to lower cranial nerves can also result from dissection of the internal carotid artery.

Some specific vascular syndromes are also associated with flaccid dysarthrias. *Wallenberg's lateral medullary syndrome* is among the more common of these. It is usually caused by occlusion in the intracranial vertebral artery or the posterior inferior cerebellar artery, which affects the lateral portion of the medulla and inferior cerebellum. It leads to ipsilateral facial and contralateral trunk and extremity sensory loss, ipsilateral cerebellar signs, ipsilateral neuro-ophthalmologic abnormalities, and ipsilateral nucleus ambiguus involvement with subsequent palatal, pharyngeal, and laryngeal weakness and associated dysarthria and dysphagia.[8] *Collet-Sicard syndrome* is characterized by unilateral involvement of cranial nerves IX through XII. It can be caused by vascular lesions of the jugular vein and carotid artery below the skull base, as well as by skull base fractures, inflammatory lesions, and tumors. Occlusion of the anterior spinal artery or its source, the vertebral artery, can injure the hypoglossal nerve *(medial medullary syndrome)* and cause lingual weakness.[8]

### Anatomic Anomalies

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

*Syringomyelia* (syrinx = a tube) is an abnormality characterized by elongated cavities lined by glia close to the central canal of the spinal cord. Cavity expansion and compression of 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 in the brainstem, where it is called *syringobulbia.* Syringomyelia and syringobulbia can reflect developmental anomalies but can also develop in response to tumor, trauma, or inflammatory conditions. When the brainstem is involved, the ninth through twelfth cranial nerves can be affected.[52,62] Flaccid dysarthrias can result from this.

### 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, mainly motor. Focal demyelinization and sometimes axonal degeneration occur in peripheral and cranial nerves. 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.[20]

*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.[48]

### Infectious Processes

*Polio (poliomyelitis)* is a now rare 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 cranial nerves IX and X most often affected, but cranial nerve V and VII involvement is 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.[2] Survivors often recover function of muscles that are not completely paralyzed, usually within 6 months.

Polio victims occasionally 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; it does not appear related to reactivation of the virus.[11]

*Herpes zoster* is a viral infection that may affect the fifth and seventh nerve ganglia, most often producing pain. When it causes facial paresis, it is known as the *Ramsay-Hunt syndrome*. The herpes virus can also cause superior laryngeal nerve paralysis and dysphonia.[3,24]

*Sarcoidosis* is a nonviral, chronic granulomatous infection that can occur in all organs and tissues. It occasionally affects the PNS or CNS, most often single or multiple cranial nerves, especially the seventh nerve. Sometimes, cranial neuropathies associated with sarcoidosis result from basilar meningitis.[48]

Individuals with *human immunodeficiency virus (HIV)* who develop *acquired immune deficiency syndrome (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 and lead 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*.[57] The involvement of cranial nerves for speech may lead to a flaccid dysarthria in such cases.



### Other Causes

*Skull base tumors* can cause cranial neuropathies and flaccid dysarthrias. *Radiation therapy* for the treatment of carcinoma in the neck, oral cavity, and tonsillar area can cause cranial neuropathies and, possibly, associated flaccid dysarthrias. Pathology usually involves axonal degeneration and fibrosis as a result of damaged vascular supply to tissues in the radiated field[37,48]; it may be difficult to separate the effects of axonal degeneration (neurologic weakness) from the effects of necrosis on reduced range of motion of affected structures. The effects of radiation on cranial nerve function may be delayed for years following radiation treatment.[34,56,58]

Cranial mononeuropathies, particularly facial (Bell's palsy) and vocal fold paralyses, are frequently *idiopathic* (of unknown origin). Recovery from such conditions is often quite good.

## SPEECH PATHOLOGY

### Distribution of Etiologies in Clinical Practice

Box 4-1 and Figure 4-1 summarize the etiologies for 154 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 dysarthrias in the general population or its distribution in many speech pathology practices. They may approximate the most frequent causes 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 dysarthrias can result from various medical conditions. Surgical trauma, most often but not always limited to the laryngeal branches of the vagus nerve, was a frequent cause. Surgical trauma to the laryngeal branches of the vagus can occur in cervical disk, thyroid, cardiac, and upper lung surgeries because of the proximity of the vagus nerve to the surgical field. *Carotid endarterectomy*—performed to remove

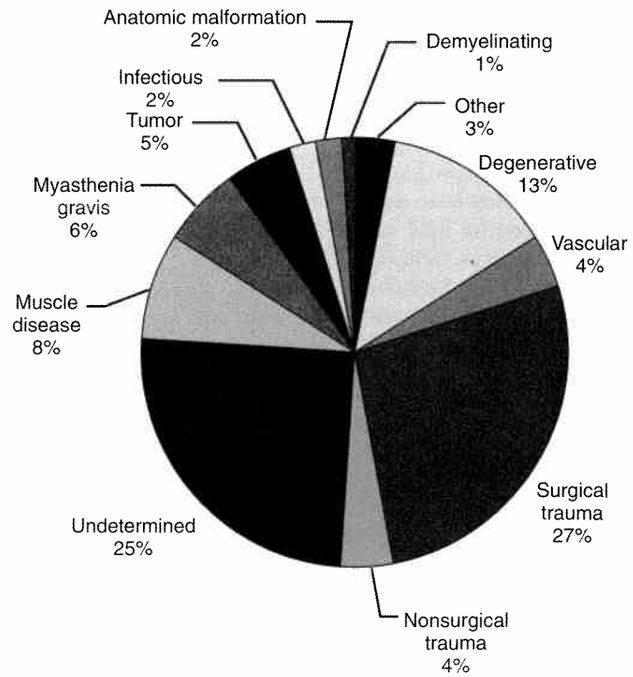


**FIGURE 4-1** Distribution of etiologies for 154 quasirandomly selected cases with a primary speech pathology diagnosis of flaccid dysarthria at the Mayo Clinic from 1969-1990 and from 1999-2001 (see Box 4-1 for details).

**table 4-2 Neuromuscular deficits associated with flaccid dysarthrias**

| 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 or slow | Normal or slow | Reduced | Reduced | Weak | Reduced |

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

occlusive or ulcerative plaque from the carotid artery in the neck—injures cranial (especially the tenth and twelfth) and cervical nerves in 12% to 14% of cases.[5,55] Such injuries are usually transient and probably result from retraction or clamping of nerves rather than nerve division and distal degeneration.

Neurosurgical trauma was more likely to result in multiple cranial nerve lesions than was otorhinolaryngologic, plastic, dental, or chest or cardiac surgery. Neck surgery, most often thyroid surgery, was the most frequent cause of isolated laryngeal nerve lesions. Nonsurgical trauma was most often due to closed head injury.

It is noteworthy that 25% of the flaccid dysarthrias were idiopathic, and that the tenth nerve was most often implicated when the idiopathic lesion was confined to a single nerve. The remaining etiologies were less frequent and represented by various conditions, including degenerative disease, muscle disease, MG, tumor, stroke, infectious processes, anatomic malformations, demyelinating disease, and the effects of radiation therapy or drug toxicity.

This retrospective review did not permit a clear delineation of dysarthria severity. However, a judgment about intelligibility was made for 74% of the last 47 patients seen in the sample; 46% were felt to have reduced intelligibility. The degree to which this figure accurately estimates the frequency of intelligibility impairments in the population with flaccid dysarthria is unclear. It is likely that many patients for whom an observation of intelligibility was not made had normal intelligibility, but the sample probably contains a larger number of mildly impaired patients than is encountered in many rehabilitation settings. In general, reduced intelligibility is more common when damage to a cranial or peripheral nerve involved in speech is bilateral or when multiple nerves are involved.

Finally, cognitive impairment is not common in flaccid dysarthria. Among the last 47 patients seen in the sample, cognitive impairment was noted in only 11%. Affected patients had diseases associated with CNS as well as PNS impairments (e.g., muscular dystrophy).

## Patient Perceptions and Complaints

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

The next several sections address the cranial and spinal nerves that may be involved in flaccid dysarthrias. The anatomic course and function of each nerve is reviewed briefly (greater detail was provided in Chapter 2), as are some of the conditions that can damage each nerve. Nonspeech findings are also discussed. Finally, the salient features of the motor speech examination will be discussed, including the primary auditory perceptual characteristics, 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 dysarthrias are summarized in Table 4-2.

## Trigeminal Nerve (V) Lesions

### *Course and Function*

The three main branches of cranial nerve V 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 brainstem.

The peripheral distribution of cranial nerve V through its three branches includes the sensory ophthalmic branch, which exits the skull through the superior orbital fissure to innervate the upper face; the sensory maxillary branch, which exits the skull through the foramen rotundum to supply the mid face; and 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 (e.g., teeth, alveolus, palate). Motor functions are associated with jaw movements during speech.

### *Etiologies and Localization of Lesions*

Damage to cranial nerve V is usually associated with involvement of other cranial nerves. *It is rarely the only cranial nerve involved in flaccid dysarthrias* (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 aneurysm, infection, arteriovenous malformation (AVM), tumors in the middle fossa or cerebellopontine angle, and surgical (e.g., posterior fossa, acoustic neuroma, temporomandibular joint) or nonsurgical 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.[8] 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 will 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. Patient complaints may include chewing difficulty, drooling, and 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 patient's eyes are closed by asking him or her to indicate when light touch or pressure applied to the affected areas is detected. Decreased sensation of undetermined origin in one or more of the peripheral branches of the fifth 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.[49]

### *Speech*

Effects of cranial nerve V lesions on speech are most apparent during reading, conversation, and alternate motion rates (AMRs). During AMRs, imprecision or slowness for "puh" should be greater than that for "tuh" or "kuh." Vowel prolongation may be normal. In MG, progressive weakening of jaw movements during speech may be observed.

Unilateral damage to the motor division of the fifth nerve generally does not perceptibly affect speech. In contrast, bilateral lesions can have a devastating impact on articulation. The inability to elevate a bilaterally weak jaw can *reduce precision or make impossible bilabial, labiodental, lingual-dental, and lingual-alveolar 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 fifth 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, lingual-alveolar, and lingual-palatal 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 primarily neuromotor. However, because the source is neurologic and does affect the precision of motor activity, it could be viewed as a *"sensory dysarthria";* the use of such a


**FIGURE 4-2** **A,** Partially recovered unilateral right facial weakness during spontaneous smile. **B,** Voluntary lip retraction. **C,** Lip pursing, with, **D,** paradoxical, involuntary right lip retraction (synkinesis) when voluntarily closing the eyes. Synkinetic eye closing is also apparent during, **B,** voluntary lip retraction and, **D,** pursing.

term should be accompanied by a statement that the speech deficits are presumed to reflect decreased oral sensation.

Individuals with relatively isolated severe jaw weakness 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 Nerve (VII) Lesions

### *Course and Function*

Cranial nerve VII 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 cranial nerve VIII, 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

**table 4-3** Effects on speech of unilateral and bilateral cranial nerve and spinal respiratory nerve lesions. The ninth and eleventh nerves are not included because of the negligible or unclear effects of lesions of them on speech.

| | Respiratory-Phonatory | | Resonance | | Articulation | | Prosody | |
|---|---|---|---|---|---|---|---|---|
| **Cranial Nerves** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** | **Unilateral** | **Bilateral** |
| V | None | None | None | None | None | Imprecise<br>• bilabials<br>• labiodentals<br>• lingual-dentals<br>• lingual-alveolars<br>• vowels<br>• glides<br>• liquids | None | Slow rate (compensatory or primary) |
| VII | None | None | None | None | Mild distortion of bilabials & labiodentals<br>? Mild distortion of anterior lingual fricatives & affricates | Distortion or inability to produce bilabials & labiodentals<br>? Vowel distortions<br>? Anterior lingual fricative & affricate distortions | None | Slow rate (compensatory or primary) |
| X 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 | None (? mildly weak pressure consonants) | Weak pressure consonants | Short phrases | Short phrases |

*Continued*

**table 4-3** Effects on speech of unilateral and bilateral cranial nerve and spinal respiratory nerve lesions. The ninth and eleventh nerves are not included because of the negligible or unclear effects of lesions of them on speech.—cont'd

| | 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 | Breathiness<br>Hoarseness | Breathiness<br>Hoarseness<br>Reduced<br>• loudness<br>• pitch<br>• range | None | None | None | None | Short phrases | Short phrases |
| X<br>Recurrent branch only | Breathiness<br>Hoarseness<br>Reduced loudness<br>Diplophonia | Breathiness<br>Hoarseness<br>Reduced loudness | None | None | None | None | Short phrases | Short phrases |
| XII | None | None | None | ? Altered | Mildly imprecise lingual consonants | Mild to severe imprecise lingual consonants<br>Vowel distortions | None | Slow rate (compensatory or primary) |
| Spinal respiratory nerves | None | Reduced<br>• loudness<br>• pitch<br>• variability<br>Strained voice (compensatory) | None | None | None | None | None | Short phrases<br>Reduced pitch & loudness variability |



that move the lips and firm the cheeks to permit impounding of intraoral air pressure for bilabial and labiodental articulation.

### *Etiologies and Localization of Lesions*

Cranial nerve VII can be damaged in isolation or along with other cranial nerves. Pathology in the brainstem and posterior fossa can cause seventh nerve damage, but a lesion anywhere along the nerve may affect its functions for speech.

Because cranial (abducens) nerves VI and VII are in close proximity within the pons, especially in the floor of the fourth ventricle, lesions of both the sixth and seventh nerves implicate that part of the brainstem. If cranial nerves VII and VIII 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 brainstem.

Known infectious causes of facial paralysis include, but are not limited to, infection by herpes zoster, mononucleosis, otitis media, meningitis, Lyme disease, syphilis, sarcoidosis, Guillain-Barré syndrome, and inflammatory polyradiculoneuropathy. Common neoplastic causes include acoustic neuroma, parotid tumor, cerebellopontine angle meningioma, tumor of the facial nerve, and leptomeningeal carcinomatosis.[8,31,38] Vascular lesions and trauma can also cause cranial nerve VII lesions.

*Bell's palsy* is a relatively common idiopathic condition of undetermined etiology characterized by isolated unilateral cranial nerve VII weakness. Upper and lower facial muscles are affected, and the ability to close the eye on the affected side may be limited. Some patients 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). A significant majority reportedly makes full recovery, with better recovery in those younger than age 50.[13] Proposed causes for Bell's palsy include an autoimmune-mediated inflammatory focal 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.[39]

### *Nonspeech Oral Mechanism*

The visible effects of unilateral cranial nerve VII 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 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 4-2). 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. Reduced or absent movement is apparent during voluntary, emotional, and reflexive activities. Fasciculations and atrophy may be apparent on the affected side.

Bilateral cranial nerve VII lesions are less common than unilateral lesions. With bilateral lesions, the effects of weakness are on both sides, but they 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 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, or during eating or sleep may be reported or observed.

Abnormal movements of the face sometimes occur with cranial nerve VII lesions. They are noteworthy because they are unexpected in the context of FCP disease and may be confused with hyperkinesias of CNS origin. *Synkinesis* (see Figure 4-2) is the abnormal contraction of muscle adjacent to muscle that is contracting normally (e.g., 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. It is most commonly seen after recovery from Bell's palsy.[8] *Hemifacial spasm* is characterized by paroxysmal, rapid, irregular, usually unilateral tonic twitching of the 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 may also be associated with tumor, vascular abnormalities, or multiple sclerosis.[8] *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." Such movements 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, syringobulbia, or demyelinating cranial neuropathies.[39,29]

### *Speech*

The speech tasks that are most revealing of cranial nerve VII 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 MG 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 can 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/, /b/, /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 cranial nerve VII lesions on speech are summarized in Table 4-3.

Patients with unilateral and bilateral facial weakness sometimes spontaneously compensate in an effort to improve speech and physical 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 exaggerate jaw closure in an effort to approximate the lips. If weakness is bilateral, severe, isolated to the face, and chronic, substitution of lingual articulation for bilabial consonants (e.g., t/p) may occur.[44]

## Glossopharyngeal Nerve (IX) Lesions

### *Course and Function*

Motor fibers of cranial nerve IX that are relevant to speech originate in the nucleus ambiguus within the reticular formation in the lateral medulla. The rootlets of cranial nerve IX emerge from the medulla, exit through the jugular foramen in the posterior fossa, and eventually pass into the pharynx to innervate the stylopharyngeus muscle, which 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*

Cranial nerve IX is rarely damaged in isolation (at the least; cranial nerve X is also typically involved). It is susceptible to the same pathologic influences that affect the other cranial nerves in the lower brainstem. Intramedullary and extramedullary lesion localization is usually tied to localization of cranial nerve X and XI lesions (discussed later).

### *Nonspeech Oral Mechanism*

Cranial nerve IX 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 cranial nerve IX, suggesting that cranial nerve X is also involved in pharyngeal function. It is clear, however, that cranial nerve IX may be implicated in dysphagia, with lesions to it presumably affecting pharyngeal elevation during the pharyngeal phase of swallowing.

Some individuals with cranial nerve IX 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 role of cranial nerve IX 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 cranial nerve IX lesions are usually associated with cranial nerve X lesions, and because cranial nerve X has a crucial and relatively clearly defined role in speech, cranial nerve IX's importance in the assessment of dysarthria can be considered indeterminate for practical purposes.



## Vagus Nerve (X) Lesions

### *Course and Function*

Cell bodies of cranial nerve X 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 brainstem.

Cranial nerve X exits the skull through the jugular foramen, along with cranial nerves IX and XI. 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 upper chest. Cranial nerve X 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 (cranial nerve IX), the muscles of the soft palate except the tensor veli palatini (mandibular branch of cranial nerve V), 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 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 folds for pitch adjustments.

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

### *Etiologies and Localization of Lesions*

The localization of cranial nerve X lesions is more complicated than that for other cranial nerves due to its long course and three major branches. The degree of weakness, positioning of paralyzed vocal folds, and degree and type of voice or resonance abnormality depend on the localization of the lesion along the course of the nerve and whether the lesion is unilateral or bilateral. Careful consideration of signs and symptoms stemming from cranial nerve X lesions can often distinguish among lesions that are (1) intramedullary, extramedullary, or above the pharyngeal branch; (2) below the pharyngeal branch but above the superior and recurrent laryngeal branches; or (3) below the superior laryngeal branch.

Vagus nerve lesions can be intramedullary, extramedullary, or extracranial. *Intramedullary lesions* damage the nerve in the brainstem. *Extramedullary lesions* damage the trunk of the nerve as it leaves the body of the brainstem but while it is still within the cranial cavity (i.e., 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 brainstem increases, the number of muscles, structures, and functions affected by the lesion decreases. Thus 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 (e.g., cranial nerves IX, X and XI all exit through the jugular foramen on each side of the posterior fossa). Extracranial lesions are more likely to be isolated to cranial nerve X, and perhaps only one of its branches.

