Chapter 3

NEURAL PERSPECTIVES ON MOTOR SPEECH DISORDERS

Current Understanding

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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,

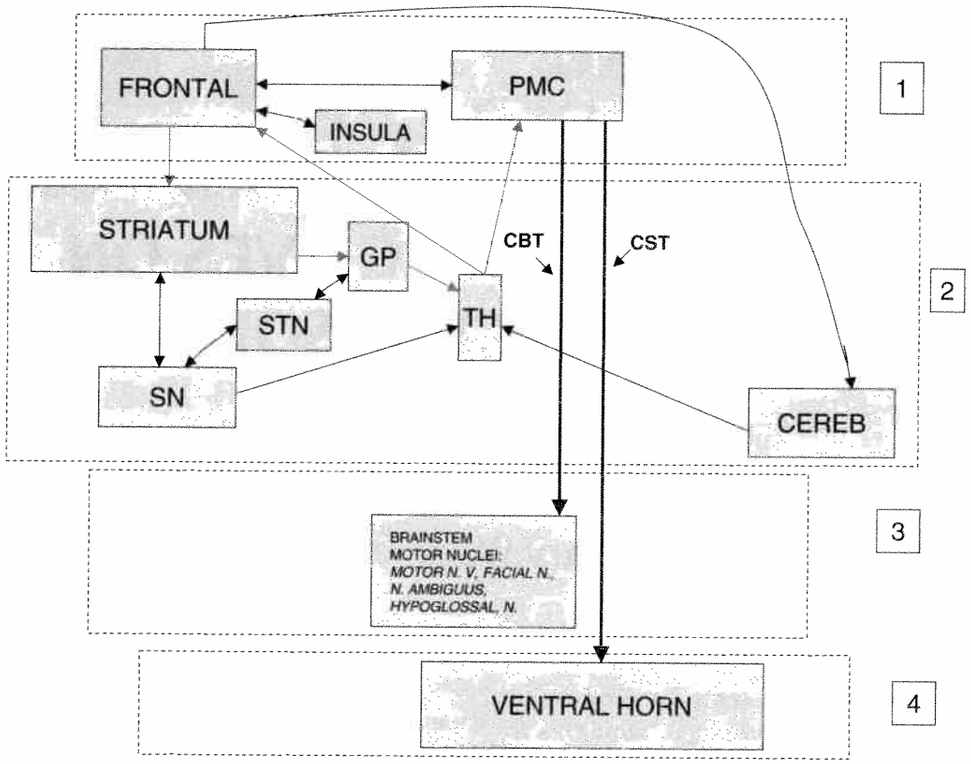

Figure 3–1. Box-and-arrows schematic diagram of central nervous system (CNS) structures involved in sensorimotor control. Section 1 contains cortical structures (PMC = primary motor cortex); Section 2, the basal nuclei and cerebellum (GP = globus pallidus; SN = substantia nigra; STN = subthalamic nucleus; 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

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

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

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

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

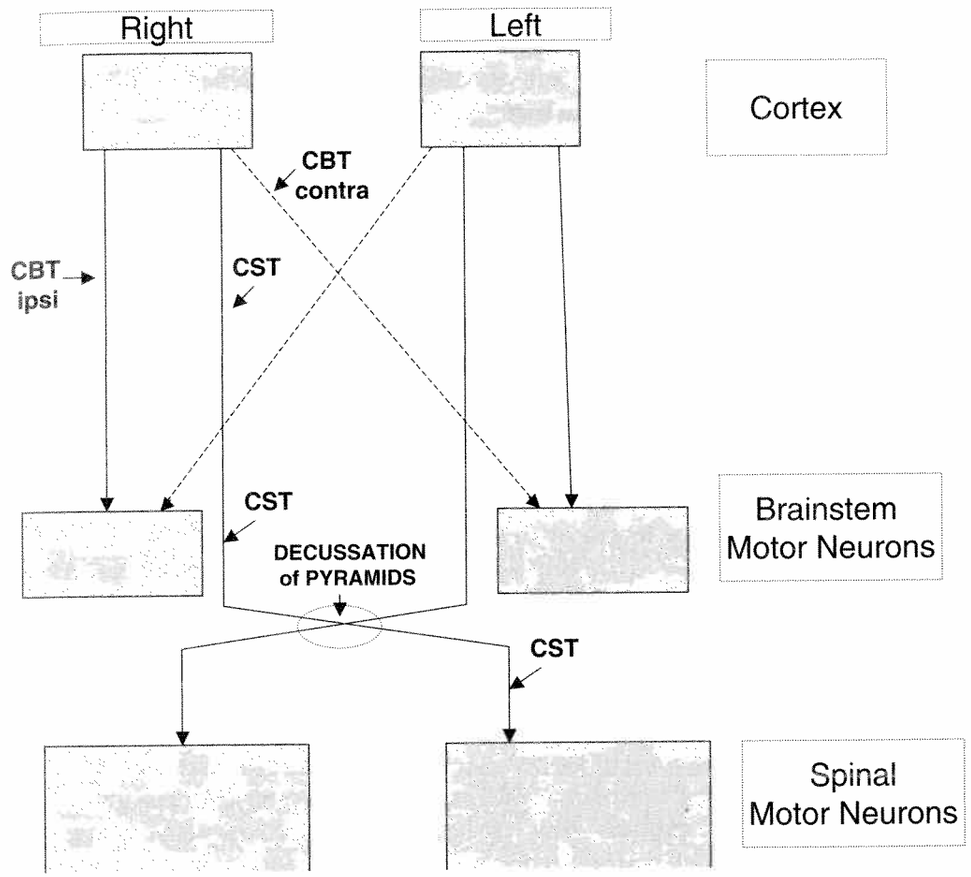

Figure 3–2. Box-and-arrows schematic diagram showing simple way to understand the difference between upper and lower motor neuron. Top two boxes represent cortical cells in primary motor cortex, middle two boxes the brainstem motor neurons, and the bottom two boxes the motor neurons in the ventral horn 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.

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.

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

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, 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.

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 temporary 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

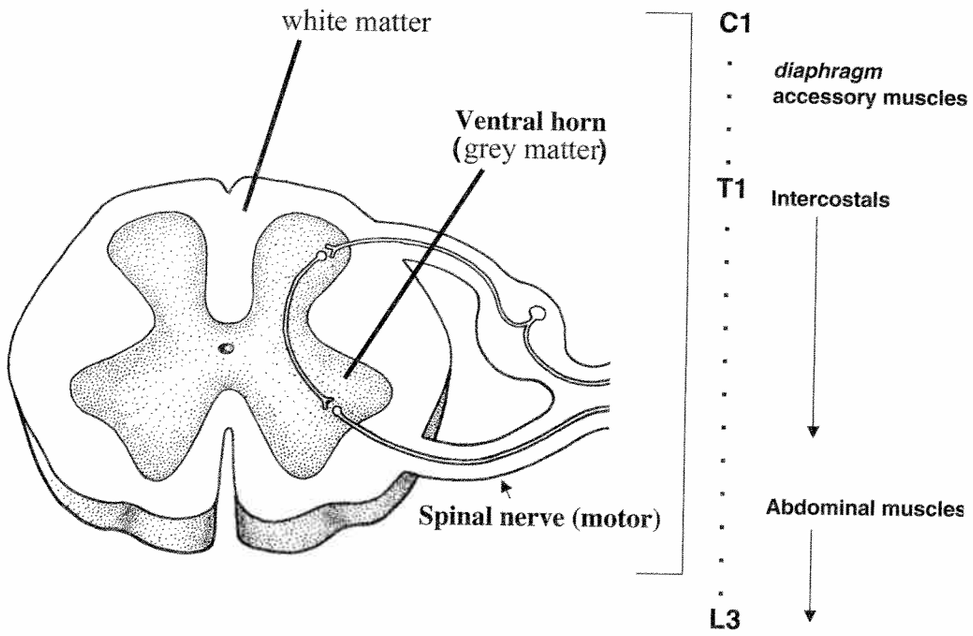

Figure 3–3. Drawing of a transverse slice of the spinal cord from a thoracic level, showing white matter (fiber tracts) and central gray matter (cell bodies), the ventral horn of which contains the motor neurons for respiratory muscles. The right side of the figure provides a sketch of the range of spinal 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

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.

