

Hypokinetic Dysarthria

*"I became conscious that my voice tended to sound flat and lacking in expression My voice had become softer, and I was unable to enunciate certain words clearly . . . if I went on talking, my voice would fail, and I could do no more than whisper"*¹⁹

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CHAPTER OUTLINE

- I. Anatomy and basic functions of the basal ganglia control circuit
- II. Clinical characteristics of basal ganglia control circuit disorders associated with hypokinetic dysarthria
- III. Etiologies
 - A. Degenerative diseases
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 - C. Toxic-metabolic conditions
 - D. Trauma
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 - A. Distribution of etiologies, lesions, and severity in clinical practice
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Hypokinetic dysarthria is a perceptually distinctive motor speech disorder (MSD) associated with basal ganglia control circuit pathology. 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 *voice*, *articulation*, and *prosody*. The disorder reflects the effects of rigidity, reduced force and range of movement, and slow individual but sometimes fast repetitive movements on speech. Decreased range of movement is a significant contributor to the disorder, hence its designation as *hypokinetic* dysarthria.

Hypokinetic 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 8.2% of all dysarthrias and 7.6% of all MSDs (see Figure 1-3).

The identification of hypokinetic dysarthria can aid neurologic diagnosis and localization. Its presence is strongly associated with basal ganglia pathology and is often tied to a depletion or relative insufficiency of the neurotransmitter *dopamine*. *Parkinson's disease (PD)* is the prototypic, but not the only, disease associated with hypokinetic dysarthria.

The clinical features of hypokinetic dysarthria reflect the effects on speech of aberrations in the maintenance of proper background tone and supportive neuromuscular activity on which the quick, discrete, phasic movements of speech are superimposed. The disorder permits inferences about the role of the basal ganglia control circuit in providing an adequate neuromuscular environment for voluntary motor activity. Hypokinetic speech often gives the impression that its underlying movements are "all there" but have been attenuated in range or amplitude and restricted in their flexibility and speed.

■ ANATOMY AND BASIC FUNCTIONS OF THE BASAL GANGLIA CONTROL CIRCUIT

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

The basal ganglia are located deep within the cerebral hemispheres. They include the *striatum*,

composed of the *caudate nucleus* and *putamen*, and the *lentiform nucleus*, composed of the *putamen* and *globus pallidus*. The *substantia nigra* and *subthalamic nuclei* are anatomically and functionally closely related to the basal ganglia. Basal ganglia activities are strongly associated with the actions of the indirect activation pathway or extrapyramidal system.

The interconnections that make up the basal ganglia control circuit are complex. The basic components include: (1) cortical, thalamic, and substantia nigra input to the striatum, with crucial cortical input coming from the frontal lobe premotor cortex; (2) striatum input to the substantia nigra and globus pallidus; (3) globus pallidus input to the thalamus, subthalamic nucleus, red nucleus, and reticular formation in the brainstem. These connections form loops in which information is returned to its origin. For example, basal ganglia input to the thalamus is relayed to the cortex and returned to its origin in the basal ganglia; striatum input to the substantia nigra returns to the striatum; globus pallidus input to the subthalamic nucleus is returned to the globus pallidus. The major efferent pathways of the basal ganglia originate in the globus pallidus.

The functions of the circuit are to *regulate muscle tone; regulate movements that support goal-directed activities* (e.g., the arm swing during walking); *control postural adjustments during skilled movements* (e.g., stabilize the shoulder during writing); *adjust movements to the environment* (e.g., speaking with restricted jaw movement); and *assist in the learning, selection, and initiation of movements*. Damage to the circuit either reduces movement or results in a failure to inhibit involuntary movement. In hypokinetic dysarthria, speech deficits are mostly associated with reductions of movement.

The primary influence of the basal ganglia control circuit on speech is through its connections with motor areas of the cerebral cortex.* Its influence on the cortex appears inhibitory; that is, it damps or modulates cortical output that would otherwise be in excess of that required to accomplish movement goals. The circuit helps to maintain a stable musculoskeletal environment in which discrete movements can occur. Excessive or insufficient damping of cortical output results in movement disorders.

Imbalances among neurotransmitters are responsible for many motor problems associated with basal ganglia control circuit malfunction. The actions of

dopamine are of particular importance to understanding PD and its associated hypokinetic dysarthria. When substantia nigra neurons are destroyed, the dopamine supply to the striatum is reduced and its role in the circuit is diminished. The functional results of this are discussed in the next section.

■ CLINICAL CHARACTERISTICS OF BASAL GANGLIA CONTROL CIRCUIT DISORDERS ASSOCIATED WITH HYPOKINETIC DYSPHARTHRIA

Parkinsonism serves as a model for discussing the clinical characteristics of basal ganglia control circuit disorders that result in hypokinesia. PD and parkinsonism are by far the most common causes of hypokinetic dysarthria (Box 7-1). The pathophysiology of PD and parkinsonism are discussed in the next section. At this point, only nonmotor characteristics of parkinsonism are addressed.

The nonspeech motor characteristics of parkinsonism are summarized in Table 7-1. The classic signs are *tremor at rest*, *rigidity*, *bradykinesia*, and *a loss of postural reflexes*.

The *tremor* in parkinsonism is a *static* or *resting tremor* that occurs at a rate of approximately 3 to 8 Hz.⁷ It is most apparent when the body part is relaxed, and it tends to decrease during voluntary movement. It is often apparent in the limbs and head but may also be evident in the jaw, lips, and tongue. A *pill-rolling* movement between the thumb and forefinger may be present.

Slowness of movement and a feeling of stiffness or tightness characterize *rigidity*. It is apparent during passive stretch on muscles and probably contributes to paucity of movement. It may be the result of excessive central nervous system (CNS) influence on alpha motor neurons, which occurs because excessive cortical motor output is not properly inhibited by the basal ganglia.⁴ Unlike spasticity, in which resistance to movement is usually greatest at the beginning of stretch and is biased in direction, rigidity is associated with resistance in all directions and through the full range of movement. *Cogwheel rigidity*, in which resistance of the limbs to passive stretch has a jerky character, is common.

Posture tends to be characterized by involuntary flexion of the head, trunk, and arms. Because postural reflexes are impaired, the patient may be unable to make adjustments to tilting or falling and have difficulty turning in bed or moving from a sitting to standing position.

Bradykinesia is a problem in the speed with which muscles can be activated. It is characterized by delays or false starts at the beginning of movement and slowness of movement once begun. Movement may also be difficult to stop, and repetitive

*Sensory motor integration is a component of the operations of the basal ganglia control circuit. Preliminary evidence indicates that these operations include modulation of auditory feedback for the control of vocalization.⁷¹ This may be relevant to clinical observations that speakers with hypokinetic dysarthria and reduced loudness are poorly calibrated in their judgments of the adequacy of their vocal loudness (i.e., they overestimate it).

box
7-1

Etiologies for 167 quasirandomly selected cases with a primary speech pathology diagnosis of hypokinetic dysarthria at the Mayo Clinic from 1969-1990 and from 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 (78%)

Parkinson's disease (36%)
 Parkinsonism (22%)
 PSP (7%)
 Unspecified degenerative CNS disease (5%)
 Shy-Drager syndrome (2%)
 Multiple systems atrophy (4%)
 Lewy body disease (1%)
 Parkinsonism + amyotrophic lateral sclerosis (1%)

Vascular (9%)

Nonhemorrhagic stroke (5%)
 Nonparenchymal bleeds (subarachnoid hemorrhage, subdural hematoma) (1%)
 Ruptured aneurysm (1%)
 Small vessel disease (1%)
 Anoxia (cardiac arrest) (1%)

Undetermined (4%)

Extrapyramidal disorder (1%)
 PD vs. PSP (2%)
 PSP vs. stroke (1%)
 Multiple (4%)
 Parkinsonism + multiple sclerosis or stroke; Alzheimer's disease or dementia + stroke; PD + subdural hematoma or stroke

Toxic or Metabolic (2%)

Drug related (phenothiazines, unspecified) (1%)
 Carbon monoxide (1%)

Traumatic (1%)

Closed head injury

Infectious (1%)

Postencephalitic parkinsonism; encephalitis

Other (1%)

Radiation necrosis; basal ganglia calcification

CNS, Central nervous system; PD, Parkinson's disease; PSP, progressive supranuclear palsy.

movements may be decreased in amplitude and speed. In spite of a desire to move, there may be intermittent "freezing" or immobility (akinesia). Bradykinesia or akinesia frequently are "the key feature of parkinsonian off-states, periods when brain levels of dopamine are inadequate."³¹

The terms *hypokinesia* (reduced movement) and *akinesia* (absence of movement) are often used

table 7-1 Common nonspeech clinical signs of parkinsonism

Primary Deficits	Examples
Resting Tremor	Head Limb Pill-rolling Jaw, lip, tongue
Rigidity	Resistance to passive stretch in all directions through full range of movement Paucity of movement
Bradykinesia or Hypokinesia	Slow initiation & speed of movements "Freezing"
Akinesia	Festinating gait Reduced: Arm swing during walking Limb gestures during speech Eye blinking Head movement accompanying vertical & horizontal eye movement Frequency of swallowing
Postural Abnormalities	Micrographia Masked facies Stooped posture (flexed head & trunk) Poor adjustment to tilting or falling Difficulty turning in bed Difficulty going from sitting to standing

interchangeably with bradykinesia. In addition to slowness, however, they also refer to underactivity or reduced range of movement, reduced use of an affected body part, and a reduction of the automatic, habitual movements that accompany natural movement. This probably cannot be attributed solely to weakness because *strength is thought to be relatively unimpaired in parkinsonism*. However, Corcos et al.²⁴ have shown that withdrawal of antiparkinsonian medications does produce muscle weakness that is attributable to reduced agonist muscle activation and, in some patients, increased antagonist muscle activation. The authors suggested that patients with PD might benefit from exercise programs designed to improve strength and power, a notion embraced by some behavioral programs for treating hypokinetic dysarthria (see Chapter 17).

The underactivity of hypokinesia is reflected in a masked or expressionless and unblinking facial expression (*masked facies*),* a classic feature of

*Studies have documented that the intensity of spontaneous emotional expression is reduced in PD, but that posed facial expression and emotional feelings generally are not.^{15, 109}

parkinsonism (Figure 7-1). Similarly, the normal arm swing during walking and the limb gestures that automatically accompany speech may be reduced. Writing may be *micrographic* (small). Walking may be initiated slowly and then characterized by short, rapid shuffling steps, a phenomenon known as *festination*.

It is possible that a number of problems associated with hypokinesia are influenced by deficits in sensory function. Kent et al.⁶⁷ reviewed the growing evidence of impaired sensory function in PD, such as difficulties estimating movement displacements on the basis of kinesthetic information, and poor temporal discrimination of auditory, tactile, and visual stimuli. Summarizing the conclusions of Demirici et al.,²⁹ they stated: "the reduced kinesthesia, combined with reduced motor output and the likelihood of reduced corollary discharges, could mean that the sensorimotor apparatus is 'set' smaller in PD." The possible role of sensory disturbances in hypokinetic dysarthria is addressed in some speech therapy programs for the disorder (see Chapter 17).

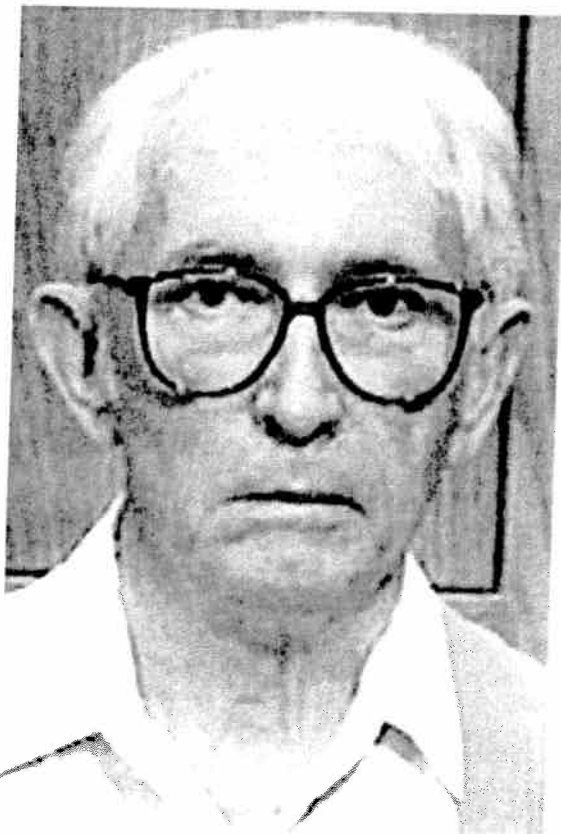


FIGURE 7-1 Masked facial expression associated with Parkinson's disease and hypokinetic dysarthria.

