Chapter 110: Neurolaryngology

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Laryngology has been a discipline primarily involved in examination and identification of anatomic abnormalities, tumors, cysts, or paralysis of the vocal cords. Until recently, little has been taught regarding dysphonia related to disorders of movement of the larynx. Many of the disorders of motion are neurologic in origin.

Patient History

As with other disorders, the history of the patient's disorder is vital. The circumstances of the development of the dysphonia and/or dysphagia are important, that is, whether the symptom is persistent or intermittent, when the voice is normal, and whether the patient has a normal laugh, cry, singing voice, humming voice, or yawn. Does the patient have dyspnea, shortness of breath, stridor? Is swallowing normal for liquids, solids, or all foods? Is there any sign of dysarthria? Are there activities that can make the symptom disappear? Does the patient have weakness, tremor, or spasms of other body parts? Has the patient taken medications (such as phenotiazines, which may cause tardive movement disorders)? Has the patient had head trauma, surgery, laryngeal trauma, or endocrine disease symptoms (particularly of the thyroid)? The history of patients who have neurologic disabilities of the larynx should emphasize changes in phonation, respiration, and swallowing.

Physical Examination

Examination of the morphology of the larynx and information about vocal cord mobility can easily be obtained using indirect laryngoscopy. This method tethers the tongue and hyomandibular complex, interfering with speech. Therefore this method is relatively useless in the diagnosis of movement disorders of the larynx. The fiberoptic laryngoscope allows direct visualization of the laryngeal function during speech without having to interfere with the tongue and hyomandibular complex. Respiration, cough, phonation, and swallowing can all be visualized. Altered motion, discoordination, tremor, and spasm are often readily apparent utilizing fiberoptic laryngoscopy. The procedure can be carried out painlessly with minimal or no topical nasal anesthesia.

A video camera is an invaluable adjunctive tool for the neurolaryngologist. The entire patient examination can be recorded to allow review and more precise, thoughtful classification. It also allows accurate record keeping for comparison over time or after therapy. For certain conditions, a video printer can be utilized to produce hard copy images for inclusion in a patient record. The single-image and video-image records are also useful in patient education about the patient's condition and the proposed therapy and potential hazards. In addition, the video image has been used as a biofeedback method in voice and singing therapy (Brewer and McCall, 1974; Davidson et al, 1974; Yanagisawa, 1992).

Videolaryngeal stroboscopy adds yet another dimension to examining, documenting, and understanding movement disorders of the larynx (Alberti, 1978; Hirano et al, 1985; Kitzing, 1985; von Leden, 1961). The stroboscope produces intermittent flashes of light. If these flashes are at the same frequency as the movement of the vocal cords the stroboscope

will produce a frozen image. If the frequency of the light is slightly less than that of the vocal cord motion, there is a phase delay of the consecutive light flashes, producing a slow-motion effect. The strobe image is therefore a visual averaged signal over many vibratory cycles. Stroboscopy results in an estimate of the abnormal motion or vibration. The parameters that can be measured using the stroboscope include the fundamental frequency, the regularity or periodicity of successive waves, the symmetry of the vocal fold motion, the glottic closure, the amplitude of the horizontal excursion, tremor, and the character of the mucosal wave (Hirano, 1992; Sessions, 1992).

Photoglottography and electroglottography have also become important tools for the neurolaryngologist. Photoglottography is performed by shining a light on the larynx. The amount of light that passes through the glottis is proportional to the degree of glottic opening. The light transmission can be measured by a photosensor overlying the subglottic neck skin (Baken, 1987; Hanson et al, 1983; Kitzing and Lofquist, 1979).

Electroglottography (EGG) is a technique based on a change of electrical resistance related to movement of the vocal cords. Low-voltage electrical current is applied through surface electrodes placed on the skin overlying the thyroid alae. The sensors then measure a change in the electrical resistance that is representative of the surface area of contact of the vocal folds. Changes in the EGG amplitude and duration of the opening and closing phase may characterize abnormal function. The EGG is suggestive of a pathologic condition by variance from normal rather than pathognomonic signs (Baken, 1987; Fourcin, 1981; Hanson et al, 1983; Kitzing, 1982).

