The Voice and Voice Therapy

ELEVENTH EDITION

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PREFACE

Updates in This Edition

From the first publication of *The Voice and Voice Therapy* in 1971, the authors have attempted to maintain the book's relevance to students and voice clinicians alike. Each edition has incorporated the most current scientific evidence from a variety of disciplines that supports the behavioral approaches to voice assessment and intervention that are core to the text. In this 11th edition, videos demonstrating voice assessment and voice facilitation approaches appear liberally throughout the chapters. These videos show the authors performing each voice facilitation approach with real clients, demonstrating in real time how each technique improves or normalizes voice. Clinically relevant sidebars guide the reader through a series of thought-provoking exercises, illustrating the principles of voice anatomy and mechanics. The 11th edition also features hundreds of self-check quizzes that map onto the chapters' learning outcomes. Each quiz is followed by feedback for all correct and incorrect answers. These pedagogical enhancements, along with the end-of-chapter Praxis questions, support the book's use in the classroom and clinic. The culmination of these elements ensures that students and their instructors, or clinicians and their colleagues, have the most current resources they need for voice and voice-related disorders in a single package. Some chapter-by-chapter highlights include the following:

- Chapter 1, "An Introduction to Voice Disorders and Their Management," features new data on the incidence and prevalence of voice disorders in the general population and in specific populations, such as children, teachers, and those who use teleconferencing. There are expanded discussions of the classification of voice disorders, approaches to assessment and stimulability probes, and managing the person with dysphonia.
- Chapter 2, "Normal Voice: Anatomy and Physiology Throughout the Lifespan," has been expanded considerably. It features a more comprehensive description of the anatomy and physiology of normal voice production. The detailed, colored anatomical illustrations and accompanying captions make it easy for the reader to "put it all together." This chapter can stand alone, thus eliminating the need for students, instructors, or clinicians to refer to outside source material.
- Chapter 3, "Functional Voice Disorders," presents practical approaches to identifying and managing behaviorally based voice disorders across the age spectrum. The chapter includes expanded discussions of excessive laryngeal muscle tension and the benign laryngeal pathology that may develop as a result, as well as voice disorders with a psycho-emotional basis or overlay. Special attention is given to the emotional and/or behavioral issues that might result in children with functional voice disorders. Several perceptual measures of voice administered to adults with dysphonia are also explored.

We also review evidence-based practice (EBP) studies supporting the value of our voice facilitating approaches in treating persons with functional or psychogenic dysphonia.

- Chapter 4, "Organic Voice Disorders," presents practical approaches to identifying and managing organic voice disorders. The chapter includes significant updates on the risks for, and assessment and treatment of, gastroesophageal reflux diseases and laryngopharyngeal reflux. Discussions of endocrine changes and juvenile recurrent respiratory papilloma have also been expanded. We present current literature on the medical management of all organic disorders and on the role of the voice clinician in evaluation and therapy.
- Chapter 5, "Neurogenic Voice Disorders," presents the latest research in the behavioral, pharmacological, and surgical management of neurogenic voice disorders. Emphasis is placed on medical and behavioral interventions for unilateral vocal fold paralysis and laryngeal dystonia, the most common being adductor laryngeal dystonia. Updated research on the diagnosis and management of organic voice tremor is also covered. We review numerous EBP studies supporting the value of our voice facilitating approaches in treating the respiration, phonation, and resonance subsystems in persons with dysarthria in general and in hypokinetic dysarthria in particular. Behavioral approaches that increase the respiratory support, vocal quality, and intelligibility in Parkinson's disease are described and supported by strong clinical research.
- Chapter 6, "Evaluation of the Voice," has been updated extensively. It features the latest approaches to the auditory-perceptual evaluation of the voice and to assessment of voice-related quality of life. Multiple case studies illustrate both instrumental and noninstrumental assessment of the voice across medical and educational settings. These case studies also provide a framework for report writing and special considerations for voice populations across the lifespan. New figures illustrate instrumental approaches to identifying and quantifying voice and resonance disorders. Over a dozen tables present the student and clinician with the latest normative data across the lifespan for a variety of acoustic, aerodynamic, and related voice measures. This chapter can stand alone, thus eliminating the need for students, instructors, or clinicians to refer to outside source material.
- Chapter 7, "Voice Facilitating Approaches," continues to be the bedrock of this textbook. We retained our core set of 25 voice facilitating approaches with updated procedural approaches and EBP summaries that support their value in treating persons with dysphonia. Many of the cases illustrating the approaches have been updated to reflect the types of patients seen in current clinical practice, including applications for audiovisual feedback in therapy. We also discuss current literature on patient compliance and barriers to treatment and present updates on voice intervention via telepractice.
- Chapter 8, "Therapy for Specific Patient Populations," features expanded discussions of the identification and management of older adults and children with dysphonia. We explore direct versus indirect therapy approaches and the contribution of family members to the therapeutic process. We discuss in greater detail the professional voice user, notably educators, and the management of dysphonia in this increasing population of patients, including vocal hygiene programs. We discuss the management of dysphonia in children and adults with hearing impairment and in those with a variety

