Flaccid dysarthria is a perceptually distinguishable motor speech disorder produced by injury or malfunction of one or more of the cranial or spinal nerves. It reflects problems in the nuclei, axons, or neuromuscular junctions that make up the motor units of the final common pathway (FCP), and it may be manifest in any or all of the respiratory, phonatory, resonatory, and articulatory components of speech. Its primary deviant speech characteristics can be traced to muscular weakness and reduced muscle tone and their effects on the speed, range, and accuracy of speech movements. The primacy of weakness as an explanation for the speech characteristics of this disorder leads to its designation as flaccid dysarthria.

Flaccid dysarthria is encountered in a large medical practice at a frequency comparable to that of the other major single dysarthria types. From 1987 to 90 at the Mayo Clinic it accounted for 10.5% of all dysarthrias and 9.6% of all motor speech disorders seen in the Section of Speech Pathology (Figure 1–3).

Unlike most other dysarthria types, flaccid dysarthria sometimes results from damage confined to isolated muscle groups. As a result, it is justifiable to think of subtypes of the disorder, each characterized by speech abnormalities attributable to unilateral or bilateral damage to a specific cranial or spinal nerve or combination of cranial or spinal nerves. Accurate identification of the cranial or spinal nerve source for the deviant speech features can help localize the offending lesion that, in flaccid dysarthria, will always be somewhere between the brain stem or spinal cord and muscles of speech.

Close attention to the clinical features of flaccid dysarthria can help solidify the clinician’s knowledge of peripheral nervous system (PNS) anatomy and physiology. More than any other dysarthria type, it can teach us about the course and muscular innervations of the cranial nerves, the roles of specific muscle groups in speech production, and some of the remarkable and often spontaneous ways in which people adapt and compensate for weakness in order to maintain intelligible speech.

**CLINICAL CHARACTERISTICS OF FLACCID PARALYSIS**

Because flaccid paralysis reflects FCP damage, reflexive, automatic, and voluntary movements all are affected. Recognition of this principle is important to distinguishing lower motor neuron (LMN) lesions from lesions to other parts of the motor system.

Weakness, hypotonia, and diminished reflexes are the primary characteristics of flaccid paralysis. Atrophy, fasciculations, and fibrillations commonly accompany them. Occasionally, rapid weakening with use and recovery with rest are distinguishing features. The presence or absence of these characteristics is dependent to some extent on the portion of the motor unit that has been damaged. These characteristics are discussed below and summarized in Table 4–1.
Weakness

Weakness in flaccid paralysis stems from damage to any portion of the motor unit, including cranial and spinal nerve cell bodies in the brain stem or spinal cord. The peripheral or cranial nerve leading to muscle, and the neuromuscular junction. It can also result from pathology of the muscle itself. When damaged, motor units are inactivated and the ability to contract is lost or diminished in the muscles. When motor unit inactivation affects all of the LMN input to a muscle, paralysis, the complete inability to contract muscle, is the result. If some input to muscle remains viable, paresthesia, or reduced contraction and weakness, is the result.

The effects of weakness on muscle can be observed during single (phasic) contractions, during stepping, walking, and during sustained (tonic) contractions.

Hypotonia and reduced reflexes

Flaccid paralysis is also associated with hypotonia (reduced muscle tone) and reduced or absent normal reflexes. In flaccid paralysis, the ability of a muscle to contract in response to stretch is compromised because the motor component of the stretch reflex* operates through the FCP. This results in the flabbiness that can be seen or felt in muscles with reduced tone. Weakness and hypotonia, therefore, are roughly synonymous with the concept of flaccidity.

Atrophy

Muscle structure can be altered by FCP and muscle diseases. When cranial or spinal nerve nuclei (cell bodies), the peripheral nerve (axons), or muscle fibers are involved, muscles will eventually atrophy or lose bulk. Atrophy is almost always associated with significant weakness.

Fasciculations and fibrillations

When motor neuron cell bodies are damaged (and less prominently, when their axons are damaged), fasciculations and fibrillations may develop. Fasciculations are visible, arhythmic, isolated twitches or dimplings in resting muscle that result from spontaneous motor unit discharges in re- spiratory and abdominal muscles, and are visible to the examiner. Fibrillations are visible, spontaneous, independent contractions of individual muscle fibers that reflect slow repetitive action potentials. They can be detected electromyographically (EMG) with about 1 to 3 weeks after a muscle is deprived of motor nerve supply. Fasciculations and fibrillations are generally not present in muscle disease.

Progressive weakness with use

When disease affects the neuromuscular junction, progressive and rapid weakening of muscle with use, and recovery with rest, can occur. Even though fatigue is common in people with flaccid paralysis, there is no recovery or improvement with rest. Fatigue is prominent only in neuromuscular junction disease (for example, myasthenia gravis).

