

Unilateral Upper Motor Neuron Dysarthria

"I didn't even know anything happened except I was talkin' to this gal, and I said, 'somethin's happened to me and I can't talk real good!'"

(82-year-old woman describing the onset of her right internal capsule lacunar stroke)

CHAPTER OUTLINE

- I. Anatomy and basic functions of the upper motor neuron system
- II. Clinical characteristics associated with unilateral upper motor neuron lesions
- III. Etiologies
- IV. Speech pathology
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Unilateral upper motor neuron (UUMN) dysarthria is an often distinguishable motor speech disorder associated with damage to the upper motor neuron (UMN) pathways that carry impulses to the cranial and spinal nerves that supply the speech muscles. It may be manifest in any component of speech but is most often apparent in articulation, phonation, and prosody. Its deviant characteristics usually reflect the effects of weakness on speech, but sometimes spasticity and incoordination are implicated. The identification of a UUMN dysarthria can aid the diagnosis of neurologic disease and its localization to central nervous system (CNS) motor pathways.

In contrast to other dysarthria types, the label for this dysarthria is anatomic rather than pathophysiologic. This is because only in recent years have we begun to carefully describe its clinical perceptual characteristics and understand its anatomic and physiologic correlates. We do know that the disorder's clinical features and anatomic and physiologic correlates can vary considerably among affected people. It thus seems best to avoid a single physiologic label until clinical characteristics and their underpinnings are better defined and to use a label that conveys what is most certain about it, hence its designation as *UUMN dysarthria*. The reasons for the variability associated with UUMN dysarthria, as well as some related practical clinical issues, are tied together at the end of this chapter.

Why has UUMN dysarthria received limited attention? One reason is that historically it has been considered a mild and temporary problem (e.g., Darley, Aronson, and Brown [DAB]¹⁸; Metter¹⁸). Although this is not always the case, disorders that frequently are mild and short-lived in their clinical manifestations are naturally difficult to study. In addition, UUMN dysarthria often occurs simultaneously with aphasia or apraxia of speech when the lesion is in the left hemisphere and with cognitive or nondysarthric speech deficits when the lesion is in the right hemisphere. Such disorders can be devastating in their effects on communication: as a result, a dysarthria may be masked by them or made more difficult to study because of their presence. In general, therefore, UUMN dysarthria has probably received little attention because of its presumed mildness and short duration, and its frequent

cooccurrence with deficits that may mask or overwhelm its manifestations, minimizing its functional importance and making it difficult to isolate and study.

It should be recognized, however, that UUMN dysarthria is sometimes a person's only or most obvious communication disorder and sometimes the only or most obvious manifestations of neurologic disease.* Its recognition is especially important when it is a relatively isolated sign, because the offending lesion tends to be small and can escape detection by neuroimaging techniques, especially early after onset. An understanding of UUMN dysarthria's characteristics is also important, because it can occur simultaneously and be difficult to distinguish from other speech disorders associated with unilateral CNS disease, such as apraxia of speech (left hemisphere lesions) and aprosodia (right hemisphere lesions).

UUMN 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 8.5% of all dysarthrias and 7.8% of all motor speech disorders (MSDs) (see Figure 1-3). This is almost certainly an underestimate of its actual prevalence in clinical practice, because it occurs frequently as a secondary diagnosis for people with aphasia, apraxia of speech, or nonaphasic cognitive-communication deficits.

The clinical features of UUMN dysarthria nearly always reflect, at least in part, the effects of unilateral UMN weakness in the face and tongue, and sometimes other levels of the speech system. In some cases, however, deviant speech characteristics also suggest effects of spasticity, incoordination, or both, sometimes making the overall speech pattern difficult to distinguish from spastic or ataxic dysarthria. These perceptual ambiguities can usually be clarified by additional clinical data.

■ ANATOMY AND BASIC FUNCTIONS OF THE UPPER MOTOR NEURON SYSTEM

The UMN system includes the *direct and indirect activation pathways*. They were described in detail

*Urban et al.⁷⁴ reported that, in 69 consecutive patients with the sudden onset of dysarthria due to a single stroke, isolated dysarthria or dysarthria with central facial and lingual paresis occurred in 3% and 10%, respectively; dysarthria-clumsy hand syndrome in 12%; and dysarthria with pure motor hemiparesis or ataxic hemiparesis in 28%. Lesions were in the lower part of the primary motor cortex, the centrum semiovale, the internal capsule, the cerebral peduncle, the base of the pons, or the ventral pontomedullary junction. All of these locations are along the course of the pyramidal tract.

in Chapter 2 and reviewed again in Chapter 5 when the effects on speech of bilateral UMN lesions (spastic dysarthria) were addressed. These pathways are reviewed here only with reference to their implications for understanding UUMN dysarthria and the neurologic deficits that frequently accompany it. Their relevant anatomy and functions can be summarized as follows:

1. The UMN system is bilateral, half originating in the right hemisphere and half in the left hemisphere.
2. The *direct activation pathway* of the UMN system passes directly as corticobulbar and corticospinal tracts to the cranial and spinal nerves, respectively, mostly to the side opposite their origin. It emerges from the *cerebral cortex* and begins its descent in the *corona radiata*. The corona radiata converges into the *internal capsule* in the vicinity of the basal ganglia and thalamus (corticobulbar fibers are grouped primarily in the *genu*, or midportion, of the internal capsule). From there it descends to the brainstem, where corticobulbar fibers cross to the opposite side just before reaching the cranial nerve nuclei they are to innervate; corticospinal fibers cross in the pyramids of the medulla. The impulses traveling in the direct pathway appear *crucial for finely coordinated skilled movements*.
3. The *indirect activation pathway* of the UMN system has the same predominantly contralateral destinations, and it crosses in the brainstem in the same general areas as the direct activation pathway. However, along its route to the cranial and spinal nerves are synaptic connections in several intervening structures, lying mostly in the *reticular formation* and *other brainstem nuclei*. This pathway appears *crucial for regulating reflexes and controlling posture and tone*, upon which skilled movements are superimposed.
4. In the bulbar speech muscles of most people, *the general principle of contralateral innervation holds true only for the lower face and, to a lesser and probably variable degree, the tongue*. The trigeminal nerve, the fibers of the facial nerve going to the upper face, and the glossopharyngeal, vagus, accessory, and, at least in some individuals, hypoglossal nerves receive both contralateral and ipsilateral UMN innervation. This bilateral input to most of the speech cranial nerves provides a degree of redundancy that helps to preserve breathing, feeding, and motor speech functions when UMN lesions are confined to one

box

9-1

Primary clinical features of UUMN lesions. All features are present on the side of the body contralateral to the lesion.

Direct Activation Pathway (Pyramidal Tract)

Hemiplegia or hemiparesis
Loss/impairment of fine, skilled movements
Absent abdominal reflex
Babinski's sign
Hyporeflexia
Central facial weakness at rest and during voluntary movement
Lingual weakness

Indirect Activation Pathway (Extrapyramidal Tract)

Increased muscle tone
Spasticity
Clonus
Hyperactive stretch reflexes
Decerebrate or decorticate posturing
Central facial weakness apparent during emotional expression

UUMN, Unilateral upper motor neuron.

side of the brain. However, this redundancy is not always all protective. Evidence suggests that *at least some individuals with UUMN lesions have detectable contralateral weakness of the jaw, palate, vocal folds, and, most frequently and obviously, the tongue*; in some cases, even ipsilateral weakness can be measured. This is important to remember, because it helps explain several of the deviant speech characteristics that can be present in UUMN dysarthria. This is addressed later in the speech pathology section.

CLINICAL CHARACTERISTICS ASSOCIATED WITH UNILATERAL UPPER MOTOR NEURON LESIONS

The distinctive effects of UUMN lesions affecting the direct and indirect activation pathways are summarized in Box 9-1.* Briefly, such lesions are often associated with contralateral hemiplegia or hemiparesis. A *Babinski reflex* is usually present on the affected side.

A combination of weakness and spasticity is usually present in the affected limbs. Weakness, hyporeflexia, and hypotonia in the limbs tend to predominate shortly after the onset of acute lesions,

with spasticity, hyperactive stretch reflexes, and increased muscle tone often emerging over time. Limb motor deficits tend to be worse when muscle flaccidity (as opposed to spasticity) is prolonged following stroke. Evidence suggests that prolonged flaccidity is associated with a higher prevalence of structural involvement of the lentiform nucleus and reduced cerebral blood flow in the lentiform nucleus, thalamus, and contralateral cerebellum.⁵⁸ Whether or not structural or physiologic involvement of these basal ganglia and cerebellar control circuits predicts specific deviant features of UUMN dysarthria, or its severity and prognosis, has yet to be determined.