of respiratory-based conditions. Gender-affirming voice therapy supports gender-diverse individuals as they explore and develop a healthy voice that feels congruent with their gender and sense of self. This expanded section provides an extensive overview of those aspects of voice and speech most salient to the perceptions of a speaker's gender and cites studies on expert consensus as to the recommended approaches to voice training.

- Chapter 9, "Management and Therapy Following Laryngeal Cancer," features detailed discussion of the medical management of patients with laryngeal cancer and the role of the voice clinician in evaluation and therapy. The illustrations and photographs that accompany the chapter are powerful learning tools for the student and clinician and can also serve as effective teaching tools for the patient. Communication options post laryngectomy are discussed in detail, including the artificial larynx, esophageal speech, and tracheoesophageal speech.
- Chapter 10, "Resonance Disorders," features both the instrumental and noninstrumental assessment of persons with disorders of nasal or oral resonance. Hypernasality, hyponasality, and assimilative nasality are discussed in depth, along with medical and behavioral approaches to each disorder. We expanded the chapter's discussion of the team management of persons with cleft palate speech. Application of our voice facilitating approaches to treatment of resonance disorders is illustrated.

Close to 1,200 references to research studies are included throughout the text. Cardinal literature from the past 50 years of voice science and care is included as well as the most current literature from a variety of disciplines. Greater than half the references are new in this edition, with the majority representing advances in our field from the year 2018 to the present.

Pedagogical elements supporting the use of the book for teaching include the following:

- The *Learning Objectives* at the beginning of each chapter have been expanded.
- *Self-check multiple-choice quizzes* are embedded in each chapter, complete with answer feedback for all correct and incorrect answers.
- *Clinical Sidebars* reinforce clinical application of material.
- *Clinical Concepts* at the end of select chapters reflect many of the learning objectives.
- *Guided Reading exercises* at the end of select chapters reference key clinical articles. The articles might be accessible via your university's library, ASHA, or can be purchased online.
- *Multiple-choice questions (Preparing for the Praxis)* at the end of select chapters help readers master the type of content covered in the Praxis II examination in speech-language pathology.
- A PluralPlus companion website is provided with updated and robust supplemental teaching materials, including PowerPoint slides for each chapter, an instructor resource manual, and a test bank.

For students, the 11th edition comes with the ancillary materials on a PluralPlus companion website. The study aids include the following:

- *Video examples.* Embedded videos provide an illustration of a key principle or concept in action. These video examples show the authors demonstrating the use of voice facilitating approaches with persons with voice disorders.

PROLOGUE

The Voice and Voice Therapy was first published by Prentice-Hall in 1971, emerging before cellphones or any hint of help from digital technology. At that time, Dr. Daniel Boone wrote that much of the early voice literature was dominated by otolaryngology for "organic" voice problems and by psychiatry for "functional" voice problems. Much of the voice disorders literature in the middle-1960s and earlier references were based on this organic–functional dichotomy. Prominent in the speech pathology literature were two "organic" voice disorders: (a) resonance problems with focus given to hypernasality and cleft palate and (b) the clinical management after laryngectomy centered on the teaching of esophageal speech.

The "functional" voice disorders literature in the 1960s was based on the premise of dysphonias being caused by continuous abuse and misuse of the voice. Reactive to such excessive vocal effort, vocal fold tissue changes could develop, producing such physical lesions as nodules, polyps, or contact ulcers. However, many children and adults with such hyperfunctional voice problems seen in our clinics showed no laryngeal tissue changes. Their common voice problems were alterations in loudness, inappropriate pitch, poor voice quality, and faulty resonance.

