# 4 Flaccid Dysarthria

**Chapter Outline**



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.

**Table 4–1** Components of the motor unit associated with characteristics of flaccid paralysis (+ = present; – = absent; +/– = may or may not be present)

| Feature | Damaged component | | | |
|---|---|---|---|---|
| | Cell body | Axon | Neuromuscular junction | Muscle |
| Weakness | + | + | + | + |
| Hypotonia | + | + | + | + |
| Diminished reflexes | + | + | + | + |
| Atrophy | + | + | – | + |
| Fasciculations | + | +/– | – | – |
| Fibrillations | + | +/– | – | – |
| Rapid weakening & recovery with rest | – | – | + | – |

### 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 disease inactivates 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, *paresis*, or reduced contraction and weakness, is the result.

The effects of weakness on muscle can be observed during single (phasic) contractions, during repetitive contractions, 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, arrhythmic, isolated twitches or dimplings in resting muscle that result from spontaneous motor unit discharges in response to nerve degeneration or irritation. Fibrillations are invisible, spontaneous, independent contractions of individual muscle fibers that reflect slow repetitive action potentials. They can be detected electromyographically (EMG) within about 1 to 3 weeks after a muscle is deprived of motor nerve supply. Fasciculations and fibrillations are generally not present in muscle disease.

*The stretch reflex was discussed in Chapter 2.

### 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 paresis, *rapid* weakening and recovery with rest 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, neoplastic, traumatic, and vascular diseases. FCP diseases are associated with these broad etiologic categories with varying frequency, however, and this 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 disease of nerve, regardless of cause, although usually of noninflammatory etiology.

*Neuritis*—An inflammatory disorder of nerve.

*Peripheral neuropathy*—Any disorder of nerve in the PNS. Peripheral neuropathies can affect motor, sensory, or autonomic fibers. They may be axonal, demyelinating, or mixed in their effects.

*Cranial neuropathies*—Peripheral neuropathies involving the cranial nerves.

*Mononeuropathy*—Neuropathy of a single nerve.

*Polyneuropathy*—A generalized process producing widespread bilateral and often symmetric effects on the PNS.

*Radiculopathy*—A PNS 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 (in plexuses) before forming nerves that go to the extremities.

*Myelitis*—A nonspecific term that indicates inflammation of the spinal cord.

*Myelopathy*—Any pathologic condition of the spinal cord, but most often refers to those that result from compression, or toxic or altered metabolic states (Kincaid 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 relatively unique to FCP or muscle diseases whose presence have a strong association with flaccid but not other forms of dysarthria. The conditions discussed 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 abnormally rapid weakening of voluntary muscles with use and improvement with rest. It appears that antibodies destroy acetylcholine (ACh) receptors on muscle, in effect making muscle less responsive to the ACh that triggers muscle contraction. As a consequence, muscle contractions become progressively reduced with repeated use. Strength may improve with rest as nerves are able to replenish the supply of ACh. In men, MG occurs most often after age 50; women are affected most often between the ages of 20 and 40.

*Ptosis* (drooping of the eyelid), 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, pulmonary function tests, ACh receptor antibody blood tests, and a Tensilon (edrophonium chloride) test. Injection of Tensilon produces temporary recovery from weakness brought on by prolonged muscular effort. Sometimes speech stress testing is the task used for the Tensilon test. Patients with MG will show rapid development or worsening of flaccid dysarthria during stress testing, but rapid improvement after Tensilon injection, even as they continue to speak. Occasionally, a placebo (saline) will be used instead of Tensilon 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 suspicions about a psychological disorder.

*Eaton-Lambert 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 or with slow rates of stimulation; strength increases with repetitive rapid stimulation, apparently because high rates of activation facilitate release of ACh. The syndrome occurs mostly in men with oat cell carcinoma of the lung, less frequently in the absence of neoplasm but with evidence of immune system abnormality (Penn, 1989).

*Botulism* is a deadly disease in which botulinum toxin acts on presynaptic membranes for the release of ACh, thus blocking neuromuscular transmission. Contaminated food is the most common cause. Facial, oropharyngeal, and respiratory paralysis can be among presenting signs (Penn, 1989). Botulism toxin in very small doses is an effective treatment for a number of movement disorders, including certain forms of spasmodic 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. *Wallenberg'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 posterolateral 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 ambiguus involvement with subsequent palatal, pharyngeal, and laryngeal weakness and associated dysarthria and dysphagia (Brazis, 1992).

*Collet-Sicord syndrome* is characterized by unilateral involvement of cranial nerves IX–XII. It can be caused by vascular lesions of the jugular vein and carotid artery below the skull base (and also by carotid artery dissection, skull base fractures, inflammatory lesions, and various tumors such as neurinomas, metastases, and jugular glomus tumors) (Waespe and others, 1988).

