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[Note: Throughout the text all terminology introduced will be defined and explained to ensure readability at a first year introductory level.]

# Preface

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The world was a different place in 1861. The Civil War raged in the United States and brothers fought a horrific battle at Ball's Bluff in Virginia. The Canellas meteorite struck the earth near Barcelona, Spain. The telegraph left the Pony Express obsolete and horses with mail pouches stopped racing across the American West. Charles Darwin burst on the scene with the publication of *Origin of the Species*. Xian Feng, a Chinese Emperor, died and was succeeded by his 6-year-old son. Explorers Burke and Wills set out with camels and horses and 18 men to cross Australia's outback from South to North. Both died on the return trip of exhaustion and hunger. San Francisco was rocked by a series of small tremors, followed by a major earthquake on July 4.

In France, history was being made as well. Tremors across the intellectual communities were created by Pierre Paul Broca, who opened a dialogue on brain and language by presenting the first of two landmark papers (Broca, 1861a, 1861b). These papers have since served as a clear demarcation and turning point in the history of understanding brain-based disorders of language and speech. Broca is one of our heroes. He encapsulates all that a good clinician, scholar, and neuroscientist should be. He cared for and about his patients. Broca understood the whirlpool of despair into which a shoemaker from rural France could be plunged by an

unseen stroke in his brain. He guided, nurtured, and studied the small victories of recovery from torn brains in his practice. Clearly, 1861 was a big year. So it came to pass that Carl Sagan (1979) rhapsodized in *Broca's Brain* about the exquisite experience of standing in the *Musée de l'Homme* as he contemplated and gazed upon the preserved brain of Pierre Paul Broca (June 28, 1824–July 9, 1880) this scientific giant of the 19th century who contributed so much of what we know about the localization of articulate speech in the brain of humans. Broca was a French physician, anthropologist, and eventually a senator who believed that by studying the brains of cadavers and correlating the known experiences and behaviors of the former possessor of the organs, human behavior could be revealed, associated with brain function, and better understood. For that purpose, he collected hundreds of human brains in jars of the preservative formalin. Upon his death in 1880, with exquisite irony, his own brain was preserved in formalin and added to the collection in the museum, along with hundreds of skulls that Broca had used in his comparative cephalometric studies. When Sagan happened upon Broca's brain in the museum, along with that of Broca's milestone patient "Tan" Leborgne, he was awestruck by the irony of it all. Here was Broca, for whom the region of the frontal lobe of the cortex that he had described was subsequently

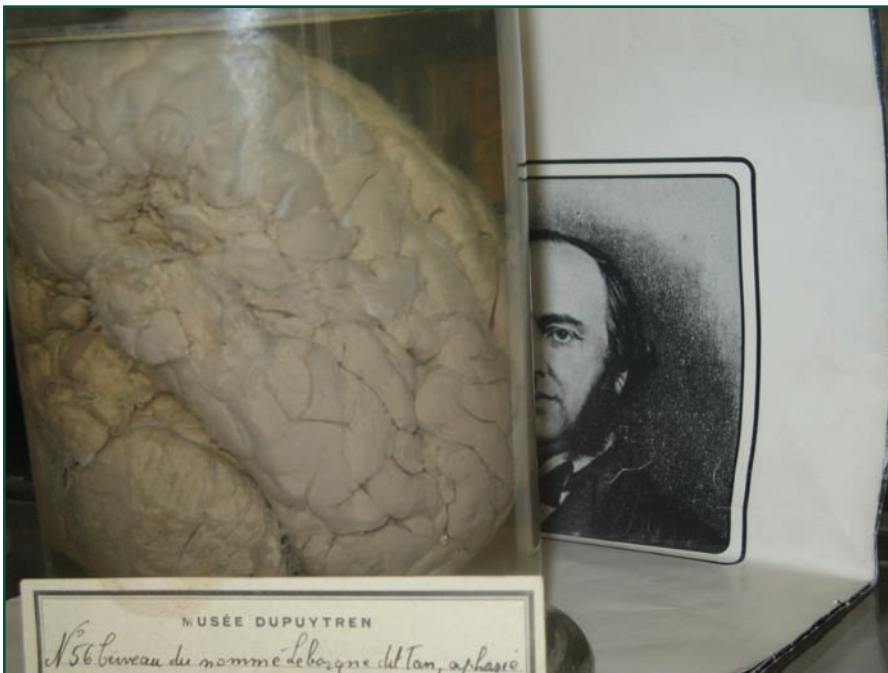
## X Brain-Based Communication Disorders

named, with his own Broca's area discernible. Sagan was mesmerized by the incongruity of all of this. In *Broca's Brain*, he used that visit to the Museum of Man to launch philosophical questions that challenge some core ideas of human existence and consciousness such as "How much of that man known as Paul Broca can still be found in this jar?"

These questions were pondered again as we looked upon the brains of Broca's patients in the Dupuytren Museum in Paris in July of 2009. This question and other brain puzzles are pondered in the essay, "Broca's brain: Brother, wherefore art thou?" (LaPointe, 2010). Indeed, we are indeed in the debt of this French scientist and scholar as well as of his patients whose brains rest even today in these jars.

In 2010 the world is different and the world is the same. Twitter, social

networking, texting, and 24-hour information access have created digital autism and consume us as we text our way off of the curbs into the traffic of perpetual connectivity. Neural imaging that would have appeared to be magic in 1861 is now commonplace. Diffusion tensor imaging (DTI) and magnetoencephalography (MEG) are guiding us to better visions of broken brains and their consequences. Sophisticated ways of viewing brain activity have led to speculations about the study of different gamma oscillation patterns for true versus false memories and even into the seeming brain science fiction of "mind gaming" or thought controlled activities. What was rudimentary in 1861 and speculative in 2010 may be commonplace in days to come. But people with brain-based communication disorders still will require attention and intervention by clever and



The brain of Broca's patient LeBorgne ("Tan") in formalin at the Duputryn museum in Paris (LaPointe photo).

educated experts. That is what this book is all about.

*Brain-Based Communication Disorders* introduces the reader to the major clinically recognized types of acquired speech/language, cognitive, and swallowing disorders encountered by clinicians working with child and adult neurologic cases. The text provides contemporary and state-of-the-art content on these disorders in terms of their neuropathologic bases, clinical symptomatology, and prognosis. Basic anatomy and physiology of human communication and swallowing are introduced, as well as the neural mechanisms controlling speech, language, cognitive, and swallowing functions. In addition to the traditional acquired speech/language disorders of the nervous system (aphasia; neuromotor speech disorders), content including communication impairments caused by traumatic brain injury, multi-system blast injuries, and degenerative disorders of the nervous system also is provided. The reader also is introduced to the principles that govern the assessment and treatment for both pediatric and adult populations. We are indebted to

those who passed before, such as Pierre Paul Broca who showed us the path. We are indebted as well to all those who have undergone the slings and arrows of brain damage from whom we have learned. We hope the future generations of brain scientists and clinicians will grow in their ability to understand and to help.

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## Motor Speech Disorders in Adults: Dysarthrias and Apraxia of Speech

*Jason is sitting in a therapy room talking about his workout regimen. He is a young man of 22, with a sleeveless shirt to show off his “guns.” He is enrolled part-time as a student at a local university where he also attends speech therapy. When he talks, the reason for speech therapy becomes clear. He is difficult to understand. In fact, listening to him and understanding requires a good bit of concentration on the part of the listener. His speech rate is slow; he stops occasionally to adjust his palatal lift prosthesis, which sometimes rattles in his mouth. “It needs an adjustment,” Jason says. He also must work very hard to coordinate breathing and speech, getting a few words out then pausing to take another lengthy breath for a few more words. On his neck is a telltale scar, indicating that, at one time, a machine had to do the breathing for him.*

*Jason is the classic picture of a young person whose life was dramatically changed after a motor vehicle accident. When asked to tell his story, Jason reflects . . .*

*Three years ago . . . I went to a party . . . and got really drunk . . . then decided to drive home . . . 30 miles. I made it 29 . . . when I passed the officer . . . (makes a car steering motion) I went off . . . hit three trees . . . going 90 miles an hour (another big breath). I was lucky he was there . . . it was the middle of the night . . . I never would have made it . . .*

Such is the story for many of the people we work with. An accident, a disease, a stroke, can change life in an instant. So many of the things that were once taken for granted—the ability to speak clearly, to breathe without effort—are now a constant challenge. Communication/speech is so integral to everything we do, and when it becomes difficult to produce clearly, what a dramatic change it makes to the overall quality of a person’s life.