The relationships between cranial nerve X lesion loci and impairment of muscle function are summarized 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 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 muscles supplied by the recurrent laryngeal branch are weak or paralyzed on the side of the lesion (Figure 4-3).
2. Lesions below the pharyngeal branch, but still high enough in the neck to affect the superior and recurrent branches, spare the upper pharynx and velopharyngeal mechanism but cause paralysis or weakness of the cricothyroid and other intrinsic muscles on the side of the lesion.
3. Lesions of the superior laryngeal branch but not the recurrent laryngeal or pharyngeal branches affect the cricothyroid but not the velopharyngeal mechanism or the remaining intrinsic laryngeal muscles.
4. Lesions affecting only the recurrent laryngeal nerve cause weakness or paralysis of the

**table 4-4** Effects on the vocal folds and soft palate cranial nerve X lesions. Note that many lesions do not cause complete paralysis, so the vocal folds and soft palate may be weak but capable of some movement.

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

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

intrinsic laryngeal muscles on the side of the lesion, except the cricothyroid.

Intramedullary and extramedullary lesions affecting cranial nerve X can be caused by primary or metastatic tumor, infection, stroke, syringobulbia, Arnold-Chiari malformation, Guillain-Barré syndrome, polio, motor neuron disease, and other inflammatory or demyelinating diseases.[4] 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 a *jugular foramen syndrome*.

Extracranial cranial nerve X disorders can be caused by myasthenia gravis, tumors in the neck or thorax, aneurysms in the aortic arch or internal carotid or subclavian artery, aortic dissection, and internal carotid artery dissection.[8,19,22] Surgery is a common cause of vocal fold paralysis, most often associated with thyroidectomy; carotid endarterectomy; anterior approach for cervical fusion; and cardiovascular, pulmonary, and skull base procedures.[32] Vagus nerve degeneration and dysphonia have been reported in individuals with severe alcoholic neuropathies.[21]

### *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 (see Figures 4-3 and 4-4). 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:

1. The palate hangs low in the pharynx at rest and moves minimally or not at all during phonation.
2. The gag reflex may be difficult to elicit or absent (recall that this may be normal in some individuals).
3. 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 folds can appear normal. However, in unilateral lesions, even though both folds adduct, the affected vocal fold appears shorter than normal, and the epiglottis and anterior larynx are shifted toward the intact side. In bilateral cricothyroid paralysis, both folds appear short and bowed, and the epiglottis overhangs and obscures the anterior portion of the vocal folds.[4]

Unilateral lesions of the recurrent laryngeal nerve but not the pharyngeal or superior laryngeal nerve leave the affected vocal fold fixed in the paramedian position. When bilateral, both folds are in the paramedian position. The folds are not completely abducted, because the intact cricothyroid maintains its adductor function and pulls the fold closer to the midline. In unilateral paralysis, dysphagia may be present, the cough and glottal coup can be weak, and 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 tracheotomy.



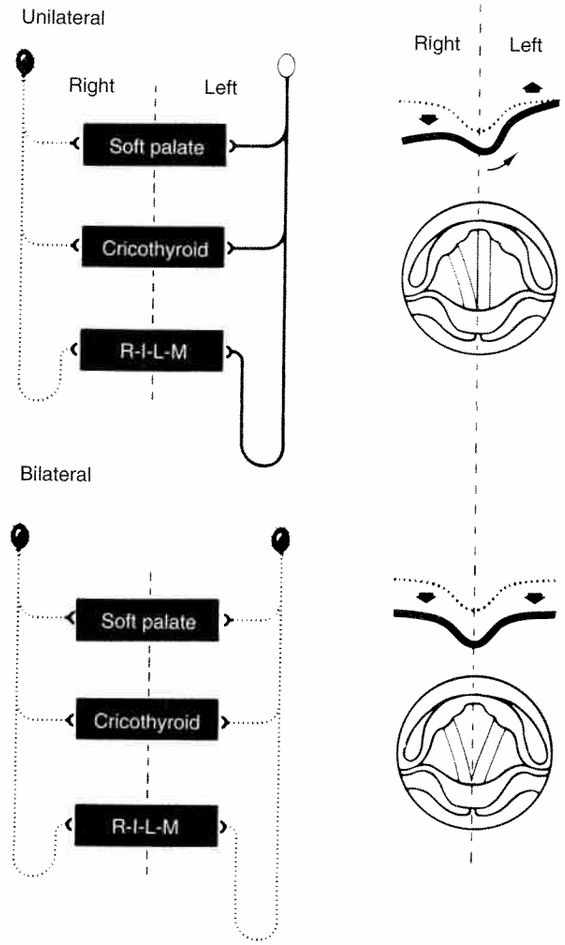


**FIGURE 4-3** Effects of unilateral (right) and bilateral cranial (vagus) nerve X lesions above the origin of the pharyngeal, superior laryngeal, and recurrent laryngeal branches of the nerve. When unilateral, the soft palate hangs lower on the right and pulls toward the left on phonation. The right 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 remain in the abducted position on phonation. (From Aronson AE: *Clinical voice disorders*, New York, 1990, Thieme, with permission).

Lesions affecting both the recurrent and superior laryngeal branches of the vagus leave the affected vocal folds 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 fold lesions.

### *Speech*

Table 4-3 summarizes the effects of unilateral and bilateral cranial nerve X 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 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 due solely to velopharyngeal incompetence, occluding the nares during speech facilitates intraoral pressure for articulation and aids assessment of the adequacy of the other articulators.

Unilateral lesions of cranial nerve X below the pharyngeal branch but including the superior and recurrent laryngeal branches can result in *breathiness* or *aphonia, hoarseness, reduced loudness, diplophonia, reduced pitch,* and *pitch breaks*. A *rapid vocal flutter* may be present during vowel prolongation. *Phrases may be short* because of air wastage through the incompletely adducted glottis during phonation; when glottal air wastage is substantial, *speaking on inhalation* is sometimes spontaneously adopted as a compensatory strategy. In bilateral paralysis these characteristics are usually 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 cause *breathy-hoarse voice quality, decreased loudness,* and sometimes *diplophonia* and *pitch breaks*. Bilateral weakness or paralysis causes *inhalatory stridor,* but the voice may be relatively unaffected because the folds are adducted close to the midline. *Airway compromise,* however, can be a serious problem.

#### *Acoustic and Physiologic Findings*

Videofluoroscopy or nasoendoscopy are useful for documenting weakness of the velopharyngeal valve during speech. Bilateral velopharyngeal weakness can be demonstrated by nasoendoscopy and by videofluoroscopy in lateral, frontal, and base views. Laryngoscopic examination is essential in cases with suspected vocal fold weakness, not only for diagnostic purposes but also for management considerations.

The visible characteristics of weak vocal fold activity have been described beyond simple observations of paralysis. Videostroboscopy and high-speed laryngeal photography in patients with unilateral vocal fold 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 fold approximation by the Bernoulli effect; irregular vocal fold vibration; exaggeration of the mucosal wave in the affected fold during phonation; and abnormal frequency and amplitude perturbations in vocal fold activity.[26,63] Greater vibratory amplitude and exaggerated mucosal waves are consistent with hypotonicity. These observations are consistent with the perception of breathiness (lack of firm glottal closure), hoarseness, and perhaps diplophonia associated with vocal fold weakness.

Aerodynamic studies of people with unilateral or bilateral vocal fold weakness have identified increased airflow rates during speech. These findings are consistent with neuromuscular weakness of the vocal folds, with subsequent incomplete vocal fold adduction and excessive air escape through the glottis during phonation.[9,26,60] Relatedly, it has been documented that dysarthric speakers with laryngeal "hypovalving" inspire considerably more volume of air per minute than normal speakers, mostly through increased breaths per minute; have mean speech duration per breath group that is considerably less than normal; expire more air than normal during pauses; and tend to have reduced pause frequency and duration, possibly secondary to poor vocal fold valving or a compensatory effort to increase speaking time.[60] People with inspiratory airway compromise (including unilateral and bilateral vocal fold paralysis) also have increased mean inspiratory duration during speech.[61] Many of these findings are consistent with the perception of breathiness and short phrases in people with laryngeal weakness. They also define some of the efforts that may be made to compensate for vocal fold 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 fold paralysis or weakness have documented the following characteristics: a breakdown of formant structure, reflected in a long-term average acoustic spectrum characterized by high $f_0$ amplitude with a marked drop-off of harmonics above the first formant; random noise in spectrograms and increased spectral energy levels in high-frequency regions, possibly reflecting turbulent airflow through a partially open glottis; and restricted standard deviation and range of fundamental frequency, suggesting reduced ability to reach upper pitch ranges.[23,43,51] Some studies have noted a relationship between some of these characteristics and perceptual judgments of breathiness and hypofunctional voice.[23,50] Findings of restricted $f_0$ range and variability[43] are consistent with Darley, Aronson, and Brown's (DAB's)[14] finding that monopitch is frequently perceived in flaccid dysarthrias.

Aerodynamic, acoustic, videofluoroscopic, and nasoendoscopic studies have repeatedly shown a relationship among velopharyngeal insufficiency (VPI) and hypernasality, nasal emission, and weak pressure consonants. Although most published studies have examined people with palatal clefts or undefined or mixed dysarthrias, their general findings can probably be generalized to those with velopharyngeal weakness associated with cranial nerve X lesions. In addition to increased nasal airflow with VPI, there are numerous acoustic correlates of listeners' perceptions of hypernasality. These include decreased energy and higher frequency of the first formant, change or shift in center frequencies of formants, increased formant bandwidth, reduced vowel intensity and dynamic intensity range, reduced vocal pitch range, and extra resonances.[12,30] Reduced formant and overall intensity probably reflect the damping characteristics of the nasal cavity. Finally, the connection of the pharyngeal tube to a side branching tube (nasal cavity) leads to the development of antiresonances in the spectrum (i.e., a sharp drop in intensity in a portion of the spectrum where energy is expected). Because these acoustic attributes are correlated with VPI and perceptions stemming from it, they represent quantifiable indices of velopharyngeal weakness that may be useful for documentation of deficits, comparisons over time, and the effects of management.

## Accessory Nerve (XI) Lesions

### *Course and Function*

The cranial portion of cranial nerve XI arises from the nucleus ambiguus, emerges from the side of the medulla, and exits the skull through the jugular foramen along with cranial nerves IX and X. It intermingles with fibers of cranial nerve X to help innervate the uvula, levator veli palatini, and intrinsic laryngeal muscles. The spinal portion arises from the first five to six cervical segments of the spinal cord, ascends and enters the posterior fossa through the foramen magnum, and then leaves the skull with fibers of cranial nerves IX and X, and cranial portion of cranial nerve XI, where it innervates the sternocleidomastoid and trapezius muscles.

### *Etiologies and Localization of Lesions*

Etiologies of lesions to the cranial portion of cranial nerve XI are similar to those described for cranial nerve X. The spinal portion can be damaged by lesions in the cervical spinal cord and by compression from lesions in the area of the foramen magnum. Radical neck surgery is another source of eleventh nerve lesions.

### *Nonspeech Oral Mechanism*

Lesions of the spinal portion of cranial nerve XI reduce shoulder elevation on the side of the lesion and weaken head turning to the side opposite the lesion. Such lesions do not generally affect speech. If bilateral weakness causes significant shoulder weakness and head drooping, then respiration, phonation, and resonance may be indirectly and mildly affected by the postural deficit.

Because it is clinically impossible to separate the effects of cranial nerve X lesions from those of lesions to the cranial portion of cranial nerve XI, and because some people argue that the cranial portion of cranial nerve XI is more appropriately considered part of cranial nerve X, it is unnecessary to treat cranial nerve XI as distinctly important to motor speech function.

## Hypoglossal Nerve (XII) Lesions

### *Course and Function*

Cranial nerve XII originates in the medulla. Its fibers exit the brainstem as a number of rootlets that converge and pass through the hypoglossal foramen just lateral to the foramen magnum. The nerve travels medial to cranial nerves IX, X, and XI in the vicinity of the common carotid artery and internal jugular vein and passes above the hyoid bone to reach the intrinsic and extrinsic muscles of the tongue.

Cranial nerve XII innervates all of the intrinsic and extrinsic muscles of the tongue, except the palatoglossus (cranial nerve X). It is crucial for

lingual articulatory movements, as well as chewing and swallowing.

### *Etiologies and Localization of Lesions*

Hypoglossal nerve lesions can be intramedullary, extramedullary, and extracranial. They can be caused by any condition that can affect the lower cranial nerves. Lesions to it often damage other cranial nerves, especially IX, X, and XI, but it can be damaged in isolation. Common causes of isolated hypoglossal lesions include infection and basilar skull or neck tumor, trauma, or surgery. Approximately 5% of carotid endarterectomies are associated with usually temporary hypoglossal nerve injury.[5] The nerve can also be damaged by carotid and vertebral artery aneurysms; carotid artery dissection; tumors in the neck, salivary glands, or base of the tongue; and radiation therapy.[8,34,47,59]

### *Nonspeech Oral Mechanism*

In unilateral hypoglossal lesions the tongue may be atrophic and shrunken on the weak side (see Figure 4-4). Fasciculations may be apparent. The tongue deviates to the weak side on protrusion, because the action of the unaffected genioglossus muscle is unopposed (Figure 4-5). The ability to curl the tip of the tongue to the weak side inside the mouth is diminished, as is the ability to push the tongue into the cheek against resistance. Voluntary tongue lateralization within the mouth occasionally yields paradoxical results, with the ability to push the tongue into the cheek on the weak side sometimes appearing normal. It may be that some people push the tongue to the weak side with the unaffected side instead of attempting to use the longitudinal fibers on the weak side to turn the tongue to the weak side.

With bilateral lesions the tongue may be atrophic bilaterally, with bilateral fasciculations. It may protrude symmetrically, but with limited range, or not at all. Lateralization and elevation may be impossible. Saliva may accumulate in the mouth, and food may squirrel in the cheeks. Patients may note an inability to move food around in the mouth and may alter their diet to accommodate to this problem. They may complain that the tongue feels "heavy," "thick," or "big," or that it does not move well for eating and speaking. Drooling can be related to lingual weakness.

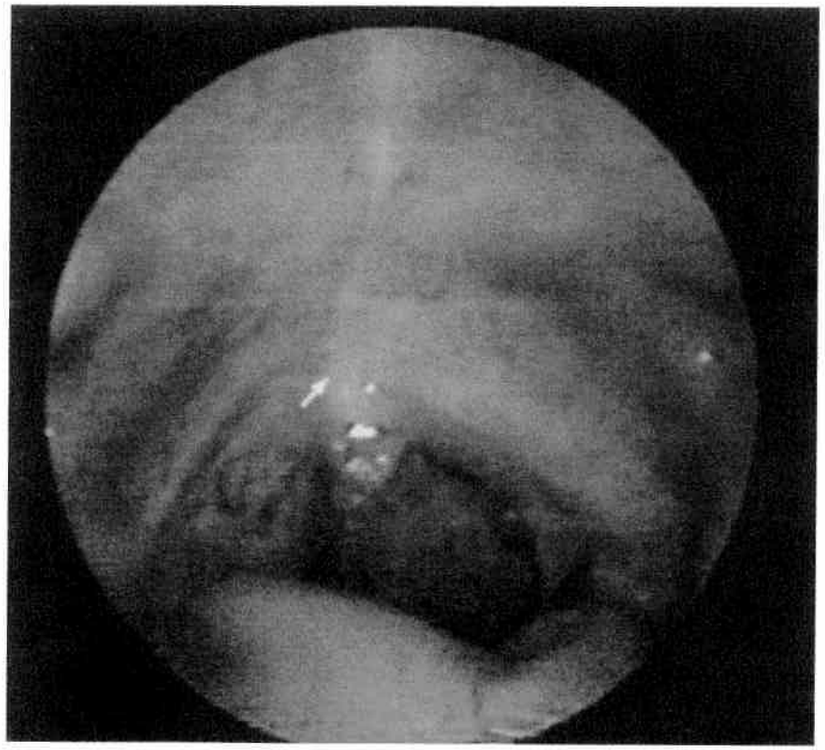

**FIGURE 4-4** Palatal movement during phonation in a patient with left palatal weakness. The palate pulls to the right. The *arrow* identifies the levator eminence (dimple), which is also displaced to the right. This patient also has left lingual weakness secondary to a left cranial (hypoglossal) nerve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



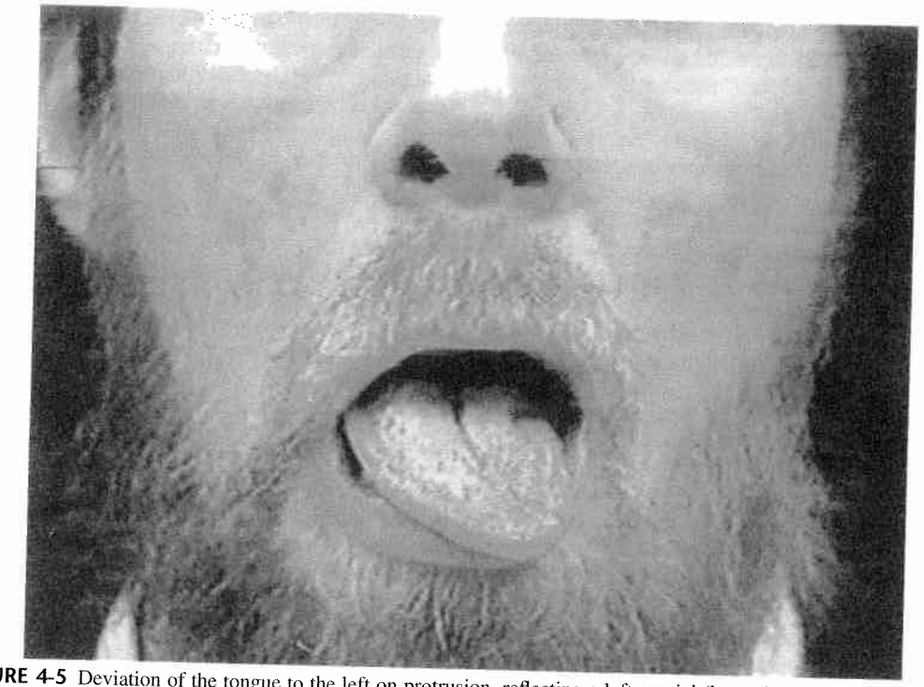

**FIGURE 4-5** Deviation of the tongue to the left on protrusion, reflecting a left cranial (hypoglossal) nerve XII lesion.

### *Speech*

The overriding speech characteristic in unilateral and bilateral cranial nerve XII lesions is *imprecise articulation* that can be isolated to lingual phonemes. Table 4-3 summarizes the effects of unilateral and bilateral cranial nerve XII lesions on speech.

Isolated unilateral twelfth nerve lesions are often compensated for to a degree that allows perceptually normal speech. Articulatory distortions are generally mild and do not affect intelligibility.

Bilateral lingual weakness causes difficulty with sounds requiring elevation of the tip or back of the tongue. When weakness is mild, anterior lingual consonant distortion is often detected more readily than velar distortions because of their greater number and frequency of occurrence in the language. The movements for the relatively difficult /s/, /ʃ/, /tʃ/ and their voiced cognates, as well as /r/ and /l/, are most susceptible to lingual weakness and may be the "first to go" when weakness develops. When weakness is pronounced, however, velars may be particularly devastated, perhaps because a greater mass must be moved to produce them relative to that for anterior lingual consonants.