Section 3: Brainstem Mechanisms

Section 3 of the model includes the brainstem nuclei containing the motor neurons and sensory cells associated with muscles of the larynx, pharynx, tongue, velum, jaw, lips, and other facial muscles. These nuclei are located in the pons and medulla and contribute to five cranial nerves that have a motor component. Figure 3–4 shows a schematic diagram of the brainstem, in coronal view. The positions of the paired nuclei are shown, as are the fiber tracts running within the brainstem (shown as dotted-lines) before they exit as cranial nerves. The rough location of the cranial nerve exits from the brainstem are labeled. Also shown is the accessory nucleus, which is located in the upper cervical spinal cord and is the origin of cranial nerve XI. As in the spinal cord, damage to these nuclei or the nerves issuing from them will generally result in paresis or paralysis, and atrophy, of the relevant muscle tissue. Sensory nuclei within the brainstem receive information about touch, proprioception (position of a structure in space), pain, temperature, and of course taste. Here the nuclei are described briefly, followed

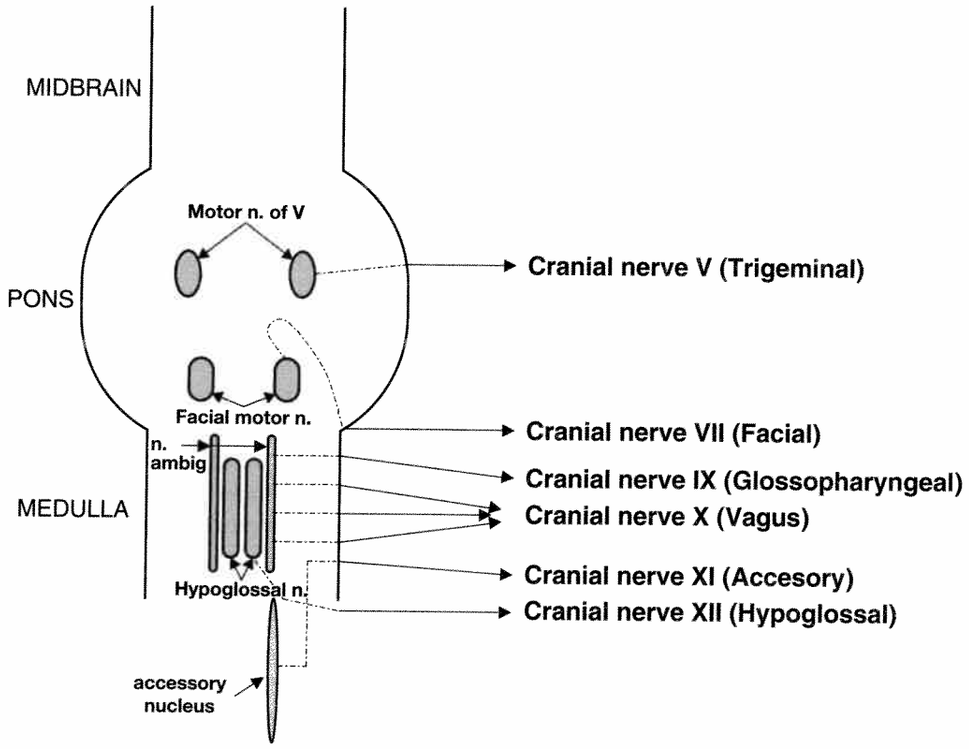

Figure 3–4. Schematic diagram of the location of brainstem and spinal motor nuclei for head and neck musculature, as well as the cranial nerves associated with those nuclei. The view is coronal.

by a brief description and discussion of the cranial nerves typically tested in the speech mechanism examination.

Motor Nucleus of V

The motor nucleus of V is paired (like all the nuclei to be discussed), with one nucleus on each side of the brainstem midline. As depicted in Figure 3–4, the nucleus is an oval-shaped, compact group of cells located approximately midway between the superior and inferior borders of the pons. The input to the motor nucleus of V—the information delivered to it from cortical levels via the corticobulbar tract—is bilateral, meaning that both cerebral hemispheres send fibers to the nucleus on its same and opposite side. Axons from the motor nucleus travel laterally and ventrally (forward) and exit the brainstem approximately at mid-pons level as part of cranial nerve V (trigeminal). Soon after it exits the pons the trigeminal nerve separates into three major divisions—ophthalmic, maxillary, and mandibular—but motor fibers are found only in the mandibular division. The motor fibers innervate the muscles that close and open the mandible, the mylohyoid muscle (floor of the mouth), the tensor veli palatini (a muscle of the velopharynx important for opening the auditory tube), and the tensor tympani (a muscle of the middle ear whose action can damp vibration of the ossicular chain).

Jaw motions during speech have been studied more than other structures of the speech mechanism. This is probably because the jaw is easily accessible for monitoring of motions (unlike, say, the tongue and velum) that are fairly simple for speech (unlike, say, the motions of the tongue). It is widely assumed, and probably for good reason, that unilateral, upper motor neuron damage will not have much or any effect on jaw motions because the motor nuclei of V are innervated bilaterally. It is not known how unilateral, lower motor neuron damage might affect jaw motions for speech.

Evaluation of Motor Integrity of Motor Nucleus of V. Evaluation of the integrity of the motor nucleus of V can be performed using the masseter bulge test. The patient is asked to relax with her mouth closed, and the tester places the index and middle fingers of both hands roughly at the ear lobes of a patient and slowly moves them forward along the side of the face, toward the mouth. As the fingers are moving forward the examiner will reach a point where her fingers move inward, as if a ridge has been reached where the fingers "drop off" toward the midline of the mouth. This ridge is the anterior edge of the masseter muscle, one of the important jaw closers for both mastication and speech. If the fingers are positioned against this ridge and a patient is asked to bite (clench), the masseter muscles will bulge as they shorten, pushing the fingers toward the examiner. It is quite easy to feel this movement in a healthy masseter muscle.

A unilateral, lower motor neuron lesion will result in an asymmetric masseter bulge when the teeth are clenched, with a weak or absent bulge on the affected side and a normal bulge on the healthy side. If both sides have a weak or absent bulge, there is some ambiguity in interpretation because either bilateral lower motor neuron or bilateral upper motor neuron damage could produce this result. One way to resolve the ambiguity of lesion location is to test the jaw-jerk reflex, mentioned above. The normal response is very subtle, and perhaps absent to the naked

eye, but in cases of upper motor neuron lesions a strong, upward movement of the mandible will follow the downward tap. This is consistent with the general symptom of hyperexcitable reflexes in upper motor neuron disease. The combination of a hyperexcitable jaw-jerk reflex with observation of fasciculations of the tongue can be diagnostic of ALS, which often has both upper and lower motor neuron damage.

The presence of a unilateral, lower motor neuron lesion can also be evaluated by asking the patient to slowly open her mandible while the clinician offers some resistance to the motion. During the opening motion the jaw will deviate toward the affected side if there is a unilateral lesion of the motor nucleus of V.

Facial Motor Nucleus

The facial motor nucleus is located in the lower pons, slightly more medial than the more superior motor nucleus of V (see Figure 3–4). The nucleus sits approximately halfway between the fourth ventricle (at the posterior border of the pons) and the ventral surface of the pons. Axons emerging from the facial motor nucleus run within the brainstem posteriorly toward the floor of the fourth ventricle (shown for the sake of illustration as an upward course in the schematic of Figure 3–4), loop around the abducens nucleus (associated with cranial nerve VI which supplies motor innervation to muscles of the eye), and then course ventrolaterally to emerge at the junction of the pons and medulla as cranial nerve VII. Cranial nerve VII innervates all muscles of facial expression as well as several other muscles (including the stapedius muscle in the middle ear, the contraction of which is the end product of the acoustic reflex).

The facial motor nucleus receives a fairly complex input from cortical and subcortical parts of the brain. The motor neurons that control muscle fibers from approximately mid-face and up receive bilateral innervation from cortical cells, but motor neurons serving the lower facial muscles receive only contralateral innervation from the cortex. As explained more fully below, this innervation pattern means that a unilateral cortical lesion in the face area is likely to have little effect on upper facial muscles (such as those of the forehead), but will result in weakness and possibly paralysis of the lips and other central/lower facial muscles on the side of the face opposite to the lesion location. The facial motor nucleus also receives complex innervation from parts of the limbic system, which plays a major role in emotional expression. The separation of input to the facial motor nucleus from cortex and limbic system most certainly explains the clinical observation in certain stroke patients whose capacity for facial expressions elicited in emotional situations is at odds with their relative inability to produce volitional manipulation of facial muscles.