## Introduction

In reading through these chapters and the many individual stories about brain-based communication impairments, you have learned more about the complexities of communication. We hope that your interest as a future clinician has been piqued by the fascinating array of neurogenic disorders, even though your heart aches for the suffering those individuals experience (welcome to the world of allied health). Throughout this process, you probably began to develop an appreciation for the differences between speech and language. They are inextricably linked, yet can be differentially affected by damage to the brain. The previous chapters of this textbook were devoted primarily to those disorders of language and cognition (aphasia, disorders of a nonfocal nature, and right hemisphere dysfunction) that deal with the organization, formulation, and social rules of communication. What you will find in the following chapters is a review of impairments that relate purely to the motor execution of speech (i.e., muscle capability, motor plan). A motor speech impairment indicates problems with one or more aspects of

the speech mechanism as reviewed in Chapter 2. Just to remind you,, those component systems include respiration, phonation, resonance, and articulation. The speech difficulties experienced by a person with a motor speech impairment fall into one of two categories: some type of disruption in (1) the speech musculature or (2) the motor plan that arrives from the brain, which directs the muscles how to move. These categories each carry a specific diagnosis:

**Dysarthria:** A group of neurologic speech disorders resulting from abnormalities in the strength, speed, range, steadiness, tone, or accuracy of movements required for control of the respiratory, phonatory, resonatory, articulatory, and prosodic aspects of speech production.

**Apraxia:** A neurologic speech disorder reflecting an impaired capacity to plan or program sensorimotor commands necessary for directing movements that result in phonetically and prosodically normal speech. (Duffy, 2005, p. 5)

When you consider the following excerpt from the definition of dysarthria, “. . . abnormalities in the strength, speed, range, steadiness, tone or accuracy of movements . . .” you can derive that this definition is really referring to the status of the speech musculature, something about the muscles of the speech mechanism is causing a problem with the coordination and execution of speech. On the other hand, the definition for apraxia makes no mention of how speech is produced, but how it is planned or programmed. Therefore, although both are considered motor speech disorders, they are two distinctly different problems.

## Scope of the Problem

In Chapter 3, we learned that 49 million Americans have a communication disorder. That number reflects both developmental (problems you are born with) and acquired (a problem that occurs after normal development of speech/language has occurred) causes. An extensive study of acquired communication disorders in over 14,000 cases that occurred in the Department of Neurology Mayo Clinic from 1987 to 1990 and 1993 to 2001 revealed that roughly 41% suffered from motor speech disorders (Duffy, 2005). Based on that number, it is probably safe to say that you will encounter an individual with a motor speech disorder at some point in your practice as a speech-language pathologist. It is just as likely that currently you may know someone who has motor speech impairment, perhaps a friend of the family with Parkinson disease, or a neighbor who was in a car accident, or a grandparent who suffered a stroke.

## Definition

From the above definition of the dysarthrias as a “group of disorders,” it becomes obvious that there is more than one type of dysarthria. In the late 1960s, a group of master clinicians (Darley, Aronson, & Brown, 1969a, 1969b) suggested that dysarthria, which up to that point was only differentiated from aphasia and perhaps apraxia of speech, actually seemed to contain several subtypes. Their careful analysis of over 200 patients with dysarthria led to six specific types (flaccid, spastic, ataxic, hyperkinetic, hypoki-

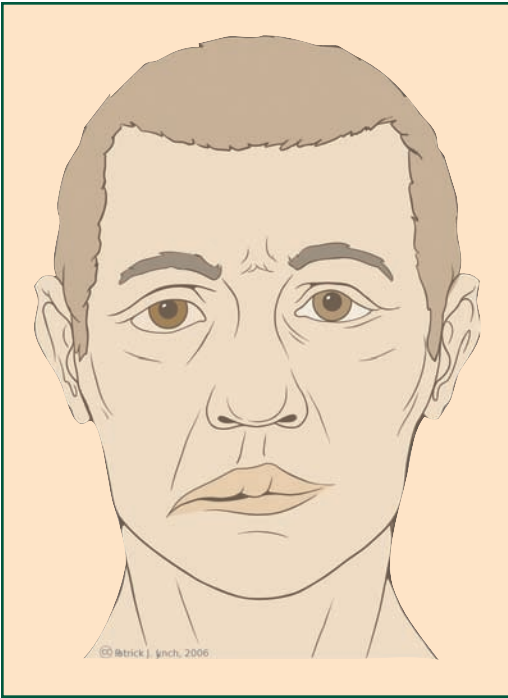
netic, and mixed), classified by common lesion sites as well as unique characteristics of motor (muscle) performance.

## Dysarthria

### Flaccid Dysarthria

Flaccid dysarthria is named for the nature of the muscle in those who suffer from this diagnosis. Flaccidity means a lack of muscle firmness or tone so muscles will appear to be somewhat floppy or droopy. The general lack of tone in the muscles is also known as hypotone (hypo- meaning less than optimal) and it can be an indication of muscle weakness. A look at Figure 8–1 reveals characteristics of flaccid paralysis. The individual in Figure 8–1 has a condition Bell’s palsy, which is a flaccid paralysis of the hemiface (half the face). Notice the droopiness or sagging of the structures surrounding the eye, nose, and mouth on the right side of the individual’s face. Another example of the condition (Bell’s palsy) is seen in Figures 8–2A through 8–2E. Notice that, in this case, the condition is less obvious when the facial structures are at rest. When the structures are functioning, however, you can see that movement is compromised and the weakness becomes more apparent.

In Chapter 1, you learned about the general organization of the nervous system into the central nervous system (CNS) and the peripheral nervous system (PNS). As a reminder, the CNS contains the brain and spinal cord, and the PNS is composed of the cranial and spinal nerves that arise primarily from the brainstem and spinal cord. Flaccid



**FIGURE 8–1.** An illustration of Bell's palsy, paralysis of the facial nerve (cranial nerve VII). From Wikimedia Commons. Patrick J. Lynch; illustrator; C. Carl Jaffe; MD; cardiologist Yale University Center for Advanced Instructional Media Medical Illustrations by Patrick Lynch, generated for multimedia teaching projects by the Yale University School of Medicine, Center for Advanced Instructional Media, 1987–2000. Patrick J. Lynch (<http://patricklynch.net>), Creative Commons Attribution 2.5 License 2006.

dysarthria is a result of damage to the PNS. More specifically, flaccid dysarthria is present following damage to the cranial nerves that are involved in speech and/or the spinal nerves that supply the respiratory system. Table 8–1 contains a listing of the cranial nerves that contribute to speech production. In reviewing this list, you should recognize that these cranial nerves are responsible for a large portion of the sensory and motor function of speech execution.

In Chapter 2, the structures of the speech mechanism were reviewed, including the respiratory, phonatory, and resonance systems and the articulators. Flaccid dysarthria can affect just one, a combination of, or even all of the systems that contribute to speech. The systems that are affected are dependent on where the damage lies in the PNS. Seven of the twelve cranial nerves in the PNS contribute in some way to speech production. Functionally, these nerves provide sensation and movement to those structures responsible for phonation (larynx), resonance (soft palate or velum), and the articulators (lips, teeth, hard palate, jaw, and tongue). It is easy to imagine, then, that damage to these nerves would result in problems with speech execution, making individuals who suffer from flaccid dysarthria difficult to understand (unintelligible). Although there are many speech characteristics that can fall under the umbrella of flaccid dysarthria, those most frequently noted are hypernasality, imprecise articulation, breathy voice quality, and shortened utterances.

