Unit 1. The Role of the Speech-Language Pathologist in Dysphagia / Causes and Incidence: Stroke

Speech-language pathologists teach patients techniques for eating and swallowing. They also make recommendations about diets, specifying the consistency of foods that a patient can safely consume. e.g. thick/thin liquids, puree/mechanical soft etc.

Assessment of Swallowing and Remediation Techniques

Speech pathologists assess the swallowing abilities of patients through bedside evaluations and also through the use of radiographic techniques when necessary.

The speech pathologist should be aware that a swallowing patient's neurological status may change due to additional stroke(s). A patient who was previously able to swallow safely may begin to aspirate and require re-evaluation. Silent aspiration, or aspiration of food and liquids without coughing, can occur as a result of neurological damage. (If food or liquid enters the respiratory system of a normal, healthy individual, it induces coughing.) Silent aspiration can cause pneumonia, with a temperature spike being the first indication that food or liquids are entering the lungs.

Causes and Incidence of Dysphagia
(Logemann, 1989; Miller and Groher, 1984)

Patients may be treated for dysphagia after suffering a stroke or after traumatic brain injury. Other causes of dysphagia include brain tumor, spinal cord injuries, and progressive diseases like Parkinson's Disease and Multiple Sclerosis.

The exact incidence of dysphagia in the population of adults with neurogenic disorders is unknown. However, when the Rehabilitation Institute of Chicago surveyed their records, they found that about one third of such patients in their facility had some type of dysphagia (Cherney, 1994).

According to Cherney (1994), stroke was the most common cause of dysphagia, accounting for about half of the cases. Bilateral cortical stroke was the type of CVA most likely to cause dysphagia. Unilateral right hemisphere lesions cause more dysphagia than do unilateral left hemisphere lesions. This probably occurs because right hemisphere lesions often cause impulsivity, poor judgment, and reduced ability to follow compensatory strategies. Approximately one third of stroke patients have some type of dysphagia.

Traumatic Brain Injury (TBI) was the second most common cause of dysphagia, being the cause of about 20% of swallowing problems seen at that facility. Overall, about 25% of patients suffering from Traumatic Brain Injury have some type of dysphagia. As with CVA it seems reasonable to assume that those with right hemisphere lesions would have more problems.

Stroke tends to cause mild or moderate dysphagia. However I have worked with many CVAs who were quite severe. TBI more often causes severe dysphagia.

Stroke

A stroke or cerebral vascular accident is the temporary or permanent loss of functioning brain tissue due to an interruption in the blood supply. There are two types of stroke; those that result from a full or partial blockage of an artery and those caused by hemorrhages, or ruptures of intracranial blood
vessels.

Approximately 300,000 people have strokes in the U.S. each year. The following factors predispose an individual to stroke:

**Primary Hypertension** greatly increases a person's risk of suffering a CVA. Primary hypertension refers to elevated *diastolic blood pressure*. Diastolic blood pressure is measured when the heart is relaxing, while *systolic* blood pressure is measured when the heart muscle is contracting. Normal blood pressure is 120/80 (mm. of mercury Hg.), while upper limits of normal are 140/90 (Tabor's Cyclopedic Medical Dictionary). During strenuous exercise, a normal person's blood pressure will go up into the high range temporarily. In cases of primary hypertension, blood pressure remains in the high range regardless of activity level. Untreated hypertension increases the likelihood of stroke by pushing plaque up against arterial walls, causing *stenosis* which sometimes leads to *thrombosis*. The cause of primary hypertension is unknown, but is seems to be inherited.

Smoking and obesity further increase the risk of vascular problems in those who have hypertension. Mild hypertension seems to respond to exercise.

**High Cholesterol Levels** (hypercholesterolemia) also increase the risk of stroke. HDL or *High Density Lipoprotein* is the "good" cholesterol. LDL or *Low Density Lipoprotein* is the "bad" cholesterol. It is all right to have elevated levels of HDL, but having a high concentration of LDL in the blood is a health risk. Overall cholesterol levels should be under 200.

**Hypercholesterolemia** can cause stroke even in very young people.

There are two principle types of stroke, *ischemic* and *hemorrhagic*.

**Ischemic strokes**

The term "ischemia" refers to a lack of blood-borne oxygen. Ischemic strokes are more common than hemorrhagic strokes and may be caused by *stenosis*, *thrombosis*, or *thrombo-emboli*.

**Stenosis** is a general term that means "narrowing." In this case, it refers to the narrowing of an artery due to the build-up of plaque. As the artery is not completely blocked, some blood does pass through it. However, if at least 50% of normal blood pressure is not maintained, brain damage will occur.

**Thrombosis** refers to the total blockage of an artery due to plaque build-up.

**Thrombo-emboli** are pieces of plaque which break lose from thrombi and travel through the arterial system of the brain until they reach a narrowed area and lodge there, cutting off blood supply to brain tissue beyond that point. This sometimes occurs when a normally sedentary person engages in strenuous activity.

Two different type of ischemic events are warning signs that a stroke is likely to occur in the near future.

**TIA or Transient Ischemic Attack**

This is a transient disturbance of the blood supply to a localized part of the brain which produces a *temporary*, focal lesion. Unlike strokes, TIAs resolve in *spontaneous* and *complete* recovery within one day. TIAs typically last between two and fifteen minutes, although such an event could conceivably last as long as twenty-four hours. It is also possible to have a series of many brief TIAs during one day. For example, a patient might have 20 transient ischemic attacks within a twenty-four
Symptoms of TIAs mimic those of stroke. These attacks may cause temporary aphasia, numbness, and impairments of speaking, reading, and writing abilities. Dizziness and visual problems, such as blindness in part of the visual field, also occur. Sometimes TIA’s are very mild and involve only numbness in a limb, or loss of sight in one eye. Severe TIAs can not be differentiated from a stroke until recovery occurs. I had one patient whose first TIA occurred while he was eating lunch with his wife. She noticed that he had stopped eating and was just sitting there with food falling out of his mouth. He recovered completely within twelve hours.

**RIND or Reversible Ischemic Neurological Defect**

Some authorities (I use that term loosely) feel that the term RIND is no longer applicable. That it is really a stroke. A RIND is a lengthy TIA. The term RIND is usually applied to attacks that continue for more than twelve hours without interruption, although some RINDs endure for several days. As is the case with TIAs, patients make a complete recovery from RINDs. (There is some evidence that RINDs do cause some extremely subtle neurological damage, but these minor changes are nothing like the disabilities seen after an actual stroke.)

Sometimes, events that last for twenty-four hours are called TIAs rather than RINDs, so there is some inconsistency in the application of this terminology.

Strokes, TIAs and RINDs are most likely to occur in the morning, when blood pressure is at its lowest. When a person gets out of bed, the change in activity level causes a change in blood pressure.
UNIT 2

Causes and Incidence: Traumatic Brain Injury

I. Traumatic Brain Injury

According to Adamovich, Henderson, and Auerbach, (1984) as many as 400,000 head injuries occur in the United States each year. The severity of head injury has generally been classified as mild, moderate and severe. Mild head injury has been defined as concussion, while severe head injury means being in a coma for at least six hours. There does not appear to be a consensus for a definition of moderate head injury. Jennett and Teasdale developed the Glasgow Coma Scale in 1974 (Bach-y-Rita, 1989). It is a numerical scale that quantifies level of consciousness in response to three categories: response to pain, ability to open eyes, and ability to speak.

II. Types of Brain Injury (Urosevich, 1984)

A. Coup-contrecoup

B. Coup can occur due to hitting one's head on a hard surface after a fall. There is a bruising of the brain and a laceration of blood vessels immediately below the injury site. Contre-coup injury occurs as the brain hits against the inside of the head just opposite the injury site. More serious hemorrhaging occurs when there are bony prominence where the brain comes in contact with the inside of the skull. Direct impact injury typically results in focal lesions.

C. Acceleration-deceleration

Motor vehicle accidents are the primary cause of this type of TBI. When there is a sudden stoppage of the vehicle the passenger's head is thrown forward violently, then thrown backward. The brain rebounds against the bony prominence of the skull, and may go through several oscillations. This often results in severe hemorrhaging. In addition, shearing injuries occur as the head twists dragging the brain along. This can result in diffuse white matter (axonal) injury.

III. Typical Lesions Occurring with TBI (Pires, 1984)

Many of the following lesions result in edema of brain tissue. It is extremely important that intra cranial pressure is managed. Your patients may be in a barbiturate induced coma, have their heads elevated, be hyperventilated, or heavily medicated (Bach-y-Rita, 1989).

A. Epidural Hematoma

Bleeding occurs above the dura mater, usually from meningeal arteries. The blood quickly accumulates,
creating a space between the skull and the dura. The typical course for the patient is instantaneous loss of consciousness followed by a short period of lucidity, then a lapse into unconsciousness. The hematoma frequently exerts pressure on cranial nerves which can result in ipsilateral lower motor neuron paralysis. Because cranial nerves innervate muscles of swallowing and speech as well as those for eye movement, the paralysis can result in unilateral pupillary dilation and paralysis of the jaw, face, throat, larynx, or tongue.

B. **Subdural Hematoma**

Bleeding occurs into the potential space below the dura mater and above the arachnoid mater. It is typically venous and found above the frontal and temporal cerebral lobes. Since the primary and association motor cortices as well as pre-frontal cortex are in the frontal lobes damage there can result in swallowing, speech, language or cognitive problems. It is common for the appearance of symptoms to be delayed for several days or a week or more. A coma or less severe symptoms may develop. The latter include: seizures, unilateral or bilateral weakness or paralysis.

C. **Intracerebral Hematoma**

Bleeding is within the cerebrum and often includes sub cortical structures. It most often occurs because of penetrating head wounds. The frontal and temporal lobes are frequently involved. Of course this type of injury could occur anywhere in the brain. Dysphagia, aphasia, apraxia, dysarthria, paralysis, as well as visual and other problems can occur.

D. **Tentorial Herniation**

Edema of the brain which occurs due to TBI, forces neural tissue through the tentorial notch between the cerebrum and cerebellum. This results in the squeezing of the brain stem (mid brain, pons, medulla). The cranial nerves are affected resulting in dysphagia, and speech problems, or even in death, as the medulla is important to respiration and circulation. Some of the initial symptoms of tentorial herniation are weakness, paralysis (often on one side), and visual disturbances (pupillary dilation, double vision). Respiration may be affected, the heart may be quite slow (bradycardia), or decortication (removal of cortical tissue) may be necessary.

Study Questions

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Other courses in the Neuroscience on the Web series:

SPPA 362 Syllabus (Neuroanatomy) | SPPA 336 Syllabus (Neuropathologies of Language and Cognition)
Unit 3. Anatomy of the Swallow

Basic anatomical structures for swallowing and speech

**The Oral Cavity**

The Oral Cavity is defined as the space between the lips and pharynx.

The muscles involved in chewing are all innervated by the trigeminal nerve. They include: the temporalis which elevates, retracts, and assists in closing the mandible, the masseter which elevates and closes the mandible, the medial which also elevates the mandible and aids in its closure, the lateral pterygoid which depresses, opens, and protrudes the mandible, as well as moving it laterally.

Other muscles involved in chewing are the obicularis oris and the zygomaticus. Both are lip muscles. The buccinator holds food in contact with the teeth. All three are innervated by the facial nerve (CN. VII).

Five muscles control the movements of the velum. The palatoglossal and the levator veli palatini both raise the velum. They are innervated by the vagus nerve (CN. X). The tensor veli palatini tenses the velum. It receives innervation from the trigeminal (CN. V).

The palatopharyngus depresses the velum and constricts the pharynx. It is innervated by the spinal accessory (CN. XI). The muscularis uvula shortens the velum. It is also innervated by the spinal accessory.

Poor velopharyngeal closure will affect speech but is not a matter of great concern in regard to swallowing. Patients may be concerned about it and believe that it is very important. While the entrance of food into the nasopharynx may be unpleasant, it is certainly not life-threatening.

**The Pharynx**

The hypopharynx is the lower portion of the pharynx. It is also known as the laryngopharynx.

There are three pharyngeal recesses: Food boluses can lodge in these recesses.

The **vallecula** is the space or depression between the base of the tongue and the epiglottis.

The **two pyriform sinuses** are located in the pharynx, beside the larynx. They are formed by the shape of muscle attachments to the pharyngeal walls.

The **superior, middle, and inferior pharyngeal constrictor muscles** make up the external **circular layer** of the pharynx.

The **stylopharyngus m.** and the **salpingopharyngus m.** make up the internal longitudinal layer of the pharynx.
The pharyngeal constrictor muscles help move food down toward the esophagus via a **stripping action**. (This process should not be confused with **peristalsis** which is the wave-like motions of muscles that occur in the esophagus. In some of the literature, the action of the pharyngeal constrictor muscles is mistakenly called peristalsis.)

The latest research on swallowing suggests that the action of the pharyngeal constrictor muscles is not the most critical factor in the movement of food down the pharynx. It seems that the **plunger action** of the tongue, or the **tongue driving force**, plays a major role in this process.

The **cricopharyngus m.** or **pharyngeal-esophageal (P.E)** segment separates the pharynx from the esophagus. At the end of the pharyngeal stage of the swallow, it must relax to allow the bolus to enter the esophagus. (It is normally closed to prevent the reflux of food and to keep air out of the digestive system.) If the P.E. segment does not relax, food will build up in the pharynx and eventually spill over the top of the larynx into the airway. The cricopharyngus is innervated by the vagus (CN. X).

Problems with the P.E. segment are rare. According to Logemann (1983, 1989), only 5% of dysphagias are caused by malfunction of the cricopharyngus muscle. In the past, physicians frequently treated all types of swallowing problems by cutting the P.E. segment. This procedure is called **myotomy**.

**The Neuroanatomy of Swallowing**


**Definition**

In the past, the swallow was classified as a reflex. Now most sources agree that swallowing is a pattern-elicited response.

The gag reflex, in contrast, is a good example of a true reflex. It is "triggered" whenever a noxious substance touches the back of the tongue, back of the pharynx, or soft palate. The swallow response, on the other hand, cannot be initiated by touching any particular area in the oral cavity. The gag reflex and the swallow response also differ in terms of neurological control. The gag reflex is completely controlled by the brain stem. The swallow, on the other hand, is only partially controlled by the brain stem. It also receives cortical input, and input from muscle spindles, including feedback from tongue movements.

(It is important to note that the gag reflex and the swallow response are not related. In the past, many physicians would determine feeding status based on the presence or absence of a patient's gag. Actually, the presence or absence of a gag reflex does not predict the status of the swallow response.)

**Neurological Control**

Both sensory and motor information are necessary for the initiation of the swallow response; swallowing is dependent on both sensory and motor control or on information from both afferent and efferent systems. Sensory feedback plays a more important role in swallowing than it does in speech. Sensory input involved in the initiation in the swallow comes from the trigeminal, facial, and glossopharyngeal nerves. Information about motor movement is received from the muscle spindles in the tongue via the hypoglossal nerve.

Sensory and motor information from these sources is carried to the swallowing center, which is believed to be located in the medulla, within the nuclei of the reticular formation; specifically the nucleus ambiguous. When the swallow response is initiated, this center causes messages to be sent to the glossopharyngeal, the vagus, and the hypoglossal nerves. The glossopharyngeal is considered
the major nerve for the swallowing center.

Six of the cranial nerves provide the innervation for both swallowing and speech:

1. CN. V The Trigeminal Nerve
2. CN. VII The Facial Nerve
3. CN. IX The Glossopharyngeal Nerve
4. CN. X The Vagus Nerve
5. CN. XI The Spinal Accessory Nerve
6. CN. XII The Hypoglossal Nerve

**The Trigeminal Nerve (CN. V):**

**Motor Component**

The efferent portion of the trigeminal nerve innervates the muscles involved in chewing. These include the temporalis, the masseter, the medial, and the lateral pterygoid.