The integrity of cranial nerve VII is important in speech production because of the role it plays in control of lip motions and the resulting configurations of the labial orifice. Lip motions are not only important for English consonants such as bilabial stops and labiodental fricatives, but the shaping of the lip orifice—what is often called the rounding-spreading dimension, but is better understood as the area and length of the space between the lips—is crucial to vowel production. This is especially the case in languages where there are vowel contrasts critically dependent on the shape of the lip orifice, such as the /i/-/y/contrast in Swedish (/y/ is very much like an /i/ produced with rounded lips). Here the tongue configuration for the two vowels is essentially identical and the goodness of the articulatory contrast depends on the ability to create a difference in the area of the labial orifice (English does not have these kinds of vowel contrast). To date, there are no studies of labial configuration characteristics for speech in persons with upper or lower motor neuron damage affecting facial muscle control.

Evaluation of Motor Integrity of Facial Motor Nucleus and Cortical Facial Area. There are simple rules for evaluating the integrity of the motor pathways associated with facial muscle control. A unilateral, lower motor neuron lesion will cause *all* ipsilateral muscles of the face to be weak or paralyzed. A lesion in the facial motor nucleus or on cranial nerve VII near its exit from the brainstem will therefore affect all facial muscles from forehead to chin. This will be apparent by viewing the patient's face at rest, where the smoothing of the furrows of the forehead and of the nasolabial fold on the affected side, as well as drooping of the corner of the mouth on the same side, will indicate loss of facial muscle control. This scenario can be contrasted with the case of a unilateral, upper motor neuron lesion, where the primary deficit will only be seen in the central/lower face on the side opposite the lesion. Thus, even though the forehead on the side contralateral to the lesion will be normally furrowed, the nasolabial fold will be smoothed and the corner of the mouth will droop. The apparently normal forehead appearance coupled with a weak or paralyzed lower face is indicative of an upper motor neuron lesion of the side opposite the facial evidence.

There are additional tests for the integrity of facial motor pathways. It is often said, for example, that individuals with unilateral, upper motor neuron lesions will show some asynchrony of the two corners of the mouth when asked to smile voluntarily (with the affected side, opposite the lesion, lagging the lateral movement of the healthy side). These same patients, however, may show symmetric movements when smiling spontaneously. In contrast, patients with unilateral, lower motor neuron damage will present with a deficit on the affected side that will be the same in voluntary and spontaneous smiles. Also, facial muscle weakness can be demonstrated by asking the patient to lift her eyebrows, where marked asymmetry of the gesture indicates weakness on the side with the lower eyebrow; or to have the examiner attempt to open the patient's eyes when the latter forcefully closes them—intact musculature for closing the eyes should prevent the examiner from lifting the eyelid and exposing the eye. Because these are tests of upper-face control, weakness confined to one side would only be expected with an ipsilateral, lower motor neuron lesion. Weakness on both sides of the upper face could indicate either a bilateral lower or upper motor neuron lesion.

A test of central/lower facial control is to ask the patient to forcefully close the lips while the examiner attempts to open them. Any weakness of the relevant muscles should allow the examiner to separate the lips, but a healthy facial motor pathway should allow the patient to maintain lip closure against this effort. A unilateral, upper motor lesion would affect the control of lip muscles on the side opposite

the lesion and could reduce the patient's ability to maintain lip closure for this test; the same situation would apply to a unilateral lower motor neuron lesion. Bilateral lesions, either of the lower or upper motor neuron, should clearly weaken the muscles of the central/lower face and prevent the patient from resisting the examiner's attempt to separate the lips.

Nucleus Ambiguus

The nucleus ambiguus is a column of cells running almost the entire length of the medulla (see Figure 3-4). This column of cells is located about midway between the ventral and dorsal surfaces of the brainstem, and somewhat lateral to the midline. Motor neurons in the nucleus ambiguus innervate the constrictor muscles of the pharynx, muscles of the velopharynx (such as the levator veli palatini), intrinsic muscles of the larynx, and a single muscle of the tongue (the palatoglossus, or anterior faucial arch). These motor neurons receive bilateral input from the cortical areas in which the muscles are represented. Motor axons exit the nucleus ambiguus and run more or less laterally and somewhat ventrally within the brainstem until they emerge as a series of rootlets from the lateral aspect of the medulla as parts of cranial nerves IX (glossopharyngeal) and X (vagus). Fibers in cranial nerve IX innervate only one muscle, the stylopharyngeus; the remaining muscles are innervated by cranial nerve X.

Evaluation of the Motor Integrity of the Nucleus Ambiguus and Cranial Nerves IX and X. The motor function of cranial nerves IX and X cannot be evaluated separately because there is no unique test for the stylopharyngeus muscle. Thus, evaluation of the motor function of the two nerves and their common brainstem nucleus is combined. Tests of the integrity of the relevant muscle groups, and by inference cranial nerves IX and X and the nucleus ambiguus, typically focus on the velopharynx and larynx. The weakness or paralysis associated with a unilateral lesion in the nucleus or cranial nerve X would likely produce a breathy voice quality and some hypernasality, depending on the extent of the damage. A suspected, unilateral lower motor neuron lesion involving the nucleus ambiguus/cranial nerve X can also be evaluated by asking the patient to open her mouth and breathe quietly so the position of the relaxed soft palate can be observed. At rest the velum should have a symmetric appearance. When the patient is asked to phonate a vowel, the observable movement of the soft palate and posterior wall of the pharynx—raising of the soft palate and tensing of the pharyngeal muscles—should be symmetrical if there is no damage to the nucleus or nerve. In the case of a unilateral, lower motor neuron lesion, when the patient phonates the soft palate will raise asymmetrically, with higher elevation on the healthy side and the uvula pulled off the midline in the same direction (the same patient may show a similar, but more subtle asymmetry with the velum at rest). Normally the primary muscles of velopharyngeal closure, the levator veli palatini and the superior pharyngeal constrictor, apply equal force on the two sides of the velopharynx and therefore produce symmetric movements around the orifice. A unilateral, lower motor neuron lesion will produce weakness or paralysis of the muscles on the same side, allowing the healthy muscles on the opposite side to "overpower" the affected muscles and pull the structures in the unaffected direction.

The interpretation of a breathy voice quality as a sign of a unilateral, lower motor neuron lesion is more complicated, as are some of the other laryngeal tests for the motor integrity of cranial nerves X and the nucleus ambiguus. Vocal fold vibration is produced by a combination of aerodynamic, mechanical, and muscular forces, the latter being required for overall settings of the larynx but not strictly for the vibration, even if they are desirable for "good" phonation and the full range of phonatory ability (see Chapter 5). In other words, the vocal folds can vibrate in response to aerodynamic and mechanical forces even when the laryngeal muscles are weak or paralyzed, and especially when the muscles on only one side are affected by a unilateral, lower motor neuron lesion. Moreover, the range of "normal" voice qualities is enormous, with some people having breathy qualities in the absence of any known neural pathology. Breathy voice quality as a sign of lower motor neuron disease is probably most reasonable when co-occurring signs (such as hypernasality) are present.

A more direct evaluation of laryngeal muscular integrity is to have the patient cough forcefully or produce a series of staccatolike /i/ sounds, the latter sometimes called laryngeal diadochokinesis. Both tasks require forceful adductory efforts, produced by some combination of the lateral cricoarytenoid, interarytenoid, and vocalis muscles. An inability to produce a sharp cough and "hard" onsets of successive /i/ sounds could indicate a lower motor neuron lesion although there is no way to identify which side the lesion is on (including the possibility of bilateral lesions) unless the vocal folds are imaged with indirect or direct laryngoscopy studies (e.g., stroboscopy, high-speed digital imaging).