Corticobulbar involvement is often manifest by varying degrees of *contralateral lower facial weakness*. This is usually called *central (or supranuclear) facial weakness* to distinguish it from peripheral cranial nerve VII lesions that usually affect the upper and lower face. Similarly, when contralateral lingual weakness is present, it is often called *central lingual weakness*.

A combination of direct and indirect pathway lesion effects are usually present, at least in the limbs. Depending on the specific site of lesion, however, there may be relative sparing of the upper or lower limb or bulbar muscles. For example, some lesions affect only the bulbar muscles or only the bulbar muscles and hand.

ETIOLOGIES

Any process that can damage UMNs unilaterally can cause UUMN dysarthria. Because degenerative, inflammatory, and toxic-metabolic diseases usually produce diffuse effects, they are rarely associated with focal unilateral signs, including UUMN dysarthria. Tumors confined to one side of the CNS can cause UUMN dysarthria when they invade or produce mass effects on UMN structures and pathways unilaterally. Trauma, particularly surgical trauma, can produce focal deficits, including UUMN dysarthria; the typical multifocal, bilateral, or diffuse deficits associated with closed head injury are usually associated with other dysarthria types.

Stroke is by far the most common cause of UUMN damage, and dysarthria is a frequent consequence of stroke, occurring in 29% of patients with stroke associated with hemiparesis.⁴⁶ It is thus appropriate to review some of the vascular conditions that can produce relatively isolated UMN deficits.

Left carotid or middle cerebral artery occlusions are the most common causes of strokes leading to UMN deficits that are also accompanied by aphasia or apraxia of speech. Right carotid or middle cerebral artery occlusions are the most common cause of

*These features are discussed in more detail in Chapter 5.

strokes leading to UMN deficits that are also accompanied by neglect and cognitive disturbances characteristic of right hemisphere pathology. Unilateral strokes in the distribution of the posterior cerebral, basilar, and, less frequently, anterior cerebral arteries can also cause UUMN deficits.

Sometimes small infarcts occur in the brainstem or cortical or subcortical areas of the cerebral hemispheres as the result of occlusion of the small penetrating branches of the large cerebral arteries. These small infarcts are often called *lacunes* or *lacunar infarcts* because, in healing, they leave behind a small cavity (lacune).^{*} They most often involve the lenticulostriate branches of the anterior and middle cerebral arteries, the thalamoperforant branches of the posterior cerebral arteries, and the paramedian branches of the basilar artery. The most common sites of lacunar strokes are the putamen, caudate nucleus, thalamus, pons, internal capsule, and white matter below the cerebral cortex.²³ These locations establish the relevance of lacunes as a mechanism for producing UUMN dysarthria (and spastic dysarthria, when lesions are bilateral); that is, most of them are part of the UMN pathways.[†] In addition, because of their location, lacunes often are not associated with aphasia, neglect, visual field deficits, severe memory impairment, or alterations in consciousness; their signs usually are primarily motor or sensorimotor. Lacunar stroke is probably the most frequent cause of UUMN dysarthria when dysarthria is a relatively isolated sign of stroke.[‡]

Fisher²⁵ has outlined a number of "lacunar syndromes." Dysarthria (presumably UUMN dysarthria) is among the defining characteristics for several of them.[§] The most relevant of these are:

1. *Pure motor hemiparesis.* A pure motor stroke involving the face, arm, and leg on one side in the absence of sensory deficit, homonymous hemianopia, aphasia, agnosia, or apraxia. The lesion may be in the corona radiata, internal capsule, cerebral peduncle,

or pons. The vascular origin is usually a branch of the middle cerebral artery or verte-brobasilar system.

2. *Ataxic hemiparesis.* This involves signs of pure motor hemiparesis plus cerebellar dysmetria in the affected limbs. The lesion is often in the pons, internal capsule, or corona radiata.^{22,32,41,44} It has also been reported in people with thalamic lesions, often including the posterior limb of the internal capsule.^{47,76}
3. *Dysarthria clumsy hand syndrome.* Facial weakness, dysarthria, and dysphagia are prominent, but there is also slight weakness and clumsiness of the hand. The lesion is usually in the pons; genu or posterior limb of the internal capsule; or the adjacent corona radiata, caudate nucleus, or cerebral peduncle.^{27,41,42,44,62,71} This syndrome may account for 6% of lacunar infarcts.¹³
4. *Pure dysarthria.* The sudden onset of dysarthria without other signs (except for face and tongue weakness). This syndrome, which may be a variant of the dysarthria clumsy-hand syndrome,⁴⁰ was found in approximately 1% of 670 consecutive cases of stroke.⁴ The genu of the internal capsule or the adjacent corona radiata are probably the most frequent lesion sites in cases of pure dysarthria resulting from a unilateral stroke, but lesions in the basal ganglia,^{*} base of the pons, and cortical-subcortical motor area have also been reported.^{9,31,34,40,41,44,56,57,73} Urban et al.⁷³ concluded, "interruption of the cortico-lingual pathways is crucial in the pathogenesis of isolated dysarthria after extracerebellar lacunar stroke"; affected patients had no involvement of corticospinal tracts and no cerebellar diaschisis.

■ SPEECH PATHOLOGY

It is unfortunate that the neurology literature's often-refined descriptions of lesion loci associated with dysarthria are not matched by clear descriptions of specific speech deficits. Beyond describing speech as dysarthric, description is usually limited to vague terms such as "slow dysarthria," "slurred," "unintelligible," or "thick."

Systematic prospective studies of UUMN dysarthria providing detailed descriptions of speech and oral mechanism findings are few in number, and their data are based on small numbers of subjects. In this section, the results of a relatively large retrospective study of 56 patients with UUMN

^{*}Lacunes account for approximately 25% of all strokes in some clinical practices.¹³ They range in size from 0.2 to 15 mm³; the smallest lacunes may escape detection by computed tomography (CT) scan.⁵⁰

[†]Dysarthria has been found in 25% of patients with lacunar infarcts³ and occurs in approximately 30% of patients with stroke in the internal capsule.²⁴

[‡]For example, Urban et al.⁷⁴ reported that in 69 consecutive patients with the sudden onset of dysarthria due to a single stroke, lacunar stroke was the cause in 53%.

[§]Dysarthria can also be a defining feature of nonlacunar infarcts. For example, Kataoka et al.³⁵ found that it was the most common clinical sign among 49 patients with acute paramedian pontine infarcts, occurring in 55% of the patients.

^{*}Facial weakness is apparently common (50%) in unilateral putaminal lacunar strokes.²⁶

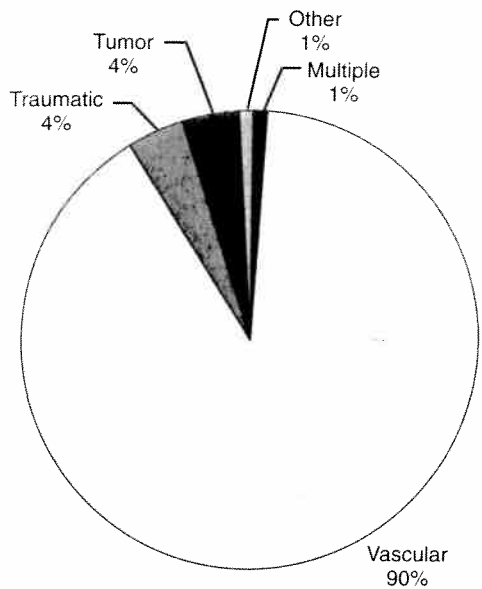


FIGURE 9-1 Distribution of etiologies for 98 Mayo Clinic patients with a primary speech pathology diagnosis of unilateral upper motor neuron dysarthria (see text for description of data sources and Box 9-2 for other details).

box
9-2

Etiologies for 98 Mayo Clinic patients with a primary speech pathology diagnosis of UUMN dysarthria (see text for description of data sources). Percentage of cases for broad etiologic headings is given in parentheses. Specific etiologies under each heading are ordered from most to least frequent.