■ ETIOLOGIES

Any process that can damage the basal ganglia control circuit can cause hypokinetic dysarthria. These include degenerative, vascular, traumatic, inflammatory, neoplastic, toxic, and metabolic diseases. The exact distribution of causes of hypokinetic dysarthria is unknown, but degenerative diseases are undoubtedly the most frequent known causes (see Figure 7-1 and Box 7-1).

PD is almost certainly the most frequent cause of hypokinetic dysarthria. Also, in the absence of other influences (e.g., medication effects), hypokinetic dysarthria is *the* dysarthria of PD. This sometimes leads to the use of such terms as "the dysarthria of PD" or "parkinsonian dysarthria." The term *hypokinetic* dysarthria is preferable, however, because conditions other than PD can be associated with it. In addition, patients with PD may have more than hypokinetic dysarthria. For example, medication used to treat PD sometimes causes involuntary movements that result in hyperkinetic dysarthria. Also, some patients with an initial diagnosis of PD ultimately receive a different diagnosis, one indicating the presence of more than basal ganglia dysfunction (e.g., progressive supranuclear palsy [PSP]).

Some of the common neurologic conditions associated with hypokinetic dysarthria with noticeably greater frequency than other dysarthria types are discussed later. Other diseases that can produce it but are more frequently associated with other dysarthria types, especially mixed dysarthrias, are discussed in the chapters that address those specific dysarthria categories.

Degenerative Diseases

PD is a common, slowly progressive idiopathic neurologic disease that affects approximately 50 people per 100,000 older than the age of 50. It usually begins in mid-to-later life; survival from symptom onset is approximately 9 years. Its occurrence can be sporadic, but nearly one third of people with two or more affected first-degree relatives are likely to acquire the disease; unidentified environmental toxins such as herbicides and pesticides are other possible causes.⁸⁸ Although dysarthria usually does not emerge for several years after the onset of other signs of PD, Müller et al.⁹⁵ found that it did become evident in approximately 90% of autopsy-confirmed cases during the course of the disease, nearly always preceding the onset of dysphagia, which occurred in approximately 40% of cases.

PD may affect more than motor function. Ten to thirty percent of affected people eventually develop significant dementia, and depression occurs in 40%

to 60%.³¹ Sometimes akinesia and bradykinesia are mistaken for depression, and the diagnosis of PD is missed.

The pathologic changes of PD most often involve nerve cell loss in the substantia nigra and locus ceruleus, as well as decreased dopamine content in the striatum. The imbalance between dopamine and acetylcholine caused by the depletion of dopamine in the striatum is thought to be responsible for clinical signs of the disease. PD tends to be responsive to *dopaminergic drugs* (known as *dopamine agonists*), but such drugs are not curative. *Carbidopa-levodopa (Sinemet)* is the cornerstone of treatment of PD. It works by increasing dopamine levels in the striatum: the carbidopa component prevents destruction of levodopa in the bloodstream and minimizes side effects. Direct-acting dopamine agonists, including *bromocriptine (Parlodel)*, *pergolide (Permax)*, *pramipexole (Mirapex)*, and *ropinirole (Requip)* are sometimes used in place of Sinemet. Some newly diagnosed, mildly impaired PD patients are treated with *amantadine (Symmetrel)*, or *selegiline (Eldepryl)*, *trihexyphenidyl (Artane)*, or *benztropine (Cogentin)*, but those drugs are less potent than Sinemet or direct-acting dopamine agonists.⁷ *Anticholinergic drugs* may be used for resting tremor.

Unfortunately, the medications used to treat PD have side effects that include dystonia and dyskinesias, confusion, and *on-off effects*. On-off effects are symptom fluctuations that occur during a dosage cycle; they may include shifts from worsening of parkinsonian symptoms to the development of dystonia or dyskinesias at the beginning, peak, or end of a dosage cycle. Worsening of hypokinetic dysarthria or the emergence of hyperkinetic dysarthria may occur as reflections of on-off effects. Thus the dysarthria encountered in people with PD can represent the effects of the disease itself, as well as the effects of medications used to treat it. The design of clinical and laboratory investigations of hypokinetic dysarthria in PD must take into account medication effects, and they need to control for the time at which observations are made during the dosage cycle, especially in longitudinal investigations.

The term PD is usually reserved for parkinsonism of unknown cause that is responsive to levodopa treatment. In contrast, *parkinsonism* is a more generic term that refers to the clinical signs of the disease regardless of etiology. Parkinsonism is often used to refer to conditions with etiologies and pathophysiology that are different from PD (e.g., vascular, Alzheimer's disease [AD], drug induced), or when symptoms are not responsive to medications that are effective in managing PD.

Degenerative neurologic diseases that include but go beyond signs and symptoms of parkinsonism are

often called *parkinsonism-plus syndromes* or *atypical parkinsonian disorders*. They include *multiple system atrophy* (with subtypes of *Shy-Drager syndrome*, *olivopontocerebellar atrophy*, and *nigrostriatal degeneration*), *PSP*, and *corticobasal degeneration*. Although hypokinetic dysarthria can be the only MSD encountered in each of these disorders, it is generally more common for a mixed dysarthria to be associated with them. Because of this, further discussion of the parkinsonism-plus conditions is deferred until Chapter 10, which addresses mixed dysarthrias.

Some primary dementing illnesses can be associated with parkinsonian signs. Although AD is marked clinically by its progressive effects on memory, thought, language, and personality, parkinsonian signs have been noted in 35% to 50% of patients with AD.^{80,89} *Diffuse Lewy body disease*, characterized early in its course by relatively mild parkinsonian and more severe cognitive symptoms (e.g., dementia, visual hallucinations, paranoid delusions), straddles the boundary between AD and PD. In it, Lewy bodies, a pathologic hallmark of PD, are found not only in the substantia nigra, as in PD, but also in the cerebral cortex.²² Finally, *Pick's disease*, a dementing illness with primary effects on the frontal and temporal lobes, although not usually associated with signs of parkinsonism late in its course,⁹⁰ Thus the development of hypokinetic dysarthria is possible in some degenerative diseases whose primary manifestations are in the cognitive domain.

Vascular Conditions

Although strokes usually do not cause parkinsonism or hypokinetic dysarthria, diffuse frontal lobe white matter lesions and basal ganglia lesions* occasionally are associated with parkinsonian signs; gait difficulty and postural instability, dementia, corticospinal signs, and pseudobulbar affect are more prevalent than in PD.^{121,125,126} This entity is frequently called *vascular parkinsonism*. It is not generally responsive to levodopa therapy.¹²⁵

Cerebral hypoxia, including that induced by carbon monoxide poisoning, can also produce parkinsonian syndromes.

Toxic-Metabolic Conditions

Antipsychotic (neuroleptic) and *antiemetic[†] medications*, known as *dopamine antagonist drugs*, can

*Dysarthria with hypokinetic features has even been reported in bilateral thalamic strokes.³

[†]Drugs to relieve nausea or prevent or arrest vomiting.

have prominent blocking effects on dopamine receptors*: such drugs lead to parkinsonism in an estimated 10% to 20% of patients treated with them.⁹² Parkinsonism can also be caused by drugs that interfere with the brain's ability to store dopamine (*dopamine depletors*); *reserpine* and *tetrabenazine*, used to treat tardive dyskinesia and Tourette's syndromes, are such drugs.[†] *Bupropion (Wellbutrin)*, an antidepressant, has infrequently been associated with bradykinesia, pseudoparkinsonism, and dysarthria¹⁰⁸; the dysarthria has not been described, but it is probably hypokinetic. Parkinsonism induced by dopamine antagonist drugs usually develops within the first 2 months of treatment⁸ and tends to resolve within weeks to months after withdrawal.^{4,92}

Chronic or toxic exposure to heavy metals (e.g., manganese) or to chemicals such as carbon disulfide, cyanide, and methanol can create a parkinsonian syndrome through their effects on the basal ganglia. Temporary parkinsonism can occur during alcohol withdrawal.⁹²

Acquired metabolic disorders, including those associated with *liver failure*, *hypoparathyroidism*, and *central pontine myelinolysis* (discussed in Chapter 10) can damage the basal ganglia and cause parkinsonism.⁹²

Wilson's disease, which can lead to abnormal copper depositions in the liver and brain, can produce parkinsonian signs, including hypokinetic dysarthria. Because it may also affect structures outside the basal ganglia, Wilson's disease is frequently associated with mixed dysarthria; it is discussed further in Chapter 10.

Trauma

Bradykinesia, rigidity, and tremor are among the many neuromotor deficits that may be caused by a single-event traumatic brain injury (TBI) that causes loss of consciousness. Repeated head trauma, as can occur in boxers (*dementia pugilistica*), can damage the substantia nigra. Over time, this can lead to parkinsonian-like motor abnormalities (including hypokinetic dysarthria), as well as dementia and ataxia.

Neurosurgery, including stereotactically guided lesioning and deep brain stimulation of the thalamus and globus pallidus, have been effective in relieving limb tremor and dyskinesias associated with PD.

*These drugs include phenothiazines (e.g., chlorpromazine), butyrophenones (e.g., haloperidol), thioxanthenes (e.g., thioxene), dibenzazepines (e.g., loxapine), and substituted benzamides (e.g., metoclopramide).⁹²

†See Molho and Factor⁹² for a list of other medications that may cause or worsen parkinsonism.

However, such treatments, especially when bilateral, carry risks for temporary or persisting speech deficits, including the development of dysarthria or worsening of a preexisting dysarthria.^{46,107,115}

Infectious Conditions

Many cases of parkinsonism emerged in the aftermath of a viral encephalitis epidemic during and after World War I; this no-longer-occurring condition is known as *postencephalitic parkinsonism*. Today, other viral encephalitides are sometimes associated with parkinsonism.⁴² *Acquired immunodeficiency syndrome (AIDS)* is thought to be the most common infectious cause of parkinsonism. Uncommon infectious causes include *Creutzfeldt-Jacob disease*, *syphilis*, *tuberculosis*, *Whipple's disease*, and *mycoplasma pneumoniae*.⁹²

Other

Normal pressure hydrocephalus (NPH) (defined in Chapter 6) and *obstructive hydrocephalus* can be associated with parkinsonism, including hypokinetic dysarthria. Ataxic features (including ataxic dysarthria), dementia, and incontinence are also often present.

Parkinsonism can be a significant or minor component of many inherited diseases. Some examples include Wilson's disease, Huntington's disease, familial basal ganglia calcification, some dominantly inherited spinocerebellar ataxias, and some rare inborn errors of metabolism.⁹² Some of these conditions are addressed in Chapters 6, 8, and 10.

■ SPEECH PATHOLOGY

Distribution of Etiologies, Lesions, and Severity in Clinical Practice

Box 7-1 and Figure 7-2 summarize the etiologies for 167 quasirandomly selected cases seen at the Mayo Clinic with a speech pathology diagnosis of hypokinetic dysarthria. The cautions expressed in Chapter 4 about generalizing these data to the general population or to all speech pathology practices also apply here.

The data establish that hypokinetic dysarthria can have numerous etiologies, the distribution of which are different from that associated with several other dysarthria types. Degenerative diseases accounted for 78% of the cases, of which three quarters had diagnoses of PD or parkinsonism. Unspecified "CNS degenerative disease" was the diagnosis for several cases; parkinsonism was suspected in some of these, but others had signs of more than basal ganglia degeneration (e.g., dementia or cerebellar findings).

