visualización endoscópica mediante fibrolaringoscopio, que permita una evaluación dinámica de la voz, es una parte esencial de la valoración y en algunas ocasiones se emplean otras pruebas complementarias. La evaluación otorrinolaringológica es importante en el diagnóstico y el tratamiento de las enfermedades neurológicas con expresión al nivel laringeo.

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Introduction

Neurolaryngology deals with the study of electrical and biochemical events occurring during the execution of the phonatory, respiratory and swallowing functions of the larynx. This new branch of otolaryngology has emerged as a result of progress in the understanding of laryngeal neuroanatomy, neurophysiology and biomechanics. Therefore, neurolaryngology comprises the body of knowledge necessary to understand and treat laryngeal diseases arising due to motor and sensory disorders of the larynx. But neurolaryngology does not only bring together the contributions of biomechanics, neurology, phonology, phonosurgery and speech and language pathology, it also constitutes a new, multidisciplinary field which studies the neural mechanisms of laryngeal functions through various approaches. As such, neurolaryngology is useful not only for understanding disease, but also to offer effective treatments in many cases which are difficult to solve. In fact, recent interest in laryngeal neuroanastomosis techniques, laryngeal reconstruction techniques, laryngeal pacemakers, larynx transplants and neurosurgery of spasmodic dysphonia is a result of the development of neurolaryngology. In general, neurolaryngological disorders are mainly characterised by motor dysfunctions of the larynx, although some diseases are also associated with sensory alterations.

Neurophysiology

The neuroanatomy of voice and speech is complex; there is an intricate neural network which enables the larynx to exercise its primary functions, that is, airway maintenance and protection and the production of cough, Valsalva and voice. The complexity is greater when we consider all the supporting joints and muscles which must be coordinated to produce speech. Humans have a greater voluntary control over laryngeal and articulator muscles than other animal species. Although more primitive, visceromotor, neural pathways are also involved, the human voice has a notable cortical control. Like other voluntary motor systems, the neuromuscular pathway of voice and speech production is constituted by upper and lower neurons, the basal ganglia and cerebellum, myoneural junctions and muscles.

Pyramidal System

The first or upper motor neurons carry signals to initiate a voluntary movement from the cortex to the second or lower motor neurons, which are located in the brainstem or spinal cord. Numerous cortical areas contribute to phonation; movement of the vocal cords is carried out by bilateral cortical signals, so a unilateral lesion of the cortex does not lead to complete vocal cord paralysis. Although the cortex of the left hemisphere is dominant, there is a hemispheric specialisation in which the right side plays an important role in the prosody of speech. The bodies of the neurons of laryngeal nerves are located in the nucleus ambiguus, where they also receive signals from other brainstem nuclei within a complex network of connections supporting different reflexes. Other synapses are recognised in the periaqueductal grey matter, the final pathway of the visceromotor system, which are involved in the involuntary functions of voice such as crying and involuntary screams. Lower motor neurons represent the common final pathway to the effector muscles.

Extrapyramidal System

The basal ganglia and cerebellum are part of the extrapyramidal system, which controls gross motor function, inhibits erratic movements and helps maintain muscle tone. The basal ganglia inhibit rapid discharges from motor neurons, receive inputs from most cortical areas and project towards the frontal cortex, where movement is planned. The cerebellum improves the accuracy of movement by comparing central motor commands with peripheral sensory input, manages vocal self-control and performs fine tuning of airflow and tone. Neurodegenerative processes affecting the extrapyramidal system result in the appearance of abnormal movements such as tremor, dystonia, dysdiadochokinesia and abnormal muscle tone.