***Infectious processes.*** *Polio (poliomyelitis)* is a viral disease with an affinity for LMN cell bodies, most often in the lumbar and cervical regions of the spinal cord. Bulbar involvement occurs in 10 to 15% of cases, with the IXth and Xth cranial nerves being affected most often, but involvement of the Vth and VIIth nerves not uncommon. The dorsal area of the medulla is generally involved in the bulbar form of the disease; respiratory and circulatory centers in the medulla can also be affected (Adams and Victor, 1991). Survivors often recover function of muscles that were not completely paralyzed, usually within six months. Occasionally, polio victims will develop the insidious onset of progressive weakness long after the acute attack ("*post-polio syndrome*"); this may occur by chance alone, but it may be that previously involved nerves are more susceptible to the effects of aging (Kincaid and Dyken, 1987).

*Herpes zoster* is a viral infection that may affect the Vth and VIIth nerve ganglia. It most often produces pain. When it causes facial paresis, it is known as the *Ramsay-Hunt syndrome*. The herpes virus may also cause superior laryngeal nerve paralysis and dysphonia (Andour, Schneider, and Hilsinger 1980; Hartman, Daily, and Morin, 1989).

*Sarcoidosis* is a nonviral, chronic granulomatous infection that can occur in all organs and tissues. It occasionally affects the PNS or central nervous system (CNS), most often single or multiple cranial nerves, especially the VIIth nerve. Sometimes, cranial neuropathies associated with sarcoidosis result from basilar meningitis (Pleasure and Schotland, 1989).

Individuals infected with HIV who develop AIDS may develop neurologic complications as the result of opportunistic infections. *Cryptococcal meningitis* is the most common fungal infection in AIDS. The resulting meningeal inflammation can affect posterior fossa structures, leading to multiple cranial nerve palsies. Other neurologic complications of AIDS that can lead to cranial nerve involvement include *CNS lymphoma* (the most common CNS tumor in AIDS) and *neurosyphilis* (Singer, 1991). The involvement of cranial nerves for speech may lead to flaccid dysarthria in such cases.

***Demyelinating disease.*** *Guillain-Barré syndrome* is a disorder of unknown cause but is frequently preceded by viral infection. It is characterized by the acute or subacute onset of PNS dysfunction. Histologically, focal demyelinization occurs in peripheral and cranial nerves. When severe, axonal degeneration may occur. Proximal muscles are affected more severely than distal muscles. Facial, oropharyngeal, and ocular muscles are occasionally affected first, and more than half of affected individuals have facial weakness, dysphagia, and flaccid dysarthria. Recovery is sometimes rapid and complete but may take several months in others. Some individuals are left with permanent weakness (Griffin, 1983).

*Chronic demyelinating polyneuritis* is similar to Guillain-Barré syndrome but less acute in onset and more prolonged in course. Affected individuals may suffer frequent, recurrent attacks (Pleasure and Schotland, 1989). Weakness may be asymmetric.

***Muscle disease.*** *Muscular dystrophy* is a genetic degenerative disease. There are several types, including Duchenne, fascioscapulohumeral, and limb girdle. They are associated with degeneration of muscle fibers and proliferation of connective tissue. The effects are usually diffuse, chronic, and progressive. The fascioscapulohumeral form may affect bulbar muscles; facial weakness may be prominent.

*Myotonic muscular dystrophy* is an inherited autosomal dominant disease that can affect several organ systems. A characteristic diagnostic sign is failure of muscle to relax promptly after forcible contraction. *Percussion myotonia* is a persistent myotonic contraction that follows strong percussion. It may be observed in the tongue, after pressure is exerted on it, as an obvious depression that persists for several seconds (Roses, 1989). Muscle atrophy gives the face a characteristic long, lean, and expressionless appearance, with weak voluntary and emotional facial movements. Articulation, phonation, and resonance may be affected. Velopharyngeal incompetence has been reported as the presenting symptom in one case (Salomonson, Kawamoto, and Wilson, 1988).

*Polymyositis* is a disease of striated muscle that can be idiopathic or associated with a number of infectious processes. The tongue, jaw, pharyngeal, and laryngeal muscles may be affected, causing dysarthria and dysphagia (Adams and Victor, 1991).

***Degenerative disease.*** *Motor neuron diseases* are a group of disorders that involve degeneration of motor neurons. *Progressive bulbar palsy* is a motor neuron disease that primarily affects LMNs supplied by cranial nerves. Although it may also include upper motor neurons (UMNs) that supply the bulbar muscles, it can be limited to LMNs. *Amyotrophic lateral sclerosis (ALS)*, the most common motor neuron disease, affects the bulbar, limb, and respiratory muscles. By definition, ALS is a disease of both UMNs and LMNs; its initial manifestations may be confined to the LMNs of bulbar muscles, however. Therefore, progressive bulbar palsy and ALS may produce flaccid dysarthria associated with multiple cranial nerve involvement. These conditions will be discussed in more detail in Chapter 10 which discusses mixed dysarthrias.