Unilateral, upper motor neuron lesions in cell bodies or fiber tracts associated with velopharyngeal and laryngeal musculature will likely produce little effect on resonance or phonation because of the bilateral innervation of the lower motor neurons, described above. As reviewed by Duffy (2005), however, a unilateral upper motor neuron lesion *can* result in phonatory and resonatory effects despite the bilateral innervation. The interested reader is encouraged to read the careful coverage of unilateral, upper motor neuron dysarthria in Duffy (2005) and Chapter 2 of the present text.

Accessory Nucleus

The accessory nucleus is a column of cells in the ventral horn of the upper five or six cervical segments of the spinal cord. These cells give the appearance of a spinal continuation of the nucleus ambiguus (see Figure 3-4), the two nuclei being more or less in line with each other. The accessory nucleus supplies innervation to the sternocleidomastoid and the trapezius muscles. Cortical input to the accessory nucleus is ipsilateral for the motor neurons supplying the sternocleidomastoid muscle, and contralateral for the trapezius muscle.

The accessory nucleus is included here because it is the origin of cranial nerve XI, the accessory nerve. Fibers from the motor neurons of the accessory nucleus exit the spinal cord and ascend along its surface until reaching the level of the medulla where the most caudal fibers of cranial X exit the brainstem (see the dotted-line from the accessory nucleus). There the accessory fibers join with vagus fibers and together (also with cranial nerve IX) they exit the skull and then separate to their respective muscle targets. The main functions of the two muscles innervated by the accessory nucleus and

cranial nerve XI are to rotate the head to the side opposite the contracting muscle (sternocleidomastoid) and to elevate the shoulder on the same side of the contracting muscle (trapezius). These muscles are potentially of interest to evaluation of the speech mechanism because both have points of attachment on the clavicle, upper ribs, and sternum and so can function as secondary (accessory) muscles of inspiration. Such muscles do not typically come into use in normal respiration or speech breathing, but may be recruited for deep inspiration during exercise or in cases of spinal cord injury that weaken diaphragmatic mechanisms of inspiration. The standard neurologic tests for the integrity of these muscles are described in many textbooks (see Wilson-Pauwels, Akesson, Stewart, & Spacey, 2002).

Hypoglossal Nucleus

The hypoglossal nucleus is a column of cells running on either side of the midline of the medulla (Figure 3–4). The nucleus is in the posterior part of the medulla with its rostral end in the floor of the fourth ventricle. The motor neurons of the hypoglossal nucleus innervate all intrinsic muscles of the tongue and three extrinsic muscles of the tongue (genioglossus, styloglossus, hyoglossus; recall that palatoglossus is innervated from the nucleus ambiguus via cranial nerve X). Axons from the hypoglossal nucleus first run forward (ventrally) in the medulla; midway between the dorsal and ventral surfaces the fibers bend obliquely to exit the brainstem in a series of rootlets as cranial nerve XII (hypoglossal nerve). The cortical input to the hypoglossal nucleus is primarily bilateral, with the exception of the genioglossus muscle which is innervated contralaterally (left side of cortex innervates hypoglossal motor neurons on the right side of the medulla, right side of cortex innervates hypoglossal motor neurons on the left side of the medulla).

Evaluation of the Integrity of the Hypoglossal Nucleus and Cranial Nerve XII. The distinction between intrinsic and extrinsic tongue muscles is a long-debated aspect of speech motor control. Some scientists believe the intrinsic muscles play more of a role in shaping the tongue (as in the case of grooving for lingual fricatives) and the extrinsic muscles in positioning the tongue within the vocal tract (as in the position difference between front and back vowels). There is probably more cooperation than strict difference between the two sets of muscles in creating tongue shapes and positions, and this may be reflected in the fact that there are no neurologic evaluations permitting isolated evaluation of intrinsic and extrinsic tongue muscles. Like evaluations of the motor integrity of other cranial nerves, the focus for the tongue is on differentiating upper from lower motor neuron lesions.

If a healthy patient is asked to protrude her tongue it will emerge from the mouth along the midline, without remarkable deviation to either the right or left side. The genioglossus muscle—the paired muscle that inserts along the entire length of the tongue, and is often said to compose its bulk—produces the protrusion motion by applying equal force on its two sides. If one side of the genioglossus muscle is weak because of an upper or lower motor neuron lesion the protruding tongue will deviate markedly to the weak side, pushed that way by the healthy, stronger muscle. Upon seeing such a deviation, the examiner can be reasonably certain there is a neurologic problem but cannot, in the absence of other evidence, pinpoint the location of the lesion. This is because unilateral weakness of the genioglossus could result from two different lesion locations: (1) an ipsilateral, lower motor neuron lesion that affects the hypoglossal nucleus and/or cranial nerve XII, or (2) a contralateral, upper motor neuron lesion affecting the cortical tongue cells or the corticobulbar fibers that run from the cortical tongue area of one hemisphere and cross in the brainstem before terminating in the hypoglossal nucleus on the other side.

For example, a unilateral, lower motor neuron lesion of the left hypoglossal nucleus and a unilateral, upper motor neuron lesion of the cortical tongue area in the right cerebral hemisphere will both result in weakness of the left genioglossus. Both lesion locations, therefore, produce the same sign, a deviation of the protruded tongue to the weak (left) side. What, then, distinguishes a unilateral upper from lower motor neuron lesion of motor neurons and fibers serving the tongue? SLPs and neurologists typically rely on the appearance of the tongue at rest, inside the mouth, where fasciculations and atrophy of one side of the tongue will indicate an ipsilateral lower motor neuron lesion. As described above, upper motor neuron lesions are not typically associated with either of these symptoms. Therefore, a protruded tongue that deviates to the left and shows no atrophy or fasciculations is suggestive of a cortical or cortibulbar tract lesion (upper motor neuron), on the side opposite the deviation.

Lower Motor Neuron Disease and Speech Production

As reviewed above, flaccid dysarthria is typically associated with lower motor neuron disease. Presumably, the weakness and/or paralysis following damage to motor neurons or the peripheral nerves to muscles results in the symptoms of flaccid dysarthria: breathiness because weak adductory forces makes the closing phase of vocal fold vibration relatively less effective; hypernasality as a result of the weak musculature (levator veli palatini, superior constrictor, musculus uvuli) typically involved in closure of the velopharyngeal orifice; and imprecise consonants as a result of weakness of the lingual muscles. Flaccid dysarthria associated with either isolated brainstem damage or specific lesions of peripheral nerves has not been studied as much as some of the other types of dysarthria, so the precise speech production characteristics are not well understood. Moreover, lesions whose effects are restricted to specific parts of the speech mechanism are unusual because the motor nuclei of the brainstem are contained within a very small volume of tissue. Thus, structural damage or deterioration within the brainstem is unlikely to involve a single motor nucleus. One interesting feature of flaccid dysarthria to emerge from the Mayo studies was the absence of speaking rate abnormalities among this group of patients. Certainly some patients with damage to brainstem structures or peripheral nerves will produce abnormally slow speaking rates, but available data do not implicate rate as a major feature of flaccid dysarthria. This would suggest that the slow speaking rates characteristic of some of the other types of dysarthria and in some cases of apraxia of speech cannot be attributed to weakness of speech mechanism structures.

Upper Motor Neuron Disease and Speech Production

Spastic dysarthria has traditionally been associated with bilateral, upper motor

neuron lesions. The hypertonicity—specifically the spasticity—of upper motor neuron disease has been thought to contribute to the strain-strangled voice quality (too much adductory force) and perhaps the slow speaking rate observed so often in spastic dysarthria. The role, if any, of hyperexcitable reflexes in the specific speech characteristics of spastic dysarthria is unknown.