The trigeminal nerve also innervates the tensor veli palatine muscle, which tenses the velum.

In addition, the trigeminal assists the glossopharyngeal nerve in raising the larynx and pulling it forward during the laryngeal substage of the pharyngeal swallow.

**Sensory Components**

The trigeminal nerve carries feedback about all kinds of sensation, except taste, from the anterior 2/3 of the tongue.

CN. V also carries sensory information from the face, mouth and mandible.

**The Facial Nerve (CN. VII)**

**Motor Components**

The facial innervates the lip muscles including the orbicularis oris and the zygomaticus. The muscles must contract during the oral preparatory and oral transport stages of the swallow to prevent food from dribbling out of the mouth.

The facial also innervates the buccinator muscles of the cheeks. These must remain tense during the oral component of the swallowing process to prevent the pocketing of food between the teeth and the cheeks.

**Sensory Component**

The facial carries information about taste from the anterior 2/3 of the tongue.

**The Glossopharyngeal Nerve (CN. IX)**

**Motor Components**

It innervates the 3 salivary glands in the mouth. The saliva from these glands mixes with the chewed up food to form a bolus.

CN. IX has motor, sensory, and autonomic nervous system nerve fibers. It, along with the vagus (CN.
X), provides some innervation to the upper pharyngeal constrictor muscles (Zemlin, 1997).

It innervates the **stypopharyngeus** muscle which elevates the larynx and pulls it forward during the pharyngeal stage of the swallow. This action also aids in the relaxation and opening of the cricopharyngeus muscle.

**Sensory components**

The glossopharyngeal nerve mediates all sensation, including taste, from the posterior 1/3 of the tongue.

CN. IX also carries sensation from the velum and the superior portion of the pharynx. A lesion may have impaired the gag reflex unilaterally (Zemlin, 1997).

**The Vagus Nerve (CN. X)**

**Motor Components**

The vagus is responsible for raising the velum as it innervates the glossopalatine and the levator veli palatine muscles.

The vagus along with CN. IX innervates the pharyngeal constrictor muscles.

The vagus along with CN. XI innervates the intrinsic musculature of the larynx. It is responsible for vocal fold adduction during the swallow.

The vagus also innervates the cricopharyngeus muscle.

The vagus controls the muscles involved in the esophageal stage of the swallow as well as those that control respiration. (This is the only cranial nerve that influences structures inferior to the neck.)

**Sensory Component**

The vagus carries sensory information from the velum and posterior and inferior portions of the pharynx.

The vagus also mediates sensation in the larynx.

**The Spinal Accessory Nerve (CN. XI)**

**Motor Components**

CN. XI innervates the palatopharyngeus muscle which depresses the velum and constricts the pharynx.

It also innervates the muscularis uvula which tenses the velum. It, along with CN.X, innervates the levator veli palatini.

(CN. XI is strictly a motor nerve.)

**The Hypoglossal Nerve (CN. XII)**

**Motor Components**

The hypoglossal innervates all extrinsic and intrinsic tongue muscles
(It is strictly a motor nerve.)
Unit 4. Physiology of the Swallow

Stages of the Swallow

The oral preparatory phase

This part of the swallow is voluntary. It is a mechanical phase that can be by-passed by dropping liquid or food into the back of the throat.

In this stage, the food is chewed into smaller pieces and tasted. It is also mixed with saliva from three pairs of salivary glands, which are innervated by the glossopharyngeal nerve. The food and saliva form a bolus of material.

The bolus is kept in the front of the mouth, against the hard palate by the tongue. The front of the tongue is elevated with its tip on the alveolar ridge. The back of the tongue is elevated and the soft palate is pulled anteriorly against it to keep the food in the oral cavity (the airway is open and nasal breathing continues during this phase). Labial seal is maintained to prevent food from leaking out of the mouth. Buccal muscles are tense. This prevents pocketing of food. Duration of the oral-preparatory stage is variable (Logemann, 1983, 1997).

The oral transport stage

This stage of the swallow is also voluntary. It starts with the jaws and lips closed, and the tongue tip on the alveolar ridge. The pattern-elicited response is initiated at the end of this phase.

Inspiration is reflexively inhibited at the beginning of this stage. The food is moved to the back of the mouth by the tongue via an anterior to posterior rolling motion. The anterior portion of the tongue is retracted and depressed while the posterior portion is retracted and elevated against the hard palate. When the bolus passes the anterior faucial pillars.touches the posterior wall of the pharynx, the oral stage ends and the pharyngeal stage begins as the tongue driving force or the tongue's plunger action, forces the bolus into the pharynx. Logemann (1997) describes the "pharyngeal tongue" which extends from the velum to the hyoid bone and valleculae. The "oral tongue" which extends from the tip to the back, adjacent to the velum, functions during the oral stage of the swallow while the "pharyngeal tongue" functions during the pharyngeal stage. The oral transport stage lasts one second (Logemann, 1989, 1998; Dobie, 1978).

The pharyngeal stage

The pharyngeal phase of the swallow is involuntary. It is the most critical stage of the swallow; airway closure must occur to prevent the bolus from entering the respiratory system. A number of things occur almost simultaneously.

1. Sensory information from receptors in the back of the mouth and in the pharynx goes to the swallowing center in the medulla via CN. IX. The palatopharyngeal folds pull together medially to form a slit in the upper pharynx. The bolus passes through this slit.
2. The velum is raised, primarily by the levator and tensor veli palatini muscles. This prevents the
entry of food into the nasopharynx. The narrowing of the upper pharynx due to the contraction of superior pharyngeal constrictor muscle helps to close the velopharyngeal port.

3. The tongue is retracted, preventing the food from re-entering the mouth.

**The laryngeal substage**

Three actions occur simultaneously to protect the airway. (Obviously, inspiration is inhibited during the pharyngeal stage of the swallow.)

1. The larynx and the hyoid bone are pulled both upward and forward. This movement enlarges the pharynx. It also creates a vacuum in the hypopharynx, pulling the bolus downward. Finally, it contributes to the relaxation of the cricopharyngeous muscle (Dobie, 1978; Logemann, 1983, 1989, 1997).

2. The true and false vocal folds adduct. (Closure begins at the level of the true vocal folds and progresses up to the false vocal folds and then to the ari-epiglottic folds.)

3. The epiglottis drops down over the top of the larynx, protecting the airway and diverting the bolus into the pyriform sinuses. The bolus passes down on both sides of the epiglottis. If the bolus is liquid, the epiglottis acts as a ledge to slow its movement through the pharynx, giving the vocal folds time to adduct and the larynx time to elevate. (Nevertheless, the action of the epiglottis is the least important of these three movements.)

Three factors cause food to move down the pharynx during the rest of the pharyngeal stage (Cherney, et al., 1994; Logemann, 1983, 1989, 1997):

1. The tongue driving force using the "pharyngeal tongue"

2. The stripping action of the pharyngeal constrictors

3. The presence of negative pressure in the laryngopharynx

It is believed by some that the tongue driving force (TDF) is the most important of these factors. This generates pressure in the upper pharynx.

The pharyngeal stage ends when the cricopharyngus muscle relaxes, allowing the bolus to enter the esophagus. It is believed that the following three factors affect the opening of the p.e. segment, although the process is not currently well-understood:

1. Innervation by the vagus nerve

2. The timing of the stripping action in the pharynx may somehow trigger the relaxation of the p.e. segment.

3. The elevation of the larynx may pull the muscle upward, causing it to open by stretching it and therefore causing it to relax.

**The esophageal stage**

In this phase, which is of course involuntary, the bolus is moved down the esophagus via peristaltic wave motion with some help from gravity.

At the beginning of the phase, the larynx lowers, returning to its normal position. The cricopharyngeus muscle contracts to prevent reflux and respiration resumes.

This stage normally lasts between three and twenty seconds, but in elderly persons peristalsis is slower (Logemann, 1989, 1997; Dobie, 1978).

Esophageal problems can cause the reflux of food back into the pharynx, leading to aspiration. Speech pathologists do not treat esophageal problems, but should be aware of them, and be able to differentiate them from problems that are within their scope of practice.
Note that 90% of the swallow occurs during expiration; an apneal pause between 1 and 3.5 seconds in duration occurs during the oral and pharyngeal stages (Logemann, 1989; 1997).

Aspiration occurs whenever food enters the airway below the true vocal folds. Aspiration can occur before, during, or after the swallow.

Aspiration before the swallow

Aspiration occurs before the swallow in the case of a delayed or absent swallow initiation. It may also be the result of poor tongue control, which allows food to trickle into the pharynx while the patient is still chewing. Apparently, a "neurological override" exists which prevents the initiation of the swallow while one is still chewing (Logemann, 1983, 1989, 1997).

Aspiration during the swallow

Aspiration occurs during the swallow when the vocal folds fail to adduct or the larynx fails to elevate. (Remember that this type of dysphagia is uncommon. Only 5% of dysphagias involve problems with airway closure).

Aspiration after the swallow

Aspiration can occur after the swallow in several different circumstances:

The patient may pocket food in the oral cavity. Later, when he or she lies down to sleep, the food will fall down into the airway.

Food may get stuck in the pharyngeal recesses. This happens to everyone, but someone with a normal system would realize that the food was there and swallow again. A CVA or TBI patient may have a sensory impairment and allow the food to drop into the larynx.

Due to reduced laryngeal elevation, food may remain on top of the larynx (Logemann, 1989).

Types of Dysphagia

The most common type of dysphagia is delayed/absent initiation of the pharyngeal stage of the swallow. 80% of CVA patients who have dysphagia have this type of problem (Logemann, 1989, 1997).

It is common for patients with this type of disorder to keep trying to push the bolus into the pharynx with the tongue. Eventually, they will succeed. Where the food goes when this happens depends on three things: the posture of the patient, the consistency of the food and size of the bolus. Smaller amounts of thick substances will generally lodge in the pharyngeal recesses rather than going directly down the airway.

As a patient moves the tongue and tries to push the bolus into the pharynx, the movements of the tongue and the hyoid bone look a lot like a swallow. It will be difficult to tell whether or not the patient is aspirating. A number of patients aspirate without coughing. Also, food may be lodging in the pharyngeal recesses, which will hold several teaspoons of material, before being aspirated.

Disorders of the pharyngeal stage of the swallow are the most prevalent type of dysphagia among the CVA population; over 90% have pharyngeal stage problems. Reduced tongue driving force or poor pharyngeal
stripping action is an especially common problem among those who have had CVAs. This causes food residue to accumulate in the valleculae and may lead to aspiration after the swallow. Pharyngeal stripping action is usually the last part of the swallowing process to recover. No specific site of lesion is associated with this problem.

The majority of patients who are NPO have pharyngeal stage problems.

Fifty percent of those who have pharyngeal stage problems also have oral stage problems.

Half of CVA patients with dysphagia have problems that affect the **oral stage of the swallow**. Fifty percent of those have reduced or abnormal tongue movements that affect the initiation of the swallow. Typically, tongue control problems are not sufficiently severe to cause aspiration. No specific site of lesion is associated with tongue movement problems.

Only 5% of CVA patients have problems with vocal fold adduction. This type of difficulty only occurs with **brain stem lesions**. There are generally no problems with airway closure following a cortical stroke, unless there are bi-lateral upper motor neuron lesions. This is principally due to the fact that upper motor neurons bi-laterally innervate most of the cranial nerves.

Only 5% of CVA patients have swallowing problems caused by the **failure of the cricopharyngeus muscle** (p.e. segment) to relax. If the p.e. segment does fail to relax, food will build up in the pharynx and may be aspirated. In this case food residue will be accumulated in the **pyriform sinuses**, or in cases of severe problems, throughout the lower portion of the pharynx, and may cause aspiration after the swallow.

*** Typically, each patient will have more than one type of swallowing problem.

**Site of lesion**

There is currently enough evidence to specify the specific type of swallowing problem associated with particular sites of lesion caused by stroke.

**Brain stem** stroke typically causes the most severe cases of dysphagia. Damage to the medulla is particularly devastating as is to be expected since the "swallowing center" and the nuclei of most of the cranial nerves involved in swallowing are located there. As the cranial nerves are lower motor neurons, they form the final common pathway for all motor impulses traveling from the brain to the muscles involved in deglutition (Logemann, 1983, 1989).

Patients with unilateral medullary lesions may have functional or even normal oral control. However, they usually have significant problems with the pharyngeal stage of the swallow (the cranial nerves that innervate the pharynx and larynx originate in the medulla). They may have one or more of the following problems: extreme delay in the initiation of the swallow response (10-15 seconds) and reduction in both elevation and anterior movement of the larynx. This in turn may lead to reduced opening of the criopharyngeus muscle. They may also have unilateral pharyngeal weakness and unilateral vocal fold paralysis. In some cases, patients will not recover their swallow for 4 to 6 months or ever.

**Subcortical stroke** can affect both sensory and motor pathways. It may cause problems in both the oral and pharyngeal stages of the swallow, including:

- Mild delays in oral transit time (3-5 seconds)
- Mild delays in initiation of the pharyngeal swallow (3-5 seconds)
- Impairments in the timing of the neuromuscular components of the pharyngeal swallow. (Logemann, 1989).

**Unilateral Left Hemisphere Stroke (Cortical)**

A lesion in this area may cause apraxia of the swallow. The tongue may not respond to food or may make searching movements prior to transporting the bolus. Patients with this kind of problem may have more
success with oral feeding if they are allowed to feed themselves. This makes the swallow more “automatic.” Other problems that occur with this type of lesion include:

- Mild delays in oral transit (3-5 seconds)
- Mild delays in the initiation of the pharyngeal swallow (2-3 seconds)
- The pharyngeal stage should be normal once it is initiated since it does not require a lot of cortical input.

**Unilateral Right Hemisphere Stroke (Cortical)**

The following problems may be experienced:

- Mild oral transit delays (2-3 seconds)
- Slightly longer pharyngeal delays (3-5 seconds)
- Delayed laryngeal elevation

The dysphagias produced by right hemisphere lesions are usually more severe than those resulting from left hemisphere damage. Patients with right hemisphere damage tend to have attentional problems and exhibit poor judgment including impulsivity. These characteristics reduce their ability to use compensatory strategies for safe swallowing.

**Multiple Strokes** often cause significant swallowing problems that affect both the oral and the pharyngeal stages. Also, cognitive ability may be impaired, reducing the patient's ability to use compensatory strategies. According to Logemann (1989), the swallow is never quite the same after a stroke even when a patient is able to return to a regular diet. When a patient has another stroke later, the already compromised mechanism is further damaged.

In **recovery of the swallow**, tongue movement is generally the first part of the process to improve, followed by the initiation of the swallow. Pharyngeal stripping action is usually the last part of the process to improve in recovery.

Recovery is most rapid in the first 3 or 4 weeks after a stroke. Therefore, an SLP should always re-evaluate an NPO patient about one month after a stroke.

Generally, if the swallow is going to recover it will do so within 6 or 7 weeks after a stroke.
Bed-side evaluation

Do an oral peripheral examination. Look for strength, range of motion, and symmetry of structures. Also note any drooling as well as the patient's dentition.

1. If the patient can phonate, note the strength and quality of the voice. If the voice is hoarse, this suggests that the vocal folds are not adducting completely. If the voice sounds "wet" or "gurgly," saliva may be pooling in the larynx. (A patient may aspirate his/her own secretions.) How long can the patient sustain phonation? Also, note the patient's articulation. If it is good, this bodes well for swallowing.