These cases support the status of PD and parkinsonism as prototypes of diseases associated with hypokinetic dysarthria, and they establish that vascular, toxic-metabolic, traumatic, and infectious conditions can also produce the disorder. As might be expected, nearly all patients had nonspeech deficits that were clinically localized to the basal ganglia control circuit, although not necessarily limited to it. Neuroimaging (e.g., computed tomography [CT] scan, magnetic resonance imaging [MRI]) in most cases, however, was either negative or revealed only mild cerebral atrophy or enlarged ventricles. This is common in idiopathic PD and related degenerative diseases that include the basal ganglia. Most vascular and traumatic cases had neuroimaging evidence of lesions in or near the basal ganglia but not always bilaterally; however, patients with only unilateral lesions on neuroimaging nonetheless usually had bilateral clinical signs, indicating the presence of bilateral pathology.

This retrospective review did not permit a precise description of dysarthria severity. However, in those patients for whom a judgment about intelligibility was stated (75% of the sample), 77% had reduced intelligibility. The degree to which this figure accurately estimates the frequency of intelligibility impairments in the population with hypokinetic 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, however, it is reasonable to conclude that intelligibility is frequently reduced by hypokinetic dysarthria.

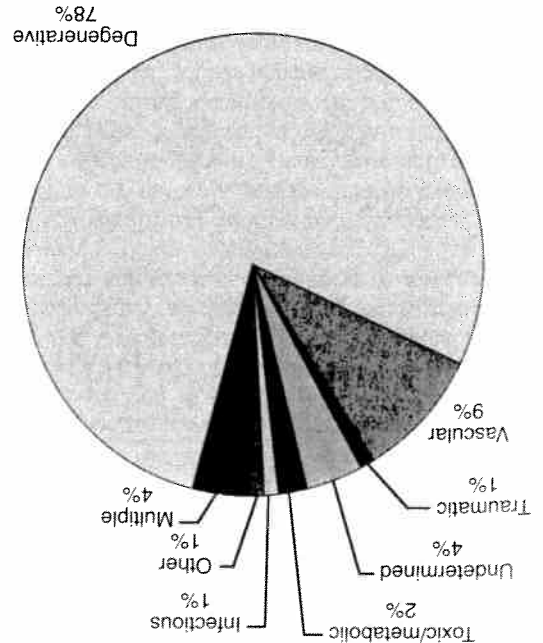
Finally, cognitive impairment was common in this sample. For patients whose cognitive abilities were explicitly commented on or formally assessed (84% of the sample), 72% had some degree of cognitive impairment.*

Patient Perceptions and Complaints

Affected people may describe their speech in ways that provide clues to diagnosis and its localization. Although they frequently report that others tell them their voice is *quieter* or *weak*, they often *deny* or *minimize* such changes themselves. Complainers that rate is *too fast* or that words are *indistinct* are common. Some report that it is *“hard to get speech started.”* Some use the word *stutter* to describe any of which might have caused the dysarthria (i.e., parkinsonism, stroke, and multiple sclerosis). The remaining causes included postencephalitic parkinsonism, encephalitis, basal ganglia calcification, and an unusual case with parkinsonian signs following radiation therapy.

*Bayles et al.¹⁰ found no evidence of language difficulty in a group of 75 people with PD who were not demented. Mildly demented PD patients did have difficulty on several measures of language performance.

FIGURE 7-2 Distribution of etiologies for 167 quasirandomly selected cases with a primary speech pathology diagnosis associated with multiple system atrophy or parkinsonism plus amyotrophic lateral sclerosis. (Most of hypokinetic dysarthria at the Mayo Clinic from 1969-1990 and from 1999-2001 (see Box 7-1 for details).)



The remaining degenerative diseases included conditions associated with multiple system atrophy or one of its subtypes, Lewy body disease, and parkinsonism plus amyotrophic lateral sclerosis. Nonhemorrhagic stroke accounted for the largest proportion of the relatively small number of vascular etiologies. This small number is consistent with clinical impressions that hypokinetic dysarthria is an uncommon result of stroke. The remaining vascular etiologies included hemorrhagic events, small vessel disease, and anoxia resulting from cardiac arrest. The 4% of cases with undetermined etiology had diagnostic possibilities that included PD, PSP, and stroke. In several of these cases, signs and symptoms were of recent onset or were subtle. It is likely that a more definitive diagnosis emerged over time. The small percentage of cases (2%) with toxic or metabolic etiology is generally consistent with preconditions from the literature (e.g., phenothiazine use, carbon monoxide poisoning). Two cases had closed head injuries, illustrating that the frequent significant effect of head injuries on subcortical structures can produce hypokinetic dysarthria. A few cases had more than one disease, any of which might have caused the dysarthria. The parkinsonism, stroke, and multiple sclerosis). The remaining causes included postencephalitic parkinsonism, encephalitis, basal ganglia calcification, and an unusual case with parkinsonian signs following radiation therapy.

initiating speech. It is rare that a patient associates such dysfluencies with anxiety, anticipation of difficulty, or specific word or sound fears.

Complaints about negative effects of *fatigue* on speech are not uncommon. Those with drug-responsive parkinsonism sometimes note *variations in speech during their medication cycle*, frequently characterized by deterioration just before their next dose. *Drooling* and *swallowing complaints* are not uncommon. Some report that their upper lip feels stiff, perhaps reflecting a perception of reduced movement flexibility.

Clinical Findings

Hypokinetic dysarthria usually occurs with other signs of basal ganglia disease, and it occurs frequently enough in parkinsonism for its recognition to serve as confirmatory evidence for the neurologic diagnosis. Even more important, it sometimes is the presenting complaint and only sign of parkinsonism. In such cases, recognition of the dysarthria as hypokinetic can be essential to localization and diagnosis.

Nonspeech Oral Mechanism

The oral mechanism examination can be revealing and often confirmatory of a diagnosis of hypokinetic dysarthria. The eyes may have a *reduced blink frequency*. The face may be *unsmiling, masked, or expressionless* at rest (see Figure 7-1) and *lack animation* during social interaction. Movements of the eyes and face, hands, arms, and trunk that normally accompany speech and complement the emotions and indirect meanings conveyed through prosody may be attenuated. Chest and abdominal movements during quiet breathing may be reduced, and excursion may remain reduced even when the patient attempts to breathe deeply.

As the eyes may blink infrequently, so may the patient *swallow infrequently*, perhaps another reflection of rigidity or reduced automatic movements. This may lead to excessive saliva accumulation and *drooling*. When moving the eyes to look to the side or up or down, the normal tendency for head turning to accompany the gaze may be reduced.

A tremor or *tremulousness* of the jaw and lips may be apparent at rest or during sustained mouth opening or lip retraction. Similarly, the tongue is often strikingly tremulous on protrusion or at rest within the mouth. The lips (particularly upper) can appear tight or immobile at rest and during movement, including speech. Jaw, face, and tongue strength may be grossly normal, often surprisingly so given their limited movement during speech. Nonspeech alternating motion rates (AMRs) of the jaw, lips, and tongue may be slowly initiated and

completed or rapid and markedly restricted in range. In contrast, range of motion for single movements (e.g., lip retraction) may be normal or distinctly greater than that observed during speech or expected emotional responses.

The occurrence of swallowing problems in PD ranges from approximately 40% to 80%⁶⁶; they are usually preceded by dysarthria. The median latency between disease onset to development of dysphagia is generally longer in PD (130 months) than other degenerative diseases associated with parkinsonism, and latency from disease onset to onset of dysphagia is strongly correlated with overall survival.⁹⁵

The overall impression derived during casual observation and formal oral mechanism examination is one of a lack of vigor or animation in the absence of a degree of weakness that might explain it. At rest, as well as during social interaction and speech, the patient's facial affect appears restricted, unemotional, and sometimes depressed. These appearances may not accurately reflect the inner emotional state. Unfortunately, speech usually mirrors these nonverbal characteristics.

Speech

Conversational speech or reading, speech AMRs, and vowel prolongation all provide useful information about salient and distinguishing speech characteristics. Conversational speech and reading are essential for identifying the prosodic abnormalities that can be so prominent in the disorder. Speech AMRs are particularly useful for observing reductions in range of movement and rate abnormalities; although not always present, *rapid, accelerated, and sometimes "blurred" speech AMRs are distinguishing perceptual characteristics of hypokinetic dysarthria* (Figure 7-3). Vowel prolongation is useful for isolating some of the disorder's phonatory characteristics, especially those associated with loudness and quality.

Hypokinetic dysarthria usually reflects neuromuscular abnormalities at all levels of the speech system, usually related to restriction in the range or speed of movements. The effects of these abnormalities give hypokinetic dysarthria its distinctive characteristics, most of which are associated with phonatory and articulatory activities and the effects of those abnormalities on prosody.

Table 7-2 summarizes the neuromuscular deficits presumed by Darley, Aronson, and Brown (DAB)^{27,28} to underlie hypokinetic dysarthria. Speech movements and their timing are generally accurate. Individual movements are slowed, but repetitive movements may be fast, especially when range of movement is limited. The range and force of individual and repetitive movements are reduced.

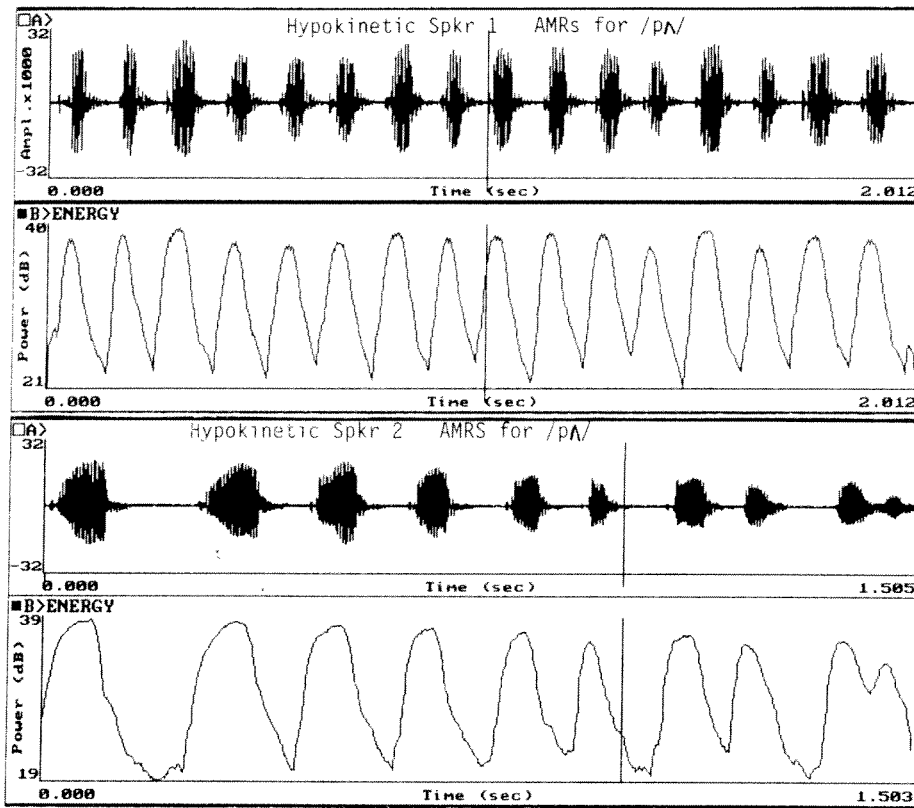


FIGURE 7-3 Raw waveform and energy tracings of speech alternate motion rates (AMRs) for /pʌ/ by two speakers with hypokinetic dysarthria. Speaker 1's AMRs (2 seconds) are regular but rapid (=8 Hz). Speaker 2's productions (1.5 seconds) are normal in rate (=6 Hz in the first second) but show a trend toward increased overall rate and reduced amplitude and duration of each pulse, the acoustic correlate of perceived accelerated rate.