Resonance differences are occasionally noted in people with bilateral lingual weakness. The altered resonance is sometimes labeled *hypernasality* or *hyponasality*, but this is probably inaccurate. Although the reason for resonance alterations is unclear, it may be that the weak tongue tends to fall back into the pharynx, altering the shape of the pharynx and, hence, resonance characteristics; reduced tongue movement reduces variability of oral cavity shapes during speech, thus reducing normal resonance variability, leading to a perception of abnormal resonance; or that atrophy alters the size of the oral and pharyngeal cavities, leading to resonance changes.

The most useful tasks for assessing lingual movement for speech are connected speech (including stress testing if MG is suspected) and speech AMRs. Connected speech places heavy demands on rapid, variable movements and may be most useful for identifying lingual distortions. If weakness is limited to the tongue, AMRs for "puh" should be normal, while those for "tuh" and "kuh" may be imprecise or slow. A noticeable mismatch in precision or rate between bilabial and lingual AMRs usually suggests isolated or relatively greater lingual weakness or, if the difference is in favor of lingual AMRs, isolated or relatively greater bilabial weakness. Imprecision and slowness for "kuh" generally exceeds that for "tuh" when the tongue is weak, possibly because

elevation of the back of the tongue, with its greater mass, places increased demands on strength (note, however, that AMRs for "kuh" are usually somewhat slower than "tuh" in normal speakers).

Speakers with bilateral lingual weakness often compensate well if other muscles are intact. For example, they may exaggerate jaw movement to facilitate lingual articulation, or they may restrict jaw movement to keep the tongue closer to articulatory targets in the maxilla. Compensatory exaggerated movements occasionally are mistaken for hyperkinetic movement disorders, although the physical mechanism examination usually clarifies the issue.

### *Acoustic and Physiologic Findings*

Studies have demonstrated reduced lingual strength or endurance in individuals with flaccid dysarthria[9,18] and, in some affected individuals, slower than normal lingual AMRs.[18] It is noteworthy, however, that lingual strength in dysarthric individuals may not be related to rate or ratings of intelligibility. It thus appears that tongue strength measures may be useful for quantifying lingual weakness but may not have a strong predictive relationship with speech rate or intelligibility. The lack of relationship between tongue strength and speech rate is consistent with the general perceptual impression that speech rate is noticeably reduced in flaccid dysarthrias only when weakness is quite severe.

## Spinal Nerve Lesions

### *Course, Function, and Localization of Lesions*

Upper cervical spinal nerves supplying the neck are indirectly implicated in voice, resonance, and articulation (see discussion of the spinal component of cranial nerve XI). Effects on speech of lesions to these nerves are indirect, usually mild, and poorly understood.

Spinal nerves more directly involved in respiration are spread from the cervical through the thoracic divisions of the spinal cord. Those supplying the diaphragm arise from the third through fifth cervical segments. They combine to form the phrenic nerves, each of which innervates half of the diaphragm, the most important inspiratory respiratory muscle. Remaining inhalatory muscles are supplied by branches of the lower cervical nerves, intercostal nerves, and phrenic nerves. Muscles of forced exhalation, important for control of exhalation during speech, are innervated by motor fibers of the thoracic and intercostal nerves.

Diffuse impairment of spinal nerves supplying respiratory muscles is required to interfere significantly with respiration. The exception is damage to the third through fifth segments of the cervical spinal cord that can paralyze the diaphragm bilaterally and severely compromise breathing.

### *Etiologies*

Spinal cord injuries above C3 can isolate the respiratory muscles from the brainstem respiratory control centers and cause respiratory paralysis. Diseases such as MG, ALS, Guillain-Barré syndrome, and spinal cord injuries affect respiration by weakening muscles or interfering with their innervation.

### *Nonspeech Oral and Respiratory Mechanisms*

Compromised respiratory nerve function can result in rapid, shallow breathing. Flaring of the nasal alae and use of upper chest and shoulder neck muscles to elevate and enlarge the rib cage suggest respiratory compromise. Chest wall and abdominal expansion may be visibly restricted during inhalation, and patients may be unable to hold their breath for more than a few seconds. They may be unable to generate or sustain subglottal air pressure sufficient to support speech as measured by a U-tube or water glass manometer.

### *Speech*

Flaccid dysarthria due to isolated respiratory disturbance is uncommon in many speech pathology practices. It is not clear if this is because the incidence of such disturbances is low, if such patients rarely complain of the effects of such disturbances on speech or spontaneously compensate for them, or if the respiratory compromise for basic life support is so overriding that its effect on speech is of low priority to the patient and his or her medical caregivers. Such speech problems certainly exist and have been described in published reports.[27,28] Patients with respiratory weakness sufficient to affect speech usually also have weakness that interferes with quiet breathing or breathing during other physical activities. These deficits have usually been identified before speech examination. Table 4-3 summarizes the effects of respiratory weakness on speech.

Respiratory weakness reduces the amount and force of expelled air. Reduced vital capacity and control of expiration can result in *short phrases* and *reduced loudness*. Prosodic abnormalities secondary to *altered phrasing* may result, as may *decreased pitch and loudness variability*. Such problems are not universally present, but are not uncommon. For example, in a study of 10 adults with cervical spinal cord injury, three were perceived as normal speakers; three had reduced loudness; two were breathy; two had short phrases; and one had prolonged inspiration, which presumably affected prosody.[28]



Patients with respiratory weakness may inhale with obvious effort, sometimes raising their shoulders and extending their neck in compensation for diaphragmatic weakness. They may attempt to speak on residual air, which may cause the voice to actually sound *strained*, probably secondary to efforts to achieve vocal fold adduction with limited subglottic pressure or to maximize efficient use of the restricted respiratory supply. Many of these characteristics are also evident in people with severe asthma, chronic obstructive pulmonary disease, and other respiratory disturbances of a nonneurologic nature. Finally, inability to extend the duration of exhalation for normal phrase length in speech leads some patients to *speak on inhalation*.

Respiratory weakness in combination with cranial nerve weakness in flaccid dysarthrias is not unusual, and distinguishing between phonatory and prosodic abnormalities due to respiratory versus laryngeal weakness can be difficult. Some clues that help to identify which level is more involved include:

1. Gasping for air, nares flaring, shoulder elevation, and neck retraction on inhalation during speech are rare in isolated laryngeal weakness but not uncommon in respiratory weakness.
2. Patients with isolated laryngeal adductor weakness do not complain of shortness of breath at times other than during speech. Those with respiratory weakness do.
3. Patients with isolated respiratory weakness may have reduced loudness and breathy or strained voice quality but not hoarseness, harshness, or diplophonia. Those with laryngeal weakness are frequently hoarse or harsh and sometimes diplophonic.
4. Patients with greater laryngeal than respiratory weakness may have a glottal coup that is less adequate than their cough (good respiratory force during coughing may overcome vocal fold weakness). The opposite can occur when respiratory weakness exceeds laryngeal weakness (less respiratory force is required for a glottal coup than cough).

### *Physiologic Findings*

Acoustic and physiologic studies of speech in people with isolated respiratory weakness are few. Hixon, Putnam, and Sharp[27] conducted a detailed kinematic analysis of respiratory movements in a man with flaccid paralysis of respiratory muscles. He had considerable capacity for compensatory speech respiratory activities in the form of "neck breathing" and "glossopharyngeal breathing" (discussed in Chapter 17) The data support contentions that reduced vital capacity need not result in speech difficulty if valving of the air stream can be made more efficient.

Hoit et al.[28] documented abnormal chest wall movement consistent with loss of abdominal muscle function in individuals with cervical spinal injury. They also found speech breathing patterns that reflected compensations for expiratory muscle weakness. Speakers inspired to larger lung and rib cage volumes (they inhaled more deeply) and terminated speech at larger volumes than nonimpaired speakers, presumably to take advantage of higher elastic recoil pressure at those volumes that could drive the upper airway and larynx during phonation. Speakers also used larger lung volumes when asked to increase loudness. These compensatory strategies were developed spontaneously in most cases.

## Multiple Cranial Nerve Lesions

When several cranial nerves are damaged, the condition is often referred to as *bulbar palsy*. Damage to more than a single cranial nerve is not unusual. The jaw, face, lips, tongue, palate, pharynx, and larynx can be affected in varying combinations and to varying degrees depending on the particular cranial nerves involved and whether damage is unilateral or bilateral.

Conditions that affect multiple cranial nerves tend to be associated with intracranial pathology. This is because the smallest lesion that can do the most damage is in the brainstem where the cranial nerves are closer together than anywhere else along their course. This is not always the case, however, because multiple cranial nerves may be involved in neuromuscular junction diseases (e.g., myasthenia gravis), and myopathies can affect muscles in the distribution of more than one cranial nerve.

### *Etiologies*

Multiple cranial nerve involvement can be caused by many of the same conditions that affect single cranial nerves. Multiple rather than single cranial nerve involvement is more common in certain diseases, however, including ALS, MG, and brainstem vascular disturbances or tumors.

### *Nonspeech Oral Mechanism*

Clinical examination findings for patients with multiple cranial nerve involvement are no different than those with damage to single cranial nerves. The cumulative effects on function, however, can be much more devastating than the effects of single cranial nerve lesions.

**table 4-5** Distribution of involvement of cranial nerves V, VII, X, and XII, and spinal respiratory nerves, in 151 quasirandomly selected cases with a primary speech diagnosis of flaccid dysarthria at the Mayo Clinic from 1969-1990 and 1999-2001. Number of instances in which each nerve was the only speech nerve involved, and the number of instances in which each nerve was involved along with other speech nerves, are given. Forty-three percent of the cases had isolated unilateral or bilateral involvement of a single cranial nerve. Fifty-seven percent had more than one cranial nerve involved. As a result, the total number of different nerves reported is 221.

| Nerve | Isolated Unilateral | Isolated Bilateral | Multiple Unilateral | Multiple Bilateral | Percent of Total |
|---|---|---|---|---|---|
| V | — | — | 2 | 4 | 3 |
| VII | — | 3 | 5 | 22 | 14 |
| X—Pharyngeal branch only | — | 2 | — | 2 | 2 |
| Laryngeal branch(es) only | 52 | 7 | — | 4 | 29 |
| All branches | 10 | 11 | 5 | 35 | 28 |
| XII | 3 | 6 | 10 | 33 | 24 |
| Respiratory | — | 1 | — | 4 | 2 |
| Percent of total | 29 | 14 | 10 | 47 | 100 |

### *Speech*

Deviant speech characteristics associated with multiple cranial nerve lesions are similar to those associated with isolated cranial nerve damage, but the effects are heard in combination. Consequently, they may be more difficult to isolate. In general, the dysarthria is perceived as more severe than in single cranial nerve lesions, but this is not always the case, especially if the measure of severity is intelligibility. For example, a bilateral lesion of the facial nerve could have a greater impact on intelligibility than combined unilateral lesions of cranial nerves V, VII, and X. In general, effective compensatory strategies for maintaining intelligibility are more difficult when multiple cranial nerves are involved than when impairment is to only a single cranial nerve.

## Distribution of Speech Cranial Nerve Involvement in Flaccid Dysarthrias

The distribution of cranial nerve involvement in the population of people with flaccid dysarthrias is unknown, but a retrospective review of cases seen in a large medical setting provides clues about the distribution encountered in some practices.

Table 4-5 summarizes the distribution of involvement of cranial nerves V, VII, X, and XII in 151 of 154* of the cases whose etiologies are summarized in Box 4-1. Cautious interpretation should be exercised regarding the representativeness of these data for the general population or for all speech pathology practices. In addition, these data represent speech pathologists' judgment about the contribution of cranial nerve weakness to the dysarthria and not necessarily all of the cranial nerves that might have been involved (e.g., unilateral cranial nerve V weakness was not included if it did not appear relevant to the speech deficit).

Several characteristics of the distribution are of interest. First, cranial nerve V and respiratory contributions to flaccid dysarthrias were infrequent. This probably means that they are usually not affected in flaccid dysarthrias or are not often judged to contribute to deviant speech characteristics. Cranial nerves VII and XII were involved much more frequently, and cranial nerve X more often than any other speech cranial nerve. Among the branches of cranial nerve X, the pharyngeal branch was only infrequently implicated without suspected involvement of the superior or recurrent laryngeal branches. In contrast, the laryngeal branches were frequently implicated without pharyngeal branch involvement; this reflects the high frequency of surgery-related or idiopathic vocal fold paralyses below the pharyngeal branch of cranial nerve X. Finally, more than 40% of the sample had unilateral or bilateral involvement of a single cranial nerve (most often cranial nerve X). The majority of the sample had unilateral or bilateral involvement of more than one cranial nerve.

*The three missing cases were insufficiently documented to identify specific cranial nerves.

## Clusters of Deviant Speech Dimensions

DAB[16] found three clusters of deviant dimensions among 30 patients with bulbar palsy. These clusters are useful in understanding the presumed neuromuscular deficits, the components of the speech system that are most prominently involved, and features of flaccid dysarthrias that distinguish it from other dysarthria types (Table 4-6).



The first cluster was *phonatory incompetence*. It included *breathy voice, audible inspiration,* and *short phrases*. The cluster represents incompetence at the laryngeal valve, including inadequate vocal fold adduction (breathiness due to inadequate vocal fold adduction, as well as short phrases due to air wastage through the glottis) and abduction (audible inspiration due to inadequate vocal fold abduction during inspiration).

The second cluster was *resonatory incompetence*. It included *hypernasality, nasal emission, imprecise consonants,* and *short phrases*. The relationships among these features reflect weakness of the velopharyngeal valve leading to excessive nasal resonance (hypernasality) and nasal air flow during attempts to produce consonants requiring intraoral pressure (nasal emission). Imprecise consonants in this cluster reflect the secondary effect of nasal emission on pressure consonant precision. Short phrases reflect the effect of air wastage through the velopharyngeal port during speech.

The final cluster was *phonatory-prosodic insufficiency*. It consisted of *harsh voice, monopitch,* and *monoloudness*. DAB felt these characteristics reflected hypotonia in laryngeal muscles. This hypothesis receives support from acoustic and physiologic studies and from direct observation of weak or paralyzed vocal folds.

The phonatory and resonatory incompetence clusters are especially important for differential diagnosis, because they were not found in other dysarthria types. Thus the presence of phonatory or resonatory incompetence is suggestive of flaccid dysarthria and implicates LMN weakness at the laryngeal and velopharyngeal valves (cranial nerve X). The third cluster, phonatory-prosodic insufficiency, is of less value to differential diagnosis, because it is also found in other dysarthria types.

The reader may be struck by the restriction of these clusters to cranial nerve X abnormalities. This does not mean that speech abnormalities attributable to weakness of other cranial nerves do not occur in flaccid dysarthrias, nor does it imply that recognition of other abnormalities is not helpful to diagnosis. The absence of obvious effects of other cranial nerves in the cluster analysis of DAB probably reflects several influences. First, the distribution of cranial nerve involvement in their sample (and those with flaccid dysarthrias in general, as suggested by the findings summarized in Table 4-5) may have been biased toward cranial nerve X lesions. Second, the grouping of all articulatory deficits under the global designation of imprecise consonants and vowel distortions may have masked the specific effects of cranial nerve V, VII, and XII lesions on speech. Third, imprecise consonants can occur in all dysarthria types, so their presence is not likely to be distinctive within clusters that distinguish among types of dysarthria. Finally, the primary purpose of

**table 4-6** Clusters of abnormal speech characteristics in flaccid dysarthrias

| Cluster Name | Speech Characteristics |
|---|---|
| Phonatory Incompetence | Breathiness, short phrases, audible inspiration |
| Resonatory Incompetence | Hypernasality, imprecise consonants, nasal emission, short phrases |
| Phonatory-prosodic Insufficiency | Harsh voice, monoloudness, monopitch |

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

**table 4-7** Most deviant speech characteristics encountered in flaccid dysarthrias by Darley, Aronson, and Brown,[15] listed in order from most to least severe. Also listed are the cranial nerves and muscle groups most likely associated with the deviant speech characteristics.

| Dimension | Primary Cranial Nerve | Level |
|---|---|---|
| Hypernasality* | X | Velopharyngeal |
| Imprecise consonants | | Articulatory |
| | V | • Jaw |
| | VII | • Face |
| | X | • Velopharyngeal |
| | XII | • Tongue |
| Breathiness (continuous)* | X | Laryngeal |
| Monopitch | X | Laryngeal |
| Nasal emission* | X | Velopharyngeal |
| Audible inspiration* | X | Laryngeal |
| Harsh voice quality | X | Laryngeal |
| Short phrases* | X | Laryngeal or respiratory |
| | Spinal respiratory | |
| Monoloudness | X | Laryngeal or respiratory |
| | Spinal respiratory | |

*Tend to be distinctive or more severely impaired in flaccid dysarthrias than any other single dysarthria type.

**table 4-8** Summary of direct observations and acoustic and physiologic findings in flaccid dysarthrias (based on literature summarized in text). Some findings may reflect efforts to compensate for weakness and not just the primary effects of weakness.

| Level | Direct, Acoustic, and Physiologic Observations |
|---|---|
| **Respiratory** | Reduced vital capacity |
| | Termination of speech at larger than normal lung volumes* |
| | Larger than normal inspiratory and rib cage volumes* |
| | Abnormal chest wall movements* |
| | Neck and glossopharyngeal breathing* |
| **Laryngeal or Respiratory** | Vocal fold immobility or sluggishness (unilateral or bilateral) |
| | Incomplete glottal closure (unilateral or bilateral) |
| | Abnormal vocal fold frequency and amplitude perturbations |
| | Increased amplitude of vocal fold mucosal wave (unilateral or bilateral) |
| | Increased airflow rate |
| | Increased inspiratory volume* |
| | Increased breaths per minute* |
| | Reduced pause frequency and duration* |
| | Reduced speech duration or syllables per breath group* |
| | Reduced range and variability of $f_o$ |
| | High amplitude of $f_o$ with reduced energy of harmonics |
| | Reduced formant intensity and definition |
| | Increased high-frequency spectral energy (noise) |
| **Velopharyngeal†** | Reduced or absent palatal movement (unilateral or bilateral) |
| | Reduced or absent pharyngeal wall movement (unilateral or bilateral) |
| | Increased nasal airflow |
| | Decreased energy in $f_o$ |
| | Increased frequency of $f_o$ |
| | Reduced pitch range |
| | Increased formant bandwidth |
| | Reduced overall intensity and intensity range |
| | Extra resonances |
| | Antiresonances |
| **Lingual** | Reduced sustained lingual force |

*Compensatory or possibly compensatory.
†Includes findings from studies of velopharyngeal incompetence associated with cleft palate.

the studies by DAB focused on distinctions among dysarthria types rather than the differential effects on speech of damage to specific cranial nerves within a specific dysarthria type (i.e., flaccid dysarthrias).

The important point here is that investigating the functions of each cranial nerve and the loci of specific speech characteristics is important to examination, description, and diagnosis. Also, because flaccid dysarthrias can be manifest by damage to only a single cranial nerve, and because other dysarthrias are rarely manifest through a single cranial nerve, identification of the offending muscle group is important to differential diagnosis and treatment decisions.