2. Listen to the patient's voluntary cough. If it is weak, this is another indicator of poor vocal fold adduction.

3. Examine chewing. Use a roll of gauze that has been dipped in a good-tasting liquid. Check chewing on both sides of the mouth.

4. Check the palatal reflex by stimulating the anterior surface of the velum. It should elevate and retract, closing against the pharyngeal wall.

5. Check tactile sensation in the oral cavity. (Food should be presented to areas where sensation is most intact.)

6. Check the dry swallow. This is a test for apraxia of the swallow. You can also note laryngeal excursion by placing your fingers on the larynx and hyoid bone.

7. Be careful when you check the gag reflex. Although it does not predict the status of the swallow a reduced or absent gag does indicate neurological dysfunction. If the patient vomits, he/she may aspirate the regurgitated food.

8. Note attentional abilities. How alert are they? Can they use compensatory strategies? Can they cooperate with a radiographic exam?

Radiographic studies

The use of radiographic techniques provides a direct view of events in the oral cavity and the pharynx. The procedure used to image the swallowing process is a modified barium swallow, not a true barium swallow. A true barium swallow is used to view the esophagus, which is collapsed unless a bolus is moving through it. It is also used to view the stomach. The patient drinks a barium milkshake which coats the esophagus and the stomach lining. (Barium is not radioactive. It is a contrast material.

In a modified barium swallowing study, the patient consumes foods of varying consistencies that have been coated with barium. Usually, the patient swallows a liquid of water-like consistency. This kind of material may be aspirated if there are swallowing problems, but it will not block the airway. The patient may drink straight from a cup. Drinking from a straw is the easier of the two. If it can be
done safely, the patient also swallows food with a paste-like consistency and a cookie or bread during the radiographic study. Logemann (1989) particularly likes the Lorna Doone cookies.

It is a good idea to experiment with different food consistencies and with compensatory postures during the radiographic studies. This allows you to see exactly what is happening when each one is used and helps you to decide what will help the patient the most. If the patient is cognitively intact, you can also try out some airway closure techniques like the supraglottic swallow during the study. Logemann (1989) recommends checking for the "dry swallow" during radiographic studies. When there is still food left in the pharynx after a swallow, does the patient realize this and swallow again?

Sometimes it is helpful to have family members watch the study. This will help them to understand why the patient is NPO or on a special diet and they will not be inclined to bring the patient food that violates his dietary restrictions. Many family members feel that water is harmless. This is not so. A good number of patients aspirate thin liquids (Cherney, 1994; Logemann, 1989).

Other procedures include videonasendoscopy (fiberoptics), manometry (measures pressure), ultrasound (imaging technique), ultrafast computerized tomography, scintigraphy (radioactive liquid, galium, is swallowed), and electromyography (examines muscle tissue) (Logemann, 1997).
Unit 7. The Remediation of Dysphagia

*Therapy Strategies*

These techniques are designed to actually change the physiology of the swallow.

**Icing**

This is a technique developed by occupational therapists. It may or may not be effective but is currently in use. The external surface of the lower cheeks and the mouth are typically iced and the velum and uvula may also be treated.

Icing some areas could possibly have dangerous consequences for the elderly, the frail, or those with heart problems. (Icing these areas would not affect a young healthy person.)

Icing above the mouth can cause a sympathetic nervous system response which increases heart rate. This might cause plaque to break lose from a thrombus and lead to another stroke or to a heart attack.

Icing behind the ear can lower blood pressure to dangerous levels in some patients.

Icing near the pinna can cause irregular heart beat and respiratory problems.

**Thermal Stimulation (Logemann, 1989)**

Evidence regarding the efficacy of this procedure is mixed, but it is commonly used. Logemann (1989) has promoted the use of this technique.

Thermal stimulation involves tapping or rubbing the patient's anterior faucial pillar with an iced dental mirror. In each treatment “set” the tapping/rubbing is done about five times. As immediately as possible after a set is completed the patient is instructed to swallow and may be given a small amount of liquid through a straw, even carbonated. The extra stimulation provided by the iced mirror is supposed to somehow alert the nervous system, allowing the swallow response to occur more rapidly.

Logemann (1989) recommends doing thermal stimulation three times per day. She suggests stimulating only the anterior faucial pillar on the patient's good side. I like to do both sides and only when the patient is taking a break in between doing oral-motor exercises. I have grave doubts about the efficacy of thermal stimulation. The research is quite conflicting and from my own experience after working with swallowing patients for many years I am quite unconvinced as to its efficacy.

**Sensory stimulation (for apraxia of swallowing or reduced sensation) (Logemann, 1989)**

It may involve presenting a warm or cold bolus, presenting foods with strong tastes or textures, or pressing the spoon on the tongue when food is presented.

For patients with apraxia of the swallow, it may be best to let them feed themselves, allowing the swallow to be more automatic.

**Suck-swallow technique**
The patient produces an exaggerated suck with the lips closed followed by an exaggerated vertical back-tongue motion prior to swallowing attempts. (Have the patient suck on a popsicle stick.)

The sucking action pulls saliva to the back of the mouth, and this seems to help trigger the swallow more rapidly. So, this technique is also based on the idea that increased oral sensation will help to trigger the swallow.

**Chewing**

For some patients, this provides the extra oral sensation necessary to trigger the swallow. If this technique is used, the patient must also chew liquids prior to swallowing them.

**Motor exercises**

Exercises can be done to improve the range of motion of the lips, tongue, and jaw, to improve coordination, to improve vocal fold adduction, laryngeal elevation, or tongue base retraction.

Range of motion exercises involve moving target structures as far as possible from rest position, holding them at the most distal point for a few seconds and then relaxing.

Resistance exercises involve moving against pressure. For the tongue, use a tongue blade. Falsetto exercises will improve laryngeal elevation.

**Posture to facilitate the swallow**

(Logemann, 1989)

Specific postures are used to compensate for particular types of dysphagia by changing the way that the food moves through the pharynx. It is a good idea to have the patient try using these postures during the radiographic study; this way you can get an idea of how well they will really work.

If the patient's problem is **delayed initiation of the swallow**, have them tuck their chin while they eat. This **head-down** posture moves the tongue forward, enlarging the vallecula. The vallecula can then contain the bolus a little longer than usual, allowing more time for the larynx to elevate and the vocal folds to adduct. It also narrows the airway and puts the epiglottis in a more over-hanging position.

If the patient has **poor tongue control**, have him/her swallow in a **head-back** position, allowing for more drainage. When a patient swallows in this position, it may be dangerous to give him/her thin liquids. Also, if the patient has both poor tongue control and problems with airway closure, he/she could swallow in this position using the **supraglottic swallow**. (Remember that only patients with fairly intact cognition can effectively use the supraglottic swallow.)

In the case of unilateral paralysis of the pharynx, the patient should **turn** his head **toward the paralyzed side** before swallowing. This closes the pyriform sinus on the bad side and keeps food on the functioning side of the pharynx.

If there is a **unilateral paralysis** of both the oral cavity and the pharynx, the patient should swallow while **tilting** the head **toward the better side**. This technique prevents pocketing and also sends the bolus down the functioning side of the pharynx.

Note that the head should only be turned toward the bad side in the case of unilateral paralysis of the pharynx alone. If the oral cavity is also affected, use the tilting technique. (Tilting could be used for pharyngeal problems alone if turning is too difficult for the patient.)

If the patient is pocketing food in the oral cavity, it may be necessary to teach him/her to sweep the buccal cavity with a finger.

**Food consistencies to increase safety while swallowing**

(Logemann, 1989)
Again, there is no "typical" dysphagia diet. The consistency of food should be chosen based on the specific nature of the problem.

In the case of **reduced stripping action**, patients will do better with liquids rather than with thicker foods. Liquid will move more easily through the pharynx.

If the problem is **reduced tongue control**, use thickened liquids. They should be sufficiently viscous to prevent splashing.

If the patient has problems with **airway closure**, use thickened liquids (you may use carbonated beverages during trial feedings only). They will travel more slowly, allowing more time for laryngeal elevation and closure.

In the case of problems with the **cricopharyngus**, thinner consistencies are better. Thin liquids can drain into the esophagus through even a small opening in the p.e. segment.

In cases of **reduced laryngeal elevation**, use thin liquids because of crico pharyngeal problems. Remember, laryngeal elevation stretches the cricopharyngeous resulting in relaxation of the muscle.

In addition to changing the type of food that the patient eats, you can also ask them to regulate the size of the bolus that they try to swallow. Have them take small bites.

**Techniques for protecting the airway (Logemann, 1989)**

**The supraglottic swallow**

The patient is told to take a breath and hold it while swallowing and then coughs after the swallow. This results in the voluntary closure of the vocal folds before, during and after the swallow.

From my experience with swallowing patients this technique is rarely effective. In the first place, it is seldom necessary as only 5% of dysphagia patients have problems with airway closure. Of this group, many will have cognitive impairments that prevent them from using this strategy. In addition, many elderly patients are unable to hold their breath and open their mouths at the same time. (It is a better strategy to have put food in their mouths first and have them start holding their breath only when they are ready to swallow).

**The supra supra-glottic swallow**

This technique closes the entrance to the airway at the level of the arytenoid cartilages. The patient follows the same procedure as with the supra-glottic swallow, but "bears down while holding his breath."

**The Mendelsohn Maneuver**

This technique helps the patient gain some voluntary control over the opening and closing of the p.e. segment.

The patient is told to pay attention to the way the thyroid cartilage (Adam's apple) goes up and down during swallowing. Then he learns to use muscles to keep the larynx elevated for several seconds after the swallow. This should facilitate the opening of the cricopharyngus muscle.

**The Effortful Swallow**

This increases the tongue driving force by causing exaggerated retraction of the tongue. This helps to get food past the valleculae.
The patient is directed to squeeze hard with his throat and neck muscles during the swallow.

*** The above techniques are most often used with patients who have had brain stem lesions and so have severe dysphagia, but still have good cognitive ability.

**NG tubes and swallowing**

According to Logemann (1989), it's not necessary to wait until tubes are removed to begin therapy. Others, including the instructor believe that NG tubes can affect swallowing. It may be difficult for the patient to realize that food is stuck in the pharynx as an NG tube touches the pharynx all the way down through the esophagus.

**Other Personnel Involved in the Treatment of Dysphagia**

- Primary Care Physician
- Radiologist
- Physical Therapist
- Occupational Therapist
- Dietician
- Gastroenterologist

**Patient/family counseling and followup**

Assessment may involve the SLP, the gastroenterologist (manometry), the otolaryngologist (endoscopy), and the radiologist (videofluoroscopy). Non oral feeding may be necessary. Rehabilitation may be required to bring the patient back to full oral intake, and nutritional support should be provided to facilitate recovery. Physical and occupational therapy will provide assistive devices for feeding, as well as for seating and positioning.

Patient and family counseling and follow-up will inform the family about the details of the problem. Family members and the patient, if feasible, will be taught how to do thermal stimulation, oral motor exercises, etc.
Before we get into apraxia of speech (AOS) you will need to review your neuroanatomy notes on the motor system. The motor system is present at all levels of the nervous system. It includes “afferent connections of the cortex, especially the frontal lobes; the basal ganglia, cerebellum, and related CNS pathways; descending pathways to motor nuclei of cranial and spinal nerves; efferent fibers within cranial and spinal nerves, and striated muscle” (Duffy, 1995, p. 21). In addition, you must remember that the planning/programming associated with motor speech is thought by many to occur in Broca's area on the third frontal convolution, although a recent study has found that the Island of Reil was involved in 25 stroke patients who had a disorder involving the motor planning of speech (muscle movements) (Dronkers, 1996). Apraxia of speech (AOS) is believed by most researchers to result from a Broca's area lesion. According to Duffy (1995), who is with the Mayo Clinic, AOS as the primary speech pathology accounted for 9% of patients with motor speech disorders seen at the clinic between 1987 and 1990. In addition it is seen as a secondary diagnosis with aphasias and other neurological disorders. Remember, it is not a neuromuscular disorder. It is a planning/programming problem. According to Duffy, 1995, vascular lesions are the most prevalent cause of AOS. In addition he states that most cerebral vascular accidents (cva's) occur in the middle cerebral artery of the left hemisphere.

I like the neurological model of communication developed by Meitus and Weinberg, 1983, It enables one to clearly see the steps involved in oral communication from the development of the concept to the production of speech.

Meitus & Weinberg (1983, p.227) have four ordered steps or levels in their neurological model of oral communication. They are:

- **Ideation** - when the concepts we wish to express are generated
- **Symbolization** - when these concepts are put into a symbolic system congruent with the rules of the speaker's language;
- **Translation** - when these linguistic units or symbols are translated into neuromotor commands that result in the innervation of the motor nerves.
- **Execution** - the actual movements of the speech mechanism.

According to Meitus & Weinberg (1983), impairments at each of these levels will result in a different disorder:

- Impairment at the ideation level will result in mental-verbal dysfunction (or mental confusion, dementia, confused language)
- Impairment at the level of symbolization will result in aphasia
- Impairment at the level of translation will result in apraxia of speech
- Impairment at the level of execution will result in dysarthria.

What Is Apraxia of Speech?
According to Halperin (1986 in Chapey, 1986, p.422), "apraxia of speech is an articulation disorder that results from impairment due to brain damage, of the capacity to order the positioning of speech musculature and the sequencing of muscle movements for volitional production of phonemes and sequences of phonemes; but it is not accompanied by significant weakness, slowness, or incoordination of these same muscles in reflex and automatic acts." There is a great difference between motor speech programming and the neuromuscular execution of speech.

With an acquired motor disorder of speech (as is dysarthria), basic language processes are intact, (although some would consider apraxia to be a type of phonological aphasia) but the mechanical production of speech is impaired because of nervous system damage.

Apraxia of speech is a disruption of the capacity to program the skilled oral movements necessary for speech. The problem is with the programming associated with incorrect neural commands at higher, more central levels.

"Apraxia" comes from the Greek word "praxis," which means action.

In the past, apraxia was often classified as a type of articulation disorder and was in fact called central dysarthria by some. Now, it is considered a motor planning/programming deficit. According to Wertz (1984), apraxia of speech "is a neurogenic phonological disorder, resulting from the sensorimotor impairment of the capacity to select, program and execute coordinated movements of the speech musculature for the production of voluntary speech." In other words, the part of the brain that generates the motor programs for speech/phonology is damaged. Darley et.al. 1975 have referred to the motor planning aspect of language/speech as the motor speech programmer (MSP). Kearns and Simmons (1989, in Northern, 1989) reported that research using spectrographic analysis of voice onset time (VOT) supports the programming position. The following physiological studies support the spectrographic results: fiberoptic observation, electromyography, voicing measures, and muscular movement.

### Other Apraxias

**Limb apraxia** is another planning deficit that impairs the voluntary movements of the arms, hands, legs and feet. Many tests used to diagnose receptive aphasia require patients to respond to questions by pointing at items or following commands involving limb movement like "scratch your nose." For this reason, speech pathologists must be aware that a person's poor scores on some sections of an aphasia battery may be due to limb apraxia rather than aphasia. Lesions that cause limb apraxia are usually in the pre-motor area, Brodmann's area 6. According to Duffy, 1995, limb apraxia is a disorder of the dominant hemisphere, i.e. the left hemisphere for most.

As with apraxia of speech, voluntary movements of the limbs are affected by this disorder, but often involuntary movements remain intact. A patient with this problem would be unable to follow the command "scratch your nose" but could perform this action with ease if his/her nose were itching.

