

THE VOICE AND VOICE THERAPY (with Free DVD), 7/e

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CHAPTER 4

Neurogenic Voice Disorders

In previous chapters we have talked about the normal anatomy and physiology required for voice (Chapter 2). We have considered the causes and treatment of a number of voice disorders (Chapter 3). In this chapter, we review the neurological structures and processes that must function in coordinated balance to produce what we perceptually consider as normal voice. By gaining an appreciation of the neurophysiological bases of voice, we can then begin to recognize and pinpoint the causes of neurogenic dysphonias. As Duffy (1995) suggests, changes in speech can be the first or only manifestation of neurogenic disease. Recognition of these changes can have a significant impact on medical diagnosis and care. Indeed, on numerous occasions the voice clinician has been the first to identify the salient features of myasthenia gravis, Parkinson's disease, and even progressive supranuclear palsy. Only through early detection and differential diagnosis are the voice professional and the patient's health care team able to generate an intervention program that directly addresses the patient's deficits.

To understand the complexities of neurogenic dysphonias, it is necessary to have an understanding of the innervation of the larynx and resonators from the central and peripheral nervous system structures. A comprehensive discussion of the neuroanatomy and neurophysiology of phonation is beyond the scope of this book; readers are directed to excellent texts by Duffy (1995), Yorkston et al. (1999), and Darley, Aronson and Brown (1975) for a discussion of speech science, anatomy and physiology, and motor speech disorders. However, we do offer a working view of the central and peripheral nervous system and innervations of the muscles necessary for voice.

A Working View of the Nervous System

The central nervous system (CNS) and the peripheral nervous system (PNS) coordinate all laryngeal operations, from the elevation of the larynx for swallowing, to the triple valving closure (true vocal folds, ventricular folds, and aryepiglottic folds) required for a cough, to the delicate nuance sung by 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 in all mammals (including the human) of such laryngeal vegetative functions as breathing, coughing, or swallowing. The human not only has all the sensory–motor structures and functions of most mammal species, but has added abilities to subdue or augment response (for example, suppress crying when the situation is not appropriate), and the ability to use phonatory functions for expression of emotions, or in verbal and nonverbal communication or artistic expression. The expanded cerebral cortex unique to the human species has much to do with the enabling of the human to use voice in a controlled sequential pattern as heard or said in spoken language, or in the exact pitch and loudness requirements of singing, or in the voicing cues (inflection, loudness changes, etc.) we use in spoken communication.

The Central Nervous System (CNS), the Cortex, and Its Projections

The central nervous system is composed of the brain and spinal cord and is housed in the bony, protective structures of the cranium and vertebral column. Sophisticated sensory–motor function, such as formulation of speech and voice, appears to be directed by the cerebral cortex, a six-layer composite of millions of neurons, interconnected by their dendrites and axons. Researchers suggest that both the frontal and temporal lobes are primarily involved with the production of voice, although these areas do not represent the only structures involved in sensory–motor programming for voice. The motor cortex for laryngeal control is in the inferior and lateral aspects of the motor cortex and primary motor strip. The third frontal convolution, or Broca’s area, in the left hemisphere, has much to do with preplanning a motor speech act, including voice response. For example, in regional cerebral blood flow studies, Broca’s area shows greater density of blood just before something is said. The actual production of the utterance at the cortical level activates bilaterally at specific locations along the precentral gyrus. The projections from the cortex are polysynaptic, passing to the midbrain and then to the brainstem.

It appears that the insula (older cortex phylogenetically) medial to the temporal lobe plays an active role in motor planning for voice and speech (Dronkers, 1996; Dronkers, Redfern, and Shapiro, 1993). Later review by Bennet and Netsell (1999) suggests that the insula may be involved in far more than motor planning and that it may be associated with all aspects of speech and language processing. The temporal lobes, in turn, provide cortical input for audition. Heschl’s gyrus, the

primary auditory cortex, receives tonotopic frequency input from the medial geniculate bodies of the thalamus. New research investigating the relationship of linguistic (phonetic) and extralinguistic (voice) information in preattentive auditory processing suggests a parallel and contingent process (Strouse et al., 1998).

Speech comprehension is associated with Wernicke's area, which communicates directly with Broca's convolution via the bundle of association fibers known as the arcuate fasciculus. The actual execution of voice may be dependent on temporal cortical connections to lower brain centers, such as from the temporal planum of the cortex to the pulvinar body of the thalamus (Minckler, 1972).

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 4.1, the pyramidal tract

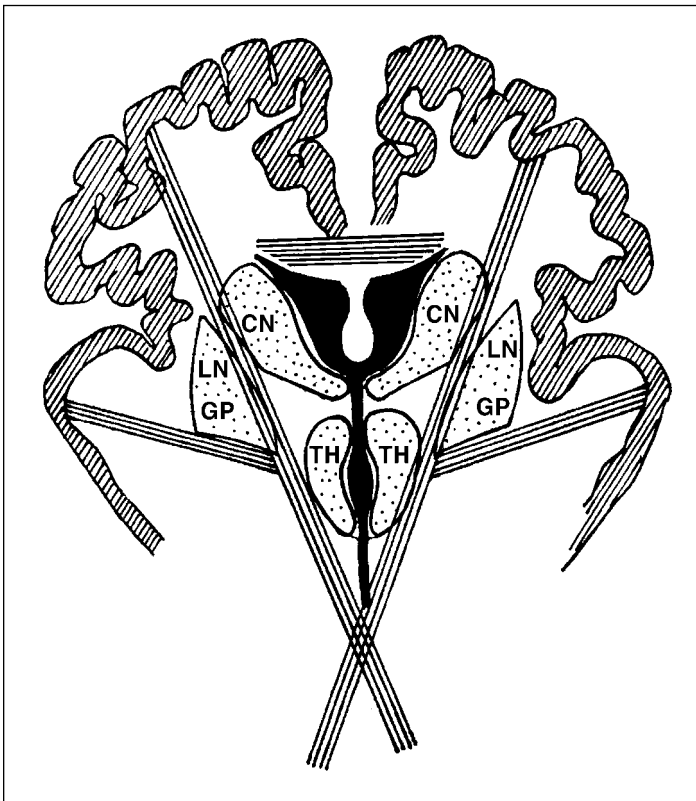



FIGURE 4.1 A Schematic View of the Pyramidal Tract. The pyramidal tract is like a neural turnpike with fibers descending uninterrupted via the internal capsule from their cortical origins to their terminations at cranial nerve nuclei in the brain stem. This line drawing shows basal ganglia (including CN, caudate nucleus; LN, lentiform nucleus; GP, globus pallidus) and TH, thalamus. Pyramidal fibers are depicted as .

is composed of white matter nerve fibers (corticobulbar and corticospinal) that pass in a bundle between the basal ganglia and the thalamus, which is called the internal capsule.

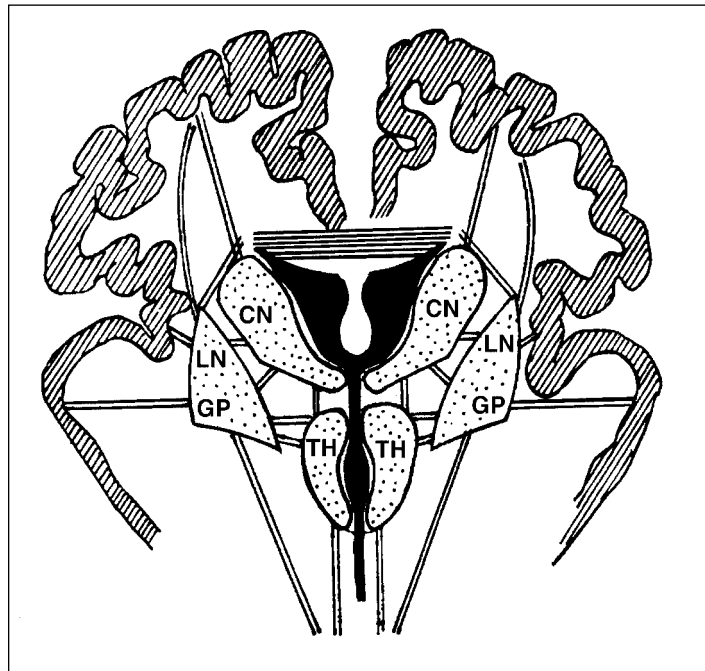
One way to think of the pyramidal tract is that it functions like a neural turnpike, permitting the transmission of impulses from the cortex to the cranial nerve nuclei without interruption of local neural traffic. Conversely, the extrapyramidal tract (Figure 4.2) is similar to a country road, with fibers stopping in many locations, bringing neural transmissions to synapse 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 between the thalamus and the basal ganglia. It is suggested that 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 the 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. It 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

FIGURE 4.2 A

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 ————. This line drawing shows the basal ganglia (including CN, caudate nucleus; LN, lenticular nucleus; GP, globus pallidus); and TH, thalamus.



most of the sensory–motor 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, which receives neural impulses from the auditory tract via the medial geniculates, the most inferior–posterior of the pulvinar. From the medial geniculates, 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, and exit in a bundle and go directly to primary visual cortex in the occipital lobes.

There is also some speculation (Boone, 1996, 1998; Minckler, 1972) 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 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 cerebral vascular accidents, 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 would cause white matter projections to die, resulting in contra-unilateral symptoms of paralysis (note: such a high-level lesion would not cause contralateral vocal fold paralysis). Any lesion (disease, CVA, or trauma) to the internal capsule could cause contralateral sensory–motor symptoms of skeletal muscles, classified as upper motor neuron lesions. Sensory loss could include hypesthesias and motor loss would be seen in hemiparesis or hemiplegia (paralysis with spasticity).

The basal ganglia utilizes the sensory information provided by the thalamus. The main nuclei of the basal ganglia are the caudate nuclei and the lenticular nuclei, which includes 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).

Neurotransmitters. It should be acknowledged at this point that the transmission of neural impulse between 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 are dependent on dopamine as the primary neurotransmitter. The facial, neck, and skeletal muscles are dependent 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 neurotransmitter solutions.

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, discussed later in this chapter. The symptoms of Parkinson's are vastly ameliorated with levodopa, a synthetic dopamine.

The Brain Stem and the Cerebellum. The projection fibers from both the pyramidal and extrapyramidal 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 on to the cerebellum and connections from the peduncle area into the cerebellum. The medial hypothalamus is the lowest structure of the midbrain, under which are the lesser (in number) gray bodies and myelinated nerve tracts (innumerable) that compromise the brain stem. The hypothalamus forms the lateral walls of the central third ventricle and connected to it are some gray bodies hugging the third ventricle aqueduct, containing important vegetative respiratory areas known as the periaqueductal gray (Davis et al., 1996). Hypothalamic fibers, pyramidal, and extrapyramidal projections communicate anteriorly in the brain stem 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, 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), changes in resonance, and inarticulate 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 brain

stem; 20% remain ipsilateral. 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 at this point what constitutes an upper motor neuron lesion or a lower motor neuron lesion. Functionally, an upper motor lesion produces symptoms of spasticity, such as in a CVA in which the patient may experience hemiplegia (one-sided spastic paralysis of extremities). A lower motor lesion, such as the cutting of the recurrent laryngeal nerve, causes unilateral vocal fold flaccid paralysis. Upper motor neuron function begins at the cerebral cortex and ends at the nucleus ambiguus; lower motor neuron function begins at the nucleus ambiguus and travels down the spinal cord, ending at the lowest spinal nucleus. Also included as lower motor neuron structures 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 what is known as the peripheral nervous system (PNS).