Table 4-7 summarizes the most deviant speech characteristics that were found by DAB in their patients with flaccid dysarthria.[15] The cranial or spinal nerve and the component of the speech mechanism that is most likely implicated in the production of each of the characteristics are also given. Table 4-8 summarizes the acoustic and physiologic correlates of flaccid dysarthrias that were reviewed within the discussion of deficits associated with each of the speech cranial nerves.



## Cases

The following cases review the histories, examination findings, and diagnoses for several patients with flaccid dysarthria. As a group, they illustrate some of the similarities and differences that exist among people with this type of dysarthria. Several cases illustrate the prominence of speech deficits in neurologic disease and the importance of speech diagnosis to medical or neurologic diagnosis.

### Case 4-1

A 44-year-old woman presented with an 8-month history of speech difficulty that she thought was caused by ongoing stress. The neurologic examination was normal. The neurologist was uncertain but wondered if her complaint was stress related. Speech pathology consultation was requested.

During speech evaluation the patient said that her speech deteriorated when she was tired or under stress, and that it frequently changed while she was coaching a volleyball team. She described it as "slurred, almost like my mouth freezes . . . almost sounds like it goes nasal." She vaguely described an alteration of chewing and swallowing at such times but denied choking or drooling. The speech problem would persist until she rested. Her primary sources of stress were a busy schedule caring for her three school-age children and coaching a high school volleyball team. She described her family life and work as stable and happy but busy.

Speech was initially normal. After 6 minutes of continuous reading aloud, mild sibilant distortions became apparent, along with equivocal hoarseness and intermittent vocal flutter. Speech AMRs were normal, and she did not become hypernasal, but inconsistent nasal airflow was detected on a mirror held at the nares during repetition of nonnasal sounds and phrases. After another 4.5 minutes of reading, she began to interdentalize /s/ and /z/, distort affricates, and mildly distort /r/. AMRs remained normal, and hypernasality was not perceived. The oral mechanism examination immediately following stress testing demonstrated only equivocal lingual weakness. The patient was upset and cried when her speech changed, making it difficult to separate the effects of her emotional response from weakness. Speech returned to normal after 30 seconds of rest.

She was asked to return the following day at 5 PM, following volleyball practice. Although speech was initially normal, it deteriorated quickly and significantly, but its character was the same as that noted the day before. In addition, pitch breaks and some fluttering of the cheeks during speech were apparent.

The speech diagnosis was "flaccid dysarthria characterized by weakness of, at the least, cranial nerves VII, X, and XII, bilaterally, with rapid deterioration with stress testing, consistent with the pattern of breakdown seen in myasthenia gravis." Subsequent EMG and improvement of her symptoms with Mestinon treatment confirmed the diagnosis of MG. She subsequently did well with Mestinon treatment.

**Commentary.** (1) Speech difficulty can be the first sign of neurologic disease. (2) The presence of psychologic distress at the onset of speech difficulty is insufficient proof of psychogenic etiology. Patients often attribute their physical problem to stress when neurologic disease presents insidiously. In such cases, neurologic and psychologic factors deserve equal attention until a clear cause emerges. (3) Speech diagnosis can localize disease in the motor system. In some cases, speech diagnosis provides strong evidence for a specific neurologic diagnosis.*

*An informative case of a person with MG masking as stroke can be found in Duffy.[17]

## Case 4-2

A 37-year-old man presented with a complaint of speech difficulty, problems with "tongue control," and headache and neck pain of 2 months' duration. He described his speech as "slurred" and complained of excess saliva accumulation and difficulty moving food with his tongue.

Oral mechanism examination identified a bilaterally atrophic tongue but no fasciculations. He was barely able to move his tongue in any direction, and tongue strength was rated -4 bilaterally. Saliva pooled in his mouth. Phonation and resonance were normal, as were AMRs for "puh" and "tuh," but those for "kuh" were equivocally slowed and reduced in precision. Lingual sounds were distorted. Nonlingual sounds, rate, and prosody were normal. Jaw and facial movements during speech were exaggerated in apparent compensation for his lingual weakness. Intelligibility was good.

Neurologic examination was otherwise normal except for mild weakness of neck flexor muscles. Skull radiographs showed destruction of the interior portion of the clivus (the bony part of the posterior fossa anterior to the foramen magnum) and an associated nasopharyngeal soft-tissue mass. Magnetic resonance imaging (MRI) and computed tomography (CT) scans identified a destructive tumor mass in the anterior rim of the foramen magnum bilaterally. The patient underwent neurosurgery for radical subtotal removal of a chordoma tumor of the clivus. Postoperatively, articulatory imprecision was mildly worse, but no other speech deficits developed. The patient underwent radiation therapy, and his speech gradually improved, though not to normal. Lingual atrophy and weakness persisted. He did well but 2 years later developed headache, nausea, vomiting, and double vision. There was evidence of tumor recurrence, but further radiation therapy or surgery was not advised because of risks and unlikely benefit. The patient lived outside of the geographic area of treatment and was not seen for further follow-up.

**Commentary.** (1) Flaccid dysarthrias can be caused by damage to a single cranial nerve, unilaterally or bilaterally. (2) Speech difficulty can be the first sign of neurologic disease. (3) Speech intelligibility can be remarkably preserved in isolated bilateral tongue w eakness.

## Case 4-3

A 40-year-old millwright presented with an 8-month history of voice difficulty. His dysphonia began after anterior-approach cervical disk surgery. He had been unable to return to work, because coworkers were unable to hear him in the noisy work environment. He occasionally coughed and choked after swallowing and had to clear his throat frequently.

Speech and oral mechanism examination were normal except for a markedly breathy-hoarse voice quality, moderately decreased loudness, and short phrases secondary to presumed air wastage through the glottis. He could sustain "ah" and "z" for only 2 seconds but sustained "s" for 12 seconds. His cough and glottal coup were markedly weak. There was no evidence of palatal asymmetry, the palate was mobile, and the gag reflex was normal.

The speech pathologist's impression was "suspect vocal cord paralysis secondary to recurrent laryngeal nerve damage caused by surgical trauma." Subsequent laryngeal examination identified a right vocal fold paralysis (paramedian position) and agreed it was probably secondary to surgical trauma. Teflon injection (rarely used currently) of the right vocal fold resulted in normal conversational loudness, ability to sustain "ah" for 14 seconds, /s/ for 12 seconds, and /z/ for 10 seconds. The patient remained unable to produce a loud, shouting voice. He was, however, pleased with his voice improvement and returned to work as a millwright, although with some fatigue in his voice by the end of the workday.

**Commentary.** (1) Flaccid dysarthrias can result from damage to a single cranial nerve. (2) Flaccid dysarthrias can be caused by surgical trauma. (3) The degree of impairment perceptually does not always predict the impact of the problem on a person's day-to-day functioning (this patient could not work). (4) Some speech deficits can be managed effectively with medical intervention.



## Case 4-4

A 76-year-old mildly retarded man presented with a 10- to 11-week history of speech and swallowing difficulty. A swallowing study conducted elsewhere was normal. An ear, nose, and throat (ENT) examination was normal. The physician thought the patient might have amyotrophic lateral sclerosis. He was referred for speech and neurologic examinations.

Speech examination the following day was difficult because of the patient's immature affect, anxiety, and difficulty following directions. He did report that his swallowing problem was present upon awakening one morning and that his speech difficulty followed 1 to 2 days later. He had greater difficulty swallowing food than liquids but he did have nasal regurgitation when swallowing water. He thought all of his problems had worsened since onset.

Oral mechanism examination revealed left ptosis and difficulty closing both eyes completely. His face was moderately weak bilaterally. The tongue was tremulous on protrusion, but there were no fasciculations or atrophy; it was -2,3 weak bilaterally. Palatal movement gradually decreased over repetitions of "ah ah ah . . . ." There was consistent nasal air escape during speech. There was some reduction in speed and range of motion during alternating retraction and pursing of the lips. Cough and glottal coup were weak. Gag reflex was normal.

Speech examination was difficult because of his anxiety and difficulty following directions. However, the following characteristics were apparent: hypernasality (3), weak pressure consonants (3,4), imprecise articulation (2), and reduced rate (0,1). Prolonged "ah" was breathy (0,1), and inhalatory stridor was apparent following maximum vowel prolongation. The patient prolonged "ah" for 20 seconds initially, but over multiple trials this decreased to 12 seconds. It was difficult to get him to persist in speaking for stress testing, but hypernasality and weak pressure consonants appeared to increase over time.

The speech pathologist's impression was "flaccid dysarthria implicating, at the least, cranial nerves X, XII, and VII, bilaterally. There is no evidence of a spastic dysarthria or other CNS-based dysarthria. There is some deterioration of speech during stress testing, raising suspicions about neuromuscular junction disease (does this patient have MG?)."

Subsequent clinical neurologic examination, EMG, and an ACh receptor antibody test confirmed a diagnosis of MG. The patient improved rapidly when treated with Mestinon, but within 3 months his bulbar symptoms worsened and he developed respiratory compromise. He died 1 month later.

**Commentary.** (1) Speech difficulty can be among the first signs of neurologic disease. (2) Careful speech examination often is more enlightening than anatomic examination of speech structures. (3) The presence of cognitive deficits can make examination difficult. (4) The value of accurate localization and disease diagnosis by speech examination, unfortunately, is not always matched by long-term benefit to the patient.

## Case 4-5

A 45-year-old man presented with a 3-month history of dysphagia that began with a choking episode, followed by continuing difficulty swallowing solid foods but not liquids. Speech difficulty, which he described as "slurring" and "difficulty with pronunciation," began approximately 1 month later. The neurologic examination was normal with the exception of possible palatal and tongue weakness. EMG failed to find evidence of neuromuscular junction disease but did find an abnormality of the hypoglossal nerve or its nuclei. MRI scan failed to find evidence of abnormality in the brainstem or posterior fossa. A video swallow study was normal. ENT examination was normal.

During speech evaluation, he complained of some dull, aching pain in his ears, tongue, jaw, and gums that he attributed to increased effort to chew food completely before swallowing. He noted mild difficulty with chewing and a tendency to put food to the left in his mouth. He felt he was able to initiate a swallow but often gagged and had to bring food back up and reinitiate a swallow. He was not aware of drooling during the day but noted that his pillow was frequently wet upon awakening in the morning.

During the examination, he cleared his throat frequently. Jaw strength was normal. There was equivocal weakness of lip rounding. The tongue was moderately weak bilaterally. Tongue protrusion and lateralization were limited (2,3), and there were equivocal fasciculations on the right side of the tongue. The palate elevated more extensively toward the right. There was a trace of nasal emission during pressure sound production. Cough and glottal coup were normal. Speech was characterized

## Case 4-5—cont'd

by imprecise articulation, primarily on lingual consonants (0,1) and by hypernasality with occasional audible nasal emission (1). Voice quality was hoarse-breathy (0,1). He was able to sustain a vowel for 25 seconds. Speech AMRs for "puh" and "tuh" were normal, but "kuh" was slow (1). There was no significant deterioration of speech during stress testing.

The clinician's impression was "flaccid dysarthria associated with, at the least, weakness of cranial nerves XII and X, most likely bilateral. There was no significant deterioration of speech during stress testing, as might be encountered in MG. Finally, I hear no evidence to suggest the presence of a spastic component to his dysarthria."

All other laboratory and imaging tests, including tests for MG, were normal. The patient received counseling for management of his dysphagia and was discharged. He returned 3 months later complaining of increased dysphagia and tongue pain. ENT examination revealed a tender, swollen tongue. CT scan of the head and neck identified a mass extending posteriorly from the posterior aspect of the left superior tongue. Subsequent surgery identified extensive squamous cell carcinoma of the tongue with neck metastases. Right and left neck dissection and total glossectomy and laryngectomy were carried out.

**Commentary.** (1) Speech difficulty can be among the first signs of neurologic and other organic disease. (2) The apparent involvement of more than one cranial nerve does not always place the lesion inside the skull, even when muscle disease and neuromuscular junction disease are not present. (3) Neurologic signs and symptoms do not always mean the patient has primary nervous system disease. Although cranial nerves were affected, the neoplasm in this case was nonneurologic.

## Case 4-6

A 24-year-old farmer was hit by a falling piece of heavy farm machinery. He sustained complex skull base, bilateral petrous ridge, and bilateral carotid canal fractures. The accident caused bilateral otorrhea, cranial nerve V palsy, and bilateral cranial nerve VII palsies. EMG and nerve conduction studies demonstrated near-complete paralysis of both cranial nerves VII, with some fibrillation potentials. Surgical management of cranial nerve VII palsies was deferred in the hope that there would be spontaneous regeneration.

The patient initially had significant difficulties with chewing and speech, primarily because he was unable to open his jaw. When seen for speech examination approximately 1 month after onset, his restricted jaw movement had cleared, and he no longer had any chewing or swallowing complaints. He admitted, however, that liquids would sometimes escape his mouth. He recognized that his speech difficulty was related to his facial weakness, but he did not feel that people were having significant difficulty understanding him. He complained that his mouth and lips would get dry easily and that he frequently needed to protrude his tongue to moisten his lips.

Oral mechanism examination was normal with the exception of bilateral facial paralysis. Relative to lower face movements relevant to speech, he was completely unable to make any isolated lip movements toward retraction or rounding. Attempts to puff his cheeks resulted in some flutter of the lips due to air escape. He was able to approximate his lips with his jaw closed. Conversational speech was characterized by distortion of all bilabial and labiodental sounds. He had some mild distortion of anterior lingual fricatives and affricates that the clinician felt was secondary to his facial weakness. There occasionally was mild distortion of /r/ in phonemic environments requiring lip rounding. He did achieve some lip approximation for bilabial sounds, and bilabials and labiodentals were distorted rather than omitted. Speech intelligibility was remarkably adequate in the evaluation setting, although it was felt that it would be mildly reduced in some phonetic environments or under adverse environmental conditions.

It was concluded that the patient had a flaccid dysarthria that was consistent with his bilateral cranial nerve VII paralyses. There was no evidence of speech difficulty that could not be explained by his bilateral facial nerve paralyses. He compensated well, primarily with jaw movement, for his facial weakness.

The patient received training for exercises to help promote lower facial movement and was instructed to do them twice daily. Speech exercises included materials with consonant-vowel syllables containing /b/, /p/, and /m/ sounds. He was not seen for further follow-up in speech pathology, but his records showed that within 2 months he began to have some recovery of both facial nerves. Approximately 11 months later, he had made further recovery, with mild to moderate persistent weakness. Nearly 2 years after onset, he had made further recovery, but bilateral facial weakness was still evident.



## Case 4-6—cont'd

**Commentary.** (1) Bilateral facial weakness can cause articulatory imprecision for phonemes requiring facial movement. (2) When a single cranial nerve is damaged, even if bilaterally, considerable compensation is possible if paralysis is not complete and other cranial nerves are functioning normally. (3) The specific speech deficits encountered in flaccid dysarthria depend on the specific cranial nerves that are involved. In this case, all of the patient's speech distortions could be explained by his bilateral facial weakness. (4) Oromotor exercise to improve strength is sometimes justified for people with flaccid dysarthria. In this case, however, it is not possible to conclude that such exercises were responsible for improved strength or speech.

## Case 4-7

A 62-year-old woman presented with an 8- to 10-year history of mild swallowing difficulties and a 2- to 3-year history of speech problems. Her history was significant only for radiation treatment to the face for acne at age 13. Clinical neurologic examination was normal with the exception of bilateral weakness in the face, tongue, and sternocleidomastoid muscles.

Speech pathology evaluation revealed normal symmetric jaw movement and strength. The lower face was lacking in tone, but lip retraction and rounding were grossly normal. The tongue was full and symmetric, without atrophy or fasciculations, but it was mild to moderately weak bilaterally. Lateral lingual AMRs were slow. Palatal movement was symmetric, and cough and glottal coup were normal. There were no pathologic oral reflexes. The patient's speech was characterized by equivocally slowed rate and imprecise articulation, particularly for anterior lingual fricatives, liquids, and bilabial sounds. There was some fluttering of the cheeks during production of bilabials. She had some exaggerated lip movements during speech that were judged to be compensatory. Voice quality was normal. Speech AMRs and sequential motion rates were normal. Speech intelligibility was normal.

The speech pathologist concluded that the patient had a "mild flaccid dysarthria whose deviant speech characteristics are consistent with facial and lingual weakness." The clinician stated, "I do not hear anything in her speech to suggest significant weakness in muscles in the distribution of cranial nerves V, IX, X, or XI. I do not hear anything to suggest the presence of a spastic component to her dysarthria, or any other CNS-based dysarthria." It was felt that the patient was compensating adequately for her mild dysarthria. Speech therapy was not recommended.

After a comprehensive neurologic workup, it was concluded that the most likely cause of the patient's cranial and peripheral nerve deficits was her radiation treatment.

**Commentary.** (1) Flaccid dysarthria can develop in response to radiation-induced cranial nerve weakness. Such effects can be delayed for many years following radiation treatment. (2) Speech evaluation can help rule out certain neurologic diagnostic possibilities. In this case, it was possible to state that there was no evidence of any CNS-based dysarthria and that the speech deficit reflected LMN involvement alone. (3) Speech therapy for dysarthria is not always necessary. In this case, the patient was compensating very well for her impairments and had no difficulty with intelligibility or efficiency of verbal communication. Her need was to establish the etiology of her mild speech and swallowing difficulty.

## SUMMARY

1. Flaccid dysarthrias result from damage to the motor units of cranial or spinal nerves that serve the speech muscles. They occur at a frequency comparable to that of other single dysarthria types. They sometimes reflect weakness in only a small number of muscles and can be isolated to lesions of single cranial or spinal nerves. Weakness and hypotonia are the underlying neuromuscular deficits that explain most of the abnormal speech characteristics associated with flaccid dysarthrias.
2. Lesions anywhere within the motor unit can cause flaccid dysarthrias, and various etiologies can produce such lesions. Surgical trauma and

degenerative diseases are common known causes, but etiology is frequently undetermined, particularly when only a single cranial nerve is involved. Muscle disease, MG, tumor, stroke, infection, neuroanatomic malformations, demyelinating diseases, and radiation therapy effects represent other known causes.