**Oral apraxia** is an inability to make voluntary, non-speech oral movements. A person with this problem would be unable to stick out his tongue if told to do so, but could perform this action without struggle if given an icecream bar to eat. As the auditory comprehension sections of some aphasia batteries include commands involving oral structures like "lick your lips," oral apraxia may also be mistaken for aphasia.

**Apraxia of gait** refers to difficulty with programming the motor movements involved in walking.

**Apraxia of the swallow** refers to a patients inability to swallow volitionally.
According to Darley (1978, as cited in Meitus & Weinberg, 1983, p. 266), the characteristics of apraxia of speech are as follows:

The patient struggles to avoid errors and to correct articulatory positioning, leading to prosodic alterations (slow rate, equalization of syllabic stress, etc).

Substitution errors predominate, but some prolongations, additions, and repetitions occur. Distortions and omissions are less frequent. Recent studies using instrumentation and narrow transcription reveal that distortions are actually the predominant error, but using broad transcription and listening, substitutions errors seem most common (Wertz, et al., 1984, p. 53).

Sound or syllable transpositions may be present.

Sometimes, articulatory errors are complications rather than simplifications.

Errors are often inconsistent off-target approximations. There is variability in type of articulatory errors, and instances of error-free fluent speech on some words, phrases, and occasionally on sentences.

Anticipatory errors may be present. The patient anticipates a speech sound which will occur further on in the word or sentence.

There is greatest difficulty in initiating speech, as noted by hesitations, and nonfluencies, visible searching, groping movements of the articulators, numerous trials, and false starts (this resembles stuttering).

Speech comprehension and word recognition abilities are often disproportionately better than speech production abilities.

Patients recognize their articulatory errors.

The number of errors increases as a function of word length.

More errors occur on consonants, and sounds which require precise articulatory adjustments, e.g. fricatives and affricates. Consonant clusters are most difficult of all. Palatal and dental phonemes are associated with a high rate of errors (Halperin, 1986 in Chapey, 1986).

Greater difficulty is noted during purposeful speech than during the production of reactive, automatic, or emotional speech.

There are increased errors in the production of words such as nouns, verbs, adjectives and adverbs, which carry more linguistic weight (Meitus & Weinberg, p. 268).

Accuracy on frequently occurring phonemes is higher (Wertz et al. p. 62).
Accuracy is better with **meaningful speech** compared to nonsense stimuli (Wertz et al. p. 63).

Fewer errors are made during **unstructured spontaneous language** than in word repetition or imitation tasks (Chapey, 1981). However, there is debate about this, with Wertz et al., 1984, (p. 66) believing the opposite to be true.

Patients do better with stimuli presented by a **visible examiner** (auditory-visual mode) than with stimuli presented on a tape recorder (Halperin, 1986).

Patients do better given repeated trials of a word rather than with increased stimulus presentations. In other words, **one stimulus presentation with several opportunities to imitate it** is better than the converse. (Johns and Darley, 1970, in Halperin, 1986) This has important implications for therapy!!

Some report **no benefit** when using the following:

- having patients observe themselves in a **mirror**,
- introducing **masking noise** to prevent patients from hearing their own speech,
- **delaying** the patient's imitative response,
- varying the instructional set given to the patient as to the task's difficulty,
- using a **metronome** (Halperin, 1986).

Apraxia of speech is **often**, but not always, accompanied by **oral motor apraxia**, or an inability to voluntarily move the mouth, tongue, lips, cheeks, larynx, and pharynx as directed (Halperin, 1986).

Some patients who suffer from apraxia of speech have **impaired oral sensation and perception** (Wertz et al., 1984 p. 75).

The following taken from Halperin (1984), who himself draws from numerous sources, is somewhat redundant but it may help you better understand apraxia of speech:

Involuntary speech is relatively preserved.

Because apraxia involves problems with motor planning for voluntary or purposeful speech, involuntary speech or the recitation of common, over-learned phrases and sayings remains remarkably intact. A patient with apraxia may struggle and appear to "grope" for sounds when trying to produce simple sentences or even single words during a conversation but might be able to recite the Lord's prayer or the Pledge of Allegiance clearly and easily.

For apraxics, imitative responses are even more difficult to produce than spontaneous speech, possibly because this kind of speech is especially "voluntary."

Articulatory errors increase as the complexity of the motor adjustment required of the articulators increases.

Therefore, it is easier for the apraxic patient to produce vowels than consonants. Single consonants are more readily produced than blends or clusters. Fricatives and affricates are the hardest manner of production for apraxics to master. As for place of production, palatal and dental phonemes seem to be the most difficult sounds. In general, phonemes that occur with high frequency in the language reproduce more accurately.
than those that occur less frequently.

Articulation errors will increase as the length of words increases. When saying a multi-syllable word, an apraxic may produce the syllables out of order.

Some errors are anticipatory.

Like stutterers, apraxics will sometimes realize that they are going to have difficulty producing an upcoming sound and hesitate. Such pauses make apraxic speech dysfluent and impairs prosody.

Some errors are perseveratory.

The apraxic patient will have difficulty virtually every time he/she tries to produce certain sounds or certain words. According to Deal (1974, as cited in Halpern, 1984), apraxic patients demonstrate a "consistency" effect when asked to read the same passage several times. That is, they make errors in the same places during all trials.

Errors are inconsistent.

While apraxics will error repeatedly on the same word, the specific off-target approximations produced will vary. For example, if an apraxic patient has difficulty saying the word "telephone" he/she may first err by saying "pelephone" but then mispronounce the target as "kelephone" or "felephone" on the next trial.

Initial consonants are misarticulated more frequently than are consonants in other positions.

This is another way in which apraxic speech resembles stuttered speech. Initial consonants may be more difficult for apraxics because they are more affected by the anticipation of errors. Also, it may be that once the apraxic has started a word and produced a vowel sound, this helps them to ease into the production of the remaining consonants.

Articulatory errors occur more often on words that carry a lot of psychological or linguistic weight and are therefore most essential for communication.

This is yet another way that apraxic speech resembles stuttered speech.

Errors are often off-target approximations of the desired articulatory production.

Errors are more likely to differ from targets in terms of place and manner of articulation rather than voicing.

Although omissions, distortions, additions and transpositions all occur, substitutions are the type of error most frequently made by apraxics.

According to Johns and Darley (1970, as cited in Halpern, 1981), patients are more likely to produce an articulatory target correctly when they are given several opportunities to do so.

Repeated presentation of the target by the clinician, on the other hand, is not helpful. This means that the clinician should present a stimulus once and then give the patient four or five chances to repeat it.

The modality in which a stimulus is presented influences the articulatory success of apraxic patients.

The repetitions of an individual with apraxia are more accurate when they are able to
watch a clinician produce the target (speech reading the clinician) than when they only hear or read the stimulus.

Finally, it should be noted that there are several different types of apraxia. The form of the disorder that affects speech should really be called apraxia of speech or verbal apraxia to distinguish it from these other types.

**Site of lesion**

There is some controversy but apraxia of speech appears to result from damage to the inferior or third frontal convolution of the dominant hemisphere (the region of Broca's area). This region is also known as Brodmann's area 44/45. Frequently Broca's aphasia and apraxia co-occur. Some feel that the basal ganglia may have programming functions similar to those attributed to Broca's area.

Such lesions can be produced by stroke or traumatic brain injury (or other types of brain damage) tumors, metabolic encephalopathy and etc. According to McNeill and Kent, 1990 (in Duffy, 1995), apraxia of speech may be an integral part of the Broca's aphasia syndrome. Many others disagree.
Unit 9. Diagnosis of Apraxia

Primary diagnostic methods are listening to the patient's speech and observing oral motor movements. Present a series of speech and oral motor tasks to the patient. Of course, damage to Broca's area can also cause Broca's aphasia. As Broca's aphasia is a non-fluent form of the disorder in which speech is labored, choppy and poorly articulated, it may be hard to differentiate between this syndrome and apraxia. One instrument that can help a clinician to make this distinction is the Token Test (DeRenzi & Vignolo, 1966). This test has five parts involving the manipulation of objects of different colors, shapes and sizes. It tests receptive grammar by requiring the patient to follow instructions like "put the red square on the yellow circle." Since patients with Broca's aphasia have difficulty comprehending grammatical morphemes like the preposition "on" they will do poorly on the test. Apraxics, on the other hand, will have no difficulty following the commands unless they have limb apraxia and therefore cannot volitionally follow commands. Another test I give routinely is the Boston Examination for Aphasia. It's a good test that just got better (2001). I was hired by Williams and Wilkins Publishing Company to review the new edition and was very favorably impressed.

It should be noted that a patient may well have both apraxia and Broca's aphasia.

Both limb and oral apraxia may be mistakenly diagnosed as an auditory comprehension deficit.

Non-Speech Tasks

Begin with vowel production. Go all the way around the vowel triangle (quadralateral). Now have the patient perform diadochokinetic tasks: repeat /p/, /t/, /k/ then /ptk/ several times (rapid alternating task).

Speech tasks

- produce words of progressively increasing length (e.g. hope, hopeful, hopefully; thick, thicker, thickening); apraxic patients performance tends to deteriorate as words increase in length;
- repeat several multisyllabic words three times (e.g. butterfly, butterfly, butterfly); production may improve with some apraxic patients;
- repetition of sentences
- conversation
- picture description
- oral reading
- counting to 20
- days of the week
- months of the year

Motor Speech Evaluation
(suggested by Wertz et al., 1984, p. 98):

This is a screening tool which should take less than 20 minutes to administer. Three steps may be required to obtain useful data. If the patient does not respond in such a way as to give diagnostic information, repeat the stimuli. If the second response is still ambiguous, cue (e.g. "Listen, watch me, and do what I do").
Scoring the Motor Speech Evaluation can be descriptive:

Use "A" for apraxic productions, "P" for paraphasias, "D" for dysarthria, "U" for nondiagnostic errors, "O" for other errors, and "N" for normal responses. It can also utilize the PICA 16-point scale, or narrow or broad phonetic transcription.

The tasks are conversation, vowel prolongation, repetition of monosyllable /p/, /t/, /k/; repetition of those in sequence, repetition of multisyllabic words; multiple trials with the same word; repetition of words that increase in length; repetition of monosyllabic words that contain the same initial and final sound; repetition of sentences; counting forward and backward; picture description; repetition of sentences used volitionally to determine consistency of production; and oral reading.

Generally, apraxic patients will reveal their deficits in conversation by producing apraxic articulatory errors and abnormal prosody. Usually they have no problem with vowel prolongation or repeating single monosyllables. With the sequence of /ptk/, there may be initiation difficulty, substitution, omission, or rearrangement of the syllables; slow rate; equal and even stress; stops, starts, and reattempts to produce the sequence. Similar errors should be evident with multisyllabic words and short phrases. Repeated trials on the same word may show inconsistent errors. Words of increasing length should show more errors on the longer words. Monosyllabic words beginning and ending with the same sound may show more errors in initial position, but not necessarily. Sentences and picture description will produce apraxic errors in articulation and prosody. Counting forward and backward contrasts automatic speech versus volitional speech, with more errors expected on the latter. Having the patient repeat sentences he/she produced earlier allows the examiner to check for consistency, with the apraxic expected to be inconsistent. Oral reading of the "Grandfather Passage," which contains most of the sounds of English, allows comparison with repetition tasks and more volitional tasks.

Some oral movement tasks (suggested by Darley, 1978 and DeRenzi et al, 1966; cited in Meitus & Weinberg, p. 269): (use verbal instruction alone at first; if patient cannot perform, then demonstrate and observe his/her imitation)

- stick out your tongue
- puff or blow
- pucker up your lips
- try to touch your nose with the tip of your tongue
- bite your lower lip
- whistle
- move your tongue in and out of your mouth
- lick your lips
- clear your throat
- click your teeth together once
- smile
- click your tongue as if imitating the sound of a galloping horse
- chatter your teeth as if you are cold
- try to touch your chin with the tip of your tongue
- cough
- puff out your cheeks
- wiggle your tongue from side to side
- show how you would kiss someone
- alternately pucker and smile
- yawn

The clinician should make qualitative judgments of the patient's performance, eg., consider the presence of:

- accurate movement patterns preceded by trial and error
- searching movements of the tongue or lips
- accurate movement preceded by pauses
- crude, awkward, erratic, or extraneous oro-facial movements
- overall gesture patterns which are grossly acceptable, but defective in terms of amplitude, accuracy, or speed
- perseverated movement

**Commercial Instruments Available**

Apraxia Battery for Adults (ABA) by Barbara Dabul (1979)

According to Wertz et al. (1984, as cited in Halperen, 1986), several factors affect the prognosis of an apraxic patient. These include:

**Size of lesion**

The smaller the lesion, the more favorable the prognosis.

**Presence of concomitant disorders**

If the patient has only minimal aphasia and does not display severe oral apraxia, the prognosis is better.

**Health and attitude of the patient**

The better the state of the patient's over-all health and the more motivated he/she is to practice drills and improve his/her speech, the better the prognosis.

**Time post-onset**

Patients who begin treatment within one month of the onset of apraxia have a better chance of recovery.

According to Wertz et al. (1984), patients with a better prognosis are those who: are less than one month post-onset; have a small lesion confined to Broca's area, have minimal coexisting aphasia, do not have significant oral apraxia, are in good health, and have the strength to endure intensive treatment.

Prognosis for functional recovery is poor without treatment; fair with treatment for the severe patient; and good with treatment for the moderate to mild patient.

**Remediation/Therapy for Apraxia of Speech**

*Halperen, 1981, pp. 425-426*

Emphasis should be on the auditory and visual modalities, but especially the visual.

Using the **phonetic placement method** of describing the correct manner and place of articulation and the correct voice and voiceless components of phoneme production is useful.

Dabul and Bollier (1976 in Halperen, 1981) outlined the following sequential program:

- Mastery of individual consonant phonemes.
- Rapid repetition of each consonant plus the vowel /a/.
- The buildup of sounds into syllables using CV CV combinations, such as /fa ta/, and CVC combinations such as /p a p/.
- Once the patient has a solid base of articulatory positions, he can master words by breaking them down into individual phonemes, then blending them into syllables and words.
A visual technique (speech reading the clinician) and a placement technique (using oral directions and physical placements) are helpful. Words can be broken down into syllables using graphic materials, with part of the word covered.

Therapy can begin with vowel sounds, and then successive consonants, e.g. /m/, which is visible and manipulable. Once the /m/ can be produced in isolation, it can be combined in the initial position with the vowels that were learned (e.g. /mi, me, ma, mo/ etc.). Next /m/ is produced in the final position in one-syllable words (e.g. am, I'm, arm, etc.); two syllable words (mamam, memo, etc.); and then in word lists with /m/ in all positions (omen, madam, etc.). Then the therapist can build up from simple phrases (my man, my mama), to longer ones (e.g. miles from Montana) to sentences (e.g. The mailman will get the mail.) Further exercises might contrast words with /m/ in the initial position with a minimal pair (e.g. man-pan, mare-pear), and then /m/ in the final position (e.g. comb-coat, came-cape).

**Melodic Intonation Therapy** (Sparks and Holland, 1976) may be a useful therapy tool. This therapy, originally developed by Sparks and Holland for the treatment of severe non-fluent aphasia. Currently, however, it is most frequently used to treat apraxics. This technique involves teaching the patient to sing words or phrases set to simple melodies. It is hypothesized that this therapy is effective because the use of music helps involve the right hemisphere in the production of speech.

For severe apraxics, alternative or supplementary methods of communicating may be needed. A gestural system such as Amer-Ind can be taught to the patient and his family. Communication boards or computer programs may likewise be helpful.