***Anatomic anomalies.*** *Arnold-Chiari malformation* is a congenital anomaly of undetermined etiology characterized by downward elongation of the brain stem and cerebellum into the cervical spinal cord. Signs and symptoms reflect injury to the cerebellum, medulla, and lower cranial nerves. Onset of symptoms is infrequently delayed until adulthood. The damage to the brain stem may lead to flaccid dysarthria.

*Syringomyelia* (syrinx = a tube) is a developmental abnormality characterized by elongated cavities lined by glia close to the central canal of the spinal cord. Cavity expansion and compression of the anterior horns of the gray matter cause atrophy of the anterior horn cells and axonal degeneration in the spinal cord. The condition may extend upward into the fourth ventricle, where it is called *syringobulbia*. When the brain stem is involved, Vth nerve sensory loss, vertigo, and cranial nerve weakness can occur (Bannister, 1985). Flaccid dysarthria may be associated with the condition.

***Other.*** Radiation therapy for the treatment of carcinoma can cause neuropathy, including cranial neuropathies and, possibly, associated flaccid dysarthria. Pathology usually involves axonal degeneration and fibrosis (Pleasure and Schotland, 1989).

Cranial mononeuropathies, particularly facial and vocal cord paralyses, are frequently idiopathic (of unknown origin). Recovery from such conditions is often quite good.

## SPEECH PATHOLOGY

### Distribution of etiologies in clinical practice

The box on page 104 summarizes the etiologies for 107 quasirandomly selected cases seen at the Mayo Clinic with a primary speech pathology diagnosis of flaccid dysarthria. The reader is cautioned that these data may not represent the distribution of etiologies of flaccid dysarthria in the general population or its distribution in many speech pathology practices. They probably approximate the most frequent etiologies encountered in speech pathology practices within large multidisciplinary primary and tertiary medical settings where patients are referred for diagnosis as well as management of communication disorders.

The data establish that flaccid dysarthria can result from a wide variety of medical conditions. Surgical trauma, most often but not always limited to the laryngeal branches of the vagus nerve, was a frequent cause. Trauma to the laryngeal branches of the vagus occurred in cervical disk, thyroid, cardiac, and upper lung surgeries because of the proximity of the vagus nerve to the surgical field. Carotid endarterectomy—to remove occlusive or ulcerative plaque from the carotid artery in the neck—reportedly damages the VIIth, Xth, or XIIth cranial nerves in about 15% of cases (Massey and others, 1984); such injuries are usually transient and probably the result of retraction or clamping of nerves rather than nerve division. Neurosurgical trauma was more likely to result in multiple cranial nerve lesions than was otorhinolaryngologic, plastic, dental, or chest surgery. Neck surgery, most often thyroid surgery, was the most frequent cause of isolated laryngeal nerve lesions.

It is noteworthy that a substantial proportion of flaccid dysarthrias were due to cranial neuropathies of undetermined origin, and that the Xth nerve was most often implicated when the lesion was confined to a single nerve (note that even though facial palsy occurs relatively frequently in the general population (Katusic and others, 1986), it apparently either did not often result in dysarthria or did not lead to referral for speech pathology assessment or management). The remaining etiologies were associated with muscle disease, tumor, myasthenia gravis, ALS, stroke, infectious processes, anatomic malformations, demyelinating disease, or the effects of radiation therapy.

### Patient perceptions and complaints

People with flaccid dysarthria may offer complaints that differ from those of people with other dysarthria types; these complaints may provide clues to speech diagnosis and its localization, especially when they can be attributed to muscles supplied by a single cranial nerve. Such complaints will be noted in the review of deficits associated with each of the cranial nerves because they provide clues about some of the questions that should be asked when weakness is suspected as the primary cause of speech difficulty.

The next several sections will address the cranial and spinal nerves that may be involved in flaccid dysarthria. The anatomic course and function of each nerve will be reviewed briefly (greater detail was provided in Chapter 2), as will some of the conditions that can damage each nerve. Nonspeech findings that may be encountered will also be discussed. Finally, the salient features of the motor speech examination will be discussed, including the primary auditory perceptual character-

**ETIOLOGIES OF FLACCID DYSARTHRIA FOR 107 QUASIRANDOMLY SELECTED CASES WITH A PRIMARY SPEECH DIAGNOSIS OF FLACCID DYSARTHRIA 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%)
   Cervical disk
   Carotid endarterectomy
   Carotid artery tumor, posterior fossa tumor, pontine tumor
   Carotid aneurysm, brainstem vascular
   Jugular and acoustic tumors
2. Otorhinolaryngologic/plastic and dental surgery (8%)
   Thyroid
   Parathyroid, maxillectomy for carcinoma, dental surgery, facelift with liposuction
3. Chest/cardiac surgery (7%)
   Left upper lobectomy for lung carcinoma
   Cardiac

***Nonsurgical*** (5%)
Closed-head injury, skull fracture
Neck injury

**Neuropathies of undertermined origin** (27%)
Xth nerve, superior and/or recurrent laryngeal branches
X (all branches) + XII
X, all branches
(VII), (XII), (VII + X), (IX + X + XI, jugular foramen syndrome)

