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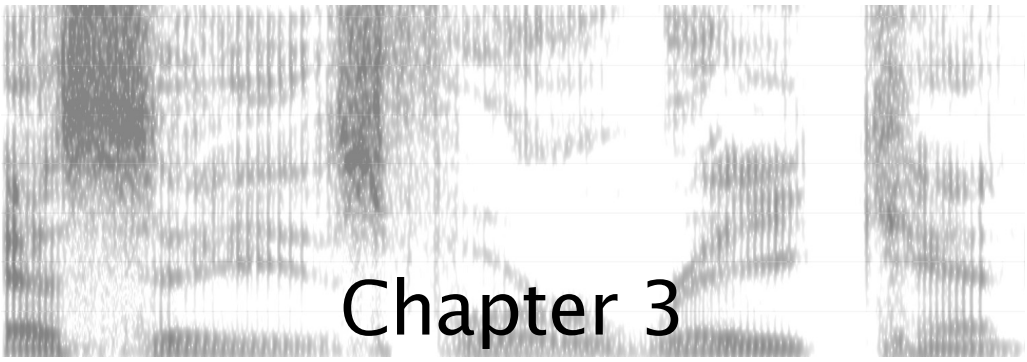

Chapter 3

NEURAL PERSPECTIVES ON MOTOR SPEECH DISORDERS

Current Understanding

Gary Weismer

This chapter presents a neuroanatomic and *functional neurologic* framework for the student of motor speech disorders, but does not present detailed information typically covered in a chapter on the neurologic underpinnings of speech production. For example, material on the anatomy and physiology of neurons, the structure and variation of motor units, and the detailed structure of fiber tracts and nuclei is assumed to be generally familiar or easily accessible to readers. Rather, the focus of this chapter is on a model of sensorimotor structures, pathways, and functions of the brain that can be referenced to the Mayo classification system described in Chapter 2 and summarized below under *Preliminaries*. Students interested in alternative presentations of material on brain mechanisms for speech should consult Duffy (2005, Chapter 2), Kent and Tjaden (1997), and an older text by Kuehn, Lemme, and Baumgartner (1989).

The organization of this chapter is as follows. First, a model of brain structures relevant to sensorimotor control is presented, accompanied by a general discussion of what the term "speech motor control" means in this text. Parts of the model are then taken one at a time and discussed in further detail not only in terms of general structure and function but also with respect to possible pathophysiology and its effect on speech production. Throughout the chapter, components of the traditional speech motor examination are described and discussed.

The Model: A Brief Description

Figure 3–1 is a box-and-arrows diagram of the parts of the brain thought to be involved in sensorimotor control in general,

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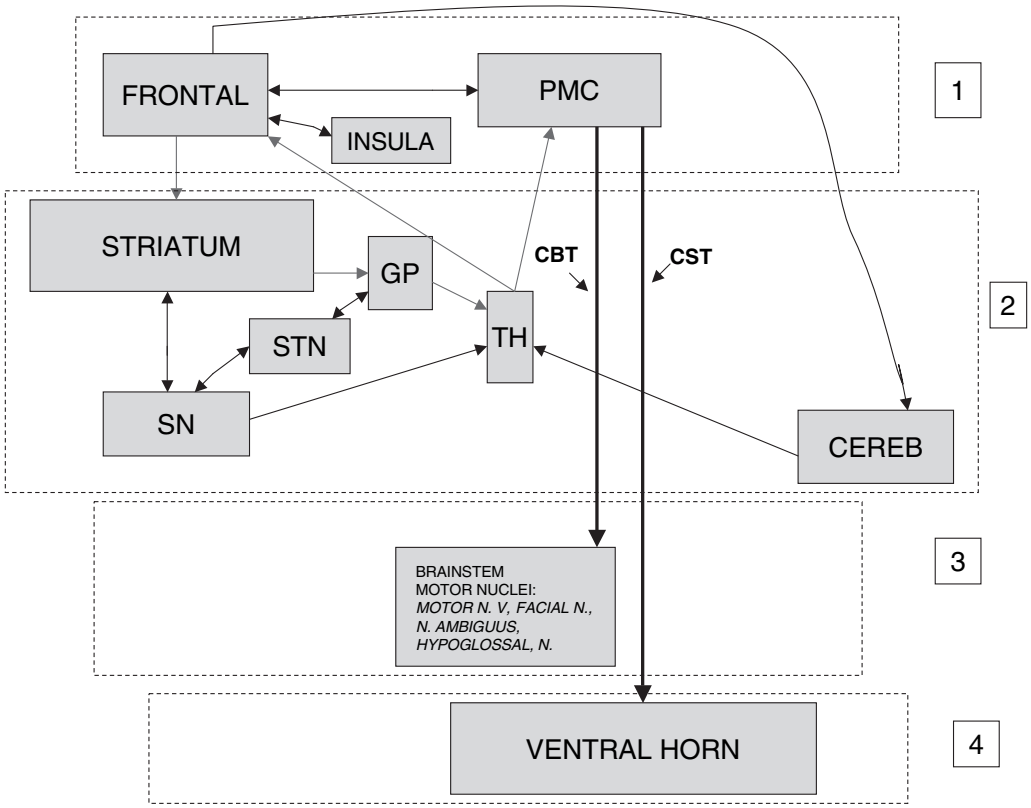