3. The speech characteristics and nonspeech examination findings differ among lesions of cranial nerves V, VII, X, and XII, and spinal respiratory nerves. Examination can localize the effects of disease to one or a combination of these nerves.
4. Lesions of the mandibular branch of cranial (trigeminal) nerve V lead to weakness of jaw muscles. When bilateral, jaw weakness can have significant effects on articulation. Lesions of cranial nerve V that affect sensation from the jaw, face, lip, and tongue and stationary points of articulatory contact may also affect speech, primarily articulatory precision.
5. Lesions of cranial (facial) nerve VII can lead to facial weakness and flaccid dysarthria. Unilateral weakness of the face can be associated with mild articulatory distortions. Bilateral lesions may lead to significant distortion of all consonants and vowels requiring facial movement.
6. Lesions of cranial (vagus) nerve X can lead to weakness of velopharyngeal and laryngeal muscles and to some of the most frequently encountered manifestations of flaccid dysarthrias. Lesions of the pharyngeal branch of the nerve can lead to resonatory incompetence, with hypernasality, nasal emission, and weakening of pressure consonant sounds. Lesions of the superior laryngeal and recurrent laryngeal branches can lead to various dysphonias whose perceptual attributes are consistent with weakness and hypotonia of laryngeal muscles. Lesions above the pharyngeal branch of the vagus nerve can lead to both resonatory and laryngeal incompetence, whereas lesions below the pharyngeal branch are associated with laryngeal manifestations only.
7. Lesions of cranial (hypoglossal) nerve XII cause tongue weakness. The resulting flaccid dysarthria is reflected in imprecision of lingual articulation, with severity dependent upon the degree of weakness and whether the lesion is unilateral or bilateral.
8. Lesions affecting spinal respiratory nerves can reduce respiratory support for speech. Weakness at this level can lead to reduced loudness and pitch variability, as well as reduced phrase length per breath group.
9. Phonatory and resonatory incompetence are commonly encountered distinguishing features of flaccid dysarthrias. Although they are tied to involvement of cranial nerve X, it is nonetheless important to attend to speech movements generated through cranial nerves V, VII, and XII. This is important both for a complete description of the speech disorder and because speech deficits isolated to single cranial or spinal nerves are possible in flaccid dysarthrias and unusual in other dysarthria types.
10. Flaccid dysarthrias can be the only, the first, or among the first and most prominent manifestations of neurologic disease. Their recognition and localization to motor units subserving speech can aid the localization and diagnosis of neurologic disease. Their diagnosis and description are important to decision making for medical and behavioral management.

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chapter **5**

# Spastic Dysarthria

*"It's slower, and sometimes it tires me, and I just don't want to talk anymore . . . the kids don't really say that much about it . . . I think they're in denial."*

*(79-year-old woman with an unambiguous but mild spastic dysarthria of undetermined origin)*

**CHAPTER OUTLINE**



Spastic dysarthria is a perceptually distinctive motor speech disorder (MSD) produced by bilateral damage to the direct and indirect activation pathways of the central nervous system (CNS). It may be manifest in any or all of the respiratory, phonatory, resonatory, and articulatory components of speech, but it is generally not confined to a single component. Its characteristics reflect the combined effects of weakness and spasticity in a manner that slows movement and reduces its range and force. Spasticity, a hallmark of upper motor neuron (UMN) disease, seems to be the crucial contributor to the distinctive features of the disorder, hence its designation as *spastic* dysarthria. The identification of a dysarthria as spastic can aid the diagnosis of neurologic disease and its localization to CNS motor pathways.

Spastic dysarthria is encountered in a large medical practice at a rate comparable to that of the other major single dysarthria types. Based on data for primary communication disorder diagnoses in the Mayo Clinic Speech Pathology practice, it accounts for 8.2% of all dysarthrias and 7.6% of all MSDs (see Figure 1-3).

The clinical features of spastic dysarthria reflect the effects of excessive muscle tone and weakness on speech. They illustrate well the distinction between speech deficits attributable to weakness alone (as in flaccid dysarthria) from those in which the barriers to normal speech also include neuromuscular resistance to movement.

## ANATOMY AND BASIC FUNCTIONS OF THE DIRECT AND INDIRECT ACTIVATION PATHWAYS

The direct activation pathway, also known as the *pyramidal tract* or *direct motor system,* forms part of the UMN system. Its activities lead to movements through the final common pathway (lower motor neurons [LMNs]). The pathway includes the *corticobulbar tracts,* which influence the activities of cranial nerves, and the *corticospinal tracts,* which influence the activities of spinal nerves.

The direct activation pathways are bilateral, one originating in the cortex of the right cerebral hemisphere, the other in the cortex of the left cerebral hemisphere. The pathways from the cortex lead rather directly to the cranial and spinal nerve nuclei in the brainstem and spinal cord. Their fibers

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primarily innervate muscles on the side of the body opposite the cerebral cortex of origin, but, for the speech muscles, this applies only to the muscles of the lower face, and, to a lesser extent, the tongue. The remaining cranial nerves subserving speech receive bilateral input from the direct (and indirect) activation pathways. This neural redundancy helps to minimize the effects of unilateral UMN lesions on speech and chewing, swallowing, and airway protection functions. Unilateral UMN lesions generally do not have a pronounced effect on jaw, velopharyngeal, laryngeal, or lingual movements for speech.

The direct activation pathway is predominantly *facilitatory.* That is, impulses through it tend to lead to movement, particularly *skilled, discrete movements.*

The indirect activation pathway, also known as the *extrapyramidal tract* or *indirect motor system,* is also part of the UMN system. It originates in the cortex of each cerebral hemisphere, but its course is more complicated than the direct activation pathway's because synapses occur between the cortex and the brainstem and spinal cord. Crucial interneuronal connections include those in the basal ganglia, cerebellum, reticular formation, vestibular nuclei, and red nucleus. The indirect activation pathway is crucial for *regulating reflexes and maintaining posture, tone,* and *associated activities that provide a framework for skilled movements.* Many of its activities are *inhibitory.*

## CLINICAL CHARACTERISTICS OF UPPER MOTOR NEURON LESIONS AND SPASTIC PARALYSIS

Damage to the direct activation pathway leads to a loss or reduction of fine, discrete movements. Following acute lesions, reduced muscle tone and weakness are evident, but they generally evolve to increased tone and spasticity. Weakness is usually more pronounced in distal than proximal muscles; the distal and speech muscles are those most involved in finely controlled skilled movement. Reflexes tend to be diminished initially but become more pronounced over time.

Direct activation pathway lesions are also associated with a *positive Babinski sign,* a pathologic reflex elicited by applying pressure with a relatively sharp point from the sole of the foot on the side of the heel forward to the little toe and across to the great toe. The normal response is a planting of the toes. A Babinski response is an extension of the great toe and fanning of the other toes. When present in adults, the Babinski sign is associated with CNS disease, reflecting the release of a primitive reflex from CNS inhibition (Babinski's reflex is normal in infants). *Pathologic oral reflexes* are also common in bilateral UMN disease, including suck, snout, palmomental, and jaw jerk reflexes (defined in Chapter 3).

Damage to the indirect activation pathway affects its predominantly inhibitory role in motor control. As a result, lesions tend to lead to overactivity *(positive signs)* such as increased muscle tone, spasticity, and hyperactive reflexes. These signs are interrelated. Spasticity, for example, is the result of hyperactivity of stretch reflexes, and it goes hand in hand with increased muscle tone. It results in resistance to movement that is generally more pronounced at the beginning of movement or in response to quick movements (i.e., it is velocity dependent). In the limbs, spasticity tends to be biased toward lower extremity extension (i.e., the legs resist bending) and upper extremity flexion (i.e., the arms resist straightening). Physical therapists sometimes hope for spasticity to develop in the legs of patients with UMN lesions, because it facilitates standing.

Patients with UMN lesions and hyperactive reflexes sometimes exhibit *clonus,* a kind of repetitive reflex contraction that occurs when a muscle is kept under tension (stretch) by an examiner (e.g., when the foot is continuously dorsiflexed by the examiner). The reflex response may look like a rhythmic tremor.[30]

Selective damage to only the direct or only the indirect activation pathway is uncommon, because both pathways arise in adjacent and overlapping areas of the cortex and travel in close proximity through much of their course to LMNs. As a result, people with spastic paralysis commonly exhibit decreased skilled movement and weakness from direct activation pathway damage, as well as increased muscle tone and spasticity from indirect activation pathway damage.

Direct and indirect activation pathway signs of UMN lesions are summarized in Table 5-1. The

**table 5-1** Direct and indirect activation pathway signs of upper motor neuron lesions

| Damage to | |
|---|---|
| **Direct Activation Pathway (Pyramidal Tracts)** | **Indirect Activation Pathway (Extrapyramidal Tracts)** |
| Loss of fine, skilled movement | Increased muscle tone |
| Hypotonia | Spasticity |
| Weakness (distal > proximal) | Clonus |
| Absent abdominal reflexes | Decorticate or decerebrate posture |
| Babinski's sign | Hyperactive stretch reflexes |
| Hyporeflexia | Hyperactive gag reflex |



major abnormalities that affect movement in spastic paralysis include *spasticity, weakness, reduced range of movement,* and *slowness of movement.* These abnormalities also appear to represent the most salient features of disordered movement in patients with spastic dysarthria.

## THE RELATIONSHIP OF SPASTIC PARALYSIS TO SPASTIC DYSARTHRIA

The neuropathophysiologic underpinnings of spastic dysarthria are more complex and less well understood than those of flaccid dysarthria. This is partly a product of the complexity of the CNS motor pathways and the fact that spastic dysarthria is usually associated with damage to two components of the motor system, the direct and indirect activation pathways. In addition, the degree to which the concept of spasticity can be applied to the cranial nerve innervated portion of the speech system has been questioned.[1,2,7]

Most of what is known about the clinical manifestations of spastic paralysis is based on studies of limb movements that require the movement of joints in agonist and antagonistic relationships with each other.*[1] Many speech movements do not involve the movement of joints, and different speech structures have varying numbers of muscle spindles that are important in the mediation of stretch reflexes. For example, the jaw is well endowed with spindles, the intrinsic muscles of the tongue have some, and the face has none.[7] Furthermore, lip movements do not require the movement of bone, and the tongue is a muscular hydrostat, the movements of which do not involve joints. Relatedly, it seems that different speech structures are affected in somewhat different ways by UMN lesions.[1] Finally, unlike the limbs, speech requires symmetric movements of bilaterally innervated structures. That is, jaw, face, tongue, palate, and laryngeal movements require the synchronous movement of each of their halves so that the structures move as a single unit.

In spite of the differences between bulbar and limb movements, and uncertainty about the degree to which understanding spastic paralysis in the limbs can explain what occurs in the bulbar muscles during speech, it appears that, for practical clinical purposes at least, several of the *general* principles and observations about spastic paralysis discussed in the previous section can be usefully applied to our clinical conceptions of spastic dysarthria.

*See Sheean[42] for a concise overview of the pathophysiology of spasticity.

## ETIOLOGIES

Any process that damages the direct and indirect activation pathways bilaterally can cause spastic dysarthria. These include degenerative, inflammatory, toxic, metabolic, traumatic, and vascular diseases. These etiologic categories produce bilateral CNS motor system damage and spastic dysarthria with varying frequency, but the exact distribution of causes of spastic dysarthria is unknown. It does appear, however, that degenerative, vascular, and traumatic disorders are the predominant causes.

Although no general etiologic category is uniquely associated with spastic dysarthria, vascular disorders are more frequently associated with it than with most other dysarthria types. Some of those vascular disorders are discussed as follows. A few other conditions that have a relatively specific association with spastic but not other forms of dysarthria are also addressed. Note that all of the following conditions represent only a few of the potential etiologies of spastic dysarthria. Other diseases associated with spastic dysarthria but that are more frequently associated with other dysarthria types are discussed in the chapters that deal with those specific dysarthria types.

### Vascular Disorders

Strokes in the distribution of the internal carotid and middle and posterior cerebral arteries, and less frequently the anterior cerebral artery, can produce spastic dysarthria. However, because these arteries mostly supply structures within the cortex and subcortical structures of the cerebral hemispheres—where the UMN pathways on the left and right are not in close proximity to one another—lesions in both the left and right hemispheres are required to produce the bilateral UMN damage usually associated with spastic dysarthria. In the brainstem, where the right and left UMN pathways are in close proximity to one another, a single infarct in the vertebrobasilar arterial distribution may be sufficient to produce the bilateral UMN damage associated with spastic dysarthria. In general, therefore, *a single brainstem stroke can produce a spastic dysarthria, whereas a single cerebral hemisphere stroke usually does not.**

Some patients with spastic dysarthria have had multiple *lacunes* or *lacunar infarcts*—small, deep infarcts in the small penetrating arteries of the basal ganglia, thalamus, brainstem, and deep cerebral

*Single cerebral hemisphere stroke sometimes leads to speech characteristics associated with spasticlike dysarthria. This issue is addressed further in Chapter 9.

white matter.* They are usually associated with hypertension. *Lacunar state* is a term applied to patients with numerous lacunar infarcts who frequently have dementia, dysarthria, pseudobulbar affect, dysphagia, hyperreflexia, and incontinence.[8] However, dysarthria (perhaps plus dysphagia) can be the only sign of lacunar strokes. Okuda et al.[35] reported that 11 of their 12 patients with "pure dysarthria" due to stroke had magnetic resonance imaging (MRI) evidence of multiple, bilateral lacunar strokes involving the internal capsule or corona radiata, with 8 of the 11 patients also having evidence (based on single photon emission computed tomography [SPECT]) of frontal hypometabolism.

Relatedly, *Binswanger's disease (subcortical arteriosclerotic encephalopathy)* is a term sometimes applied to patients with vascular dementia. It is characterized by periventricular demyelination of subcortical white matter and is often associated with a history of hypertension.[40] The major lesions are in the subcortical white matter with relative sparing of the cortex and basal ganglia. These bilateral lesions can affect UMN pathways and lead to spastic dysarthria. The association of spastic dysarthria with dementia is an important diagnostic observation, because dysarthria is not commonly associated with common degenerative cortical dementias such as Alzheimer's and Pick's diseases.

Not all occlusive vascular diseases are due to arteriosclerosis or emboli, nor are they solely a disease of the elderly. *Moyamoya disease,* for example, is a chronic, progressive nonatherosclerotic occlusive vascular disease of unknown cause that most frequently affects children, adolescents, or young adults.[9] It can cause stroke and intracranial hemorrhage, with resulting neurologic deficits including speech and language impairments.[21] Because it is associated with bilateral stenosis of the arteries, a resulting dysarthria may be spastic in character. *Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (often referred to as CADASIL)* is a hereditary disorder that often presents with stroke manifestations in early adulthood. It is frequently associated with pseudobulbar palsy and cognitive deficits.[15] Because the pathology can be bilateral, an associated dysarthria may be spastic.

### Degenerative Disease

*Primary lateral sclerosis (PLS)* is a rarely occurring subcategory of motor neuron disease (of which amyotrophic lateral sclerosis [ALS] is a major subcategory). It is manifest by corticospinal and corticobulbar tract signs alone,* with no evidence of LMN involvement. It seems that dysarthria occurs frequently in PLS, and it sometimes is the presenting problem.[39] When dysarthria and dysphagia are the primary manifestations in PLS, the disorder is sometimes referred to as *progressive pseudobulbar palsy.*† Because clinical motor findings are limited to descending UMN tracts, it can be assumed that the type of dysarthria is typically spastic. The distinction between PLS and ALS is of more than academic interest because the median disease duration until death for PLS (19 years, as reported by Pringle[39]) is much longer than for ALS (only a few years). Because the dysarthria of PLS is presumably spastic only, the correct distinction between spastic dysarthria and the mixed spastic-flaccid dysarthria often associated with ALS can be of some assistance to neurologic differential diagnosis.

### Inflammatory Disease

*Leukoencephalitis* is an inflammatory demyelinating disease that affects the white matter of the brain or spinal cord. In acute *hemorrhagic leukoencephalitis* the white matter of both hemispheres is destroyed, with similar changes in the brainstem and cerebellar peduncles. This destruction is associated with necrosis of small blood vessels and surrounding brain tissue, with inflammatory reactions in the meninges. There is a tendency for large focal lesions to form in the cerebral hemispheres.[4] The bilateral and multifocal effects of this white matter disease can affect UMN pathways and cause spastic dysarthria (or mixed dysarthrias).

## SPEECH PATHOLOGY

### Distribution of Etiologies, Lesions, and Severity in Clinical Practice

Box 5-1 and Figure 5-1 summarize the etiologies for 144 quasirandomly selected cases seen at the Mayo Clinic with a primary speech pathology diagnosis of spastic dysarthria. The cautions expressed in Chapter 4 about generalizing these observations to the general population or all speech pathology practices apply here as well.

*Lacunar stroke syndromes are discussed in detail in Chapter 9.

*Mild cognitive impairment has been demonstrated in some patients with PLS.[10]

†Windebank[45] uses PLS to refer to a motor neuron disease characterized initially by lower limb spasticity secondary to UMN degeneration. He distinguishes it from progressive pseudobulbar palsy, in which UMN degeneration is characterized primarily by dysarthria and dysphagia.



**box 5-1** Etiologies for 144 quasirandomly selected cases with a primary speech pathology diagnosis of spastic dysarthria at the Mayo Clinic from 1969-1990 and 1999-2001. Percentage of cases for each etiology is given in parentheses. Specific etiologies under each heading are ordered from most to least frequent.

**Degenerative (40%)**
Unspecified degenerative CNS disease (13%)
ALS (14%)
PSP (5%)
PLS (5%)
CBD (1%)
Spinocerebellar ataxia (1%)
Uncertain (PLS vs. ALS; PSP vs. CBD) (1%)

**Vascular (29%)**
Nonhemorrhagic stroke (single or multiple) (26%)
Ruptured aneurysm (1%)
Hemorrhagic stroke (1%)
Hypoxic encephalopathy (1%)

**Traumatic (10%)**
Traumatic brain injury (8%)
Neurosurgical (e.g., tumor resection) (1%)

**Undetermined (10%)**

**Demyelinating (4%)**
Multiple sclerosis

**Tumor (3%)**
CNS tumor (2%)
Paraneoplastic syndrome (1%)

**Multiple Causes (2%)**
CVA + dementia; tumor + radiation therapy; TBI + alcoholism + PSP

**Inflammatory (1%)**
Inflammatory brainstem disorder; postencephalitic

**Infectious (1%)**
Infectious encephalopathy

*ALS,* Amyotrophic lateral sclerosis; *CBD,* corticobasal degeneration; *CNS,* central nervous system; *CVA,* cerebrovascular accident; *PLS,* primary lateral sclerosis; *PSP,* progressive supranuclear palsy; *TBI,* traumatic brain injury.

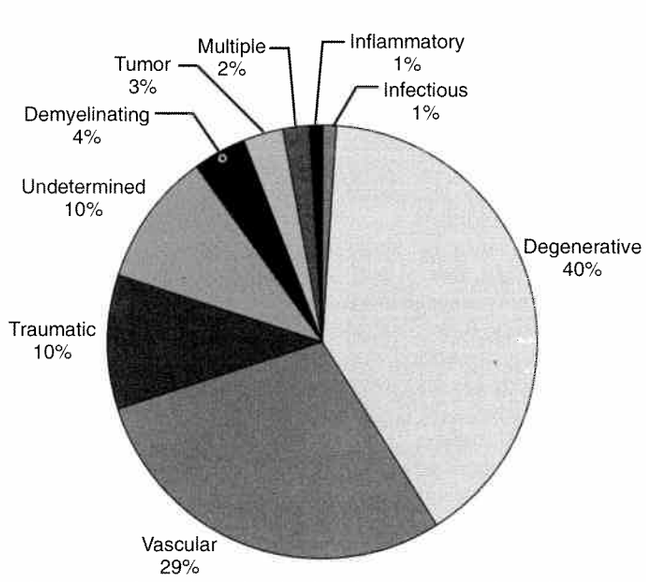


**FIGURE 5-1** Distribution of etiologies for 144 quasirandomly selected cases with a primary speech pathology diagnosis of spastic dysarthria at the Mayo Clinic from 1969-1990 and from 1999-2001 (see Box 5-1 for details).