Auditory Nuclei

A series of nuclei from the medulla to the thalamus serve as processing and relay points for the delivery of auditory information to the primary auditory cortex, located in the superior and lateral parts of the superior gyrus of the temporal lobe. Information is delivered to the first of the brainstem nuclei, the cochlear nucleus in the medulla (see Figure 3-5), from the auditory part of cranial nerve VIII. The auditory nerve codes in neural form the frequency and intensity analysis performed by the cochlea. As the coded information travels up the auditory pathway to the cortex a variety of analyses produce the perceptual experiences we know as pitch, loudness, sound localization, sound quality, and sound sequence (that is, the relative simultaneity or sequential nature of multiple auditory stimuli). The relationship between the physical attributes of a signal, such as frequency, intensity, and phase relations, and the just mentioned perceptual characteristics is fairly well understood thanks to the long-established

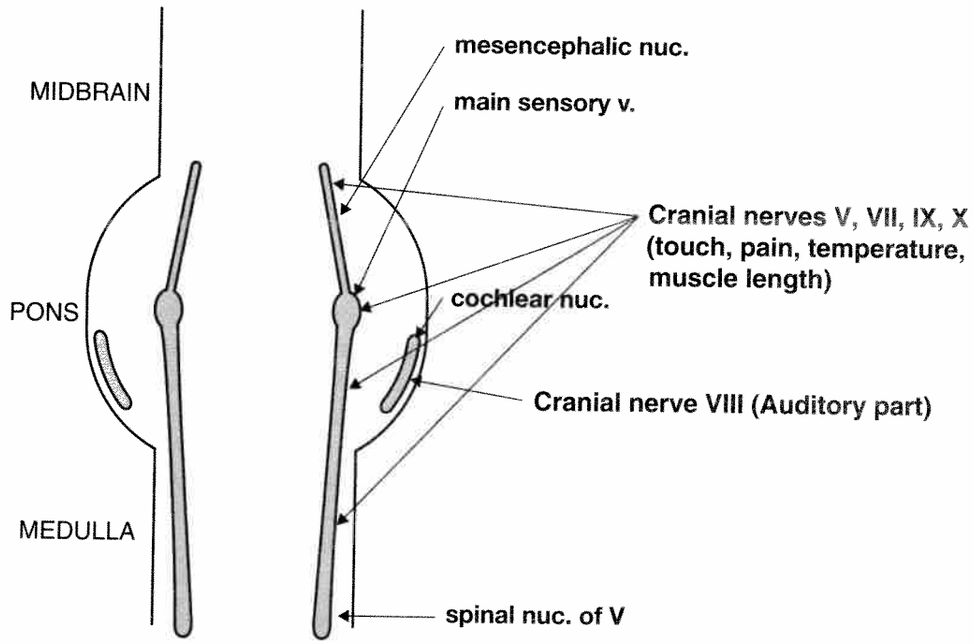

Figure 3-5. Schematic diagram of the location of the cochlear nucleus—the first brainstem structure in the central auditory pathways—and the sensory nucleus of V. See text for additional details.

and productive discipline of psychoacoustics. Moreover, the science of psychoacoustics has been translated into clinical practice; many of its findings form the basis for tests used in diagnostic audiology.

Missing from the above list of auditory analyses is speech. Clearly, analyses along the auditory pathway and in the cortex that yield percepts of pitch, loudness, and quality, are related to the perception of speech. But the discipline of speech perception, which has a history almost as long as that of auditory psychophysics, has not produced definitive explanations concerning the perception of speech and how it might be related to speech production. Standard audiologic evaluations of the speech reception threshold (SRT) or speech discrimination ability may provide some very coarse-grained information on auditory abilities, but these tests are far removed from the phenomena linking speech perception with speech production abilities. For the time being, then, we have to rely on the very gross connection between a patient's general hearing ability and their speech production capabilities. In patients with hearing impairment and no other known neurologic disease, a general expectation is that the more severe the hearing loss the greater the impact on speech production ability. Thus, the speech intelligibility deficits of persons with neuromotor disease and substantial hearing loss, as for example sometimes observed in persons with the athetoid form of cerebral palsy, most likely reflect the combined influence of hearing loss and neuromotor deficits on speech production.

Sensory and Other Brainstem Nuclei

Section 3 of the model as shown in Figure 3-4 does not include sensory nuclei associated with head and neck structures because the focus in clinical evaluation is typically on motor functions. This does not mean that sensory function is unimportant for speech production. Tactile integrity, proprioceptive function, even sensation of air pressures and flows are likely to be important to speech production skill and probably especially so during speech development. Unfortunately, clinical tests for the evaluation of orosensory integrity are controversial, partly because there is no agreement on proper tests or their interpretation. The clinician should know, however, that tactile, pain, temperature, and proprioceptive sensation is processed by the sensory nucleus of the trigeminal nerve, a long, paired nucleus that runs virtually the entire length of the brainstem. This nucleus has three subnuclei, including a top part in the midbrain called the mesencephalic nucleus of V, a middle part in the pons called the main (or primary) sensory nucleus of V, and the bottom part (in the medulla) called the spinal nucleus of V. The nucleus is named for the trigeminal nerve because so much sensory information from the head and neck is carried through the three nerve branches of cranial nerve V. For example, touch sensation from the face and the anterior two-thirds of the tongue is delivered to the brainstem through cranial nerve V. Most of this information projects to the main sensory nucleus of V, in the pons. Other cranial nerves, however, also deliver information on touch, pain, and temperature to the sensory nucleus of V. This includes cranial nerve VII (facial nerve) which carries sensory information from parts of the pinna and the ear-canal side of the tympanic membrane; cranial nerve IX (glossopharyngeal nerve), from the pharynx, the posterior one-third of the tongue, the middle ear side of the tympanic membrane, and

some skin around the ear; and cranial nerve X (vagus), from the larynx and external ear.

The sensory nerves and nuclei for audition (just the cochlear nucleus), touch, pain, and temperature are summarized schematically in Figure 3–5. Note how several cranial nerves deliver sensory information to the sensory nucleus of V. An interesting chapter on possible functions and evaluations of the sensory components of the speech mechanism is available in Kent, Martin, and Sufit (1990).

The brainstem also contains nuclei serving other functions in the head and neck region. These nuclei include those that receive taste information and control salivatory and other glandular secretions. The interested student is referred to Wilson-Pauwels et al. (2002) for a more detailed but accessible presentation.

Section 2: Basal Nuclei and Cerebellum

Section 2 of the model includes the several groups of cells in the basal nuclei, the thalamus, and the cerebellum. The schematic diagram in Figure 3–6 shows the interconnections between these structures. These structures are grouped together because of what is presumed to be their joint role in movement planning, initiation, and coordination.

Basal Nuclei

The basal nuclei include the *putamen* and *caudate*, which together are called the *striatum*; the *substantia nigra*, the *globus pallidus*, and the *subthalamic nucleus*. These structures are interconnected in complex ways as shown by connecting

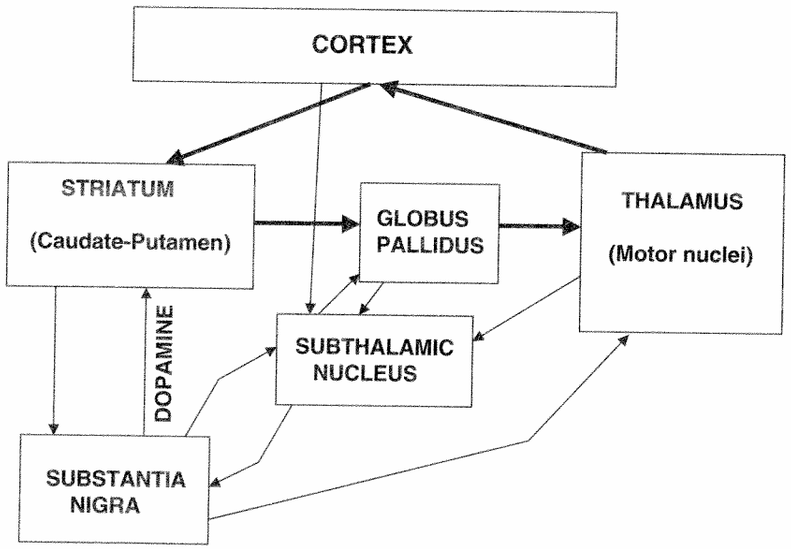

Figure 3–6. Schematic diagram showing the interconnections between basal nuclei structures, and the motor loop between the basal nuclei and cortical structures (the latter shown by the heavy arrows). Note the dopaminergic pathway between the substantia nigra and the striatum.

arrows in Figure 3–6. Many of the structures are connected reciprocally (for example, see the arrows in both directions between the substantia nigra and the striatum). Most importantly, for our purposes, most of the outputs from the basal nuclei converge on the globus pallidus which sends its output to the thalamus. The thalamus then relays this information to motor parts of the cortex (different areas of the cortex are lumped together for this schematic), which as shown in the figure sends the information back to the basal nuclei. Thus, the basal nuclei receive their own, processed output via the globus pallidus→thalamic→cortical→basal nuclei loop. This is shown in Figure 3–6 by the thick lines connecting these structures.