Laryngeal electromyography (EMG) has become a more important part of the diagnostic process in motor disturbances of laryngeal function. Diagnostic distinctions can be made between neuropathy, anterior horn cell disease, brain stem lesions, myopathy, and neuromuscular transmission disorders. Electromyography of the larynx can also be useful in determining whether an immobile vocal cord is related to paralysis or mechanical limitation. The electromyogram may be prognostic for the recovery of a paralyzed vocal cord.

Among techniques available for EMG are surface noninvasive, direct laryngeal muscle sampling by laryngoscopy, percutaneous needle electromyography, indwelling wire and hooked wire electrode placement, and stimulation of the recurrent laryngeal nerve. The hooked wire technique will not change position with motion and therefore allows the possibility of continuous recordings over a period of time with different activities. Utilizing digital computer software programs, the rate, amplitude, and duration of the motor units can be averaged to identify variation.

Quantitative measures of voice production (that is, fundamental frequency, perturbations, amplitude, harmonics, shimmer and jitter, and tremor) are available for the diagnosis of vocal pathologic conditions. These measurements can safely and easily be made using spectrography and digital computer program. Measurements of airflow during phonation can also give information about hypoadduction or hyperadduction. Some of the dysphonias seen, such as those associated with parkinsonism, are actually related to poor presentation of air to the sound generator and not an intrinsic laryngeal disability (Baken, 1987; Gramming and Sundberg, 1988; Hirano et al, 1988; Horii, 1975, 1979).

Vocal Cord Paresis/Paralysis

When there is alteration of voice and the visual examination shows vocal cord immobility, a differentiation between vocal cord fixation and vocal cord paralysis must be made. Electromyography is most useful in establishing denervation. Palpation of the arytenoid via a direct laryngoscopy, with visualization of motion at the cricoarytenoid joint, can also be utilized to observe for fixation.

Vocal cord paralysis is usually classified by either the site of the lesions (supranuclear, bulbar, peripheral nerve, or muscle) or the nature of the disorder (inflammatory, neoplastic, traumatic, postsurgical, or idiopathic) (Brin and Younger, 1988; Younger et al, 1992).

Lesions of the cortex or supranuclear corticobulbar pathways uncommonly affect the larynx. Lesions from the medulla to the muscle are more common and generally produce a flaccid paralysis. Motor and sensory losses may be seen in patients who have had a focal stroke, syringobulbia, multiple sclerosis, cranial trauma, or intermedullary tumors. However, in motor neuron disease (amyotrophic lateral sclerosis) or poliomyelitis, only the motor tracts are involved (Younger et al, 1992).

Motor neuron disorders are characterized by wasting, weakness, and fasciculations. This may be associated with bilateral corticospinal tract signs including progressive spinal muscular atrophy, progressive bulbar palsy, and amyotrophic lateral sclerosis. In progressive bulbar palsy, there is slurring of speech and hypernasality because of weakness of the tongue and palatopharynx. In amyotrophic lateral sclerosis there is a combination of spastic (supranuclear) and flaccid (lower motor neuron) dysfunction (Tucker, 1980; Younger et al, 1992).

Laryngeal dysfunction can be seen with Arnold-Chiari malformations producing choking, apnea, or aspiration. Laryngeal weakness may also be seen i patients with lateral medullary strokes (Wallenberg's syndrome). These patients have an ipsilateral loss of pain and temperature on the face and a contralateral loss on the trunk and extremities. They may also have vertigo, nausea, vomiting, dysphonia, dysphagia, ipsilateral Horner's syndrome, and paresis/paralysis of the pharynx and larynx (Younger et al, 1992).

Peripheral paralysis of the laryngeal nerves is usually caused by inflammatory, metabolic, neoplastic, compressive, traumatic, or idiopathic causes. Lesions at the skull base usually affect cranial nerve IX, X, and XI. The more central the lesion, the more disabling is the resultant weakness. Skull base lesions produce not only a vocal cord paralysis, but also a pharyngeal paralysis and a loss of sensation. Therefore aspiration is usually a consequence (Younger et al, 1992).