**Muscle disease** (8%)
Muscular dystrophy, myotonic dystrophy, myopathy

**Tumor** (6%)
Posterior fossa (foramen magnum, jugular foramen)
Tongue/neck
Nasopharynx, widespread metastases

**Myasthenia gravis** (6%)

**Degenerative** (6%)
ALS

**Vascular** (5%)
Brainstem stroke (pons, medulla)

**Infectious** (3%)
Polio
Meningitis

**Anatomic malformation** (3%)
Arnold-Chiari malformation, syringobulbia, syringomyelia

**Demyelinating** (1%)
Guillain-Barré

**Other** (1%)
Radiation therapy (palate, nasopharynx)

*ALS,* amyotropic lateral sclerosis.



istics, accompanying visible deficits, some of the compensatory behaviors that may develop in response to the neuromuscular deficit, and some of the evidence from instrumental studies that further delineate the characteristics and neurologic bases of the speech deficits. The neuromuscular deficits associated with flaccid dysarthria are summarized in Table 4–2.

### Trigeminal (Vth) nerve lesions

***Course and function.*** The three main branches of the Vth nerve arise in the trigeminal ganglion in the petrous bone of the middle cranial fossa. Central connections from the trigeminal ganglion enter the lateral aspect of the pons and are distributed to various nuclei in the brain stem.

The peripheral distribution of the Vth nerve through its three branches includes (1) the sensory ophthalmic branch, which exits the skull through the superior orbital fissure to innervate the upper face; (2) the sensory maxillary branch, which exits the skull through the foramen rotundum to supply the midface; and (3) the motor and sensory mandibular branch, which exits the skull through the foramen ovale to supply the jaw muscles, tensor tympani, and tensor veli palatini.

Trigeminal functions for speech are mediated through its maxillary and mandibular branches. Sensory roles are to provide tactile and proprioceptive information about jaw, face, lip, and tongue movements and their relationship to stationary articulatory structures within the mouth (teeth, alveolus, palate). Motor functions are associated with jaw movements during speech.

***Etiologies and localization of lesions.*** Damage to the Vth nerve is usually associated with involvement of other cranial nerves. *It is rarely the only cranial nerve involved in flaccid dysarthria* (see Table 4–5). Any pathology that can affect the middle cranial fossa can produce weakness or sensory loss in its distribution. Etiologies most often include stroke, infection, arteriovenous malformation (AVM), tumors in the middle fossa or cerebellopontine angle, and trauma to the skull or anywhere along its course to muscle. Peripheral branches are most often damaged in isolation by tumors or fractures of the facial bones or skull. 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.

***Nonspeech oral mechanism.*** In 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 face, cheek, tongue, teeth, or palate sensation. This can be assessed while the patients' eyes are closed by asking them to indicate when they detect 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. Facial numbness is occasionally a presenting symptom in multiple sclerosis (Regli, 1981).

**Table 4–2** Neuromuscular deficits associated with flaccid dysarthria.

| Direction | Rhythm | Rate | | Range | | Force | Tone |
|---|---|---|---|---|---|---|---|
| **Individual movements** | **Repetitive movements** | **Individual movements** | **Repetitive movements** | **Individual movements** | **Repetitive movements** | **Individual movements** | **Muscle tone** |
| Normal | Regular | Normal | Normal | Reduced | Reduced | Weak | Reduced |

Adapted from Darley FL, Aronson AE, and Brown JR: Differential diagnostic patterns of dysarthria, J Speech Hear Res 12:246, 1969a.

***Speech.*** Effects of Vth nerve lesions on speech are most apparent during reading and conversation and during AMRs. During AMRs, imprecision or slowness for "puh" should be greater than that for "tuh" or "kuh." 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 bilabial, labiodental, linguadental, and linguaalveolar 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, linguaalveolar, and linguapalatal sounds. This can occur without weakness and is presumably due to reduced sensory information about articulatory 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 cavity, 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 lips and firm the cheeks to permit impounding of intraoral air pressure and bilabial and labiodental articulation.

***Etiologies 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 functions for speech.

Because the VIIth and VIth (abducens) nerves are in close proximity within the pons, especially in the floor of the fourth ventricle, lesions of both the VIth and VIIth 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, mononucleosis, otitis media, meningitis, Lyme disease, syphilis, sarcoidosis, and inflammatory polyradiculoneuropathy. Common neoplastic causes include acoustic neuroma, cerebellopontine angle meningioma, neurofibroma of the facial nerve, and leptomeningeal carcinomatosis (*Clinical Examinations in Neurology*, 1991; Brazis, Masdeu, and Biller, 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 86% have been reported to make full recovery (Katusic and others, 1986). Proposed causes include an autoimmune-mediated inflammatory fo-