Vascular (90%)

Nonhemorrhagic or hemorrhagic stroke

Traumatic (4%)

Neurosurgical (4%)

Tumor resection; thalamotomy

Tumor (4%)

Other (1%)

Cerebritis

Multiple (1%)

Nonhemorrhagic stroke + tumor resection

UUMN, Unilateral upper motor neuron.

dysarthria*²¹ serve as the primary vehicle for describing the disorder. Findings from prospective studies are used to support, supplement, and qualify or modify the observations of Duffy and Folger as appropriate.

Distribution of Etiologies, Lesions, and Severity in Clinical Practice

Etiology

Figure 9-1 and Box 9-2 summarize the etiologies for 98 Mayo Clinic patients with a primary speech pathology diagnosis of UUMN dysarthria. Duffy and Folger²¹ described 56 of the cases in their retrospective study. The remaining 42 cases were quasirandomly selected from cases seen from 1999-2001. The cautions expressed in Chapter 4 about generalizing these observations to the general population or all speech pathology practices apply here as well.

*Duffy and Folger's patients were selected on the basis of their speech diagnosis and clinical or neuroimaging evidence of only a single lesion confined to one side of the brain. Patients with parkinsonism and cerebellar lesions were excluded, as were all patients with apraxia of speech. Patients with aphasia that was severe enough to preclude obtaining a sufficient speech sample also were excluded (18% of the sample had aphasia, but it was usually mild).

The data establish that UUMN dysarthria is almost always due to stroke, tumor, or neurosurgery, and stroke is the overwhelmingly predominant cause (90%). Nonhemorrhagic strokes, which account for the highest proportion of neurovascular disturbances in general, accounted for most of the vascular causes. The predominance of stroke as an etiology is consistent with most other studies that have carefully examined the dysarthria associated with UUMN lesions.*^{7,28,33,36,66,67,38}

Lesion Loci

The lesion sites for Duffy and Folger's patients are summarized in Box 9-2. Lesions were supratentorial in approximately 95% of the cases. The internal capsule, pericapsular regions, and regions affecting all or portions of cerebral hemisphere lobes were the most common lesion sites. Larger lesions in the cerebral hemispheres nearly always included the frontal lobes. A few patients had lesions in the thalamus, midbrain, or pons. These lesion loci are consistent with the anatomy of the UMN system, its vascular

*The exclusive stroke etiology in some studies may reflect a desire to study patients with small, focal lesions rather than the natural distribution of etiologies of the disorder. Small strokes are ideal for investigating UUMN dysarthria, because their anatomic boundaries are easier to define than those of diseases with more difficult-to-localize effects, such as traumatic brain injury, tumor, or infection.

supply, and the literature on the locus of lacunar strokes that can produce dysarthria. It is also important to recognize that unilateral stroke affecting regions of the basal ganglia, sometimes including portions of the internal capsule, have also been associated with dysarthria and facial weakness.^{7,12,33}

Regarding side of lesion, 61% had lesions in the left hemisphere and 34% had lesions in the right hemisphere. This is consistent with some other studies reporting that dysarthria resulting from single small strokes in UMN pathways occur more frequently when the lesions are on the left, thus suggesting a greater influence of left descending motor fibers on speech.^{33,63,69} However, the greater percentage of cases with left side lesions, at least in the Hwang et al.³³ and Duffy and Folger studies, could simply reflect referral bias (e.g., many patients may have been referred primarily because of their aphasia) or differences in the distribution of left- and right-sided strokes that come to medical attention. In addition, at least one study has reported that dysarthria occurs more frequently or is more pronounced when the lesion is in the right than in the left hemisphere.²⁵ Thus although the possibility of left side dominance for UMN pathway control of speech is intriguing, and logical to the extent that the left hemisphere is dominant for language and motor speech programming, current evidence is insufficient to support such a conclusion. What is clear is that *UUMN dysarthria can result from lesions on either side of the brain.*

Severity

The literature does not permit a precise description of dysarthria severity. Dysarthria severity in Duffy and Folger's patients could not always be ascertained from their records, but it was probably mild in many cases. For example, the median severity ratings across the individual deviant speech characteristics that were detected were almost always mild or mild to moderate. These observations are in general agreement with indices of severity reported in other studies.^{28,33,36,65,66,68} However, moderate to severe dysarthria was reported for some cases in several of the just-cited studies (and that of Ropper⁶¹), and a moderate or severe reduction of intelligibility can sometimes occur.^{33,67}

It has been suggested that UUMN dysarthria is a transient problem.^{*7,18,19,78} Although clinical experi-

ence indicates that this frequently is the case, clinical experience and published data also indicate that the dysarthria can persist. For example, approximately 45% of Duffy and Folger's patients were evaluated more than 1 month after onset, and all subjects in some studies have been evaluated at least 3 months after onset.⁶⁵⁻⁶⁸

These observations suggest that UUMN dysarthria due to stroke is often mild and that significant recovery often takes place, but it sometimes can be markedly severe, chronic, or both. Why some patients can have a markedly severe or persisting UUMN dysarthria following stroke is not entirely clear, but Takahashi et al.,⁶³ observing that 41% of their patients with small unilateral strokes had asymptomatic strokes on the contralateral side, noted that when such "silent" strokes were present, dysarthria lasted longer and dysphagia occurred more frequently. Those data, plus clinical experience, suggest that *persistent severe dysarthria following a presumed unilateral stroke should raise suspicions about a lesion or lesions on the other side of the brain.* The effects of "silent" strokes (presumably in areas relevant to speech) may be unmasked by the occurrence of a new lesion elsewhere in the brain, making the effects of the new lesion more severe than predicted by the new lesion alone.

How frequently is UUMN dysarthria associated with aphasia, apraxia of speech, aprosodia, or nonaphasic cognitive deficits? Available data are not satisfactory because studies have not been specifically designed to examine those relationships, but some inferences are possible. Duffy and Folger reported that 24% of their patients with left hemisphere lesions had evidence of aphasia, although they had excluded patients whose aphasia, apraxia of speech, or nonaphasic cognitive problems precluded valid assessment of dysarthria. Among 48 patients with a primary diagnosis of UUMN dysarthria seen in the Mayo Clinic speech pathology practice from 1999-2001, 10% had a less severe aphasia, 2% had a less severe apraxia of speech, and 10% had less severe nonaphasic cognitive impairments. It thus appears that *when UUMN dysarthria is the primary communication deficit, aphasia, apraxia of speech, and nonaphasic communication deficits are not frequently present.* The prevalence of UUMN dysarthria when aphasia or nonaphasic cognitive deficits are more prominent than the dysarthria is unknown, but it is likely to be more prevalent than when UUMN dysarthria is the primary diagnosis, at least in patients with accompanying unilateral limb motor deficits. The occurrence of UUMN dysarthria in people with apraxia of speech is addressed in Chapter 11.

*Recovery of limb motor function following unilateral capsular stroke is generally good. Limb motor recovery from unilateral stroke is less adequate when multiple motor areas, their descending pathways, or thalamic circuitry are affected.^{8,24}

Patient Perceptions and Complaints

People with UUMN dysarthria are usually aware of their speech difficulty. They may minimize its effects, however, especially when intelligibility is preserved. When the etiology is stroke, by the time they are seen for formal speech assessment in the acute setting, they are sometimes more impressed with the improvement they have made than the degree of deficit that remains. When the dysarthria is more severe, they may express distress over its effect on intelligibility or efficiency of communication. They often describe their speech as *slurred*, *thick*, or *slow*. As with most other dysarthria types, patients tend to complain that *speech deteriorates under conditions of fatigue or psychologic stress*.*

Patients frequently complain of *drooling* or a *heavy feeling* on the affected side of the face or corner of the mouth and sometimes of heaviness or thickness in the tongue, especially when speaking. *Chewing and swallowing difficulty* are not unusual,²⁹ especially early after onset. Many complain of *drooling* from the affected side of the mouth. Although less frequent than in people with bilateral UMN lesions and spastic dysarthria, some patients complain of and exhibit *pseudobulbar crying or laughter*.⁵ Patients with clinically apparent aphasia or apraxia of speech often do not complain of their dysarthria because the language or motor programming deficits overwhelm its functional effects.