With your knowledge of the cranial nerves, let's analyze these symptoms (Appendix 8-A). Hypernasality means "too much nasal resonance." You have learned from this text that resonance has to do with the movement of the velum or soft palate. You also know that the velum typically is elevated to seal off the nasal passages so that, for most speech sounds, sound resonates and airflow passes through the oral cavity, *not* the nasal cavity. Thus, hypernasality must mean that the velum is not doing its job effectively and allowing too much sound and airflow to pass through the nasal cavity. Simply put, the sound resonating in the nasal passages when it should not distorts the sound. In addi-





A



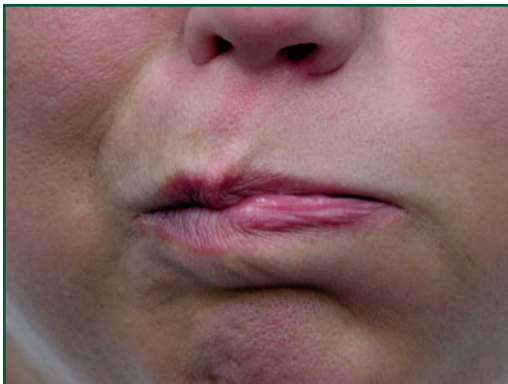
B



C



D



E

**FIGURE 8–2. A–E.** Signs of Bell's palsy. Notice that during rest (A), the face looks fairly symmetric. However, during functional tasks (B, C, D), the weakened side is pulled toward the stronger more intact side. Finally, when the cheeks are filled with air (E), the lips on the weakened left side cannot maintain a functional seal to keep the air from escaping.

tion, the airflow traveling through the nasal cavity detracts from the airflow that is needed in the oral cavity to accurately articulate many of the speech sounds. All this occurs when there is damage to

the motor portion of cranial nerve X or vagus, which supplies the velum.

Imprecise articulation indicates a problem with the function of the articulators, specifically, those articulatory

**Table 8–1.** Cranial Nerves That Contribute to Speech Production

<i>Cranial Nerve</i>	<i>Name</i>	<i>Motor/ Sensory Mixed</i>	<i>Primary Function for Speech</i>
V	Trigeminal	Mixed	Facial sensation (S) Jaw movement (M)
VII	Facial	Mixed	Oral cavity (S) Facial expression (M)
VIII	Auditory	Sensory	Hearing and balance
IX	Glossopharyngeal	Mixed	Pharyngeal sensation (S) Pharyngeal movement (M)
X	Vagus	Mixed	Laryngeal sensation (S) Phonation, Velar elevation (M)
XI	Spinal-Accessory	Motor	Head and shoulder movement
XII	Hypoglossal	Motor	Tongue movement

structures that are dynamic or moveable (lips, jaw, and tongue). In order to produce intelligible speech at a normal rate (around 140 words per minute), the articulators must move rapidly and with a great deal of precision. If they are not able to, the rate of speech production will slow and the precision or crispness of speech will be affected. The cranial nerves that are implicated for the movable articulators include trigeminal, facial, glossopharyngeal, vagus, spinal accessory, and hypoglossal (see Table 8–1). I am sure that you notice another nerve that is included in the table that is not listed here. Although not directly involved with the innervation (nerve supply) to the articulators, cranial nerve eight (vestibulocochlear) also is important because it is the auditory nerve, and hearing speech does influence how it is produced.

Another of the primary features of flaccid dysarthria is a breathy vocal quality. The quality of the voice has to

do with phonation. A breathy vocal quality is an indication of excessive air leaking through the glottis (the space between the folds). In the case of flaccid dysarthria, excessive air goes through the glottis due to incomplete closure during voicing, likely secondary to the hypotone and weakness in the laryngeal musculature. In some individuals, there may be bilateral weakness of all the muscles of phonation and in others only one side may be affected by weakness (paralysis).

A number of possible etiologies (causes) may lead to flaccid dysarthria. Traumatic injuries, which may be secondary to everyday accidents involving the head and neck, inadvertent injury during surgeries to the neck, and motor vehicle accidents that place a great deal of stress on the brainstem area, all could contribute to damage to the PNS affecting speech (Freed, 2000). Flaccid dysarthria also may be present following a stroke. The type of stroke would have to be

very specific, however. In Chapter 3, you learned about strokes that occur in the brain. In order to impact the PNS, the stroke would have to occur in the brainstem. Remember that the majority of cranial nerves originate along the brainstem so a stroke (whether ischemic or hemorrhagic) in this location likely would have a direct impact on the adjacent cranial nerves (see Figure 1–16).

Disease is another potential cause of flaccid dysarthria. There are conditions or diseases that directly impact the PNS. Several examples include Guillain-Barré syndrome, myasthenia gravis, and muscular dystrophy. The common theme to all etiologies that result in flaccid dysarthria is that they somehow impact the PNS.

### Spastic Dysarthria

Spastic dysarthria, like flaccid dysarthria, is named for the nature of the muscle impairment present in this condition. Spasticity in muscle refers to *hypertone*, or excessive muscle tone. So, whereas the muscles in flaccid dysarthria are lacking tone or floppy, muscles in an individual with spasticity have increased tone. An understanding of spasticity is important here. People may think of increased muscle tone, or tight muscles, indicate strength (aren't tight muscles what we would all like to have?). For the "buff" people working out at the gym, that may be the case. In the situation of spasticity, however, increased tone should not be equated with strength. Instead, spasticity exists in muscle after there has been bilateral damage to the motor pathways in the brain. Therefore, although spastic muscles are tight, they are not strong. In fact, they more typically demonstrate weakness. The combination of increased

tone and weakness can make it difficult to move structures (Figure 8–3); in extreme cases, tight contractures caused by spasticity can lead to changes in the structure. This phenomenon can and does occur in the speech musculature as well. As a result, the most salient speech features of spastic dysarthria are imprecise articulation, reduced pitch and stress variation, and harsh vocal quality, implicating primarily the articulatory, phonatory, and respiratory systems. Although some degree of hypernasality may be present, resonance typically is not one of the primary problems for individuals with spastic dysarthria.

Spasticity, then, is a condition that could affect all subsystems of the speech mechanism although it is likely that



**FIGURE 8–3.** Spasticity in the lower limb.

some aspects will demonstrate more impairment than others. Unlike flaccidity, however, the effects of spasticity will impact the entire system. For example, if phonation is impaired by spasticity, then muscles in the entire system will demonstrate the effects of increased or hypertone. In flaccid dysarthria, you might see just one aspect of phonation (one vocal fold) demonstrate weakness in a system that otherwise functions well. Because one of the primary features of spastic dysarthria includes imprecise articulation, it is clear that the increased tone has a direct impact on the articulatory system. Restricted movement of the articulators from the spasticity leads to difficulty in making full articulatory contacts and difficulty with the rapid movements required to transition between sounds. These restrictions translate directly to imprecision and a slowed rate of speech. The inaccuracy that occurs will affect all speech: consonants as well as vowels.

Phonation is another speech system often affected by spastic dysarthria. In fact, the phonatory characteristics may differentiate spastic dysarthria from other dysarthrias, as many of the dysarthrias also include imprecise articulation and slowed rate. Spasticity affects the intrinsic laryngeal musculature exactly in the manner you would expect. The excessive tightness of muscles leads to tight adduction (hyperadduction) of the vocal folds. The result is the perception of a “strained” vocal quality. The tightness in the musculature also impacts the muscles that are responsible for changing vocal pitch. Thus, individuals with spastic dysarthria tend to have little pitch variation or intonation in their speech (monotone).

Now let’s consider the respiratory system. Remember that the respiratory

system never rests, breathing is necessary 24/7; thus, those muscles are continuously working. Tight muscles of the respiratory system can limit the amount of movement for breathing, resulting in shallow breaths. Shallow breaths will certainly impact speech (fewer words per breath, reduced stress patterns) but also may compromise gas exchange (oxygenation of the blood and tissues). If this restricted movement occurs over a long period of time, it can actually change the shape of the rib cage, which will contribute even more to limitations in movement. Because you understand that respiration is akin to the power source that drives speech production, then you know that with more shallow breaths there is less air pressure to generate voice and shape into speech. Therefore, when spasticity affects the respiratory system, the result will be reduced breath support for speech resulting in short utterances, low volume, reduced stress patterns, and less air pressure for generating speech sounds.