**Table 4–3** Effects on speech of unilateral and bilateral cranial nerve and spinal respiratory nerve lesions. The IXth and XIth nerves are not included because of the negligible or unclear effects of lesions of them on speech.

| Cranial nerves | Respiratory-phonatory | | Resonance | | Articulation | | Prosody | |
|---|---|---|---|---|---|---|---|---|
| | Unilateral | Bilateral | Unilateral | Bilateral | Unilateral | Bilateral | Unilateral | Bilateral |
| V | None | None | None | None | None | Imprecise consonants<br>Bilabial<br>Labiodental<br>Linguadental<br>Lingual-alveolar<br>Distorted vowels, glides, liquids | None | Slow rate (compensatory or primary) |
| | | | | | Mild distortion of bilabial & labiodentals<br>Distortion of anterior lingual fricatives & affricates | Distortion or inability to produce bilabials & labiodentals<br>? Vowel distortions<br>? Anterior lingual frictative & affricate distortions | None | Slow rate (compensatory or primary) |
| VII | None | None | None | None | None<br>(? mildly weak pressure consonants) | Weak pressure consonants | Short phrases | Short phrases |
| X<br>Above pharyngeal branch | Breathiness<br>Reduced loudness<br>Reduced pitch<br>Short phrases<br>Hoarseness<br>Diplophonia | Breathiness<br>Aphonia<br>Short phrases<br>Inhalatory stridor | Mild hypernasality<br>Nasal emission | Moderate + hypernasality<br>Nasal emission | | | | |

*Continued.*

**Table 4–3** Effects on speech of unilateral and bilateral cranial nerve and spinal respiratory nerve lesions. The IXth and XIth nerves are not included because of the negligible or unclear effects of lesions of them on speech—continued.

| | Respiratory-phonatory | | Resonance | | Articulation | | Prosody | |
|---|---|---|---|---|---|---|---|---|
| **Cranial nerves** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** |
| X<br>Below pharyngeal branch | Same as above | Same as above | None | None | None | None | Short phrases | Short phrases |
| X<br>Superior branch only | Breathy hoarseness<br>Respiratory-phonatory | Breathy, hoarse<br>Reduced loudness<br>Reduced pitch range<br>Resonance | None | None | None | None | Short phrases<br>Prosody | Short phrases |
| X | Breathy, hoarse | Breathy, hoarse | None | None | None | None | Short phrases | Short phrases |
| Recurrent branch only | Reduced loudness<br>Diplophonia | Reduced loudness | | | Mildly imprecise lingual consonants | Mild-severe imprecise lingual consonants<br>Vowel distortions | None | Slow rate (compensatory or primary) |
| XII | None | None | None | ? altered | None | None | None | Short phrases<br>Reduced pitch & loudness variability |
| Spinal Respiratory Nerves | None | Reduced loudness<br>Reduced pitch variability<br>Strained voice (compensatory) | None | None | | | | |



cal 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).

***Nonspeech oral mechanism.*** The visible effects of unilateral VIIth nerve lesions can be striking. At rest, the affected side sags and is hypotonic. The forehead may be unwrinkled, the eyebrow drooped, and the eye open and unblinking. 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 squirrel between the teeth and cheek 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 VIIth 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 reflexive 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 overcome easily by the examiner. Fasciculations in the perioral area and chin may be present; patients are usually unaware of them. Patients may complain that their face or the lips do not move well during speech and that they lose food or liquid out of their mouth when eating. Drooling during speech, when concentrating on another activity, during eating, or during sleep may be reported or observed.

Abnormal movements of the face sometimes occur with VIIth 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 (Brazis, Masdeu, 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 AVM (*Clinical Examinations in Neurology*, 1991; Levin and Lee, 1987; Nishi 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 "bag 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 VIIth 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 intraoral air pressure peaks during pressure sound production. Poor bilabial closure on one or both sides may be apparent. There may be a noticeable mismatch between speech AMRs for "puh" versus those for "tuh" and "kuh," with reduced precision and perhaps slowness of "puh" because of lip weakness. In general, precision is reduced more than speed, unless weakness is bilateral and severe. If myasthenia gravis is present, stress testing may generate visible and auditory perceptual deficits attributable to lower-face weakness.

The effect of unilateral facial nerve lesions on speech may be more visible than audible. There may be mild perceptible distortion of bilabial 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/, /m/, /w/, /hw/, /f/, and /v/. The distortion of bilabial stops is often in the direction of frication or spirantization. If lip rounding and spreading are markedly reduced, vowels may be distorted. The effects of VIIth 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