Peripheral Innervation

Cranial and spinal motor nerves establish synapses in the myoneural junction of the target muscle. The neurotransmitter acetylcholine is the mediator released in the nerve terminal to trigger the action potential which contracts the muscle. The intrinsic muscles of the larynx are innervated by the recurrent nerve and the superior laryngeal nerve, both of which are branches of the tenth cranial nerve or vagus nerve. Although the intrinsic muscles of the larynx are classically classified into adductors and abductors, their functions are more complex, since antagonistic muscles contract simultaneously during vocal function, showing different muscle activation patterns depending on the purpose of the action. In addition to motor function, the superior laryngeal nerve is also a sensory nerve, as it contains fibres from the sensory neurons of the ganglion nodosum of the tenth cranial nerve. This sensory function is closely related to the protective function of the airway during swallowing. The introduction into clinical practice of the method described by Aviv for measuring laryngeal sensory thresholds revealed the role that the sensitivity of the larynx has in phonatory and swallowing functions. Stimulation of the laryngeal mucosa causes, in addition to sensation, a reflex adductor response of the larynx, which is related to the clinical finding of a pharyngolaryngeal sensory deficit in patients with dysphagia and aspiration.

Laryngeal Manifestations of Neurological Diseases

From our knowledge of the neurophysiology of the upper airway we can infer that neurological disorders may be reflected in many forms in the voice, speech and swallowing functions of patients. Neurological diseases can produce a series of signs and symptoms localised in the upper aerodigestive tract, caused by varied lesions ranging from respiratory muscle weakness due to denervation to oropharyngeal muscle dysfunction, and including direct effects on glottal closure patterns.
but associating a marked dysarthria which may also be accompanied by emotional lability, cognitive impairment and limb involvement with Babinski sign. Lower motor neuron diseases include progressive spinal atrophy, caused by degeneration of cells in the anterior horn of the spine, and progressive bulbar palsy, which is essentially ALS limited to cranial nerve involvement.

**Multiple Sclerosis**

This is a diffuse inflammatory disease of the central nervous system which causes demyelination. Its specific signs and symptoms depend on the affected areas. Approximately 50% of patients may present ENT manifestations such as vertigo, dysphagia, dysarthria and spasmatic voice. These symptoms may show exacerbations and remissions, but with an overall progression of the disease.

**Extrapyramidal Diseases**

Parkinson’s disease, spasmodic dysphonia and vocal tremor are extrapyramidal diseases of interest for neurolaryngology. The first results from the degeneration of dopamine-producing cells in the substantia nigra of the brainstem, which disrupts movement control pathways, and is manifested by the appearance of tremor, rigidity, bradykinesia and postural instability. Over 70% of patients with Parkinson’s disease have voice and speech alterations, with weak and aerial monotonous dysphonia (Fig. 1) due to glottic failure and poor respiratory support, laryngeal tremor, dysarthria and dysphagia. Spasmodic dysphonia is focal dystonia of the larynx, characterised by involuntary muscle contractions or spasms during the execution of movement. Spasms of the vocal cords can take place in adduction, causing a harsh voice with intermittent interruptions (80% of patients) (Fig. 2) or in abduction, causing an arrhythmic and intermittent aerial voice (Fig. 3). The extension of vowels shows characteristic voice breaks in the acoustic level. The pathophysiology of spasmodic dysphonia (SD) is unknown. It is attributed to a dysfunction of laryngeal feedback systems which disinhibits laryngeal muscle action. There have been reports of focal lesions with loss of myelin in the white

**Table 1** summarises the characteristics of various neurological diseases, and the laryngeal findings thereof.

<table>
<thead>
<tr>
<th>Location of Lesion</th>
<th>General Findings</th>
<th>Alterations of Voice and Speech</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper neuron</td>
<td>Spasticity with hyperreflexia and rigidity Babinski sign</td>
<td>Spastic vocal paralysis, paresis, spastic dysarthria, laryngeal myoclonus</td>
</tr>
<tr>
<td>Myoclonus</td>
<td>Weakness, flaccidity, fasciculations</td>
<td>Flaccid vocal paralysis, paresis, glottic failure, hypernasality, flaccid dysarthria</td>
</tr>
<tr>
<td>Extrapyramidal</td>
<td>Tremor, dystonia, dyskinesia, dysdiadochokinesia</td>
<td>Bowing of vocal cords, tremor, laryngeal dystonia, dysdiadochokinesia</td>
</tr>
<tr>
<td>Peripheral nerve</td>
<td>Weakness, atrophy, sensory deficit</td>
<td>Hypomobility or immobility with atrophy</td>
</tr>
<tr>
<td>Myopathy</td>
<td>Weakness, flaccidity</td>
<td>Hypomobility with flaccidity</td>
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</tbody>
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Source: Rubin.  