The data establish that spastic dysarthria can result from various medical conditions, the distribution of which are quite different from that associated with flaccid dysarthria. More than 90% of the cases were accounted for by degenerative, vascular, traumatic, demyelinating, and undetermined etiologies. Nearly 70% were accounted for by degenerative and vascular diseases.

Nonhemorrhagic strokes accounted for most of the vascular causes. This is not surprising because such strokes account for the highest proportion of neurovascular disturbances in general. Many of these

patients had multiple strokes. Most who had only a single stroke had a brainstem lesion.

Patients with only a single confirmed (by computed tomography [CT] or MRI) stroke in one of the cerebral hemispheres usually had nonspeech clinical signs of bilateral involvement, suggesting the presence of "silent" or undetected infarcts or other pathology in the "intact" hemisphere or brainstem. A few patients with a diagnosis of stroke had no identifiable lesion on CT or MRI, suggesting that *spastic dysarthria may be the only evidence of stroke in some individuals.* It is also possible that characteristics of spastic dysarthria can sometimes result from a unilateral UMN lesion.*

Degenerative diseases associated with spastic dysarthria were often nonspecific. It is not unusual for neurodegenerative disease to defy a more specific diagnosis, especially early in its course. This sometimes remains the case until autopsy. ALS, PLS, and progressive supranuclear palsy (PSP) were the most commonly diagnosed neurodegenerative diseases. It is noteworthy, however, that ALS and PSP can be associated with other dysarthria types and frequently with mixed dysarthrias. ALS, PSP, and other degenerative diseases listed in Box 5-1 are discussed further in Chapter 10.

Traumatic brain injury (TBI) was a fairly frequent cause of spastic dysarthria. Although Yorkston et al.[47] indicate that most TBI-associated dysarthrias are mixed spastic-ataxic or flaccid-spastic, data from the sample reviewed here establish that spastic dysarthria can be the only dysarthria type following TBI. Trauma from intracranial surgery is another possible traumatic cause of spastic dysarthria.

Numerous patients had spastic dysarthria of undetermined etiology. Some of them had several possible diagnoses (e.g., stroke versus degenerative CNS disease) or diagnoses compatible with bilateral UMN involvement. Some had only dysarthria and dysphagia and received only a descriptive diagnosis.

Several patients had multiple sclerosis (MS) (discussed in Chapter 10). Some had tumors. CNS tumors, particularly if localized in the brainstem, can cause spastic dysarthria, as can unilateral hemispheric tumors if they exert mass effects on the brainstem or opposite hemisphere. Only a few patients had inflammatory or infectious disorders.

The data also illustrate that spastic dysarthria can arise from multiple causes or events in the same patient (e.g., TBI plus alcoholism plus PSP). This observation is important, because some patients being evaluated for a condition that ordinarily might not be associated with spastic dysarthria might develop it because their current illness is added to the effects of a previous or concurrent condition. It is not unusual, for example, to discover in a patient who has developed signs of unilateral stroke and spastic dysarthria that there is a history of prior stroke on the opposite side of the brain (with or without speech disturbance).

The distribution of lesions for the cases summarized in Box 5-1 was spread through the course of the UMN system, including the *cortex, corona radiata, basal ganglia, internal capsule, pons,* and *medulla.* Focal lesions were most obvious when the etiology was vascular. Generalized or diffuse atrophy was frequently the only anatomic abnormality in TBI, degenerative disease, and undetermined etiologies. Approximately one fourth of the patients had no evidence of cerebral pathology on neuroimaging studies. It is important to note that the only clinical sign of bilateral pathology in some patients was their spastic dysarthria and frequently accompanying dysphagia and pathologic oral reflexes.

This retrospective review did not permit a precise delineation of dysarthria severity. However, in a review of the 144 patients reviewed in Box 5-1, intelligibility was specifically commented on in 81%; in those cases, 56% *were judged to have reduced intelligibility.* The degree to which this percentage accurately estimates intelligibility impairments in the population with spastic dysarthria is unclear. It is likely that many patients for whom an observation of intelligibility was not made had normal intelligibility, but the sample probably contains a larger number of mildly impaired patients than is encountered in a typical rehabilitation setting.

Finally, because of its association with bilateral, multifocal, or diffuse CNS disease, it is not uncommon for spastic dysarthria to be accompanied by cognitive disturbances that may include dementia or cognitive-communication deficits associated with right hemisphere impairment, TBI, or aphasia. For the patients in this sample whose cognitive abilities were explicitly judged or formally assessed (83% of the sample), *36% had some impairment of cognitive ability.*

## Patient Perceptions and Complaints

People with spastic dysarthria sometimes offer complaints or descriptions that provide clues to the speech diagnosis and its localization. Some of these are only infrequently associated with other dysarthria types.

A frequent complaint is that speech is *slow* or *effortful.* When asked, patients often confirm that it

*In the author's experience, apparent spastic dysarthria in cases of presumed unilateral stroke, with no other clinical evidence of bilateral pathology, is encountered most frequently early after onset of a single unilateral stroke. If true, the reasons for this occurrence are unclear.



feels as if they are speaking against resistance. The descriptors "slow" and "effortful" are not often heard from patients with other dysarthria types (with the exception of some hyperkinetic dysarthrias). Patients often complain of *fatigue* with speaking, sometimes with accompanying deterioration of speech. With the exception of myasthenia gravis (MG), complaints of fatigue occur more frequently in spastic than flaccid dysarthria, even though deterioration of speech in spastic dysarthria is not usually dramatic and almost never rapid.* Patients also often note that they must speak more slowly to be understood but often admit that they really are unable to speak any faster. Finally, they often complain of *nasal* speech, although this complaint is heard more frequently in people with flaccid dysarthria.

*Swallowing complaints* are common and often can be associated with both oral and pharyngeal phases of swallowing.† In some patients, a precursor to dysphagia and evidence of a lowered gag reflex threshold is increased gagging when brushing teeth. Patients also complain of *drooling,* more so than for most other single dysarthria types. Finally, many patients complain of or admit to *difficulty controlling their expression of emotion,* especially laughter and crying. This *pseudobulbar affect* is rarely encountered in other single dysarthria types. It is discussed in detail in the next section.

## Clinical Findings

Spastic dysarthria is often associated with bilateral motor signs and symptoms in the limbs that make the presence of bilateral CNS involvement obvious.‡ However, it can occur in the absence of bilateral or even unilateral limb findings, and it may, sometimes along with dysphagia and pathologic oral reflexes, be the only sign of neurologic disease. This is not unusual in certain degenerative nervous system diseases.

Bilateral spastic paralysis affecting the bulbar muscles traditionally has been called *pseudobulbar palsy,* and many neurologists use the term to describe the speech of spastic dysarthria. Pseudobulbar palsy is a clinical syndrome that derives its name from its superficial resemblance to bulbar palsy (associated with LMN lesions and flaccid dysarthria). It reflects bilateral lesions of corticobulbar fibers and is most commonly associated with multiple or bilateral strokes, CNS trauma, degenerative CNS disease, encephalopathies, and CNS tumors. Its clinical features include dysarthria, dysphagia, and other oral mechanism abnormalities that are discussed as follows.

### Nonspeech Oral Mechanism

Several oral mechanism findings are frequently associated with spastic dysarthria. *Dysphagia* is common and sometimes severe. Although some patients deny chewing or swallowing difficulties, on questioning they may admit that they are careful when swallowing, that chewing meat has become more difficult, or that they chew more slowly or more carefully than before. Nasal regurgitation is unusual in pure spastic dysarthria, but *drooling* is common, and patients often attribute it to excessive saliva production; it is more likely due to decreased frequency of swallowing or poor control of secretions. It may occur when the patient concentrates on some nonspeech activity, particularly if the neck is flexed (e.g., during writing). Patients with or without daytime drooling sometimes note that their pillow is wet upon awakening in the morning, or that saliva has dried around the mouth during the night. Obviously slowed jaw, lip, and facial movement may characterize reflexive swallowing of secretions; the swallow is occasionally audible.

At rest, the nasolabial folds may be smoothed or flattened, or the face may be held in a somewhat fixed, subtle smiling or pouting posture. Reflexive or emotional facial movements frequently emerge slowly but may then overflow and be excessive.

Lability of affect, often called *pseudobulbar affect* or *pathologic laughing and crying,* is frequently apparent. When subtle, patients may have an "on the verge of tears" facial expression. When more obvious, they may cry or laugh in a stereotypic manner for no apparent reason, may fluctuate between laughing and crying, or may have difficulty inhibiting laughter and crying once they begin. The ease with which the response is elicited tends to be

*Fatigue is a common complaint in people with neurologic disease. In those with spastic paresis of the limbs, it is usually assumed to be of CNS origin, secondary to impaired recruitment of alpha motor neurons, but it is recognized that mechanisms underlying fatigue can include all elements of the motor system.[18] For example, there is some evidence that biochemical changes in muscles of patients with UMN lesions may contribute to excessive fatigability.[32] The etiology of the muscle changes may be due to disuse, a problem known to reduce muscle volume and weight.[16]

†In degenerative or gradually developing neurologic disease, speech and swallowing problems very often emerge concurrently. In the author's experience, which could be subject to referral bias, when one precedes the other, speech difficulty tends to develop first.

‡Unilateral UMN lesions produce a syndrome of signs and symptoms that affect movements on the contralateral side of the body. This syndrome sometimes includes unilateral UMN dysarthria, which is addressed in Chapter 9.

**table 5-2 Neuromuscular deficits associated with spastic 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 | Slow | Slow | Reduced (weak) | Reduced (biased) | Reduced | Excessive |

Modified from Darley FL, Aronson AE, Brown JR: Clusters of deviant speech dimensions in the dysarthria, *J Speech Hear Res* 12:462, 1969.

related to the emotional loading of the interaction, although the emotional response can occur spontaneously or simply in response to being asked if they have difficulty controlling emotional expression. Patients sometimes report that their inner emotional state does not match their physical expression of emotion. These affective responses can occur during speech, sometimes with significant effects on intelligibility or efficiency of communication. Pseudobulbar affect can convey an impression of emotional instability or dementia but can be present without any clear evidence of those disorders and sometimes without other evidence of pseudobulbar palsy,[6] including dysarthria. These uncontrollable emotional responses are often upsetting to patients. Aronson[5] points out that "the reduced threshold for crying and laughter has clinical diagnostic importance and needs to be recognized as one of the great social and psychological burdens borne by patients with pseudobulbar palsy."

Examination of nonspeech oromotor functions usually demonstrates normal jaw strength. The face may be weak bilaterally, and range of lip retraction and pursing may be decreased; however, lower facial weakness is usually not as pronounced as with LMN lesions. The tongue is usually full and symmetric, but range of movement may be reduced and weakness apparent on strength testing. Nonspeech alternate motion rates (AMRs) for jaw, lip retraction and pursing, and lateral or anterior tongue movements are often slow and reduced in range of movement, but they are generally regular in rhythm.

The palate is usually symmetric but may move slowly or minimally on phonation. The gag reflex is often hyperactive.* The cough and glottal coup may be normal in sharpness if respiratory and laryngeal movements are not too slowed, but they may lack sharpness if slowness is prominent.

Pathologic oral reflexes are common. *Sucking, snout, palmomental,* and *jaw jerk reflexes* are frequently present and are suggestive of bilateral UMN involvement.

### Speech

Conversational speech and reading, speech AMRs, and vowel prolongation are the most useful tasks for eliciting the salient and distinguishing characteristics of spastic dysarthria.* Speech stress testing and sequential motion rates (SMRs) are not particularly revealing.

The deviant speech characteristics associated with spastic dysarthria are not easily or usefully described by listing each cranial nerve and the speech characteristics associated with its abnormal function. This is because *spastic dysarthria is associated with impaired movement patterns rather than weakness of individual muscles.* This reflects the organization of CNS motor pathways for the control of movement patterns rather than isolated muscle movements, and it represents an important distinction between LMN and UMN lesions. Therefore *spastic dysarthria is usually associated with deficits at all of the speech valves and for all components of the speech system,* although not always equally. The involvement of multiple speech valves may explain why intelligibility is so frequently affected.

Table 5-2 summarizes the neuromuscular deficits assumed by Darley, Aronson, and Brown (DAB)[12-14]

*Some patients with bilateral damage to the lower part of the precentral and postcentral cortex of the cerebral hemispheres may have an absent gag reflex. The constellation of deficits with such lesions is discussed in the section on biopercular syndrome in Chapter 12.

*Speech AMR and vowel prolongation tasks are also sensitive to differences between "developmental" spastic dysarthria and nondysarthric speech. Wit et al.[46] found that performance on such tasks reliably distinguished children with spastic dysarthria associated with cerebral palsy (age 6 to 11 years) from a matched control group. The dysarthric children had reduced maximum sound prolongation and $f_0$ range on vowel prolongation tasks and slower and more variable syllable durations on AMR tasks.





to underlie spastic dysarthria. In general, direction and rhythm or timing of movement are unaffected. The chief disturbances are *slowness and reduced range of individual and repetitive movements, reduced force of movement, and excessive or biased muscle tone or spasticity.* The bias of muscle tone is most apparent at the laryngeal valve, in which the bias is toward hyperadduction during phonation. The relationship between these neuromuscular deficits and the prominent deviant clusters and speech characteristics of spastic dysarthria is apparent in subsequent descriptions of those characteristics. Experimental support for the presumed underlying neuromuscular deficits, especially slowness and reduced range of movement, is reviewed in the section on acoustic and physiologic studies.

### Clusters of Deviant Dimensions and Prominent Deviant Speech Characteristics

DAB[13] found four clusters of deviant dimensions in their group of 30 patients with pseudobulbar palsy. These clusters are useful to understanding the neuromuscular deficits presumed to underlie spastic dysarthria, the components of the speech system that are most prominently involved, and the features of spastic dysarthria that distinguish it from other dysarthria types (Table 5-3).

The first cluster is *prosodic excess,* represented by *excess and equal stress* and *slow rate.* These characteristics are probably related to slowness of individual and repetitive movements. Slowness of movement logically reduces speech rate. It probably also contributes to excess and equal stress by reducing the speed of the muscular adjustments necessary for the rapid pitch, loudness, and duration adjustments associated with normal prosody. Slow overall speech rate can also lead to a perception of excess and equalized stress, because longer syllable duration is associated with stressed syllables.

The second cluster is *articulatory-resonatory incompetence,* represented by *imprecise consonants, distorted vowels,* and *hypernasality.* This cluster represents the probable effects of reduced range and force of articulatory movements (presumably including the tongue, jaw, and face) and velopharyngeal movements. The strong interrelationships among velopharyngeal and articulatory features in this cluster implicate the velopharyngeal mechanism's articulatory role, not its resonatory role (i.e., inadequate velopharyngeal closure can result in weak, imprecise pressure consonants).

The third cluster is *prosodic insufficiency,* consisting of *monopitch, monoloudness, reduced stress,* and *short phrases.* For the most part, these characteristics are attributable to reduced vocal variability, with stressed syllables left unstressed or insufficiently different from unstressed syllables, and reduced pitch and loudness variability. Decreased range of movement is a likely explanation for this cluster.

The fourth cluster is *phonatory stenosis,* characterized by *low pitch, harshness, strained-strangled voice, pitch breaks, short phrases,* and *slow rate.* These phonatory characteristics seem to reflect efforts to produce voice through a narrowed glottis with secondary reduction of phrase length and speech rate. The assumption is that laryngeal hypertonus is present with a bias toward excessive adduction or resistance to abduction. The features of slow rate and short phrases may also be related to slowness of movement and inefficient valving at the velopharyngeal and articulatory valves.

DAB noted the presence of *breathiness* in some patients with spastic dysarthria, a characteristic that was not correlated with any of the clusters found for the disorder. This breathiness could reflect a degree of vocal-cord weakness, but it could also represent a compensatory response rather than a primary problem. For example, some patients may actively maintain incomplete adduction to prevent laryngeal stenosis, or, alternatively, may intermittently actively abduct the cords to facilitate exhalation or provide relief from the effort induced by laryngeal stenosis.

Table 5-4 summarizes the most deviant speech dimensions found by DAB.[12] It is noteworthy that the rankings in Table 5-4 represent the order of prominence (severity) of the speech characteristics, not the features that are most distinctive of spastic

**table 5-3 Clusters of abnormal speech characteristics in spastic dysarthria**

| Cluster | Speech Characteristics |
|---|---|
| Prosodic Excess | Excess & equal stress<br>Slow rate |
| Articulatory-Resonatory Incompetence | Imprecise consonants<br>Distorted vowels<br>Hypernasality |
| Prosodic Insufficiency | Monopitch<br>Monoloudness<br>Reduced stress<br>Short phrases |
| Phonatory Stenosis | Low pitch<br>Harshness<br>Strained-strangled voice<br>Pitch breaks<br>Short phrases<br>Slow rate |

Modified from Darley FL, Aronson AE, Brown JR: Clusters of deviant speech dimensions in the dysarthria, *J Speech Hear Res* 12:462, 1969b.

**table 5-4** Most deviant speech dimensions encountered in spastic dysarthria by DAB,[13] listed in order from most to least severe. Also listed is the component of the speech system associated with each characteristic. The component "prosodic" is listed when several components of the speech system may contribute to the dimension.

| Dimension | Speech Component |
|---|---|
| Imprecise consonants* | Articulatory |
| Monopitch | Laryngeal |
| Reduced stress | Prosodic |
| Harshness | Laryngeal |
| Monoloudness | Laryngeal-respiratory |
| Low pitch* | Laryngeal |
| Slow rate* | Articulatory-prosodic |
| Hypernasality | Velopharyngeal |
| Strained-strangled quality* | Laryngeal |
| Short phrases | Laryngeal-respiratory-velopharyngeal or articulatory |
| Distorted vowels | Articulatory |
| Pitch breaks | Laryngeal |
| Breathy voice (continuous) | Laryngeal |
| Excess & equal stress | Prosodic |

*Tend to be distinctive or more severely impaired in spastic dysarthria than other single dysarthria types.

dysarthria. For example, imprecise consonants, although rated as the most severely impaired characteristic in spastic dysarthria, are found in all major dysarthria types and therefore are not a *distinguishing* characteristic of spastic dysarthria.

Numerous studies support DAB's identification of slow rate as a pervasive and perceptually salient feature of spastic dysarthria. For example, Kammermeier[25] (as summarized by DAB[14]) found a mean reading rate of 104 words per minute in patients with spastic dysarthria, slower than in those with bulbar palsy, Parkinson's disease, cerebellar disease, and dystonia. Slow speech AMRs have been documented in several studies.[17,22,28,38] In studies in which comparisons have been made to other dysarthria types, patients with spastic dysarthria have had the slowest AMRs. Linebaugh and Wolfe[29] documented slow rate of syllable production in spastic dysarthria, as well as a moderate relationship between rate and intelligibility and speech naturalness ratings.