The putamen is thought to be primarily involved with learning and execution of complex movements. The caudate nucleus is believed to have a similar function, but is often implicated in more cognitive aspects of planning and execution of complex movement sequences. Lesions in either the putamen or caudate nucleus will result in movement disorders, which may be accompanied by personality and/or mood disorders. The substantia nigra is found in the midbrain and contains cells that manufacture the neurotransmitter dopamine. Dopamine is transported to the striatum (see the labeled pathway in Figure 3–6) where it plays a critical role in the regulation of movements. To a first approximation, Parkinson's disease has its onset and progression as a result of cell death in the substantia nigra and the ensuing loss of sufficient amounts of dopamine in the striatum.[1] The classic symptoms of Parkinson's disease are tremor, rigidity (a type of hypertonus, differing from spasticity because the excessive tone is not dependent on the direction of limb movement), and bradykinesia (slowed preparation, initiation, and production of movements). The subthalamic nucleus receives input from the cortex and thalamus, as well as other parts of the basal nuclei, and sends its output primarily to the globus pallidus. The output of the subthalamic nucleus is thought to modulate the activity of the globus pallidus. Because the globus pallidus is the terminal nucleus in the basal nuclei-thalamic-cortical-basal nuclei loop, the subthalamic nucleus may play an important role in shaping the output of the basal nuclei and, therefore, the form of motor behavior. Lesions in the subthalamic nucleus cause choreiform movements (brief, tic- or jerklike movements of the distal extremities [like the fingers] and the face, sometimes resembling fragments of movements but not having a real purpose) and ballism (involuntary, large-scale jerks involving the proximal limbs, sometimes in the form of "throwing" movements). Interestingly, lesions or deep-brain stimulation of the subthalamic nucleus may be used therapeutically to relieve symptoms of Parkinson's disease. Finally, the globus pallidus is, as mentioned above, the main output of the basal nuclei; this output is delivered to the thalamus, which in turn sends the information to motor areas of the cortex. Isolated lesions of the globus pallidus are very rare (Kuoppamäki et al., 2005), but when they have been reported they seem to produce some symptoms like those seen

[1]This is an oversimplification of the neurotransmitters that play an important role in the basal nuclei. There are other neurotransmitters of importance, but the scope of this chapter does not permit full coverage of their role in normal and disordered movement. See Kandel, Schwartz, and Jessell (2000) for excellent treatment of neurotransmitters and movement.

in Parkinson's disease. Deep-brain stimulation of the globus pallidus (particularly of the more medial, internal segment) has also been used to relieve symptoms of Parkinson's disease.

Three or four nuclei in the thalamus receive the output of the globus pallidus and send that information to various motor areas of the cortex, such as the supplementary motor cortex, the premotor cortex, and the primary motor cortex. In this way, the basal nuclei are thought to influence motor commands in the descending corticospinal and corticobulbar tracts. Isolated lesions of these thalamic regions—typically referred to in the contemporary literature as the motor thalamus—are rare, but consequences for complex motor behavior can be assumed. Parts of the motor thalamus have also been targets for neurosurgery to relieve the tremor symptoms of Parkinson's disease and other neuromotor disorders.

Symptoms of Basal Nuclei Disease

The concepts of upper versus lower motor neuron disease are not typically applied to signs and symptoms associated with diseases of the basal nuclei. In the older neurologic literature and even in some contemporary writings diseases of the basal nuclei are said to produce *extrapyramidal signs and symptoms*. *Extrapyramidal* refers to structures and lesion effects outside of the *pyramidal tract*, the term used to describe the corticospinal and corticobulbar tracts which form the "direct" pathways from cortex to motor neurons in the spinal cord and brainstem. The basal nuclei have only indirect connection to these "direct" tracts, via the loop described above (Figure 3-6), hence the term extrapyramidal.

There are many symptoms of basal nuclei disease. There are also some predictable effects of basal ganglia disease on speech production. These are summarized here.

Tone. Parkinson's disease is the result of dopamine deficiency in the basal nuclei. One of the classic symptoms of Parkinson's disease is *rigidity*, a form of hypertonus. A rigid limb, when passively displaced, will feel stiff and offer a great deal of resistance to the motion. This high degree of resistance will be felt when the limb is displaced either away from, or into, the body, and is not likely to be sensitive to speed or range of displacement (contrast this with spasticity, another form of hypertonus discussed earlier).

Certain diseases of the basal nuclei may be associated with tone that fluctuates from hyper- to hypotonia. Diseases such as athetosis (one of the categories of cerebral palsy, thought to involve primary damage to the putamen) and Huntington's disease (a genetic disease affecting cells in the striatum—most notable the caudate nucleus) may produce this kind of fluctuating tone.

Dyskinesias. Dyskinesias are impairments of voluntary movement and may be seen in a variety of neurologic diseases and sometimes as a result of treatment of those diseases. Some of the more common dyskinesias include *tics*, *jerks*, and *dystonias*. The impaired movements may be spasmodic, as in a slow buildup of muscular contraction that is maintained for an unusual amount of time. When this happens, the disorder is called a dystonia. In other cases the impaired movements may be sudden and brief, as in the facial tics of Tourette's syndrome, or jerky and repetitive as in *myoclonus*.

Basal Nuclei Disease and Speech Production. The many diseases of the basal nuclei and their connections may result in a variety of speech symptoms. This discussion is limited to a selected few. In the Mayo classification system, basal nuclei disease may be associated with hypokinetic and hyperkinetic dysarthria, and in cases where there is basal nuclei disease plus damage to other parts of the brain as well, mixed dysarthria. Hypokinetic dysarthria is typically associated with Parkinson's disease, and is often characterized by reduced voice loudness with breathy quality, imprecise consonants which may be a function of a faster-than-normal speaking rate (episodic or chronic), and a general reduction of articulatory gestures. The extent to which these speech symptoms are directly related to, or can be explained by, the triad of classic neurologic signs of the disease—tremor, rigidity, and bradykinesia—is unknown. There seem to be logical links between rigidity and bradykinesia and the speech symptoms of reduced articulatory gestures and voice loudness; after all, it makes sense that stiff muscles would result in smaller-than-normal movements and difficulty initiating and moving speech structures. But the logic of the link shows some weakness when the effects of therapeutic drugs on nonspeech (e.g., limb) and speech symptoms are observed. Drugs meant to supplement dopamine often relieve the classic symptoms of Parkinson's disease, but have little effect on the severity of the hypokinetic dysarthria (for a good review of drug effects on hypokinetic dysarthria, see Schulz, 2002; and Pinto et al., 2004). This suggests a more complex relationship, or perhaps an indeterminate relationship, between the signs and symptoms of Parkinson's disease and the nature of hypokinetic dysarthria.

Hyperkinetic dysarthria is a cover term for dysarthrias associated with a number of basal nuclei structures and may encompass several, unique forms of the speech disorder. A good illustration of the difficulty of relating the classic motor signs of neurologic disease to speech motor symptoms is found in the dysarthria of athetosis, a subtype of cerebral palsy usually associated with damage to the striatum. Athetosis is characterized by slow, writhing movements, often but not exclusively of the hands and feet, and may be accompanied by a related movement disorder called chorea in which rapid, jerky, and often large movements seem to travel across the body with no apparent purpose. When chorea and athetosis occur together the movements are called choreoathetotis. In the case of speech, the random movements of athetosis or choreoathetosis have been thought of as a form of "motor noise" that contribute in an important way to the dysarthria seen in this form of cerebral palsy. Such a view would predict that speakers with the athetoid form of cerebral palsy and dysarthria would, if asked to produce multiple repetitions of a single utterance, have much more variable articulatory behavior than neurologically normal controls. In this view, the random motions of athetosis or choreoathetosis—the motor noise—would create abnormal movement variability on each repetition. Many years ago Neilsen and O'Dwyer (1984) tested this notion by recording electrical activity from speech muscles in a group of adults with the athetoid form of cerebral palsy and in neurologically healthy (control) speakers. Neilsen and O'Dwyer's findings were surprising and provocative: Athough the speakers with athetosis had clearly abnormal muscle activity for repeated utterances, the variability of the muscle activity across repetitions

was no greater than observed for the control speakers. Neilsen and O'Dwyer's conclusion was that the random movements and fluctuating tone typically said to be classic signs of athetosis did not explain the speech disorder, or at least not the observed muscle activity of articulators producing speech.