Clinical Findings

The lesions leading to UUMN dysarthria usually produce a constellation of physical signs and symptoms on the side of the body contralateral to the lesion (see Box 9-1). For example, 79% of Duffy and Folger's patients had hemiplegia or hemiparesis, and 20% had sensory deficits (Box 9-3). Language and other cognitive disturbances may be present and can and often do have a greater impact on spoken communication than the dysarthria. When aphasia results from left subcortical lesions, an accompanying dysarthria is frequently present.^{17,55}

*Brodal,¹⁰ an anatomist, and Aronson,⁵ a speech pathologist, made observations following their own right hemisphere strokes that provide sophisticated testimonial support for many common patient complaints. Brodal spoke of feelings of decreased force of innervation and problems with skilled movements, as if they were no longer automatic, requiring increased volitional energy to generate movement. Even 6 months after his stroke, he felt that his speech deteriorated under conditions of fatigue. Aronson spoke of his "emotional incontinence" and its similarities and differences from normal crying and laughter. He also described his sense of a spastic voice, with an accompanying feeling of overpressure in the thorax and abdomen, and its exacerbation by stress and fatigue.

Box 9-3

Primary oral mechanism, clinical neurologic findings, and confirmed or presumed lesion locus for 56 cases with a primary speech diagnosis of UUMN dysarthria.²¹ Percentage of cases is given in parentheses.

Oral Mechanism Findings

Unilateral lower facial weakness (82%)
Unilateral lingual weakness (52%)
Unilateral palatal weakness (5%)

Clinical Neurologic Findings

Hemiplegia/hemiparesis (79%)
Sensory deficits (20%)
Dysarthria and clumsy hand only (13%)
Dysarthria and bulbar weakness only (lower face or tongue) (5%)

Lesion Locus*

Internal capsule (34%)
Internal capsule or pons (4%)
Pericapsular (11%)
Lobar, cortical, and subcortical (nearly always including frontal lobe) (27%)
Lobar, cortical (always including frontal lobe) (7%)
Lobar, subcortical (always including frontal lobe) (7%)
Pericapsular, subcortical, and lobar (7%)
Brainstem (2%)
Thalamus and midbrain (2%)

UUMN, Unilateral upper motor neuron.

*Lobar—region affecting all or portions of a lobe in a cerebral hemisphere, divisible into cortical and subcortical subcategories when possible; pericapsular—region of the internal capsule plus adjacent structures projecting to or from the cerebral cortex, including the corona radiata.

Thirteen percent of Duffy and Folger's patients had dysarthria and a clumsy hand only, and 5% had dysarthria and face and tongue weakness as their only neurologic abnormality (see preceding discussion of related lacunar syndromes).

Nonspeech Oral Mechanism

Box 9-3 summarizes the primary oral mechanism findings in Duffy and Folger's patients. Unilateral central facial weakness was present in 82% of patients, a figure comparable to that reported in other studies.* This weakness is often apparent at rest and

*This is comparable to the 75% and 79% frequency of central facial weakness in large series of patients with unilateral stroke studied by Willoughby and Anderson⁷⁷ and Melo et al.,⁴⁶ respectively, and the 86% frequency of facial weakness in 14 dysarthric patients with single unilateral stroke prospectively studied by Hwang et al.³³ Melo et al.⁴⁶ reported that 93% of their dysarthric

during movement. If components of both the direct and indirect activation pathways are involved, weakness is apparent during voluntary and emotional facial movements. If the indirect pathway is relatively spared, emotional facial expression, such as smiling, may be relatively symmetric, reflecting the ability of the indirect pathway to drive emotional expression even when voluntary control is impaired. The converse can also occur. These disparities between voluntary and emotional facial expression are not unusual in UUMN dysarthria. In general, *unilateral central facial weakness seems to be a fairly good predictor of dysarthria in people with stroke.*

It is rare to find unilateral central lingual weakness in the absence of unilateral central facial weakness, and unilateral lingual weakness appears to be a good predictor of dysarthria and a fairly good predictor of dysphagia in people with acute stroke.* Unilateral lingual weakness was apparent in 52% of Duffy and Folger's patients.⁷ It is most easily detected as deviation of the tongue to the weak side on protrusion. It can also be detected on attempts to lateralize the tongue, or on lateral strength testing. Difficulty turning or pushing the tongue to one side is occasionally detectable when tongue deviation on protrusion is not apparent.

The reason lingual weakness is observed less frequently than facial weakness may reflect individual variability in the degree to which the twelfth cranial nerve receives contralateral versus bilateral UMN innervation. This variability may also explain some of the variability in deviant speech characteristics among people with UUMN dysarthria.⁴

patients had unilateral facial weakness. The frequency of unilateral facial weakness has been comparably high or higher in other (small N) prospective studies of patients with dysarthria and UUMN lesions that have made clinical observations of facial weakness.^{28,36}

*For example, Umapathi et al.⁷⁰ found a 29% incidence of tongue deviation in 300 patients with acute stroke that did not include the lower brainstem (i.e., the weakness was central). All patients with tongue deviation also had a central facial weakness on the same side. Dysphagia occurred in 43% and dysarthria in 90% of those with tongue deviation.

⁴This is somewhat less than the 64% frequency of lingual weakness in 14 patients prospectively studied by Hwang et al.³³ Unilateral lingual weakness was also present frequently in the six patients studied by Hartman and Abbs.²⁸

⁷The existence of bilateral UMN input to the hypoglossal nerve, but with variability in degree among individuals, receives support from recent studies using motor-evoked potential and magnetic stimulation methods.^{14,53} In addition, Umapathi et al.⁷⁰ noted that tongue deviation in people with acute unilateral stroke occurred more frequently in those with a history of prior stroke on the contralateral side, suggesting that bilateral involvement may be necessary to produce clinically obvious lingual weakness in some people.

The jaw is usually normal on clinical examination. For example, jaw weakness was not reported for any of the patients studied by Duffy and Folger or Hwang et al.³³ However, contralateral jaw weakness is occasionally apparent clinically,^{9,77} usually as reduced ability to clench or deviation to the weak side on opening. It has also been demonstrated electrophysiologically.^{15,28}

Velopharyngeal function has usually been assumed to be normal in UUMN lesions, but recent observations indicate this is not always the case. For example, Duffy and Folger observed palatal weakness (usually manifest as asymmetry at rest or during movement) in 5% and Hwang et al.³³ in 29% of their patients with UUMN strokes. Thompson and Murdoch⁶⁵ reported mildly impaired soft palate movement in three out of seven patients with a single unilateral stroke, and Kennedy and Murdoch³⁶ made similar observations in all four of their patients with unilateral subcortical stroke. Thus palatal asymmetry is clinically apparent more often than predicted by the presumed protective redundancy of bilateral UMN neuron supply to the vagus nerve.

Vocal fold weakness has also been assumed to be rare or nonexistent, but an assumption that bilateral UMN supply to the vagus nerve invariably spares laryngeal functions in UUMN lesions is questionable. For example, Bogousslasky and Regli⁹ reported hypotonia and decreased movement of the vocal fold contralateral to an internal capsule lesion in one patient. More convincingly, in a study of patients within 48 hours after a first-ever ischemic stroke, 11% of 35 patients with a lacunar stroke in the internal capsule, corona radiata, or paramedian pons, and 16% of 12 patients with cortical or large subcortical stroke had evidence of contralateral vocal fold paresis on flexible endoscopic examination. All patients with dysphonia had vocal fold weakness, and several also had palatal weakness. The weakness resolved in a majority of patients within 1 month. The authors concluded, "the long-held belief of the invariable bilateral innervation of the nucleus ambiguus may be incorrect."⁷⁵ It thus appears that *a minority but not insignificant percentage of patients with UUMN lesions can have contralateral vocal fold weakness.* These are important observations because they help to explain at least some of the deviant voice characteristics that can occur in UUMN dysarthria (described in the next section).

It has been well documented that dysphagia, as well as audible or silent aspiration, can occur with UUMN lesions.^{9,29,30,45} Similar to the dysarthria, these problems are often mild and recovery is good.

Motor impersistence may be apparent during oral mechanism examination, especially in patients with right hemisphere lesions. Motor impersistence is discussed in Chapter 3.

Deviant speech characteristics observed in 2 or more of 56 cases with a primary speech diagnosis of UUMN dysarthria (modified from Duffy and Folger²¹). Percentage of cases exhibiting each characteristic is given in parentheses. Confirmatory observations from other studies* are referenced, and observations from those studies not noted by Duffy and Folger and usually noted in only one or a few patients are listed under "Other characteristics."