ETIOLOGIES

Flaccid dysarthria can be caused by any process that damages the motor unit. These include degenerative, inflammatory, toxic, metabolic, necrotic, traumatic, and vascular diseases. FCP diseases are associated with these broad etiological categories with varying frequency, however, and is also true for flaccid dysarthria. The exact distribution of causes of flaccid dysarthria is unknown and, in fact, probably varies as a function of the particular cranial or spinal nerves involved, the involvement of multiple versus single nerves, and the location of the pathology within the motor unit.

Some common terminology

A number of terms are used to describe pathologies of the FCP and muscle. The following definitions may facilitate comprehension of information presented in the remainder of this chapter:

Neuropathy—A general term that refers to any disorder of nerves, regardless of cause, although usually of noninflammatory etiology.
Neuritis—An inflammatory disorder of nerve. It may involve any nerve in the PNS. Peripheral neuropathies may affect motor, sensory, or autonomic fibers. They may be axonotmesis or axonotmesis or, mixed in their effects.
Crural neuropathies—Peripheral neuropathies involving the cranial nerves.
Motor neuropathy—Neuropathy of a single nerve.
Polynuropathy—A generalized process producing widespread bilateral and often symmetric effects on the CNS.
Radiculopathy—A CNS disorder involving the root of a spinal nerve, often just proximal to the intervertebral foramen.
Plexopathy—PNS involvement at the point where spinal nerves intermingle (inplexuses) before they become branches of a major plexus.
Myelitis—A non-specific term that indicates inflammation of the spinal cord.
Myositis—Any pathologic condition of the spinal cord, but most often refers to those that result from compression, or toxic or altered metabolic states (Kincad and Dyken, 1987).
Myopathy—Muscle disease. Myopathies are not associated with sensory disturbances or CNS pathology; the most common types of myopathy affect proximal rather than distal muscles.
Myositis—Inflammatory muscle disease.

Some associated diseases and conditions

At this point, it may be useful to review some conditions that are described here represent only a few of the potential etiologies of flaccid dysarthria.

Neuromuscular junction disease. Some diseases affect only the neuromuscular junction. Myasthenia gravis (MG) is the most common of these. MG is a chronic autoimmune disease characterized by abnormal repetitive weakening of voluntary muscles with use and improvement with rest. It appears that antibodies destroy acetylcholine receptors on muscle cells, such effect making muscle fibers more responsive to the ACh that triggers muscle contraction. When involved, muscles will progressively become progressively reduces with repeated use. Strength may improve with rest as nerves are able to replenish the supply of ACh. In women, MG occurs most often after age 50; women are affected most often between the ages of 20 and 40.

Plexus (drooping of the eyelids), weakness of facial muscles, flaccid dysarthria, and dysphagia are very frequent presenting signs of MG (Penn and Rowland, 1989). Beyond clinical neurologic examination, MG is commonly diagnosed by EMG, pulmonatory function tests, ACh receptor antibody blood tests, and a Tension (cholinergic) test. Injection of Tension precures temporary recovery from weakness brought on by prolonged muscular effort. Some patients undergoing stress testing is the task used for the Tensilon test. Patients with MG may show rapid development or worsening of flaccid dysarthria during stress testing, but rapid improvement after Tension injection, even as they continue to speak. Occasionally, a placebo (saline) will be used in stead of Tension injection when there is suspicion that a patient’s symptoms are psychogenic in origin. Such patients may improve with the placebo, ruling out MG and increasing suspicion about a psychological disorder.

Epstein-Barr syndrome is a disorder of neuromuscular transmission in which there is inadequate release of ACh from nerve terminals. It is characterized by an incremental (improved) response to repetitive nerve stimulation, a pattern opposite of MG. That is, weakness is greatest at the initiation of muscle use and with increased stimulation. Strength increases with repetitive stimulus, apparently because high rates of activation facilitate release of ACh. This occurs mostly in men with oat cell carcinoma o’ the lung, less frequently in the absence of neoplasms but with evidence of immune system abnormality (Penn, 1989).

Botulism is a deadly disease in which totalinum neurotoxin acts on presynaptic membranes to block release of ACh, thus blocking neuromuscular transmission. Contaminated food is the most common cause. Fa- cial, cranial, and respiratory muscles are affected, but the presence of signs (Penn, 1989). Botulism toxin in very small doses is an effective treatment for a number of movement disorders, including certain forms of spastic dysphonia. Its therapeutic use will be discussed in Chapter 17.