Figure 3–1. Box-and-arrows schematic diagram of central nervous system (CNS) structures involved in sensorimotor control. Section 1 contains cortical structures (PMC = primary motor cortex); Section 2, the basal nuclei and cerebellum (GP = globus pallidus; SN = substantia nigra; STN = subthalamic nucleus; TH = Thalamus; CEREB = cerebellum); Section 3, the brainstem motor nuclei; Section 4, the ventral horn of spinal cord. CBT = corticobulbar tract; CST = corticospinal tract. Single-headed arrows indicate fiber tracts going to the structure where the arrowhead points; double-headed arrows indicate pairs of structures with fiber tracts going in both directions. See text for additional details.

and speech motor control in particular. Two general points about this diagram and its interpretation should be made at the outset of this discussion. First, we use the term *sensori*motor control to designate the likely role of different forms of sensory information in shaping and maintaining "good" motor behavior. The most obvious case for speech is auditory and tactile/proprioceptive information (e.g., in the latter case, tongue-palate contact in the accurate production of certain high vowels and many consonants), but there are other more subtle forms of sensory information that enter into the production of smooth, accurate movements. Second, much of our current information on sensorimotor control is based on studies of limb and hand (paw) movements produced by humans and animals. Knowledge of the

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precise mechanisms of *speech* motor control lags that of limb motor control—and even orofacial control in animals—precisely because there is no animal model of speech production. Much of what is currently thought to be known about speech motor control, therefore, is inferential, based on "natural" experiments in which humans suffer localized brain damage and have speech production deficits specific to the affected structures. In fact, as reviewed in Chapter 2 and discussed below, this is the conceptual basis of the Mayo Clinic classification system of dysarthria. Knowledge of speech motor control mechanisms is increasing with brain imaging techniques. One of the claims from this work, in its infancy, is that speech motor control mechanisms in the brain are widely distributed across many structures. The possibility of speech production requiring diverse and sometimes unexpected brain mechanisms is reflected by the several cortical mechanisms shown in Figure 3–1. To add to the complication but to make a clinically relevant point, the precise mechanisms involved in speech production may depend on the kind of speech being produced (Blank, Scott, Murphy, Warburton, & Wise, 2002).

It is useful to separate the model into four general sections, as shown by the dashed-line rectangles in Figure 3–1. Sections 1 and 2 include cortical and subcortical structures known to be important to motor control. The cortical "boxes" in the model represent several areas, including the primary motor cortex, the premotor cortex, the supplementary motor area and Broca's area, the latter three gathered in the box labeled "Frontal," and parts of the insular cortex. Not shown among the cortical areas are the primary sensory cortex and Wernicke's area, both of which have been implicated in some studies as active during speech production. Cells in the primary motor cortex and the premotor cortex receive either direct or indirect connections from the other cortical areas. Primary and premotor cortex send axons to make direct connections to the motor cells in the brainstem and spinal cord. The corticobulbar tract (CBT in Figure 3–1) makes these connections between cortex and brainstem, the corticospinal tract (CST in Figure 3–1) between cortex and spinal cord. These direct connections carry the processed and integrated output of many sources of cortical and subcortical activity. Note also the lack of specification for the hemisphere affiliation of the cortical areas shown in Figure 3–1. It is, of course, well accepted that for most individuals the left hemisphere is dominant for speech and language production and reception, but both hemispheres show activity during speech production as would be expected because many of the muscles of the head and neck receive bilateral innervation (see below) from the cortical primary and premotor areas.

Section 2 of the brain model in Figure 3–1 shows the basal nuclei, the thalamus, the cerebellum, and connections between these structures. The doubled-headed arrows between certain structures indicate information flowing in both directions; these connections are described more fully below. The basal nuclei (often called the basal ganglia) include the caudate and putamen which together constitute the striatum, the substantial nigra, the subthalamic nucleus, and the globus pallidus (other structures not mentioned here are sometimes included as part of the basal nuclei). The thalamus is made up of many nuclei that relay information from several different locations throughout the

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brain and spinal cord to the cortex; sometimes it is called the main sensory relay for neural signals in transit to the cortex. The thalamus also contains several "motor nuclei" which are described later in this chapter. The cerebellum, a massive, phylogenetically older part of the brain located beneath the cerebral hemispheres and posterior to the brainstem, contains nuclei connected by large fiber tracts to many different parts of the brain.

Section 3 of the model includes nuclei in the brainstem associated with the cranial nerves that innervate head and neck muscles, as well as nuclei for audition. The motor neurons (neuron cell bodies connected to muscle fibers by means of an axon) associated with cranial nerves V (trigeminal), VII (facial), IX (glossopharyngeal), X (vagus), XI (accessory), and XII (hypoglossal), can be considered the final common pathways for motor control. In other words, signals originating in these brainstem motor neurons and conducted via the cranial nerves to muscles represent the combined influences of all cortical, subcortical, and cerebellar processing reflected in the descending input signal delivered to a motor nucleus in the brainstem. Auditory nuclei in the brainstem receive information from the auditory nerve, one of the two divisions of cranial nerve VIII (the other is the vestibular portion, important for balance and orientation in space). Auditory mechanisms are included in our model because they have a prominent role in our definition of speech motor control. In the simplified diagram of Figure 3–1, connections between the brainstem and other structures of the brain, including the cerebellum, subcortical nuclei, and spinal cord, have been omitted.