Diabetes, lupus, rheumatoid arthritis, polyarteritis nodosa, drug toxicity, chronic alcoholism, sarcoidosis, and tuberculosis may cause a laryngeal neuropathy, although there is usually involvement in other organs as well. Guillain-Barre syndrome (acute inflammatory polyradiculoneuritis) causes demyelination of peripheral nerves and may well affect the recurrent laryngeal nerves. These patients are at risk for aspiration but usually have a spontaneous recovery (Asbury, 1981; Hughs et al, 1981; Younger et al, 1992).

Myasthenia gravis and Eaton-Lambert syndrome are examples of disorders of the neuromuscular junction. Patients produce antibodies to acetylcholine receptors and frequently have fluctuating ocular or oropharyngeal weakness, often with limb weakness. The diagnosis is made with injections of edrophonium, repetitive nerve stimulation tests, electromyography, and acetylcholine receptor antibody titers (Schmidt-Nowara et al, 1984; Younger et al, 1992).

Many primary muscle disorders can cause laryngeal dysfunction, including toxic and metabolic myopathies, polymyositis, dermatomyositis, and the muscular dystrophies (Younger et al, 1992).

Patients who have a vocal cord paralysis should also have an examinatio of their thyroid to look for an occult carcinoma that may have destroyed the recurrent laryngeal nerve. Chest x-rays and computerized tomographic (CT) scans will evaluate the possibility of a thoracic tumor destroying the recurrent laryngeal nerve. Skull base x-ray films should also be taken to look for a lesion affecting the vagus nerve at its exit from the cranium (Titche, 1976; Tucker, 1987; Tucker and Lavertu, 1992).

In patients with a mechanically fixed vocal cord, the voice may be relatively normal if the cord is fixed in the midline, but they may have dyspnea on exertion. If the vocal cord is fixed in a more lateral position, the patient will have a brethy, weak voice with little or no dyspnea.

A unilateral recurrent laryngeal nerve paralysis usualy produces a breathy voice, which at times has diplophonia. The patient may complain of shortness of breath when speaking, caused by air escape. Careful inspection, as well as additional testing, is often necessary, since visual examination may show some motion related to cricothyroid and/or interarytenoid function (Titche, 1976).

A unilateral superior laryngeal nevre paresis/paralysis usually produces easy fatigability, diminished high-pitch production, and diplophonia. Since the vocal folds may be vibrating at different tensions, they may produce different frequencies of sound, thereby the diplophonia. These weaknesses are most often related to either surgical trauma (for example, thyroid surgery) or presumed viral neuropathy. Approximately 60% of patients with isolated superior laryngeal nerve weakness will recover function spontaneously within 1 year. Many of those who do not, learn to compensate with normal or near normal voice (Abelson and Tucker, 1981).

Some patients may present with bilateral vocal cord paralysis. These patients will have a breathy or aphonic voice and a variable degree of stridor. They may develop life-threatening airway compromise. Many of these patients have been misdiagnosed as having asthma, bronchitis, or abductor spasmodic dysphonia (Tucker and Laveru, 1992).

Motion Disorders of the Larynx

Patients may have dysphonia related to abnormal motion of the vocal cords (that is, tremor, dyssynchrony, hypoadduction, or hyperadduction). Patients are classified as having a movement disorder if they have a disorder of motor programming resulting in either a paucity of movement (akinesia or bradykinesia), excessive movement (hyperkinesia), or a

combination thereof (Table 110-1).

Table 110-1. Movement disorders of larynx

Movement disorder	Bradykinetic	Hyperkinetic
Parkinsonism	X	
Chorea	X	X
Essential tremor		X
Dystonia		X
Stuttering		X
Myoclonus		X
Tics (Tourette's syndrome)		X
Tardive dyskinesia		X.

Parkinsonism

A common example of a motion disorder that may affect the larynx is *parkinsonism.* This is a neurologic syndrome manifested by any combination of tremor at rest, rigidity, bradykinesia, and loss of postural reflexes (Fashn, 1986; 1989). At least two of these four cardinal features should be present before the diagnosis of parkinsonism is made. There are many causes of parkinsonism, and they can be divided into three major categories: idiopathic, symptomatic, and parkinsonism-plus disorders (see box). The specific diagnosis depends on details of the clinical history, the neurologic examination, and laboratory tests. Tremor is usually the first symptom recognized by the patient; however, the disorder can begin with slowness in movement or shuffling gait. In the early stages, the symptoms and signs tend to remain on one side of the body, but with time, the other side slowly becomes involved as well (Fahn, 1986, 1989a).