Vascular disorders. Brainstem stroke affecting cranial nerve nuclei can lead to flaccid dysarthria. A number of specific vascular syndromes are associated with flaccid dysarthria. Wernicke’s lateral medullary syndrome is among the more common of these. It is usually caused by ischemia in the territory of the posterior inferior cerebellar artery and affects the posteroslateral portion of the medulla. It leads to ipsilateral facial sensory loss, contralateral trunk and extremity sensory loss, ipsilateral cerebellar signs, ipsilateral neuroophthalmologic abnormalities, and ipsilateral nucleus ambiguous involvement with subsequent palatal, pharyngeal, and laryngeal weakness and associ- ated dysarthria and dysphagia (Brazis, 1992).
Common motor neuron disease, affects the bulbar, limb, and autonomic systems. In ALS, the most frequent injury is to the spinal cord and peripheral nerves. In ALS, the most frequent injury is to the spinal cord and peripheral nerves. The disease is characterized by the progressive degeneration of motor neurons in the brainstem and spinal cord, resulting in muscle weakness and atrophy. The disease is usually progressive and fatal, with an average duration of 3-5 years from the onset of symptoms.

Cerebrospinal fluid (CSF) analysis is often used to diagnose ALS, as it can help rule out other conditions that mimic the symptoms of ALS. CSF analysis typically shows an increase in the levels of certain proteins, such as tau and amyloid beta, which are associated with neurodegeneration. Other diagnostic tests may include electromyography (EMG) to assess nerve function, magnetic resonance imaging (MRI) to rule out other causes of muscle weakness, and nerve conduction studies (NCS) to evaluate the function of motor and sensory nerves.

Treatment for ALS is typically symptomatic and may include medications to manage muscle weakness, respiratory support, and physical therapy. There is no cure for ALS, and the disease is currently untreatable. However, research is ongoing to develop new treatments and therapies to slow the progression of the disease and improve the quality of life for patients with ALS.

The natural history of ALS is variable, and the disease can progress at different rates. Some patients may experience rapid progression, while others may have a more gradual decline. The rate of progression is influenced by a combination of genetic, environmental, and lifestyle factors. There is currently no known cure for ALS, and the disease is currently untreatable. However, research is ongoing to develop new treatments and therapies to slow the progression of the disease and improve the quality of life for patients with ALS.
ETIOLOGIES OF FLACCID DYSPHARIA FOR 107 QUASIRANDOMLY SELECTED CASES WITH A PRIMARY SPEECH DIAGNOSIS OF FLACCID DYSPHARIA AT THE MAYO CLINIC FROM 1969–90. PERCENTAGE OF CASES UNDER EACH BROAD ETIOLOGIC HEADING IS GIVEN IN PARENTHESES. SPECIFIC ETIOLOGIES UNDER EACH HEADING ARE ORDERED FROM MOST TO LEAST FREQUENT.

Traumatic (34%)
Surgical (29%)
1. Neurosurgical (14%)
   a. Cervical disk
   b. Carotid endarterectomy
   c. Carotid artery tumor, posterior fossa tumor, pontine tumor
   d. Carotid aneurysm, brainstem vascular
   e. Jugular and acoustic tumors
2. Otohinoselarlyngologic/plastic and dental surgery (8%)
   a. Thyroid
   b. Parathyroid, maxillectomy for carcinoma, dental surgery, facelift with liposuction
3. Chest/cardiac surgery (7%)
   a. Stent upper lobectomy for lung carcinoma
   b. Cardiac
Non-surgical (5%)
   a. Closed-head injury, skull fracture
   b. Neck injury
Neurotropics of undetermined origin (27%)
   a. Vth nerve, superior and/or recurrent laryngeal branch
   b. X (all branches) + Xll
   c. X (all branches) + Xlll, X (all branches) + X + Xll, jugular foramen syndrome
Muscle disease (8%)
   a. Muscular dystrophy, myotonic dystrophy, myopathy
Tumor (6%)
   a. Posterior fossa (foreman magnum, jugular foramen)
   b. Tongue
   c. Nasopharynx, widespread metastases
Myasthenia gravis (6%)
Degenerative (6%)
ALS (5%)
   a. Vascular
   b. Brainstem stroke (pons, medulla)
Infections (3%)
   a. Polio
   b. Meningitis
Anatomic malformation (3%)
   a. Arnold-Chiari malformation, syringobulbia, syringomyelia
Demyelinating (1%)
Guillain-Barre
Other (1%)
   a. Radiation therapy (palate, nasophraynx)

ALS, amyotrophic lateral sclerosis.

Disease of the neuromuscular junction can cause jaw weakness, as can disease affecting the jaw muscles themselves (myopathies).

Pain of trigeminal origin can indirectly affect speech. Trigeminal neuralgia (tic douloureux) is characterized by sudden brief periods of pain in one or more of the sensory divisions of the trigeminal nerve. It is often idiopathic but many cases may be due to compression or irritation of the trigeminal sensory roots (Brazis, Masdeu, and Biller, 1985). Pain can be triggered by sensory input from facial or jaw movements, sometimes leading to restricted lip, face, or jaw movements during speech to avoid triggering pain.