What features of spastic dysarthria help distinguish it from other types of MSDs? Among the many abnormalities that may be detected, *strained-harsh voice quality, slow speech rate, and slow and regular speech AMRs are the most distinctive clues to the presence of spastic dysarthria.*

**table 5-5** Primary distinguishing speech and speech-related findings in spastic dysarthria

| | |
|---|---|
| **Perceptual** | |
| ***Phonation*** | Strained-strangled voice quality |
| ***Articulation-prosody*** | Slow rate |
| | Slow & regular alternating motion rates |
| **Physical** | Dysphagia, drooling |
| | Weak face & tongue |
| | Pathologic reflexes (suck, snout, palmomental, jaw jerk) |
| | Pseudobulbar affect |
| **Patient Complaints** | Slow speech rate |
| | Increased effort to speak |
| | Fatigue when speaking |
| | Chewing-swallowing difficulty |
| | Poor control of emotional expression |

Table 5-5 summarizes the primary distinguishing speech characteristics and common oral mechanism examination findings and patient complaints encountered in spastic dysarthria.

## Acoustic and Physiologic Findings

This section focuses primarily on acoustic and physiologic studies of acquired spastic dysarthria, but a few studies of children and adults with cerebral palsy are also relevant. The results of these studies are summarized in Table 5-6. Figure 5-2 illustrates some acoustic correlates of perceived slow and regular AMRs. Figure 5-3 illustrates some acoustic correlates of perceived slow speech rate and prosodic abnormalities commonly associated with spastic dysarthria.

### *Respiration*

Little is known about speech-related respiratory characteristics in acquired spastic dysarthria. It is quite possible, however, that they bear a resemblance to some of the documented respiratory difficulties of children and adults with spastic cerebral palsy. These abnormalities include reduced inhalatory and exhalatory respiratory volumes leading to shallow breathing; paradoxical breathing in which abdominal muscles fail to relax during inhalation with resultant restriction of respiratory intake; and reduced vital capacity.[5,14]



**table 5-6** Summary of acoustic and physiologic findings in studies of spastic dysarthria*

| Speech Component | Acoustic or Physiologic Observation |
|---|---|
| **Respiratory** (or respiratory or laryngeal) (based on studies of spastic cerebral palsy) | Reduced:<br>Inhalatory & exhalatory volumes<br>Respiratory intake<br>Vital capacity<br>Maximum vowel prolongation<br>Poor visuomotor tracking with respiratory movements |
| **Laryngeal** | Decreased:<br>Harmonic-to-noise ratio<br>Laryngeal airflow<br>Fundamental frequency variability<br>Increased:<br>Shimmer & jitter<br>Standard deviation of $f_o$<br>Subglottal pressure<br>Glottal resistance<br>Nonsyntactic breaks<br>Hyperadduction of true & false cords during speech<br>Poor visuomotor tracking with pitch variations |
| **Velopharyngeal** | Slow velopharyngeal movement<br>Incomplete velopharyngeal closure |
| **Articulatory or Rate or Prosody** | Reduced:<br>Overall rate (words per minute, syllables per second, phoneme duration)<br>Alternate motion rates (AMRs)<br>Speed & range of tongue, jaw, & palatal movements<br>Acceleration & deceleration of articulators<br>Maximum speed of lip movements<br>Rate & slope of F2 transitions<br>Rate of amplitude variation<br>Tongue strength<br>Ability to sustain maximum tongue contraction<br>Vowel space<br>Completeness of articulatory contacts<br>Completeness of consonant clusters<br>Sharpness of voiceless stops<br>Spectral tilt for /s/ (imprecision)<br>Oral pressures<br>Sound pressure level contrasts in consonants<br>Amplitude of release bursts for stops<br>Frequency & intensity increases for initial word stress<br>Articulatory effort for final word stress<br>Increased:<br>Duration of nonphonated intervals<br>Variability of noise amplitude or spectrum shape during /s/<br>Noise before closure for /s/<br>Duration of phoneme-to-phoneme transitions<br>Intersyllable duration<br>Temporal & amplitude variability for AMRs<br>Centralization of vowel formants<br>Acoustic energy during intersyllable gaps (imprecision)<br>Voicing of voiceless stops<br>Incomplete lingual articulatory contacts<br>Spirantization |

*Note that many of these observations are based on studies of only one or a few speakers, and not all speakers with spastic dysarthria exhibit all of these features. Note also that these characteristics may not be unique to spastic dysarthria; many can be observed in other motor speech disorders or nonneurologic conditions.

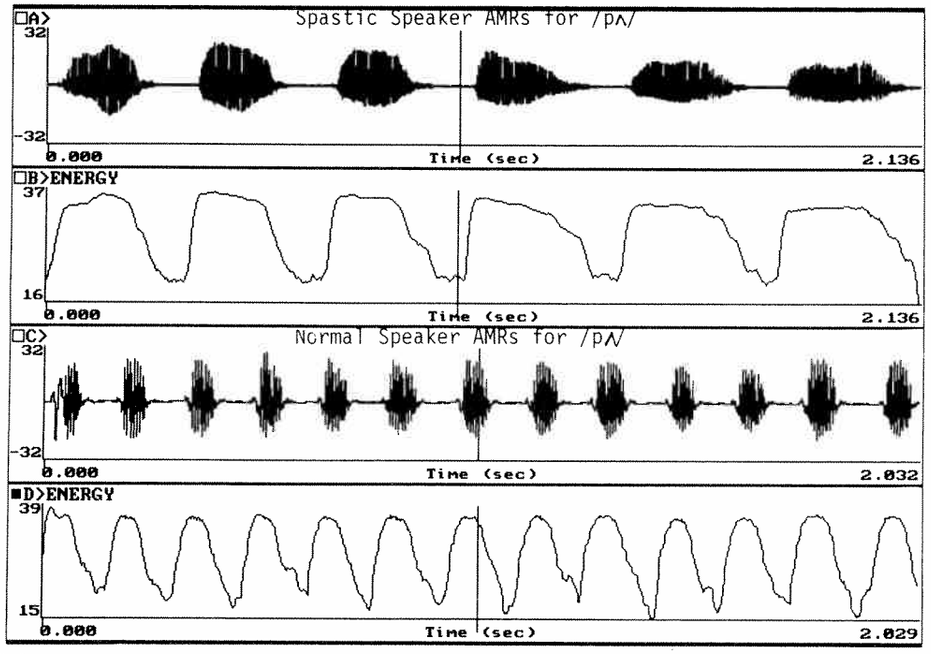


**FIGURE 5-2** Raw waveform and energy tracings of speech alternate motion rates (AMRs) for /p⇒/ by a normal speaker *(bottom two panels)* and a speaker with spastic dysarthria. The normal speaker's AMRs are normal in rate (≈6.5 Hz) and relatively regular in duration and amplitude. In contrast, the spastic speaker's AMRs are slow (≈3 Hz) and regular. These attributes represent the acoustic correlates of perceived slow and regular AMRs that are common in spastic dysarthria.

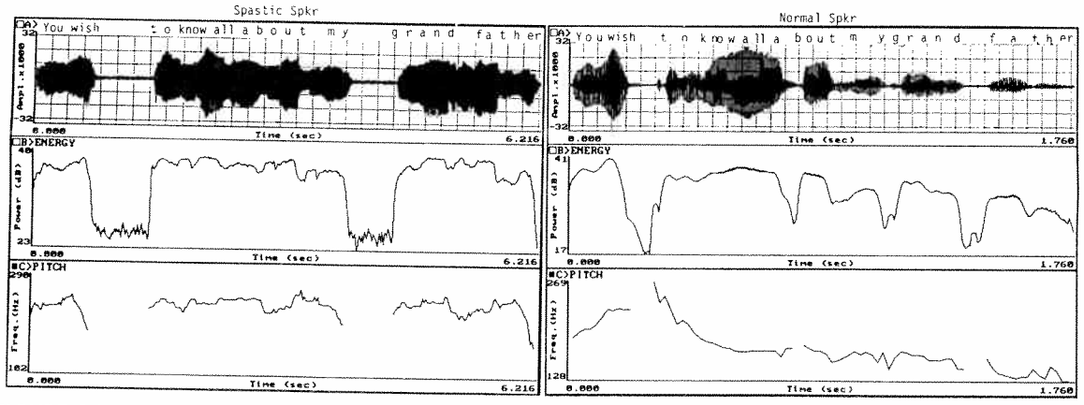


**FIGURE 5-3** Raw waveform and energy and pitch ($f_0$) tracings for the sentence "You wish to know all about my grandfather" by a normal female speaker *(tracings on right)* and a female speaker with spastic dysarthria *(tracings on left)*.

The normal speaker completes the sentence in less than 2 seconds with normal variability in syllable duration and amplitude (energy tracing) and normal variability and declination in $f_0$ across the sentence (pitch tracing).

In contrast, the spastic speaker is slow (≈6.2 seconds for the utterance). The silent breaks evident in all tracings between "wish" and "to" and between "my" and "grandfather" are considerably lengthened and reflect slowness in achieving and releasing stop closure for /t/ and /g/, respectively. Other portions of the utterance in the energy and pitch tracings show little syllable distinctiveness, reflecting continuous voicing and restricted loudness and pitch variability. These acoustic attributes reflect the perceptible slow rate and monopitch and monoloudness that are characteristic of many speakers with spastic dysarthria.



The degree to which respiratory abnormalities affect speech in spastic dysarthria is unclear. Complicating their understanding is the fact that laryngeal valve hyperadduction is usually present, so even normal expiratory capacity must work against laryngeal resistance to airflow. In some cases, efforts to overcome severe glottic constriction during speech are so great that the speaker will seek momentary relief by volitionally releasing a considerable quantity of air. The result is intermittent breathiness and air wastage that can lead to reduced utterance length per breath group. Therefore deviations of respiratory activity may reflect the primary effects of underlying respiratory deficits but also secondary effects from abnormal laryngeal (and possibly resonatory and articulatory) activities as well.

### *Laryngeal Function*

Visual examination of the larynx at rest may reveal normal-appearing vocal folds. However, bilateral hyperadduction of the true and false vocal cords during speech may be apparent.[5,48]

Studies of patients with pseudobulbar palsy or multiple bilateral strokes have examined connected speech and vowel prolongation using various acoustic measures related to laryngeal function. They have found evidence of increased shimmer and jitter,* increased nonsyntactic breaks, increased standard deviation of fundamental frequency ($f_0$), decreased harmonic-to-noise ratio, decreased pitch variability, decreased words per minute and syllables per second, and decreased maximum vowel prolongation[25] (as reported by DAB[14]; Sherard, Marquardt, and Cannito[43]).

Using electromyography and aerodynamic measures, Murdoch, Thompson, and Stokes[33] reported that approximately half of their 14 subjects with stroke had hyperfunctional features such as increased subglottal air pressure, increased glottal resistance, and decreased laryngeal airflow. Other subjects had hypofunctional activity (including perceived breathiness in some subjects), thought possibly to reflect compensation or laryngeal hypertonus and muscle stiffness. Perceptual results did not concur with instrumental findings in approximately half of the subjects (e.g., some subjects with perceived hyperfunctional features had instrumental findings suggestive of laryngeal hypofunction, and vice versa). The authors questioned if this could be due to inadequacies of perceptual or instrumental methods, different tasks used for the two methods, or different compensatory strategies.

The findings of these studies generally align well with several of the primary perceptual features of spastic dysarthria, including monopitch, strained-harsh voice quality, and slow rate. Evidence of hypofunction from aerodynamic studies raises the possibility of weakness at the laryngeal level but might also reflect compensatory strategies, variations in the muscular dynamics of laryngeal spasticity, or methodological artifacts. Incongruities between perceptual and instrumental findings could also reflect methodological artifacts but might also reflect the sensitivity of instrumental methods to abnormalities that are dismissed or escape detection perceptually.

### *Velopharyngeal Function*

On oral inspection, the palate may move sluggishly or not at all during vowel prolongation. Palatal immobility, slow movement, and incomplete velopharyngeal closure may be apparent during videofluoroscopy and nasoendoscopy.

Thompson and Murdoch[44] found that 7 of 19 subjects with "UMN dysarthria" had hypernasality based on accelerometric recordings. Ziegler and von Cramon,[48] noting the tendency of some of their spastic subjects to voice voiceless stops, speculated that such distortions might be facilitated by incomplete velopharyngeal and oral cavity contacts that prevent interruption of phonation, even if vocal fold capacity is normal.* This explanation was supported by one of their subject's ability to produce voiceless stops when air wastage through the velopharyngeal port was decreased with the nares occluded. This observation illustrates the interactions at different levels of the speech system that may affect articulatory outcomes.

### *Articulation, Rate, and Prosody*

Numerous acoustic and physiologic studies have contributed to a better understanding of the articulatory dynamics and rate and prosodic impairments in spastic dysarthria. A few of the studies summarized here are detailed to illustrate the logic behind them and how they relate to clinical perceptual findings.

*Shimmer and jitter are "short-term" measures of departures from regularity (perturbation) in the voice. Shimmer reflects "cycle-to-cycle variations in the peak amplitude of the laryngeal waveform." Jitter reflects "cycle-to-cycle variation in the fundamental period."[27]

*The rapid laryngeal adjustments necessary for producing voiceless consonants are another source of voicing errors.

Acoustic studies support conclusions that rate of movement is slow and that range and precision of movement are reduced. Evidence of slowness comes from findings of reduced overall speech rate, increased word durations, increased syllable durations, prolonged phonemes, slow transitions from one phoneme to another, lengthened intersyllable pauses, reduced rate of amplitude variations, and slow speech AMRs. Evidence of imprecision and reduced range of movement derives from findings of acoustic energy within intersyllable gaps (imprecise articulation, spirantization) and centralization of vowel formants indicating restricted range of movement.[17,22,28,29,36,38,48] Some findings,[28,38] while confirming perceptual judgments of slow AMRs, suggest a degree of variability in timing and amplitude that has not generally been noted in perceptual studies.

Several other acoustic attributes suggest that imprecise articulation may be related to slowness, reduced range of movement, or weakness at the articulatory, velopharyngeal, or laryngeal valves. These include reduced sharpness of voiceless stops with a tendency toward voicing and reduced sound pressure level (SPL) contrasts in consonants (Alajouanine, Sabouraud, and Gremy, 1959, as summarized by DAB[14]; Ziegler and von Cramon[48]). Ziegler and von Cramon[48] attributed reduced SPL differences to inadequate voicing and hypernasality, as well as to the presence of friction noise *(spirantization)* with decreased amplitude of release bursts during production of stops. They noted that adequate production of stops and vowels was usually accomplished at the expense of articulatory rate. It is also instructive to note that voice onset time (VOT)—an acoustic reflection of timing control between laryngeal and supralaryngeal movements—is measurable less frequently in stop consonants of people with spastic dysarthria (84% measurable) than neurologically normal speakers (95% measurable).[37] This is most often due to lack of a burst signifying release of stop consonants, suggesting imprecision or a lack of firm articulatory contact. This implies that the greater than normal inability to make certain acoustic measurements in dysarthric speakers is an indirect way to document abnormality and, depending on the measure, may permit inferences about abnormal movement dynamics.

Chen and Stevens[11] used spectral analysis and spectrographic observations of /s/ in the initial position of words in a study of two normal and eight dysarthric subjects. Four of the dysarthric speakers had spastic cerebral palsy and can be presumed to have had spastic dysarthria. They had abnormal values on a number of the acoustic measures, but three deserve mention because together they predicted speech intelligibility: (1) spectral tilt, a measure (in dB) of the high-frequency prominence relative to the mid-frequency spectrum amplitude for /s/, served as an indirect measure of the proximity of the tongue blade to the lips and hence an indirect measure of articulatory precision; (2) time variation, a measure of variability of noise amplitude or spectrum shape during the /s/, was an indirect measure of the maintenance of a reasonably fixed tongue blade position and shape, and possibly fixed jaw position and relatively constant intraoral pressure; (3) precursor, a measure of the amount of inadvertent noise or voicing energy before the closure for the /s/, was an indirect measure of coordination among expiratory pressure, vocal fold configuration for the voiceless /s/, and the placement and shaping of the tongue blade for /s/.

A few studies have found different effects across speech structures. Ziegler and von Cramon[48] acoustically analyzed consonant-vowel-consonant (CVC) sequences and found disproportionate impairment of tongue-back movements relative to tongue-blade movements. Some studies have found relative preservation of range and control of jaw movement,[22,31] and Hirose[22] speculated that this might permit the jaw to compensate to some degree for inadequate tongue and lip articulatory movements. In a nonspeech visuomotor tracking study, McClean, Beukelman, and Yorkston[31] required subjects to track a sinusoidal wave with lower-lip and jaw movement (using strain gauge transducers), respiratory activity (by transducing air pressure changes in a face mask), and laryngeal activity (by altering $f_o$). Their one subject with spastic dysarthria had subnormal levels of respiratory tracking and greatly reduced tracking with the larynx but normal control of the jaw and lip. These observations suggest that spastic dysarthria may be associated with fine motor control difficulties that may vary across levels of the speech system.

In a kinematic study of lower lip trajectories during sentence production, Ackermann et al.[3] found reduced maximum speed of lip opening and closing gestures, as well as reduced peak velocity to maximum amplitude of lip movements, in three speakers with spastic dysarthria. These findings were interpreted as a reflection of "stiffness" and "central paresis due to an impairment of the upper motor neurons." Several other studies, using various physiologic methods, have documented slowness and reduced range of movement of the tongue, jaw, or palate.[22-24,26]

Using a rubber bulb tongue pressure transducer system, Thompson, Murdoch, and Stokes[44] examined tongue strength, rate of repetitive tongue movements, and ability to sustain maximum tongue contraction in 16 adults with stroke-related "UMN type dysarthria." Three of the subjects had bilateral lesions and may have had spastic dysarthria. In comparison to normal speakers, the dysarthric speakers had reduced tongue strength, reduced rate of repetitive tongue movements, and reduced ability to sustain maximum tongue contractions (i.e., reduced endurance or fatigue). Of interest, the transduced measures of tongue function were not significantly related to perceived articulatory adequacy. The authors suggested that the lack of relationship may have been because only some of the subjects had reduced strength beyond a critical level at which speech is affected, or that the relationship is not a linear one. Dworkin and Aronson[17] also found reduced tongue strength in speakers with spastic dysarthria, although not more so than in individuals with other dysarthria types.

Electropalatography has been employed to measure lingual-palatal contact during speech in a small number of people with spastic dysarthria. Detected abnormalities have included incomplete patterns of articulatory contact, smaller areas of contact, and greater numbers of contacts.[19,20] These abnormalities could reflect spatial as well as timing disturbances, and they imply reduced precision and accuracy of lingual speech movements.