Box: **Classification of parkinsonism**

1. Idiopathic (primary): Parkinson's disease
2. Symptomatic (secondary)
    a. Drugs - neuroleptics
    b. Postencephalitis
    c. Toxins - manganese, carbon monoxide, cyanide
    d. Vascular
    e. Brain tumor
    f. Head trauma
3. Parkinsonism-plus syndromes
    a. Progressive supranuclear palsy
    b. Multiple system atrophy
        (1) Striatonigral degeneration
        (2) Shy-Drager syndrome
        (3) Olivopontocerebellar degeneration
    c. Dementia syndromes
        (1) Alzheimer's disease
        (2) Normal-pressure hydrocephalus

d. Hereditary disorders
    (1) Wilson's disease
    (2) Huntington's disease
    (3) Hallervorden-Spatz disease.&

Tremor is present in the distal parts of the extremities and the lips while the involved body part is "at rest" (see box). "Pill-rolling" tremor of the hands is the most typical. The tremor ceases with active movement of the limb. Bradykinesia is manifested by masked facies; decreased blinking; drooling of saliva from decreased spontaneous swallowing; loss of spontaneous movement such as gesturing; smallness and slowness of handwriting (micrographia); difficulty with hand dexterity for shaving, brushing teeth, and putting on make-up; short-stepped, shuffling gait with decreased arm swing; and difficulty arising from a chair, getting out of automobiles, and turning in bed. Rigidity is an increased resistance to passive movement, is equal in all directions, and usually is manifested by a ratchety "give" in the range of motion, so-called *cogwheel rigidity*. Depression is a frequent feature in patients with Parkinson's disease. Dementia occurs in about 10% ofr patients with Parkinson's disease. More common is bradyphrenia, in which the patient is not demented but slow in responding to questions (Fahn, 1986).

Box: **Criteria for diagnosis of Parkinson's disease**

Inclusions

At least two of the following:

Tremor at rest
Rigidity
Bradykinesia
Loss of postural reflexes

Exclusions

History of:

Encephalitis
Exposure to carbon monoxide, manganese, MPTP, or other toxins
Recent exposure to neuroleptic medication

Onset of parkinsonian symptoms after:

Head trauma
Stroke

Presence on examination of:

Cerebellar ataxia
Loss of downward ocular movements
Pronounced postural hypotension not caused by concurrent medication

Vocal cord paralysis

Magnetic resonance imaging or computed tomography of head reveals:

Lacunar infarcts
Capacious cerebral ventricles
Cerebellar atrophy
Atrophy of midbrain or other parts of brainstem

Failure to respond to levodopa therapy.&

In Parkinson's disease, speech production is compromised because of hypokinetic dysarthria with poor presentation of air to the vocal apparatus (sound generator) as a result of decreased flow associated with a bradykinetic bellows mechanism. The dysphonia is characterized by a decreased loudness with monopitch, monoloudness, and prosodic insufficiency. Voice is decreased in loudness and tends to fade out at the end of breath groups. Breath groups are shortened, and pauses for breaths may occur at inappropriate times. Speech is produced in short rushes with inappropriate silences between words and syllables. Articulation is produced with reduced range of articulation for both lingual and labial sounds (Darley et al, 1975; Ramig et al, 1988).

Laryngoscopy often reveals bowing of the vocal cords with a midcord opening of the glottis on phonation. The vocal cord motion is often slowed. There may be pooling of secretions in the hypopharynx.

The vocal cords are not infrequently paralyzed in multiple system atrophy, such as in striatonigral degeneration or in Shy-Drager syndrome (Fahn and Greenberg, 1972). When paralysis is severe, tracheostomy may be required. There may also be a sensory aberration causing a diminished or absent cough reflex and intermittent aspiration. The respiratory laryngeal function may deteriorate during sleep with stridor and obstructive, at times life-threatening, events (Kavey and Whyte, 1992.