Non-speech oral mechanisms. It patients with unilateral mandibular branch lesions, the jaw may deviate to the weak side when opened, and the partly opened jaw may be pushed easily to the weak side by the examiner. The degree of masseter or temporalis contraction felt on palpation when the patient bites down may be decreased on the weak side.

With bilateral weakness, the jaw may hang open at rest. The patient may be unable to close it or may move it slowly or with reduced range. The patient may be unable to resist the examiner's attempts to open or close the jaw and may be unable to clench the teeth strongly enough for normal masseter or temporalis contraction to be felt (Figure 3–2). Patient complaints that may relate to jaw weakness include chewing difficulty, drooling, and overt recognition that the jaw is difficult to close or move.

If sensory branches to speech structures are affected, the patient may complain of decreased facial, cheek, tongue, teeth, or palate sensation. This can be assessed while the patients' eyes are closed by asking them to indicate when they feel touch or pressure applied to the affected areas. Decreased sensation of undetermined origin in one or more of the peripheral branches of the Vth nerve is often referred to as trigeminal sensory neuropathy. Viral etiology is common, but association with diabetes, sarcoidosis, and connective tissue disease has also been noted (Roglis, 1981).

### Table 4-2 Neuronal muscular deficits associated with flaccid dysphoria.

<table>
<thead>
<tr>
<th>Direction</th>
<th>Rhythm</th>
<th>Rate</th>
<th>Range</th>
<th>Force</th>
<th>Tone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individual movements</td>
<td>Repetitive movements</td>
<td>Individual movements</td>
<td>Repetitive movements</td>
<td>Individual movements</td>
<td>Repetitive movements</td>
</tr>
<tr>
<td>Normal</td>
<td>Regular</td>
<td>Normal</td>
<td>Normal</td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
</tbody>
</table>

Speech. Effects of Vth nerve lesions on speech are most apparent during reading and conversation and during AMRs. During AMRs, imprecision or slowness for "th" should be greater than that for "uh" or "kah." Vowel prolongation may be normal. In myasthenia gravis, progressive weakening of jaw movements during speech may be observed.

Unilateral damage to the motor division of the Vth nerve generally does not perceptibly affect speech. In contrast, bilateral lesions can have a devastating impact on articulation. The inability to elevate the bilaterally weak jaw can reduce precision or make impossible palatal, labiodental, lingualpalatals, and lingualpalatal articulation, as well as lip and tongue adjustments for many vowels, glides, and liquids. Speech rate may be slowed; this may be either a direct effect of weakness or reflect compensation for weakness. The effects of Vth nerve motor weakness on speech are summarized in Table 4—3. Lesions to the sensory portion of the mandibular branch, especially if bilateral, can cause loss of face, lip, lingual, and palatal sensation sufficient to result in imprecise articulation of bilabial, labiodental, lingualpalatal, and lingualpalatal sounds. This can occur without weakness and is presumably due to reduced sensory information about articulator movements or contacts. Technically, the articulatory distortions resulting from decreased sensation should not be classified as a dysarthria because the source of the speech deficit is not neuromotor. However, because the source is neurologic and does affect the precision of motor activity, it could be viewed as a "sensory dysarthria." Nonetheless, it is probably best simply to describe these difficulties as deficits resulting from decreased oral sensation, rather than using the term dysarthria or sensory dysarthria for them.

Individuals with relatively isolated severe jaw weakness will sometimes manually hold the jaw closed to facilitate articulation. Patients with mandibular branch sensory loss sometimes produce exaggerated movements of the jaw, lips, and face during speech, presumably in an attempt to increase sensory feedback. These movements can sometimes be mistaken for, or difficult to distinguish from, hyperkinetic movement disorders. However, sensory loss is usually detectable on touch or pressure sensation testing in patients with trigeminal sensory loss and not in patients with true hyperkinesias.

Finally, as noted previously, patients with trigeminal neuralgia may restrict jaw movement during speech to reduce sensation that might trigger pain. Although apparent visually, this compensatory restriction of movement may not be apparent auditorily. Mild articulatory distortions and decreased loudness or altered resonance, however, could result from such a strategy.

Facial (VIIth) nerve lesions

Course and function. The VIIth nerve is motor and sensory in function, but only its motor component has a clear role in speech. Motor fibers originate in the facial nucleus in the lower third of the pons and exit the cranial canal, along with fibers of the VIIIth nerve, through the internal auditory meatus. They pass through the facial canal, exit at the stylomastoid foramen below the ear pass through the parotid gland, and innervate the muscles of facial expression. The facial muscles crucial for speech are those that move the lip and firm the cheeks to permit impounding of intralabial air pressure and bilabial and labiodental articulation.