The Peripheral Nervous System (PNS)

We will limit our discussion of the peripheral nervous system primarily to those cranial nerves that have direct impact on voice, and, in particular, two branches of cranial nerve X, the vagus, that innervate the larynx: the superior and recurrent laryngeal nerves.

While cranial nerves V, VII, and VIII have direct impact on speech, 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 will give special attention to cranial nerves IX, X, XI, and XII as each has some role in phonation and voice resonance. For each

nerve, we will 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, 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). In the next section of this chapter, we will present in greater detail the origins and functions of the SLN and the RLN. The vagus nerve originates in the nucleus ambiguus in the medulla from which it emerges laterally and courses its way, with continuous branching along the way, with particular branches terminating at the various innervation sites from the pharynx to the abdominal viscera. Affecting voice are the sensory components of the vagus with sensory innervation of the pharynx and larynx; motor aspects affecting voice include innervation of the velum, base of tongue, superior, middle, and inferior pharyngeal constrictors, larynx, and autonomic ganglia of the thorax (affecting the respiratory aspects of phonation).

Cranial Nerve XI, Spinal Accessory. Cranial nerve XI is a motor nerve that has innervation of the neck accessory muscles as its primary function. It is composed of two sections, the cranial portion and spinal portion. The cranial branch originates in the nucleus ambiguus and emerges from the side of the medulla with five successive small rootlets. Some fibers are distributed to the superior branches of the vagus nerve, innervating the levator veli palatini and uvula. Fibers from the spinal portion of the nerve originate from the anterior horn of the spinal cord and merge with lower spinal portion fibers to innervate the major muscles of the neck, such as the sternocleidomastoid and the trapezius muscles. Lesions to cranial XI can cause obvious problems of resonance and in the contribution of neck accessory muscles to respiration.

Cranial Nerve XII, Hypoglossal. The hypoglossal nerve is a motor nerve innervating (as the name suggests) the extrinsic and intrinsic muscles of the tongue as well as some of the neck strap muscles. The nerve originates in its own nucleus, the hypoglossus nucleus, in the lower medulla, exiting laterally and entering the hypoglossal canal in the occipital bone, descending and then moving laterally into its many innervation sites. The muscles it innervates are the omohyoid, sternothyroid, styloglossus, hyoglossus, genioglossus, geniohyoid, sternohyoid, and all of the in-

trinsic muscles of the tongue. Cranial nerve XII has much to do with positioning of the larynx, that is, depression or elevation of the total laryngeal body, and is essential for all intrinsic movements of the tongue. Its primary impact on voice is on resonance and quality.

Superior and Recurrent Laryngeal Nerves. As the vagus nerve leaves the nucleus ambiguus and exits laterally from the superior medulla and descends down the neck, it soon begins a series of branches. The first and most superior nerve branch off the vagus is the pharyngeal branch that contains both sensory and motor branches that supply the mucous membrane and selected muscles of the pharynx and soft palate. The next branch off the vagus bilaterally is the superior laryngeal nerve.

The superior laryngeal nerve branches off the vagus at about the level of the carotid sinuses in the neck (above which begins the carotid artery bifurcation) and angles medially toward the superior larynx. The superior laryngeal nerve divides into two branches (internal and external). The internal branch provides sensory innervation to the mucous membrane at the base of the tongue and to the mucous membrane of the supraglottal larynx. The external branch provides motor innervation to part of the lower pharyngeal constrictor and to the cricothyroid muscles. Although presented briefly in Chapter 2, let us consider the function (and symptoms of disorder) of the cricothyroid muscle:

Cricothyroid (CT). Like all intrinsic laryngeal muscles (except the transverse arytenoids), the cricothyroid muscles are paired (L and R). The muscle is divided into two parts, the recta and the obliqua. Contraction of the cricothyroid muscles increases the distance between the cricoid and thyroid cartilages, increasing the length of the vocal folds, which decreases their cross-sectional mass. This action results in an increase of vibratory frequency and is heard as a rise in pitch. This stretching action also contributes to an adducting action of the vocal folds. Lesions to the CT are relatively rare and are seldom due to trauma but more often are related to some form of viral neuropathy (Tucker and Lavertu, 1992). Inability to elevate vocal pitch is the primary symptom of CT disease or trauma; in the case of unilateral CT paralysis, there may also be extreme hoarseness and occasional diplophonia (because of the disparate tension between the two vocal folds).

The next nerve branching off the descending vagus nerve is the recurrent laryngeal nerve (RLN). The RLN branches off the vagus considerably below the level of the larynx, almost at the level of the middle of the trachea. The right RLN loops “behind the right common carotid and subclavian arteries at their junction and courses vertically to the larynx” (Zemlin, 1998, p. 375). The left RLN leaves the vagus at a lower level than the right RLN, looping under and behind the aortic arch before making its vertical ascent to the larynx. Of some relevance to its frequent accidental cutting during surgery is its precarious location in the neck, ascending to the larynx in a groove between the trachea and the esophagus; the RLN then divides into three branches that enter the larynx through the cricothyroid membrane. The

RLN is vital to the abductory–adductory function of the larynx as it innervates the five intrinsic muscles of the larynx, as first introduced to the reader of this text in Chapter 2. At this point, however, we will reintroduce the five intrinsic muscles of the larynx that are innervated by the RLN, with a brief description of their function and voice symptoms if they do not receive their innervations:

Thyroarytenoid (TA). The thyroarytenoid muscle is the main mass of the vocal fold. The muscle originates on the posterior side of the thyroid cartilage, which is known as the *anterior commissure*. The medial portion of the muscle is often described as the *vocalis muscle*, inserting posteriorly in the vocal process of the arytenoid. The larger muscle portion of the TA, known as the *thyromuscularis*, leaves the inner thyroid cartilage wall and extends posteriorly to the anterior surface of the arytenoid muscular process. The TA muscle mass, its ligament, and its cover (known collectively as the *vocal fold*) when adducted serves as the primary protective valve of the airway. Airway protection appears to be the primary role of the larynx and the TA is certainly a primary valve in this protection. Second, the vibrating mass of the vocal fold produces phonation. Changes in pitch are related to changes in tension of the thyroarytenoid muscle, either from its internal muscle contraction or stretching from external causes. TA contraction also contributes to medial vocal fold adduction. Flaccid paralysis of this muscle resulting from cutting or trauma to the RLN will in time lead to vocal fold atrophy resulting in weakness in vocal fold approximation, mid-fold bowing, and dysphonia. Subtle changes of pitch variation required in normal talking and singing will also be compromised with lack of TA innervation.

Posterior Cricoarytenoid (PCA). The paired PCA is the lone abductor muscle of the vocal folds. Originating on the posterior surface of the cricoid cartilage, the muscle rises laterally and obliquely to the posterior muscular process of the arytenoid. When the muscle contracts, it rocks and slides the arytenoid on its cricoid mount, parting the arytenoids and abducting the vocal folds. The primary symptom of PCA paralysis is the inability to open the glottis on the involved side, creating a unilateral abductor paralysis.

Lateral Cricoarytenoid (LCA). The paired LCA is the primary adductor muscle of the vocal folds. The LCA originates on the superior, lateral surface of the cricoid arch and rises to the lateral muscular process of the arytenoids. When the LCA contracts, it slides the arytenoids together, which adducts the vocal folds. The LCA is an antagonist to the PCA: LCA relaxation facilitates PCA action and, conversely, relaxation of the PCA makes LCA adductory action easier. The primary symptom of LCA paralysis is vocal fold paralysis in the fixed, abducted, paramedian position.

Transverse Arytenoids. The transverse arytenoid muscles are the only unpaired muscles among the laryngeal intrinsic muscles. They are bilaterally innervated, crossing over the surfaces and space between the two arytenoid cartilages. When they contract, they have the effect of sliding the arytenoid cartilages together, contributing to vocal fold adduction. An RLN lesion may pro-

duce weakness or paralysis not only in transverse arytenoid function but in other adductory muscles as well.

Oblique Arytenoids. The oblique arytenoids are paired muscles, originating from the base of one arytenoid cartilage and rising obliquely to the apex of the opposite arytenoid. When they contract, they assist in bringing the arytenoids together, contributing to vocal fold adduction. It appears that, among those few people who can, with intent, produce ventricular phonation, differential contraction of the oblique arytenoids enables the more superior-lateral surfaces (the insertion points of the false folds) to approximate, resulting in ventricular voice. If a unilateral oblique arytenoid is paralyzed from lack of RLN innervation, this further contributes to unilateral adductor paralysis.

Having had a quick but intense look at the central and peripheral nervous systems and the innervation of muscles responsible for voice, let us now consider various voice problems of a neurogenic origin using a dysarthria classification framework introduced by Darley, Aronson, and Brown (1975) and subsequently revised by others (Duffy, 1995; Dworkin and Culatta, 1996; Yorkston et al., 1999). This revised classification system includes seven dysarthria subtypes, the categories of which are primarily based on neuromuscular abnormalities unique to each form: flaccid, unilateral upper motor neuron, spastic, ataxic, hypokinetic, hyperkinetic, and mixed.

Dysarthria is a disturbance of muscular control over the speech mechanism due to damage to the central or peripheral nervous system. Therefore, it is reasonable to expect that irregularities in speed, strength, timing, and accuracy may be observed in the systems of respiration, voicing, resonance, articulation, and prosody, either singly or in combination. Because voice is so intimately associated with respiration, resonance, articulation, and prosody in all seven dysarthria types, each subtype will be addressed.

Flaccid Dysarthria

Flaccid dysarthria is usually caused by unilateral or bilateral damage to specific cranial nerves, irrespective of whether the disturbances occur at their nuclei in the brainstem or somewhere along their extracranial route to the speech subsystem muscles that they innervate. Damage to the peripheral nervous system causes a flaccid paralysis with underlying involvement of weakness, reduced force or muscle contraction, and reduced range of motion.

Vocal Fold Paralysis

Flaccid dysarthric patients who suffer damage of the cranial nerve X anywhere along its path from the medulla to the larynx have voice difficulties as a result of vocal fold paralysis. The type and extent of dysphonia largely depends on the lesion site and whether the damage is unilateral, bilateral, partial, or complete.