Etiologies that lead to spastic dysarthria include any condition or injury that results in bilateral brain damage. Remember that, in order to demonstrate spasticity, damage has to occur in the motor pathways in both left and right hemispheres. The usual culprits are stroke (most likely a second stroke as it has to occur in both hemispheres), a lack of oxygen to the brain (anoxia), traumatic brain injury, multiple sclerosis, and the list goes on. Although this list looks diverse in the type of injury, the common thread is the diffuse nature of brain injury that occurs. Widespread injury, such as what occurs with traumatic brain injury or anoxia, most certainly will include both hemispheres. Conditions like stroke or multiple sclerosis, on the other hand, can impact

smaller, more discrete areas, but if they occur in both hemispheres, spasticity can result.

## Ataxic Dysarthria

Another of the dysarthrias is called ataxic dysarthria. Ataxic dysarthria is named for the condition of *ataxia*, which is defined as a general lack of coordination in muscle movements. In Chapter 1, you learned about the cerebellum, a structure that is located at the back and below the cerebral hemispheres. The cerebellum is “highly connected” to motor pathways on several levels. First, there are connections to and from the cerebral hemispheres, there is proprioceptive feedback from the speech mechanism, connections with the brainstem, and interaction with the basal ganglia. Functionally, the cerebellum helps to coordinate and refine motor movements, an activity that actually sounds much simpler than it really is.

Let’s consider an example. On a late spring day, you come in from a hot, steamy walk around campus and make what of course would be your first stop, the refrigerator for a nice cold beverage. You approach the refrigerator and reach out for the handle. Instead of slamming your hand into the door of the fridge, you slow your movements so that you grasp the handle with just the right amount of force. Then you open the door and look inside. Once you spy the object you want, you once again reach forward to grasp the can/bottle. Now, chances are you won’t knock the can or bottle over in your attempt to pick it up. Instead, your hand slows to a stop just as you reach the object and you will grasp it with just enough force to keep it contained in your hand as you pull it from

the fridge. This describes such an easy task, something we do every day without even considering how difficult it is. At each phase of movement, there is a great deal of planning, refining, coordinating, and smoothing movement so that the end result occurs without incident. The cerebellum is the structure that facilitates and oversees this process in its entirety. More specifically, the cerebellum assists with the timing, degree, and coordination of motor movement. Consequently, damage to the cerebellum will result in movements that are halting, jerky, and lacking in precision and timing, with a general lack of coordination that affects entire movements.

The movements just described are ataxic in nature. Following cerebellar damage, ataxic characteristics will be seen across all motor systems of the body, those responsible for walking (ambulation), standing, speech, or any motor activity that you might attempt. Another example of ataxia is in the speech of individuals who are inebriated. Excess alcohol is toxic to the cerebellum and its effects, although transient (short-term) are presented as ataxia. The speech of individuals who have ataxic dysarthria has been described as similar to “drunken” speech. The distinguishing features of ataxic dysarthria then, relate to the irregular movements that can occur within the speech mechanism. In particular, the articulatory system demonstrates the most salient features of ataxic dysarthria.

When disruptions in the articulatory timing and force of movement are present, the production of consonants, vowels, and the transitions between them are uncoordinated. Consequently, the precision we have discussed as necessary for intelligible speech is sorely lacking. Instead, the articulators tend to

overshoot or undershoot their placement targets, sounds may be prolonged, distorted, or possibly even omitted altogether. Another important feature is that the pattern of imprecision is not always predictable. An example can be seen in one of the common assessment tasks, diadochokinetics. In this task, speakers are asked to produce a syllable (usually /puh/, /tuh/, or /kuh/; see Chapter 11 for a full description of this task) as accurately and rapidly as possible. Performance on this task in a “normal” system yields approximately 5 to 7 repetitions per second (Tomblin, Morris, & Spriestersback, 2000). An individual with flaccid or spastic dysarthria would perform much slower but the productions would be steady and rhythmic. However, an individual with ataxic dysarthria would have productions that vary. They might produce two syllables slowly followed by three rushed and one prolonged syllable. Because the underlying problem of ataxia is systemic, the coordination of all the motor systems for speech is a difficult task. Additionally, the demand for coordination can change based on the speaking task, context, even the emotional state of the speaker. As such, articulatory disruptions are irregular and perhaps unpredictable; for example, a word that may be produced perfectly in one sentence may be distorted in another.

The effects of ataxia also may disrupt movements of the respiratory system, more specifically, the coordination between respiration and phonation. Phonation occurs on the exhalation cycle of breathing, and when the systems are in sync, speech is an efficient, almost effortless product. When the two systems are not well coordinated, the result may be speech that is not so easy. As an illustration, try the following: Breathe in, and as you breathe out, begin

counting at a comfortable rate. How far did you get? Counting to 10 should be relatively easy for an intact system. Now count to 10 again but breathe in and before you begin to count let about half of the air escape before you begin to count. How did that go? Counting to 10 on less air is more difficult, isn't it? You probably had to push harder at the end of the utterance to keep speech going. That concept of “pushing harder” means that you were engaging additional muscle systems (probably the abdominals) just to complete the utterance. This simple comparison of using air efficiently and not so efficiently shows how “out of sync” systems can impact speech. During that task you may also have noticed that stress patterns were atypical. Did you notice stressing each number equally? Abnormal stress patterns that overlie speech are another of the features that differentiate ataxic dysarthria from the other dysarthria types. Careful orchestration of all the systems working together is what makes speech so easy for most individuals. For individuals with cerebellar damage, however, imagine systems that are out of sync and working less efficiently all the time (Murdoch, Chenery, Stokes, & Hardcastle, 1991). Ataxia is a condition that makes that scenario a reality. Speech for individuals with ataxic dysarthria, then, is hard work!

The conditions that contribute to cerebellar damage resulting in ataxic dysarthria are similar to what you might find with the other dysarthrias with a few exceptions. First, the primary contributors are degenerative conditions. Examples include unspecified cerebellar degeneration, olivopontocerebellar atrophy, multiple systems atrophy, and Shy-Drager syndrome to mention a few. Demyelinating diseases, such as multiple sclerosis, can also cause cerebellar

damage. Stroke or CVA is another cause that is actually common across almost all dysarthria types. Of interest to note, however, is that if a stroke occurs in one cerebellar hemisphere, the prognosis for rapid recovery is favorable. Strokes that occur in the vermis (midline) or that affect both hemispheres will have more lasting effects and disability. One cause of ataxic dysarthria that is unique relates to toxic or metabolic disturbances. Alcohol toxicity, alluded to earlier, is one example, but there are other substances/medications that can reach toxic levels and result in ataxia. For the most part, the ataxia that comes from toxic or metabolic influences can be reversed. However, if the toxicity continues or is severe, there may be permanent damage with irreversible effects.

### Hypokinetic Dysarthria

The diagnosis “hypokinetic dysarthria” is similar to some of the other dysarthrias in that the name itself describes what to expect in speech features. As a reminder, “hypo-” means *a lack of*, or *less than expected*, and “kinetic” means *movement*. Right away, understanding the meaning of the diagnosis reveals that individuals who suffer from hypokinetic dysarthria will demonstrate generally less movement, as seen throughout the components of the motor speech mechanism. The general lack of movement stems from disruptions with the basal ganglia control circuit as described in Chapter 1. The basal ganglia and its connections really function to regulate muscle tone, support motor behaviors, make postural adjustments during movement, respond to environmental changes, and assist with the initiation of motor movements and new motor learning. A listing of these functions illustrate that

damage to the basal ganglia circuit definitely will impact motor control and movement. In hypokinetic dysarthria, damage to the basal ganglia is secondary to a depletion of the neurotransmitter dopamine, which is provided by the substantia nigra. The prototype example for this diagnosis is Parkinson disease. Cardinal features of Parkinson disease include resting tremor, rigidity, bradykinesia (also known as hypokinesia or akinesia), and reduced or lack of postural reflexes. Considering these features, bradykinesia/hypokinesia is perhaps the condition that contributes the most to the clinical signs of hypokinetic dysarthria.