**Motor Neuron Diseases**

These diseases are characterised by lesions of the upper motor neurons, lower motor neurons or both. Lesions of the upper motor neurons present spasticity of laryngeal and orofacial muscles, with rough and tense dysphonia and notable dysarthria, as well as occasional laryngeal spasms and myoclonus. These lesions are characterised by the Babinski sign. Lesions of the lower motor neurons present flaccid paralysis, muscle atrophy and fasciculations, leading patients to develop an aerial and hypernasal voice, bowing of the vocal cords and ineffective cough. Swallowing is affected by nasal regurgitation and dysphagia by alteration of the oral and pharyngeal phases, often causing aspiration pneumonia. Involvement of both the upper and lower motor neurons characterises amyotrophic lateral sclerosis (ALS), which is an idiopathic, progressive degeneration of both motor neurons with atrophy, fasciculations and muscle weakness. The resulting voice is hypernasal, weak and aerial, with a weak cough. Articulation is slow due to lazy tongue movements which also cause oral dysphagia and tongue fasciculations can be observed. In advanced stages of the disease there is significant alteration of the swallowing function and in terminal stages there is severe dysphagia and aspiration pneumonia of own secretions. Pseudobulbar palsy arises from bilateral corticospinal tract lesions resulting from cerebrovascular events, multiple sclerosis or tumours. Although neuronal bodies are not injured, there are manifestations typical of upper motor neuron lesion, such as muscle spasticity and hyperreflexia, with a rasping and strangled voice resembling spasmodic dysphonia, but not associating a marked dysarthria which may also be accompanied by emotional lability, cognitive impairment and limb involvement with Babinski sign. Lower motor neuron diseases include progressive spinal atrophy, caused by degeneration of cells in the anterior horn of the spine, and progressive bulbar palsy, which is essentially ALS limited to cranial nerve involvement.

![Figure 1](http://www.elsevier.es/) Parkinson syndrome. Narrow-band spectrogram. Extremely aerial voice.
the matter of the corticobulbar tract, which is responsible for central control during voluntary voice production. \(^{15}\) Currently, the diagnosis of SD is based on 3 types of tests: a screening questionnaire, a dynamic exploration of voice and speech and a fibroscopic examination of the larynx. \(^{16}\) A laryngeal tremor known as dystonic tremor may be associated with dystonia. \(^{17}\) This disease is difficult to distinguish from dysphonia due to muscle tension and even from functional dysphonias. Laryngeal tremor is characterised by the presence of regular oscillations of the laryngeal and pharyngeal muscles. The resulting voice is unstable and with frequent breaks. It can be seen as a manifestation of other neurological diseases, such as Parkinson’s disease, ALS and cerebellar pathology. It is often impossible to distinguish it from spasmodyc dysphonia. When there are no other accompanying signs, vocal tremor is usually a manifestation of essential tremor, one of its most common causes. It can occur in isolation as a vocal tremor, but is normally observed together with tremor in the limbs or head. \(^{18}\) Essential tremor is probably as common as Parkinson’s disease, although its prevalence has undoubtedly been underestimated due to its scarce functional effects. It is a postural tremor whose diagnostic criteria are: (a) it appears with the maintenance of a posture; (b) it disappears with rest; (c) it does not interfere with movement, and (d) it is not accompanied by cerebellar or parkinsonian symptoms. It can occur at any age and has no specific neuropathological substrate.