Cricothyroid Muscle Paralysis. The SLN branches first or higher than the RLN from the descending vagus nerve and soon branches again into its internal and external branches that insert directly into the larynx. Because of its relatively direct course after it has branched out of the vagus down to the larynx, the SLN is rarely injured by trauma. Although other etiologies may be considered, viral infection appears to be the most common cause of SLN involvement, unilateral or bilateral, causing paralysis of left or right (or both) cricothyroid muscles (Dursun et al., 1996). Cricothyroid function is primarily in the tensing of the vocal fold, essential for elevation of pitch as well as contributing to vocal fold adduction. On examination, the patient displays a slight rotation of the involved vocal fold to the normal side as well as a slight bowing of the vocal fold on the involved side (Tanaka, Hirano, and Umeno, 1994). The patient's primary voice symptoms are an inability to elevate or lower pitch and some breathiness (due to the bowing). The virally caused cricothyroid paralysis is usually temporary, with the patient responding well to corticosteroids and antiviral agents; voice therapy has been found helpful in correcting the effects of the anterior glottal rotation (Dursun et al., 1996).

Bilateral Vocal Fold Paralysis. Bilateral paralysis of the vocal folds is usually the result of lesions high in the trunk of the vagus nerve or at the nuclei of origin in the medulla. If the lesion is above the nodose ganglion, other muscles innervated by the vagus, as well as muscles supplied by other cranial nerves, will be affected as well. These high lesions include tumors at the base of the skull, carcinoma, or trauma. In the case of children, bilateral vocal fold paralysis is a common cause of neonatal stridor (Baker, Sapienza, and Collins, 2003). Most cases are associated with intracranial pathology such as meningomyelocoele, hydrocephalus, or Arnold–Chiari malformation. Other reports of rare etiologies are motor axonal neuropathy (Marchant et al., 2003) and familial clustering with autosomal recessive mode of inheritance (Raza et al., 2002).

Bilateral vocal fold paralysis may be of the abductory or adductory type; both are life threatening. Voice per se is of secondary concern to respiratory survival and feeding. In bilateral adductor paralysis, neither vocal fold is capable of moving to the midline, thus making phonation impossible and placing the individual at risk for aspiration. In abductor paralysis, the vocal folds remain at the midline, causing serious respiratory problems for which most patients will need a tracheostomy. Andrews (2002) offers specific procedures for the SLP to use in working with young children with bilateral vocal fold paralysis, that is, how to manage the tracheostomy, the use of tracheal valves, and the need for minimizing the negative effects of the vocal fold dysfunction on the child's expressive language and speech development.

Continued bilateral vocal fold paralysees may require surgery to improve greater airway competence in both children and adults. Surgical reinnervation of the muscles of the vocal folds has been successfully reported by Crumley and Izdebski (1986). Perhaps used more often is the unilateral removal of one arytenoid with cauterization of muscular attachments to stimulate eventual contracture, resulting in more anterior glottal closure with posterior airway dilation (Tucker and Lavertu, 1992).

Zealer et al. (2003) recently reported in a pilot study that electrical stimulation to the posterior cricoarytenoid muscle via an implant resulted in improved vocal fold movement for three patients. Laser surgery has been successful in decreasing open glottal space for bilateral adductor fold paralysis (Prasad, 1985), and laser arytenoidectomy for bilateral abductor paralysis has successfully opened the glottis (Lim, 1985). An alternative to surgery for some patients with abductor vocal fold paralysis may be inspiratory pressure threshold training. Baker, Sapienza, and Collins (2003) reported reductions in dyspnea during speech and exercise for a six-year-old child with congenital bilateral abductor paralysis after eight months of respiratory muscle strength training.

Unilateral Vocal Fold Paralysis (UVFP). Disease or trauma to the recurrent laryngeal nerve (RLN) on one side is the most common form of laryngeal paralysis (Case, 2002; Hirano and Bless, 1993; McFarlane, Watterson, and Von Berg, 1999). Because of the extended course of the left RLN, traveling down the neck and looping around the aortic arch in the chest and then traveling up again to the larynx, the left RLN appears to be more prone to traumatic or surgical injury than the right RLN. Bhattacharyya, Kotz, and Shapiro (2002) reported that of 64 patients presenting with UVFP 53 cases were left-sided. In a retrospective review of patient cases, researchers at Georgetown University reported that isolated right VFP comprised only 3.1% of 778 laryngeal evaluation cases (Hughes et al., 2000). Surgical trauma predominated as an etiology, followed by viral and idiopathic causes. The authors commented that an unexpectedly high number of cases of VFP were caused by anterior laminectomy, accounting for 36% cases. Etiologies of UVFP, of course, may also be location specific. Researchers in Scotland found a high rate of vocal fold palsy secondary to bronchogenic carcinoma, likely, the authors speculate, associated with the high levels of smoking in Scotland (Loughran, Alves, and MacGregor, 2002).

When the RLN is compromised on one side, the laryngeal adductor muscles (particularly the lateral cricoarytenoid) are not able to perform their adductory role. This keeps the paralyzed fold fixed in the paramedian position, that is, neither fully abducted nor adducted. The vocal fold remains at the paramedian position for both inspiration and expiration (including attempts at phonation).

On endoscopy, we see the paralyzed fold remaining abducted as the normal vocal fold moves to midline. Because of the proximity of the folds at the anterior commissure, there is usually some anterior approximation, which helps to set the two folds into vibration during phonation attempts. Colton and Casper (1996) discount that there is any slight crossover of the normal fold to meet the paralyzed fold, and, generally, what we observe on phonation attempts is the vibration of the paralyzed fold set in motion by the outgoing airflow passing between the two folds, particularly at the anterior one-third. Also, the Bernoulli effect, described in Chapter 2, plays a role here in drawing the two folds together.

The voice in UVFP is markedly dysphonic or aphonic. Perceptual characteristics include breathy, hoarse vocal quality, reduced phonation time, decreased

loudness, monoloudness, diplophonia, and pitch breaks. The breathy vocal quality, reduced loudness and short phonation times can be attributed to air escape through an open glottis during phonation. Hoarseness, pitch breaks, and diplophonia can be associated with reduced ability to adjust the internal tension of the paralyzed vocal fold. Excessive supraglottal constriction (hyperfunction) may contribute to the perception of hoarseness.

Because many traumatic vocal fold paralyses have spontaneous recovery within the first 9 to 12 months post onset, permanent corrective procedures should be delayed until voice intervention has been tried. In many cases, strengthening the vocal muscles and improving speaking technique result in very good voice quality and surgery is unnecessary (Sataloff, 1997b). Behavioral voice therapy may be the only treatment required or it may suffice as a temporary measure until medical intervention is feasible. We have found behavioral voice therapy to be superior in voice quality to Teflon injection and was judged by speech pathologists, otolaryngologists, and lay listeners to be comparable with muscle nerve reinnervation surgery (McFarlane et al., 1991). Another study found that voice therapy reduced mean airflow rate in 16 patients with UVFP by nearly 50% (McFarlane et al., 1998). The techniques we normally introduce in clinic are half-swallow boom, head positioning, tuck chin, digital manipulation, focus, tongue protrusion /i/, yawn-sigh, pitch shift up, and inhalation phonation (McFarlane, Watterson, and Von Berg, 1999). Each technique affords an anatomical and physiological rationale for improving voice in individuals with UVFP, as demonstrated in the following case study.

Mr. S. was a Korean-born gentleman who underwent surgery and subsequent radiation therapy for thyroid cancer. Post surgery and radiation, Mr. S. reported a deterioration of voice, and his surgeons reported that he had sustained a right vocal fold paralysis. He reported that, in addition to speaking difficulties, on occasion he experienced dysphagia, especially on thin liquids. Swallow dysfunction secondary to UVFP is not unusual; indeed, researchers at Harvard Medical School reported that, for 64 patients presenting with UVFP, radiographically significant penetration or aspiration occurred in approximately one-third (Bhattacharyya, Kotz, & Shapiro, 2002).

Perceptually, phonation was breathy and diplophonic at times. Phrases were brief and tended to decline in volume toward the ends. Head turn right with right digital manipulation of the thyroid cartilage increased vocal loudness while maintaining vocal quality. Mr. S. extended the chin and tensed the neck strap muscles, notably when beginning a phrase. New techniques of chin down with focus eliminated these maladaptive behaviors.

Acoustic measures using a sustained /a/ revealed an F_0 of 104 Hz, with a RAP of 2.98% and shimmer of 12.2% (see Chapter 5 for acoustic assessment of the voice). F_0 was within normal limits; however, RAP and shimmer were above normal limits. Head turn right and right digital pressure revealed reduced RAP of 1.7% at 121 Hz, which is not within normal limits, but improved from baseline.

Shimmer was reduced to 4.37% employing these techniques. Transglottal airflow was reduced to 266 mL/s from 468 mL/s with head turn right and right digital manipulation.

Rigid videoendoscopy with stroboscopy revealed a right vocal fold at the paramedian position. Phonation revealed an immobile right vocal fold and longitudinal glottal gap, even though the left vocal fold adducted to the midline. Medial compression was observed, as was seen by the bulging of the left ventricular fold.

Mr. S. received six hours of intensive voice therapy focused on eliminating maladaptive behaviors and improving vocal quality and intensity. Yawn–sigh followed by vowel initial productions were successful for increasing amplitude and vocal quality. This was followed by focus with nasal glide stimuli. We were sensitive to the multicultural nature of this case, and the phonemes that comprise the Japanese and Korean languages were explored. Mr. S. was able to identify voiced and voiceless sounds and to make phonation and resonance adjustments for smooth voiced to voiceless transitions. The primary clinician was assisted by an undergraduate clinician who spoke Japanese.

When Mr. S. felt comfortable with the techniques, he was introduced to new speaking situations within the clinic. He lectured for 15 minutes before an undergraduate class. Students reported full intelligibility. Mr. S. observed that he is asked to speak in large rooms, often without a microphone. For these occasions we recommended that he use a personal voice amplifier. Personal voice amplifiers vary widely in features and corresponding costs. In a recent study involving voice amplification as a control condition, Roy et al. (2002) used the ChatterVox portable amplifier; however, many others are available over the Internet and at local electronics stores. Finally, Mr. S was provided with a video and written home program to help to maintain the gains made in the clinic.

Nonbehavioral Approaches to UVFP. Since Arnold (1962) introduced the injection of Teflon as a surgical approach for promoting better medialization, there are numerous reports (Dedo and Carlsöö, 1982; Lewy, 1983) in the literature citing advantages, problems, and precautions. In general, Teflon injection no longer appears to be the procedure of choice for unilateral vocal fold paralysis (Sataloff, 1997b). In greater use today is collagen, reported to be successfully used in 119 patients with glottic insufficiency (Ford, Bless, and Loftus, 1992) who were injected with soluble bovine collagen. A distinct advantage of the collagen injection is that it can be custom contoured or recontoured to fit the glottal deficit without appreciable damage to surrounding tissues. Gelfoam (another often-used injection compound), autologous fat, Teflon, and collagen have been compared with thyroplasty in the treatment of unilateral paralysis (D'Antonio, Wigley, and Zimmerman, 1995; Lacourrege et al., 2003); one disadvantage of injection approaches is that the vocal cover is often violated, resulting in increased stiffness.