The goal of therapy for apraxia is to help the patient regain conscious control over articulatory programming so that speech can be produced voluntarily.

Some, including Darley, recommend compensatory types of therapies for apraxia. Such therapies focus on helping the patient take full advantage of his remaining communication abilities. A number of people urge compensatory techniques. In contrast, the therapies for apraxia presented in this course are those that attempt to "re-train" the brain. Such techniques endeavor to stimulate another part of the cortex to take over the motor programming function previously served by Broca's area.

**Sensory Modalities**

According to most sources, apraxics must receive multimodality input or multisensory information, including visual, auditory, tactile and kinesthetic feedback, in order to benefit maximally from therapy. Virtually all research has found that visual input is the most important source of feedback for apraxics. This means that patients should be instructed to focus on how their speech sounds, on how it feels to produce speech targets and, most importantly, on how the articulators look during the production of speech targets. To encourage focus on the visual modality, patients should be told to watch as the therapist produces target sounds or words and then to observe their own production of these targets by using a mirror.

**Repetition**

When asking an apraxic patient to imitate your production of a target, allow him to make four or five attempts before repeating the stimulus. This groping, or trial and error articulatory process is believed to be essential in "re-training" the brain to do the motor programming necessary for voluntary speech.

**Hierarchy of Speech Tasks**

For an apraxic, the difficulty involved in producing a speech target depends on its length as well as on the types of speech sounds that it contains. Therefore, both these factors can be used to develop a hierarchy of speech drills for the apraxic patient. Of course, the hierarchical level at which therapy begins for any particular patient depends upon the extent of that individual's impairment. If a patient can produce CVC words, there is no need to start work at the single phoneme level.

**Phonemic Categories**

Below, classes of phonemes are listed in order from least to most difficult for an apraxic patient to produce. Note that this arrangement is the same as the order in which children acquire different categories of speech
sounds. Also note that it is harder for apraxics to produce blends than single consonants. Consonant clusters are even more difficult to produce than blends.

Vowels

Semivowels

Nasals

Plosives

Fricative

Affricates

With therapy with a severely apraxic patient, it may be necessary to begin working on sound production by teaching the person to phonate voluntarily. To do this, hold the patient's hand against your larynx while you cough. Then ask them to hold their hand against their own larynx while they cough. Try to move from coughing to the production of /a/. (As first, the patient may have to start /a/ with a cough.)

Once the patient can phonate voluntarily, work on the production of a few vowels. It is best to start with /u/, /i/ and /a/ as these are the most visible. It may also be helpful for the patient to produce some diphthongs, moving back and forth between the two components.

When the patient has mastered some vowels, move on to the nasals. It is a good idea to begin with /m/ as the production of these sounds can be seen very easily. As therapy continues, sounds should be introduced in order from least to most difficult. So, after the nasals have been learned, the semivowels should be presented. These should be followed by the plosives, fricatives and affricates. Later, blends and clusters can be practiced.

**Utterance Length**

Remember, the shorter the utterance, the easier it is for an apraxic patient to produce it. For this reason, drills should be arranged based on utterance length as well as on phoneme type. As each new phoneme is incorporated into the drills, the length of targets including that sound should be increased gradually. Sounds might initially be produced in isolation and finally in phrases consisting of multi-syllabic words.

Here is an example of how drills involving the phoneme /m/ might gradually increase in length (Dabul and Bollier, 1976, in Halperen, 1981):

- **Work on /m/ in isolation.** To facilitate production, tell the patient to hum. If that doesn't work, the patient might be instructed to use her fingers to put her lips together. (The clinician could use her fingers to do this if necessary.)

- **Work on /m/ in the context of CV syllables like /mi/, /ma/, /mu/ and /mo/ . (These four vowels are maximally contrasted.)**

- **Ask the patient to slowly shift from the production of one of these CV's to another.** When he becomes proficient at doing this slowly, have him do the task more rapidly.

- **Use double syllables.** Have the client produce two successive repetitions of the CVs practiced in step #2. For example, the target might be /mi/ /mi/. As before, have the client shift from the production of one syllable to another ( /mi/ /mi/ -- /mu/ /mu/).

- **Use CVC forms.** The initial and final consonants of these syllables should both be /m/. For example, the patient might be asked to produce the syllables /mim/, /mum/, and /mam/.
Use words rather than nonsense syllables. Of course, the patient should begin with CV words like "my" and "me."

Use CVC words like "more", "mayor", "mine", "map", and "mat."

Use two-word segments like "more money," "my monkey"

Use two-word segment in which the components end with /m/ instead of beginning with it. Some examples are:

- some dam
- same pram
- lame ram
- game time

Use two-word segments in which one word begins with /m/ and the other word ends with /m/. Some examples are:

- money game
- Mary's lamb
- miserable Sam

Repeat steps 7, 8 and 9 using multi-syllabic words.

**Contrasting**

It will be helpful for the apraxic patient to practice drills involving contrasting phonemes. At first, exercises should include maximally contrasting phonemes, or those that differ by the greatest number of distinctive features. For example, the patient might practice saying some words that begin with the labiodental /f/ and others that begin with the palatal sound /k/ (phil vs. kill). The degree of contrast between the phonemes paired in exercises should be gradually reduced until minimal pairs, or two phonemes that differ by only one distinctive feature, are used.

**Exaggeration of Intonation and Stress**

As apraxics often have abnormal prosody due to the dysfluent nature of their speech, it may be a good idea to have them practice using intonation and stress to change the meaning of utterances.

For example, a patient could practice contrasting phrases like "You must read that book" and "You must read that book."

**Integral Stimulation**

This simply means that the patient watches and listens to the therapist's productions and then attempts to imitate this model. According to Rosenbeck et al. (1973, in Halpern, 1981), this is the primary procedure to be used in the remediation of apraxia. It is an 8 step "listen and watch me" technique.

**Phonetic Placement**

The therapist may verbally instruct the patient on the correct placement of the articulators for the production of a particular sound. The therapist may also physically manipulate the patient's articulators. For example, the clinician might place the patient's lips together for the production of /m/ or use a tongue depressor to facilitate lingual movements.
**Automatic Speech**

One of the hallmarks of apraxia is the relative preservation of automatic or over learned speech sequences such as greetings, leave-takings and proverbs. Automatic speeches are used in therapy for two reasons; first, it gives the patient a break from the hard and frustrating work of drills and secondly, because it provides the brain with sensory feedback from fluent speech.

Types of automatic speech that may be used in therapy include:

- Prayer
- nursery rhymes
- counting
- recitation of the alphabet
- familiar song
- the pledge of allegiance
- advertising jingle
- proverbs
- greetings (Hi, how are you)
- leave-takings (Bye, see you later)

Of course, the clinician should find out what things will be most automatic for each individual patient.

**Some Ideas from Duffy and Others on Remediation of AOS**

Duffy (1995) recommends the following treatment principles:

- Make sure of a high success level.
- Use extensive and intensive drill.
- Work on articulation as well as prosody.
- Introduce meaningful and functional material asap.
- Systematically manipulate variables which effect response accuracy.

He feels that principles of motor learning are crucial to the treatment of AOS. Furthermore he feels that drill is paramount. According to Darley, Aronson and Brown (1975); Wertz, LaPointe and Rosenbeck (1984), and others, apparently many apraxics have lost some of the pre-programmed sub routes for movement sequences that make normal speech so automatic. Accordingly, systematic and extensive drill is used to help the patient regain or learn lost speech skill instructions.

Self learning and instruction is used by moderately involved patients as early as feasible in their treatment program. Patients are encouraged to monitor their speech, grope for correct targets, and self correct their errors.
Feedback, according to Wertz, LaPointe and Rosenbeck (1984), should be a general principle of AOS treatment. Rosenbaum (1991) and Singer (1980) feel that knowledge of results is crucial for patients. Patients should be encouraged to judge the accuracy of their responses reliably. When responses are inaccurate they should attempt to self-correct. Clinician feedback to the patient should be done from the beginning. Mirrors, or even video and audio-tapes may help. Some people have worked directly on deficiencies in oral sensation. However there are conflicting studies as to whether those approaches are helpful. Rubow (1982) has used vibrotactile stimulation for intersystemic reorganization to treat apraxia of speech. I'm quite familiar with vibrotactile research. My doctoral dissertation was in that area and I have published a number of articles on that topic. Others, Deutch (1981); Square and Weidner (1976), feel that work on oral sensation is unlikely to be of much help to apraxic patients.

Intersystemic reorganization used in the treatment of apraxia of speech has been discussed by Rosenbeck, Collins and Wertz (1976); and Rosenbeck (1978). Brookshire (1995) has quite a good description of this treatment in his text. Another type of reorganizational approach to apraxia remediation is gestural reorganization. It uses manual gestures paired with speech. Be aware too that speaking may not be a realistic goal for some severe apraxics. Augmentative communication may help as long as there is not another problem such as a severe aphasia that could preclude that. Duffy (1995) has a lot of additional material on remediation in his text.
Unit 11. Dysarthria: Definition and Description; Etiology

According to Darley, Aronson and Brown (1975), dysarthria is a speech disorder resulting from a weakness, paralysis, or incoordination of the speech musculature that is of neurological etiology. There are several different types of dysarthria. All result from damage to the central or peripheral nervous system that impairs the transmission of neural messages to the muscles involved in speech. In contrast to apraxia which affects the brain's capacity to produce the "programs" necessary for coordinated motor movements, dysarthria results from an inability to send the proper messages to the musculature. While apraxia affects articulation and, to some extent, prosody, dysarthria can impair all processes involved in speech production including respiration, phonation, articulation, resonance and prosody.

Based on etiology, Darley, Aronson and Brown (1969), identified six different types of dysarthria. These include four forms of the disorder which are caused by damage to upper motor neurons. They include spastic, hyperkinetic, hypokinetic, and ataxic dysarthria. Spastic dysarthria results from lesions to the pyramidal tract, while both hypokinetic (substantia nigra) and hyperkinetic (basal ganglia) dysarthria occur when the extrapyramidal tract is damaged. Cerebellar lesions cause ataxic dysarthria.

**Flaccid dysarthria** is the only form of the disorder that results from damage to lower motor neurons.

**Mixed dysarthria**, the sixth type described by Darley, et al., occurs when both upper and lower motor neurons are injured.

**Etiology**

**Upper Motor Neurons vs. Lower Motor Neurons**

A review of the difference between upper and lower motor neurons may make it easier to understand the causes of the various types of dysarthria.

Upper motor neurons carry information from brain centers that control the muscles of the body. The pyramidal tract, which is the most important of the upper motor neuron tracts, transmits messages directing voluntary motor movements. It is primarily facilitatory (Duffy, 1995). The extrapyramidal tract consists of neurons that regulate involuntary/automatic movements. The neurons found in the cerebellum play an important role in the coordination and smoothness of movements required for speech.

Although they carry messages intended for the muscles of the body, upper motor neurons cannot exit the central nervous system. For this reason, they synapse with the lower motor neurons which are able to pass outside the neuraxis and carry information directly to the muscles.

In order to reach the muscles of the body, motor commands generated in the central nervous system must travel along upper motor and lower motor neurons.

Upper motor neurons are a type of **first order neuron**. They are unable to leave the central nervous system. The pyramidal tract is the most important of the upper motor neuron tracts, but the
extrapyramidal tract also consists of upper motor neurons.

As upper motor neurons must remain inside the neuraxis, they synapse with neurons of another type called lower motor neurons which can carry messages to the muscles of the rest of the body.

**Lower motor neurons**, a type of second order neuron, are cranial or spinal nerves. The cell bodies of these neurons are located in the neuraxis, but their axons can leave the central nervous system and synapse with the muscles of the body.

All lower motor neurons are either spinal or cranial nerves. All spinal nerves are lower motor neurons as they are mixed nerves and therefore all have a motor aspect. However, not all cranial nerves are lower motor neurons. Some of the cranial nerves contain only sensory fibers and therefore cannot be classified as lower motor neurons. For example, CN I, the olfactory nerve, CN II the optic nerve, and CN VIII, the auditory nerve, do not have motor components.

Lower motor neurons are sometimes called the final common pathway for neural messages traveling to the muscles of the body because these nerves are the only route by which information from any of the upper motor tracts can reach the periphery. Thus, when lower motor neurons are damaged, the parts of the body that they innervate are deprived of input from the pyramidal and extrapyramidal tract as well as cerebellar pathways. Thus, voluntary, automatic and reflexive movements are all affected.

**The Pyramidal Tract**

This group of upper motor neuron fibers carries messages for voluntary motor movement to the lower motor neurons in the brain stem and spinal cord.

Approximately 80% of the cell bodies of the pyramidal tract are located on the precentral gyrus of the frontal lobe which is also known as the motor strip. Particularly large cells located here whose axons are part of the pyramidal tract are called pyramidal cells. Approximately 20% of the pyramidal tract fibers also originate in the postcentral gyrus of the parietal lobe, in Brodmann's areas 1, 2, and 3. Regardless of the location of their cell bodies, pyramidal tract fibers descend from the cortex in a fan-shaped distribution which converges inside the internal capsule (Fitz Gerald, 1996).

This tract is direct and monosynaptic, meaning that the axons of its neurons do not synapse with other cells until they reach their final destination in the brain stem or spinal cord. These direct connections between the cortex and the lower motor neurons allow messages to be transmitted very rapidly from the central nervous system to the periphery.

The fibers of the pyramidal tract that synapse with spinal nerves sending information about voluntary movement to the skeletal muscles form the corticospinal tract. These axons are among the longest in the central nervous system, as some of them travel all the way from the cortex to the inferior part of the spinal cord. As they descend through the brain, they form part of the posterior limb of the internal capsule.

At the pyramids in the inferior part of the medulla, eighty-five to ninety percent of corticospinal fibers decussate, or cross to the other side of the brain. The remaining ten to fifteen percent continue to descend ipsilaterally. The fibers that decussate are called the lateral corticospinal tract or the lateral pyramidal tract because they descend along the sides of the spinal cord. The uncrossed or direct fibers that synapse with spinal nerves on the ipsilateral side of the body are called the direct pyramidal tract. They may also be referred to as the ventral pyramidal tract or the corticospinal tract since they travel down the ventral aspect of the spinal cord.
The spinal nerves receive only contralateral innervation from the corticospinal tract. This means that unilateral pyramidal tract lesions above the point of decussation in the pyramids will cause paralysis of the muscles served by the spinal nerves on the opposite side of the body. For example, a lesion on the left pyramidal tract could cause paralysis on the right side of the body.

The fibers of the pyramidal tract that synapse with cranial nerves located in the brainstem form the corticobulbar tract. Obviously, this is the part of the pyramidal tract that carries the motor messages that are most important for speech and swallowing. Corticobulbar axons descend from the cortex within the genu or bend of the internal capsule.

Almost all of the cranial nerves receive bilateral innervation from the fibers of the pyramidal tract. This means that both the left and right members of a pair of cranial nerves are innervated by the motor strip areas of both the left and right hemispheres. This redundancy is a safety mechanism. If there is a unilateral lesion on the pyramidal tract, both sides of body areas connected to cranial nerves will continue to receive motor messages from the cortex. The message for movement may not be quite as strong as it was previously but paralysis will not occur.

The two exceptions to this pattern are the portion of CN XII that provides innervation for tongue protrusion and the part of CN VII that innervates the muscles of the lower face. These only receive contralateral innervation from the pyramidal tract. This means that they get information only from fibers on the opposite side of the brain. Therefore, a unilateral upper motor neuron lesion could cause a unilateral facial droop or problems with tongue protrusion on the opposite side of the body. For example, a lesion on the left pyramidal tract fibers may cause the right side of the lower face to droop and lead to difficulty in protruding the right side of the tongue. The other cranial nerves involved in speech and swallowing would continue to function almost normally as both members of each pair of nuclei still receive messages from the motor strip.