Slow speech rate helps explain the presence of prosodic abnormalities in spastic dysarthria, but few investigations have examined vocal-stress patterns. Murry[34] tested the ability of five individuals with spastic dysarthria to vary stress during multiple productions of three-word sentences in which stress was placed on varying words. Peak intraoral pressure, duration of the pressure pulse, $f_o$, vowel duration, and vowel intensity were measured. In contrast to normal speakers, spastic speakers conveyed phrase final word stress only with frequency and intensity changes. They also generally conveyed stress less adequately than normal and usually by compensation. For example, spastic speakers seemed to use increased articulatory effort for phrase initial word stress. For final word stress, they increased $f_o$ and intensity, but articulatory effort was compromised. Murry[34] concluded that when spastic dysarthric subjects use consonant-related cues to stress an initial word, vowel-related cues are decreased relative to baseline. For final word stress, they switch to a vowel strategy and reduce articulatory effort. They did not generally use vowel duration cues to vary stress in any position.

Finally, Roy et al.[41] examined several perceptual, acoustic, and physiologic parameters in a man with severe spastic dysarthria from a TBI. Before initiation of treatment, acoustic analyses identified slow and shallow F2 format transitions (i.e., slow movement and reduced range of movement) and reduced vowel space (i.e., reduced acoustic distinctiveness among different vowels). Nasometry and aerodynamic measures of velopharyngeal function identified reduced oral pressures, increased nasal airflow, and increased nasalance. All findings are consistent with auditory perceptual features of spastic dysarthria. The study is noteworthy because it illustrates the value of combining perceptual, acoustic, and physiologic measures to understand specific speech subsystem contributors to reduced intelligibility and their contribution to treatment decisions and measurement of change.

To summarize, acoustic and physiologic studies have documented the presence of impairments at all levels of the speech system in spastic dysarthria, and, for the most part, they provide strong support for many of the perceptually recognizable features of the disorder. Within each speech subsystem there is evidence of slowness, reduced range and precision of movements, and sometimes variability of movement control. The studies support and refine perceptual observations of imprecise articulation and indicate that at least some affected people lack precision and control for articulatory placement. Physiologic studies have defined some of the movement dynamics underlying the perception of slow rate, and they support inferences that spastic dysarthria reflects a combination of spasticity and weakness. There is some evidence that the motor control difficulties associated with the disorder can vary across levels of the speech system. Finally, there is evidence that some acoustic correlates of precision, steadiness, and coordination in spastic dysarthria are related to intelligibility. Chen and Stevens[11] concluded that one goal of ongoing acoustic analyses should be "to assemble a set of parameters that, in combination, can predict the intelligibility of a dysarthric speech signal and can be interpreted in terms of deviations in control of the speech production system." If this goal can be met, and if the related analyses can be relatively automated and cost-effective, acoustic analysis will become highly valuable in many clinical settings.

## Cases

### Case 5-1

A 65-year-old woman presented with a 6-month history of "slurred speech" and dysphagia. Prior history was unremarkable, except for hypertension that did not require medication. Her initial difficulty with swallowing was greater for liquids than solids and had progressed to a point where she had extreme difficulty with liquids. She had not lost weight, nor had she had difficulty with aspiration or nasal regurgitation. A short time after her dysphagia developed, she noted speech difficulty that had also gradually progressed. She had been placed on Mestinon for MG by a neurologist at another institution, without benefit.

The neurologic examination, beyond her speech difficulty and dysphagia, revealed mild bilateral facial weakness and bilaterally increased deep tendon and Babinski reflexes. Arm and leg AMRs were diminished slightly on the left. Laboratory tests were essentially normal, as were screenings for hereditary demyelinating syndromes. Nerve conduction studies and electromyogram (EMG) were normal, including EMG examination of the tongue. MRI of the head was normal.

During speech examination, the patient said she initially attributed her swallowing difficulty to her dentures. At onset, her tongue felt "thick," and she was aware of a "nasal tone" to her voice. Psychologic stress and prolonged speaking made speech worse. She admitted to occasionally biting her cheek when chewing; food sometimes squirreled in her cheeks. She had compensated by chewing more slowly and eating smaller amounts to prevent choking. She admitted to difficulty controlling emotional expression.

She frequently had an "on the verge of crying" facial expression. Jaw strength was normal. The lower face was weak (−1) on voluntary lip retraction. The tongue was full and symmetric, but lateral tongue movements were slow (−2, 3). The tongue was moderately weak bilaterally, slightly more so on the left. The palate was symmetric and mobile. Gag reflex, cough, and glottal coup were normal. A sucking reflex was equivocally present.

Conversational speech and reading were characterized by reduced rate (2), monopitch and monoloudness (2), strained-harsh-groaning voice quality (1,2), occasional pitch breaks, hypernasality (0,1), and imprecise articulation (1,2). Prolonged "ah" was sustained for 11 seconds and was equivocally strained. Her speech AMRs were slow (2,3) but regular. Intelligibility was judged normal in the quiet one-to-one setting but probably mildly compromised in noise.

Acoustic analysis showed $f_0$ (242 Hz) and measures of jitter and shimmer to be grossly normal. Speech AMRs for /pʌ/, /tʌ/, and /kʌ/ were 2.8, 2.8, and 2.5 Hz, respectively.

The clinician concluded: "Spastic dysarthria, suggestive of bilateral UMN involvement affecting the bulbar muscles. There are no clear-cut features of flaccid dysarthria, nor do I note characteristics that could be interpreted as ataxic." Speech therapy and management for her dysphagia were recommended.

The neurologist concluded that the patient had progressive UMN dysfunction of undetermined etiology but wondered about primary lateral sclerosis. Reevaluation in 3 to 6 months was recommended. She did not return for follow-up.

**Commentary.** (1) Degenerative neurologic disease can present as dysarthria or dysphagia. (2) Diagnosis of spastic dysarthria places the lesion in the CNS, bilaterally, and can help to rule out disease isolated to LMNs (e.g., MG). (3) Early during their course it is not unusual for degenerative diseases in which spastic dysarthria and dysphagia are the primary signs to defy more specific neurologic diagnosis, and for neuroimaging studies to be normal.



### Case 5-2

A 41-year-old right-handed man from Saudi Arabia was hospitalized for management of hypertension and speech and swallowing difficulties. According to his family, he had fairly adequate English language skills.

The patient had a 2-year history of hypertension for which he had refused to take medication. Eleven months previously, over the course of an evening, he developed left hemiplegia. Ten days later he lost consciousness and upon awakening 17 days later was unable to speak or swallow. His left hemiplegia persisted, but he had no motor signs on the right side of the body. With therapy his left-sided weakness improved, but swallowing and speech remained significantly impaired. He had been fed through a nasogastric tube, but more recently he had been eating puréed foods while lying supine.

Neurologic examination revealed a left hemiparesis. Upper limb reflexes were hyperactive bilaterally, left greater than right. He was unable to speak. Questions were raised about whether the patient had an "expressive aphasia," or if a component of his speech difficulty was psychogenic. It was assumed that his lesion was unilateral (right).

On speech examination, he was nearly *anarthric* (speechless due to severe dysarthria). He could produce a nasally emitted and resonated, quiet but strained-strangled undifferentiated vowel with great effort but little else. With his lips closed he produced a prolonged and strained /m/. Voluntary lip and jaw movements were slow and limited in range but were more extensive during reflexive swallowing; the jaw opened widely during a reflexive yawn. Suck, snout, and jaw jerk reflexes were present. At rest the tongue sat in a relatively retracted position. Tongue movement was minimal and slow; he was unable to extend it beyond the edge of the lower teeth and unable to elevate or move it laterally. The palate hung so low in the pharynx that the uvula could not be seen; a gag reflex could not be elicited. Surprisingly, his cough was sharp.

There was no evidence of language difficulty. He followed two-step commands and communicated effectively through writing, although with occasional spelling errors.

It was concluded that he had a "severe spastic dysarthria without any evidence of aphasia or apraxia of speech, and no clear evidence of a psychogenic contribution to his speechlessness. To produce a dysarthria like this, the lesion should be bilateral."

Subsequent CT scan revealed old infarcts in the centrum semiovale of both hemispheres, as well as an infarction in the right posterior parietal cortex (Figure 5-4).

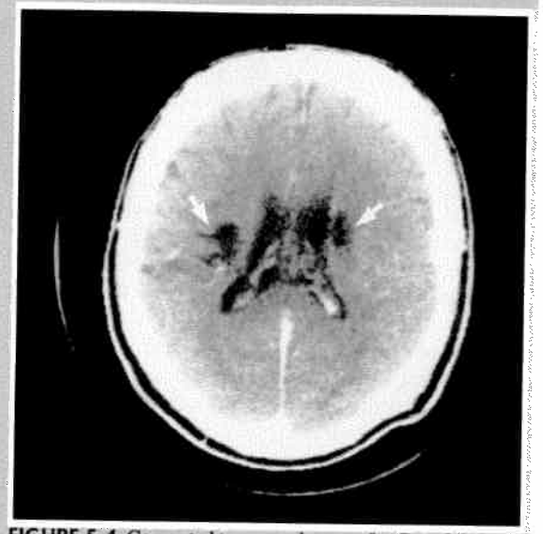

**FIGURE 5-4** Computed tomography scan for Case 5-2. Relatively small infarcts in the centrum semiovale bilaterally *(arrows)* were associated with a severe spastic dysarthria.

A brief period of speech therapy was undertaken, but it was soon apparent that intelligible speech would not be achieved. Vocal loudness increased and hypernasality decreased when the palate was elevated from the surface of the tongue with a tongue depressor. A palatal lift prosthesis was made to ease swallowing, but the weight of the velum on the device made it impossible to keep the prosthesis securely fastened. The patient underwent pharyngeal flap surgery and was then able to eat puréed food while sitting in an upright position, although it took 2 hours for him to complete a meal. He could breathe orally. Writing was an effective, portable, but somewhat inefficient means of communication for him. He returned to his home before other means of augmentative communication could be thoroughly investigated.

**Commentary.** (1) The presence of significant spastic dysarthria should raise questions about bilateral UMN involvement, even when limb findings suggest that the lesion is only unilateral. (2) Lesions do not have to be large to produce devastating consequences for speech. The patient's centrum semiovale lesions were small, but their locus was sufficient to interrupt UMN pathways to the bulbar speech muscles bilaterally. (3) Severe spastic dysarthria is almost always accompanied by significant dysphagia. (4) Accurate diagnosis of the speech deficit helped to rule out aphasia, as well as significant psychogenic influences. This information was useful in counseling the patient and family, particularly their understanding of the nature of the problem and their acceptance of limitations on future recovery of speech.

## Case 5-3

A 71-year-old woman presented with a 3-month history of "lost voice." Prior medical history was unremarkable. The only abnormality on ear, nose, and throat (ENT) examination was decreased tongue mobility. "Neurologic dysphonia" and possible "LMN disease" were suspected. Speech pathology and neurology consultations were arranged.

During speech evaluation, the patient recalled that her progressing speech difficulty had been present for approximately 15 months. She complained that her voice was strained, that speech was slow, and that speaking was effortful. She had difficulty swallowing liquids, with occasional choking and infrequent nasal regurgitation. She had not had to modify her diet, nor had she lost weight. She denied change or difficulty controlling emotional expression, drooling, and difficulty with memory or other cognitive skills.

Speech AMRs of the jaw, lower face, and tongue were slow (2) but regular. Jaw and lower face strength were normal; the left side of the tongue was equivocally weak. There was a slight droop at the right corner of the mouth and a subtle "snarl" of the left upper lip at rest. The palate was symmetric and relatively immobile during vowel prolongation but moved normally during gagging. Nasal emission was apparent during pressure sound production. Her cough was normal. Suck, snout, and jaw jerk reflexes were not detected.

A strained-harsh-groaning voice quality (2), reduced rate (1,2), hypernasality (1,2), imprecise articulation (1), and monopitch and monoloudness (1,2) characterized connected speech. Lip and jaw movements were slightly exaggerated during speech, possibly reflecting compensatory efforts to maintain intelligibility. Speech AMRs were slow (2,3). "Ah" was strained (3) and sustained for only 6 seconds.

The clinician concluded, "Spastic dysarthria, moderately severe. No clear evidence of a flaccid (LMN) component. Speech characteristics are strongly suggestive of bilateral UMN dysfunction affecting the bulbar musculature." She was referred for speech therapy and management of her dysphagia, which she pursued closer to home.

Neurologic examination noted brisk muscle stretch reflexes but no pathologic reflexes. No fasciculations were detected. Subsequent EMG failed to identify fibrillations or fasciculation potentials. MRI of the head, with special attention to the brainstem, was normal. The neurologist concluded that the patient had a pseudobulbar palsy with spastic dysarthria plus minimal findings in the upper limbs. ALS was suspected, but a diagnosis could not be confirmed. She was not seen for subsequent follow-up.

**Commentary.** (1) Speech difficulty can be the presenting complaint in neurologic disease. (2) Spastic dysarthria can occur in the absence of other significant neurologic deficits and can progress without significant clinical findings in the limbs. (3) Spastic dysarthria is frequently accompanied by dysphagia. (4) Dysarthria affecting the bulbar muscles, in the absence of limb findings, is sometimes misinterpreted as LMN disease (frequently MG). Careful speech examination can help establish the presence of bilateral UMN involvement in such cases.

## Case 5-4

An 80-year-old woman was admitted to the hospital neurology service following the sudden onset of speech difficulty. She had a 10-year history of hypertension. Approximately a year before the current admission she had the sudden onset of dysarthria, dysphagia, and right-hand clumsiness, all which resolved within 10 days.

Neurologic examination identified significant dysarthria, dysphagia, and left-hand weakness, as well as hyperactive reflexes on the left. A diagnosis of a right internal capsule or pontine infarct was made. Subsequent MRI and CT scans identified moderate generalized atrophy and multiple focal areas of abnormality in the hemispheric white matter bilaterally, consistent with subcortical ischemic disease.

Speech examination revealed both left and right lower facial weakness with reduced range of movement on smiling, lip rounding, and lip puffing. Tongue protrusion and lateralization were limited in range. Gag reflex was hypoactive. A sucking reflex was not present. A hoarse, strained voice quality, reduced loudness, imprecise articulation, hypernasality, and monopitch and monoloudness characterized contextual speech. Speech AMRs were slow (2) but regular. Speech intelligibility was reduced. There was no evidence of aphasic language impairment or apraxia of speech.

The clinician concluded that the patient had a "marked spastic dysarthria with significantly reduced speech intelligibility. The tongue is markedly weak, but this is probably on a bilateral UMN basis."

Speech therapy was recommended, which she pursued closer to home. Neuropsychological assessment identified moderate generalized cognitive dysfunction, most evident in areas of attention and concentration, new learning and memory, and reasoning and problem solving.

**Commentary.** (1) Although excellent recovery from unilateral UMN lesions causing dysarthria is possible, additional lesions on the other side of the brain can result in spastic dysarthria with significant reduction of speech intelligibility. (2) When spastic dysarthria is present following an apparent unilateral cerebral event, suspicions should be raised about bilateral lesions. In this case, the history and current event helped establish the presence of more than one lesion.



## SUMMARY

1. Spastic dysarthria results from damage to the direct and indirect activation pathways (UMNs) bilaterally. It occurs at a frequency comparable to that of other single dysarthria types. Its deviant speech characteristics reflect impaired movements and movement patterns, usually at all levels of speech production. The combined effects of spasticity and weakness on the speed, range, and force of movement seem to account for most deviant speech characteristics of the disorder.
2. Clinical signs that accompany spastic dysarthria usually include weakness, loss of skilled movement, spasticity, hyperactive reflexes, and pathologic reflexes. The salient effects of UMN lesions on speech movements include spasticity, weakness, reduced range of movement, and slowness of movement.
3. Degenerative and vascular etiologies probably account for a majority of cases of spastic dysarthria, but traumatic, demyelinating, neoplastic, and undetermined etiologies are not uncommon. Most patients with spastic dysarthria have other clinical signs or neuroimaging evidence of bilateral UMN dysfunction, but spastic dysarthria can be the only neurologic sign in some cases. The distribution of offending lesions can be widespread in the UMN system, including pathways ranging from the cortex to brainstem.
4. Dysphagia and pseudobulbar affect are common in people with spastic dysarthria. Complaints that speech is slow and effortful and deteriorates with fatigue are also common.
5. The major clusters of deviant speech characteristics in spastic dysarthria include prosodic excess, articulatory-resonatory incompetence, prosodic insufficiency, and phonatory stenosis. Although many deviant speech characteristics can be detected in spastic dysarthria, strained-harsh voice quality, slow speech rate, and slow and regular speech AMRs are the most distinctive clues to the presence of spastic dysarthria.
6. In general, acoustic and physiologic studies of individuals with spastic dysarthria have provided quantitative support for its clinical perceptual characteristics. They have helped to specify more completely the location and dynamics of abnormal movements that lead to the perceived speech abnormalities.
7. Spastic dysarthria can be the only, the first, or among the first or most prominent manifestations of neurologic disease. Its recognition and correlation with bilateral UMN dysfunction can aid the localization and diagnosis of neurologic disease and may influence decision making for medical and behavioral management.

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chapter **6**

# Ataxic Dysarthria

*"Well, I slur the 'ph' and the 'th' and some of the harsh sounds. And they come real slurred, almost like I was drunk . . . and it's like I can't control my lips and tongue, and they'll occasionally get in my way. I know this could be carelessness, but it very seldom used to happen. Now it happens quite often!"*

*(62-year-old man with degenerative cerebellar disease and ataxic dysarthria)*

**CHAPTER OUTLINE**



Ataxic dysarthria is a perceptually distinctive motor speech disorder (MSD) associated with damage to the cerebellar control circuit. It may be manifest in any or all of the respiratory, phonatory, resonatory, and articulatory levels of speech, but *its characteristics are most evident in articulation and prosody*. The disorder reflects the effects of incoordination and reduced muscle tone, the products of which are slowness and inaccuracy in the force, range, timing, and direction of speech movements. Ataxia is an important contributor to the speech deficits of patients with cerebellar disease, hence the disorder's designation *ataxic* dysarthria. The identification of a dysarthria as ataxic can aid the diagnosis of neurologic disease and its localization to the cerebellum or cerebellar control circuit.

Ataxic dysarthria is encountered as the primary speech pathology in a large medical practice at a rate comparable to that for most other major single dysarthria types. Based on data for primary communication disorder diagnoses within the Mayo Clinic Speech Pathology practice, it accounts for 10.8% of all dysarthrias and 9.9% of all MSDs (see Figure 1-3).

The clinical features of ataxic dysarthria illustrate the important role of the cerebellum and its connections in speech motor control. Of all the individual dysarthria types, it most clearly reflects a breakdown in timing and coordination. When one listens to the speech of a person with ataxic dysarthria, the impression is not one of underlying weakness, resistance to movement, or restriction of movement, but rather one of an activity that is poorly timed and coordinated.

## ANATOMY AND BASIC FUNCTIONS OF THE CEREBELLAR CONTROL CIRCUIT

The cerebellar control circuit consists of the cerebellum and its connections. Its components are described in detail in Chapter 2. Here its structures, pathways, and functions that are most relevant to speech are briefly summarized.

The vermis forms the midportion of the anterior and posterior lobes of the cerebellum. To the sides of the vermis are the right and left cerebellar hemispheres, each of which is connected to the opposite thalamus and cerebral hemisphere. Each cerebellar