Thyroplasty I is a surgical approach to medialization of the paralyzed fold, using a free-moving wedge to move the paralyzed fold to midline (Blaugrund,

Isshiki, and Taira 1992). The surgeon cuts a rectangular window (4 by 12 mm) out of the thyroid cartilage on the side of the paralyzed vocal fold. The patient is conscious during the procedure and produces voice when the surgeon places the wedge at various sites against the paralyzed fold. When it is confirmed that a certain site produces the best phonation, the wedge is fixed surgically at that point. Thyroplasty in the hands of a competent surgeon produces excellent results (Lu et al., 1996), and patients should expect “voice improvement as early as 1 month postoperatively and should remain stable with slight fluctuations for at least 6 months” (p. 576).

Dean et al. (2001) recently introduced a modification of the thyroplasty technique by introducing a titanium implant with a micrometric screw that allows for secondary adjustment of medialization, if necessary. Titanium is MRI safe.

Although there are few reports following thyroplasty patients and their voices over many years, it would appear anecdotally by these text authors (DB, SMF) that the vocal gains after thyroplasty appear to last for several years. Some patients, after injection or surgery, continue to display the hyperfunctional vocal behaviors they were using before treatment. Direct symptom modification can usually reduce such problems as squeezing the words out, using pushing behaviors, and using excessive glottal attack. Following injection or surgical forms of medialization, the SLP may help the patient reestablish a normal voice, giving some attention to adequate breath support, phonation free of effort, with some attention given to voice focus and adequate loudness.

Another procedure for unilateral paralysis, reported by Crumley and Izdebski (1986), involves reinnervating the paralyzed muscles by nerve grafts from the phrenic nerve, or by grafting a section of the superior laryngeal nerve with a portion of the hypoglossus nerve into the vocal fold adductor muscles.

Myasthenia Gravis

Although vocal paralysis is the most common voice problem associated with flaccid dysarthria, myasthenia gravis is not an uncommon dysarthria, with an incidence of 1 in 10,000. Some patients with myasthenia gravis experience problems of severe voice fatigue with associated problems in adequate breath support. MG is an autoimmune disease in which the neuromuscular junction becomes impaired as the patient uses that particular muscle or muscle group, resulting in extreme muscle fatigue. Muscles innervated by the cranial nerves in the head and neck are particularly vulnerable to the disease. In MG, the immune system (for reasons unknown) produces antibodies that attack the receptors that lie on the muscle side of the neuromuscular junction (Berkow, Beers, and Fletcher, 1997). Symptoms occur because there is damage to the receptors at the neuromuscular junction, preventing the normal transfer of impulse from the nerve into the particular muscle. The disease occurs twice as often in women over men with females reporting the onset in their thirties and men reporting onsets in their sixties.

Typically, sustained repetitive performance of a particular muscle group will lead to a complete performance fatigue: Tapping two alternate notes repeatedly on a piano will result in a progressively slower tapping rate with eventually an in-

ability to continue the task. For the myasthenia gravis patient with voice problems, the patient experiences a vocal change with voice usage from a normal voice to a breathy, weak, barely audible voice. With a few minutes of complete voice rest, the voice will be restored, but after a few minutes of usage, the weak voice will return. In severe cases, the patient will report difficulty swallowing, with occasional nasal regurgitation (Hopkins, 1994).

The diagnosis of myasthenia gravis should always be suspected in patients who experience weakness after usage of the muscles of the eye, face, and throat, but with some recovery after rest. Because acetylcholine receptors are blocked, drugs that increase the presence of acetylcholine in the neuromuscular junction are useful in helping to confirm the diagnosis. “Edrophonium (Tensilon™) is most commonly used as the test drug; when injected intravenously, it temporarily improves muscle strength in people with myasthenia gravis” (Berkow et al., 1997, p. 333).

Accordingly, the treatment of MG is primarily medical, giving anticholinesterase medications, immunosuppressants, antimetabolite agents, and corticosteroids. Other options available to the patient are plasmapheresis, which removes AchR antibodies from the circulating plasma of patients with MG, intravenous immunoglobulin, and thymectomy (Armstrong and Schumann, 2003).

The speech–language pathologist often plays the primary role in the discovery of the disease. Patient complaints of deteriorating voice after usage, particularly when coupled with other visual symptoms, such as a drooping eyelid (ptosis), or new problems in swallowing, should be referred through the patient’s primary care physician to a neurologist. At the time of the evaluation, the SLP should give the patient sustained oral reading tasks; a determination should be made of how long oral reading must continue before vocal–speech deterioration is heard. From the onset of voice change, time measurements should be made of continued oral reading until further voicing is almost impossible. Then determine the amount of time required before there is some restoration of vocal strength. Beyond oral reading, the SLP might take measures of airflow and pressure, spirometric determination of air volumes, test diadochokinetic rates for various oral tasks, make glottographic determinations of vocal fold approximation, and administer articulation tests. Once the patient is receiving treatment with some form of drug regimen, the SLP should select any of these measures for comparison over time, giving objective evidence of medication effectiveness. The SLP’s role is one of discovery and comparison of motor response data over time, and not one of providing voice therapy. With appropriate acetylcholine levels achieved through medication, MG patients in early stages of the disease process will usually experience the levels of speech and voice competence they had before the onset of symptoms.

Guillain–Barré

A brief mention of Guillain–Barré is warranted at this point, because the onset of the disease is often expressed in dysphonia and dysphagia. GB is a disorder of unknown cause, but is frequently preceded by viral infection. It involves the focal demyelination of spinal and cranial nerves. The disease process usually begins

symmetrically in the lower extremities and advances superiorly, but other researchers have suggested that facial, oral–pharyngeal, and ocular muscles occasionally are affected first. The patient often requires a tracheostomy and ventilary support. Often the patient receives plasmapheresis as part of medical intervention. Approximately 65% of individuals recover from GB while the remainder are left with residual dysarthria and altered psychosocial situations (Bernsen et al., 2002).

Unilateral Upper Motor Neuron Dysarthria

UUMN dysarthria is caused by a unilateral lesion to the CNS, involving both the pyramidal and extrapyramidal tracts. It is often observed in patients who have experienced a cerebrovascular accident (CVA), but it could be caused by other etiologies, such as tumor or trauma. A CVA, known more commonly as a *stroke*, is a temporary impairment of blood flow to the brain. There are generally recognized three types of CVA: thrombosis (the most common obstruction, a clot forms within an artery obstructing the flow of blood); embolus (a traveling blood clot that lodges within an artery preventing the flow of blood); or hemorrhage (blood flows out of a break in an arterial wall). Because the distribution of blood for most of the higher areas of the brain (cortex through thalamus–basal ganglia) is distributed superior to the circle of Willis, the blood supply to each of the two cerebral hemispheres is unilateral. That is, each hemisphere has its own blood supply. This is why most strokes appear to involve motor and sensory function on one side of the body. A CVA in the left hemisphere will produce a right-sided weakness or paralysis; a right hemisphere stroke will involve the left side of the body.

Voice was once thought to be seldom affected by a single, unilateral cerebral lesion, because of the bilateral nature of corticobulbar innervation of the vagus nerve. Rather, imprecise articulation due to unilateral facial and lingual weakness is a primary deviant characteristic of UUMND. Nevertheless, Duffy and Folger (1986) reported a high percentage of dysphonia occurring in 56 cases of individuals with UUMND. Thirty-nine percent of patients had a moderate dysphonia, which was described as harsh or strained-harshness and 9% had reduced loudness. Duffy suggests that the dysphonia could reflect the effect of subtle vocal cord weakness, mild spasticity from the lesion, or possible spasticity from an *undetected* lesion in the contralateral hemisphere. UUMND is also associated with dysphagia, due to lip, buccal, and lingual involvement. These sensory–motor disturbances are normally mild and transient (Darley, Aronson, and Brown, 1975; Logemann, 1998; Metter, 1985).

Spastic Dysarthria

Two or more strokes that result in bilateral cerebral lesions may produce severe voice symptoms due to lesions to the pyramidal and extrapyramidal tracts bilat-

erally. Common neuromuscular symptoms include hypertonicity, exaggerated reflexes, paresis, and bilateral weakness of various speech and voice muscle groups. The voice symptoms are characteristic of a spastic dysarthria, also known as pseudobulbar palsy. Voice may be strained and strangled, brief in phonation time, low in pitch, and monopitch with variable loudness. Hypernasality may be present in some patients (Murdoch and Chenery, 1997) due to the slow and weak range of movement of the velum. Symptomatic of patients with bilateral pyramidal and extrapyramidal tract damage is emotional lability, which may severely influence voice quality and resonance. The patient will laugh or cry easily, inappropriately to the intensity of the stimulus. For example, we remember a 55-year-old patient with pseudobulbar palsy who, when meeting an old friend, would appear to be crying on inhalation and laughing on exhalation. His lability-influenced voice would have severe posterior focus with extreme hypernasality. He had so much massive escape of airflow through his nasal passages that his oral articulation was severely compromised. When he cried or laughed, his speech was unintelligible. Management of his voice was best helped by attempting to reduce his lability.

Voice therapy for patients with spastic dysarthria is highly individualized, depending on the speech subsystems that are compromised. Dworkin and Culatta (1996), Duffy (1995), and Yorkston et al. (1999) have discussed hierarchical approaches to voice and speech therapy, based on the comprised subsystem(s).

Hypokinetic Dysarthria

Hypokinetic dysarthria is associated with a depletion of or functional reduction in the effect of the neurotransmitter dopamine on the activities of the basal ganglia. As described earlier, the basal ganglia are associated with providing proper background and tone for quick, discrete movements. The clinical features underlying basal ganglia pathology are rigidity, slow movement (bradykinesia), limited range of motion, and a resting tremor that is normally ameliorated through intentional movement. Although a number of etiologies may cause hypokinetic dysarthria, idiopathic Parkinson's disease (PD) is known as the prototypical hypokinetic dysarthria, as 98% of hypokinetic dysarthrias are of the Parkinson's type (Berry, 1983).

Parkinson's Disease

The Parkinson patient exhibits a hypokinetic dysarthria characterized by reduced loudness, breathy voice, monotony of pitch, intermittent rapid rushes of speech, and soft production of consonants. Some investigators of PD have found diminished function in one or more components of speech-voice; for example, Solomon and Hixon (1993) found significant respiratory difficulties as possibly contributing to the PD patient's voice symptoms; Ramig and others (1994) found that 35 of 40 PD subjects had bowed vocal folds. Duffy (1995) writes that many of these

abnormalities can be related to the underlying neuromuscular deficits of rigidity, reduced range of movement, and slowness of movement in the laryngeal muscles.