Muscle rigidity in the speech musculature will restrict movements. You might see parallels between the muscle rigidity as a feature of hypokinetic dysarthria and the hypertone seen in spastic dysarthria. Indeed, the impact on speech systems can be similar, but there is an underlying difference in muscle rigidity and spasticity. Increased muscle tone or spasticity is a condition that results in muscle tightness. However, spasticity can be alleviated through deep massage or range of motion (ROM) activities. When a structure with spasticity is moved, the initial resistance of spasticity will ease as the structure continues through the movement. Although muscle rigidity also results in muscle tightness, it does not respond to massage or ROM exercises. The tightness demonstrated by muscle rigidity will resist all the way through a movement.

The speech characteristics that best represent hypokinetic dysarthria reflect the underlying primary features of Parkinson disease. The most common speech impairments include reduced pitch, loudness and stress variation, short rushes of speech, dysphonia, and the perception of rapid speech. (It is

worth noting that hypokinetic dysarthria is the *only* motor speech impairment in which speech rate is perceived as fast rather than slow.) Muscle rigidity is another muscle condition that is systemic in nature. Thus, components of speech production may have decreased functionality because of the “tightness” of the musculature. With regard to respiration, the symptoms are similar to what may be present in spastic dysarthria. The restricted movements of the respiratory muscles result in a reduced lung capacity. Reduced capacity, and thus air support for speech, will result in an overall reduced intensity (volume) as well as less variation of intensity (used for stress patterns). The phonatory system also demonstrates the effects of muscle rigidity in a general lack of pitch variation. The muscles that manipulate laryngeal structures to change vocal fold tension (thus pitch) are tight, not making the fine adjustments to the system for normal pitch contours, like the rising pitch you typically hear at the end of an utterance when an individual asks a question. Muscle rigidity leading to the lack of pitch variation is somewhat expected in these individuals. However, another aspect of laryngeal function in hypokinetic dysarthria has been a bit perplexing. Dysphonia commonly has been listed as a feature of the diagnosis. Dysphonia is a voice quality also known as hoarseness, which includes some degree of breathiness. Any presence of breathiness in a system with rigidity in the musculature is counterintuitive. If the muscles of phonation are rigid, then you might expect an excessive or hyperadduction of the vocal folds, which would lead to more of a “strained” vocal quality. Instead, the vocal folds actually are hypoadducted, which results in that

hoarse, breathy voice quality. The underlying mechanism for this unexpected clinical sign is unclear and keeping some researchers busy trying to find an explanation. Articulatory function also is affected by the nature of the muscles in hypokinetic dysarthria. The effects of Parkinson disease lead to articulators that are restricted in their range of motion; thus, they tend to undershoot targets. The imprecision in meeting articulatory contacts results in speech that is difficult to understand. The strength of the articulators may be in the normal range (at least until the advanced stages of the disease) but muscle rigidity limits movements. The restricted movement also is noted in the muscles of facial expression. The lips are an active articulator of the face, but the rest of the face also adds content to what we express through speech. Individuals with hypokinetic dysarthria have what is known as “masked facies” or a general lack of facial expression during speech or to express emotion. The face may have features that are still, like the immovable features on a mask.

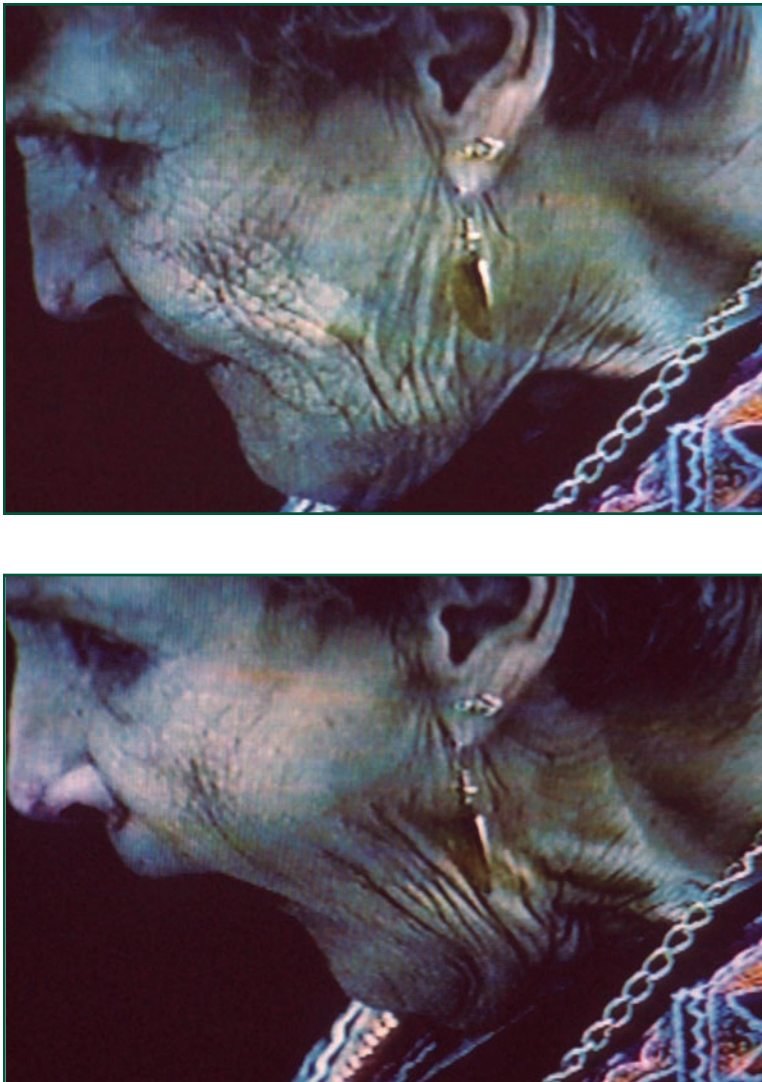
Degenerative conditions are by far the primary cause of hypokinetic dysarthria. Parkinson disease is the major contributor, but there are other conditions that share some of the characteristics of Parkinson disease that have additional symptoms as well. Those diseases are often called Parkinson-plus syndromes and contain such diagnoses as multiple systems atrophy, progressive supranuclear palsy (PSP), and corticobasal degeneration. Involvement of the substantia nigra and dopamine depletion are all common elements, although most of these other diagnoses demonstrate more of a mixed dysarthria (hypokinetic plus another dysarthria type).



## Hyperkinetic Dysarthria

As you have just learned, the diagnosis hypokinetic dysarthria means too little or reduced movement of the speech musculature resulting in impairment. At the other end of the spectrum is “*hyperkinetic dysarthria*,” which is defined as excessive movement of the speech musculature also resulting in disrupted

speech. A large number of movement disorders result in excessive, involuntary movements of the body. When those movements affect the components of speech, hyperkinetic dysarthria is the result. Depending on the movement disorder, a single speech component (as in the jaw opening dystonia in Figure 8–4) may interfere with speech, or it is possible that the entire system will be



**FIGURE 8–4.** An example of jaw opening dystonia, one type of hyperkinetic dysarthria.

affected by the involuntary movements making speech difficult to produce.

The movement disorders that result in hyperkinetic dysarthria range from slow to fast movements, small tics of a single body part to whole body movements. Each diagnosis demonstrates a unique pattern of movements. The wide range of disturbance makes it difficult to predict speech characteristics based on simply “hyperkinetic dysarthria” as you can with other dysarthria types (flaccid, spastic). However, there is one unifying symptom that spans the diagnoses: involuntary movement.

The movement disorders that result in hyperkinetic dysarthria are often associated with damage to the basal ganglia. Remember that *hypokinetic* dysarthria also is linked to the basal ganglia; however, the mechanism for hypokinetic dysarthria (i.e., depletion of the neurotransmitter dopamine) is fairly well understood and is isolated to basal ganglia connections, specifically the substantia nigra. The mechanisms for the wide range of movement disorders linked to hyperkinetic dysarthria are not as well understood. The importance of the basal ganglia in regulating the movements planned by the motor cortex is not disputed, but the mechanism of injury/disease that results in such a wide variety of impairments remains a question and a source of ongoing inquiry.