As physicians began referring more people with either "organic" or "functional" voice disorders, the typical speech-language pathologist (SLP) experienced a larger caseload of patients with voice disorders. With this growing demand for voice therapy, many SLPs increased their search for more knowledge about voice disorders and their treatment. Literature search was supplemented by increasing attendance at voice disorder workshops scattered around the country. Dr. Boone wrote that he remembered well attending such workshops—receiving excellent descriptions of the vocal mechanisms but sorely lacking information about voice therapy strategies and demonstrations of therapy procedures.

As a young professor in the 1960s teaching voice disorders, Dr. Boone developed a voice therapy file for each of three clinical voice components: respiration, phonation, and resonance disorders. His sources for the therapy procedures came from workshops, a scant literature including clinical texts, other SLPs and voice scientists in his profession, drama and singing teachers, and observations of a few superior users of voice. Since there were no smartphones, emails, web pages, or Google-type resources available for therapy suggestions, his therapy file showed what to do and how to do it.

Dr. Boone used this therapy file and other parts of his voice course materials when writing *The Voice and Voice Therapy*. He selected 25 therapy techniques, labeling them in the text as "Facilitating Techniques." Four subheadings under each technique illustrated the kind of problem for which the approach could be useful, its procedures, a case history example, and an evaluation of the approach. The SLP would apply a therapy technique with the patient. If it worked, it would remain as part of the therapy regimen. If it was not helpful, it was replaced by another one of the techniques. The 25 techniques featured in the first edition eventually grew into voice facilitating

CHAPTER 5

NEUROGENIC VOICE DISORDERS

LEARNING OUTCOMES

After reviewing this chapter, one should be able to:

- Describe the roles of the central and peripheral nervous systems in the innervation of the vocal mechanism.
- Identify the major role of each cranial nerve involved with voice production.
- Describe the origin in the nervous system and typical course for vocal fold paralysis. Identify the latest in surgical, pharmacological, and behavioral management of vocal fold paralysis.
- Identify the origin in the nervous system and typical course for spasmodic dysphonia and essential voice tremor. Identify the latest in surgical, pharmacological, and behavioral management of these disorders.
- Identify the origin in the nervous system and typical course for Parkinson's disease, cerebrovascular accident, and traumatic brain injury. Identify the latest in surgical, pharmacological, and behavioral approaches to these neurogenic-based voice and speech disorders.

In Chapter 2, we reviewed the normal anatomy and physiology required for voice. We considered the causes and treatment of several non-neurogenic voice disorders in Chapters 3 and 4. In this chapter we review the neurological structures and processes that must function in coordinated balance to produce normal voice. By gaining an appreciation of the neurophysiological bases of voice, we can then begin to recognize and pinpoint the causes of neurogenic dysphonia, the focus of this chapter. As Duffy (2020) suggests, speech changes can be the first or only a manifestation of neurogenic disease. Recognition of speech changes can have a significant impact on medical diagnosis and care. Indeed, on numerous occasions, the speech-language pathologist (SLP) has been the first to identify the salient features of myasthenia gravis (MG), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), and even progressive supranuclear palsy (PSP). Only through early detection and differential diagnosis are the SLP and others on the patient's health care team able to generate an intervention program that directly addresses the patient's deficits and long-term communication options.

To understand the complexities of neurogenic dysphonia, it is necessary to have an understanding of the innervation of the larynx and resonators from the central and peripheral nervous systems. Note that a comprehensive discussion of the neuroanatomical and neurophysiological bases of phonation is beyond the scope of this text. Readers are directed to textbooks by Brookshire (2014), Abou-Khalil and Webb (2023), and Duffy (2020). In this chapter, however, we offer a working view of the central nervous system (CNS), the peripheral nervous system (PNS), and innervation of the muscles necessary for voice.

A Working View of the Nervous System

The CNS and the PNS coordinate all laryngeal operations, from the elevation of the larynx for swallowing, to the tri-level valve closure required for a cough, to the nuanced vocal fold vibrations of the operatic lyric soprano. We know far less about the neural controls required for human singing and talking than we do about the neural governing of laryngeal vegetative functions such as breathing, coughing, or swallowing. The human not only has all the sensorimotor structures and functions of most mammals but also has added abilities to subdue or augment response (e.g., suppress crying when the situation is not appropriate) or to use the voice for emotional or artistic expression. The expanded cerebral cortex unique to humans enables one to use voicing cues (e.g., pitch and loudness inflections) for speaking, singing, and other forms of verbal communication.