It would appear that isolation of any one speech component for study in the PD patient will find a deficit in function. Fortunately, the most effective voice therapy approach is a holistic one, finding that to exaggerate one component helps improve function in all other components.

When patients attempt to speak in a quick conversational pattern, speech is often unintelligible due to the rapid and accelerated movement of the articulators. When they speak with intent, however, their speech can be slower, louder, have better voice quality, and better articulation. Following the model of intention used in physical therapy for gait training (thinking where you are going to place each foot as you walk makes walking easier), the same model of intent works to improve speech. Using intention with these patients, the writers have asked PD patients to speak with an accent, or use a different pitch, or speak slower, or speak louder (Boone and Plante, 1993). Taking the automatic motor-set out of speaking by speaking intentionally different seems to help the patient's speech in all parameters: loudness, voice quality, appropriate pitch, and rate. More recent in-clinic trials have found that instructing patients to deliberately pronounce the final sound of each word has yielded increases in vocal loudness and intelligibility.

Ramig (1994) and others (Sapir et al., 2002; Liotti, et al., 2003) have studied the model of intention in a formal voice and speech improvement program that is driven by a number of perceptual features of phonation in Parkinson's disease. The main goal of the Lee Silverman Voice Treatment (LSVT) program is to increase vocal fold adduction and respiratory effort ("think loud, think shout"), which, in turn, is intended to increase loudness, vocal quality, and, subsequently, intelligibility.

One study of LSVT effectiveness had 40 PD patients receive one hour of voice therapy four times a week for one month, receiving 13 to 16 hours of individual voice therapy. There were three general therapy tasks: increasing vocal fold adduction, increasing respiratory support, and increasing maximum fundamental frequency range. Of all the therapy tasks, speaking "louder shout" seemed to be the most unique and beneficial part of the Silverman therapy approach. There was significant improvement in all variables studied between pre- and posttherapy treatment.

Sapir and others (2002) investigated whether increased loudness is maintained over several months after conclusion of the LSVT program. Judges listened to reading samples produced by two groups: one that had undergone LSVT and one that had undergone a high-effort respiratory treatment program. The speech samples in the LSVT group, but not the high-effort respiratory group, were significantly more likely to be perceived louder and of better quality at follow-up.

Researchers in the Netherlands have suggested that increased respiratory-phonatory effort raises vocal pitch and laryngeal muscle tension (de Swart et al., 2003). These researchers generated an intervention program called Pitch Limiting Voice Treatment (PLVT), which instructs patients to increase respiratory support, but to phonate at a low pitch. A study comparing LSVT and PLVT revealed the

same increases in loudness for both groups, but the authors suggested that PLVT limited increases in vocal pitch, thus preventing strained and pressed voicing.

For those patients who are initially stimulable for behavioral voice programs, but who experience difficulty generalizing the gains beyond the clinic, we have offered delayed auditory feedback (DAF), with mixed results. DAF is an instrumental procedure that feeds an individual's speech trace back to the individual's auditory system via earphones at a delayed rate. The effect of the delay is to slow speech rate, increase vocal loudness, and increase articulatory accuracy. Several case studies at this clinic and reports in the literature have suggested improved speech using DAF for individuals presenting with hypokinetic dysarthria (Downie, Low, and Lindsay, 1981; Hanson and Metter, 1983; Yorkston et al., 1999). These authors suggest that the benefits included marked reduction in speech rate, increased loudness, reduced phonetic errors, and increased acoustic distinctiveness.

DAF intervention produced remarkable results in the vocal intensity, rate, and intelligibility of an individual seen at our clinic with PD. This patient, a former physician, had received a thalamic (deep brain) stimulator four years earlier to reduce tremors and was taking Sinemet (combined levodopa and carbidopa). Nevertheless, speech was rapid and blurred and the voice was hyphonic. Various clinic probes of speaking to a metronome, pacing, respiratory–phonatory training, and hyperarticulation were not effective. He was fitted with the Kay Facilitator (Kay Elemetrics, 1998) set at the 170 ms feedback mode and the effect was dramatic. He increased vocal intensity, extended the vowels, and increased articulatory contacts, thus increasing intelligibility. He subsequently purchased a pocket-sized DAF unit that he wore at all times. He said the system was so innocuous that people thought he was listening to the ball game. Even though he did not return to his former practice, he did begin to deliver lectures at the medical school and spoke at Parkinson's disease support group meetings.

The diagnosis of PD and its medical management belongs to the neurologist, although we and many other voice clinicians have been instrumental in alerting health professionals to patients who present to our clinics with hypokinetic features that may have previously gone undetected. We have found that an interdisciplinary health care team, consisting of the SLP, physician, nurse, nutritionist, and various rehabilitation experts, comprises the best medical care for patients with this complex disorder. With respect to conducting voice therapy, speech therapy, or dysphagia intervention, the timing of dopaminergic medication should be monitored to ensure that the maximum effect of the medications can be used to facilitate motor performance. Schulz (2002) reported that when a person is optimally medicated, speech therapy has proved to be the most efficacious therapeutic treatment for improving voice and speech function for people with Parkinson's disease. Over time, the period of relief from continued dopaminergic administration becomes shorter, requiring new medication protocols and possible neurosurgical approaches to reduce tremor, such as anterior thalamotomies (Stacy and Jankovic, 1992), pallidotomies (Schulz, Greer, and Friedman, 2000), and stereotactic surgery deep-brain stimulation. In deep-brain stimulation, a thin stimulator is surgically placed in the

thalamus, the part of the brain that is believed to activate tremors. The stimulator is powered by a tiny generator implanted in the patient's chest. Different researchers have identified different areas of the thalamus that are most receptive to stimulation, resulting in reduced tremors. Hamel and colleagues (2003) suggest that stimulation of the subthalamic nucleus results in marked improvement in levodopa-sensitive Parkinsonian symptoms and levodopa-induced dyskinesias.

Hyperkinetic Dysarthria

Hyperkinetic dysarthria is difficult to define because of its many different clinical presentations, but it is generally associated with damage to the basal ganglia or an imbalance of neurotransmitters therein, specifically acetylcholine and dopamine. Hyperkinetic means involuntary and uncontrolled movements, and it may be manifest in any or all of the subsystems of speech. Unlike most CNS-based dysarthrias, hyperkinetic dysarthria can manifest itself at only one level of speech production, sometimes only a few muscles at that level (Duffy, 1995).

Spasmodic Dysphonia

Spasmodic dysphonia is a relatively rare voice disorder that can be classified as a focal dystonia (Case, 2002), which falls under the cluster of hyperkinetic dysarthria. The patient exhibits a strangled harsh voice with observable effort in pushing the air out during most voicing attempts. Patients' voices sound strained, choked off with the attempts to voice, as if they are trying to push the outgoing airstream through a tightly adducted laryngeal opening. Endoscopic examination (Davis et al., 1988) shows that the tight voice is indeed produced by hyperadduction (severe approximation) of the true folds, often accompanied by tight closure of the false vocal folds (ventricular folds) with supraglottal constriction of the aryepiglottic folds and contraction of the lower pharyngeal constrictors. The total laryngeal and lower pharyngeal airway appears to close down (McFarlane, 1988; McFarlane and Lavorato, 1984). No wonder we hear a strained, strangled voice in such patients. Nevertheless, Hirano and Bless (1993) caution that the voice clinician should not anticipate seeing one particular laryngeal pattern. They suggest that spasmodic dysphonia can be very heterogeneous, with presentation ranging from spasmodic hyperfunction to hypofunction to irregular twitching of the vocal folds.

In addition to the problem of voicing, patients with spasmodic dysphonia complain about the difficulties they experience trying to force expiratory air out whenever they desire to phonate. Aronson (1990) comments that the tight voice during adductor spasmodic dysphonia "occurs only during voluntary phonation for communication purposes and not during singing, vowel prolongation, laughing, or crying" (p. 161). However, in patients who have carried the diagnosis of spasmodic dysphonia for some period of time and who are more severe, we see symptoms of

this disorder in prolonged vowels as well. The patients soon learn to expect phonation difficulties whenever they attempt to speak. In this sense, spasmodic dysphonia resembles stuttering. In European writings, in fact, the condition is sometimes called the *laryngeal stutter*. McFarlane and Shipley (1979) make a case for spasmodic dysphonia *not* being considered as laryngeal stuttering based on there being a greater number of important dissimilarities than important similarities between the two disorders. Most patients with spasmodic dysphonia experience some normal voice in certain situations. Case histories of these patients reveal that such situations as “talking to my cat” or “speaking to others in a pool while I tread water” are times when patients have experienced normal voice.

The most common type of spasmodic dysphonia appears to be related to tight laryngeal adduction (known as adductor spasmodic dysphonia ADSD). Aronson (1990), however, also describes a second form of the disorder, known as abductor spastic dysphonia (ABSD). Patients with this disorder exhibit normal or dysphonic voices that are suddenly interrupted by temporary abduction of the vocal folds, resulting in fleeting aphonia. After such momentary aphonia, the patients’ voice patterns are restored again (until the next aphonic break). Endoscopy shows that the vocal folds of such patients abduct suddenly, “exposing an extremely wide glottic chink” (Aronson, 1990, p. 185). More often than not, the abductor spasms appear to be triggered by unvoiced consonant sounds. The abductor-type disorder is a much rarer form of spasmodic dysphonia. For example, Davis and others (1988) reported that of 25 successive cases of spasmodic dysphonia observed in a Sydney, Australia, hospital, 24 were adductor type and one was an abductor type. The abductor spasm can often be treated successfully as a phonation break. Watterson and McFarlane (1992) make a strong case for considering the abductor type as a different disorder altogether and not a subtype of spasmodic dysphonia. Colton and Casper (1990) also seem to indicate that the two are different in pathophysiology and require different treatments. The symptoms and the treatment between adductor and abductor spasmodic dysphonia are so different, the authors of this text will confine our further comments about spasmodic dysphonia to the adductor type. Symptoms and treatment for the sudden abductory spasms described by Aronson (1990) may be found in Chapter 7 under abductor spasms.

Spasmodic dysphonia (SD) today is classified as a form of focal dystonia. Dystonia is a neurological dysfunction of motor movements, either more generalized to major body movements or seen in focal disorders, such as in the eyelids (blepharospasm), in the neck (torticollis), or in the larynx (spasmodic dysphonia). The site in the brain where a lesion might occur that would cause spasmodic dysphonia is still not definitively known. One of the first studies using MRI, SPECT, and BEAM for identifying possible SD lesion sites was reported by Finitzo and Freeman (1989) who concluded that “SD is a supranuclear movement disorder primarily, but not exclusively, affecting the larynx. Fully half of our subjects evidence isolated functional cortical lesions” (p. 553). While there is increasing consensus among medical and voice pathologists that SD is a neurological problem (Blitzer and Brin, 1991; Chhetri et al., 2003), the treatment of SD has not embraced symptom-modifying

medication or intracerebral neurosurgery treatments. Of all the intrinsic laryngeal muscles (except the important adductor, the lateral cricoarytenoid, which was not studied), it has been clearly demonstrated using simultaneous EMG recordings that only the thyroarytenoid (the vocal fold) has abnormal muscle activation during SD-voicing (Nash and Ludlow, 1996).