Table 7-2 Neuromuscular deficits associated with hypokinetic 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	Fast	Reduced	Very reduced	Reduced (paretic)	Excessive (balanced)

Modified from Darley FL, Aronson, AE, Brown JR: Clusters of deviant speech dimensions in the dysarthrias, *J Speech Hear Res* 12:462, 1979.

Muscle tone is often excessive (i.e., rigid) with resistance to movement in all directions, a condition that contributes to decreased range of movement. *Reduced range of movement may be the most significant underlying neuromuscular deficit in hypokinesia as it affects speech.* The relationships among these characteristics and the specific deviant characteristics associated with hypokinetic dysarthria are discussed in the next section. Experimental support for the presumed

underlying neuromuscular deficits are reviewed in the section on acoustic and physiologic findings.

Prominent Deviant Speech Characteristics and Clusters of Deviant Dimensions

A general profile of hypokinetic dysarthria was established by Logeman et al.,⁸² who determined the frequency of deviant speech characteristics in a

table 7-3 Deviant cluster of abnormal speech characteristics found in hypokinetic dysarthria

Cluster	Speech Characteristics
Prosodic insufficiency	Monopitch Monoloudness Reduced stress Short phrases Variable rate* Short rushes of speech* Imprecise consonants*

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

*Considered a component of prosodic insufficiency in hypokinetic dysarthria but not in other dysarthria types with prosodic insufficiency.

group of 200 people with PD. Approximately 90% had a speech deficit, attesting to the high prevalence of dysarthria in the disease. Eighty-nine percent had voice disorders characterized by *hoarseness*, *roughness*, *tremulousness*, and *breathiness*, and 45% had articulation problems. Twenty percent had rate abnormalities characterized by *syllable repetitions*, *shortened syllables*, *lengthened syllables*, and *excessive pauses*. Ten percent were *hypernasal*. Of interest, 45% had voice abnormalities only, and all patients with articulation problems had voice problems. The authors noted that this might reflect the existence of subgroups of dysarthrias in PD or the tendency for dysarthria to begin with laryngeal manifestations and eventually include articulation and other abnormalities. Zwirner and Barnes¹²⁹ also noted the higher frequency of laryngeal than articulatory impairment in PD.

DAB²⁸ found only one cluster of deviant speech dimensions in their group of parkinsonian patients. They labeled it *prosodic insufficiency* to represent the attenuated patterns of vocal emphasis that result from the combined effects of speech characteristics that make up the cluster. The characteristics include *monopitch*, *monoloudness*, *reduced stress*, *short phrases*, *variable rate*, *short rushes of speech*, and *imprecise consonants* (summarized in Table 7-3). Together, these features give hypokinetic dysarthria its distinctive gestalt of a flat, attenuated, and sometimes accelerated quality. The neuromuscular basis for the cluster was attributed by DAB to reduced range of movement and to the fast repetitive movements that are unique to parkinsonism.

Table 7-4 summarizes the most deviant speech characteristics encountered in hypokinetic dysarthria,²⁷ as well as the component of the speech system most prominently associated with each characteristic. The rankings in the table represent severity

The most deviant speech dimensions encountered in hypokinetic dysarthria by Darley, Aronson, and Brown,²⁷ listed in order from most to least severe. Also listed is the component of the speech system associated with each speech characteristic. The component "prosodic" is listed when several components of the speech system may contribute to the dimension.

table 7-4

Dimension	Speech Component
Monopitch*	Phonatory-prosodic
Reduced stress*	Prosodic
Monoloudness*	Phonatory-respiratory-prosodic
Imprecise consonants	Articulatory
Inappropriate silences*	Prosodic
Short rushes of speech*	Articulatory-prosodic
Harsh voice quality	Phonatory
Breathy voice (continuous)	Phonatory
Low pitch	Phonatory
Variable rate*	Articulatory-prosodic
Other	
Increased rate in segments*	Prosodic
Increase of rate overall*	Prosodic
Repeated phonemes*	Articulatory

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

ratings of the speech characteristics and not necessarily the features that best distinguish hypokinetic dysarthria from other dysarthria types.

A few additional observations help to complete the picture of the disorder:

1. Logeman and Fisher⁸¹ described the specific features of imprecise consonants. They found a predominance of manner errors, which occurred most frequently for stops, fricatives, and affricates. Stops, especially velars, were most frequently in error and were perceived as fricatives, presumably because of incomplete articulatory contact and continual emission of air during what should have been a stop period; this was also perceived for the stop portion of affricates. Fricatives were perceived as reduced in sharpness, presumably due to a reduced degree of articulatory constriction. These features are related to the acoustic feature of *spirantization* and may be the result of *articulatory undershooting* resulting from accelerated rate or reduced range of movement, or both.
2. Some prominent features of hypokinetic dysarthria are not captured in the cluster of prosodic insufficiency (compare the speech characteristics in Tables 7-3 and 7-4). For

example, frequently occurring *inappropriate silences* are not logically related to the neuromuscular deficits presumed to underlie prosodic insufficiency; they more likely reflect difficulty in initiating movements.

3. Harshness, breathiness, and reduced loudness are sometimes the first sign of hypokinetic dysarthria and parkinsonism. When marked, this dysphonia can have a *mildly strained, tight, aphonic, or whispered quality*. Even when not pervasively present, a strained-whispered aphonia will sometimes emerge from a breathy-harsh quality and persist for several seconds toward the end of a maximum vowel prolongation task; in the author's experience, this rarely occurs in other dysarthria types. In general, *dysphonia can be the presenting and most prominent and debilitating speech feature in people with hypokinetic dysarthria*.
4. Rate abnormalities can be a striking and highly distinctive feature of hypokinetic dysarthria. These often are apparent during AMRs, in which rate may be *rapid or accelerated*; combined with reduced range of articulatory excursions, they may have a "blurred" quality, as if all syllables are run together. In conversation or reading, patients may demonstrate *short rushes of speech* in which several words are uttered together, sometimes rapidly, and are separated from the remainder of the utterance by pauses that may occur at inappropriate intervals. Some patients demonstrate an apparent *increased speech rate within segments*, a characteristic that appears analogous to the festinating gait so often present in parkinsonism. Finally, some patients' *overall speech rate is rapid*. Although not always present, features that lead to a perception of rapid rate in hypokinetic dysarthria are unique among the dysarthrias.*
5. Dysfluencies in the form of *repeated phonemes* are not uncommon,[†] and some-

times they are prominent enough to be designated as stuttering.⁷² They tend to occur at the beginning of utterances or following pauses. They are usually rapid and sometimes blurred and restricted in range of movement. When vowels and some consonants are "repeated," they may sound more like a prolonged vowel with a tremulous character. These features may be analogous to parkinsonian patients' difficulty in initiating walking ("freezing"), and the rapid, short shuffling steps that may occur as walking begins. Although dysfluencies or stuttering-like behavior can occur in several neurologic conditions (see Chapter 13), the specific character of repeated phonemes in hypokinetic dysarthria tend to be distinctive.

6. A disorder that can be associated with hypokinetic dysarthria and may be strongly related to phoneme repetitions is *palilalia*, a problem characterized by "compulsive reiteration of utterances in a context of increasing rate and decreasing loudness."⁷⁴ The repetitions usually involve words and phrases; phoneme repetitions are generally not subsumed in the disorder's definition. Palilalia is usually associated with bilateral subcortical pathology, especially involving the basal ganglia, but has also been noted in bilateral frontal lobe pathology.¹⁴ It is discussed further in Chapter 13.
7. True voice tremor is uncommon in hypokinetic dysarthria. However, the voice may be unsteady and tremorlike in character secondary to the prominent head and upper limb tremor present in some patients. In addition, the voice during vowel prolongation is sometimes characterized by a rapid, low amplitude *tremulousness*, sometimes known as *flutter*. Logeman et al.⁸² found vocal tremulousness in 14% of their 200 parkinsonian patients.
8. Abnormal resonance is not usually prominent, but mild hypernasality is probably present in 10% to 25% of patients with hypokinetic dysarthria.^{27,28,82} Thus hypernasality and mild "weakening" of pressure consonants because of nasal airflow are acceptable abnormalities in the disorder; that is, they need not raise strong suspicions about another dysarthria type (particularly flaccid or spastic dysarthria) in people whose other deviant speech characteristics are consistent with hypokinetic dysarthria.

What features of hypokinetic dysarthria help distinguish it from other MSDs? Among all of the abnormal characteristics that may be detected,

*Note, however, that rapid speech rate can be idiosyncratic or associated with some nondysarthric neurologic and psychiatric conditions.

†Dysfluencies, including repetitions of sounds, syllables, and words, sound prolongations, and inappropriate silences and excessive pauses, have been observed in several group studies of hypokinetic speakers with PD.^{11,27,82} Some suggest that dysfluencies are exacerbated by levodopa,⁸¹ others suggest that increased dysfluency is associated with increased or decreased dopamine levels,⁴¹ and still others report that dysfluencies are more frequent in PD patients with advanced disease regardless of whether they are in an on or off levodopa state.¹¹

monopitch, monoloudness, reduced loudness, reduced stress, variable rate, short rushes of speech, overall increases in rate, increased rate within segments, rapid speech AMRs, repeated phonemes, and inappropriate silences are the most common distinctive clues to the presence of the disorder.

Table 7-5 summarizes the primary distinguishing, distinctive speech characteristics and common oral mechanism findings and patient complaints encountered in hypokinetic dysarthria.

Acoustic and Physiologic Findings

Although hypokinetic dysarthria is often clearly distinguishable from other dysarthria types, there is perceptual heterogeneity among patients with the disorder. This variability is also apparent, and perhaps even greater, within and among many acoustic and physiologic measures. Acoustic and physiologic abnormalities often exist in only some dysarthric patients under study, and a number of the abnormalities may also be found in other dysarthria types. In fact, some “abnormalities” may be normal if they are compared to appropriate age and gender-matched normative data. This may be the case for

hypokinetic dysarthria more than for any other dysarthria type, because several of its salient perceptual features (e.g., reduced loudness, hoarseness, breathiness) and acoustic correlates are common in elderly people without neurologic disease. With these caveats in mind, acoustic and physiologic measures have contributed to a richer description and better understanding of the disorder.

Respiration

Respiratory abnormalities occur frequently and are a common cause of death in parkinsonism.⁵⁰ Although respiration has received comparatively little attention in acoustic and physiologic studies of speech, it could logically contribute to some of the prominent features of the disorder, particularly those related to loudness and prosody. Reduced vital capacity, reduced amplitude of chest wall movements during breathing, reduced respiratory muscle strength and endurance, irregularities in breathing patterns, and increased respiratory rates have been documented.^{55,69,76,111,122} Many abnormalities have been attributed to alterations in the normal agonist-antagonist relationships among respiratory muscles during breathing.

Of direct relevance to speech are data from speech and maximum performance vocal tasks. Reduced maximum vowel duration, reduced airflow volume during vowel prolongation, fewer syllables per breath group, use of greater than average percentage of vital capacity per syllable, and increased breath groups during reading have been documented in some patients with parkinsonism and presumed hypokinetic dysarthria.^{12,20,55,69,94} It should be noted, however, that such characteristics could also reflect abnormalities at the laryngeal level. That respiratory abnormalities contribute to these characteristics in at least some patients is suggested by findings of abnormally small rib cage volumes and abnormally large abdominal volumes at the initiation of speech breath groups in PD speakers who produced fewer words per breath group and spoke for less time per breath group than normal speakers.¹¹¹ In addition, Murdoch et al.⁹⁷ found abrupt movements of chest wall parts and paradoxical movements of the rib cage and abdomen during vowel prolongation and syllable repetition tasks in approximately half of their 19 subjects with hypokinetic dysarthria; there was no unambiguous explanation for the abnormalities, but the authors speculated that rigidity of respiratory muscles might have been responsible. Finally, the presence of impaired respiratory control in some speakers is suggested by documentation of longer latencies before beginning exhalation following forceful inhalation, delayed initiation of phonation once exhalation begins, difficulty altering automatic

table 7-5 Primary distinguishing speech and speech-related findings in hypokinetic dysarthria

Perceptual	
Phonatory-respiratory	Reduced loudness
Articulatory	Repeated phonemes, palilalia, rapid or “blurred” AMRs
Prosodic	Reduced stress, monopitch, monoloudness, inappropriate silences
	Short rushes of speech, variable rate, increased rate in segments, increased overall rate
Physical	Masked facial expression
	Tremulous jaw, lip, tongue
	Reduced range of motion on AMR tasks
	Head tremor
Patient Complaints	Reduced loudness, rapid rate, “mumbling,” “stuttering,” difficulty initiating speech (often reported as what listeners tell them as opposed to their own perception)
	Stiff lips

AMR, Alternate motion rate.

respiratory rhythms for speech, and difficulty tracking a sinusoidal target with respiratory movements.^{35,69,90}

Reduced respiratory excursions, reduced vital capacity, paradoxical respiratory movements, rapid breathing cycles, and difficulty altering vegetative breathing patterns for voluntary activities seem consistent with patterns of rigidity, hypokinesia, and difficulty initiating movements that occur in other muscle groups in parkinsonism. Such difficulties could contribute importantly to reduced physiologic support for speech and some of the disorder's phonatory and prosodic abnormalities, especially reduced loudness, short phrases, short rushes of speech, and inappropriate pauses.