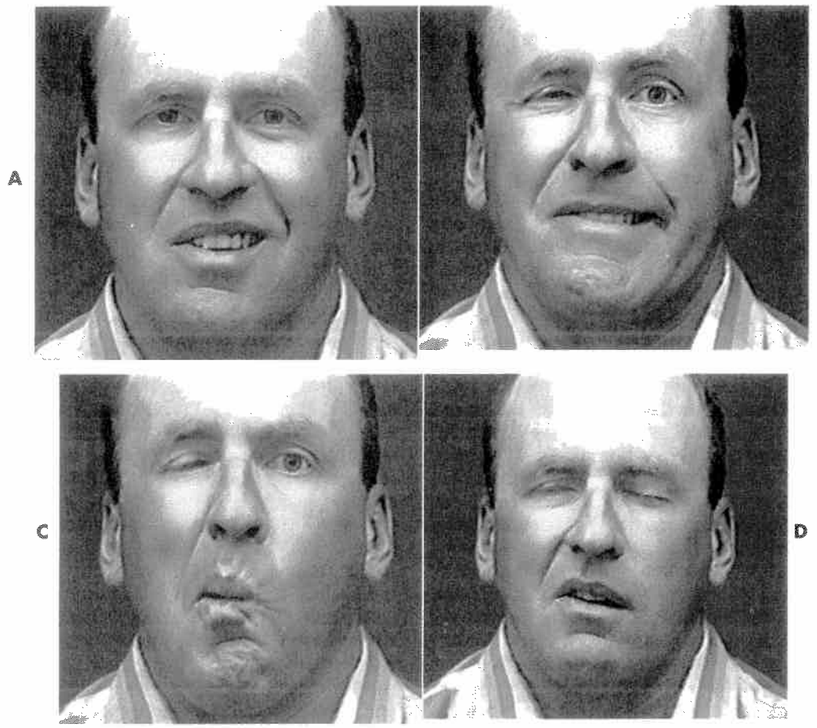


**Figure 4–1 A,** partially recovered unilateral right facial weakness during spontaneous smile; **B,** voluntary lip retraction; and **C,** lip pursing, with **D,** paradoxical, involuntary right-lip retraction (synkinesis) when voluntarily closing the eyes. Synkinetic eye closing is also apparent during voluntary lip retraction (**B**) and pursing (**C**).

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 bilabial and labiodental sounds. Some patients will exaggerate jaw closure in an effort to approximate the lips. If weakness is bilateral, severe, isolated to the face, and chronic (as may occur in some cases of myopathy), substitution of lingual alveolar consonants for bilabial consonants (for example, t/p) may occur.

### Glossopharyngeal (IXth) nerve lesions

***Course and function.*** Motor fibers of the IXth nerve that are relevant to speech originate in the nucleus ambiguus within the reticular formation of the lateral medulla. The rootlets 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 inferior 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 Xth 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 Xth and XIth nerve lesions.

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

### Vagus (Xth) 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 XIth 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 Vth nerve), and the palatoglossus muscle. It is responsible for pharyngeal constriction and palatal elevation and retraction during speech and swallowing.

The internal laryngeal nerve, a component of the superior laryngeal nerve, transmits sensation from mucus 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.

***Etiologies 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 nerve lesions can be intramedullary, extramedullary, or extracranial. *Intramedullary lesions* damage the nerve in the brain stem. *Extramedullary lesions* damage the trunk of the nerve as it leaves the body of the brain stem but while it is still within the cranial cavity (that is, before it exits from the jugular foramen). *Extracranial lesions* damage the nerve after it exits the skull. It is generally the case that as the distance of a lesion from the brain stem increases, the number of muscles, structures, and functions affected by the lesion decreases. Therefore, intracranial lesions are more likely than extramedullary and extracranial lesions to be bilateral or associated with multiple cranial nerve involvement. Extramedullary lesions are more likely to be unilateral but may still affect several cranial nerves (for example, the IXth, Xth, and XIth nerves all exit through the jugular foramen on each side of the posterior fossa). Extracranial lesions are more likely to be isolated to the Xth nerve and perhaps only one of its branches.

The relationships between Xth nerve lesion loci and impairment of muscle function are summa-

rized in Table 4–4. The most important relationships include the following.

1. Intramedullary, extramedullary, and extracranial lesions above the separation of the pharyngeal, superior laryngeal, and recurrent laryngeal branches will affect all muscles supplied by the nerve below the level of the lesion. Therefore, pharyngeal and palatal muscles supplied by the pharyngeal branch, the cricothyroid muscle supplied by the superior laryngeal branch, and the remaining intrinsic laryngeal musculature supplied by the recurrent laryngeal branch will be weak or paralyzed on the side of the lesion (see Figure 4–2).
2. Lesions below the pharyngeal branch, but still high enough in the neck to affect the superior and recurrent branches, will spare the upper pharynx and velopharyngeal mechanism, but will cause paralysis or weakness of the cricothyroid and remaining intrinsic muscles on the side of the lesion.
3. Lesions of the superior laryngeal branch but not the recurrent laryngeal or pharyngeal branches will affect the cricothyroid but not the velopharyngeal mechanism or the remaining intrinsic laryngeal musculature.
4. Lesions affecting only the recurrent laryngeal nerve will cause weakness or paralysis of the intrinsic laryngeal musculature on the side of the lesion, except the cricothyroid.