Exploring beyond the suspected site of lesion, Schweinfirth, Billante, and Courey (2002) attempted to identify risk factors and demographics in patients with adductor spasmodic dysphonia (ADSD). Results of a retrospective survey of 168 patients revealed that “There appears to be no significant environmental or hereditary patterns in the etiology of spasmodic dysphonia” (p. 220). The authors did identify some trends, however: The majority of patients were females (79%). A significantly higher incidence of childhood viral illness was found in the patients with SD (65%). Patients with SD had a significant incidence of both essential tremor (26%) and writer’s cramp (11%), but no history of major illness or other neurological disorder.

Judgment Scales for Spasmodic Dysphonia. The description and quantification of the symptoms of spasmodic dysphonia require administration of perceptual judgment scales and instrumental measures. Because SD is a rare disorder, patients with SD may not be easily identified by clinicians who do not have extensive experience with its symptomology. Barkmeier, Case, and Ludlow (2001) point out that SD can easily be misinterpreted as an essential tremor or vocal hyperfunction, behaviors whose distinguishing features and etiologies we will attempt to isolate later in this section (Table 4.1). Barkmeier et al. (2001) investigated whether voice clinicians with infrequent exposure to patients with SD could learn to identify speech symptoms of ADSD and ABSD compared with voice clinicians with extensive experience with these disorders. Results revealed that while the nonexpert judges tended toward false positive judgments for the speech symptoms of interest, the overall speech symptom profiles for each type of voice disorder appeared comparable to those obtained from the expert judges. Readers are encouraged to review this study to become familiar with the identification scale used.

Another excellent judgment scale was developed (Stewart et al., 1997) for assessing the SD patient, known as the Unified Spasmodic Dysphonia Rating Scale (USDRS). The scale offers the SLP a standardized way of asking for speech–voice responses and a 7-point rating scale for evaluating such SD-voice parameters as overall severity, aspects of voice quality, abrupt voice initiation, voice arrests, loudness variations, tremor, expiratory effort, speech rate, speech intelligibility, and related movements and grimaces (Stewart et al., 1997, p. 100). The administration of the perceptual rating scale should precede such instrumental assessments as airflow and pressure data, fundamental frequency values, perturbation measures, and intensity documentation (see Chapter 5).

What treatment options are available today for reducing the hypertonic approximation of the vocal folds during SD voicing attempts? Let us consider separately several treatment options that can be offered to the SD patient: voice therapy,

Table 4.1 Differences between Spasmodic Dysphonia, Essential Tremor, and Vocal Hyperfunction

Disorder	Age of Onset	Gender	Suspected Etiology	Presentation	Intervention
Essential tremor	Any age	Predominantly females	CNS neurogenic	Regular rhythmic vocal arrest; may involve supraglottal structures	Behavioral; Botox not recommended due to numerous supraglottal structures involved
Vocal hyperfunction	Any age	Predominantly male children and adolescent and adult females	Functional	Various: anteroposterior and medial squeezing of supraglottal structures	Behavioral
Adductor SD	Two-thirds of onset between 40 and 60	Adult females	CNS neurogenic	Irregular vocal arrests involving TA muscle	Botox injection, surgery

surgical resection of the recurrent laryngeal nerve, botulinum toxin (Botox) injection, and surgical modification of the vocal folds.

Voice Therapy for SD. For a clinical lifetime, this writer (DB) encountered each new SD patient with the optimism that the strangled-sounding, harsh voice could be modified by voice therapy, only to find repeatedly with each new patient that apparent success in producing an easy normal voice temporarily in the voice clinic seemed to have no carryover out of the clinic.

Case (2002) writes of similar poor outcomes of traditional voice therapy, noting that many patients have made slight improvements when speech is produced in small units, such as monosyllabic utterances, but rarely in contextual speech. “Historically, the poor prognosis is one of the most significant symptoms of this disorder and has been pathognomonic and diagnostic to it” (p. 189). There have been a few reported positive outcomes for SD patients who have received voice therapy, such as Cooper’s (1990) “direct voice rehabilitation” and the more conventional voice therapy reported by Shulman (1991). For the typical speech–language pathologist, our role with the SD patient is careful, meticulous assessment to permit evaluation

of treatment outcomes and to combine voice therapy efforts with pharmacological or surgical treatment, before and after intervention (Murry and Woodson, 1995).

Some trial voice therapy should follow assessment, used at least as diagnostic probes. Many SD patients experience an easier voice with less effort “pushing voice out” through working on an easy breath cycle, employing the yawn-sigh, relaxation methods, coupled with hierarchy analysis. Boone (1998) has found both real-time amplification, auditory feedback, and masking (so patients cannot hear their own voicing) are facilitative for some SD patients. Speaking on inhalation is reported as less likely to reduce the symptoms of “long-standing adductor spasmodic dysphonia” (Harrison et al., 1992, p. 000). Roy, Ford, and Bless (1996) have employed the musculoskeletal tension reduction techniques recommended by Aronson (1990) with over 150 cases of muscle tension dysphonia, described under Massage in Chapter 6 of this text. Included in the group of muscle tension dysphonia patients were some SD patients (the number was not specified in the article) who received the manual lowering of their larynx but experienced only “transient improvements in voice that could not be stabilized or generalized” (Roy et al., 1996, p. 855).

In summary, from long clinical experience and in reviewing the literature, there are scarce efficacy data to show that SD patients who struggle to get air out while producing harsh, strangled voice is resolved solely with voice therapy. Rather, those patients who do respond positively to voice therapy may likely have originally presented with a vocal hyperfunction, which often masquerades as SD. It would appear that voice therapy coupled with surgery or Botox injections offers the best therapeutic management of spasmodic dysphonia. These interventions are described in the following sections.

Recurrent Laryngeal Nerve (RLN) Sectioning. Introduced by Dedo (1976), the RLN section (Izdebski, Dedo, and Boles, 1984) was the first surgical procedure for SD that was widely used. Patients are selected for RLN section after a thorough diagnostic evaluation by both the surgeon and the SLP, which includes an injection of Xylocaine into the RLN, which produces a temporary unilateral adductor paralysis. The patient’s airflow, relative ease of phonation, and change of voice quality are assessed. If there is marked improvement in airflow (greater flow rates with less glottal resistance) and in both ease and quality of phonation, the decision may be made to cut the RLN permanently. Postoperatively, then, the patient usually has an easily produced but breathy voice, similar in sound to the patient with unilateral adductor paralysis. Voice therapy focusing on a slight elevation of pitch, some ear training, head positioning and digital manipulation have all been effective in developing a better-sounding voice.

The long-term results of RLN resection have been mixed. Wilson, Oldring, and Mueller (1980) reported a woman who had received RLN cut 13 months previously who then experienced a regeneration of the severed RLN and a return of spasmodic dysphonia; a second RLN resection again produced immediate relief from her phonatory struggle. Over three years Aronson and DeSanto (1983) fol-

lowed 33 patients with spasmodic dysphonia who had each received RLN cut. Although all experienced improved voice and ease of airflow immediately after surgery, three years later 21 of them, or 64%, had failed to maintain their gains and were considered failures. Much different results were reported by Dedo and Izdebski (1983) on over 306 patients who had received RLN cut for spasmodic dysphonia; they reported that 92% of the patients maintained voice improvement and required less effort to phonate.

The arguments over the long-term effectiveness of RLN section as posed by Aronson and DeSanto (1983) versus Dedo and Izdebski (1983) contributed to a significant reduction in the use of RLN sectioning as a treatment for SD. Regeneration of the severed RLN appears to be the primary factor in symptoms of tight voice coming back a few months or years after RLN section. To meet this regeneration problem, Weed and others (1996) recommended the use of avulsion (tearing out or entire removal) of as much of the recurrent laryngeal nerve as is surgically possible. In the Weed study, long-term follow-up of RLN avulsion patients revealed that “72 to 78 percent of patients retained clear benefit from the procedure beyond 3 years” (p. 600).

Berke and colleagues (1999) described a new surgical technique for ADSD that paralyzes the thyroarytenoid and lateral cricoarytenoid muscles bilaterally by denervating the recurrent laryngeal nerve branches to these muscles. To prevent unwanted reinnervation and to preserve muscle tone, the TA nerve branch is reinnervated with a branch of the ansa cervicalis. The procedure, the authors note, obviates the breathy voice and other typical sequelae of unilateral vocal fold paralysis. The long-term results of 21 sequential cases were reported, with 19 patients judged to have an “absent to mild” dysphonia following the procedure and one patient requiring further Botox treatments. The opposite vocal behaviors were reported by one patient presenting to our clinic for voice therapy. Twenty months earlier he underwent bilateral laryngeal adductor denervation with ansa cervicalis reinnervation. He reported that although he no longer had to worry about strained and strangled phonation, he now had to worry about a soft voice that was insufficient for many social and professional activities of daily living. This patient responded well to intervention for unilateral vocal fold paralysis (see Chapter 6) (digital manipulation, focus, head positioning).

Even though higher airflow rates and lower pressures are noted after RLN surgery, many patients persist in maladaptive hyperfunctional postures, such as pushing and working hard to produce outgoing expiration, grimacing, and continuing to experience a marked reduction in normal prosody. Most of these hyperfunctional postures are unlearned with voice therapy, allowing us to agree with Dedo and Izdebski (1983) that the vast majority (92% in their study) of patients continue for years to enjoy improved voices with ease of airflow after RLN section.

Botulinum Toxin (Botox) Injections. The primary approach today for treating SD appears to be the injection of botulinum toxin (Botox) in one or both vocal folds (Miller, Woodson, and Jankovic, 1987). For many years, injection of Botox into

muscles in spasm has been found effective for eyelid spasms (blepharospasm) and for severe neck muscle spasms (torticollis). More recently Botox is being used to treat limb contractures secondary to stroke (O'Brien, 2002) and chronic headaches (Loder and Biondi, 2002), not to mention the smoothing of hyperfunctional lines (read *wrinkles*) (Semchyshyn and Sengelman, 2003).