The Central Nervous System, the Cortex, and Its Projections

The CNS is composed of the brain and spinal cord and is located within the bony, protective structures of the cranium and vertebral column. Sensory and motor areas within the cerebral cortex, cerebellum, and basal ganglia contribute to production of voice (Abou-Khalil & Webb, 2023; Duffy, 2020). Researchers suggest that both the frontal and left temporal lobes are primarily, though not exclusively, involved with the motor aspects of voice production, while the bilateral parietal lobes provide important sensory feedback about voice production (Baldo et al., 2011; Chen et al., 2021; Dronkers, 1996). Initiation of voice begins in the inferior and lateral aspects of

the primary motor cortex (Castellucci et al., 2022; Parkinson et al., 2012). Nerve impulses are then sent primarily via the corticobulbar tract to brainstem nuclei, in particular, the nucleus ambiguus. Other cortical areas, such as the premotor cortex, the supplemental motor cortex, and Broca's area, contribute to planning and programming phonation, relying on input from the cerebellum and basal ganglia (Ferrand, 2012).

Normal voice also depends on one's ability to hear and process ongoing voice production. The temporal lobes provide cortical input for audition. Heschl's gyrus, the primary auditory cortex, bilaterally receives tonotopic frequency input from the medial geniculate bodies of the thalamus. Other auditory areas, such as the auditory association area and Wernicke's area, may also play a role in processing one's own voice production (Friederici, 2011; Kapsner-Smith et al., 2024).

Pyramidal and Extrapyramidal Tracts

The pyramidal and extrapyramidal tracts are part of the CNS. The pyramidal tract is composed of long axons that extend from the cortical neurons located in the primary motor strip and travel uninterrupted until they reach their corresponding cranial nerve nuclei in the brainstem. As illustrated in Figure 5–1, the pyramidal tract is composed of white-matter nerve fibers (corticobulbar

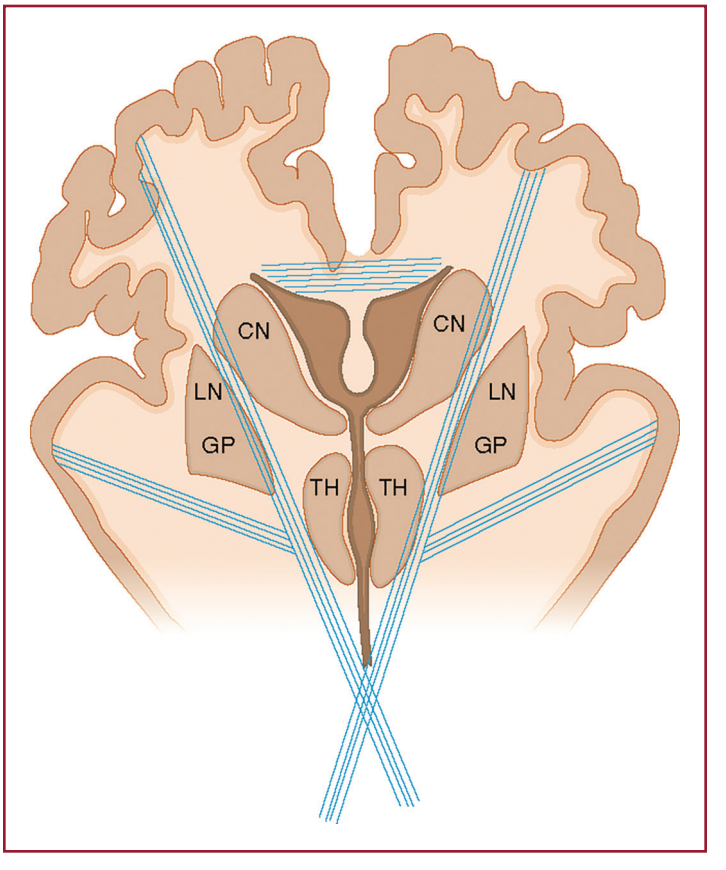

FIGURE 5–1. Schematic view of the pyramidal tract. The pyramidal tract is like a neural turnpike with fibers descending uninterrupted via the internal capsule from their cortical origins to their terminations at cranial nerve nuclei in the brain stem. This line drawing shows basal ganglia (including CN, caudate nucleus; LN, lenticular nucleus; GP, globus pallidus), and thalamus (TH). Pyramidal fibers are depicted as a thickened line.

and corticospinal) that pass in a bundle between the basal ganglia and the thalamus, which is called the internal capsule.