### Cerebrovascular Event

Patients who survive a cerebrovascular event often present varying degrees of disability secondary to motor, sensory and cognitive deficit, as well as alteration of the specific functions of speech, swallowing, walking and writing. Cerebrovascular events in the brainstem can cause vocal paralysis if they affect the nucleus ambiguus, although their isolated presentation is rare. They are usually combined with ipsilateral hemifacial anaesthesia for pain and temperature, contralateral hemihybody anaesthesia for pain and temperature, dysphagia, dysarthria, vertigo, Horner’s syndrome and ataxia, within Wallenberg syndrome due to an occlusion of the posteroinferior cerebellar artery. \(^{8}\)

### Myoneural Junction

Myasthenia gravis is an autoimmune disease characterised by muscle weakness exacerbated by repetitive movement and which improves with rest. The antibodies which cause it attack the post-synaptic acetylcholine receptor at the myoneural junction. Eye muscles are most often affected and laryngeal muscles may be involved in isolation or with other muscular groups. \(^{19}\) In the larynx, the disease is manifested as an abnormal vocal fatigue upon repetitive tasks, often associated with dysarthria and dysphagia of varying severity.

### Peripheral Nerve

Lesions of the vagus nerve or its recurrent or superior laryngeal branches typically appear as vocal paresis or paralysis. Proximal vagal lesions are accompanied by pharyngeal and velopalatal paralysis. Clinically, unilateral recurrent paralysis is manifested as aerial dysphonia with diplopia and aspiration. Superior laryngeal nerve paralysis presents subtler symptoms, such as loss of the highest voice registries, which may go unnoticed in those patients who are not voice professionals. Lesions of this nerve are accompanied by hypoesthesia of the laryngeal vestibule, which can cause aspiration. In some cases it is possible to observe a recovery of peripheral nerve function due to reinnervation phenomena. Such cases do not take place before 4 months of the injury and can cause synkinesias, abnormal muscle function due to non-selective reinnervation of the adductor and abductor muscles. \(^{20}\)

### Myopathies

These diseases may be hereditary and are caused by metabolic or inflammatory processes. There are no cases of isolated laryngeal myopathy, although their existence has been suggested in relation to chronic use of inhaled steroids. \(^{21}\)
Clinical Assessment

History

Neurological diseases can cause vocal symptoms which may be the main reason for consultation. Table 2 summarises the signs which indicate that a vocal disorder has a neurological cause.\(^\text{13}\)

Physical Exploration

General physical examination is an essential step in the diagnostic process: posture, muscle tone, gait and limb trembling are signs to consider. Listening to voice and speech is critical: dysarthria, voice breaks, spasms, aerial voice and vocal trembling are very common perceptual characteristics in neurological diseases. There are specific tasks that can help to identify a neurological disease, some of which are summarised in Table 3. The examination should include an assessment of cranial nerve function, seeking signs which point to the underlying neurological disorder. Endoscopy using a fiberoptic laryngoscope enables a dynamic assessment of phonation by allowing the patient a physiological phonation, thus being preferable to endoscopy using a telelaryngoscope or laryngeal mirror, which requires tongue protrusion and a non-physiological position of the vocal apparatus. It is important to assess symmetry, hyperfunction, tremor, spasms, dysdiadochokinesia and irregular movements.\(^\text{4}\)

Special Tests

Acoustic Analysis

The quantification of short-term alterations has little usefulness in the study of those voice disorders observed in neurolaryngology, since most dysphonia cases are type II and III. These cases present subharmonics and modulations in which the jitter and shimmer parameters and the harmonics/noise ratio are of limited use.\(^\text{22}\) Therefore, in neurolaryngology, signal analysis with spectrograms\(^\text{23}\) and perceptual voice analysis supplemented with the instability parameter (\(I\)) described by Dejonckere added to GRABS are much more useful.\(^\text{24}\) The most notable observation in narrow band spectrograms is the fact that alterations are not constant throughout the sample in most cases, but instead are determined by voice fragments which are qualitatively different from the rest. Perceptually, these correspond to especially strained phonation fragments or voice breaks. The most typical case is that of adductor dysphonia, in which subharmonics are in the phases of vocal spasm, although it is not necessary for subharmonics to exist in order to observe patterns which identify spasms (Fig. 4). Vocal tremor is a regular fluctuation of the intensity and tone of phonation (Fig. 5). In its most severe form it can cause voice breaks with a sudden drop in tone or sudden interruptions in vocal production, which correspond with spasmodic dysphonia, both adductor and abductor. The existence of an intermittent movement of the internal and external intercostal muscles has been observed in these patients and is