Articulation (98%)

Imprecise consonants (95%)^{7,28,33,36,61,66-68}
 Irregular articulatory breakdowns (14%)³³
 Imprecise consonants and irregular articulatory breakdowns (11%)

Other characteristics: vowel distortions

Speech AMRs (91%)

Slow (72%)^{28,33,36,61}
 Imprecise (33%)
 Irregular (33%)^{28,33,38}
 Two or more of above (50%)

Phonation (57%)

Harshness (39%)^{7, 33,54,61,68}
 Reduced loudness (9%)^{7,33,61,68}
 Strained-harshness (5%)^{33,54,68}
 Wet hoarseness (4%)^{33,54,68}
 Breathiness (4%)^{33,54}

Monopitch (4%)^{33,68}

Monoloudness (4%)³³

"Unsteady" voice (4%)³³

Two or more of the above (13%)

Other characteristics: high pitch, low pitch, glottal fry, pitch breaks, increased loudness, loudness decay, reduced maximum vowel duration

Rate and Prosody (23%)

Slow rate (18%)^{7,28,33,68}
 Increased rate in segments (4%)
 Excess and equal stress (4%)
 Two or more of above (4%)

Other characteristics: variable rate, short phrases, reduced stress, excess loudness variations

Resonance (14%)

Hypernasality, nasal emission, or both (14%)^{7,33,36,65}
 Other characteristics: hyponasality

AMRs, Alternate motion rates; UUMN, unilateral upper motor neuron.

*Benke and Kertesz studied 35 patients with left hemisphere lesions and 35 patients with right hemisphere lesions and speech deficits (their observations of dysarthria are confounded by the presence of apraxia of speech and aphasia, and perhaps "aprosodia," in some patients). Hartman and Abbs²⁸ studied six patients with UUMN lesions and dysarthria. Hwang et al.³³ described the dysarthria in 14 patients with a single, unilateral stroke. Kennedy and Murdoch³⁶ described the speech and language disorders in four patients with left hemisphere subcortical stroke. Ropper⁶¹ described 10 patients with severe dysarthria resulting from right hemisphere lesions. Thompson and Murdoch⁶⁵ studied disorders of nasality in seven dysarthric patients (among others) with single, unilateral stroke. Thompson, Murdoch, and Stokes⁶⁶ studied lip function in eight dysarthric patients (among others) with single, unilateral stroke. Thompson, Murdoch, and Theodoros⁶⁸ studied the perceptual and physiologic attributes of four dysarthric patients (among five patients) with single, unilateral stroke.

Speech

The speech characteristics of UUMN dysarthria, as identified by Duffy and Folger, are summarized in Box 9-4. Confirmatory observations from studies that have provided more than vague descriptions of speech are also referenced. Some speech characteristics noted in those studies but not noted by Duffy and Folger are also listed.

The most pervasive deficit, present in 98% of the patients, was *imprecise consonants*. A smaller percentage of patients had *irregular articulatory breakdowns* in contextual speech, and approximately one third had *irregular alternate motion rates (AMRs)*. *Imprecise AMRs* were also apparent in one third of patients. When severity of these characteristics was noted, it was usually rated as mild, although some patients had moderate to marked imprecision. Imprecise articulation is often attributed to the unilateral lower facial and tongue weakness that is apparent in many patients.

Irregular articulatory breakdowns and irregular AMRs are usually associated with ataxic dysarthria.* The reasons for their presence in UUMN dysarthria are not entirely clear. They could reflect clumsiness that occurs as a normal byproduct of weakness⁴³ or because of imbalance of muscle forces in midline structures (jaw, tongue) or structures that move asynchronously (right and left face) when unilateral weakness is present. They could also reflect *ataxic-like incoordination* resulting from damage to cerebello-cortical fibers that intermingle with UMN fibers in white matter pathways.[†] Regardless of the reason

*It is interesting in this regard that Ropper's⁶¹ description of dysarthria in patients with right hemisphere lesions noted that "the overall pattern had some resemblance to the speech of an intoxicated individual."

†The internal capsule and white matter pathways between the thalamus and cortex, for example, contain cerebello-cortical and proprioceptive pathways that might, when damaged, contribute to

for such irregularities, these clinical observations indicate that *some patients with UUMN lesions and dysarthria can exhibit perceptual speech attributes that are ataxic.*

The second most prominent deviant feature was *slow AMRs*, which were usually mildly slowed. Such slowness was not as striking in contextual speech, where it was noted in only 18% of patients. The reasons for slowness are not entirely clear, but weakness, compensatory efforts to maintain precision and regularity, and spasticity are possible explanations. These clinical observations suggest that *some patients with UUMN lesions and dysarthria can exhibit perceptual speech attributes that are spastic.*

Somewhat surprisingly, 57% of patients had phonatory abnormalities, 39% with a mild to moderate dysphonia that was described as *harsh* or, less frequently, *strained to harsh*. Nine percent of the patients had *reduced loudness*, possibly also reflecting phonatory or respiratory-phonatory dysfunction. In addition, several characteristics noted in other studies (see Box 9-4) are suggestive of phonatory dysfunction. These findings are surprising in light of the common assumption that the vocal folds are spared from deficits because the tenth nerve receives bilateral UMN input.* The observation nonetheless suggests several possible explanations, none of which are mutually exclusive, including (1) unilateral vocal fold weakness; (2) spasticity[†]; (3) age-related dysphonia, because the elderly are the most

ataxic-like movements. Attig⁶ and Mori et al.⁵¹ have suggested that limb ataxia induced by capsular or corona radiata lesions may reflect disruption of cerebello-cortical or thalamo-cortical projections. Brodal¹⁰ stated: "it is extremely likely that the interruption of pathways other than the direct corticobulbar pathways is of importance . . . the cerebrocerebellar pathways are presumably important and involved in achieving smooth movements."

*Metter's⁴⁸ clinical description of UUMN dysarthria included breathiness, hypophonia, and sometimes reduced loudness. Reduced loudness, breathiness, and hoarseness have also been noted in studies of aphasia resulting from unilateral subcortical lesions (e.g., Alexander and LoVerme²; Damasio, Eslinger, and Adams¹⁶; Damasio et al.¹⁷; Metter et al.⁴⁹). The lesions in these studies usually included the basal ganglia, thalamus, and internal capsule or corona radiata.

Dysphonia may be an important marker of dysphagia for patients with UUMN lesions. For example, Horner and Massey²⁹ found dysphonia in 91% of their aspirating patients with UUMN lesions. Dysphonia was less frequently present in nonaspirating patients.

[†]The possibility of an undetected lesion or lesions in the contralateral hemisphere in some cases cannot be excluded. It has also been shown that cerebral blood flow can be diminished in the hemisphere contralateral to a unilateral stroke.²⁰ This evidence of "transhemispheric diaschisis" might explain the presence of a strained to harsh voice quality in some people with UUMN lesions, especially early after onset.

frequent victims of stroke and (4) other factors unrelated to the specific effects of UUMN lesions on speech (e.g., the general effects of illness or inactivity). Relative to the first possibility, vocal fold weakness in some patients with UUMN lesions has been documented (see discussion in the previous section). Similar mechanisms could also contribute to spasticity. It is reasonable to assume that the dysphonia in at least some people with UUMN dysarthria is neurologic in origin, and that many of the observed perceptual attributes can be linked to laryngeal hypo-function or hyperfunction. It thus appears that *some or many of the phonatory abnormalities in at least some people with UUMN dysarthria reflect weakness, spasticity, or both.*

Mild *hypernasality* or *nasal emission* was present in 11% of patients and has been observed in several other studies, again somewhat surprising in light of the presumed bilateral UMN supply to cranial nerve X. The reasons for its occurrence are probably similar to those offered in the previous paragraph for the occurrence of dysphonia.

Rate and prosodic abnormalities were present in 23% of patients, and most often were reflected in mildly *slowed rate*. Other prosodic abnormalities were uncommon, but they did encompass features tied to rate, loudness, pitch, and duration. The presence of irregular articulatory breakdowns almost certainly altered prosody in some patients.