One way to think of the pyramidal tract is that it functions like a neural turnpike, permitting the transmission of impulses from the cortex to the cranial nerve nuclei without interruption of local neural traffic. Conversely, the extrapyramidal tract (Figure 5–2) is similar to a country road, with fibers stopping in many locations, bringing neural transmissions to synapses with the basal ganglia, across to the thalamus and the subthalamus, and to the cerebellum, among other structures. The extrapyramidal tract enables extensive checking and balancing of sensory and motor information with its many interconnections among the cortex, thalamus, and the basal ganglia. The many checks and balances afforded by the extrapyramidal system are crucial for maintaining posture, tone, and associated activities that provide a foundation for skilled movements executed by the pyramidal tract.

Thalamus, Internal Capsule, and Basal Ganglia

The subcortical areas occupied by the thalamus, which is medial in the hemisphere; the internal capsule that runs laterally adjacent to it; and the more lateral basal ganglia are known collectively as the corpus striatum, which gets its name from the contrast of the gray matter nuclei and the white-matter projections between them. The corpus striatum is the site of most of the sensorimotor integrations of the cerebrum. The thalamus is to sensation what the basal ganglia are to motor behavior.

Even the thalamus has its posterior (pure sensory) and anterior (sensory-influenced motor) divisions. The posterior thalamus is known as the pulvinar body and receives neural impulses

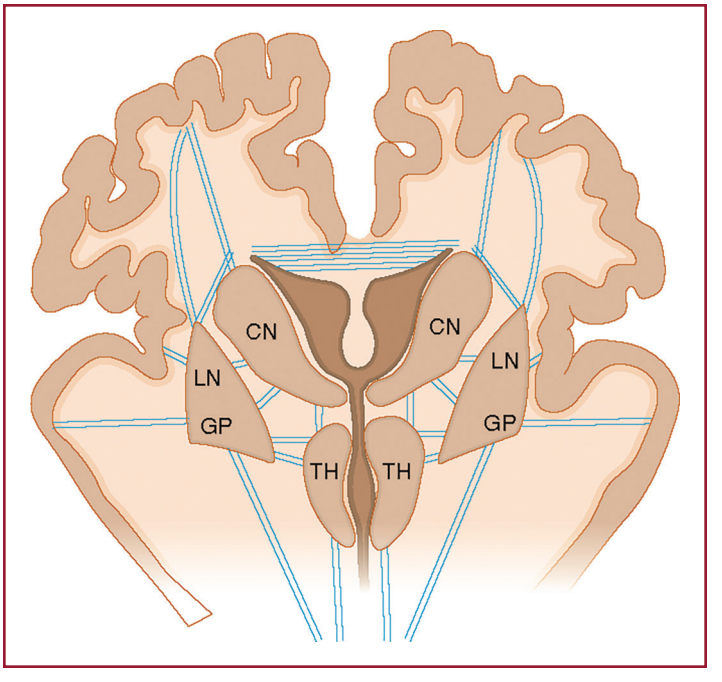

FIGURE 5–2. Schematic view of the extrapyramidal tract. The line drawing of the extrapyramidal tract depicts its neural fibers like a neural country road, starting and stopping at various cortical, basal ganglia, and thalamic sites and ending (or starting) at lower brainstem sites. These extrapyramidal fibers are depicted as a thickened line. This line drawing shows the basal ganglia (including CN, caudate nucleus; LN, lenticular nucleus; GP, globus pallidus); and TH, thalamus.

from the auditory tract via the medial geniculates, the most inferior–posterior of the pulvinar. From the medial geniculates and after some central mixing within the thalamus, the auditory fibers radiate in a bundle superiorly to the primary auditory cortex, Heschl's gyrus. Similarly, the visual fibers come into the lateral geniculate bodies of the pulvinar section of the thalamus, undergo central mixing, exit in a bundle, and go directly to the primary visual cortex in the occipital lobes.