Because most cranial nerves receive bilateral innervation, lesions of the upper motor neurons of the pyramidal tract must be bilateral in order to cause a serious speech problem. (The effects of the inability to protrude the tongue and of paralysis of the lower face on speech are negligible.)

On the other hand, unilateral lesions of the lower motor neurons may cause paralysis. This occurs because the lower motor neurons are the final common pathway for neural messages traveling to the muscles of the body. At the level of the lower motor neurons, there is no alternative route which will allow messages from the brain to reach the periphery. Muscles on the same side of the body as the lesion will be affected.

Injuries to the nuclei of the cranial nerve nuclei located in the brain stem are called bulbar lesions. The paralysis that they produce is called bulbar palsy. It is usually bi-lateral.

Lesions to the axons of the cranial nerves are called peripheral lesions.

As cranial nerves are lower motor neurons, both bulbar and peripheral lesions are lesions of the final common pathway (FCP), although some sources consider the FCP to be axonal only.

When bilateral lesions of the upper motor neurons of the pyramidal tract occur, they produce a paralysis similar to that which occurs in bulbar palsy. For this reason, the condition is known as pseudo-bulbar palsy.
If a lesion occurs in the brain stem and damages both the nucleus of a cranial nerve and one side of the upper motor neurons of the pyramidal tract, a condition known as alternating hemiplegia may result. This involves paralysis of different structures on each side of the body. The lesioning of the nucleus of the cranial nerve will cause a paralysis of the structures served by that nerve on the same side of the body as the injury. Because the pyramidal tract provides only contralateral innervation to the spinal nerves, damage to the upper motor neurons will meanwhile cause a paralysis of different structures on the other side of the body. For example, a lesion that affected the right nucleus of the trigeminal cranial nerve and the right side of the pyramidal tract would cause paralysis of the right side of the jaw and of part of the left side of the body.

Both the corticospinal and corticobulbar tracts send some axons to the pontine nuclei in the pons as they descend to synapse with lower motor neurons. These fibers that end in the pons form the corticopontine tract. This pathway carries information about the type and strength of the motor impulses generated in the cortex to the cerebellum. The copy sent to the cerebellum actually leaves the cortex before the command is sent to the muscles. While the corticopontine fibers actually end in the pontine nuclei, second order neurons carry their message to the cerebellum via the middle cerebellar peduncle. I consider this tract to be part of the extrapyramidal system rather than a component of the pyramidal tract since it does not synapse directly with lower motor neurons.
Spastic Dysarthria

As noted above, spastic dysarthria results from damage to the pyramidal tract. Recall that most pyramidal tract cells originate in the cortex of the precentral gyrus of the frontal lobe which is also known as the motor strip. The pyramidal tract fibers that carry messages to the spinal nerves are called the corticospinal tract. The part of the pyramidal tract that sends information to the cranial nerves involved in speech and swallowing is called the corticobulbar tract. According to Dworkin (1991, p.188), spastic dysarthria is characterized by “Generalized hypertonicity, weakness, immobility, abnormal force physiology, and exaggerated reflexes of virtually all muscles of the speech mechanism produce obvious dysfunction of the articulation subsystem. Speech is slow-labored, and imprecise articulatory efforts, compounded by disturbances of respiration; resonation, and phonation often render speech unintelligible.”

The spinal nerves receive only contralateral innervation from the corticospinal tract. This means that spinal nerves on the left side of the body receive input only from the motor strip of the right hemisphere while those on the right side of the body are innervated by the half of the motor strip located in the left hemisphere. Therefore, unilateral lesions of the pyramidal tract can cause paralysis of the muscles innervated by the spinal nerves on the opposite side of the body. For example, damage to the left half of the corticospinal tract could result in paralysis of the right side of the body.

On the other hand, almost all of the cranial nerves receive bilateral innervation from the fibers of the corticobulbar tract. This means that both the left and right members of most pairs of cranial nerves are innervated by the motor strips of the right and left hemispheres of the brain. This redundancy is a safety mechanism. If there is a unilateral lesion of the pyramidal tract, both sides of body areas connected to the cranial nerves will continue to receive messages about voluntary motor movement from the cortex. These messages may not be quite as strong as they were previously, but paralysis will not occur. This means that the muscles involved in speech would continue to function adequately in spite of damage to one side of the pyramidal tract. Therefore, only bilateral lesions of the pyramidal tract will cause a serious, spastic dysarthria. When such bilateral lesions occur, they produce a condition known as pseudo-bulbar palsy. This name is derived from the term bulbar palsy which is used to describe the symptoms of brain stem lesions.

(The two exceptions to the innervation scheme described above are the portion of CN XII that controls tongue protrusion and the part of CN VII that innervates the lower face. Like spinal nerves these two cranial nerve nuclei receive only contralateral innervation from the pyramidal tract. Thus, a unilateral pyramidal tract lesion could cause drooping of the lower part of the face or problems with tongue protrusion on the opposite side of the body. However, neither a unilateral weakness of the facial muscles nor the inability to protrude one half of the tongue are likely to cause a significant speech impairment. For this reason it can still be said that only bilateral pyramidal tract lesions seriously affect speech.)

It should be noted that although speech will not be much impaired by unilateral pyramidal tract damage, swallowing may be compromised by this type of lesion. Unilateral lesions are unlikely to cause a serious dysphagia, but they may produce abnormalities that require treatment. Remember that swallowing requires great precision as does speech and is therefore easily impaired by neurological damage. Many of my dysphagia patients over the years had been diagnosed with unilateral upper motor neuron lesions. Swallowing is less forgiving than speech of neurological damage.
The Extrapyramidal Tract

This system is involved in automatic motor movements, and in gross rather than fine motor movement. It works with the autonomic nervous system to help with posture and muscle tone and has more influence over midline structures than over those in the periphery. It regulates reflexes and maintains posture and tone (Duffy, 1995). Facial expression is one important communicative behavior that is mediated by the extrapyramidal tract. In contrast to the pyramidal tract, the extrapyramidal tract is an indirect, multisynaptic tract that is primarily inhibitory.

Components of the extrapyramidal tract include the basal ganglia, the red nucleus, the substantia nigra, the reticular formation, and the cerebellum. All of these structures send information to the lower motor neurons in an indirect, multi-synaptic fashion.

The basal ganglia acts to inhibit the release phenomenon, or the rapid firing of motor neurons. It is aided in this function by the substantia nigra of the midbrain. The muscles most often affected by this inhibitory function are those controlling the head, the hands, and the fingers.

The neurotransmitters involved in the inhibitory function of the basal ganglia include dopamine, which is produced by the substantia nigra, acetylcholine, and GABA, which is a glutamate. Dopamine is an especially powerful inhibitor.

Extrapyramidal Projections to Lower Motor Neurons

The rubrospinal tract passes through the red nucleus. The cerebellum sends messages to the spinal nerves along this tract. Information flows from the superior cerebellar peduncle to the red nucleus and finally to the spinal nerves. This information is very important for somatic motor, or skeletal muscle control and the regulation of muscle tone, smoothness of movement and for posture.

The reticulospinal tract runs from the reticular nuclei of the pons and medulla to the spinal nerves. It is involved in somatic motor control like the rubrospinal tract and also plays an important role in the control of autonomic functions.

The tectospinal tract has points of origin throughout the brain stem, but especially in the midbrain area, and ends in the spinal nerves. It is involved in the control of neck muscles and also in visual and auditory reflexes. So, when you jump after hearing a noise or duck when you see something coming toward you, this tract helps to mediate these reactions.

The vestibulospinal tract runs from the vestibular nuclei located in the lower pons and medulla to the spinal nerves. It is involved in balance.

(Note that all of these tracts receive input from the cerebellum.)

Extra Pyramidal Diseases and Syndromes Affecting Communication/Swallowing

Lesions in the extrapyramidal tract cause various types of diskinesias or disorders of involuntary movement.

The problems mostly common anomalies affecting the extrapyramidal tract include degenerative diseases, encephalitis, and tumors.

Parkinson's Disease, which is a degenerative disease, is probably the most frequently occurring illness that results from extrapyramidal tract lesions. It occurs when the dopaminergic neurons of the substantia nigra are destroyed. Its symptoms include:

Tremor
Festinating movements, especially a festinating gait, but these movements can also affect speech. (Festinating movements are movements which become increasingly rapid and uncontrolled).

Hypokinetic dysarthria

Weak Voice

Mask-like facial expression

This will be discussed in greater detail a little later.

Diseases associated specifically with lesions of the basal ganglia include Huntington's Chorea and Sydenham's Chorea. The word "chorea" comes from the Greek "khoros" which means to dance. Both of these diseases are associated with jerky, uncontrolled movements of the limbs. Sydenham's chorea was probably the cause of the malady that was known as St. Vitus' Dance during the middle ages. Huntington's Chorea is an inherited degenerative disease. These will be discussed further a little later.

Essential Tremor Syndrome, which is associated with Spastic Dysphonia may also be the result of basal ganglia lesions.

Lesions of the basal ganglia will also cause hyperkinetic dysarthria.

Note that not only is the definition of the extrapyramidal system controversial, but also many sources say that it is very difficult to make functional distinctions between the extrapyramidal and pyramidal systems. When upper motor neuron lesions occur, it is often difficult to determine which tract has been damaged.

Hyperkinetic Dysarthria

Hyperkinetic dysarthria results from damage to the extrapyramidal tract. It specifically results from lesions of the basal ganglia. The extrapyramidal system is involved in automatic motor movements and works with the autonomic nervous system to control posture and muscle tone. Components of the extrapyramidal system include the basal ganglia (caudate and lenticular nuclei), the red nucleus, the substantia nigra, the reticular formation and the cerebellum. Also included are the rubro spinal, reticulo spinal, and vestibulo spinal tracts. The latter runs from the vestibular nucleus in the pons to the spinal cord. The vestibular nucleus also connects to the semicircular canals in the ear and to the cerebellum for balance and orientation in space. Again, hyperkinetic dysarthria most often results from damage to the basal ganglia specifically. Dworkin (1991) describes hyperkinetic movements as quick, jerky and unsustained as with Huntington's chorea, or slow, writhing and sustained as observed in people with athetosis and dystonia. He describes those with essential tremor and myoclonus as having tremorous movements. As you can see there are several different types of speech movement abnormalities in people with hyperkinetic dysarthria. Dworkin's 1991 text is exclusively on remediation of motor speech disorders. While being mindful that there is a good deal of diversity among patients, Dworkin (1991, p.189), generally recommends the following type of sub-system exercises for hyperkinetic dysarthria: "1. Lingual, labial, and mandibular force physiology training. 2. Phonetic stimulation in various contexts." He also feels that this treatment in general can also apply to ataxic patients after they receive treatment of the respiratory and phonation sub-systems.

Conditions that may cause hyperkinetic dysarthria include essential tremor syndrome,
chorea, athetosis, and dystonia and tardive dyskinesia. All result in the suprimposition of automatic movements upon volitional movements.

1. **Essential tremor syndrome** is also known as heredofamilial tremor. Usually, various parts of the body, including the jaw, head and hands are affected, displaying oscillatory movements superimposed upon voluntary and involuntary movements. When only the laryngeal musculature is affected, the condition is called organic voice tremor. In this case, phonation is abnormal but resonance and articulation are not impaired. The voice disorder resulting from organic voice tremor may involve alternations of pitch and loudness at regular intervals. It may also involve irregular, complete stoppage of voicing. When the later symptom occurs, organic voice tremor may resemble spastic dysphonia. However, there is no strong concensus as to whether the two disorders are actually related.

2. **Chorea** is a "disorder characterized by irregular, spasmodic, involuntary movements of the limbs or facial muscles" (Love & Webb, 1992, p. 289). "Choreic movements are rapid and coordinated but purposeless." These "fidgets" can cause dysarthria by impairing the coordination of respiration with speech and interrupting the movements of the articulators during speech (Love & Webb, 1992, p. 149). There are two types of chorea, **Sydenham's Chorea** and **Huntington's Chorea**.

3. **Sydenham's chorea** is a non-hereditary form of the disorder that occurs during childhood following infections like strep throat, rheumatic fever or scarlet fever. The symptoms of this disorder will resolve spontaneously within six months of onset. (It is believed that this is the disease that was known as St. Vitus' Dance during the Middle Ages.)

4. According to Fitz Gerald (1996), **Huntington's chorea** is transmitted genetically (chromosome 4 ). Because the disorder is transmitted by a single, dominant gene, a patient's children have a fifty percent chance of developing it themselves. This form of chorea is progressive and fatal, causing the loss of neurons in the cortex as well as in the caudate nucleus and globus pallidus. The onset of Huntington's chorea typically occurs in the 50's although it may begin much earlier, even in childhood.

5. **Athetosis** is a disorder that causes slow writhing movements of the entire body but especially of the arms, face and tongue. These movements can affect speech by interrupting the action of the articulators and of the respiratory system. Athetosis most often results from lesions of the putamen. The caudate nucleus may also be involved. It is a rare disorder that usually occurs as a form of cerebral palsy brought on by perinatal anoxia. An especially rare form of athetosis begins during adolescence and follows a progressive course. According to Fitz Gerald (1996), spontaneous movements are considered an escape phenomenon caused by damage to the striatum and/or subthalamic nuclei. This silences the globus pallidus permitting the ventral lateral (VA) nucleus of the thalamus to fire spontaneously.

6. **Dystonia** causes slow jerky movements which are most likely to occur in the trunk, neck and proximal parts of the limbs. These may be exacerbated by voluntary movements, like those involved in speech (Love & Webb, 1992). Rosenfeld (1991) feels that it results from a combination of dopaminegric and cholinergic overactivity in the basal ganglia. When oralfacial muscles are involved the condition is called focal mouth dystonia (Duffy, 1995).

Some have speculated that spastic and spasmodic dysphonia, a condition "characterized by effortful, strained voice quality with voice arrests" (Love & Webb, 1992), may be a localized form of dystonia. Not much evidence supports this view, however. Although it has been believed that the intermittent aphon
seen in cases of spastic dysphonia is the result of hyperadduction or spasm of the vocal folds. Indirect laryngoscopy generally shows that the folds continue to move normally during these episodes (Love & Webb, 1992). It should also be noted that some cases of spastic dysphonia appear to be psychogenic in origin and are thus not related to any hyperkinetic movement disorder.

As its etiology has not yet been identified, the treatment of spastic dysphonia remains controversial. In the past, the recurrent laryngeal nerve was cut to ameliorate symptoms. While this operation improves voice for a time, symptoms generally reappear. At this time, injections of botulinum-A toxin (Botox) seem to be the most promising treatment for spastic dysphonia.

7. **Tardive Dyskinesia** is a condition which results from longterm use of antipsychotic drugs called phenothiazines. These drugs are sometimes used to treat schizophrenia. This movement disorder causes "choreiform and peculiar rhythmic movement" (Love & Webb, 1992), note that these are especially likely to affect the tongue and lips. Involuntary movements of the tongue in and out of the mouth, which are known as **fly-catcher's movements**, are characteristic of tardive dyskinesia. It should be noted that the symptoms of this form of hyperkinesia do not always resolve when the use of phenothiazines is discontinued.