One early report of successful use of Botox injections for SD patients (Blitzer and Brin, 1991) found that Botox injection into the thyroarytenoid (TA) experienced by 210 SD patients was “a relatively safe and effective mode of therapy for laryngeal dystonia” (p. 88). Botox is injected into the TA, unilaterally or bilaterally in very low dosages (1 to 3 U). While the typical site of injection for SD is on the vocalis section of the TA, there is strong research (Inagi et al., 1996) that found the best postinjection voice and the voice that lasted the longest was the result of unilateral injection toward the posterior end of the TA with some absorption occurring in the lateral cricoarytenoid (LCA). These authors concluded that Botox appears to have the best effect with a single “unilateral injection placed strategically at the posterior portion of the TA and directed toward the LCA so that both muscle groups are affected” (p. 306). Other Botox teams continue to use bilateral injections with slightly lower Botox dosages injected into each TA (*Advance*, 1998). The amount of Botox required and the site(s) of injection vary according to the experience of the individual team members and the patient’s response to the drug.

It is commonly observed in patients who have received Botox injection in the TA that they experience for a short time (two to three weeks) some mild symptoms of aspiration, coughing, and breathiness. Instead of tight phonation with low air-flow rates with high subglottal pressures, the SD patient now displays temporarily the symptoms of a patient with unilateral vocal fold paralysis, that is, high flow rate, low pressure, and a breathy voice. The patient at this time requires counsel from the SLP that the aspiration and breathiness are temporary. About three weeks after injection, the patient should return to the SLP for voice therapy. Murry and Woodson (1995) found in 27 patients that those who received both injection plus voice therapy had significantly better flow rates and acoustic improvement than those patients who received only Botox without follow-up voice therapy. Typically, those patients who received both Botox and follow-up voice therapy will maintain good, functional voice from four to six months. As the patient experiences increasing adductory tightness while phonating, reinjection of Botox will be required.

Murry and Woodson (1995) usually begin with “five voice-therapy sessions planned for each patient” (p. 462). Some patients may require less therapy and some may need more. Beginning therapy is designed to reduce continued vocal hyperfunction. The typical SD patient has used for many years hyperfunctional behaviors in an attempt to push voice out. Even though such excessive effort is no longer needed after injection, the patient’s habit set of vocal hyperfunction continues. Counseling, showing the patient differential airflow rates, and listening to pre- and postinjection recordings can be used to help the patient cognitively recognize that effort for voicing is no longer required. A useful task is to model in front of a mirror or on videotape the saying of “ah” with no discernible effort, no

visible neck muscle activity, resulting in a slightly easy, breathy voice. Stay with this task until the patient can demonstrate taking the work out of voicing. Therapy then follows with learning to find the optimal breath for saying a series of syllables on one expiration, perhaps reducing voice production in the beginning to saying only six to eight syllables per breath. If the patient is observed to squeeze out the last syllable or two, the syllable target per breath should be reduced further. Practice should be given to developing the number of syllables that can be comfortably voiced on one expiration. When breath volume gets low, the patient should pause; during the pause breath will renew without the patient doing anything consciously but pausing (Boone, 1997).

We have established a voice therapy practice plan that monitors the patient's vocal behaviors post-Botox injection. This program is individualized for each patient and normally begins with several follow-up telephone calls. We ask the patient whether the postinjection aspiration has resolved; we can hear any latent vocal hyperfunction, which would necessitate a follow-up visit to the clinic. Several weeks after injection, we listen for spasmodic vocal behaviors, which would also necessitate a return to clinic. If we detect the return of the dystonia, we ask the patient to return for acoustic and airflow assessment and possible reinjection.

Although no definitive information is available on the long-range effects of continued Botox injection, Lundy et al. (1998) did report on voice outcomes following Botox injections for 68 patients over a five-year period. Voice quality was significantly correlated with the underlying severity of vocal symptoms recorded prior to injection, incidence of breathiness, and unilateral versus bilateral injection. The length of favorable response was greater in males and following bilateral injections. An increased period of breathiness significantly correlated with bilateral injections.

Essential Tremor

Organic or essential voice tremor is often viewed as a disorder separate from the other dysarthrias, yet it can be classified as a hyperkinetic dysarthria of tremor (Duffy, 1995). Essential tremor is the most common of the movement disorders and is considered a benign autosomal dominant condition with variable penetrance (Jankovic, 1986). The tremor may appear present in tongue, velar, pharyngeal, and laryngeal structures, producing a vocal tremor in the 4 to 7 per second range. Other patients with voice tremor may show similar tremorous movements in the hands, arms, neck, and face (Aronson, 1991). A common form of essential tremor is familial tremor (approximately 50% of all cases), which may begin in early adulthood, in which the patient shows exaggerated tremorous behavior, showing more than the normal tremor that may be observed in people who are overworking particular muscles, such as may be felt or seen while carrying a heavy weight, "like carrying a case of twenty-four quarts of milk." Another form of essential tremor appears to be related to aging, although Colton and Casper (1996) and Greene and Mathieson (1991) report the senile form of tremor begins generally in the patients' late fifties.

Vocal tremor may also be heard in other neurogenic voice disorders, such as in spasmodic dysphonia and in Parkinson's disease. Such tremors must be differentiated from a diagnosis of essential tremor, which is basically intention tremor that appears to exist independently of other neurogenic conditions. The diagnosis of essential tremor is best made by eliminating contextual speech, asking the patient to sustain the production of vowels in isolation. The longer duration of the vowel, the more severe the tremor. On prolonged vowel production, the tremor is well isolated, permitting the frequency count and an acoustic evaluation of the tremorous voice. Endoscopic examination of the vocal folds while prolonging the vowel will show a structurally normal larynx with the vocal folds producing the alternate tension changes that are part of the overall tremor production. Flexible endoscopy can also reveal velar, pharyngeal, and tongue movements in absolute tremorous synchrony with one another, all contributing to the acoustic observation of voice tremor.

There is little in the literature to suggest adequate management of essential tremor, either medically or by voice therapy. The speech–language pathologist who first encounters an essential tremor patient, either of the familial or aging type, should make a referral to a consulting neurologist who might offer some medication control, reducing the severity (amplitude) of the tremor (but not its frequency). Professional meeting papers and anecdotal reports by voice clinicians offer three therapy approaches that seem to minimize voice symptoms: (1) reducing voice intensity levels appears to minimize tremor identification; (2) elevating voice pitch a half-note seems to change the tension level of the vocal folds sufficiently to reduce severity of the tremor; and (3) attempting to shorten vowel duration while speaking minimizes the identification of voice tremor (we are less likely to hear it).

When clients understand the nature of voiced versus nonvoiced phonemes, they discover that by abbreviating the vowels and overarticulating the nonvoiced phonemes the tremor is less noticeable. In addition, we encourage the client to produce an “easy” /h/ at the beginnings of vowel initial words, such as /h/apples, to reduce the amplitude of tremor. This technique worked well for a young client who worked as a telephone receptionist at Andressen Towing. Prior to intervention, when announcing her company's name on the telephone, she produced the initial /a/ with extended vowel duration, which only served to announce the tremor. With intervention, she softened and abbreviated the /a/ by making the voice breathy, devoiced the /d/, and anticipated the production of the nonvoiced /ss/. Using these strategies, the perceptual features of the tremor were not eliminated but certainly attenuated.

Differences Between Spasmodic Dysphonia, Essential Tremor, and Vocal Hyperfunction

Spasmodic dysphonia can easily be misinterpreted as an essential tremor or vocal hyperfunction. This comes as no surprise as the three conditions may present very

similarly (Barkmeier, Case, and Ludlow, 2001). In Table 4.1 we have attempted to identify some of the differences of the disorders, although it should be noted that the disorders may, and do, overlap. For example, a patient with severe ADSD may attempt to control the capricious vocal fold movements by squeezing down on the supraglottal structures. SD is only differentially identified when the individual undergoes trial voice therapy that eliminates the hyperfunctional posturing.

Huntington's Disease

Up to this point, we have described hyperkinetic dysarthrias that are progressive in severity in symptomology, yet not life threatening. Huntington's disease, in contrast, is an inherited autosomal dominant degenerative, neurological disease in which the first symptoms begin to emerge in middle age (40 to 50 years). Each child of an affected parent has an even 50% chance of inheriting the disease, with the onset of symptoms delayed until middle age. The disease is an extrapyramidal disorder of the basal ganglia, characterized by an overabundance of dopamine. This results in the onset of the disease beginning with occasional jerks or spasms in either the extremities or more centrally in speech and voice, progressing rapidly into chorea, athetosis, and mental deterioration (Berkow, Beers, and Fletcher, 1997, p. 000). The typical voice symptoms include strained or strangled voice quality, monopitch, excessive loudness variations, equal stress on ordinarily unstressed words, with sudden forced changes in breath control (Aronson, 1985). Among the most prominent symptoms are the jerky, irregular bursts of loud voice (Colton and Casper, 1996) and obvious interruptions of prosody.

In the early stages of HD, the SLP can guide the patient into maintaining better speech and voice, permitting good, functional communication. Voice seems to remain more normal when the patient works on easy, forward prosody, maintaining a rate near 150 syllables per minute. Both the DAF and metronomic pacer of the Facilitator can help the patient develop and maintain a slower controlled speaking rate. The slower rate seems to smooth out some of the unacceptable jerkiness. The yawn-sigh has been found a useful technique for opening up the vocal tract and developing ease of voice production. Similar to the patient with Parkinson's disease (another disease of the extrapyramidal tract), speaking with greater intention often enhances the patient's speech and voice behavior (Boone and Plante, 1993).

As HD progresses, with death occurring fifteen to twenty years after onset (Yorkston, Miller, and Strand, 1995), the patient usually begins experiencing some cognitive decline. At about this time, attempts at modification of speech and voice are no longer successful. Extreme choreic interruptions of airflow and flailing athetoid movements make speech intelligibility impossible. Because of both cognitive decline and severe motor control limitations, "there are no reports in the literature of successful application of augmentative communication technology in Huntington's disease" (Yorkston et al., 1995, p. 158). Therefore, improving speech, voice, and functional communication in the HD patient is usually possible only in the first few years after onset of the disease.

The hyperkinetic movements that interrupt respiration, voice, and articulation in Huntington's disease are the same that underlie the severe dysphagia that patients experience in the moderate and severe stages of the disease process. Yorkston et al. (1995) identify notable swallowing disruptions, such as tachyphagia, respiratory chorea, and eructation, and recommend positioning, dietary, and assistive feeding strategies to maximize oral intake success and safety (p. 167).

Ataxic Dysarthria

Ataxic dysarthria is a CNS disturbance caused by damage to the cerebellum or the cerebellar control circuit, with resultant respiratory, phonatory, and articulatory dyscoordination that may make the patient sound inebriated. Common etiologies for ataxic dysarthria are degenerative disease (see MS below), vascular disorders, tumors, and trauma.