Finally, section 4 represents spinal motor neurons and their innervation of the muscles of respiration. The cell bodies of these motor neurons are located in the ventral horn of the spinal cord. As in the brainstem, they and the spinal nerves that issue from them can be considered the final common pathway to the muscles of the thorax, diaphragm, and abdomen.

The Model and Speech Motor Control

The model in Figure 3–1 is a schematic representation of the mechanisms involved in motor control in general, and speech motor control in particular. The following question can (and should) be asked, "Is a model of general motor control, based largely on studies of limb and hand behavior sufficient for and appropriate to an understanding of *speech* motor control?" For the purposes of this text, we believe the answer to this question is "no," even though principles of general motor control most certainly apply to aspects of speech motor control. Speech motor control is different from limb motor control because the goals of speech production appear to be the acoustic results of particular vocal tract shapes and changes in those shapes over time. In this conception of speech motor control, consistent with recent research and emerging theories of speech production behavior (e.g., Guenther, Hampson, & Johnson, 1998), the acoustic signal produced by the speech mechanism is part of the motor control process, not separate from it; this is why parts of our model shown in Figure 3–1 contain auditory processing components. As children are learning and refining the acoustic consequences of the changing configurations of the vocal tract they store

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these associations as a set of expectations, namely, that certain movements will produce certain acoustic consequences. The mature speaker uses this set of expectations as a form of quality control over her speech movements. If there is a mismatch between a movement and the acoustic consequence, some updating of this particular expectation would be required to re-establish the correct relationship. This perspective on the nature of speech motor control also explains why evaluation of the speech mechanism by oromotor, nonverbal tests (e.g., such as generating maximum strength efforts with the lips, jaw, or tongue, or wagging the tongue laterally at maximum speed) has limited application to the understanding of a *speech* motor control deficit: a task with no acoustic output, even if performed by parts of the speech mechanism, is not speech and therefore is subject to different control strategies and potential deficits. The acoustic output of the vocal tract is not the result of speech motor control, it is an integral part of it.

In the remainder of this chapter we consider each section of the model in Figure 3–1 in somewhat more detail, with emphasis on how damage to its components may assist in diagnosis of specific neurologic disease and how it may affect speech motor control. To "build up" the system, we begin with section 4, and work our way "up" the nervous system to the cortex. Because signs and symptoms of different types of neurologic damage are discussed within the framework of the model, some preliminary material is discussed to allow detailed consideration of the model. These preliminaries are standard concepts of neurologic description as available in any basic text on neurologic disease and diagnosis.

Preliminaries

This section presents some general concepts useful to understanding the link between neuroanatomy and neuropathology. These include the notion of *signs and symptoms* of neurologic disease, and the specific differences between upper and lower motor neuron disease. These specific differences, in fact, are mostly defined in terms of unique signs and symptoms. Finally, a brief review of the Mayo Clinic classification system of motor speech disorders is presented, stressing the presumed, underlying neuroanatomic damage associated with each of the categories in the system.

Signs and Symptoms

There is a technical difference between a sign and a symptom of a neurologic disease (or any disease). *Signs* are observable, by visual examination and in some cases through more formal testing. *Symptoms* are complaints made by patients when telling health care professionals about their problem. Signs and symptoms taken together typically constitute the basis for diagnosis of disease. A good example of a neurologic sign that is relevant to the current discussion is the patient who enters an SLP's office with feet widely spaced and a slightly staggering gait. As described below, this is a sign of cerebellar disease. A symptom of cerebellar disease might be the patient's complaint of frequently losing his or her balance without warning.

For the remainder of this chapter the terms signs and symptoms are used interchangeably or jointly, as in, "The signs and

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symptoms of Parkinson disease are tremor, rigidity, and bradykinesia." This recognizes that certain symptoms may technically become signs when they are observed (e.g., the observation of a sudden loss of balance by a patient with cerebellar disease is technically both a sign and, by the patient's report, a symptom).

Classical Signs of Neurologic Disease

The concept of "classical signs of neurological disease" is central to an understanding of motor speech disorders as categorized within the Mayo classification system. Different neurological diseases are often associated with unique signs and symptoms, the latter most typically based on limb characteristics. For example, when a patient has damage to the fiber tracts connecting cortical cells with motor neurons in either the brainstem or spinal cord, the lesion is said to be in the upper motor neuron (explained in detail immediately below). Patients with this kind of damage often have a group of limb signs/symptoms that include an excess of tone, overactive reflexes, and weakness. These classical symptoms of upper motor neuron disease, although based on limb characteristics, are used in the Mayo classification system to "explain" the speech characteristics of spastic dysarthria, the kind of dysarthria typically seen in persons with upper motor neuron damage. This is a case, then, where limb motor characteristics are taken as directly applicable to speech motor control. As stated earlier, this is a controversial and unproven aspect of the discipline of motor speech disorders. The concept of "classic" signs/symptoms of neurologic diseases as based primarily on limb characteristics should be kept in mind for the remainder of this chapter.