Etiology and localization of lesions. The VIIth nerve can be damaged in isolation or along with other cranial nerves. Pathology in the brain stem and posterior fossa can cause VIIth nerve damage, but a lesion anywhere along the nerve may affect its function for speech. Because the VIIth and VIIIth (abducens) nerves are in close proximity within the pons, especially in the floor of the fourth ventricle, lesions of both the VIIth and VIIIth nerves implicate that part of the brain stem. If the VIIth and VIIIth nerves are involved, as they frequently are with acoustic neuromas, a lesion is suspected in the area of the internal auditory meatus where both nerves exit the brain stem.

Known causes of facial paralysis include but are not limited to infection by herpes zoster, mononeuropathy, otitis media, meningitis, Lyme disease, syphilis, sarcoidosis, and inflammatory polyradiculoneuropathy. Common neoplastic causes include acoustic neuroma, cerebellopontine angle meningioma, neuraloma of the facial nerve, and leptomeningeal carcinomatosis (Clinical Examination in Neurology, 1991; Brazis, Masdeu, and Biler, 1985). Vascular lesions and trauma can also cause VIIth nerve lesions.

Bell's palsy is a relatively common condition of undetermined etiology characterized by isolated unilateral VIIth nerve weakness. Upper and lower facial muscles are affected and the ability to close the eye on the affected side may be limited. Depending on the exact site of lesion, the patient may also have decreased lacrimation, salivation, and taste sensation, as well as hyperacusis (possibly due to involvement of the portion of the nerve that innervates the stapedius). Most cases of isolated facial paralysis have no apparent cause and 80% have been reported to recover (Katsas and others, 1986). Proposed causes include an autoimmune-mediated inflammatory fo-
neuropathy, herpes simplex viral infection of the nerve, and swelling of the nerve induced by exposure to cold or allergic factors leading to compression by the bony facial canal (Clinical Examinations in Neurology, 1991).

Nonspasm oral speech mechanism. The visible effects of unilateral VLhN nerve lesions can be summarized as follows: At rest, the affected side sags and is hypotonic. The forehead may be un wrinkled, the eyebrow drooped, and the eye open and unblink ing. The tip of the nose and corner of the mouth may be drawn toward the unaffected side. Drooling on the affected side may occur. The nasolabial fold is often flattened and the nasal ala may be immobile during respiration. During smiling the face will retract more toward the intact side (see Figure 4–1). Food may dribble between the teeth and check on the weak side because of buccinator weakness. The patient may complain of biting the cheek or lip when chewing or speaking and have difficulty keeping food in the mouth. With milder weakness, asymmetry may be apparent only with use, as in voluntary retraction, pursing, and cheek puffing, with or without resistance from the examiner. Reduced or absent movement will be observable during voluntary, emotional, and reflexive activities. Fasciculations and atrophy may be apparent on the affected side.

Bilateral VLhN nerve lesions are less common than unilateral lesions. With bilateral lesions, the effects of weakness are on both sides, but may be less striking visually because of the symmetric appearance. At rest, the mouth may be lax and the space between the upper and lower lips wider than normal. During reflective smiling the mouth may not pull upward, giving the smile a "transverse" appearance. The patient may be unable to retract, purse, or puff the cheeks, or the seal on puffing may be reduced by the examiner. Fasciculations in the perioral area and chin may be present; patients are usually unaware of them. Reduced or absent movement on both sides may be noticeable when eating or talking. Reduced or absent movement of the face does not necessarily occur with VLhN nerve lesions. They are worthy of mention because they are unexpected in the context of FCP disease and may be confused with hyperkinesias of CNS origin. Synkinesis (see Figure 4–1) is the abnormal contraction of muscle adjacent to muscle that is contracting normally (for example, a normal reflexive or voluntary eye blink may cause a simultaneous movement of lower facial muscles). It reflects aberrant branching or misdirection of regenerating axons of the facial nerve, or abnormal activity of residual motor units, and is most commonly seen after recovery from Bell’s palsy (Ozuno, Mas deu, and Biller, 1985; Clinical Examinations in Neurology, 1991). Hemifacial spasm is characterized by paroxysmal, rapid, irregular, usually unilateral tonic twitching of facial muscle. It may be due to irritation of the nerve by a pulsating blood vessel in the area of the cerebellopontine angle or facial canal, but it may also be due to tumor, aneurysm, or WML (Clinical Examinations in Neurology, 1991; Levin and Lee, 1987; Nishit and others, 1987). Facial myokymia is characterized by rhythmic, undulating movements on an area of the face in which the surface of the skin moves like a "bug of worms." These are more prolonged than fasciculations and reflect alternating brief contractions of adjacent motor units. They are often benign but, if widespread, may be associated with multiple sclerosis, brainstem tumors, or demyelinating cranial neuropathies (Clinical Examinations in Neurology, 1991; Nudelman and Starr, 1983).