It is hypothesized that afferent–efferent fibers between the lateral wall of the pulvinar body and the temporale planum play an important role in auditory comprehension of the spoken word and have some control in producing vocal response. Within the main thalamic body, there appears to be much integration of sensory information occurring, getting organized for some kind of motor response via the anterior nuclei and ventral anterior nuclei of the thalamus. From the anterior thalamus, sensory projections go either directly to the sensory cerebral cortex or to nuclei within the basal ganglia.

While there are some basal ganglia–thalamic connections crossing within the internal capsule, the main body of the internal capsule is largely composed of the descending–ascending neural projections of the pyramidal tract. The internal capsule area of the brain is highly susceptible to cerebrovascular accidents (CVAs), also known as strokes, primarily because much of its blood supply is furnished by an artery known as the lenticular striata (often called the artery of apoplexy), which for some reason seems to be blocked by thrombosis more than other cerebral arteries. Such blockage of blood causes white-matter projections to die, resulting in contra-unilateral symptoms of paralysis (note that such a high-level lesion would not cause contralateral vocal fold paralysis). Any lesion (disease, stroke, or trauma) to the internal capsule could cause contralateral sensorimotor symptoms of skeletal muscles; these lesions are classified as upper motor neuron lesions. Sensory loss could include hypothesia, and motor loss would be seen in hemiparesis or hemiplegia (paralysis with hypertonicity and spasticity) (Duffy, 2020).

The basal ganglia utilize the sensory information provided by the thalamus. The main nuclei of the basal ganglia are the caudate nuclei and the lenticular nuclei, which include the putamen and globus pallidus. Bilateral innervations of both smooth and striated muscle occur within both the caudate and lenticular nuclei, and at this level, we first see bilateral innervation of velar, pharyngeal, and laryngeal muscles. The basal ganglia utilize the continuous, multiple sensory information from the thalamus in organizing appropriate motor responses, including vocalization (Behrman et al., 2020).

Neurotransmitters

It should be acknowledged at this point that the transmission of neural impulse among various nuclei via white-matter nerves is facilitated by several enzymes known as neurotransmitters. At the termination of nerves within the cerebrum, where neural synapses occur, serotonin functions as a nervous system neurotransmitter. The sympathetic nervous system employs epinephrine and norepinephrine to aid in the transmission of neural impulses for innervation of smooth muscle, glands, and viscera. The basal ganglia depend on dopamine as the primary neurotransmitter. The facial, neck, and skeletal muscles depend on acetylcholine as the chemical mediator between the muscle's nerve nucleus and the muscle body itself. While neural transmission can be altered or

stopped by isolated lesions to the gray body or its nerve connections, many of the diseases of the CNS cause inhibition or overproduction of neurotransmitters. For example, it is well known that degenerative changes in the substantia nigra cause a deficiency in a chemical neural transmitter known as dopamine in the caudate nucleus and putamen. The disturbed basal ganglia and extrapyramidal control circuit results in a hypokinetic dysarthria observed in Parkinson's disease (PD) (discussed later in this chapter). The symptoms of PD are vastly minimized by levodopa and carbidopa, a synthetic dopamine (Mayo Clinic, 2023).

The Brainstem and the Cerebellum

The projection fibers from both the pyramidal and extrapyramidal tracts extend anteriorly into the pons and posteriorly via the cerebral peduncle terminating into the medulla oblongata. This cortical to lower center tract includes both afferent and efferent fibers. There are neural connections from the midbrain to the pons and then to the cerebellum, and connections from the peduncle area into the cerebellum. The medial hypothalamus is the lowest structure of the midbrain; under it are the lesser (in number) gray bodies and myelinated nerve tracts (innumerable) that comprise the brainstem. The hypothalamus forms the lateral walls of the central third ventricle. Connected to it are some gray bodies hugging the third ventricle aqueduct; these gray bodies contain an important vegetative respiratory area known as the periaqueductal gray (Davis et al., 1996; Duffy, 2020). Hypothalamic fibers and pyramidal and extrapyramidal projections communicate anteriorly in the brainstem to the pons, while posterior fibers form the cerebral peduncle, which extends down, forming the medulla. The medulla extends from the lowermost portion of the pons, with its upper portion forming the floor of the fourth ventricle.