Upper Versus Lower Motor Neuron

The terms *upper* and *lower motor neuron* are used to describe locations of structures within the nervous system, as well as disease types, as in *upper motor neuron disease* or *lower motor neuron disease*. The terms can be explained with reference to the simple schematic diagram in Figure 3–2. This drawing shows boxes with connecting lines, the boxes representing cell groups and the lines representing fiber tracts running between the cell groups. The top two boxes represent motor cells of the cortex in the right and left hemispheres; for the sake of simplicity, we assume these cells to be located in primary motor cortex. The middle two boxes represent motor nuclei in the brainstem, which contain the motor neurons that innervate muscles of the head and neck. The two boxes represent the two sides of the brainstem; all motor nuclei (and sensory nuclei) in the brainstem are paired, with one on the left and the other on the right side. These nuclei, as listed in Figure 3–1 and described below, include the motor nucleus of V, the facial motor nucleus, the nucleus ambiguus, and the hypoglossal nucleus. Finally, the lower two boxes represent the motor neurons in the ventral horn of the spinal cord. These cells send axons to muscles of the limbs and respiratory system, as well as other muscles of the trunk.

The tracts in Figure 3–2 include those that connect cortical motor cells with motor neurons in the spinal cord. This is

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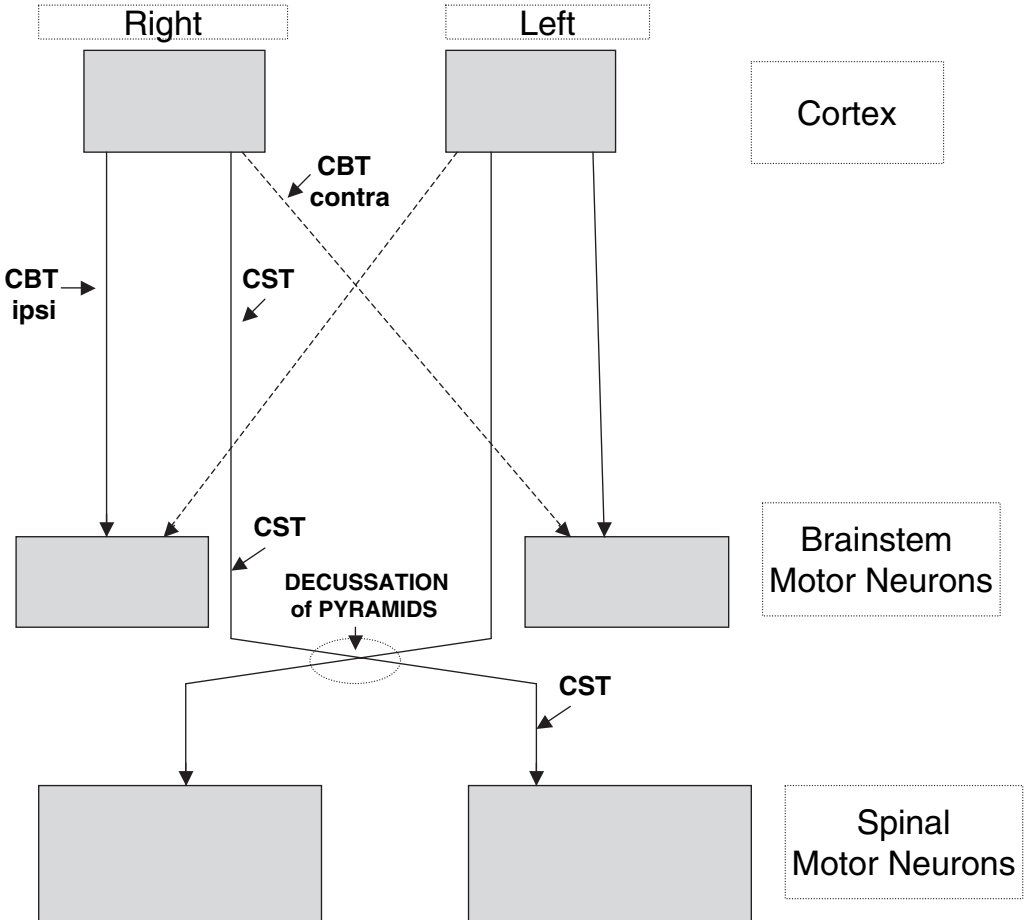

Figure 3–2. Box-and-arrows schematic diagram showing simple way to understand the difference between upper and lower motor neuron. Top two boxes represent cortical cells in primary motor cortex, middle two boxes the brainstem motor neurons, and the bottom two boxes the motor neurons in the ventral horn of the spinal cord. CST is the corticospinal tract, the fibers of which cross from the cortical side in which they begin to the opposite side to make synapses with the motor neurons in the ventral horn; the crossover point is in the lower medulla. A lesion in the cortex where the CST begins or anywhere in the tract before the synapse with the ventral horn motor neurons is an upper motor neuron lesion; a lesion in the ventral horn motor neurons or of the nerves issuing from them to the muscles they innervate is a lower motor neuron lesion. CBT ipsi is part of the corticobulbar tract that runs from one side of the cortex to the same side of the brainstem motor neurons; CBT contra is part of the corticobulbar tract that runs from one side of the cortex to the opposite side of the brainstem motor neurons. A lesion in the cortex or these tracts before the synapse with the brainstem motor neurons is an upper motor neuron lesion; a lesion in the brainstem motor neurons or the nerves issuing from them to the muscles they innervate is a lower motor neuron lesion.