Speech. The speech tasks that are most revealing of VLhN nerve lesions are conversational speech and reading, speech AMRs, and stress testing.

A flutter of the cheeks may be present during conversation because hypotonicity results in less resistance to intranasal air pressure that is necessary to ensure adequate air flow through the nose.

The effect of unilateral facial nerve lesions on speech may be more visible than audible. This may be mild perceptible distortion of slibilant and labiodental consonants and, less frequently, anterior lingual fricatives and affricates. There is usually no perceptible effect on vowels.

Bilateral facial weakness, depending on its degree, can result in distortions or complete inability to produce /p/, /t/, /k/, /b/, /d/, /g/, and /v/. The distortion of bilateral stops is often in the direction of frication or spirantization. If lip rounding and spreading are markedly reduced, vowels may be abnormal. The effects of VLhN nerve lesions on speech are summarized in Table 4–3.

Patients with unilateral and bilateral facial weakness will sometimes spontaneously compensate in an effort to improve speech and physical...
The Disorders and Their Diagnoses

The internal laryngeal nerve, 1 component of the superior laryngeal nerve, transmits sensation from mucous membranes of portions of the larynx, epiglottis, base of the tongue, and aryepiglottic folds, and from stretch receptors in the larynx. The external laryngeal nerve, the motor component of the superior laryngeal nerve, supplies the inferior pharyngeal constrictors and the cricothyroid muscles. Its innervation of the cricothyroid muscle is important because cricothyroid contraction lengthens the vocal cords for pitch adjustments.

The recurrent laryngeal branch of the Xth nerve innervates all of the intrinsic laryngeal muscles except the cricothyroid. Its sensory fibers carry general sensation from the vocal cords and larynx below them.

Etioologies and localization of lesions. The localization of Xth nerve lesions is more complicated than that for other cranial nerves, owing to its long course and its three major branches. The degree of weakness, positioning of paralyzed vocal cords, and degree and type of voice or resonance abnormality depend on the localization of the lesion along the course of the nerve and on whether the lesion is unilateral or bilateral. Careful consideration of signs and symptoms stemming from Xth nerve lesions can often distinguish among lesions that are intramedullary, extramedullary, or above the pharyngeal branch; below the pharyngeal branch but above the superior and recurrent laryngeal branches; or below the superior laryngeal branch.

Vagus (Xth) nerve lesions

Course and function. Motor fibers of the Xth nerve that are relevant to speech originate in the nucleus ambiguus within the reticular formation of the lateral medulla. The rosettes of the Xth nerve emerge from the medulla, pass through the jugular foramen in the posterior fossa, and eventually into the pharynx to innervate the stylopharyngeus muscle that elevates the pharynx during swallowing and speech. Afferent fibers originate in the interior ganglion in the jugular foramen and terminate in the nucleus of the tractus solitarius in the medulla; they carry sensation from the pharynx and posterior tongue and are important to the sensory component of the gag reflex.

Glossopharyngeal (IXth) nerve lesions

Course and function. Motor fibers of the IXth nerve that are relevant to speech originate in the nucleus ambiguous within the reticular formation of the lateral medulla. The rosettes of the IXth nerve emerge from the medulla, pass through the jugular foramen in the posterior fossa, and eventually into the pharynx to innervate the stylopharyngeus muscle that elevates the pharynx during swallowing and speech. Afferent fibers originate in the interior ganglion in the jugular foramen and terminate in the nucleus of the tractus solitarius in the medulla; they carry sensation from the pharynx and posterior tongue and are important to the sensory component of the gag reflex.

Etiologies and localization of lesions. The IXth nerve is rarely damaged in isolation (at the least, the IXth nerve is also typically involved). It is susceptible to the same pathologic influences that affect the other cranial nerves in the lower brain stem. Intramedullary and extramedullary lesion localization is usually tied to localization of IXth and Xth nerve lesions.

Non-speech oral mechanisms. The IXth nerve is assessed clinically by examining the gag reflex, particularly asymmetry in the ease with which the reflex is elicited. A reduced gag may implicate the sensory or motor components of the reflex, the sensory component if the patient reports decreased sensation in the area. However, a normal gag can be present after intracranial section of the IXth nerve, suggesting that the IXth nerve is also involved in pharyngeal function. Therefore, the gag reflex may not be a reliable test for IXth nerve function (Clinical Examinations in Neurology, 1991). It is clear, however, that the IXth nerve may be implicated in patients with dysphagia, with lesions to the nerve presumably affecting pharyngeal elevation during the pharyngeal phase of swallowing.