The cerebellum wraps around the pons and cerebral peduncle and has many interconnections with the pons, cerebral peduncle, medulla, and spinal cord. The cerebellum functions as the great regulator of the extrapyramidal tract, coordinating sensory information (proprioceptive, kinesthetic, tactile, auditory, and visual) with coordinated motor response. Lesions to the cerebellum from trauma or disease cause speech symptoms of incoordination, known as ataxic dysarthria. The voice–speech symptoms of cerebellar lesions are prosodic slowdown (scanning speech); abrupt and unpredictable changes in resonance, pitch, and loudness; and reduced articulatory accuracy for speech, all sounding like the speech of someone highly intoxicated.

Eighty percent of the descending projection fibers coming from the cerebral peduncle cross over (decussate) to the other side in the medulla just below the brainstem; 20% remain ipsilateral (on the same side). Of great importance to voice is the nucleus ambiguus in the superior medulla, located just below the pyramidal decussation. As the medulla extends downward, it begins to narrow into the spinal column. The same posterior–sensory/anterior–motor organization continues in the medulla and down into the spinal cord. Posterior nerve tracts and gray nuclei (left and right) are sensory in nature, while the anterior white-matter tracts and anterior horn nuclei (left and right) execute motor function.

Let us consider briefly what constitutes an upper motor neuron (UMN) lesion or a lower motor neuron (LMN) lesion. Functionally, a bilateral UMN lesion produces symptoms of hypertonicity, such as in a CVA (stroke) in which the patient may experience hemiparesis or hemiplegia (one-sided weakness or full paralysis of extremities, respectively). Functionally, an LMN lesion

results in flaccidity and muscle atrophy, such as when the cutting of the recurrent laryngeal nerve (RLN) during surgery causes unilateral vocal fold paralysis. UMNs begin at the cerebral cortex and end at the nucleus ambiguus; LMNs begin at the nucleus ambiguus and travel down the spinal cord, ending at the lowest spinal nucleus. Also included as LMNs are the nerves exiting from the pons and medulla (such as the cranial nerves), and the nerves that carry sensory and motor impulses to and from the various spinal nuclei for their particular muscles. The autonomic motor system and these cerebrospinal nerves, including their associated sensory receptors, constitute the PNS.

The Peripheral Nervous System

We limit our discussion of the PNS primarily to the cranial nerves that have a direct impact on voice and to two branches of cranial nerve X (vagus)—the superior and recurrent laryngeal nerves—which innervate the larynx.

Cranial nerves V, VII, and VIII have a direct impact on speech, but they do not appear primary in the production of voice. Cranial nerve V, trigeminal, emerges from the pons with its primary motor fibers innervating the muscles of mastication; the sensory components that might influence voice are the tactile sensations of the nose and oral mucosa. Cranial nerve VII, facial, leaves the lower portion of the pons and terminates in its motor innervation of facial muscles; its sensory components include taste in the anterior two-thirds of the tongue and sensation to the soft palate. Cranial nerve VIII, acoustic, has its cochlear division ending in the dorsal and ventral cochlear nuclei in the superior medulla; leaving the cochlear nuclei, the auditory pathways begin and continue to various neural stations, ending in Heschl's gyrus in the temporal lobe. As mentioned earlier in this chapter and throughout the text, the auditory system appears to play a primary role in voice production and control.

Cranial Nerves (IX, X, XI, XII)

We give special attention to cranial nerves IX, X, XI, and XII because each has some role in phonation and voice resonance. For each nerve, we look at origin and insertion with a brief statement relative to nerve function, especially as it relates to voice.

Cranial Nerve IX, Glossopharyngeal. Originating laterally in the medulla, the nerve passes through the jugular foramen coursing between the internal carotid artery and the external jugular vein and subdivides into its numerous branches that go to various innervation sites. Its functions include taste in the posterior third of the tongue and sensation to the fauces, tonsils, pharynx, and soft palate. Its primary motor innervation is to the superior pharyngeal constrictor in the pharynx and to the stylopharyngeus muscle.

Cranial Nerve X, Vagus. The vagus nerve, in addition to its many functions of control of the autonomic nervous system involving thoracic and abdominal viscera, has two important branches that innervate the larynx: the superior laryngeal nerve (SLN) and the recurrent laryngeal nerve (RLN) (Figure 5–3). In the next section of this chapter, we present in detail the origins and