Exactly how the speech mechanism is affected by movement disorders really depends on the type and extent of the condition. As previously mentioned and illustrated in Figure 8–4, an individual may have a fairly focal (isolated) dystonia that only interferes with one structure. In the case of the jaw opening dystonia, an individual might be talking and suddenly (often where there is an open mouth posture for a vowel) the jaw begins to open and perhaps stays

open on its own accord. Imagine how even a relatively isolated impairment such as this might disrupt speech. Just try saying the days of the week, and when you get to Wednesday, open your jaw slowly to an extreme open posture. Were you able to keep talking? If the jaw opening was not extreme, speech might be possible. However, remember that these are *involuntary* movements so it is unlikely that you will have a choice in how wide the jaw opens. Now let’s consider a more extreme movement disorder that affects the entire body. Huntington’s chorea is a genetic disorder characterized by random movements of the head, neck, trunk, and even the limbs. It is difficult to even simulate such a condition for the purposes of illustration as you did just now with a jaw opening dystonia. Imagine, though, what a sudden hard rotation of your entire trunk would do to the careful balance of breathing, phonation, . . . speech production in general. If you have never seen this condition, the impact can be quite dramatic. Some individuals have so little control that their flailing limbs can even strike them in the face, sending glasses flying. In spite of the wide range of impairments, there are speech features that seem to predominate in hyperkinetic dysarthria. Those features include imprecise consonants, variable speaking rate, prolonged speech segments and pauses, harsh vocal quality, distorted vowels, variable loudness, and voice interruptions. To further review how speech systems might be affected by hyperkinesias, view Table 8–2.

## Mixed Dysarthria

The dysarthrias are a group of disorders, each single diagnosis with a unique set of features that include muscle charac-

**Table 8–2.** Characteristics of the More Common Movement Disorders

<i>Movement Disorder</i>	<i>Movement Characteristics</i>	<i>Systems</i>
Chorea	Rapid, unpredictable, jerky involuntary movements	This is a progressive disease that will likely impact the entire speech mechanism
Dystonia	Slower, more gradual movements	May impact all systems; however, resonance is not typically implicated
Myoclonus	Rhythmic, periodic muscle contraction	Can affect multiple systems; however, the resonance and articulatory systems are often implicated
Tics	Brief involuntary movements (can also be sounds) during normal activity	May be isolated or inclusive of all speech components
Essential tremor	Periodic movements occurring at around 4 to 7 Hz (cycles per second)	Phonation and articulatory systems are often implicated
Spasmodic dysphonia	Involuntary muscle spasms/contractions	The phonatory system

teristics, nervous system lesions, and motor behaviors. Although the list is well described, there are some individuals who may not fit neatly into the characteristics of just one diagnosis. In fact, because damage to the brain is not always specific to one system, many individuals show signs of more than one dysarthria type. Typically, they suffer from widespread or diffuse nervous system damage. For these individuals there is a “mixed dysarthria” category. For example, the disease amyotrophic lateral sclerosis (ALS or Lou Gehrig’s disease) is a demyelinating condition that affects the CNS as well as the PNS. People who have been diagnosed with ALS frequently show signs of both spastic dysarthria (often in phonation) and flaccid dysarthria (resonance and articulation). To conclude our overview of

the dysarthrias, the mixed dysarthria category completes the group of dysarthrias. When an individual suffers from nervous system injury that is diffuse or widespread, then many features of the diagnoses discussed above may be seen in combinations, rather than fitting neatly into an isolated category.

## Apraxia of Speech

We are going to move away from the focus on dysarthria onto another motor speech disorder, apraxia of speech. The common thread of impairment that spanned the dysarthrias was muscle or motor impairment, which resulted in speech disturbance. The manifestation of speech disruption in apraxia of



# Principles of Treatment for Neurologic Communication Disorders

*“‘Sorry, Jill . . . it’s a brain tumor.’ Two of the most feared words in the English language. She broke it to me as gently as she could. Brain tumor. Unwanted growth. It wasn’t malignant and they said it probably wouldn’t grow back. But they had to do surgery to take it out. I hope they got it all. They said it was pretty much confined to the region of my frontal lobes right behind my eyes. What could make that stuff grow in my brain? I need my brain. Dr. Firlik said the surgery was a success, though they had to destroy some healthy brain tissue to get all of the “neoplasm,” their term for the unwelcome, infiltrating little bitch that grew in my head and tried to ruin my 33-year-old life. I don’t have any trouble walking or with my arms but I’m worried about whether I’ll be able to take care of my baby. I can’t seem to concentrate. My attention wanders. I don’t know why I can’t finish the things I start. My speech gets lost a little, too. Sometimes I get lost in the middle of telling them something. Even things I should know like what I did last summer at Lake Ellen during our trip to Upper Michigan. My brother and sisters were there. We made pasties, those little Cornish meat pies. I couldn’t even remember the steps. What comes first, the potatoes or the crust? Screwed up pasties. Screwed up following directions. Screwed up life. What’s going to happen? They said they could treat it. Maybe further surgery. Maybe some different medications. Maybe some speech therapy. Maybe I’ll need some counseling as well. This is not what I had in mind for my life. Maybe I’ll get better. Maybe all this treatment stuff will work. It better. I’ll need all the help I can get to raise precious little Adrienne with only part of a brain. Oh, well. Such is knife.”*

## Introduction

“Treatment” can have many meanings. In apparel and fashion design, it refers to modifying fabrics or clothing. In film, treatment refers to turning prose into a screenplay. In ecology, sewage treatment refers to processes of removing contam-

inants from wastewater. In health care, treatment is synonymous with therapy, the process of attempts to remediate medical or behavioral problems to a result that is desirable and beneficial. Treatment of health issues and disorders of communication can range widely from medical, surgical, pharmaceutical, and/or behavioral approaches (Figure 12–1).



**FIGURE 12–1.** Care of the sick. From *Care of the Sick*, Domenico di Bartolo, 1441–1442. Public domain on Wikimedia Commons.

In Jill's case, in the scenario listed above, treatment was multimodal and involved several different professions. After complaints to her family physician, her brain tumor was diagnosed by a neurologist, a medical specialist in brain diseases. She then was referred to a neurosurgeon who operated on her to remove the meningioma that had grown slowly in her brain behind her eyes. Her hospital stay necessitated association with several others on the health care team. Nurses, rehabilitation professionals in occupational therapy and physical therapy, specialists in social work who attended to her needs after discharge, the hospital pharmacist, and the speech-language pathologist all entered the picture as part of her treatment team. The focus of Jill's treatment is rehabilitation and the skills she had acquired but were lost or impaired by the tumor.

## Rehabilitation

What distinguishes habilitation from rehabilitation is that *habilitation* is intended for people with functional impairments caused by congenital injuries or disease, or to injury or disease acquired early in life. Its objective is to help a person in every way develop the best possible functional ability. *Rehabilitation* involves helping the functionally impaired person in every way regain the best possible functional ability and physical and mental well-being after the reduction or loss of a function due to injury or disease. Habilitation and rehabilitation both aim to prevent and ameliorate the difficulties that functional impairments can cause in daily life. The goal of both

is maximal function and best possible quality of life. The goal of habilitation and rehabilitation is a noble one: that people with functional impairments should be able to live their lives as independently as possible with the same rights, opportunities, responsibilities, and obligations as the rest of society. Habilitation and rehabilitation usually are used as comprehensive terms to cover all medical, psychological, social, educational, and work-oriented measures undertaken to help children and adults who are suffering from a medical condition or have been injured to develop, or regain, the best possible function and lay the foundations for a good life. In brain-based communication disorders all of the modes and venues of treatment may be necessary. We rely heavily on behavioral treatment, that is, interventions designed to change communication behaviors, but along the way many other professionals get involved.