Dystonia

Dystonia is a movement disorder characterized by sustained muscle contractions frequently causing twisting and repetitive movements or abnormal postures that may be sustained or intermittent. Dystonia can involve any voluntary muscle. Because the movements and resulting postures are often unusual, and the condition is rare, it is one of the most frequently misdiagnosed neurologic conditions. Focal dystonia symptoms involve one small group of muscles in one body part, segmental disease involves a contiguous group of muscles, and generalized dystonia is widespread. Common examples of focal dystonia (see box) include blepharospasm (forced, involuntary eye closure), oromandibular dystonia (face, jaw, or tongue), torticolis (neck), writer's cramp (action-induced dystonic contraction of hand muscles), and spasmodic dysphonia (vocal cords) (Fahn, 1989b). Aronson (1985) distinguished and reviewed two types of spasmodic dysphonia: *adductor*, caused by irregural hyperadduction of the vocal folds; and *abductor,* caused by intermittent abduction of the vocal folds. Patients with *adductor* spasmodic dysphonia exhibit a choked, strained-stranged voice quality with abrupt initiation and termination of voice resulting in short breaks in phonation.

The voice is generally reduced in loudness and monotonal. Vocal tremor is frequently observed along with a slow speech rate and decreased smoothness of speech. Patients with *adductor* spasmodic dysphonia exhibit a breathy, effortful, voice quality with abrupt termination of voicing resulting in aphonic widespread segments of speech. The voice is reduced in loudness, and vocal tremor is frequently observed (Aronson, 1985; Aronson and Hartman, 1981).

Box: **Classification of dystonia**

I. Age at onset
- A. Infantile (< 2 years)
- B. Childhood (2 to 12 years)
- C. Adolescent (13 to 20 years)
- D. Adult (> 20 years)

II. Etiology
- A. Primary
  - 1. With hereditary pattern
    - a. Autosomal dominant
      - (1) Classic types
        - (a) Childhood-onset dystonia
        - (b) Focal dystonia
      - (2) Variant types
        - (a) Dopa-responsive dystonia
        - (b) Myoclonic dystonia
    - b. X-linked recessive
  - 2. Sporadic (without a documented hereditary pattern)
    - a. Classical types
    - b. Variant types
- B. Secondary
  - 1. Associated with other hereditary neurologic disorders, for example, Wilson's disease, Huntington's disease, ceroid lipofuscinosis
  - 2. Environmental, for example, posttraumatic, postinfectious, vascular, tumor, toxic, phenothiazines (tardive)
  - 3. Dystonia associated with parkinsonism
  - 4. Psychogenic

III. Distribution
- A. Focal
  - 1. Blepharospasm (forced, involuntary eye closure)
  - 2. Oromandibular dystonia (face, jaw, or tongue)
  - 3. Torticollis (neck)
  - 4. Writer's cramp (action-induced dystonic contraction of hand muscles)
  - 5. Spasmodic dysphonia (vocal cords)
- B. Segmental (cranial/axial/crural)
- C. Multifocal
- D. Generalized (ambulatory, nonambulatory).&

Blitzer et al (1985) noted that spasmodic dysphonia is not a "spastic" disorder, electromyographic characteristics were inconsistent with those seen in pyramidal disorders. We found an irregular tremor in 25% of patients as opposed to the regular tremor of essential tremor. Dystonic movements can be rapid and repetitive, and tremor may be seen in dystonia affecting any segment of the body. Dystonic tremors are typically irregular and have a directional preponderance; symptoms are increased when the patient postures the affected body part in a position opposed to the primary dystonic contractions. Many patients with spasmodic dysphonia have an irregular vocal tremor that is both audible and can be recorded electromyographically (Blitzer et al, 1985).

Finitzo and Freeman's detailed studies (1989) support an organic basis for the condition: 35% of patients had abnormal brainstem auditory evoked responses; 47% had an abnormal gastric acid secretory response to sham feeding (an index of vagal nerve function); 46% had reduced or absent vagally mediated fluctuations in heart rate during deep inspiration; women with spasmodic dysphonia had abnormal limb motor control; 23% had brain lesions when imaged with magnetic resonance imaging (MRI) scans; 56% had abnormal brain electrical activity mapping (BEAM) scans; and 76% of patients had abnormal brain hypoperfusion on single-photon emission computerized tomography (SPECT).