Table 9-1 summarizes the primary clinical speech characteristics and common oral mechanism exami-

Table 9-1 Primary clinical speech and speech-related findings in UUMN dysarthria

Perceptual Articulation & prosody	Imprecise articulation Irregular articulatory breakdowns Slow rate Slow AMRs Imprecise AMRs Irregular AMRs
Phonation	Harshness Decreased loudness
Resonance Physical	Hypernasality (infrequent) Unilateral lower facial weakness Unilateral lingual weakness
Patient Complaints	Slurred speech/difficulty with pronunciation Drooping lower face/"heavy" lower face "Thick" or heavy tongue Drooling Dysphagia (relatively mild) May not complain of dysarthria if aphasia predominates

AMRs, Alternate motion rates; UUMN, unilateral upper motor neuron.

nation findings and patient complaints encountered in UUMN dysarthria.

Acoustic and Physiologic Findings

Acoustic and physiologic studies of patients with UUMN dysarthria are limited. In recent years, however, several such studies have been helpful in establishing the nature of speech subsystem impairment and, in combination with perceptual assessment, they have contributed to treatment planning.⁶⁸ The results of these studies are summarized in Table 9-2.

Table 9-2 Summary of acoustic and physiologic findings in studies of UUMN dysarthria*

Speech Component	Acoustic or Physiologic Observation
Respiratory	Reduced respiratory drive/weakness
Laryngeal	Unilateral vocal fold weakness Decreased: Glottal airflow Laryngeal airway resistance Rate of adduction/abduction f_0 variation Increased: Glottal airflow Laryngeal airway resistance f_0 variation Jitter Shimmer
Velopharyngeal Articulatory/Rate/ Prosody	Increased nasal airflow Reduced: Speech rate AMR rates Force of contralateral jaw movement Strength, endurance, & speed of lip & tongue movement Increased: Syllable & intersyllable gap duration & variability Variability of minimum & maximum waveform amplitude envelopes Acoustic energy during stop gap of voiceless consonants (spirantization) Irregular AMRs

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

Respiration

There is little information about respiration during speech. It is usually assumed that respiratory muscles are under bilateral UMN control and thus not significantly influenced by unilateral lesions. However, Przedborski et al.⁶⁰ established with electromyogram (EMG) recordings that most of their 25 patients with flaccid hemiplegia within 12 hours of a unilateral hemispheric stroke had abnormal neural respiratory drive of the contralateral parasternal intercostal muscles. It was not clear if their subjects were dysarthric. The data suggest that the parasternal intercostal muscles and diaphragm are predominantly under the control of contralateral corticospinal pathways in many people.

In a spirometric and kinematic study of two dysarthric patients (among others) with a single unilateral stroke, Thompson, Murdoch, and Theodoros⁶⁸ found that both patients had general respiratory impairment and one had impaired respiratory function for speech. Thus the limited data suggest that nonspeech respiratory functions can be affected in at least some patients with UUMN lesions and respiratory functions for speech can also be affected. Although clinical observation suggests that respiratory weakness is not usually of major consequence for speech, when unilateral respiratory weakness is present it could possibly contribute to the short phrases, reduced loudness, loudness decay, and reduced maximum vowel duration that are perceived in some patients.

Laryngeal Function

Endoscopic documentation of vocal fold weakness following unilateral stroke in some patients has already been discussed. Several acoustic and other instrumental measures also support a conclusion that laryngeal function can be abnormal. Similar to perceptual judgments, these findings by no means apply to all people with UUMN dysarthria; substantial variability within and among patient samples has been noted.³⁷

Acoustic analyses have documented both reduced and increased fundamental frequency (f_0) variation, as well as abnormalities on several amplitude (e.g., shimmer) and frequency (e.g., jitter) perturbation measures.^{11,37,39} Such abnormalities suggest laryngeal subsystem impairment. At least some of these abnormalities could reflect functional differences between the two vocal folds. For example, hoarseness, as reflected in the acoustic measure of jitter, could reflect asymmetric laryngeal hypotonia leading to differences between the vocal folds in overall tension or vibrating mass, with subsequent irregular vocal fold oscillation.¹

Aerodynamic and electroglottographic measures of laryngeal function during speech have documented abnormalities in a small number of patients with unilateral stroke and dysarthria.⁶⁸ The dynamics of abnormal laryngeal movement and airflow seem to vary among patients, sometimes suggesting weakness and sometimes suggesting hypertonicity. For example, Murdoch, Thompson, and Stokes³⁴ found that half of their 10 patients with dysarthria from UUMN stroke had instrumental findings suggestive of laryngeal hyperfunction, including elevated laryngeal airway resistance and subglottal air pressure, reduced laryngeal airflow, and a slower rate of adduction/abduction (the perceptual correlates of such findings would be harshness and strained voice quality). The remaining half had nearly opposite instrumental findings suggestive of laryngeal hypofunction (the perceptual correlates of such findings would be hoarseness, glottal fry, and breathiness). The authors concluded that differences between the hyperfunctional and hypofunctional subgroups could reflect differences in lesion site or compensation. For example, the instrumental evidence for hypofunction in some cases might have been attributable to increased stiffness/hypertonus preventing vocal fold approximation versus compensation for hyperadduction of the vocal folds. Although the explanation for these findings is not entirely clear, it does provide some support for perceptual voice attributes suggestive of weakness in some cases and spasticity in others.

Velopharyngeal Function

Observations that hypernasality is present only in a minority of people with UUMN dysarthria may explain why there has been only a single study of velopharyngeal function. Thompson and Murdoch⁶⁵ used nasal accelerometry and perceptual ratings to study velopharyngeal functions for speech in seven patients (among others) who had dysarthria from a single, unilateral stroke. Two of the seven patients were judged perceptually as hypernasal, and two of the seven had abnormally high nasal accelerometric indices, but for only one patient did both the perceptual and accelerometric indices identify velopharyngeal inadequacy/weakness. These findings agree with other perceptual observations of a relatively low frequency of perceived hypernasality in UUMN dysarthria. They also highlight the frequent incongruities between the results of perceptual and instrumental measures of velopharyngeal function for speech.

Articulation, Rate, and Prosody

Acoustic and physiologic measures of articulation and rate have established that at least some patients have weakness of the articulators contralateral to the side of lesion. Other patients have characteristics suggestive of spasticity or ataxia.

EMG and various other measures of strength, force, and endurance in people with UUMN lesions and dysarthria have demonstrated reduced magnitude of EMG signals and force of movement in the contralateral jaw, as well as reduced strength, endurance, and speed of lip and tongue movements.^{28,66-68} Although Thompson, Murdoch, and Stokes felt that the apparent lingual weakness in their patients could represent spasticity, they did not find evidence of lingual hypertonicity. They suggested that UMN weakness could account for reduced weakness and speed of lingual movement because reduced strength reduces the maximum shortening velocity of muscle fibers, with a subsequent reduction in speed of movement. Their results also suggest that fatigue contributes to lingual problems, consistent with frequent patient complaints that speech deteriorates with increased speaking time or general fatigue. It should be noted that their findings for lip and tongue strength, endurance, and speed did not correlate with perceptual measures of intelligibility, articulatory precision, or length of phonemes, leading them to suggest that measures of fine force control may be more relevant to perceptual measures. Nonetheless, these findings of reduced force and endurance are generally supportive of clinical observations of lower facial weakness in many patients and the presence of unilateral jaw weakness in some.

Acoustic measures have documented slow reading rate and slow and sometimes irregular AMRs,^{28,37,38} although Kent and Kent³⁷ note that the slow AMRs are generally not as slow as in ataxic dysarthria. Hartman and Abbs²⁸ found that slow AMRs were matched by perceptual ratings of AMRs, but that irregular AMRs were not perceived in their patients; irregular AMRs were perceived in some of Duffy and Folger's patients, however.

Kent et al.³⁸ examined AMRs in depth and found that syllable and intersyllable gap durations were lengthened and more variable than normal, that variability tended to increase as syllable duration increased, and that maximum and minimum waveform amplitude envelopes were more variable than normal. There was also evidence of acoustic energy during the stop gap of voiceless stops, a reflection of incomplete articulatory closure or spirantization, a correlate of perceived articulatory imprecision.