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shown in Figure 3-2 by the line labeled CST, which stands for *corticospinal tract*; the line is labeled only on a single side, but it can be seen that the tract runs on both sides of the brain. This tract, which descends from each hemisphere first as the *corona radiata*, is then gathered into a tighter bundle called the *internal capsule*, which enters and passes through the brainstem as the *cerebral peduncles* and eventually forms the columnlike *pyramids* on the ventral surface of the medulla. At the base of the medulla the great majority of fibers from one hemisphere cross over to the other side and continue running down this side of the spinal cord, giving off fibers along the entire length of the cord to the motor neurons in the ventral horn of the central gray matter. The crossover of the corticospinal tracts is indicated in the schematic drawing by the small, dotted line oval placed at the bottom of the brainstem level; the crossover point is called the *decussation of the pyramids*. This contralateral innervation of spinal motor neurons explains why a stroke that damages the left cerebral hemisphere will result in weakness or paralysis of limb muscles on the right side of the body, and vice versa.

With an understanding of how the corticospinal tract connects motor cells in the cortex with those of the spinal cord, the difference between upper and lower motor neuron disease can be explained. When a lesion occurs above a motor neuron in the spinal cord—that is, in the cortex, or anywhere along the corticospinal tract prior to a synapse with a motor neuron—it is called an upper motor neuron lesion. Damage within the motor neuron (in the ventral horn of the spinal cord) or along the peripheral nerve connecting the motor neuron with a muscle is called a lower motor neuron lesion. The diseases produced by such lesions—upper motor neuron versus lower motor neuron disease—produce different sets of symptoms.

Symptoms of Upper Motor Neuron Disease

Upper motor neuron disease is likely to produce any or all of the following signs in muscles of the limbs: spasticity (a form of hypertonia), weakness, and hyperreflexia. In addition, some patients may show emotional lability.

Spasticity

Spasticity describes the characteristics of muscle tone when an examiner asks the patient to relax and then assesses the effects of passive displacement of a limb. For example, if the patient places her arm in front of her, slightly bent with the hand roughly at mid-torso level, the examiner can displace the arm away and toward the body and evaluate how much resistance it offers to the passive motion. A limb with normal tone will offer a small amount of resistance to passive displacement, but a limb with spasticity will offer a great deal of resistance when displaced away from the body, but not toward the body. The resistance to displacement may also be sensitive to the speed of passive displacement, with greater resistance as speed increases. Spasticity is a form of hypertonicity, or abnormally high muscle tone.

Weakness

Weakness needs little description; patients with upper motor neuron disease typically cannot produce strength efforts like those of neurologically healthy persons of comparable age, gender, and general health.

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Hyperreflexia

Hyperreflexia implies a heightened sensitivity of certain reflexes. A classic example of hyperreflexia relevant to orofacial mechanisms is the jaw-jerk reflex, elicited by tapping down on the chin while the mandible is slightly open and relaxed. A neurologically healthy individual will either have no obvious response or a very small, upward movement of the mandible in response to the tap. The patient with upper motor neuron disease may have an exaggerated jaw-jerk reflex, seen as a large upward movement of the mandible in response to the tap. Hyperreflexia of the jaw-jerk, together with certain other symptoms (see below) is sometimes used as one of the diagnostic signs for amyotrophic lateral sclerosis (ALS).

Emotional Lability

Emotional lability has been observed in some patients with upper motor neuron disease, especially following strokes that damage the internal capsule. In severe cases, patients may laugh and cry for no apparent reason, and when asked if they are sad (e.g., when crying) deny the feelings. In less severe cases the emotional reaction may be tied to a meaningful situation but may be exaggerated. The reasons for this symptom of upper motor neuron disease, seen in perhaps one quarter of patients who survive stroke and more often in the earlier phases of recovery, are not well understood.

Symptoms of Lower Motor Neuron Disease

Lower motor neuron disease is likely to produce any or all of the following effects in the muscles of the limbs: reduced muscle tone (hypotonia), atrophy (wasting), hyporeflexia, weakness, and fasciculations.

Reduced Muscle Tone, or Hypotonicity

This is a symptom revealed when an examiner passively displaces a limb, as described above. In this case, the limb offers an unusually low amount of resistance to passive displacement. In extreme cases a hypotonic limb may appear to offer no resistance to displacement, giving an impression of "floppiness."

Atrophy

Atrophy, sometimes referred to as *wasting*, is the loss of muscle tissue over time. Atrophy is typically not seen in upper motor neuron disease and so can become a distinguishing characteristic between upper and lower motor neuron disease. Atrophy occurs in lower motor neuron disease because the damage to the motor neuron or peripheral nerve interferes with or eliminates the production and transport of nutrients from the nerve cell to the muscle.

Hyporeflexia

This is the condition wherein reflexes observed in neurologically healthy individuals are either reduced in magnitude or completely absent.

Weakness

Weakness is a pervasive feature of lower motor neuron disease.

Fasciculations

Observed on the surface of a muscular structure at rest, fasciculations are small, local muscle twitches. These involuntary

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contractions of small bundles of muscle fibers create an appearance on a structure's surface of rising and falling bumps. Some fasciculations are normal (as in the common experience of eyelid twitches), but when paired with atrophy and weakness are indicative of lower motor neuron disease. In the speech mechanism, fasciculations are most often observed on the tongue surface of patients with lower motor neuron disease.