**Hypokinetic Dysarthria**

Hypokinetic dysarthria is usually the result of Parkinson's Disease (discussed earlier). The disorder causes the degeneration of the substantia nigra, which is one component of the extrapyramidal system. The substantia nigra supplies dopamine to the cells of the basal ganglia, allowing it to inhibit the release phenomenon or the rapid firing of motor neurons. Thus, involuntary movements occur as a result of the shortage of dopamine caused when the substantia nigra is damaged. Dworkin (1991) recommends the same type and sequence of sub-system exercises as for hyperkinetic dysarthria described above. According to Dworkin (1991) "widespread rigidity and associated paresis of virtually all muscles of the speech mechanism justify treatments to improve resonance, phonation, articulation, and indeed prosody." It is currently believed that Parkinson's Disease results from genetically based abnormalities of the mitochondria, passed on through the female. This trait appears to be X-linked. According to Fitzgerald (1996), the disease affects about 1% of people over fifty years of age.

Parkinsonism, or Parkinson’s-like symptoms, can also cause hypokinetic dysarthria. Parkinsonism can result from carbon monoxide poisoning, arteriosclerosis, the use of tranquilizers like Haldol, or from repeated blows to the head. (Mohammad Ali has developed Parkinsonism due to injuries he received during his career as a boxer.)

Parkinson's Disease and Parkinsonism are often treated with L-dopa. Levadopa is metabolized to dopamine, a neurotransmitter, during passage through the blood-brain barrier (Fitz Gerald, 1996).

The symptoms of Parkinson's disease include:

1. **Pill-rolling tremor** - patients move their thumbs and forefingers together as if rolling a small pill between them. This movement occurs at rest, but subsides during voluntary movement and sleep.

2. **Masked facies** - Parkinson's patients usually display little facial expression, so their faces are described as "mask-like."

3. **Festinating movements** - Movements become more rapid and less coordinated over time. Festinations may affect both gait and speech. For example, a Parkinson's patient may begin to walk successfully but, as he continues, his steps will become faster and increasingly uncontrolled. As a patient continues to talk, his speech will become increasingly unintelligible.
4. **Rigidity** - If the limbs of a Parkinson's patient are moved passively, the muscles will often contract involuntarily, causing rigidity. This rigidity may be constant or intermittent. Intermittent rigidity is called **cogwheel rigidity**.

5. **Bradykinesia** - This is the "reduced speed of movement of a muscle through its range" (Love & Webb, 1992, p. 146).

6. **Hypokinesia** - This is "the reduced amplitude of muscle movement" (Love & Webb, 1992, p. 146).

7. **Micrographia** - Is the "tendency for handwriting to be very small in the height of the letters and to get progressively smaller as the person continues to write" (Love and Webb, 1992, p. 146).

8. **Dementia** - Between 30 and 40 percent of Parkinson's patients suffer from dementia.

**Ataxic Dysarthria**

Ataxic dysarthria results from cerebellar lesions. Such damage could be caused by stroke, trauma or by neurological disorders like muscular sclerosis. The cerebellum plays an important role in the coordination of motor movement due to its integration of sensory and motor information. Due to its connections with the vestibular system, it also affects equilibrium. Therefore, cerebellar lesions result in incoordination and the disruption of smooth movements (tremor) as well as problems with gait and balance. After cerebellar damage, patients are slow to initiate movements. Their movements often under or overshoot targets. For example, if an ataxic patient is trying to pick up a glass of water, she may either fail to move far enough toward the glass or may reach beyond it.
Unit 13. Types of Dysarthria: Lower Motor Neuron Damage

Lower Motor Neuron Damage

The Homunculus

There are two types of lower motor neuron lesions: bulbar lesions and peripheral lesions.

Bulbar lesions occur on the nuclei of cranial nerves, in the brain stem. These lesions cause a condition known as bulbar palsy, which is likely to result in a very severe form of dysarthria. As the cranial nerves lie very close to one another in the brainstem, a lesion will usually damage more than one pair of nuclei. Thus, a number of muscles may be affected bilaterally. Bulbar lesions can be caused by stroke, trauma or disease.

Peripheral lesions damage the axons of the cranial nerves after they have left the neuraxis and may cause what is known as a peripheral dysarthria. Typically this condition is not as serious as bulbar palsy since muscles on only one side of the body will be denervated. Also, it is possible that only the axon of one cranial nerve will be damaged. In both bulbar palsy and peripheral dysarthria, the muscles on only one side of the body will be affected cranial nerves cease to receive any input from the central nervous system, causing them to become flaccid or limp.

Myasthenia Gravis can also cause a lower motor neuron dysarthria, because the muscles most affected are those supplied by cranial nerves. This disease impairs the ability of nerve fibers to synapse with the muscles by reducing the number of acetylcholine (ACh) receptors at the neuromuscular junction. It causes weakness in muscles throughout the body, especially those involved in speech and swallowing. This weakness is progressive. In the morning or after rest, the patient's speech may sound clear. However, as he becomes fatigued, the slurring of his speech will increase. According to Fitz Gerald (1996), it is caused by the immune system producing antibodies to the ACh receptor. Dworkin (1991, p. 189) recommends the following sequence of articulation sub-system treatment: for flaccid dysarthria:

1. Lingual, labial, and/or mandibular musculature strengthening.
2. Lingual, labial, and/or mandibular force physiology training.
3. Phonetic stimulation in various contexts.

Mixed Dysarthrias

Mixed dysarthrias occur when both upper and lower motor neurons are damaged. This typically occurs as the result of disease. Amyotrophic lateral sclerosis (Lou Gehrig's Disease) is the disease that most frequently caused mixed forms of dysarthria. This condition, which usually first affects people when they are in their fifties, causes the degeneration of both upper and lower motor neurons. Its etiology is unknown. It has a very poor prognosis.

The Extrapyramidal Tract
The Pyramidal Tract
Unit 14. Dysarthria: Characteristics, Prognosis, Remediation

**Characteristics Of Dysarthria in Communication**

All types of dysarthria affect the articulation of consonants, causing the slurring of speech. In very severe cases, vowels may also be distorted. Intelligibility varies greatly depending on the extent of neurological damage. Hypernasality is frequently present as are problems with respiration, phonation and resonance.

**Spastic Dysarthria**

You will recall that it is due to damage to the pyramidal tract. This direct, mono-synaptic tract is predominantly facilitatory. That means its cell bodies send impulses that result in movement, along its axons. The principle result of damage is difficulty with fine motor movements. This is due to exaggerated stretch reflexes, resulting in increased muscle tone and incoordination. All of the cranial nerves except VII and XII are stimulated bilaterally by the cortico bulbar tracts. Bi-lateral lesions on those tracts result in a much more severe dysarthria than unilateral lesions. However, unilateral lesions on a cortico-bulbar tract will affect cranial nerves VII (jaw movement) and XII (tongue movement). Since the other cranial nerves recieve signals from both cortico-bulbar tracts, the muscles they innervate are barely affected. Thus, one would not expect problems with the lips, cheeks, throat, velum, or larynx. With bi-lateral cortico-bulbar lesions all of the above, and more, would be affected.

**Phonation**

Vocal quality is harsh. Sometimes the voice of a patient with spastic dysarthria is described as strained or strangled (Duffy, 1995). Pitch is low, with pitch breaks occurring in some cases.

**Resonance**

Hypernasality typically occurs, but is usually not severe enough to cause nasal emission.

**Prosody**

Bursts of loudness are sometimes noted in the speech of patients with spastic dysarthria.

**Articulation**

According to Duffy (1995) range of movement, tongue strength, speech rate and voice onset time for stops are reduced. There is an increase in phoneme to phoneme transitions, in syllable and word duration, and in voicing of voiceless stops.

**Hyperkinetic Dysarthria**
As described above, hyperkinetic dysarthria is usually thought to be due to lesions of the basal ganglia. Its predominant symptoms are associated with involuntary movement. There may be unilateral or bilateral damage.

**Phonation**

As with spastic dysarthria, vocal quality may be described as harsh, strained, or strangled. Voice stoppages may occur in dysarthria associated with dystonia.

**Resonance**

Hypernasality is common.

**Speech**

When voluntary speech movements are made there is often a super-imposition of involuntary movements. Speech can range from total lack of intelligibility to a mild problem. There are many syndromes, several of which are described above, associated with this problem.

**Hypokinetic Dysarthria**

Associated mainly with Parkinson's disease, it is due to a lesion in the substantia nigra. However, it can also result from anti-psychotic medications, frequent blows to the head and other etiologies described above. According to Ferrand and Bloom, 1997, in order for normal muscle movement to occur dopaminergic and cholinergic (ACh) pathways must be in balance.

**Phonation**

Hoarseness is common in Parkinson's patients. Also, low volume frequently reduces intelligibility.

**Resonance**

Hypernasality may occur

**Prosody**

Monopitch and monoloudness may occur. Pallilalia, or the compulsive repetition of syllables, is sometimes present.

The sound production pattern of patients with Parkinson's disease is sometimes described as articulatory undershoot.

**Articulation**

Bradykinesia associated with Parkinson's disease causes difficulty in the initiation of voluntary speech. This can result in delay in starting to talk as well as very slow speech. According to Duffy (1995), there may be freezing of movement during speech. Additionally, Parkinson's patient have reduced loudness, imprecise consonant production, reduced pitch variability and festinating speech. The latter can result in extremely fast speech together with short rushes of speech (Ferrand and Bloom, 1997).

**Ataxic Dysarthria**
This disorder is due to damage to the cerebellar control circuit. According to Duffy (1995), it can affect respiration, phonation, resonance and articulation, BUT its characteristics are most pronounced in articulation and prosody.

**Phonation**

Vocal quality may be harsh. As loudness may vary excessively, and increased effort is evident, ataxic speech is sometimes described as explosive speech.

**Resonance**

Hypernasality is not common, but may occur.

**Prosody**

Patients with ataxic dysarthria tend to place equal and excessive stress on all syllables spoken. The term scanning speech has been used in the past to describe this prosodic pattern. (The name was originally used by Charcot in reference to the speech of a patient who spoke very slowly and paused after each syllable). The label is no longer applied to ataxic dysarthria in order to avoid confusion as it has been used to describe a variety of different speech problems.

**Articulation**

All dysarthric speech could be described as slurred. However, due to the incoordination caused by cerebellar lesions, ataxic speech sounds especially slurred. Patients sound almost inebriated. Gait is affected in the same way. According to Duffy (1995), it is a breakdown in motor organization and control. The result is slowness and inaccuracy in range, force, timing, and direction of articulatory movements.

**Flaccid Dysarthria**

This results from damage to the lower motor neurons (cranial nerves) involved in speech.

**Phonation**

If CN X is damaged, voice will be affected as this nerve innervates the intrinsic musculature of the larynx. Occasionally, only one vocal fold is paralyzed. If the fold is paralyzed in an adducted position, the voice will sound harsh and have low volume. If the fold is paralyzed in the abducted position, this will cause breathiness along with a reduction in loudness. Bilateral vocal fold paralysis is more common than is unilateral paralysis. When the folds are in an adducted position, the voice is breathy and inspiratory stridor may be noted. (Of course, paralysis of both vocal folds in the abducted position would constitute a medical emergency; the airway would be closed off).

**Resonance**

Hypernasality will occur if the muscles involved in velar elevation have been affected. Frequently, velar movement is reduced sufficiently to cause nasal emission.

**Prosody**

Monopitch and monoloudness may both result from vocal fold paralysis.

**Associated Characteristics**
Muscles affected by flaccid paralysis may begin to atrophy or lose mass over time. Also, lack of innervation may cause fasciculations or twitching of muscle fibers. These movements are especially visible in the tongue; its surface may dimple as if worms were moving beneath its skin.

Unilateral paralysis of the oral structures may be noted. The affected side of the mouth may sag, causing drooling. The jaw will deviate toward the weakened side while the tongue veers toward its stronger side.

Articulation

According to Darley, Aronson, and Brown (1969) ataxic speech is characterized by articulatory inaccuracy, prosodic excess, and phonatory-prosodic insufficiency. This includes imprecise consonants, irregular articulatory breakdowns, vowel distortion, slow rate, prolonged phonemes and intervals, vocal harshness and monopitch and monoloudness.

Mixed Dysarthria

Characteristics will vary depending on whether the upper or lower motor neurons remain most intact. For example, if upper motor neurons are most damaged initially, the voice will sound harsh. However, if lower motor neurons are most affected, the voice will sound breathy.

Prognosis

According to Netsell (1984, as cited in Halperen, 1986), a number of factors influence the prognosis of dysarthric patients. Among them are:

Neurological status and history

Dysarthrias caused by bilateral subcortical lesions, brain stem lesions or degenerative diseases like ALS have the poorest prognosis.

Age

In general, the younger the patient, the better the prognosis. Children are especially likely to have a good outcome, because of neural plasticity.

Treatment Effects

Patients who receive treatment, especially coordinated speech, medical, physical and behavioral treatment have a better outcome than those who do not.

Personality and Intelligence

Patients who were purposeful and optimistic before injury have a better prognosis than those who were not. Those who kept their minds “active” have a better prognosis than do those who did not.

Support Systems

Obviously, patients who receive support form family members and/or friends have a more favorable prognosis than those who don’t.
Dysarthria is treated differently depending on its severity. Patients with a mild or moderate form of the disorder can be taught to use strategies that make their speech more intelligible. Before compensation is attempted every effort should be made to improve physiological support for articulation, resonance and respiration. Patients with mild to moderate dysarthria will be able to continue to use speech as their primary mode of communication. Patients whose dysarthria is more severe, however, may have to learn to use alternative forms of communication.

Remediation of Severe Dysarthria

Dworkin (1991 p.188) recommends the following sequence of treatments for spastic dysarthria that with some cautions may be used with other types such as hyperkinetic, hypokinetic and flaccid. For hyperkinetic and ataxic dysarthrias, Dworkin feels that in most cases muscular tone reduction and muscular strengthening exercises may not be necessary. For flaccid dysarthria muscular tone reduction would not be required:

1. Lingual, labial, and mandibular musculature tone reduction.
2. Lingual, labial, and mandibular musculature strengthening.
3. Lingual, labial, and mandibular force physiology training.
4. Phonetic stimulation in various contexts.

According to Dworkin, 1991, abnormal force physiology or force dyscontrol of the articulators can be seen in all types of dysarthria. These muscle forcing abnormalities affect articulation until the patient can learn to reorganize muscle forces and movements. When tone reduction or strengthening exercises are needed, Dworkin states that these treatments should be given sequentially prior to force physiology training. He further recommends that phonetic stimulation be given next followed by motor speech planning if apraxia is also present. Dworkin (1991) uses acrylic or putty blocks to inhibit disruptive hypertonic movements such as jaw elevation when the tongue is elevated. This therapy is quite involved, expensive, and time consuming. For a detailed description of his approaches to remediation refer to his book in the readings and references section.

Adaptation of the Bobath Method in Remediation of Dysarthria

Crickmay (1966) adapted the Bobath method of physical therapy for the remediation of dysarthria in children with cerebral palsy. Since cerebral palsy results from upper neural lesion problem it can present with any of the dysarthrias mentioned above, except flaccid which is caused by lesions on the cell bodies or axons of cranial nerves. The methods Crickmay advocates make sense to me. It is as timely now as when it was first published. The Bobath method has three main stages. In the first stage the patient's abnormal and pathological reflexes are inhibited. In the second stage more developmentally mature movements are facilitated. In the third stage movements are put under the voluntary control of the patient. It is important that the speech therapist have guidance and assistance from a physical therapist before he/she initiates remediation.