Phonation

A number of acoustic and physiologic studies have examined laryngeal function in hypokinetic dysarthria. In general, they confirm hypotheses generated by perceptual analyses and provide additional insights into mechanisms underlying abnormal voice and speech characteristics.

1. Fundamental frequency (f_0) and intensity.

Abnormal pitch is usually not a prominent perceptual feature of hypokinetic dysarthria, but several studies have reported elevated f_0 .^{19-21,53,58,61,68,84,85,106} However, the increase in f_0 is not always statistically significant relative to age-matched norms,⁶⁸ and in females f_0 is sometimes reduced.⁵³ For example, the median f_0 for Canter's¹⁹ male parkinsonian subjects was 129 Hz, compared to 106 Hz for age-matched male controls; Metter and Hanson⁹¹ found that f_0 fell mostly within the normal range in their PD patients, although with a tendency for it to increase with increased disease severity. These findings stand in contrast to the perceptual observation of DAB²⁷ that pitch tended to be perceived as low. The reasons for these discrepancies are not clear. It may be that there is considerable intersubject variability in f_0 /pitch, that there are gender differences, or that factors other than f_0 lead to a perception of low pitch (i.e., monopitch, monoloudness, and reduced loudness could lead to perceptions of lower pitch). The fact that pitch and f_0 are neither generally nor extremely abnormal, however, suggests that they are not reliably sensitive distinguishing features of hypokinetic dysarthria.

Measures of intensity have been less ambiguous. They generally document reduced vocal intensity during various speech, vowel prolongation, and AMR

tasks.^{33,39,51,53,58,68,106a} Ho, Iansek, and Bradshaw⁵¹ found that speakers with PD had reduced conversational loudness at various distances from their listeners but did increase loudness as listener distance increased. These results were interpreted as a reflection of normal loudness regulation but within a context of a dampened "motor set" for loudness, analogous to the reduced range of limb movement associated with PD. It is also of interest that the PD patients' perceptual judgments overestimated speaker loudness as distance increased, raising the possibility that perceptual deficits played a role in their ability to set loudness for themselves. Finally, loudness problems (e.g., overall loudness, loudness decay) may be exacerbated under conditions of divided attention, such as speaking while performing a visual-manual tracking task.⁵²

2. f_0 and intensity variability and the voice spectrum.

Measures of f_0 and intensity variability are much more revealing. They have been examined in a wide variety of tasks, including vowel prolongation, spontaneous speech, reading, word and sentence imitation, emotional expression, pitch glide tasks, and tasks requiring a range of high or low pitch productions. Specific abnormalities are somewhat task dependent, with increased variability found on some measures and decreased variability on others. In general, acoustic findings provide strong support for perceptual ratings of monopitch and monoloudness (Figure 7-4).

Many long-term measures (e.g., syllables, sentences) consistently document reduced f_0 and loudness variability or range.*[†] The relevance of these findings to clinical practice is illustrated by Bunton et al.,¹⁶ who documented reduced f_0 range during sentence production in several speakers with PD. They then used a linear predictive coding (LPC) technique to artificially flatten f_0 range during production of sentences in normal speakers and PD speakers. This resulted in reduced intelligibility in all subject groups, with the effect exaggerated in the dysarthric speakers. These findings support the perception of monopitch in hypokinetic dysarthria and

*Reduced loudness can interfere with other acoustic measures, as illustrated by Canter's²¹ observation that he was unable to measure speech AMR rate in some of his patients because of "flattened intensity peaks."

[†]References 16, 17, 19, 20, 26, 53, 61, 84, 85, 91, 105.

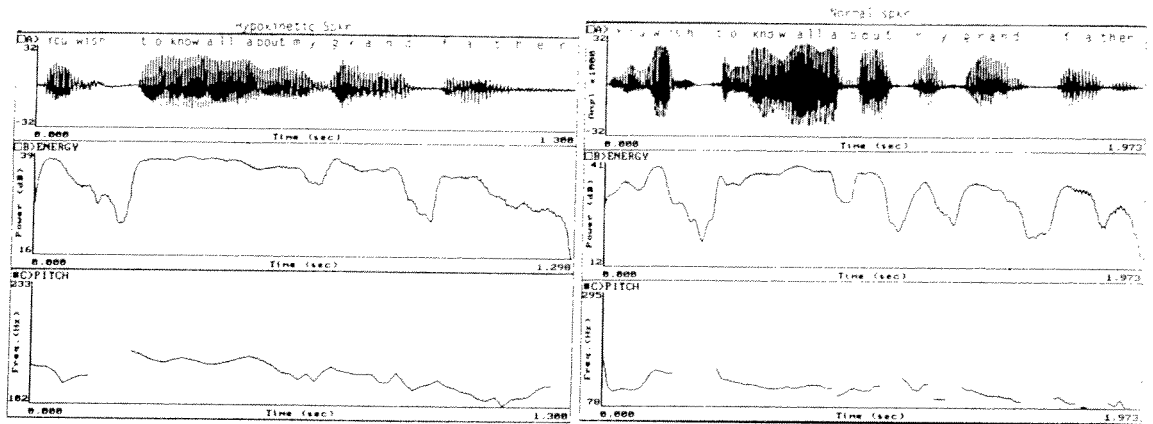


FIGURE 7-4 Raw waveform and energy and pitch (f_0) tracings for the sentence "You wish to know all about my grandfather's father" by a normal male speaker and a male with hypokinetic dysarthria. The normal speaker completes the sentence in approximately 2 seconds, the hypokinetic speaker in 1.3 seconds (66% of the normal speaker's rate, consistent with a perception of rapid rate). The energy tracing for the normal speaker has clearly defined syllables of varying duration; the hypokinetic speaker has few well-defined syllables, possibly reflecting the effects of rapid rate, continuous voicing, spirantization, and monoloudness. The speakers' pitch tracings are similar in contour, but the normal speaker has brief breaks in phonation during stop closure and voiceless consonants. The absence of breaks in phonation after the word "to" for the hypokinetic speaker probably reflects continuous voicing and spirantization.

attest to the perceptual contribution of f_0 variation to sentence-level intelligibility.*

In contrast to decreased variability of f_0 and intensity during sentence and maximum range tasks, long-term variability within vowel prolongation tasks is generally increased. For example, during vowel prolongation tasks PD speakers tend to have abnormally large standard deviations of f_0 , and that variability is correlated with perceptual judgments of dysphonia.^{32,129,130} In some studies, many hypokinetic speakers have abnormally high percentage variation in f_0 and variation in peak amplitude during sustained phonation.^{64,68} Larson, Ramig, and Scherer,⁷⁵ who found abnormally high long-term amplitude perturbation in two speakers with PD, felt the abnormality might reflect relatively slow innervation fluctuations to laryngeal abductor or adductor muscles, supraglottic structures, or a combination of these.

A promising acoustic measure for capturing some of the abnormalities perceived in hypokinetic dysarthria, especially in connected speech, may lie in long-term average spectrum (LTAS) measures, which capture the shape of the distribution of energy in the

acoustic spectrum. Dromey³³ compared dysarthric speakers with PD to age-matched controls and found that LTAS measures distinguished the two groups on vowel prolongation, reading, and monologue tasks when other acoustic measures, such as sound pressure level and f_0 variability, did not. This suggests that some of the prominent qualitative deficits perceived in the hypokinetic voice may be more readily detectable in the spectrum of the voice than in "simpler" measures of frequency and intensity variability.

3. *Voice tremor.* Voice tremor is not a prominent perceptual feature of hypokinetic dysarthria, and the tremor that can be detected seems not to differ substantially from the tremor in normal individuals.¹⁰² Nonetheless, visual and acoustic evidence of voice tremor is not uncommon. Schulz et al.¹⁰⁶ found evidence of laryngeal or arytenoid tremor on videostroboscopy in six people with PD, and Gallena et al.⁴⁰ documented laryngeal tremor in three of their six PD speakers. Perez et al.⁹¹ observed that more than 50% of their 29 patients with PD or parkinson's plus syndromes had vertical laryngeal tremor or tremor of the arytenoid cartilages during endoscopic and stroboscopic examination.

Although voice tremor in the range of 4 to 7 Hz has been reported in some speakers with PD,⁵⁹ it is not pervasively present perceptually.

*Laures and Weismer⁷⁷ also demonstrated reduced sentence intelligibility by synthetically flattening f_0 in two normal speakers.

ally or acoustically and therefore not essential for a diagnosis of hypokinetic dysarthria. For example, Holmes et al.⁵³ found tremor in their patients with later-stage but not early-stage PD. Some investigators have found both amplitude and frequency fluctuations in the tremor, but others suggest that tremor is more likely in the frequency than the amplitude domain.^{87,102,104,116}

Boutsen, Duffy, and Aronson¹³ obtained acoustic confirmation of a perceived high-frequency tremor or flutter (one component in the 5 to 6 Hz range and another in the 9 to 11 Hz range) in a woman with PD, in which the tremor was more evident in amplitude than frequency modulation. The association of flutter with hypokinetic dysarthria is important to differential diagnosis, because flaccid dysarthria is the only other dysarthria type in which it has been observed.

4. *Maximum phonation time (MPT)*. Adams⁶ suggests that MPT for vowel prolongation may not be different from normal, perhaps because of test methods and inherent variability within and across individuals. However, King et al.⁷⁰ studied a group of PD subjects and documented significant longitudinal declines (3 to 36 months) in maximum and average duration of sustained vowel prolongation. Thus reliably obtained MPT may be sensitive to changes within individuals with hypokinetic dysarthria over time but not necessarily sensitive to detection of the disorder itself.
5. *Jitter, shimmer, and other indices of quality*. Speakers with PD may have abnormally high jitter and shimmer or related measures of the regularity of vocal fold vibration, possibly reflecting reduced short-term neuromuscular control of the laryngeal abductor or adductor mechanism.^{6,53,75,85} Abnormal shimmer values have been correlated with perceptual measures of breathiness, a relationship that could be related to vocal fold bowing, with subsequent increased airflow turbulence and intensity variations.⁸⁵

Abnormal jitter and shimmer values are not always found in hypokinetic speakers. For example, although Kent et al.⁶⁵ found females with PD to differ from female controls in shimmer, measures of jitter and shimmer failed to discriminate PD males from healthy males. They concluded that "there is reason to question the sensitivity of acoustic perturbation measures [*such as shimmer and jitter*] to voice function in

dysarthria, at least for the general purposes of identifying abnormality and classifying clinical groups."