THE DISTINCTIVENESS OF UNILATERAL UPPER MOTOR NEURON DYSARTHRIA: CONCLUSIONS AND CLINICAL SUGGESTIONS

What features of UUMN dysarthria help distinguish it from other MSDs? If one attends to auditory-perceptual attributes alone, there do not appear to be any clear distinguishing features. Its most common deviant speech characteristics are not unique relative to other dysarthria types, and some are distinguishing features of other types. However, *if the speech characteristics are viewed in the context of their relative severity and other clinical findings, a cluster of distinguishing features emerges.* That is, UUMN dysarthria may best be distinguished from other dysarthria types by its common association with *unilateral central face and tongue weakness*; its predominant *stroke etiology*; its nearly-always-present but rarely-worse-than-moderate *articulatory imprecision*; and sometimes mild *irregular articulatory breakdowns, slow rate, and sometimes irregular AMRs, harsh, strained or hoarse-breathy dysphonia, and reduced loudness.* The gestalt impression from its auditory perceptual characteristics is therefore variable but most often suggestive of mild or moderate UMN weakness, sometimes spasticity or incoordination (ataxia), or sometimes various combinations of them. Indeed, studies of the dysarthria in people with UUMN lesions, while usually describing the dysarthria as UUMN in type, have sometimes labeled or at least noted its similarity to flaccid, spastic, ataxic, or mixed dysarthria.^{28,33,36,38,68}

It thus appears that a confident clinical diagnosis of UUMN dysarthria is probably best made on the basis of its auditory-perceptual features plus "the company it keeps," such as oral mechanism findings, other neurologic deficits, history, and neuroimaging results. To some extent this is how a confident clinical diagnosis of any dysarthria type is often made, but *reliance on confirmatory signs and other clinical clues is probably necessary more frequently for UUMN dysarthria than other dysarthria types.*

That a diagnosis of UUMN dysarthria is not always possible on the basis of speech features alone is not satisfying to the diagnostic purist, but the reasons this is the case *do* have a logical basis in what we know about the functions of commonly damaged structures. The reasons include the following:

1. Many patients with UUMN lesions have speech characteristics suggestive of weakness because damage to the direct activation pathways produces weakness that most often includes the face and tongue; sometimes the larynx; and less frequently the velopharynx, jaw, and respiration.
2. Some patients (perhaps fewer than those with weakness only) with UUMN lesions have speech characteristics suggestive of spasticity for several possible reasons, including (a) significant damage to the indirect activation pathway and its role in tone, reflexes, and posture; (b) individual variability in the degree to which unilateral UMN lesions have bilateral effects on speech cranial nerves* or the degree to which UMN lesions to speech cranial nerves are crossed and uncrossed; and (c) effects of altered blood flow to the contralateral hemisphere following stroke or the presence of undetected lesions in the contralateral hemisphere. If all of these explanations are valid, they suggest that speech features suggestive of spasticity sometimes result from the effects of a UUMN lesion alone on contralateral side muscles, the effects of the UUMN lesion alone on contralateral and ipsilateral side muscles, or the combined effects of the UUMN lesion plus influences (e.g., from lesions, altered blood flow/metabolism) of abnormalities on the "unaffected" side of the brain.
3. Some patients (perhaps fewer than those with weakness alone) with UUMN lesions have speech characteristics suggestive of ataxia possibly because of (a) damage to afferent cerebellocortical and proprioceptive tracts (e.g., in the internal capsule), or efferent frontopontocerebellar tracts,[†] resulting in uncoordinated speech movements, much like damage to such pathways can lead to ataxia in the limbs; or (b) clumsiness that occurs as a byproduct of weakness or imbalance of muscle forces in midline structures (jaw, tongue) or structures that move asynchronously

*In a transcranial magnetic stimulation (with CT and magnetic resonance imaging [MRI]) study of patients with dysarthria due to stroke in the lower motor cortex, corona radiata, and genu or posterior limb of the internal capsule, Urban et al.⁷² concluded that the effect of stimulation was absent or delayed bilaterally in 17 of their 18 patients.

†Ataxia has been described with lesions in the frontal lobes, presumably attributable to interruption of the frontopontocerebellar tracts. Dysarthria has been reported with such lesions, but its characteristics have not been described well.⁶⁴ Moulin et al.,⁵² in a study of 100 patients with lacunar infarcts and ataxic hemiparesis and dysmetria, found facial weakness in 60% of patients with pontine lesions, 50% with corona radiata lesions, and 40% with thalamic lesions. Dysarthria was also present with lesions in the internal capsule, pons, thalamus, corona radiata, lentiform nucleus, and cerebellum. Unfortunately, the characteristics of the dysarthria were not described, but the association of dysarthria with apparent ataxia and dysmetria in the limbs suggests that ataxic speech features could be associated with such lesions.

(right and left face) when unilateral weakness is present.

How might a clinician discuss a diagnosis of UUMN dysarthria, knowing that its perceptual characteristics may reflect weakness, spasticity, incoordination, or various combinations of them? When confident that the speech features, confirmatory signs, and clinical context are compatible with the diagnosis, the following may help frame diagnostic statements:

1. When the speech characteristics are all consistent with what can be explained by weakness, using the designation "*UUMN dysarthria with speech features consistent with (right or left side) UMN weakness,*" or, more concisely, "*UUMN dysarthria, flaccid variant,*" seems to convey information about general lesion locus and presumed pathophysiology.
2. When speech characteristics are suggestive of spasticity, using the designation "*UUMN dysarthria with predominant speech features suggestive of hypertonicity,*" or, more concisely, "*UUMN dysarthria, spastic variant,*" conveys information about general lesion locus (and implies that the lesion need not be bilateral, as is usually assumed for spastic dysarthria) and presumed pathophysiology.
3. When speech characteristics are suggestive of ataxia, using the designation "*UUMN dysarthria with predominant speech features suggestive of incoordination,*" or, more concisely, "*UUMN dysarthria, ataxic variant,*" conveys information about general

lesion locus (and implies that the lesion need not be in the cerebellum, as is often assumed for ataxic dysarthria) and presumed pathophysiology.

4. When features of two or more characteristics are present, the designations can be combined (e.g., "*UUMN dysarthria with speech features consistent with weakness and incoordination*" or "*UUMN dysarthria, mixed [specify] variant*").

In the author's experience, the most frequent designations are likely to be the first or fourth.

It is not uncommon that confidence about the diagnosis of UUMN is low. This is most often the case when the dysarthria is relatively severe and contains features suggestive of spasticity, ataxia, or both that are marked in severity, even when confirmatory signs and other clinical evidence suggest only a UUMN lesion. It is best under these circumstances to highlight the ambiguity with statements such as "although the patient's dysarthria could be explained by a UUMN lesion, the degree of spastic speech characteristics in this case is unusual for unilateral damage," or "although speech characteristics suggestive of ataxia can be present with UUMN lesions, the degree of ataxic characteristics in this case is more commonly encountered with cerebellar lesions." These qualified diagnostic conclusions are most important when lesion site is uncertain or when there are few other lateralizing signs. The ability to draw these confident or qualified conclusions probably requires considerable clinical experience.

Cases

Case 9-1

A 55-year-old right-handed man was admitted to the hospital with a 4-day history of progressive right hemiparesis and dysarthria. Neurologic evaluation revealed dysarthria, right hemiparesis, and mild sensory loss in the right face and upper limb. CT scan showed evidence of an infarct in the posterior limb of the left internal capsule.

Speech evaluation 2½ weeks after onset revealed a right central facial weakness. Speech was characterized by imprecise articulation (2.3), harsh voice quality (0,1), and slow speech AMRs (-1.2). Intelligibility was moderately reduced. There was no evidence of aphasia or any other cognitive disturbance.

The clinician concluded the patient had a UUMN dysarthria. The patient was seen for only one session of speech therapy before his discharge from the hospital. He did not return for follow-up.

Commentary. (1) UUMN dysarthria commonly affects articulation and sometimes voice quality and frequently seems predominantly explained by CNS weakness. (2) It can be associated with moderate reductions of speech intelligibility. (3) The internal capsule is a common site for lesions that cause UUMN dysarthria. Isolated internal capsule lesions in the dominant hemisphere are rarely, if ever, associated with aphasic language impairment or other cognitive disturbances.

Case 9-2

A 70-year-old right-handed man was hospitalized because of a sudden onset of inability to express himself, a right facial droop, and weakness in his right upper extremity. History and clinical evaluation were consistent with a middle cerebral artery stroke. CT scan identified an area of decreased attenuation in the left frontal lobe consistent with recent infarction.

Speech and language evaluation 3 days after onset revealed a mild to moderate aphasia with deficits apparent in verbal formulation and comprehension, as well as reading and writing. Verbal communication, however, was functional. Right facial and tongue weakness was apparent. There was no evidence of apraxia of speech. The patient's speech was characterized by imprecise

articulation (1), reduced loudness (0,1), and hoarseness (1,2). Speech intelligibility was normal. The patient began speech-language therapy. Within 1 week his dysarthria had resolved, and the only evidence of aphasia was infrequent word-finding difficulties.