## Treatment of Communication Disorders: Fundamentals

Communication is a multidimensional dynamic process that allows people who have learned language to interact with their environment. Through the marvel of communication, we are able to express our thoughts, needs, and emotions. It is so fundamental that it has been characterized as essential ingredients for the "Three Ls," Living, Learning, and Loving (LaPointe, 2005). Without it, we cannot carry out the mundane activities of daily living like ordering a cappuccino with a cranberry bagel, listening to a Stephen Lynch song about beautiful babies, or the hundreds and hundreds

of other interactions we engage in every day that require us to write, talk, listen, or speak. We cannot read directions, assignments, medicine labels, or listen to lectures. Most importantly of all, we cannot generate and nurture our interpersonal relationships; without discourse our contact with friends and family would wither and fade away. Communication is an intricate process that involves cerebration, cognition, hearing, speech production, and motor coordination. Evaluation and treatment of a communication disorder includes consideration of all aspects of the normal communication process as brain-based disorders can interrupt, corrupt, and disrupt any or all of these processes.

Language is the transformation of thoughts into meaningful symbols (sounds in the air or squiggles on paper or screen) communicated by speech, writing, or gestures. Thoughts are organized by the brain, specifically the left cerebral hemisphere, and encoded into a sequence according to whatever the learned grammatical and linguistic rules of the culture and language might be. These rules govern the way sounds are organized (phonology), the meaning of words (semantics), how words are formed (morphology), how words are combined into phrases (syntax), and the use of language in context (pragmatics) (Emedicine.medscape, 2010).

Speech involves the coordinated motor activity of muscles involved in respiration, phonation, resonance, and articulation. The entire system is modulated by both central and peripheral connections of brain cell bodies and axons to muscles and organs. Speech is greatly dependent on a specialized network of cranial nerves V, VII, VIII, IX, X, XI, and XII, as well as the phrenic and intercostal nerves. The cranial nerves

are traditionally labeled with Roman numerals, but these paired nerves also have names. For speech, the primary cranial nerves are the trigeminal (V), facial (VII), vestibulocochlear (VIII), glossopharyngeal (IX), vagus (X), spinal accessory (XI), and the hypoglossal (XII) (Emedicine.medscape, 2010).

For speech, the respiratory muscles, specifically the muscles associated with expiration, must generate enough air pressure to provide adequate breath support to make speech loud enough to hear (Emedicine.medscape, 2010). The diaphragm is the main muscle of expiration; however, the abdominal and intercostal (between the ribs) muscles help control the force and length of exhalation for speech. A great dance of muscles is necessary to produce the vibrations of phonation and these muscles of the larynx generate vibratory energy during vocal fold approximation so that sound is produced. Say “AHHHH.” It happens. Vocal pitch and intensity are modified by air pressure below the level of the vocal folds (subglottic), tension of the vocal folds, and position of the larynx. We can scream and we can whisper. We can sing in falsetto like the Bee Gees of the golden oldies or we can assume our habitual natural pitch level. Articulatory muscles within the pharynx, mouth, and nose modify and valve the tone of the sound and produce all of the little beads on the string of speech that consists of phonemes (speech sounds) and their transitions. The intricate and coordinated action of these muscles produces speech. By altering the shape of the vocal tract, we are capable of producing a tremendous range of sounds. Speech requires the balanced coordination of over 100 muscles at speeds that are a blur. Speech is our most complex, balanced, coordinated

human neuromuscular activity. It must be nearly perfectly innervated to create these time-space movement coordinates. No wonder little things, such as strokes, head banging, golf-ball sized tumors, or nasty diseases that strip the nerves of their conductivity, can throw it off.

Sound waves are transformed by the auditory system into neural input for both the speaker and the listener. The outer ear detects sound pressure waves in the air (or even under water, but not nearly as distinctly) and converts them into mechanical vibrations in the middle and inner ear. The cochlea, that magical snail-like shell full of hair cells and fluid, then transforms these mechanical vibrations into vibrations in fluid, which act on the nerve endings of the vestibulocochlear (VIII) cranial nerve. Thus, the process of communication begins and ends in the brain (Figure 12–2). Communication is what makes us human. The brain lends itself to the creation of a never ending list of metaphors. The

brain is a computer; the brain is a web; the brain is a river; the brain is an orchestra; the brain is a cookbook; the brain is a snowflake; the brain is a dream factory. No wonder we value it more than, say, our great toe or our appendix.

All of the disorders previously discussed and feared may impair a person's ability to communicate. These disorders may involve voice, speech, language, hearing, cognition, or even the basic task of taking food and swallowing it. Recognizing and addressing communication disorders is important; failure to do so may result in isolation, depression, and loss of independence. Life without communication caused by brain damage has been described as a living death by those who have recovered enough to comment on it. So we do our best to understand the clinical science of brain-based communication disorders interwoven with the clinical science and art of rehabilitation and restoration of this most human and complex function.



**FIGURE 12–2.** Brain.



## Rehabilitation Assumptions and Principles

In a European Neurology Society Meeting on neurorehabilitation, Butterworth and Howard (1990) articulated some theoretic suppositions that can serve as useful principles for rehabilitation of brain-based disorders. They listed several assumptions, some of which may appear as a firm grasp of the obvious, but nevertheless form an elemental firmament for treatment. Because their suggestions did not include as much emphasis on the acknowledgment of the effects of these impairments on life participation, we have modified them to reflect contemporary approaches to rehabilitation. These basic assumptions oblige reflection and realization.

- We must identify the exact nature of impairments and determine their impact on acceptable life participation and activities
- We must match treatment to the identified impairments and life participation needs of each unique individual
- We must be eclectic in our adoption of treatment approaches. Treatment may encompass any or all of the strategies of:
  - relearning information or procedures that have been lost
  - facilitation of access to information or skills that are intact but inaccessible
  - reconstitution or compensation for lost functions, that is, finding new ways to achieve goals
  - accommodation, adjustment, and *Aristos* (i.e., making the best of a given situation) (LaPointe, 2005)
- We must ensure that the person and families with whom we are working

have the opportunity to communicate their treatment objectives and outcomes and that these desires are incorporated overall treatment goals

- We must incorporate the principles of evidence-based practice and evaluate the effectiveness of treatment (Dollaghan, 2007)
- We must utilize principles of cross-disciplinary collaboration and cooperation by participating in professional teams.

## Other Principles Worthy of Consideration

In their book *Aphasia: A Clinical Approach* (1989), Rosenbek, LaPointe, and Wertz devoted a chapter to “Principles of Clinical Aphasiology.” These principles have broader application than just to the disorder of aphasia and may well be germane to most of the brain-based communication disorders. These authors stated:

We learned our principles from others—other professionals and the [men and women with aphasia] who have agreed over the years to spend time with us.

We have learned the same things over and over from some, and unique things from a few. Like a code for living, the principles governing our daily activities with [people with aphasia] have evolved, sometimes so subtly that they escape our notice until we sit down, as now, and take stock.

The clinical principles outlined by Rosenbek, LaPointe, and Wertz (1989) included some that have evolved a bit, some that are relatively timeless, and all that seemed to reflect the opinions of deeply committed clinical professionals

who had a passion for their work with hundreds of people with communication disorders. For example, they opined:

- Clinical aphasiology requires clinical aphasiologists—Part-time clinicians, if they are full-time clinical aphasiologists, can treat people with aphasia. But simple caring is not enough. Intellect and a subscription to *Brain and Language* also are not enough. Nor is having earned one's 10-year pin or 10 ASHA-approved CEUs in a year. Training in neurolinguistics is not enough, nor is knowing how to run a successful private practice from a garage after working all day in the public schools. [People with aphasia] deserve the care of a clinical aphasiologist. Clinical aphasiologists care about [people with aphasia]; they spend time with them every day; they read, remember, and integrate what they read; they go to workshops to stay abreast; and they specialize in the understanding, diagnosis, and treatment of aphasia and [people with aphasia].
- Treatment has three goals—The goals of treatment are: (1) to assist people to regain as much communication as their brain damage allows and their needs drive them to, (2) to help them learn how to compensate for residual deficits, and (3) to help them learn to live in harmony with the differences between the way they were and the way they are.
- The most important goal usually is to prepare persons with aphasia for a lifetime of aphasia—This is not true in all cases but certainly is true in most of the chronic conditions with which we are confronted. Too many people (and health care professionals) generalize the acute-care medical

model concept to the challenge of dealing with chronic conditions. This problem is reflected not only in the misguided or failed expectations of working with people with chronic conditions, but also gravely reflected in the reimbursement and insurance issues that permeate many health care systems. Get 'em in; get 'em out is not an adequate model for dealing with chronic conditions and many brain-based disorders indeed are chronic. Until this philosophy of economic, bottom-line approaches to health care is radically changed, professionals and sufferers must prepare for the frustrations of inadequately allocated reimbursement for chronic conditions. That will indeed "ensure" (no pun intended) that our clients must be prepared for long-term chronicity.