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Dysphonia: Signs Pointing to a Neurological Cause.</th>
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<tbody>
<tr>
<td>Vocal fatigue</td>
<td></td>
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<tr>
<td>Vocal tremor</td>
<td></td>
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<tr>
<td>Aerial or weak voice</td>
<td></td>
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<tr>
<td>Vocal tension or interruptions</td>
<td></td>
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<tr>
<td>Resonance alterations</td>
<td></td>
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<tr>
<td>Associated dysarthria</td>
<td></td>
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<tr>
<td>Associated dysphagia</td>
<td></td>
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<tr>
<td>Source: Woodson.(^\text{13})</td>
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</table>

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Dynamic Evaluation of Voice.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vocal Task</td>
<td>Findings</td>
</tr>
<tr>
<td>Count from 1 to 10 in a comfortable tone</td>
<td>Symmetry, hyperfunction, aberrant movement</td>
</tr>
<tr>
<td>Count from 1 to 10 in a high tone</td>
<td>Symmetry, hyperfunction, aberrant movement</td>
</tr>
<tr>
<td>Whistle a song</td>
<td>Symmetry</td>
</tr>
<tr>
<td>Glissando</td>
<td>Symmetry in longitudinal tension, posterior rotation of the larynx, level of the vocal apophysis</td>
</tr>
<tr>
<td>Breathe in quickly through the nose 5 times (sniff)</td>
<td>Symmetry in posterior cricoarytenoid muscle function (abductor)</td>
</tr>
<tr>
<td>Sniff-/i/-sniff-/i/-…</td>
<td>Symmetry, fatigue, abductor and adductor function</td>
</tr>
<tr>
<td>/i/-/hi/-/i/-/hi/-/i/-/hi/-…</td>
<td>Symmetry, fatigue, adductor function</td>
</tr>
<tr>
<td>/pa/-/ta/-ka/-/pa/-/ta/-ka/-…</td>
<td>Dysdiadochokinesia, rigidity, hypertonia</td>
</tr>
<tr>
<td>Sustained phonation of /i/ for stroboscopy</td>
<td>Height of vocal process, amplitude of mucosal wave, muscle tone, presence of masses</td>
</tr>
</tbody>
</table>

Source: Rubin.\(^\text{4}\)

Figure 4 Narrow-band spectrogram. Spasmodic adductor dysphonia.
most likely responsible for the fluctuations in intensity and tone. Interruptions in phonation have been attributed to the presence of spasmodic closure of the glottis, but it may also be true that the opposite phenomenon occurs: a sudden relaxation of the vocal cords, as in spasmodic dysphonia in abduction. Findings in acoustic analysis are positively correlated with the severity of the neurological disorder.\(^2\)

**Aerodynamic Assessment**

Airflow and pressure measurements can provide quantitative data on vocal function. Maximum phonation time is a simple parameter to determine which directly reports on two problems which may coexist: the existence of an insufficient glottic closure and poor respiratory support. Other measurements, such as air flow and phonation ratio, can help guide towards glottic failure rather than a breathing problem.\(^3\)