A percutaneous EMG-guided needle technique has been used to inject *Clostridium botulinum* toxin into the thyroarytenoid muscle for relief of the symptoms. Our group has injected bilateral thyroarytenoid muscles in over 300 adductor patients and posterior cricoarytenoid muscles in over 40 abductor laryngeal patients. The results have been gratifying, with few side effects (Blitzer and Brin, 1991; Blitzer et al, 1988, 1989; Brin et al, 1988, 1989, 1992b).

Essential tremor

Another movement disorder that frequently affects the larynx is *essential tremor*. According to Gresty and Findley (1984):

The appearance of tremor is that of a rhythmical movement of a part of the body. The appearance implies that the movement has a relatively fixed periodicity and possesses an amplitude and wave-form which are to some extent invariable over reasonable amounts of time. If these characteristics do not hold then movements have an irregular appearance and historically have been classified as different phenomena.

Tremor has been defined as "a series of involuntary, relatively rhythmic, purposeless, oscillatory movements" (DeJong, 1967) that have been observed in both distal and proximal musculature. The proposed underlying neural bases for tremor include a central (Hunker and Abbs, 1984; Walsh, 1969, 1979) and peripheral (Rack and Ross, 1986) mechanism. Rest tremors occur in relaxed, unsupported limbs, and action tremors occur during muscle contraction. Action tremors have been classified further as postural tremors (when holding a position against gravity), contraction tremor (produced by isometric voluntary contraction independent of gravity, such as making a fist), and kinetic or intention tremor (during goal-directed movement such as touching finger to nose) (Jankovic, 1984). The involuntary, rhythmic, oscillatory movements that affect the distal musculature in patients with tremulous diseases may also affect the muscles of the speech production mechanism and generate

rhythmic alterations in pitch and loudness called *vocal tremor*. Vocal tremor may result in rapid decreases and increases in loudness and pitch or in complete phonation stoppages. Intelligibility and rate of speech may be decreased. Vocal tremor has been described perceptually as "tremulous voice" (Brown and Simonson, 1963), "wavy voice" (Hartman et al, 1983), or "tremulous, quavering speech" (Koller et al, 1985) and has been associated with neurologic disorders such as essential tremor, Parkinson's disease, cerebellar ataxia, and flaccid dysarthria and dystonia (Aronson, 1985). The frequency, amplitude, and regularity of vocal tremor may differ among diseases of different neural subsystems (Aronson, 1985; Lebrun et al, 1982; Ludlow et al, 1986).

Analysis of vocal tremor may make important contributions to early and differential diagnosis of neurologic diseases and consequently to treatment decisions. The primary noninvasive quantification of vocal tremor has been through acoustic analysis. Most of the acoustic data on vocal tremor have been obtained from visual inspection of oscillographic displays of waveform data (Brown and Simonson, 1963; Hachinski et al, 1975) or graphic level recorder displays of amplitude contours (Hartman et al, 1983; Massey and Paulson, 1982) of sustained vowel phonation. Consequently the bulk of acoustic data on vocal tremor includes only visually quantifiable amplitude oscillations. Vocal tremor occurs in approximately 10% to 20% of patients with essential tremor (Lebrun et al, 1982). It may be the first (Brown and Simonson, 1963) or only (Massey and Paulson, 1982) sign of the disease, or it may accompany tremors in other body parts (Findley and Gresty, 1988). Vocal tremor may parallel the onset of other symptoms or have a sudden onset and cause rapid deterioration in speech intelligibility (Brown and Simonson, 1963; Findley and Gresty, 1988). It has been reported that vocal tremor is greater with emotional stress or fatigue (Aronson, 1985). Pitch breaks (octave breaks to a lower frequency) and phonation arrests have been reported in certain cases of essential tremor (Lebrun et al, 1982; Meeuwis and Baarsma, 1985) and have been associated with visible vertical oscillations of the larynx (Darley et al, 1975). We in our practice, and others have found limited clinically significant changes in vocal tremor with administration of propranolol (Duvoisin, 1984; Hartman and Vishwant, 1984; Koller et al, 1985). We have recently begun using local injections of Botox in the treatment of essential vocal tremor with a dramatic benefit noted in our preliminary series.