The tracts shown in Figure 3–2 also include the pair that connects cortical motor cells with motor nuclei in the brainstem. This is called the *corticobulbar tract* (labeled CBT in Figure 3–2), which first descends from the cortex within the *corona radiata*, is then gathered into a tighter bundle called the *internal capsule*, and enters the brainstem as the *cerebral peduncles,* which give off fibers to motor nuclei. Some of these fibers connect one side of the cortex with the motor nuclei in the brainstem on the opposite side; this *contralateral connection* (CBT-contra in Figure 3–2) is like that of the corticospinal tract, described above. Other fibers connect a side of the cortex with the brainstem motor nuclei on the same side; this is referred to as an *ipsilateral* connection (CBT-ipsi in Figure 3–2). There are many cases in which cells from both sides of the cortex connect to a motor nucleus in the brainstem via both an ipsilateral and contralateral fiber tract. A motor nucleus in the brainstem that receives such connections is said to be *bilaterally innervated* from the cortex. In a few cases, a motor nucleus in the brainstem, or a subset of the cells within a motor nucleus, will receive only contralateral connections via the corticobulbar tract. There are no reports in the literature of a corticobulbar tract having only ipsilateral connections with a paired motor nucleus in the brainstem (but see below, description of Accessory Nucleus and cranial nerve XI). A reasonable summary statement is that the motor nuclei in the brainstem receive either bilateral or contralateral innervation from the cortex, via the corticobulbar tract.

The definition of upper and lower motor neuron lesions for the corticobulbar tract and its target brainstem nuclei can be understood in exactly the same way as described above for the corticospinal tract. An upper motor neuron lesion will be in the corticobulbar tract, prior to the synapse in a motor nucleus of the brainstem; a lower motor neuron lesion will be in the brainstem motor nucleus or the nerve (cranial nerve) issuing from the nucleus to the muscle(s). To a first approximation, the signs and symptoms of upper versus lower motor neuron lesions are as described above, but with the proviso that the evaluation of certain aspects of muscle or structural function in the speech mechanism may be more difficult or complicated than in the limbs.

For example, determination of spasticity requires passive displacement of a structure, an easy task with the limbs but somewhat more challenging even with the more accessible structures of the speech mechanism such as the jaw. Passive displacement of the tongue, vocal folds, and soft palate, at least for the clinical evaluation of spasticity, is clearly not realistic. This is more than an intellectual exercise in the limits of neurologic evaluation because spasticity is often invoked as an explanation of, for example, the strain-strangled voice quality and slow speaking rate of persons with bilateral upper motor neuron lesions—those patients often diagnosed with spastic dysarthria. Very often,

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the speech-language pathologist will infer a particular neurologic sign from the speech symptoms and knowledge of lesion location. Once again, the student should note the correspondence of this aspect of diagnosis to the conceptual foundations of the Mayo Clinic classification system.

Finally, weakness is a feature of both upper and lower motor neuron disease. The degree of weakness will vary according to the severity of the disease and may not be uniform across the different structures of the speech mechanism. As described below, clinical tests of strength of speech mechanism structures are largely confined to the lips, jaw, and tongue. Many of these tests are evaluated subjectively, as when the patient is asked to push his or her tongue against the inside of the cheek with maximal effort while the examiner offers resistance by pressing against the tongue, on the outside of the cheek. Some strength tests can be implemented with instruments that measure either maximal force or pressure applied by a speech mechanism structure. Whether the tests are subjective or objective, there are two specific cautions about their use in understanding a speech production deficit in a suspected or known motor speech disorder. First, the clinician must be aware that normal speech production requires far less muscular strength than the maximal capabilities of speech mechanism structures. According to several theoretical and experimental estimates, speech requires somewhere between 5 to 20% of the maximal strength capabilities of structures such as the jaw, lips, and tongue (see Bunton & Weismer, 1994). The interpretation of weakness with respect to speech production skills, therefore, must currently be regarded as indeterminate, especially in cases where the weakness is detectable but not profound. Second, the types of orofacial muscular contractions typically used to assess weakness are quite different from the muscular contractions used in speech. Pressing the tongue into the cheek or compressing the lips or closing the jaw with maximal effort are not like the gestures used to create speech. This further complicates the use of this information for understanding the speech production deficit in motor speech disorders.

Mayo Clinic Classification of Motor Speech Disorders

The history and many other aspects of the Mayo classification system were covered in Chapter 2. Students are encouraged to read the original research papers that form the experimental basis of the Mayo classification system (Darley, Aronson, & Brown, 1969a, 1969b). The summary here is meant as a general framework for the student to return to and reflect on as the remainder of the chapter is read. The summary includes some more recent additions and modifications of the original Mayo Clinic classification system.

Flaccid Dysarthria

Associated with lower motor neuron damage, the lesions may be in the motor neurons of the brainstem or spinal cord, in the peripheral nerves leading from those motor neurons to the muscles of the head and neck and respiratory system, or at the neuromuscular junction where the peripheral nerve makes contact with the muscle. Breathiness, hypernasality, and imprecise consonants are among the frequent voice and speech problems noted with this form of dysarthria.

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Spastic Dysarthria

Associated with bilateral upper motor neuron damage, the lesions may be anywhere within the corticobulbar or corticospinal tracts, provided they are above the motor neurons. Strained-strangled (stenotic) voice, slow speaking rate, and imprecise consonants are among the "signature" speech and voice abnormalities in this dysarthria.

Ataxic Dysarthria

Associated with damage to the cerebellum or the fiber tracts connecting it to other parts of the brain (in this case, the spinal cord, brainstem, and cerebral hemispheres), ataxic dysarthria is often characterized by harsh voice, prosodic abnormalities including equal and excess stress on multisyllabic words which may contribute to a perceptual impression of scanning speech, and an overall impression of slurred, drunk-sounding speech.