Intramedullary and extramedullary lesions affecting the Xth nerve can be caused by primary and metastatic tumor, infection, stroke, syringobulbia, Arnold-Chiari malformation, Guillian-Barré syndrome, polio, motor neuron disease, and other inflammatory or demyelinating diseases (Aronson, 1990). Not infrequently, lesions in the posterior fossa affect cranial nerves IX, X, and XI in combination. When this occurs in the area of the jugular foramen it is called the *jugular foramen syndrome.*

Extracranial Xth nerve disorders can be caused by myasthenia gravis; tumors in the neck or thorax; aneurysms in the aortic arch, internal carotid, or subclavian artery; and surgery (Aronson, 1990; Brazis, Masdeu, and Biller, 1986). Thyroidectomy is a common cause of vocal cord paralysis, and Xth (and VIIth and XIIth) nerve damage can occur during carotid endarterectomy (Massey and others, 1984). Vagus nerve degeneration and dysphonia have been reported in individuals with severe alcoholic neuropathies (Guo, McLeod, and Baverstock, 1987).

***Nonspeech oral mechanism.*** Unilateral pharyngeal branch lesions are manifest by the following:

1. The soft palate hangs lower on the side of the lesion.
2. It pulls toward the nonparalyzed side on phonation (Figures 4–2 and 4–3). A palate that hangs low at rest but elevates symmetrically may not be weak. It may be asymmetric as a normal variant or the result of scarring from tonsillectomy. If palatal asymmetry on phonation is ambiguous, the clinician should look for a levator "dimple" representing the point of maximum 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 adduct, 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

**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.

| | Vocal cords | | Soft palate | |
|---|---|---|---|---|
| **Level of lesion** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** |
| I. Pharyngeal, superior, & recurrent laryngeal branches | One cord fixed in abducted position | Both cords fixed in abducted position | One side low, immobile | Both sides low, immobile |
| II. Superior & recurrent laryngeal branches | One cord fixed in abducted position | Both cords fixed in abducted position | Normal | Normal |
| III. Superior laryngeal nerve | Both cords can adduct<br>Affected cord shorter<br>Epiglottis & anterior larynx shifted toward intact side on phonation | Absent tilt of thyroid on cricoid cartilage<br>Inability to see full cord length because of epiglottis overhang<br>Bowed cords | Normal | Normal |
| IV. Recurrent laryngeal nerve | One cord fixed in paramedian position | Both cords fixed in paramedial position | Normal | Normal |

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



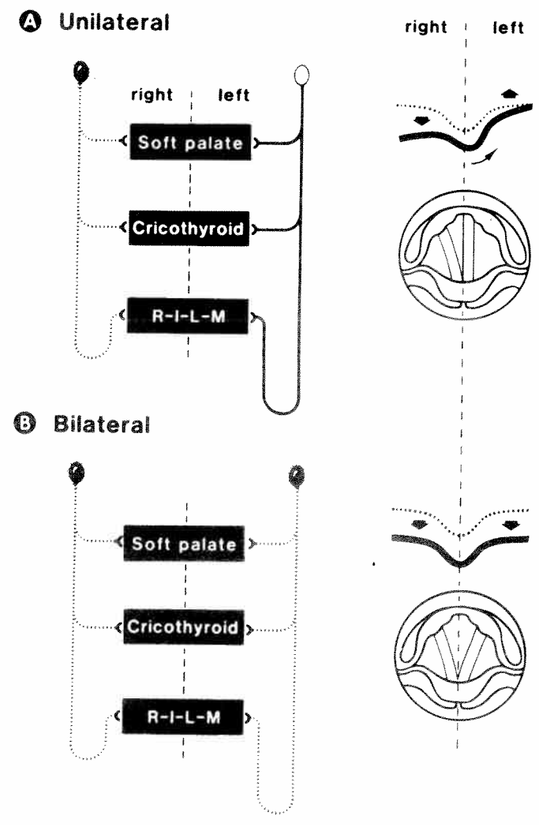


**Figure 4–2** Effects of unilateral (right) and bilateral Xth (vagus) nerve lesions above the origin of the pharyngeal, superior laryngeal, and recurrent laryngeal branches of the nerve. When unilateral, the soft palate hangs lower on the right and pulls toward the left on phonation. The right vocal fold is fixed in an abducted position, while the left fold adducts to the midline on phonation. When bilateral, the palate rests low bilaterally and does not move on phonation. Both vocal folds remains in the abducted position on phonation. (From Aronson AE: Clinical voice disorders, New York, 1990, Thieme.)