- Although abnormal signal-to-noise (S/N) ratios measured in vowels have been reported in some speakers with hypokinetic dysarthria, Adams⁶ indicates that such abnormalities are not always present. For example, Kent et al.⁶⁵ found that S/N ratio failed to discriminate males with PD from healthy males.
6. *Motor control*. Several acoustic studies suggest that laryngeal control is reduced. There is evidence that some patients are slow to initiate phonation and that such events correlate with perceived inappropriate silences.⁸⁵ Relatedly, Lehiste⁷⁹ found evidence of voiceless transitions from vowels to following consonants within syllables, possibly attributable to incoordination of articulation and voicing. Canter²¹ noted that perceived omission of final consonants in parkinsonian patients could be due to poor phonatory control. Other studies have found evidence of continuous voicing (see Figure 7-4) within sentences or on AMR tasks containing voiceless consonants.^{63,85,105} These findings suggest difficulty with the rapid termination of voicing within utterances containing voiceless phonemes. Finally, McClean, Beukelman, and Yorkston's⁹⁰ PD patients had difficulty varying vocal pitch to control a cursor in order to track a visually displayed sinusoidal target.
 7. *Laryngeal structure, movement, and airflow*. Laryngeal structure and functions for speech have received considerable attention. A comprehensive telescopic cinelaryngoscopy study of 32 unselected patients with PD by Hanson, Gerratt, and Ward⁴⁵ documented a number of visible abnormalities. Among the most striking was that only two patients were free of "abnormal phonatory posturing," and they had normal voices and no voice complaints. *Vocal fold bowing* during phonation, represented by a significant glottic gap but with tightly approximated vocal processes, was observed in 30 patients; the increased glottal gap was correlated with perceived breathiness and reduced intensity. Tremulousness of the arytenoid cartilages was apparent during quiet breathing in some subjects, but the perception of voice tremor seemed more strongly related to the secondary effects of head tremor. Laryngeal structure asymmetries were apparent in many patients, with asymmetries occurring in vocal

fold length, degree of bowing, and ventricular fold movements. Some patients exhibited approximation of the ventricular folds during phonation. Voice was often better for patients with supraglottic contraction, which may have assisted adduction and reduced breathiness. The authors noted that the vocal folds appeared solid, in spite of bowing, in contrast to the hypotonicity that may be present with lower motor neuron (LMN) paralyses. The evidence of increased adductor contraction, asymmetric contraction, and vocal fold bowing inconsistent with LMN lesions led to the conclusion that abnormalities in phonatory postures were related to laryngeal muscle rigidity.

The observations of Hanson, Gerratt, and Ward have been largely replicated and refined in subsequent studies. For example, findings of bowing and a glottal gap or incomplete vocal fold adduction during phonation have been replicated.^{40,106,110} Perez et al.¹⁰¹ observed a predominantly open phase configuration of the vocal folds during phonation (consistent with breathiness and reduced loudness); phase asymmetry (consistent with hoarseness) frequently was also evident endoscopically.

Electromyography (EMG) and aerodynamic studies provide further insight about restricted vocal fold movements and, in general, suggest that the voice characteristics of hypokinetic dysarthria may be due to problems other than weakness. Hirose,⁴⁷ in a study of EMG patterns in the thyroarytenoid (TA) muscle of a patient with PD and laryngoscopically confirmed limited vocal fold movement, observed that although neuromuscular discharges during phonation were not reduced and there were no pathological discharge patterns, there was a loss of reciprocal suppression of the TA muscle during inspiration. This suggested that limited vocal fold motion might reflect a loss of appropriate reciprocal activity between agonist and antagonist muscles, rather than weakness. In support of this, observations of vocal fold bowing and impairment of voice onset and offset control by Gallena et al.⁴⁰ were associated with increased TA and cricothyroid (CT) activity that could increase vocal fold tension and stiffness and reduce the ability of a speaker to rapidly move the folds for voice onset and offset during speech. The authors concluded that vocal fold bowing was a manifestation of "rigidity due to excessive muscle activity," although they acknowledged that a slight reduction in the abduction action of the posterior cricoarytenoid to oppose the increased action of TA and CT also might contribute to the bowed appearance of the folds.

Aerodynamic studies also suggest that subglottic pressure and laryngeal resistance are abnormally

increased during speech in some hypokinetic speakers.^{43,60} These findings suggest the presence of increased glottal or supraglottal muscle tension during phonation, caused by phonation with a smaller glottal aperture or by greater resistance to deformation of the folds with decreased pulsing of airflow. Jiang et al.⁶⁰ suggested that higher pressures and, presumably, respiratory effort confirm some patients' impressions that they are working harder to produce intensity even when the voice is not as loud as they would like.

In summary, acoustic and physiologic studies of phonatory attributes of hypokinetic dysarthria provide evidence of reduced laryngeal efficiency, flexibility, and control that are, for the most part, consistent with many perceived deviations in voice quality and prosody. Many of these abnormalities can be related to the underlying neuromuscular deficits of rigidity, reduced range of movement, and slowness of movement in the laryngeal muscles.

Resonance

There has been little acoustic or physiologic study of velopharyngeal function in hypokinetic dysarthria, possibly because resonance abnormalities usually are not perceptually prominent. However, a few studies have demonstrated that nasal airflow can be increased in the disorder,^{54,118} that nasalization may spread across several consecutive syllables,^{63,105} and that the degree and velocity of velar movements during speech tasks can be reduced.^{49,50,99} Hirose⁴⁷ noted that velar displacement (as measured by x-ray microbeam) became limited and irregular at faster rates and that the velum tended to stay in an elevated posture. He suggested that this might be due to a loss of reciprocal suppression between functionally antagonistic muscle pairs (e.g., velar lowering resisted by action of velar elevators).

Hoodin and Gilbert⁵⁴ speculated that the perception of resonance abnormalities might be masked by phonatory problems because their nasal airflow measures did not correlate strongly with perceptual ratings of hypernasality. This seems quite possible, but it is also important to note that hypernasality is perceptually evident in some hypokinetic speakers. For example, 31% of the 23 speakers studied by Theodoros, Murdoch, and Thompson¹¹⁸ were perceived as mild or moderately hypernasal. However, the fact that 71% of the speakers were more than one standard deviation above control subjects' mean nasal accelerometry scores suggests that instrumental measures may be more sensitive than perceptual measures to velopharyngeal abnormalities.

To summarize, there is acoustic and physiologic evidence of velopharyngeal dysfunction in at least some people with hypokinetic dysarthria. This seems

to reflect the effects of slow movement, rigidity, or reduced range of movement. These can lead to the perception of hypernasality and weak intraoral pressure during pressure consonant productions.

Articulation

A number of acoustic and physiologic studies of articulatory dynamics provide considerable support (with some qualifications) for the perception of imprecise articulation and rate abnormalities and reduction in range of articulatory movement. These attributes include, but are not limited to, spirantization, reduced displacements of movements, abnormal movement velocities, increased activation in muscles antagonistic to targeted movement, weakness or fatigue, and tremor or unsteadiness. In general, they support a conclusion that articulatory muscles exhibit rigidity and reduced range of motion.⁶⁶

1. *Precision.* It appears that articulatory "undershoot," or failure to completely reach articulatory targets or sustain contacts for sufficient durations, plays a significant role in imprecision in hypokinetic dysarthria. Numerous acoustic studies have detected evidence of *spirantization* during stop and affricate productions.^{2,21,63,123,128} Spirantization, usually taken as evidence of articulatory undershooting, is characterized acoustically by the replacement of a stop gap with low-intensity friction. It is attributed to a failure of complete articulatory closure for stop productions or the stop portion of affricates (see Figure 7-4) and is perceived as aperiodic, fricative-like noise.¹²³ Its effect is to reduce acoustic contrast and detail, a natural product of undershooting articulators and a reasonable explanation for at least some aspects of perceived imprecise articulation.*
2. *Range of movement.* Several studies provide evidence for reduced range of movement (which could explain articulatory undershooting), rigidity, and abnormal speed of articulator movements. Findings include kinematic evidence of lip muscle stiffness or rigidity, reduced amplitude (range) and velocity of lip and jaw movements, and electromyographic evidence of reduced duration and amplitude of lip muscle action potentials.^{18,36,37,43,48-50,56,57,99} EMG studies have also

documented poor reciprocal patterns of activity between jaw opening (e.g., anterior digastric) and jaw closing (e.g., mentalis) muscles during speech tasks^{47,93}; simultaneously active jaw opening and closing muscles would tend to slow or restrict range of movement or do both. It has been suggested that such persistent abnormal muscle contractions—reflecting difficulties with reciprocal adjustments of antagonistic muscles or a loss of reciprocal suppression between functionally antagonist muscle pairs—may represent the physiologic basis of hypokinesia and rigidity.^{47,78}

Evidence from acoustic studies also supports conclusions that range of articulator movement is reduced. For example, Forrest, Weismer, and Turner³⁷ found that some parkinsonian speakers have reduced formant transitions. Weismer et al.¹²⁴ found that speakers with PD had restricted acoustic vowel space (i.e., the acoustic space covered by the first and second formant values for the corner vowels [a/, /i/, /æ/, /u/]), suggestive of a smaller "working space" for vowels (i.e., reduced range of movement).

3. *Rate.* Numerous physiologic and acoustic studies have examined speech rate, a phenomenon of considerable interest because increased rate is often perceived in hypokinetic dysarthria. Results have been inconsistent but illuminating because they suggest that listener perceptions may not always reflect underlying movement dynamics.

Some studies demonstrate variability in rate across subjects, ranging from abnormally slow to abnormally fast.^{19,21,91} Several studies, however, have failed to find abnormalities in speech rate on various tasks.*

Some studies have found evidence of reduced rate. Dworkin and Aronson³⁴ and Ludlow, Connor, and Bassich⁸⁶ found slow AMRs or slow syllable repetition rates in some subjects. Krueel⁷³ documented slow reading rate, and Ludlow, Connor, and Bassich⁸⁶ found reduced first and second formant transition rates, suggestive of decreased articulatory speed.

As might be expected from perceptual descriptions, numerous studies report acoustic or physiologic evidence for increased or accelerated rate on speech AMR or meaningful connected speech tasks, sometimes with concurrent evidence of reduced

*The lack of firm articulatory contact signified by spirantization can make the measurement of voice onset time (VOT) during the production of stop consonants difficult due to lack of a burst signifying the release of the stop.¹⁰⁶

*References 2, 19, 21, 23, 86, 117, 123, 124, 127, 128.

amplitude of articulator movements, in at least some speakers.^{2,5,44,48-50,99} AMRs can be fast (see Figure 7-3), up to 13 per second, with associated decreased range of movement; this extremely fast rate suggests a mode of speech over which there can be no voluntary control.⁹⁹ Hirose⁴⁷ likened abnormally fast speech rate to festinating gait and speculated that this may reflect a disturbance of CNS inhibitory function, such as an abnormal release of an intrinsic oscillation mechanism.

Finally, at least some speakers with PD have difficulty altering rate when requested.^{85,86} For example, speakers studied by Ludlow, Connor, and Bassich⁸⁶ had trouble altering sentence and phrase durations when they were asked to speak at faster than conversational rates; that is, there was less of a difference between their conversational and fast rates in comparison to control speakers. The authors speculated that a major problem for these speakers is in controlling alterations in rate, even though the overall temporal organization of speech may be unaffected.

The results of these studies indicate that speech rate is heterogeneous within the population of speakers with hypokinetic dysarthria. Because such variability is probably not simply a function of severity, this raises the possibility of subtypes of the disorder, something that deserves investigation. At this time, however, it is important to recognize that hypokinetic dysarthria is the only dysarthria type in which rate is perceived as, or actually is, rapid or accelerated. The caveat, as Weismer¹²³ and Kent and Rosenbek⁶³ have suggested, is that the perception of fast rate could be an artifact of features such as articulatory imprecision and continuous voicing that reduce discrete acoustic contrasts; this "blurring" of acoustic contrasts may lead to a perception of increased rate. Some experimental support for this derives from a study by Torp and Hammen¹²⁰ that determined that speaking rate was perceived as faster in PD than control speakers even when actual speaking rates were equivalent. This supports the notion that things within the speech signal can lead to a perception of rapid rate when rate may not actually be rapid. These insights suggest that clinicians may need to tune more finely their perceptual judgments when assessing speech rate in hypokinetic dysarthria.