**Laryngeal Electromyography (L-EMG)**

Although many authors consider this technique as an essential component in laryngeal examination, others do not agree. In fact, there is no agreement as to its methodology, interpretation, validity and clinical applications. There is also a lack of scientific evidence to support its use, since most of the published studies are retrospective, without a double-blind and with a type 4 level of evidence.\(^4\) In a review of 584 articles, Sataloff et al. concluded that the only possible use of L-EMG was for botulinum toxin injection, with other uses not being based on scientific evidence.\(^5\) These conclusions conflict with the clinical use made of L-EMG by some authors, who believe that it is the most objective method for the evaluation of laryngeal neuromuscular function.\(^6\) Based on the results of the metaanalysis by Sataloff et al., a multidisciplinary panel of experts was established in order to explore and identify the parameters to consider in the development of a standard L-EMG methodology intended for use in future, prospective, double-blind studies which investigated the sensitivity, specificity and reliability of L-EMG for neurolaryngological diagnosis. The panel proposed an investigation of the following uses of L-EMG: (a) injection of botulinum toxin; (b) diagnosis of vocal paresis; (c) prognosis of recovery from acute, unilateral, vocal cord paresis or paralysis due to recurrent nerve injury; (d) diagnosis of neuromuscular diseases of the laryngeal muscles; and (e) identification and differential diagnosis of laryngeal movement disorders.\(^7\)

**Sensory Assessment**

The assessment of swallowing by means of a flexible endoscope with a sensory test is a useful tool in the identification of sensory deficits, to evaluate the risk of aspiration, to plan rehabilitation of dysphagia and to design strategies which enable patients with swallowing disorders to be fed with the least restrictive diet possible. This technique explores the sensitivity of the larynx through pressurised air pulses emitted by the tip of a fibrescope pressed against the laryngeal mucosa. Sensory capacity is measured by the threshold required to trigger an adductor reflex. The clinical interest of this test lies in its ability to predict whether a patient with dysphagia is at risk of aspiration.\(^8\)

**Algorithm for the Study of Laryngeal Motor Dysfunction**

Vocal motion abnormalities represent a common clinical problem whose correct diagnosis involves knowing if the disorder is due to neural or biomechanical causes, or both. Answering this question requires the use of endoscopy in combination with electrophysiological techniques and sometimes radiological tests.\(^9\) The diagnostic approach using the following algorithm integrates the various tests and explorations, in order to carry out patient assessment in a fast and efficient manner (Fig. 6).

The assessment should begin by endoscopic examination through videostroboscopy during normal breathing, during phonation of the vowel /i/ and performing a glissando. In patients with associated swallowing disorders, velopharyngeal function and epiglottic movement should be assessed by transnasal fiberoptic laryngoscopy whilst producing the phoneme /k/. Once inflammatory or tumoural laryngeal lesions have been ruled out, patients can be classified into 4 groups: (a) normal mobility; (b) limited mobility, when there is a lower speed in the opening or closing movement of the vocal cord or when a reduced lateral movement is observed; (c) immobility, when the vocal cord remains at the same point during the respiratory and phonatory phases; and (d) hyperactive movement, when there is a phase difference in vocal cord movement during the respiratory or phonatory phases and persistence in the vocal approach or separation position. This type of behaviour is associated with spasmodic dysphonia.

For those patients with impaired mobility, the next step in the evaluation process would be laryngeal electromyography (L-EMG), as detailed by Yin.\(^5\) The basic test battery includes assessment of the two major laryngeal muscles, the thyroarytenoid and the pars recta of the cricothyroid. The posterior cricoarytenoid muscle is also studied in cases of bilateral vocal dysfunction and the palatopharyngeal muscle in cases which associate nasal regurgitation of food.

If the L-EMG detects a neuropathic pattern, patients are classified into cases of paralysis or paresis. They then undergo electromyographic monitoring for 3–6 months, in order to monitor neuropraxia (temporary blocking of neural transmission), reinnervation (neural lesion with the appearance of new nerve fibres and recovery of function) and denervation (permanent neural lesion). In cases of reinnervation, monitoring is normal and surgery is not considered. In cases of denervation, an aetiological study is conducted...
or surgical treatment is considered (injection, thyroplasty or reinnervation). Patients in whom L-EMG detects a myopathic pattern are referred to a neurologist in order to rule out the existence of systemic disease or local inflammation. The combination of a history of orotracheal intubation, vocal paresis or paralysis observed by stroboscopy and a normal L-EMG points to a diagnosis of laryngeal joint alteration, such as arytenoid dislocation and ankylosis or synechiae of the posterior commissure.