One final observation can be made concerning hyperkinetic dysarthria and its tenuous relationship to underlying pathophysiology. The two most common forms of cerebral palsy—spastic and athetotic—are supposed to have very different underlying pathophysiologies (the characteristics of spasticity have been described above). Yet repeated attempts to demonstrate clear distinctions—that is, different types of dysarthria—between speakers with the two forms of this disease have not proved successful (see Jeng, Weismer, & Kent, 2006). If the link between neurologic signs and symptoms and characteristics of the speech production deficit were clear, the two types of cerebral palsy should be associated with fairly different types of dysarthria.[2]

Cerebellum

The cerebellum, like the basal nuclei, is part of what we can call a "motor loop." The cerebellar loop is depicted in Figure 3–7. The cortex of the cerebellum sends fibers to a nucleus deep in the cerebellum (dentate nucleus), which then sends a fiber tract via the pons to the motor thalamus, which in turn projects to the motor cortex. The loop is closed by fibers from the motor cortex that are sent back to the cerebellum, via the pons. This loop is thought to be critical in the sequencing of complex movements and in adjusting the forces applied by different muscles and, hence, the scale of motion of structures moved by those muscles. The cerebellar loop also seems to be involved in motor learning, the trial-by-trial adjustments in muscle force and movement sequencing that transform unskilled to skilled behavior.

[2]The quest to show differences between the hyperkinetic dysarthria of athetoid cerebral palsy and spastic dysarthria of spastic cerebral palsy is more complicated than space allows us to explore in this chapter. Patients with so-called "pure" forms of the disease are difficult to identify, for one thing, meaning that some studies that have failed to find different dysarthria types between the two forms may simply be reporting data on groups that are not that different neurologically. Because several different studies have failed to find the dysarthria difference between the two types, however, it is safe to conclude that an easy link between neurologic signs and symptoms and specific speech production deficits cannot be made at this time.

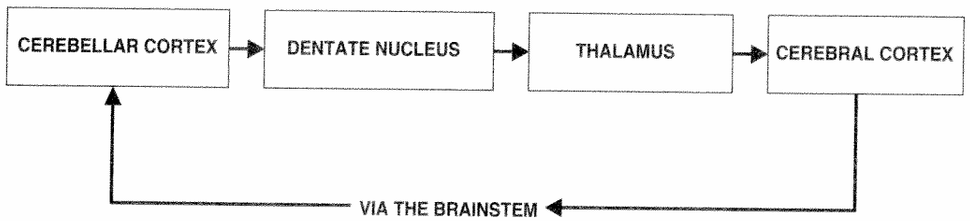

Figure 3–7. Schematic diagram showing the motor loop between the cerebellum and cortex.

Symptoms of Cerebellar Disease

As might be expected, damage to the cerebellum or parts of the cerebellar motor loop result in a disorganization of smooth movement. Muscles are not contracted at the correct times and contractile forces are not scaled appropriately to the needs of the task. Specific neurologic tests of cerebellar disease, which may be associated with strokes, tumors, head injuries, and degenerative conditions, are summarized here.

Tone. When cerebellar disease results in a tone disorder it will typically be in the form of hypotonia. Passive displacement of a limb will create the impression of little resistance, and muscles may feel flabby to the touch.

Weakness. As in most neurologic diseases with a significant motor component, patients with cerebellar disease will not be able to generate much strength and may fatigue easily.

Dysmetria. Patients with cerebellar disease will have problems in the scaling and timing of movements. If asked to point to an object the patient will likely overshoot it and may produce an oscillatory motion around the target. Dysmetria can be shown clearly when the patient is asked to produce a repetitive movement, such as making a repetitive and rhythmic motion with the wrist (typically, a pronating-supinating sequence) or closing and opening the forefinger and thumb with a highly regular pattern. The pattern produced by the patient is likely to appear irregular both in time and space; the motions will appear to lack the smoothly repetitive structure (time) of the neurologically normal person and may vary in amplitude (space) as well, with smaller and larger movements produced across the sequence.

Decomposition of Movement. In cerebellar disease, complex gestures are often broken down into their simple components, giving movements a piece-at-a-time appearance very much at odds with normal movements. The great World War I neurologist Gordon Holmes provided a detailed description of decomposition of movement by observing soldiers with penetrating head injuries to the cerebellum getting out of their field-hospital beds. Neurologically normal individuals typically swing their trunk upright from the bed as they lower their legs to stand, the whole action seeming to be one integrated gesture. In contrast to this, Holmes watched as the injured soldiers moved one leg, then the next, and only rotated their trunks when other required movements for rising from bed were completed. Decomposition of movement gives the appearance of a compensatory strategy, one allowing the patient to accomplish a goal such as getting out of bed without too much jerky movement and incoordination.

Ataxia. The term "ataxia" is often used as a general cover description for the production of clumsy, incoordinated, but purposeful movements; it is also used to designate the staggering, wide-base posture that characterizes walking movements of many persons with cerebellar disease. Reading this, the student may notice that the term "ataxia" seems to encompass the characteristics, summarized above, of dysmetria and decomposition of movement. In fact, Holmes viewed "ataxia" as a cover term for several of the commonly observed motor problems of cerebellar disease, and it is probably best to understand the term in this way.

Cerebellar Disease and Speech Production. Ataxic dysarthria has several prominent characteristics that seem to be consistent with the classic neurologic symptoms of cerebellar disease. For example, patients with ataxic dysarthria are often perceived to have "scanning speech," in which each syllable and possibly each sound seems to be metered out as an independent event, almost as if the smooth flow of speech has been broken apart. In the Mayo studies (Darley, Aronson, & Brown, 1969a, 1969b), excess and equal stress was identified as a common abnormality in patients with cerebellar disease, and it is easy to see how this would contribute to the scanning speech impression. Scanning speech seems to be a speech manifestation of decomposition of movement, wherein the apparently separate sound or syllable events is a simplification of a complex set of movements, perhaps to avoid loss of coordination among the articulators. The drunken-sounding speech and tendency for sudden bursts of voice loudness among persons with ataxic dysarthria seem to reflect the problems in cerebellar disease with control of the scale of motor behavior. Excessive changes in intonation (most notably in the fundamental frequency contour) and sudden increases in voice loudness seem to fit the idea of a motor system that is subject to sudden changes in control.

Section Summary

Section 2 of the model includes two important motor loops, one between the basal nuclei and the cortex, and the other between the cerebellum and the cortex. Both loops are clearly involved with the selection, planning, and execution of complex movements; both loops almost certainly play a role in the learning of such movements. The involvement of these loops in speech motor control is best exemplified by the effects on speech of lesions to the relevant structures and connections between them.

Section 1: Cortical Mechanisms

Section 1 of the model includes several different areas thought to be involved in either the planning or execution of speech production skills. Traditionally, cortical mechanisms have not been considered relevant to a strict definition of dysarthria.[3] Rather, these mechanisms, and their breakdown as a result of neurologic disease, have more often been associated with aphasia and apraxia of speech. Apaxia of speech, originally (and still, by many: see Hillis et al., 2004) thought to involve lesions in Broca's area, was conceived of as a disorder in which the problem was not in the execution stage of speech production, but in the planning stage. The kinds of neuromuscular control problems seen in dysarthria—paralysis, weakness, incoordination—were thought to be absent in patients with apraxia of speech, or at least not responsible for the disordered speech characteristics. Cortical damage was thought to disrupt a patient's ability to plan articulatory sequences, resulting in hesitations, articulatory gropings, impaired prosody, and disproportionate impairment with increased complexity of an articulatory sequence (such as with the addition of syllables to an utterance or increased articulatory complexity within syllables, such as complicated consonant clusters).

[3]Cortical lesions that result in dysarthria are thought to be rare, but have been reported to occur when small strokes create highly localized lesions in the motor cortex serving face and tongue areas (Kim, Kwon, & Lee, 2003).