Some individuals with IXth nerve lesions develop brief attacks of severe pain that begin in the throat and radiate down the neck to the back of the lower jaw. Pain can be triggered by swallowing or tongue protrusion. This condition is known as glossopharyngeal neuralgia.

Speech and the IXth nerve's role in speech cannot be assessed directly. It probably has some influence on resonance and perhaps phonatory function because of the effects of lesions on pharyngeal elevation. Because IXth nerve lesions are usually associated with IXth nerve lesions, and because the IXth nerve has a crucial and relatively clearly defined role in speech, the IXth nerve's importance in the assessment of dysarthria can be considered to be important for practical purposes.

Vagus nerve lesions

Course and function. Cell bodies of the Xth nerve that are relevant to speech originate in the nucleus ambiguus. Cell bodies of relevant sensory fibers originate in the inferior ganglion located in or near the jugular foramen; central processes of the sensory fibers terminate in the nucleus of the tractus solitarius in the brain stem. The Xth nerve exits the skull through the jugular foramen, along with the IXth and Xth nerves. From there it divides into the pharyngeal branch, which enters the pharynx; the superior laryngeal branch, which enters the pharynx and larynx; and the recurrent laryngeal branch, which passes down to the upper chest where it loops around the subclavian artery on the right, and around the aorta on the left before traveling back up the neck to enter the larynx.

The pharyngeal branch supplies the muscles of the pharynx, except the stylopharyngeus (IXth nerve), the muscles of the soft palate, except the tensor veli palatini (mandibular branch of the IXth nerve), and the palatoglossous muscle. It is responsible for pharyngeal constriction and palatal elevation and retraction during speech and swallowing.

appearance. In unilateral weakness, they may use a finger to prop up the sagging weak side at rest and during speech or, rarely, actually assist the movement of their lower lip in producing bilateral and occlusal sounds. Some patients will exaggerate jaw closure in an effort to approximate the lips. If weakness is bilateral, severe, isolated to the face, that chronic (as may occur in some cases of myopathy), substitution of lingual alveolar consonants for bilabial consonants (for example, /p/ for /t/).
maximal contraction of the levator veli palatini muscle. If it is centered, the palate may not be weak; if it is displaced to one side, the palate is probably weak on the opposite side.

3. The gag reflex may be diminished on the weak side.

In bilateral lesions the palate will hang low in the pharynx at rest and move minimally or not at all during phonation. The gag reflex may be difficult to elicit or absent (recall that this may be normal in some individuals), and nasal regurgitation may occur during swallowing.

Unilateral and bilateral superior laryngeal branch lesions that spare the recurrent laryngeal branch are frequently missed because the vocal cords can look normal. However, in unilateral lesions, even though both cords abduct, the affected vocal cord will appear shorter than normal and the epiglottis and anterior larynx will be shifted toward the intact side. In bilateral cricothyroid paralysis, both cords appear short and will be bowed, and the epiglottis will overhang and obscure the anterior portion of the vocal cords (Aronson, 1990).

Unilateral lesions of the recurrent laryngeal nerve but not the pharyngeal or superior laryngeal nerve will leave the affected vocal cord fixed in the paramedian position. When bilateral, both

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Table 4-4 Effects on the vocal cords and soft palate of Xth nerve lesions. Note that many lesions do not cause complete paralysis, so the vocal cords and soft palate may be weak but capable of some movement.

<table>
<thead>
<tr>
<th>Level of lesion</th>
<th>Vocal cords</th>
<th>Soft palate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharyngeal, superior, &amp; recurrent laryngeal branches</td>
<td>One cord fixed in abducted position</td>
<td>Both cords fixed in abducted position</td>
</tr>
<tr>
<td>Superior &amp; recurrent laryngeal branches</td>
<td>One cord fixed in abducted position</td>
<td>Both cords fixed in abducted position</td>
</tr>
<tr>
<td>Superior laryngeal nerve</td>
<td>Both cords can adduct</td>
<td>Afferent cord shorter</td>
</tr>
<tr>
<td>Recurrent laryngeal nerve</td>
<td>One cord fixed in paramedian position</td>
<td>Both cords fixed in paramedian position</td>
</tr>
</tbody>
</table>

Adapted from Aronson AE: Clinical voice disorders, New York, 1990, Thieme.