4. *Strength and endurance.* It appears that weakness (CNS, not peripheral nervous system [PNS]), in addition to rigidity, may contribute to reduced range of movement in hypokinetic dysarthria. Structures in which weakness (and sometimes reduced endurance) have been found include the upper lip and (probably) velum,⁹⁹ as well as the tongue.¹¹²⁻¹¹⁴ Solomon et al.¹¹⁴ found that a group of patients with mild to moderate PD had reduced tongue strength but not abnormal fatigue when asked to exert maximum lingual pressure on an air-filled bulb (the Iowa Oral Performance Instrument, as defined in Chapter 3). Others have also found evidence of fatigue or reduced tongue endurance.^{112,113}

The presence of weakness and fatigue are not necessarily linearly related to abnormal perceptual speech characteristics. For example, Solomon, Robin, and Luschei¹¹² found no significant relationship between tongue strength and endurance and articulatory precision and overall speech defectiveness, suggesting that modest degrees of tongue weakness and fatigue may not be associated with perceptible (or otherwise measurable) speech deficits. The authors noted that the operating range for speech muscles is approximately 10% to 25% of their maximum strength, so the threshold for weakness to result in functional impairment may not be a straightforward value; tongue strength may need to be impaired beyond a critical level before deficits in speech become evident.

5. *Tremor, control, and steadiness.* Hunker and Abbs⁵⁶ found evidence of pathologic tremor in the jaw and lip at rest, during sustained postures, and during active and passive movement. They speculated that prolonged reaction times (i.e., delayed initiation of movement) in PD may be due to an inability to initiate muscle contraction until it coincides with the involuntary burst of a tremor oscillation, and that tremor rate may set limits on maximum rates of syllable production that can be attained without acceleration. Abbs, Hunker, and Barlow¹ also observed lip and jaw tremor during nonspeech tasks involving muscle force. The lips and jaw were otherwise adequate in producing stable forces, although patients had difficulty producing stable tongue elevation forces. Putnam,¹⁰³ summarizing the relevant literature, noted that tremor may be involved in acceleration

phenomena in hypokinetic dysarthria, and that patients may have to contend with the effect of tremor on phasic movements during speech.

There is evidence that at least some speakers with hypokinetic dysarthria have reduced oromotor control and reduced steadiness in orofacial structures during speech and nonspeech tasks. For example, McClean, Beukelman, and Yorkston⁹⁰ found evidence of poor visuomotor tracking of a sinusoidal signal with both jaw and lip movements. Zwirner and Barnes¹²⁹ found acoustic evidence of decreased jaw stability (as reflected in first formant steadiness) during vowel prolongation. Adams,⁶ summarizing the results of relevant studies, noted that PD patients have increased instability on isometric oromotor force tasks, and that such instability may vary across orofacial structures (tongue, lip, jaw), perhaps as a function of the degree of tremor in each structure.

Stress, Pause, and Other Durational Characteristics

Findings of rate abnormalities and reduced frequency and intensity variability help explain some of the acoustic factors underlying the perception of prosodic insufficiency in hypokinetic dysarthria. Some additional factors, mostly related to stress, pause, and between-syllable durational differences, help to round out the disorder's prosodic features.

Although Canter¹⁹ found no differences between parkinsonian and control subjects in number of pauses or mean pause duration during reading, several studies have found such abnormalities. Parkinsonian subjects' pauses during speech have been shown to represent a higher percentage of the total time within speech samples.^{44,91} Pauses may also occur slightly more frequently.⁴⁴

Illes et al.⁵⁸ found an increased frequency and duration of pauses that exceeded 200 ms. These hesitations or pauses tended to be longer and occur more frequently at the beginning of sentences. There was also an increase in number of words between silent intervals, a finding that may be related to the perception of short rushes of speech. Finally, Ludlow and Bassich⁸⁵ found reduced differences in word boundary durations between separate nouns and compound nouns (e.g., the boundary between the syllables "sail" and "boats" in the sentences "They were sailboats" vs. "They will sail boats"). This was correlated with the perception of reduced stress.

Murry⁹⁸ examined the ability to vary stress in the word initial and final position when answering questions with a standard sentence that established the point of emphasis (e.g., responding "Bob bit Todd" in response to the question, "Who bit Bob?").

Normal subjects tend to increase frequency, intensity, and articulatory effort (e.g., as measured by peak intraoral pressure) to signal stress in the word initial or final position. Murry's hypokinetic speaker demonstrated only minimal increases in frequency and intensity to signal stress, and this occurred at the expense of articulatory effort. Illes et al.⁵⁸ found that hypokinetic speakers exhibited fewer interjections and "modalizations" (comments that bear on verbal behavior, such as "you know") during narrative speech. Combined with other findings, this suggests that hypokinetic speakers display silent pauses instead of fillers, and that this loss of verbal "asides" may be analogous to a reduction of the automatic movements that accompany purposeful movements in PD (e.g., masked facial expression, reduced arm swing during walking).

Kent and Rosenbek⁶³ have provided a useful summary of the acoustic "signature" of hypokinetic dysarthria. They label the pattern, in which the contour across syllables within utterances is flattened or indistinct, as *fused*. This fused or flattened profile is characterized by (1) small and gradual f_0 and intensity variations within and between syllables, (2) continuous voicing, (3) reduced variations in syllable durations, (4) syllable reduction, (5) indistinct boundaries between syllables because of faulty consonant articulation, and (6) a spread of nasalization across consecutive syllables. In general, these features represent a reduced ability to use the full range of pitch, intensity, articulatory, and durational options that are used by normal speakers (see Figure 7-4).

Sensory and Perceptual Deficits

People with PD and hypokinetic dysarthria may have sensory or perceptual difficulties that impact on speech production. For example, there is evidence that some individuals with PD have poor temporal discrimination for tactile, auditory, and visual stimuli.⁹ Dagenais, Southwood, and Mallonee,²⁵ examining responses to delayed auditory feedback (DAF) in PD speakers, concluded that "they may have reduced resources to monitor and produce speech concurrently." Forrest et al.⁸⁸ found that PD speakers had below normal word identification scores when words were spoken at a slower than normal rate. They suggested that perceptual deficits might be additional factors that contribute to rate variations in PD speech. These findings have implications for the management of hypokinetic dysarthria and are discussed further in Chapter 17.

The general observations derived from the acoustic and physiologic studies reviewed in this section are summarized in Table 7-6.

table 7-6 Summary of acoustic and physiologic findings in studies of hypokinetic dysarthria*

<u>Speech Component</u>	<u>Acoustic or Physiologic Observation</u>
Respiratory (or respiratory or laryngeal)	Reduced: Vital capacity Amplitude of chest wall movements Strength & endurance Airflow volume during vowel prolongation Intraoral pressure during AMRs Syllables per breath group Maximum vowel duration Increased: Respiratory rate Latency to begin exhalation Latency to initiate phonation after exhalation initiated Breath groups during reading Percentage of vital capacity per syllable Irregular breathing patterns Paradoxical rib cage & abdominal movements Difficulty altering automatic breathing patterns for speech Poor respiratory control for visuomotor tracking Bowed vocal folds in spite of solid, nonflaccid appearance Tremulousness of arytenoid cartilages Asymmetry of laryngeal structures & movements during phonation, especially in hemiparkinsonism Ventricular fold movement during phonation Decreased: Intensity Pitch & loudness variability Speed to initiate phonation Intensity peaks across syllables Maximum phonation time over disease course Increased: f_0 & long-term variability of f_0 Glottal resistance & subglottic pressure TA & CT activity (cocontraction)
Laryngeal	Laryngealization Shimmer & jitter Voice tremor & flutter Continuous voicing in segments with voiceless consonants Voiceless transitions from vowels to following consonants Poor pitch control for visuomotor tracking Abnormal long-term average spectrum shape Increased nasal airflow during nonnasal target productions Reduced velocity & degree of velar movement during speech Abnormal spread of nasalization across syllables Decreased: Amplitude & velocity of lip movement Amplitude & duration of lip muscle action potentials Jaw stability during vowel prolongation Tongue endurance & strength Spectrographic acoustic contrast & detail Speech rate Ability to increase rate on request F1 & F2 formant transition rates Syllable boundary durational differences between separate & compound nouns F_0 , intensity, & articulatory effort increases to signal stress Variation in syllable duration
Velopharyngeal	
Articulation or Rate or Prosody	

Table 7-6 Summary of acoustic and physiologic findings in studies of hypokinetic dysarthria*—cont'd

Speech Component	Acoustic or Physiologic Observation
	Increased or accelerated: Connected speech & AMR rates Rate variability Frequency & duration of pauses during connected speech Articulatory undershoot in lip & velum Lip rigidity or stiffness Poor maintenance of temporal reciprocity between jaw depressors & elevators Poor visuomotor tracking with jaw & lip movements Abnormal jaw & lip tremor at rest, during sustained postures, & active & passive movement Spirantization of stops & affricates Continuous voicing Indistinct boundaries between syllables Spread of nasalization across syllables Spread of nasalization across syllables Small & gradual f_0 & intensity variations within & between syllables

AMR, Alternate motion rates; CT, computed tomography; f_0 , fundamental frequency; TA, thyroarytenoid.

*Many of these observations are based on studies of only one or a few speakers, and not all speakers with hypokinetic dysarthria exhibit these features. These characteristics may not be unique to hypokinetic dysarthria; some may also be found in other motor speech disorders or nonneurologic conditions.

Cases

Case 7-1

A 69-year-old man presented with a 4-year history of progressive difficulty getting in and out of chairs and a 2- to 3-year history of speech difficulty. These had progressed to include slowness in walking and poor handwriting.

Neurologic examination disclosed generalized bradykinesia, some rigidity of the trunk and limbs, and abnormal pursuit and saccadic eye movements. He could barely walk and did so in slow shuffling steps. "Severe speech hesitancy" was also apparent.

During speech examination, the patient described himself as "stuttering" as a child, beginning at 3 years of age and resolving by his ninth year. This problem was mild, and he had never had treatment for it. However, he noted that throughout his life he would "stutter" when excited, although his family had never noticed this. The patient had a brother who also reportedly stuttered as a child, with occasional dysfluencies in adulthood.

The tongue was tremulous on protrusion and during lateral movements. No other abnormalities were noted. Conversational speech, reading, and repetition were characterized by a remarkable degree of dysfluency, characterized by rapid repetition of initial sounds, syllables, and occasionally words and phrases. Sound and syl-

lable repetitions occurred up to 30 to 40 repetitions per dysfluent moment. There was no evidence of associated struggle behavior during dysfluencies, although he did express frustration over them. In addition to the dysfluencies, articulation was moderately imprecise, pitch and loudness variability was reduced, and overall loudness was mildly reduced. Speech AMRs were rapid or accelerated. Prolonged "ah" was hoarse (2).

The clinician concluded: "(1) Hypokinetic dysarthria. (2) Marked to severe stuttering-like behavior associated with CNS disease, including some dysfluencies suggestive of palilalia. I strongly suspect the dysfluencies reflect a component of his hypokinetic dysarthria. In my opinion, this is a variant of hypokinetic dysarthria with associated dysfluencies and does not reflect the reemergence of his reported childhood stuttering."

During the patient's few days at the clinic, speech therapy was undertaken, primarily to modify his dysfluencies. Hand tapping and use of a pacing board were unsuccessful because his limb movements were as accelerated or rapid as his speech. He did, however, respond positively to DAF, with a significant reduction in speech rate and marked reduction of dysfluency. This greatly enhanced efficiency and intelligibility during

Case 7-1—cont'd

conversation. The patient left the clinic with a recommendation to pursue therapy, with consideration given to acquiring a DAF device for use in conversation.

The neurologist concluded that the patient had idiopathic PD.

Commentary. (1) Hypokinetic dysarthria may be among the prominent presenting signs of PD. (2) Dysfluencies occur commonly in hypokinetic dysarthria, and palilalia may also occur. For some patients, their dysfluencies may be the most debilitating component of their

hypokinetic dysarthria. (3) The history of early childhood stuttering was of unknown significance in this case. However, recognizing that the patient had a hypokinetic dysarthria with associated dysfluencies helped establish that his speech deficit could probably not be attributed to a reemergence or persistence of childhood stuttering. Rather, it was related to the patient's neurologic disease. (4) Marked dysfluencies associated with hypokinetic dysarthria can be responsive to speech therapy. These approaches are discussed in Chapter 17.