Neurolaryngological Treatment

Treatment of Spasmodic Dysphonia

Although there is a reasonable consensus on the use of botulinum toxin in spasmodic dysphonia, the best form of administration has not been defined. A survey aimed at North American specialists found that most used a dosage which usually started with 2.5 U of botulinum toxin into each thyroarytenoid muscle, injected through the cricothyroid membrane under EMG control, with or without fibroendoscopic aid, every 3 or 4 months according to clinical evolution. There is a dose range between 1.25 and 4.25U total in which the most prolonged vocal improvement is obtained with the shortest adverse effects. The price of botulinum toxin has led some experts to freeze the remainder of the reconstituted vial, since there have been no reports of its effects being altered. The greatest limitation of this therapy is the short period for which patients enjoy its effects, that is, between the disappearance of the adverse effects after injection and the disappearance of toxin effect. Surgical methods for the treatment of spasmodic dysphonia are less widespread than botulinum toxin. In 1976, Dedo noted that the transient paralysis of the recurrent nerve induced by lidocaine alleviated the symptoms of spasmodic dysphonia. He tested nerve section with unequal results, so this technique has been discontinued. These results led to selective denervation techniques of the terminal branches of the recurrent laryngeal nerve, with or without myectomy of the thyroarytenoid muscle. In addition, there have also been tests of laryngeal surgery, specifically type II thyroplasty, aimed at reducing spasm intensity.

Laryngeal Reinnervation

Various surgical procedures for the reinnervation of paralysed vocal cords have been described using the ansa cervicalis, phrenic nerve, sympathetic preganglionic neurons, hypoglossal nerve and nerve-muscle pedicles. The main purpose of these procedures is to prevent atrophy of laryngeal muscles due to denervation. Restoration of the mucosal wave and recovery of voice quality have been described after reinnervation with ansa cervicalis, although with occurrence of synkinesias. Nerve grafts from the normal muscle to the paralysed muscle have been employed in order to resolve these synkinesias and restore movement to the paralysed vocal cord. This technique has achieved reinnervation of the thyroarytenoid muscle and regained motion.
of the vocal cord adductor in experimental animals. Tucker\textsuperscript{39} reported recovery of adduction in a paralyzed vocal cord using a nerve-muscle pedicle obtained from the prelaryngeal muscles. This technique has recently been combined with arytenoid adduction, with excellent vocal results.\textsuperscript{40} In selected cases of severe dysphagia following central neurologic lesions there have been reports on the usefulness of sensory nerve transposition techniques, mainly during the surgical rehabilitation of patients with dysphagia by microneuromraphy between the greater auricular nerve and the superior laryngeal nerve.\textsuperscript{41}

**Laryngeal Pacemaker**

The functional electrical stimulation of the larynx or laryngeal pacemaker is still a subject of interest as a potential therapeutic option in vocal paralysis.\textsuperscript{42} Such systems have been used to restore motor function in patients with spinal lesions, in the control of heart rate and to restore sensory functions, such as in cochlear implants. In the case of laryngeal pacemakers, and contrary to heart pacemakers, an afferent arm is required in order to provide the necessary information for an efferent arm to stimulate muscles at an effective and appropriate rate.\textsuperscript{43} The efferent arm must be connected to the vagus nerve or the recurrent nerve if the latter is intact. In addition, it may also contact the denervated muscle directly, thus avoiding dependence on the attainment of a correct growth of axons which takes place after neurorrhaphy. Laryngeal pacemers have been implanted in patients suffering bilateral vocal cord paralysis, with decannulation being achieved in half of the cases.\textsuperscript{44,45} Moreover, they have also been used to restore the airway protection function of the larynx.\textsuperscript{46}

**Gene Therapy**

There are numerous growth factors that promote neuronal survival and axonal growth. So far, the gene for IGF-I has been introduced into a non-viral vector in experimental animals, showing a greater likelihood of reinnervation and reduced muscle atrophy compared to animals not receiving the gene.\textsuperscript{47,48}

**Conflict of Interests**

The authors have no conflicts of interest to declare.

**References**