- Treatment should be influenced by an attitude about what a person with aphasia is—Persons with aphasia do not talk at all or nearly as well as before becoming ill, and all their other communicative acts are impaired in varying degrees as well. In addition, they are likely to be more irritable, scared, depressed, and distractible than before they got sick. Despite these changes, they often are unchanged at the core. If they were loving before, they will be again. If they were irascible, they will remain so. If they were making their ways through life, they will continue to do so. If they were dying, regardless of how subtly, the episode causing the disorder will not be an antidote. Above all, if they were doing the best they could before, they will set about doing the best they can to adjust to their disability and to the treatments that are likely to accompany it. All of this is not fatalism or resignation. It is applause for

the resiliency of people with brain-based disorders, and it is an appeal for professionals to avoid getting in the way of each person's recovery. It is also the summary of an attitude that profoundly influences what we think. Clinicians should reinforce the client's personal strengths and support their natural processes. They should treat people with aphasia and not aphasia. Knowing what to support, what to change, what to accommodate, and what to ignore are marks of clinical maturity. Many of these thoughts and philosophies about treatment were generated and hatched by the outstanding clinician Jay Rosenbek, and the egg was then warmed if not scrambled by Wertz and LaPointe. These principles guided all three of their clinical lives.

### Specific Skills and Knowledge

The skills and knowledge involved in assessing and treating brain-based disorders of communication and swallowing require some general and some very specific competencies. These include:

- assessment, appraisal, and diagnosis skills with emphasis on interviewing proficiency, preparation and test administration, interpretation of the results with subsequent ability to integrate and present what has been learned in both oral and written form
- clear and concise report writing with emphasis on the goal of comprehensible documentation and explanation
- therapy practices that incorporate task analysis, behavioral objectives, and implementation (Figure 12–3),

and integration of information from others on the professional team

- documentation of treatment outcomes, progress, and accountability that conform to the scope of practice and ethical standards of the appropriate discipline
- implementation or referral for appropriate client and family counseling.

### Social Models of Treatment

Some of the material in this section has been considered in a chapter on social validation of treatment for aphasia that has appeared in a *Festschrift* for Chris Code in a book edited by Ball and Damico (2006). As LaPointe and Lenius (2006) have reviewed, traditionally, clinicians have used standardized tests such as the Western Aphasia Battery (Kertesz, 1982) to determine areas of impairment, select therapy goals, and possibly document change. This focus on impairment has been consistent with traditional approaches to disease characteristic of the medical model, but may not be the most efficient way to determine socially relevant goals. The seemingly ever changing terminology of the World Health Organization is being integrated into models of aphasia treatment (Rogers, Alarcon, & Olswang, 1999; Threats, 2006), and the concepts involved are important. Changes in social activities and participation are emerging as more relevant goals of treatment and are eroding the stone face of impairment-based approaches to intervention. The Life Participation Approach to Aphasia continues to have an impact on approaches to aphasia intervention and is shaping the very core of the aphasia treatment model (LPAA Group, 2000). Most stan-



**FIGURE 12–3.** Example of therapy practice.

dardized tests of aphasia are predominantly impairment based. Recently, some efforts have been made to incorporate the social model of aphasia into assessment procedures. The latest edition of Eisenson's classic *Examining for Aphasia* (LaPointe & Eisenson, 2009) incorporates principles of the social model of aphasia into assessment in an effort to guide clinicians in the quest to develop relevant participation-based treatment goals. This approach does not suggest that we discard the infant with the cleansing aqua, for impairments and cognitive-linguistic processes also may need to be targeted for treatment; but for far too long it has been impairment or nothing, with little emphasis on reintegration of the person into an active, participating milieu. This attitude and

set of values is epitomized by the recent work of Audrey Holland on the importance of counseling in communication disorders from a wellness perspective (Holland, 2008).

The traditional biographic standardized test interview needs to be supplemented or indeed replaced with specific information gathering as to the goals, values, hobbies, likes, dislikes, hopes, fears, anticipations, and motivations of each unique individual for whom we are planning treatment. This will assist the informed clinician with planning, delivering, and evaluating services (Pound, Parr, & Duchan, 2001). The LPAA Project Group (2002), Simmons-Mackie & Damico (2001), LaPointe (2002), and Cruice et al. (2005) all provide social model flesh and humanity to the bones

of treatment planning in aphasia. This entire movement of course harkens back to the birth and upbringing of language in context and person-centered concepts of aphasia intervention nurtured and influenced so thoroughly by Audrey Holland (Holland, 1982),

Interview questions appropriate to determining and creating socially relevant and valid treatment goals can be gleaned, inferred, or directly created from all of the above sources and from the assessment protocol of LaPointe and Eisenson (2008). The interview allows the client and any others present to express what life was like before the onset of aphasia, how life is impacted now, and what areas of communication breakdown generate the most stress on relationships.

In an effort to define socially valid aphasia therapy goals, Ross and Wertz (2003) set out to determine the difference in quality of life as rated by people with and without aphasia. Two groups (people with aphasia for at least six months and non-brain-damaged individuals) completed quality of life measures. The greatest difference between the groups was evident in areas of activities of daily living, opportunities to acquire new information and skills, social support, mobility, and sexual activity. Of course, it is imperative to customize treatment planning and weave specific treatment goals around the idiosyncrasies of each person, but this study shows support for language therapy focusing on enhancing communication for specific functional situations and expanding participation in society. Recent work by Ross and her colleagues (Ross, LaPointe, and Katz, 2008) has suggested that loneliness and sense of belonging also are factors that impact people with brain-based communication disorders

and should be a consideration in treatment planning and the attempt at implementing treatment outcomes.

Sometimes we tend to err on the side of overzealous treatment planning and realization. One of the authors of this book (LLL) remembers well an early treatment planning faux pas when a full court press of intervention (twice per day; intensive reading comprehension strategies integrated into the treatment schedule) was probably not in keeping with the personal goals of the person with aphasia. After two weeks of rather intensive treatment, I remember his words well: "Doc, I thank you for all you're trying to do here, but, you know, I never have completely read through a whole book. I don't really care if I ever do. I just want to go up on the St. Johns River and live with my brother in the trailer there, and maybe sit out on the dock and fish a little. I know you're trying real hard Doc, and I appreciate it, but I really don't care if I can't read very good."

If the treatment goals of the person with aphasia are not built into the treatment plan, misguided expenditures of time, effort, and money can be the result. This clinical lesson made it abundantly clear that precise activity and participation therapy must be harmonious with the expectations and needs of the person in therapy. Sometimes the people with whom we work are content to fish, plant kumquats, or join the circus (Figure 12-4).

These principles set the ground for our belief system in interacting with people with brain-based disorders of communication and swallowing. We know not what the future holds, it will surely be trials and tribulations, but just as surely it will contain family holidays, fish curry, happy weddings with henna hands (Figure 12-5), faithful pets, art,



**FIGURE 12–4.** Circus. From “Circus Amok Jugglers” by David Shankbone, New York City, 2006. Taken from Wikimedia Commons under the Creative Commons Attribution 2.5 License.



**FIGURE 12–5.** Henna.

music, and sports peak moments, walks on the beach (Figure 12–6) and new directions and accomplishments. Good treatment in both principle and implementation can help restore or compensate for these joys of living to people with brain-based disorders (Figure 12–7). As Small (2000) has implied, we can

expect not only advancement of our understanding of how the brain operates in order and disorder, but also an abundance of new mysteries that will pique our curiosity and motivate our questions. That is one of the many reasons we get up in the morning.



**FIGURE 12–6.** Sunset on the beach.



**FIGURE 12–7.** Fireworks celebrating the joy of life.

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