Damage to the face, tongue, and/or laryngeal areas of the primary motor cortex is an upper motor neuron lesion and, therefore, would be expected to result in spastic dysarthria. This expectation is consistent with preliminary evidence that unilateral, upper motor neuron lesions at *any* location along the corticobulbar tract—in the cortex, internal capsule, or cerebral peduncles (the portion of the corticobulbar tract running in the brainstem but above the level of the brainstem motor nuclei)—produces a uniform kind of dysarthria (Urban et al., 2006).[4] Whether or not this same conclusion applies to *bilateral* lesions along the corticobulbar tract is unknown. For now, the notion that bilateral cortical lesions in face/tongue/larynx areas would produce the same kind of dysarthria as bilateral lesions in the internal capsule must be considered a hypothesis, albeit a reasonable one.

In recent years the role of cortical mechanisms in speech production has been re-examined, largely due to a small number of brain imaging studies of both neurologically normal speakers and persons with speech disorders. Many different areas of the cortex, including the SMA, the insula, the postcentral gyrus (primary sensory cortex), and parts of the premotor cortex, have been implicated in speech production skills. In particular, the anterior insular cortex of the left hemisphere has been claimed to be a sort of clearing house for the coordination of the many muscles involved in normal speech production (Ackermann & Riecker, 2004). If this is so, we would expect lesions of the anterior insula to result in articulatory coordination problems, which would certainly fit with the often assumed presence of coordination difficulties in certain dysarthrias. Interestingly, the insula has also been implicated in apraxia of speech (Dronkers, 1996; but see Hillis et al., 2004, for a different view), which, like certain dysarthrias, has been claimed to have articulatory incoordination as a prominent characteristic (see the review in McNeil, Robin, & Schmidt, 1997). The student should understand that a particular characteristic of speech production, such as incoordination, cannot necessarily be used to differentiate types of dysarthria from one another, or dysarthria from apraxia of speech. The different speech disorders share many characteristics, and there is also some degree of disagreement over the proper diagnosis for some patients. For now, we can say that cortical areas are clearly involved in the production of speech, most likely from the planning to execution stages. Small cortical lesions that produce dysarthrias are likely to resolve quickly, perhaps leaving a mild, residual speech disorder.

Cortical Damage and Speech Production

Presumably, as reviewed above, cortical damage of speech mechanism cells in the primary motor cortex (that is, for the face, tongue, larynx, and so forth) causes a mild dysarthria that often resolves shortly after the onset of the lesion. It is not known how or if these dysarthrias fit into the Mayo system. Damage either in Broca's area, or to the anterior tip of the insular

[4]Urban et al. (2006) do not report a *type* of dysarthria among their patients, but simply that the nature of the dysarthria was the same regardless of the lesion location along the corticobulbar tract.

cortex has been claimed to result in apraxia of speech. Patients with apraxia of speech are said to have increasing speech production difficulty with increasing phonetic complexity; this particular observation, in the absence of easily detectable weakness or other classic neurologic signs (such as hypertonicity) has led clinicians to regard apraxia of speech as a programming disorder. The increased speech production difficulty with increasing phonetic complexity occurs, according to this view, because greater phonetic complexity requires greater programming resources. It is not known, however, if speakers with dysarthria might also produce more errors and have more difficulty initiating speech with increasing phonetic complexity.

Summary

This chapter presented information on brain structures and functions, in the context of motor speech disorders and the Mayo classification system. Emphasis was placed on relationships between what certain regions of the brain are thought to do in the control of movement, and how these functions may affect speech. There is a good deal of uncertainty about the precise relations between lesions of specific areas of the brain and their effects on speech, but a working knowledge of the material presented here is critical to an SLP's skill set, especially as a member of a healthcare team treating persons with neurogenic speech disorders. The student should also keep in mind that much of the information on structures and connections in the nervous system has been presented in schematic form. Fortunately, there are excellent, general resources on neuroanatomy, neurophysiology, and neuropathology, some of which are included in the reference list for this chapter.

References

Ackermann, H., & Riecker, A. (2004). The contribution of the insula to motor aspects of speech production: A review and hypothesis. *Brain and Language, 89*, 320–328.

Blank, S. C., Scott, S. K., Murphy, K., Warburton, E., & Wise, R. J. S. (2002). Speech production: Wernicke, Broca, and beyond. *Brain, 125*, 1829–1838.

Bunton, K., & Weismer, G. (1994). Evaluation of a reiterant force-impulse task in the tongue. *Journal of Speech and Hearing Research, 37*, 1020–1031.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969a). Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research, 12*, 246–269.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969b). Clusters of deviant speech dimensions in the dysarthrias. *Journal of Speech and Hearing Research, 12*, 462–496.

Dronkers, N. F. (1996). A new brain region for coordinating speech articulation. *Nature*, 384, 159–161.

Duffy, J. R. (2005). *Motor speech disorders* (2nd ed.). St. Louis, MO: Mosby.

Guenther, F. H., Hampson, M., & Johnson, D. (1998). A theoretical investigation of reference frames for the planning of speech movements. *Psychological Review*, 105, 611–633.

Hillis, A. E., Work, M., Barker, P. B., Jacobs, M. A., Breese, E. L., & Maurer, K. (2004). Re-examining the brain regions crucial for orchestrating speech articulation. *Brain, 127*, 1479–1487.

Hixon, T. J., & Hoit, J. D. (2005). *Evaluation and management of speech breathing disorders: Principle and methods*. Tucson, AZ: Redington Brown LLC.

Jeng, J-Y., Weismer, G., & Kent, R. D. (2006). Production and perception of Mandarin tone in adults with cerebral palsy. *Clinical Linguistics and Phonetics, 20*, 67–87.

Kandel, E., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed.). New York: Elsevier.

Kent, R. D., Martin, R. E., & Sufit, R. L. (1990). Oral sensation: A review and clinical prospective. In H. Winitz (Ed.), *Human communication and its disorders* (pp. 135–191). Norwood, NJ: Ablex Press.

Kent, R. D., & Tjaden, K. (1997). Brain functions underlying speech. In W. J. Hardcastle & J. Laver (Eds.), *The handbook of phonetic sciences* (pp. 220–255). Oxford, UK: Blackwell Publishers.

Kim, J. S., Kwon, S. U., & Lee, T. G. (2003). Pure dysarthria due to small cortical stroke. *Neurology, 60*, 1178–1180.

Kuehn, D. P., Lemme, M. L., & Baumgartner, J. M. (Eds.) (1989). *Neural bases of speech, hearing, and language*. Boston: College-Hill Press.

Kuoppamäki, M., Rothwell, J. C., Brown, R. G., Quinn, N., Bhatia, K. P., & Jahanshahi, M. (2005). Parkinsonism following bilateral lesions of the globus pallidus: Performance on a variety of motor tasks shows similarities with Parkinson's disease. *Journal of Neurology Neurosurgery and Psychiatry, 76*, 482–490.

McNeil, M. R., Robin, D. A., & Schmidt, R. A. (1997). Apraxia of speech: Definition, differentiation, and treatment. In M. R. McNeil (Ed.), *Clinical management of sensorimotor speech disorders* (pp. 311–344). New York: Thieme.

Neilson, P. D., & O'Dwyer, N. J. (1984). Reproducibility and variability of speech muscle activity in normal and cerebral palsied subjects: EMG findings. *Journal of Speech and Hearing Research, 27*, 502–517.

Pinto, S., Ozsancak, C., Tripoliti, E., Thobois, S., Limousin-Dousey, P., & Auzou, P. (2004). Treatments for dysarthria in Parkinson's disease. *The Lancet: Neurology, 3*, 547–546.

Schulz, G. M. (2002). The effects of speech therapy and pharmacological treatments on voice and speech in Parkinson's disease: A review of the literature. *Journal of Current Medicinal Chemistry, 9*, 1241–1253.

Urban, P. P., Rolke, R., Wicht, S., Keilmann, A., Stoeter, P., Hopf, H. C., & Dieterich, M. (2006). Left-hemispheric dominance for articulation: A prospective study on acute ischaemic dysarthria at different localizations. *Brain, 129*, 767–777.

Wilson-Pauwels, L., Akesson, E. J., Stewart, P. A., & Spacey, S. D. (2002). *Cranial nerves in health and disease* (2nd ed.). Hamilton, Ontario: B. C. Decker.