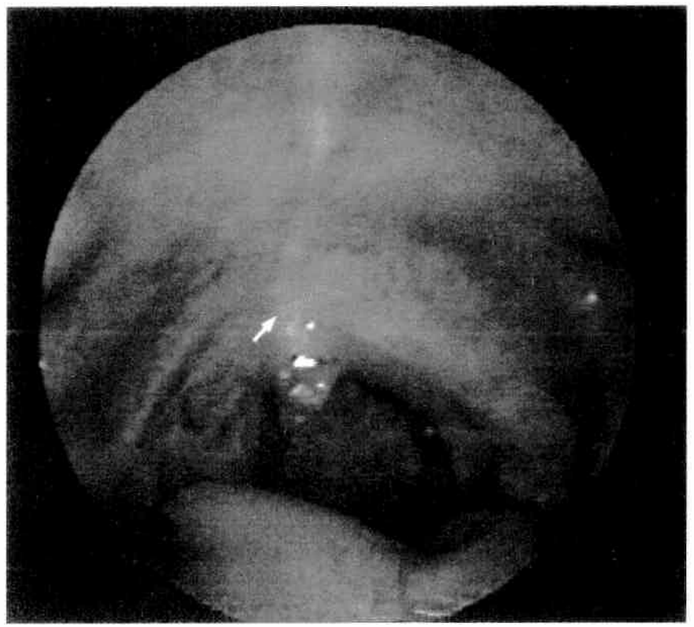

**Figure 4–3** Palatal movement during phonation in a patient with left palatal weakness. The palate pulls to the right. The arrow identifies the levator eminence (dimple), which is also displaced to the right. This patient also has left lingual weakness secondary to a left XIIth (hypoglossal) nerve lesion; note the smaller left than right side of the tongue because of atrophy on the left.

cords will be in the paramedian position. The cords are not completely abducted because the intact cricothyroid maintains its adductor function and pulls the cord closer to the midline. In unilateral paralysis, dysphagia may be present, and the cough and glottal coup will be weak. There may be airway compromise. In bilateral paralysis, airway compromise and inhalatory stridor often occur because abductor paralysis prevents widening of the glottis during inhalation. The resulting respiratory distress may require tracheostomy.

Lesions affecting both the recurrent and superior laryngeal branches of the vagus will leave the affected vocal cords paralyzed in the abducted position because all laryngeal adductors are affected. The cough and glottal coup are weak and dysphagia is common. Signs of weakness are worse with bilateral than unilateral vocal cord lesions.

***Speech.*** Table 4–3 summarizes the effects of unilateral and bilateral Xth nerve lesions on speech. The effects cross several aspects of speech production, including phonation, resonance, articulation, and prosody, but the effects on resonance and phonation are generally most pronounced. When the pharyngeal branch is affected unilaterally, there may be little or no perceptible effect or mild-to-moderate hypernasality and nasal emission during pressure-consonant production. If weakness is bilateral, *hypernasality* can be marked to severe, *audible nasal emission* may be very apparent, and *pressure consonants may be noticeably imprecise* because of inability to impound intraoral pressure. *Loudness may be mildly reduced* because of the damping effects of the nasal cavity on the emitted sound, and *phrase length may be reduced* because of nasal air wastage. Facial grimacing may develop in an effort to valve the air stream at the nares. The imprecision of pressure consonants sometimes generates suspicions about tongue, face, or jaw weakness. If consonant imprecision is solely due to velopharyngeal incompetence, occluding the nares during speech will facilitate intraoral pressure for articulation and aid assessment of the adequacy of the other articulators.



Unilateral lesions of the Xth nerve below the pharyngeal branch but including the superior and recurrent laryngeal branches can result in *breathiness* or *aphonia*, *reduced loudness*, *diplophonia*, *reduced pitch*, and *pitch breaks. Phrases 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 *breathiness* 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 breathiness and hoarseness, *decreased 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 *diplophonia* and *pitch breaks*. Bilateral weakness or paralysis will cause *inhalatory 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.*** Videofluoroscopy or nasendoscopy are useful for documenting weakness of the velopharyngeal valve during speech. Bilateral velopharyngeal weakness can be demonstrated by nasendoscopy and videofluoroscopy in lateral, frontal, and base views. Laryngoscopic 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 vibration; 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 breathiness (lack of firm glottal closure), hoarseness, and perhaps diplophonia (irregular and asymmetric vibratory characteristics) in patients with vocal cord weakness.

Aerodynamic 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; Iwata, von Leden, and Williams, 1972; Till and Alp, 1991; von Leden, 1968). Relatedly, Till and Alp (1991) have established that dysarthric speakers with laryngeal "hypovalving" 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 breathiness and short phrases in people with laryngeal weakness. They define some of the efforts that individuals may make to compensate for vocal cord weakness, such as increased breaths per minute, increased inspiratory volume, and a tendency to reduce pause frequency and duration.

Acoustic 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 energy levels 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, 1984; Murry, 1978; Rontal, Rontal, and Rolnick, 1975). These studies have noted a relationship between some of these characteristics and perceptual judgments of breathiness and hypofunctional voice (Hammarberg, Fritzell, and Schiratzki, 1984; Reich and Lerman, 1978). Findings of restricted fundamental frequency range and variability (Murry, 1978) are consistent with Darley, Aronson, and Brown's (1975) finding that