Hypokinetic Dysarthria

Hypokinetic dysarthria is most often associated with Parkinson's disease, in which the neurotransmitter dopamine is depleted in the basal nuclei as a result of cell death in the substantia nigra; hypokinetic dysarthria may also occur in diseases that produce Parkinsonism. Weak voice, possible faster-than-normal speaking rate of an episodic nature (termed "short rushes of speech"), and imprecise consonants are typical speech characteristics of hypokinetic dysarthria.

Hyperkinetic Dysarthria

Associated with damage to one of several structures of the basal nuclei, the nature of hyperkinetic dysarthria may vary according to which basal nuclei structure sustains the lesion. For this reason, it is difficult to list a central group of speech characteristics associated with hyperkinetic dysarthria because they will depend, to some degree, on the nature of the disease process.

Mixed Dysarthria

"Mixed" is a designation for dysarthrias that result from damage to two or more of the areas described above. For example, both upper and lower motor neuron lesions are typical of ALS in fully developed form. The dysarthria in these cases is referred to as a mixed flaccid-spastic type. Another example is multiple sclerosis (MS) in which lesions are typically found in the cerebellum and upper motor neuron. When a dysarthria exists in these cases, it is said to be a mixed spastic-ataxic type. In theory, any of the Mayo categories could be heard as coexisting in the same patient, hence a variety of combination (mixed) dysarthrias can occur.

Unilateral, Upper Motor Neuron Dysarthria

This is a recently documented form of dysarthria involving damage on a single side of the brain, presumably in the corticobulbar tract. At one time it was thought that dysarthria associated with upper motor neuron disease had to involve bilateral lesions. Stated otherwise, unilateral lesions of the corticobulbar tract were not expected to produce dysarthria because of the extensive bilateral innervation of speech mechanism musculature. Based on reviews of a fair number of cases, however, it now seems clear that unilateral upper motor neuron damage can produce dysarthria, albeit of a mild and often tem-

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porary kind. Interestingly, as described by Duffy (2005), when this dysarthria is diagnosed, it does not necessarily sound like spastic dysarthria.

Apraxia of Speech

Not regarded as a dysarthria because the disorder is supposed to exist in the absence of muscular weakness or paralysis, apraxia of speech is often thought to be a result of cortical lesions, but the precise location is highly controversial; in some instances apraxia of speech has been claimed to occur with subcortical lesions. The speech characteristics include difficulty initiating speech which may be evident by the patient groping for the correct articulatory posture, slow, effortful articulatory behavior, and exaggerated articulatory difficulty with phonetically complex material. These problems are thought to be a reflection of programming difficulties, wherein the patient cannot plan the articulatory sequence efficiently or correctly even though the execution part of speech production—control over the muscles—appears to be normal.

The major speech characteristics of different types of motor speech disorders are offered as typical characteristics, but these are by no means definitive. Within a given dysarthria type there will be substantial variation in the specific speech characteristics regarded as abnormal, but the *type* may still be recognizable. This is an important distinction for the aspiring and working clinician: it may be possible to group patients as having the same type of dysarthria even when their specific speech characteristics are not the same. This point is made in Chapter 2, that the identification of type of dysarthria is a complex, pattern recognition task that is not very well understood. Moreover, identification of type of motor speech disorder is not necessarily reliable, even among trained clinicians.

The Model: A Closer Look

Section 4: Spinal Mechanisms

Muscles of respiration are controlled by motor neurons spanning almost the entire length of the spinal cord; these motor neurons are located in the ventral horn of the central gray matter. As shown in Figure 3–3, motor nerves exit the ventral horn and travel as part of the peripheral nervous system to the muscles they innervate. In general, the level of motor neurons within the spinal cord correspond to the level within the torso of the muscle they innervate. For example, many of the accessory muscles of inspiration—those having origins outside the rib cage but insertions on the higher ribs, such as the scalenus group, the sternocleidomastoids, and the pectoralis major and minor muscles—have motor neurons in the cervical (C1–C8) part of the spinal cord. Similarly, the intercostal muscles (external and internal) are innervated from the thoracic parts of the spinal cord (T1–T11), at roughly the same level along the long axis of the torso as the ribs. Muscles of the abdominal wall are mostly innervated from low thoracic and high lumbar portions of the spinal cord (roughly T7–L2). The one remarkable, clinically relevant exception to this is the motor neuron pool that innervates the diaphragm, the massive muscle of inspiration that separates the thorax from the abdomen. The highest point of the domed diaphragm is roughly at the level of the 6th thoracic