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Figure 4-2 Effects of unilateral (right) and bilateral Xth (vagus) nerve lesions above the origin of the pharyngeal, superior laryngeal, and recurrent laryngeal branches of the nerve. When unilateral, the soft palate hangs lower on the right and moves toward the left on phonation. The right vocal cord is fixed in an abducted position, while the left cord adducts to the midline on phonation. When bilateral, the palate rests low bilaterally and does not move on phonation. Both vocal folds remain in the abducted position on phonation. (From Aronson AE: Clinical voice disorders, New York, 1990, Thieme.)
Unilateral lesions of the Xth nerve below the pharyngeal arch but including the superior and recurrent laryngeal branches can result in breathlessness or aphonia, reduced loudness, diplaphonia, reduced pitch, and pitch breaks. Phrasing may be short because of air wastage through the incompletely adducted glottis during phonation. A rapid vocal flutter may be present during vowel prolongation. In bilateral paralysis these characteristics are exaggerated.

Lesions of the superior laryngeal nerve that spare the pharyngeal and recurrent laryngeal nerves cause subtle changes in voice. When unilateral, mild breathlessness or hoarseness and mild inability to alter pitch may be present. Loudness may be normal or mildly reduced. The inability to alter pitch may generate complaints about decreased ability to sing. Bilateral cricothyroid paralysis can cause mild to moderate breathlessness and hoarseness with decelerated loudness, and markedly reduced ability to alter pitch.

Unilateral recurrent laryngeal nerve lesions that spare the superior laryngeal nerve and pharyngeal branch will cause breathy-hoarse voice quality, decreased loudness, and sometimes diplaphonia and pitch breaks. Bilateral weakness of the muscle will cause inspiratory stridor, but the voice may be relatively unaffected because the cords are adducted close to the midline. Airway compromise, however, is a serious problem.

Acoustic and physiologic studies. Videostroboscopy or nasendoscopy are useful for documenting weakness of the velopharyngeal valve during speech. Bilateral velopharyngeal weakness can be documented by videostroboscopy and videobronchoscopy in lateral, frontal, and base views. Laryngoscopy examination is essential in cases with suspected vocal cord weakness, not only for diagnostic purposes but also for management considerations.

The visible characteristics of weak vocal cord activity have been described beyond simple observations of paralysis. Videostroboscopy and high-speed laryngeal photography in patients with unilateral vocal cord paralysis have documented a lack of firm glottal closure during phonation. "Light touch" glottic closure, reflecting either less than complete paralysis or assistance to medial cord approximation by the Bernoulli effect; irregular vocal cord vibrations; exaggeration in the affected vocal cord of the mucosal wave during phonation; and abnormal frequency and amplitude perturbations in vocal cord activity (Hirano, Koike, and von Leden, 1968; Wattersen, McFarlane, and Menicucci, 1990). Greater vibratory amplitude and exaggerated mucosal waves are consistent with what might be expected with hypotonicity. These observations are consistent with the perception of breathlessness (lack of firm glottal closure), hoarseness, and perhaps diplaphonia (irregular and asymmetric vibratory characteristics) in patients with vocal cord weakness.

Acrodynamic studies of people with unilateral or bilateral vocal cord weakness have identified increased airflow rates during speech. These findings are consistent with neuromuscular weakness of the vocal cords, with subsequent incomplete vocal cord adduction and excessive air escape through the glottis during phonation (Hirano, Koike, and von Leden, 1968; Iwasa, von Leden, and Williams, 1972; Til and Apl, 1991; von Leden, 1968). Relatively, Til and Xip (1991) have established that dysarthric speakers with laryngeal "hypoepal" had increased mean air flow during connected speech and inspired twice the volume of air per minute than normals, mostly through increased breaths per minute. In contrast, their mean speech duration per breath group was only half of normal. They also expired more air than normal during pauses but tended to have reduced pause frequency and duration, possibly secondary to poor vocal cord valving or a compensatory effort to increase speaking time. These findings are consistent with the perception of breathlessness and short phrases in people with laryngeal weakness. They define some of the effects that may make compensation for vocal cord weakness, such as increased breaths per minute, increased inspiratory volume, and a tendency to reduce pause frequency and duration.

Acoustic and physiologic studies of people with unilateral vocal cord paralysis or weakness have documented the following characteristics: a breakdown of formant structure, reflected in a long-term average acoustic spectrum characterized by high fundamental frequency amplitude with a marked dropoff of harmonics above the first formant; random noise in spectrograms and increased spectral peaks in high frequency regions, possibly reflecting turbulent air flow through a partially open glottis; restricted standard deviation and range of fundamental frequency, suggesting reduced ability to reach upper pitch ranges (Hammarberg, Fritzell, and Schiratzki, 1994; Murray, 1995; Gelbman, and Roldack, 1978). These studies have noted a relationship between some of these characteristics and perceptual judgments of breathiness and hypo- functional voice (Hammarberg, Fritzell, and Schiratzki, 1994; Reich and Lerman, 1978). Findings of restricted fundamental frequency range and variability (Murry, 1978) are consistent with Dar- ley, Aronson, and Brown's (1971) finding that