In some patients, phonation is hoarse with a mildly tremorous overlay and respiratory function is interrupted by dyscoordinated inhalatory and exhalatory exchanges. Intervention for patients presenting with voice disturbances as a function of ataxia addresses those subsystems of speech that are most compromised. Intervention is similar to that described in the section for multiple sclerosis, which often presents with ataxic or ataxic-spastic symptoms. For patients presenting with reduced respiratory-phonatory coordination, we introduce Linebaugh's (1983) concept of optimal breath groups, that is, the number of syllables that a patient can produce comfortably on one breath. The patient is encouraged to experiment with breath support and keep utterances within the optimal breath group. One companion technique that has been particularly successful in clinic is to have the patient produce the word "boom" at the end of each breath group. This ensures that the patient has sufficient expiratory support for the entire phrase ("Take me to your summer home: *boom*"). Eventually, the "*boom*" is phased out. To regulate vocal amplitude and pitch, we introduce pacing, both tactile (Yorkston et al. 1995) and auditory (Facilitator), along with visual and auditory biofeedback on the Visi-Pitch or auditory only, using a digital or analog tape recorder.

Mixed Dysarthria

This condition is a mixture of dysarthrias characterized by two or more of the aforementioned primary types. Mixed dysarthria is caused by multiple lesion sites within the nervous system, which may involve both the central and peripheral nervous systems.

Amyotrophic Lateral Sclerosis

ALS is a progressive degenerative disease of unknown etiology involving the motor neurons of the cortex and the gray bodies within the brain stem and spinal cord.

Involving both upper and lower motor neurons, the disease is often called *motor neuron disease*. It is also known as Lou Gehrig's disease. The speech-language pathologist often sees these patients initially relative to their early complaints of difficulty in articulating rapid speech, experiencing occasional hoarseness, and complaining of occasional swallowing problems. On peripheral oral examination, on extension of the tongue, fasciculations (traveling, wavelike muscle tremors) on the surface of the tongue may be observed. The diagnosis of ALS often requires a muscle biopsy to identify lack of innervation to particular muscle groups; the diagnosis is often related to exclusion of other identifiable etiologies. As ALS progresses, the patient experiences proximal atrophy of extremities rather than distal (for example, shoulder atrophy before hand involvement or back tongue impairment more than anterior involvement). The ALS patient may develop voice harshness, hypernasality, back-pharyngeal resonance focus, breathiness, and monopitch (Silbergleit, Johnson, and Jacobson, 1997). In addition to these voice symptoms, these authors also report an articulatory deterioration and increasing dysphagia.

Of life-threatening concern is the patient's growing inability to clear the throat and to cough. Because most ALS patients are experiencing continuing bulbar involvement, more clinical focus needs to be given to swallowing and coughing, rather than to speech and voice per se. Yorkston et al. (1993), present the speech scale from their Amyotrophic Lateral Sclerosis Severity Scale, which rates the patient's speech-voice function.

In the early stages of the disease, we attempt to increase respiratory, phonatory, and resonance support for speech, which may involve building a palatal lift for patients who present with weak or spastic velar function. A palatal lift consists of a palatal portion that is attached to the teeth and a lift portion that extends posteriorly to lift the palate in the direction of velopharyngeal closure. Fitting the lift requires adequate dentition to retain the device; however, lifts have been successfully built into upper denture plates (Duffy, 1995, p. 396). Working with the prosthodontist, we view the velopharyngeal port at rest and during speech activities using flexible endoscopy. We identify the points of air escape and fashion the lift so that velopharyngeal closure is adequate for voice, speech, and swallow, while still allowing for nasal breathing. A palatal lift is shown in Figure 9.6. It should be noted that palatal lifts are difficult to fit for patients with hyperactive gag reflexes that are unresponsive to desensitization; however, on occasion we have successfully reshaped a lift to accommodate sensitive patients. Lifts are also not appropriate for patients who are not cooperative and patients who respond to the presence of the lift with increased mucous production. This latter factor is a troublesome variable, especially in the area of dysphagia.

Whether a palatal lift is indicated, some voice improvements may occur by helping the patient to renew breath more often and to develop a high front voice focus, with some attention given to increasing speaking rate (we have found metronome pacing to be helpful).

As the disease progresses, the SLP must help the patient work on diet modification and swallowing (trying different head positions with good mouth closure). These patients have good cognitive function and follow suggestions well if

the suggestions are within their motor ability to execute them. As the Yorkston scale suggests, eventually the patient may require some kind of augmentative communication aid.

Before a communication aid is selected, the SLP must determine the patient's literacy and cognitive level, specifically when selecting icon- versus text-based systems, communication needs, and hand function and mobility. A number of text-to-speech devices are available that offer scanning options as the disease progresses (DynaWrite by DynaVox; LightWRITER by Zygo).

Multiple Sclerosis (MS)

Among various demyelinating diseases, multiple sclerosis is the most common, affecting about 400,000 people in the United States (Berkow, Beers, and Fletcher, 1997). Although the cause of MS is believed to be viral, specific causes are unknown. This slowly progressive disease attacks the myelin sheath covering of nerves, literally causing breaks in transmitting axons, within the white matter of the CNS. The symptoms of the disease depend on the site of involvement. The patient may experience either sensory deficits (tingling, numbness, visual changes) or motor deficits (weakness, spasms, lack of coordination); more commonly both sensory-motor systems are involved. Citing the early work of Darley, Aronson, and Brown (1975), nearly 60% of 168 MS patients studied were judged to be normal in speech adequacy (Yorkston et al., 1995).

The increasing presence of dysarthria with problems of voice in MS is generally related to multiple neural system involvement associated "with cerebral, brain stem, and cerebellar involvement" (Yorkston et al., 1995, p. 192). The voice problems experienced by MS patients were earlier described (Darley, Aronson, and Brown, 1975; Farmakides and Boone, 1960); they are listed here in descending order of most common to least common: impaired loudness control, harsh voice quality, a scanning sameness of prosody and voice pitch control, decreased breath control, and hypernasality. If the MS patient is experiencing some or all of these symptoms, direct voice therapy can often minimize symptom effects, improving the patient's communicative effectiveness. Considering the progressive nature of the disease, communication between caregiver and the patient is increasingly needed.

Changing the patient's speaking rate (slightly slower or faster) will often have positive effects on loudness and harshness. The patient learns to pace speech or uses the metronome pacing program developed for use with the Facilitator (Boone, 1998). Improving rate control is also consistent with developing better coordinated breath support. MS patients may profit from reducing the number of words they say on one expiratory breath. Baseline measures of expiratory breath control can serve as a starting place. The SLP then instructs the patient to cut in half the number of words he or she has been saying. This reduces vocal fold tension, the tendency to squeeze out the last words of an utterance. Developing good vertical postural habits with the patient, keeping the chin down, and minimizing mouth opening (while at rest) all seem to afford the MS patient a neutral postural set be-

fore initiating speech–voice responses. This postural control and attempt to pace an even spoken response seems to inhibit the sudden jerkiness and loud voice excesses that interfere with effective communication.

In the advanced stages of the disease, natural speech–voice may not be possible. Setting up alternative means of communication, also, may have many obstacles, as the patient’s hand control for using keyboards, pointing, or pressing switches may be seriously compromised by ataxia, spasticity, and excessive tremor. Also, in advanced stages of multiple sclerosis, the patient may have severe visual problems and even blindness. Many of these obstacles can be successfully identified and addressed by performing a comprehensive augmentative and alternative communication assessment, as described by Beukelman and Mirenda (1998, p. 439).

Traumatic Brain Injury

Traumatic brain injuries are caused by external forces acting on the head. Most TBIs are caused by motor vehicle accidents; falls and assaults account for the rest (Brookshire, 2003). These injuries can cause focal or diffuse lesions, axonal shearing and hypoxia, secondary to vascular or tissue damage.

Dysarthria associated with TBI may be temporary or chronic, mild or severe, and accompanied or not by other language and cognitive disorders (Yorkston et al., 1999). Most dysarthrias are of the mixed type, and variability in the nature and severity of the physiological impairment calls for custom treatment programs based on a clear appreciation for the subsystems of respiration, phonation, resonance, articulation, and prosody. Studies of speech breathing, for example, reveal that individuals with TBI have lower vital capacities than nondisabled speakers (Murdoch et al., 1994). Kinematics of the same group revealed that the speakers with TBI had problems coordinating the actions of the rib cage and abdomen during speech. This incoordination is apparent in patients with TBI, many of whom take replenishing breaths at inappropriate phrase junctures during conversational speaking and oral reading tasks.

Victor was a 21-year-old male who experienced a TBI with bilateral basilar skull fractures, left-sided cerebral edema, and subarachnoid and subdural hemorrhages. He came to our clinic after spending 30 days in the intensive care unit on ventilatory support and one year in full-time rehabilitation. Victor presented with a mixed spastic–ataxic dysarthria; speech was characterized as slow at 82 words per minute, with reduced articulatory accuracy, reduced respiratory–phonatory coordination, and strained and strangled phonation, notably on the vowels. Voice ground to a glottal fry at the ends of phrases.

The first step in the voice and speech rehabilitation program was to familiarize Victor with the anatomical and physiological bases for voice and speech production. He learned that he indeed had sufficient breath support for speech; however, respiratory–phonatory coordination needed some adjustments. He also

learned that he could produce more words at a more rapid rate when he increased pitch and abbreviated vowel duration times. Improved voice, speech, and prosody were targeted through a variety of tasks. Using the Visi-Pitch for audio and visual feedback, Victor generated novel sentences ranging from five to eight words. He viewed the pitch traces on the screen and immediately heard his productions using the digitized feedback option. The productions were judged using a ±rating scale for articulation, breath support, vocal quality, and overall presentation of the sentence. If Victor was dissatisfied with a production, he was reminded to modify breath support, increase pitch, and reduce vowel durations. At the end of the semester, respiratory–phonatory coordination for conversational speech had increased from 57% accuracy to 85% accuracy, speaking pitch increased from 94 Hz to an average 110 Hz, words per minute had increased from 82 to 89, and intelligibility had increased from 74% to 80%, as measured by listener transcripts. He began speaking over the telephone again and soon found employment at a youth league.

Summary

At the beginning of the chapter we looked at the neurological bases of human laryngeal function. We then reviewed abnormal neurological functions in the motor speech system, and subdivided those abnormalities by site of lesion, with particular emphasis on laryngeal neurological dysfunction. We reviewed vocal pathologies associated with lower motor neuron dysfunction, notably vocal fold paralysis. We then discussed the upper motor neurons system and explored numerous pathologies that serve to interrupt normal laryngeal function: primarily, spastic, hyperkinetic, hypokinetic, and mixed dysarthrias. We reviewed the latest research in behavioral, pharmacological, and surgical management of neurogenic voice disorders, and explored voice programs from the perspective of the SLP.

THOUGHT QUESTIONS

1. Describe vocal characteristics of a unilateral vocal fold paralysis. Describe the physiological underpinnings of the characteristics.
2. Describe similarities among and differences between muscle tension dysphonia and adductor spasmodic dysphonia.
3. Describe the vocal and resonance characteristics that may be observed in an individual with TBI.
4. Describe the vocal characteristics of hypokinetic dysarthria and some intervention approaches.