Commentary. (1) UUMN dysarthria associated with dominant hemisphere lesions is frequently associated with aphasia. In this case, the dysarthria and aphasia were approximately equal in severity at onset. (2) UUMN dysarthria frequently resolves rapidly and completely (in this case, within 1 week after onset). (3) The frontal lobe is most often implicated when UUMN dysarthria is the result of a cortical lesion.

Case 9-3

A 73-year-old right-handed man was admitted to the hospital with a 1-day history of slurred speech and difficulty using his right hand. Neurologic examination demonstrated only dysarthria and mild right upper extremity weakness and clumsiness. The neurologist felt that the patient's presentation was consistent with a "dysarthria-clumsy hand syndrome." Subsequent CT scan demonstrated a lacunar infarct in the area of the left lateral basal ganglia and centrum semiovale.

Speech examination the following day identified mild right lower central facial weakness and mild deviation of the tongue to the right on protrusion. Speech was characterized by breathy-hoarse voice quality (0,1), reduced loudness (-1,2), irregular articulatory breakdowns (2),

monopitch and loudness (1,2), and equivocal acceleration of speech rate. Speech AMRs were normal in rate but imprecise (1,2) and irregular (2). Speech intelligibility was moderately reduced. There was no evidence of aphasia or apraxia of speech.

The clinician concluded that the patient had "a moderately severe UUMN dysarthria." Speech therapy was recommended, and improvement in speech was noted before discharge several days later.

Commentary. (1) UUMN dysarthria can be the only or among only a few signs of unilateral neurologic disease. (2) UUMN dysarthria is often associated with subcortical lesions.

Case 9-4

A 57-year-old man was seen in the clinic for evaluation of residual symptoms stemming from a stroke approximately 3 years earlier. Neurologic examination revealed dysarthria and left hemiparesis. The neurologist concluded that the patient had a "pure motor hemiparesis, almost like a capsular infarct."

Speech evaluation revealed mild left lower face and tongue weakness. Imprecise articulation and imprecise AMRs (1) characterized speech. Articulatory precision improved noticeably with a moderate slowing of speech rate. Phonation and resonance were normal.

The clinician concluded that the patient demonstrated a "mild UUMN dysarthria." Some time was spent demonstrating to the patient the advantages of slowing his speech rate. He appreciated the benefits of this speaking strategy but did not believe speech therapy was necessary. The clinician agreed.

Commentary. (1) UUMN dysarthria can result from lesions on the right or left side of the brain. (2) It can persist long after the spontaneous recovery period. (3) Persistent UUMN dysarthria is often mild, and clinicians and patients frequently feel that therapy is unnecessary.

Case 9-5

An 81-year-old right-handed man was admitted to the hospital with a 2-day history of "garbled speech" and left facial weakness. Neurologic examination revealed left facial weakness and mild left upper extremity weakness. A CT scan 1 week after onset revealed a lesion in the right posterior frontal lobe that was consistent with a recent stroke. A complete neurologic workup led to a right carotid endarterectomy 2 weeks later, without any deterioration in neurologic status.

Speech evaluation 12 days after surgery demonstrated a left central facial weakness (2,3) and deviation of the tongue to the left on protrusion. The patient wore loose-fitting dentures. Speech was characterized by hoarse-rough voice quality (2), imprecise articulation (1), occasional acceleration of speech rate (1,2), and slowed and imprecise AMRs (0,1). Speech intelligibility was, at worst, mildly reduced. The patient believed that his

speech was quite adequate and did not want speech therapy. His wife and daughter felt that his speech was almost back to the level before the stroke, and they had only occasional mild difficulty understanding him. Although the clinician felt that therapy might be beneficial, the patient chose not to pursue it.

Commentary. (1) UUMN dysarthria can affect voice quality as well as articulation. (2) The effects of dysarthria on intelligibility can be exacerbated by non-neurologic factors, such as loose-fitting dentures. Problems with dentures frequently become more pronounced after a stroke that affects oromotor function, and they can present additional barriers to adequate articulation. (3) Recommendations for speech therapy must consider patient needs and wishes as well as the clinician's judgment about the possible benefits of therapy.

Case 9-6

A 66-year-old right-handed man with a 20-year history of hypertension was admitted to the hospital after the sudden onset of right hemiplegia, right facial weakness, and inability to speak. A CT scan 3 weeks after onset showed an area of low attenuation in the left centrum semiovale that extended down into the adjacent lentiform nucleus, consistent with stroke.

Language examination 3 weeks after onset was normal. The patient had a right lower facial weakness. Tongue protrusion was midline, but lateral movements were mildly slowed. Voice quality was harsh-breathy (1). Articulation was imprecise (1). In addition, the patient occasionally repeated the first phoneme of a word and was mildly hesitant, but there were no obvious trial-and-error misarticulations or clear-cut substitutions of sounds.

The clinician concluded that the patient had a "flaccid UUMN dysarthria." The possibility of an accompanying apraxia of speech was considered, but evidence for it was considered equivocal. The patient received four

sessions of speech therapy that focused on improving articulation through increased self-monitoring and slowing of rate. He improved and asked that therapy be terminated so that he could devote more time to physical therapy.

Commentary. (1) UUMN dysarthria is often associated with subcortical lesions. (2) When the lesion is in the presumed dominant hemisphere, questions about the presence of aphasia and apraxia of speech often arise. There was no evidence of aphasia in this patient, but he did exhibit a few speech characteristics that were suggestive of apraxia of speech. Although it was concluded that apraxia of speech was not present, this case illustrates the difficulty that may be encountered in distinguishing between dysarthria and apraxia of speech. (3) Improvement in speech is usually noted in patients with UUMN dysarthria. It is not unusual for patients to terminate therapy on their own once speech becomes intelligible and sufficiently efficient. Evidence of dysarthria may persist, however.

SUMMARY

1. UUMN dysarthria results from unilateral damage to UMN pathways. It occurs at a frequency comparable to that of other major single dysarthria types. It is most often apparent in articulation, phonation, and prosody. Its deviant characteristics usually reflect the effects of weakness on speech, but sometimes spasticity and incoordination are implicated.
2. The anatomic designation of this dysarthria type is based on the locus of lesions associated with it. The fact that its clinical characteristics and pathophysiologic underpinnings are variable and not well understood precludes a single

pathophysiologic designation for the disorder at this time. It is possible, however, that its deviant characteristics reflect the effects of weakness and sometimes spasticity or incoordination on speech movements.

3. Stroke is by far the most common cause of UUMN dysarthria. Lesions on either side of the brain anywhere along the UMN pathways from the cortex to the brainstem can cause it, but lesions are probably most often in the cerebral hemispheres, usually in the posterior frontal lobe, internal capsule, or related white matter pathways.
4. Lower facial weakness and hemiparesis often accompany UUMN dysarthria. Contralateral lingual weakness is also common, and drooling and dysphagia may be present.
5. UUMN dysarthria is usually only mild to moderate in severity, and recovery from it is often quite good. However, it sometimes is marked in severity and can persist as a significant deficit beyond the period of spontaneous recovery.
6. The most common deviant speech characteristics are imprecise articulation and, less frequently, irregular articulatory breakdowns; slow rate; slow and sometimes irregular AMRs; harsh, strained, or hoarse-breathy dysphonia; and reduced loudness. Hypernasality occurs infrequently.
7. Physiologic studies have documented, with varying frequency, respiratory weakness, vocal fold weakness or hyperfunction, or both, with associated acoustic and aerodynamic abnormalities; velopharyngeal inadequacy; and reduced strength, endurance, or speed of jaw, lip, and tongue movements. Acoustic analyses have documented reduced speech rate and slow and irregular speech AMRs.
8. UUMN dysarthria can be the only or among the first and most prominent signs of neurologic disease. Its recognition and correlation with UUMN dysfunction can aid the localization and diagnosis of neurologic disease. Its specific diagnosis may require reliance on confirmatory clinical signs and clinical context more frequently than other dysarthria types because of the varying degree to which weakness and apparent spasticity or incoordination can be associated with it. Improved understanding of this dysarthria may assist efforts to study other speech and communication deficits that may be associated with unilateral neurologic disease, disorders whose manifestations may be masked or confounded by UUMN dysarthria.

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