In order to normalize muscle tone the patient is placed in a reflex inhibiting posture (RIP) which he/she has become accustomed to while in physical therapy. Two postures that tend to have normalizing effects are the prone (extended spine and flexed elbows-lying on your stomach while leaning on your elbows), and the supine (hips and knees extended, shoulders flexed-held by therapist, and head back-chin held. In inhibiting abnormal speech reflexes the therapist should go from gross to fine. Head control, the ability to lift and turn the head is a prerequisite for speech. Crickmay suggests that the individual be in a supine position with legs and arms at his/her sides and hips and legs flexed. This is a good time to work on desensitize the face/articulators. Remember we are talking about desensitizing the speech mechanism because when you manipulate the tongue and lips the child may react with spasms. Crickmay suggests that you help the child build up tolerance by holding him/her in the RIP while gently and carefully touching and moving the hypersensitive face. Since the mouth is the most sensitive he/she should start with facial areas furthest from the mouth and work in towards the mouth. The patient will resist and try to break out of the RIP. He/she should be held gently but firmly so that he/she can build up a tolerance and permit he speech therapist to manipulate the speech mechanism. The time taken for this to happen is quite variable-from a few days to several
The patient learns to lie quietly in an RIP keeping the face free of abnormal movement. When facial grimacing occurs it can be usually controlled by the fingertips. Those with spasticity often have an open mouth, lips drawn back grin. Crickmay suggests flexing the head forward to inhibit the extensor spasm, and close the patients mouth.

To control drooling, Crickmay recommends teaching the patient to keep the teeth closed and the tongue tip up against the alveolar ridge as he/she swallows. The clinician then inhibits any extraneous movements of cheeks and lips.

To teach a normal mouth position, Crickmay suggests the following:

Help the patient close his/her teeth in a normal jaw position. Place one hand under the patient's chin and hold that position for him/her. Use the other hand to stroke the patients lips and cheeks forward in order to relax the face. Chin pressure must be released gradually in order to permit the patient to assume control and to experience a closed mouth. Help the patient gradually increase the time the mouth remains closed.

To reinforce the closed mouth position negative practise may be used. The clinician asks the patient to go back the original open mouth position and then to deliberately assume the new closed mouth one. Repeated practise will enable the patient to feel the difference between positions. Using a mirror during practise will provide visual reinforcement.

In order to inhibit the infantile sucking reflex, Crickmay recommends the following:

Put the patient in an RIP. Gently stimulate the lips by touching them with a straw or finger. Use the other hand to prevent them from moving into a sucking position. The clinician will have to control the patient's lips by keeping them in a relaxed position despite the stimulation. Finally the therapist should help the child assume control. This can be done by the clinician removing his/her hand occasionally-gradually increasing the length of time until the patient can inhibit the reflex without assistance.

To facilitate the chewing reflex (a normal reflex, which with with sucking and swallowing is prerequisite for speech) the clinician can give hard liquorice or chocolate (absent dietary or swallowing restrictions). It should be pressed against the hard palate in order to stimulate chewing. Rubbing the gums and teeth, front, back and sides, in a rotary motion with a finger will also help.

To facilitate independent tongue movement, Crickmay suggests holding the patient's jaws apart while having him/her raise the tongue to the alveolar ridge. Next, encourage him/her to produce /t/, /d/, /l/, and /n/ sounds. Make sure the jaw doesn't become fixed. It should be immobilized, but given some freedom of movement. Finally, permit the patient to take control.

Differentiation of lip movements may be necessary for many patients. The techniques described above can be used with patients who have difficulty differentiating lip, tongue, and jaw movements. That is they cannot make the /r/ sound without moving both lips and tongue or are unable to make /t/, /l/, or /n/ sounds without also moving the jaw.

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**Remediation of Mild to Moderate Dysarthria**

**Compensatory Strategies**

According to Rosenbeck and La Pointe (1978), the goal of therapy for dysarthria is to help patients achieve compensated intelligibility. In other words, patients must learn techniques that help them to
make good use of their remaining physiological capacity to produce understandable speech.

Because dysarthria can impair respiration, phonation, resonance and prosody, therapy will often include compensatory strategies which address these aspects of speech production as well as articulation.

1. The most effective way for most dysarthrics to make themselves more intelligible is to **reduce the rate of their speech** and **produce syllables one by one**. Speaking this way will disrupt intonation patterns and may make the person sound "robot-like." However, it is the best way to maximize the clarity of dysarthric speech.

Many patients have difficulty learning to speak slowly. A **pacing board** may be helpful for such patients. Pacing boards are divided into sections and the patient must tap one section every time he pronounces a syllable. As the fingers cannot move nearly as rapidly as the articulators, this should slow the rate of speech enough to improve intelligibility. After reduced rate has been established using the board, the client can begin to count syllables on his fingers. Eventually, he should be able to maintain the proper rate without counting syllables at all. A device called a **graduated stick** may be used instead of a pacing board. Graduated sticks have bumps on them at regular intervals and the client must touch one bump every time he says a syllable. Metronomes can also be used to slow speech rate. Patients are taught to pronounce one syllable per "tick" on the metronome.

The use of a pacing board, graduated stick or metronome to slow rate may be referred to as intrasystemic reorganization. Intrasystemic reorganization for speech remediation is described by La Pointe and Rosenbeck as introducing a non speech function into the impaired act that is not normally used in the impaired act in order to facilitate speech. This new function is called an organizer. According to Duffy, 1995 using tongue protrusion to facilitate the production of interdental sounds is an example of using a lower level function for a higher level purpose.

2. Dysarthric patients should also try to **emphasize all syllables** as they speak. This will reduce vowel distortion.

3. Patients should also use **greater excursion** of the mandible, tongue and lips to improve production of both consonants and vowels. The exaggeration of jaw and tongue movements is the most crucial aspect of this strategy; it may be too difficult for some patients to increase the amplitude of their tongue movements.

4. **Consonant exaggeration** is another compensatory strategy that improves the intelligibility of dysarthric speech. Teach the patient to over-articulate in order to emphasize the sounds that s/he is slighting. (Medial and final consonants are typically most slighted in running speech.)

5. If a patient has difficulty with a particular type of phoneme, the clinician may have to teach him to use **compensatory placement** to produce those sounds. For example, if the patient has trouble with tongue tip sounds like /t/, /d/, /n/, /s/ and /z/, he/she could learn to make them with the blade rather than the tip of the tongue. If the patient can no longer put his lips together to make bilabials, he could learn to use his teeth to contact the lips.

6. **Monitoring techniques** are often used to work with patients who have problems with either reduced or excessive loudness. For example, the visipitch could be used to help Parkinson's patients who speak too softly or patients with spastic dysarthria who have bursts of very loud speech.

7. **Reducing phrase length** is another way to increase the loudness of a dysarthric's speech. When a patient with hypokinetic dysarthria stops to take a breath and rest for an instant between every few words, their vocal volume usually increases. (Slowing the rate of speech, which will be the main focus of therapy for dysarthria, may automatically reduce phrase length and improve the patient's loudness.)

8. The **yawn-sigh technique** for easy onset of voice, the use of a **breathy voice** and Froeschel's chewing method of focusing energy in the oral cavity may all be used to reduce the strained-strangled vocal quality that occurs in spastic dysarthria. Patients with this kind of voice problem are often using hard glottal attack. In this case, any technique used to ameliorate this problem will help the patient.

9. Some dysarthrics, especially those with the hypokinetic form of the disorder, may have an excessively breathy voice. **Pushing exercises** may help a patient who has this problem by facilitating glottal closure.
10. Many dysarthrics speak at an abnormally low pitch. If they continue to do so for a sufficient length of time, they may develop a functional voice disorder like nodules. For this reason, it may be necessary to address the patient's voice problem although there is usually a trade-off between improving pitch/intonation and improving intelligibility.

Prosthetic Devices

If a Parkinson's patient with hypokinetic dysarthria cannot learn to speak more loudly through the use of monitoring techniques or the use of short phrases, he or she might be provided with an electro larynx or a computer hardware/software as a way of increasing vocal volume.

Usually, therapy does not effectively reduce the hyper nasality that accompanies most types of dysarthria. Patients can be given blowing exercises or exercises that contrast nasal sounds with oral sounds (eg., /n/ vs. /a/). Perceptual training, such as having the client listen to a tape recording of his voice, is also used. Froeshel's chewing method may help to some extent by concentrating energy in the oral cavity. But, if the velum is paralyzed, none of these techniques will do anything to alleviate the problem.

If hypernasality is severe, the patient should be referred to an ENT to explore the possibility of pharyngeal flap surgery or the use of an obturator and speech bulb prosthesis. (Although these were developed as treatments for cleft palate, they could also be used with a dysarthric patient.)

Isometric Exercises

Oral motor exercises may be used in therapy with dysarthric patients. Programs must also include speech exercises as the movements involved in speech are different from other types of oral-motor movements.

Feedback

In addition to the use of the visipitch to monitor loudness levels, other kinds of feedback are employed in therapy with dysarthric patients.

1. Tactile and kinesthetic feedback can be used to teach compensatory articulation strategies.
2. Electromyographic (EMG) feedback can be used to reduce the hypertonicity of muscles.
3. Delayed auditory feedback (DAF) has been used successfully with Parkinson's patients.

Rosenbeck, et al. (1973), developed an eight step continuum for treating apraxia of speech. I have modified this approach for use with dysarthric patients:

Step 1

Integral stimulation-the therapist asks the patient to look and listen AND imitate. (watch me and do the same).

Step 2

Same as 1 except the patient is asked to delay the response; then the therapists silently mimes the response while the patient is producing the target stimulus.
Step 3

Integral stimulation followed by imitation WITHOUT cues—miming or otherwise.

Step 4

Integral stimulation with several successive productions without simultaneous cues or intervening stimuli

Step 5

Printed stimuli are presented by the therapist without auditory or visual cues followed by patients production while looking at the written stimuli.

Step 6

Written stimuli, with delayed production after removal of the written stimuli.

Step 7

The therapist elicits a response by asking a question.

Step 8.

Role playing is used to elicit responses.
Unit 15. Dysarthria vs. Apraxia: A Comparison

Etiology

**Dysarthria**

Dysarthric errors result from a disruption of muscular control due to lesions of either the central or peripheral nervous systems. In this way, the transmission of messages controlling the motor movements for speech is interrupted. Because it involves problems with the transfer of information from the nervous system to the muscles, dysarthria is classified as a neuromotor disorder.

**Central Nervous System Lesions**

Damage to the pyramidal tract causes spastic dysarthria. Lesions of the substantia nigra cause hypokinetic dysarthria. Disruption of feedback loops involving the cerebellum cause dysarthria.

**Peripheral Nervous System Lesions**

Finally, damage to any part of the peripheral nervous system serving the muscles of speech causes flaccid dysarthria.

**Apraxia**

Apraxia results from an impaired ability to generate the motor programs for speech movements rather than from the disordered transmission of controlling messages to the speech musculature. Apraxia is a planning/programming problem, not a movement problem like dysarthria.

Apraxia occurs following damage to Broca's Area, or Brodmann's area 44, which is located on the third gyrus of the left frontal lobe. Thus, apraxia is always the result of a central nervous system lesion. It is a cortical problem, not a motor impulse transmission problem like dysarthria.

Type Of Errors

**Dysarthria**

In dysarthria, errors are consistent and predictable.

There are no islands of clear speech; no matter what the speaking task or materials used, the patient will exhibit the same amount and types of errors.

Errors are mainly distortions and omissions. Distortions are the most common type of error in dysarthria.

**Apraxia**
In apraxia, errors are inconsistent and unpredictable. Different error patterns occur in spontaneous speech versus repetition. Patients’ spontaneous speech contains fewer errors than does his/her speech in repetition tasks.

There are islands of clear speech; when producing over-learned material or material that has become automatic, the patient will speak clearly.

Substitutions are the most common type of error, with others normally being approximations of the targeted phoneme.

Other types of errors found in apraxic speech, listed from most to least common, include:

- Repetitions
- Additions
- Transpositions
- Prolongations
- Omissions
- Distortions

Errors are often perseveratory or anticipatory in nature. As in stuttering, the anticipation of errors causes dysfluent speech.

The speech of apraxics is full of groping, trial and error types of articulatory movements. This is probably related to the anticipation of errors.

**Speech Sounds Affected**

**Dysarthria**

Consonants are consistently imprecise, with the production of final and initial consonants being equally impaired. Vowels are not affected as much although, due to problems with tongue movement, they may sound too much alike.

**Apraxia**

For an apraxic, vowels are easier to produce than consonants. Single consonants are easier than blends. As in stuttering, final consonants are easier than those in the initial position. This may occur because initial consonants are affected by anticipatory errors. Also, perhaps once an apraxic gets speech started with the production of a vowel, production continues in a more automatic fashion. Fricative and affricates are the most difficult phonemes for apraxics to produce. (These sounds require very complex articulatory movements.)

**Aspects Of Speech Affected**

**Dysarthria**

All aspects of speech, including articulation, phonation, resonance, prosody, rate and respiration, may be affected by dysarthria. Dysphagia frequently accompanies dysarthria.

**Apraxia**

Apraxia is mainly a disorder of articulation. Some prosodic problems may occur as a result of the hesitations caused by the apraxic speaker's anticipation of errors. However, problems with voice, resonance, etc., are not symptomatic of this disorder.
**Related Problems**

**Dysarthria**

Changes in muscle tone may accompany dysarthria. The movements of the soft palate, lips, tongue and jaw may be impaired not only during speech, but also in the context of vegetative functions. As such, changes may affect the oral and pharyngeal stages of the swallow, dysarthria and dysphagia often co-occur.

Diadochokinesis will be slow. However, it will be normal within the limitations of the neuromuscular disorder; the patient may distort or omit phonemes but syllables will be produced in the correct order.

**Apraxia**

As lesions of Broca's area do not cause changes in muscle tone, apraxia may occur without such symptoms. Therefore, apraxia of speech may occur without concomitant swallowing problems. The movement of the velum, lips, tongue and jaw will only be impaired during speech. Of course, a stroke may damage Broca's area and motor tracts or other areas involved in swallowing simultaneously. In such a case, dysphagia and apraxia would be seen in the same patient.

Diadochokinesis will be slow and abnormal; syllables may be produced out of order.

**Effects Of Utterance Complexity**

**Dysarthria**

Utterance complexity does not directly affect the degree of imprecision present in a dysarthric patient's speech. Such a patient will produce single syllable and multi-syllabic words with approximately the same amount of distortion.

**Apraxia**

Increases in utterance complexity cause increases in the complexity of apraxic symptoms. For example, it is much easier for a patient with apraxia to produce single syllable versus multisyllable words and sentences.

**Effects Of Speech Rate**

**Dysarthria**

As the rate of a dysarthric's speech increases, the intelligibility of that person's speech will decrease proportionally. In order to improve intelligibility, the dysarthric must learn to slow rate by articulating complex words syllable by syllable.

**Apraxia**

As the rate of an apraxic's speech increases, the intelligibility of that person's speech may actually increase.

Noting the effect of increased rate on speech intelligibility is one way to differentiate between dysarthria and apraxia.

**Therapy**
**Dysarthria**

Therapy for dysarthria is strictly compensatory. If motor pathways are damaged, they cannot be repaired. The dysarthric patient must learn to use techniques that increase the intelligibility of his/her speech.

**Apraxia**

Although some recommend compensatory techniques most apraxics benefit more from therapy that focuses on retraining more than on compensation. Melodic Intonation Therapy (Sparks and Holland, 1976) is one technique that is frequently used with apraxic patients. It is believe that, through the use of melody and rhythm, this method stimulates the creation of more neuronal connections in the right hemisphere. This may allow the “potential Broca's area” located in the right hemisphere to begin generating motor programs that control speech production. Duffy (1995) describes the type of patient for whom MIT might help best.