Case 7-2

A 69-year-old man presented with a 5-year history of difficulty getting in and out of chairs, stiffness during walking, and difficulty turning in bed. He also had voice and handwriting difficulty. There was no history of encephalitis, toxic exposure, or drug use that might be related to his symptoms, nor was there any family history of neurodegenerative disorder.

On neurologic examination, the arm swing was diminished and the neck and extremities were rigid. He had a mild static tremor of the left hand, and upper limb movements were bradykinetic. Facial expression was masked, and postural reflexes were mildly impaired. An MRI scan was normal. He was referred for a speech assessment "to see if there are any clues in his voice as to the type of problem that he has."

During speech examination, the patient described a 1-year history of uncertainty if "words would come out." He believed his speech had become quieter and perhaps slower, more so in the evening or after extended speaking. He reported occasional difficulty "getting going" with his speech, even though he knew what he wanted to say.

The jaw, lips, and tongue were mildly tremulous during sustained postures. Breathy-hoarse voice quality

(2), reduced loudness (1), and a tendency toward accelerated rate (0,1) characterized speech. Very infrequently, there were rapid repetitions or prolongations of initial phonemes. There was some nasal emission during production of pressure sound-filled sentences, but he was not obviously hypernasal. Speech AMRs were normal. Prolonged "ah" was breathy-hoarse (1.2). Speech did not deteriorate during stress testing.

The speech clinician concluded "hypokinetic dysarthria, mild."

The neurologist concluded that the patient had parkinsonism. However, his symptoms were unresponsive to Sinemet. Because of this, the neurologist believed he might have striatonigral degeneration, "which can appear much like PD at onset but is not Sinemet responsive."

Commentary. (1) Speech change is often associated with parkinsonism and may be among the signs encountered during initial neurologic evaluation. (2) Changes in voice quality and loudness are often among the initial complaints of patients with hypokinetic dysarthria. (3) Identification of hypokinetic dysarthria can provide confirmatory evidence for a diagnosis of parkinsonism.

Case 7-3

A 74-year-old woman presented with a 4-year history of progressive "wobbling" when walking and a tendency to fall backward. Neurologic examination initially suggested prominent proximal muscle weakness. Polymyositis, myasthenia gravis, and myopathy were suspected. Because she complained of "slurred" speech and "hesitation" when speaking, she was referred for speech assessment.

During speech examination she stated, "When I speak, I don't know how it will come out. Sometimes words do not come out at all." Conversational speech was characterized by prolonged silent intervals, occasional whole word repetitions, and repeated syllables (e.g., "I took dic-ta-ta-ta-tion from him"). Rate was mildly accelerated, and articulation was often mildly imprecise, with slighting of consonants when she spoke rapidly. Resonance was normal, but voice quality was harsh. There was no evidence of speech deterioration during 4 minutes of continuous talking.

The clinician concluded, "Speech features are most suggestive of hypokinetic dysarthria. At times, the

pattern is almost that of palilalia, also seen in parkinsonian patients. This is not a speech pattern of flaccid dysarthria; no suggestion of myasthenia gravis."

The speech diagnosis prompted additional neurologic investigation. CT scan was normal with the exception of mild generalized cerebral atrophy. Consultation with other neurologists ruled out PNS disease and myopathy and detected postural instability, slight rigidity, and brisk reflexes. Neurologic diagnosis was uncertain, but it was concluded that she had several parkinsonian symptoms but without classic idiopathic PD. A diagnosis of PSP was entertained, but evidence for its diagnosis was considered equivocal.

Commentary. (1) Hypokinetic dysarthria is common in parkinsonism. (2) Diagnosis of hypokinetic dysarthria can be helpful to neurologic diagnosis. In this case, it raised suspicions about CNS degenerative disease, specifically parkinsonism. It helped focus attention on the CNS as opposed to the PNS. (3) Dysfluencies and palilalia can be associated with hypokinetic dysarthria.

Case 7-4

A 29-year-old woman presented to a rehabilitation unit 14 months after cerebral anoxia that developed secondary to cardiac arrest during a tubal ligation. Neurologic examination revealed neck and left upper extremity rigidity, upper extremity dystonia, diffuse hyperactive reflexes, and weakness in all extremities. Gait was slow with short steps. She had difficulty with chewing and swallowing and frequently choked on solid foods.

Speech examination revealed reduced loudness (3), imprecise articulation (3), accelerated speech rate (3), little variation in pitch, loudness, and syllable duration, and reduced range of articulatory movement (3,4). Speech AMRs were "super fast and blurred."

The clinician concluded "hypokinetic dysarthria, severe." There was no evidence of aphasia, but neuropsychological assessment revealed deficits in attention, concentration, new learning, and short-term recall. She received speech therapy while at the rehabilitation unit, with subsequent improved speech intelligibility as long as she was cued to increase loudness and slow rate.

Commentary. (1) Hypokinetic dysarthria can occur in conditions other than parkinsonism and can be encountered in anoxic encephalopathy. In such cases the dysarthria may not be distinguishable from hypokinetic dysarthria associated with idiopathic PD. (2) Cognitive deficits can be present in individuals with hypokinetic dysarthria.

Case 7-5

A 75-year-old man presented with a 3- to 4-year history of shuffling gait, stooped posture, loss of facial expression, tremor, and voice change. Neurologic examination confirmed the presence of these symptoms. The patient also admitted to occasional confusion and reduced memory. Neuropsychological assessment revealed mild to moderate generalized, organic cognitive decline consistent with mild dementia.

During speech examination, the patient complained of "hoarseness" and noted that his voice occasionally "gets to a whisper." Voice quality was characterized by reduced loudness (1,2), continuous breathiness (1,2), and monopitch and monoloudness (1,2). Articulation was equivocally fast during conversation. Speech AMRs were normal.

The clinician concluded, "Mild hypokinetic dysarthria, primarily characterized by reduced pitch and loudness variability, reduced volume, and breathiness."

Because the patient cleared his throat frequently and had prominent dysphonia, he was referred for laryngeal examination; bowing of the vocal folds was observed.

The neurologist concluded that the patient had a degenerative CNS disease that did not fit well with classic idiopathic PD. Sinemet was prescribed. Two years later, although improved on Sinemet, the neurologic examination was unchanged and there was no other evidence of deterioration. Speech was also unchanged, with the exception that tongue tremulousness was apparent on protrusion.

Commentary. (1) Hypokinetic dysarthria frequently manifests as dysphonia and prosodic insufficiency. Such difficulties can remain the only speech symptoms for extended periods. (2) The dysphonia of hypokinetic dysarthria is frequently associated with bowing of the vocal folds. (3) People with hypokinetic dysarthria can also have cognitive impairments.

Case 7-6

A 51-year-old man presented for another opinion about his neurologic deficit. His difficulties began 3 years previously, over approximately 10 days, when he had several suspected myocardial infarctions. His symptoms at that time included speech difficulty and problems with gait.

Neurologic examination showed a loss of facial expression, generalized loss of associated movements, generalized bradykinesia, and generalized rigidity, greater on the left than right side.

During speech examination, the patient stated, "I can't talk in long sentences; I repeat myself; bad volume; out of breath fast." He had had three periods of speech therapy, benefiting only temporarily from each.

Examination revealed facial masking, reduced range of movement of the jaw, lips, and tongue, and, perhaps, mild left tongue weakness. Connected speech was characterized by imprecise articulation (3), accelerated rate within utterances (2,3), monopitch and monoloudness (3,4), and breathy-harsh-strained voice quality (2,3). In addition, during conversation he exhibited numerous phoneme and syllable repetitions and fairly frequent word and phrase repetitions, usually with associated accelerated rate, consistent with the characteristics of palilalia. At times, however, these repetitions appeared voluntary, based on his perception that he had not been understood, while at other times they appeared involuntary. Speech AMRs were markedly imprecise and

blurred. Intelligibility was significantly reduced but improved with slowing of rate, which was facilitated by hand tapping.

The clinician concluded, "Marked hypokinetic dysarthria and palilalia."

It was recommended that he resume speech therapy. It was felt that he might benefit from efforts to more consistently slow his rate and prepare himself respiratorily for each utterance. Development of a backup augmentative system was also recommended. The patient had been under the impression that speech therapy was intended to completely remediate his speech difficulty. During a lengthy discussion, it was stressed that speech therapy would not likely restore normal speech but could focus on maximizing intelligibility. The patient accepted this explanation, with disappointment, and did pursue additional speech therapy at a facility near his home.

Additional neurologic workup included an MRI scan that identified small lacunar infarcts in the right putamen and external capsule. The neurologist concluded that the patient had extrapyramidal disease as a result of a previous cerebrovascular event and, perhaps, diffuse cerebral ischemia that was secondary to an episode of hypotension of undetermined etiology. Although clinical findings were somewhat asymmetric and only a unilateral lesion was present on neuroimaging, the clinical picture appeared to reflect bilateral involvement of the basal ganglia.

Case 7-6—cont'd

Commentary. (1) Hypokinetic dysarthria and palilalia can result from cerebral ischemia and infarction. (2) Hypokinetic dysarthria can be among the most debilitating deficits stemming from basal ganglia disease. (3) Although neuroimaging evidence suggested only a unilateral lesion, the dysarthria and associated neurologic findings were strongly suggestive of bilateral involve-

ment. (4) Dysarthric patients sometimes have unrealistic expectations about speech therapy. It is crucial that patients understand the goals of therapy when therapy is recommended. Counseling in this regard is helpful to managing patients' acceptance and understanding of their deficits, as well as to develop an understanding of what may and may not be achieved with treatment.

SUMMARY

1. Hypokinetic dysarthria results from damage to the basal ganglia control circuit. It probably occurs at a rate comparable to that of other single dysarthria types. Its characteristics are most evident in voice, articulation, and prosody. The effects of rigidity, reduced force and range of movement, and slow individual and sometimes fast repetitive movements seem to account for many of its deviant speech characteristics.
2. Parkinsonism, the prototypic condition associated with hypokinetic dysarthria, is most often due to PD, a degenerative condition associated with a depletion of dopamine in the striatum of the basal ganglia. Several symptoms of the disease are often managed by medications that restore the balance between dopamine and acetylcholine within the basal ganglia. Several other neurodegenerative diseases may also cause parkinsonian symptoms and hypokinetic dysarthria.
3. Hypokinetic dysarthria may also result from nondegenerative conditions, most often including vascular disease, neuroleptic and illicit drugs, certain metabolic diseases, chronic exposure to heavy metals, trauma, and infection.
4. Patients or, more often, their significant others frequently complain that their voice is weak or quiet, and sometimes that their rate is too rapid. They may also note dysfluencies and difficulty initiating speech. They often are aware of deterioration with fatigue or toward the end of an antiparkinsonian medication cycle. Drooling and swallowing complaints are common. Facial masking and a general reduction in the visible range of articulator movement during speech are common.
5. Several deviant speech characteristics combine to give many patients a distinctive flat, attenuated, and sometimes accelerated speech pattern. This has been called prosodic insufficiency and is characterized by monopitch, monoloudness,

- reduced stress, short phrases, variable rate, short rushes of speech, and imprecise articulation. Additional distinctive characteristics that may be present include inappropriate silences, breathy dysphonia, reduced loudness, and increased speech rate. Dysfluencies and palilalia may also be present.
6. In general, acoustic and physiologic studies have provided support for the auditory-perceptual characteristics of the disorder; have specified more precisely the disorder's acoustic and physiologic characteristics; and have documented the role of rigidity, reduced range of movement, slowness of movement, and acceleration phenomena during speech. Data suggest that the perception of accelerated rate may sometimes be an artifact of listener expectations and reduced acoustic contrast.
 7. Hypokinetic dysarthria can be the only, the first, or among the first and most prominent manifestations of neurologic disease. Its recognition can aid neurologic localization and diagnosis and may contribute to the medical and behavioral management of the individual's disease and speech disorder.

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