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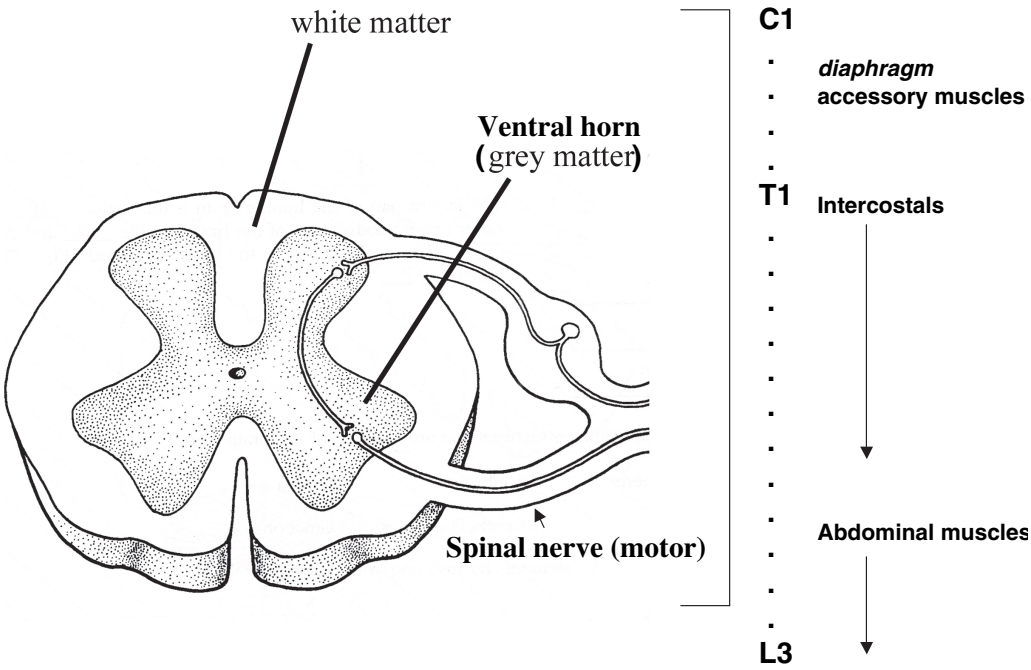

Figure 3–3. Drawing of a transverse slice of the spinal cord from a thoracic level, showing white matter (fiber tracts) and central gray matter (cell bodies), the ventral horn of which contains the motor neurons for respiratory muscles. The right side of the figure provides a sketch of the range of spinal segments that innervate respiratory muscles, most notably the innervation of the diaphragm from cervical segments of the cord. See text for additional detail.

vertebra (T6), but its motor neurons are located in the cervical region of the spinal cord, between C3 to C5. Thus, a transection of the spinal cord below C5 will paralyze all the "main" thoracic and abdominal muscles, but will leave the diaphragm intact. Because the diaphragm is a powerful muscle of inspiration, a patient with a spinal transection below C5 will be able to breathe for life without assistance, and will also be able to inflate the lungs for speech production, albeit of a type where voice loudness cannot be maintained throughout an utterance. A spinal transection between C3 and C5 will result in some paresis (weakness) of the diaphragm and such a patient may need some assistance in breathing for life. Obviously, a transection higher than C3 will paralyze the diaphragm and a ventilator will be required to sustain life.

Spinal cord damage involving motor neurons at any level of the spinal cord will result in paresis or paralysis of the affected muscles. Over time, the muscle tissue innervated by the damaged or destroyed motor neurons may atrophy as well. Weakness or paralysis of major inspiratory and expiratory muscles may affect speech breathing. When it does, the patient will have a flaccid dysarthria. The specific effects on speech production of the kind of flaccid dysarthria associated with damage restricted to the spinal cord may include

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problems with voice loudness and phrasing, and may create difficulty in the production of certain kinds of stress contrast that depend on rapid changes in lung pressure. An excellent presentation of speech breathing problems in cases of spinal cord injury is available in Hixon and Hoit (2005). What follows here is a brief summary of speech breathing manifestations of dysarthria.

Voice Loudness

When a patient has thoracic and abdominal muscle weakness and/or paralysis as a result of damage to spinal motor neurons, voice loudness will generally be insufficient. This is because the loss of muscular ability undermines the patient's ability to make and *sustain throughout an utterance* the muscular contribution required for normal voice intensity. The physiology of normal voice loudness for speech is described in Chapter 4.

Phrasing

Weakness or paralysis of respiratory muscles may result in a reduced number of syllables per utterance. Speech breathing is characterized by quick inspirations to prepare the respiratory system for an utterance. The utterance is produced on expiratory airflow and therefore a decreasing volume of air within the lungs, until the next preparatory inspiration is made. "Phrasing" is a term applied to speech breathing that can have several meanings, one of which is the number of syllables produced during an utterance, that is, during one of the expiratory events whose beginning and ending boundaries are the inspiratory "refills" just described. A reduced number of syllables per utterance may be a result of respiratory muscle weakness because the loss of muscular control makes it difficult to control the pressure developed in the lungs, resulting in utterances that are terminated after an unusually brief duration. The patient's ability to produce only a few syllables per utterance may also result in the termination of utterances at unusual locations—such as within, rather than at the end of a grammatic phrase—that adds to the communication difficulty experienced by both listener and speaker.

Stress Contrasts

Multisyllabic words in English typically have one syllable that is more prominent than the other(s). The prominence of one syllable relative to another is heard by listeners as a stress contrast. Syllable prominence or stress within a word is related to complex speech production events, including changes in fundamental frequency (F_0), vowel duration, vowel quality, and voice loudness. Similarly, speakers often choose to make a word within an utterance more prominent than the other words, to emphasize a point or indicate a contrast with something already spoken or assumed as part of the conversation. This is called sentence stress or prominence, and it is implemented by roughly the same production mechanisms as word stress. The increased loudness associated with prominent/stressed syllables is accomplished by small but rapid increments in lung pressure relative to the overall lung pressure used to produce an utterance. These pressure increments require rapid and precise contraction of expiratory muscles, which may be compromised in cases of respiratory muscle weakness or paralysis resulting from spinal motor neuron damage.