Myoclonus

Myoclonus is a movement disorder. Myoclonus refers to sudden, brief, shocklike involuntary movements caused by muscular contractions (positive myoclonus) or inhibitions (negative myoclonus, "asterix") arising from the central nervous system (Fahn et al, 1986; Marsden et al, 1982; Young and Shahani, 1986). Laryngeal involvement can become problematic in patients with myoclonic involvement of the primary laryngeal structures or muscles of respiration. Because of the irregular and often unpredictable nature of the myoclonic movements, aspiration can occur (Brin and Younger, 1988). "Branchial" or "oculo-palatal" myoclonus refers to myoclonic symptoms affecting cranial structures. When peering into the oral cavity, the movements are characterized by involuntary, usually unconscious, movements of the soft palate and pharynx (Lapresle and Ben Hamida, 1970). Further exploration will often document synchronous jerks affecting the eyes, face, palate, larynx, diaphragm, neck, shoulder, and arm, giving rise to the syndrome of "myoclonies velo-pharyngo-laryngo-oculo-diaphragmatiques" (Guillain, 1938).

The pharyngeal movements are most commonly bilateral and symmetric but can be unilateral with the palate and uvula being drawn to one side. The rhythmicity nearly always persists in sleep, usually between 1.5 and 3 Hz, with a range of 0.3 to 10 Hz (Lapresle and Ben Hamida, 1970); variation with respiration has been documented (Dubinsky and Hallett, 1988). Laryngeal involvement may produce a broken speech pattern, simulating the speech pattern heard in laryngeal dystonia or tremor (personal observation). Examination of the vocal cords often shows slow rhythmic adduction and abduction of the vocal cords at the same timing and frequency as the palatal, pharyngeal, and occasional diaphragmatic contractions. This causes the broken speech pattern and a respiratory dysrhythmia. Ventilaty dysfunction has been documented (Andrews et al, 1987).

Stuttering

Stuttering is a neurologic disorder that includes abnormal, involuntary, and inappropriate use of the muscles of speech production, resulting in dysfluency. Freeman measured muscle activity during fluent and nonfluent utterances. Analysis revealed that stuttering is accompanied by high levels of laryngeal muscle activity and disruption of normal reciprocity between abductor and adductor muscle groups. The results have been interpreted to demonstrate a strong correlation between abnormal laryngeal muscle activity and stuttering. Fiberoptic examination of stutters often reveals glottal blocks during the stutter. Motor abnormalities in other body segments have been documented in stutter patients (Brin et al, 1992a).

Other motion disorders

Other motion disorders that may produce abnormal laryngeal function include the tic disorders (including Tourette's syndrome), the choreic disorders (including Huntington's disease, which may produce inspiratory grunts, dysarthria, dysphagia, and aspiration), and laryngeal fluttr (which may produce a clicking sound).

Some of the patients who have dysphonia and possible abnormal movements may have a psychogenic disorder. These symptoms could be conscious or unconscious conversion reactions. Historically, however, if a patient had a dysphonia without visual evidence of a vocal cord lesion, the patient was felt to have a psychogenic condition. Most of these patients really had movement disorders that were missed and could not be treated with psychotherapy. Now that there are new therapeutic approaches to laryngeal motion disorders, including local and systemic therapy and *Clostridium botulinum* toxin injections, it is very important to diagnose motion disorders (Brin et al, 1992a).

Summary

Neurolaryngology has evolved in the past decate as a discipline involved with the diagnosis and therapy of disorders of the larynx related to neurologic conditions. Newer tools have allowed better evaluation of abnormalities of laryngeal movement, and various scans have produced a better understanding of brainstem abnormalities. The collaboration of laryngologists and neurologists has produced the neurophysiologic basis for neurolaryngology. Since new treatments are available for patients with motion disorders, it is imperative for people who evaluate voice patients to be able to make accurate neurolaryngologic diagnoses.