Tutorial

Expanding our expertise

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Expanding Our Expertise

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SECTION I. *State of the Art: Oral Myology – Redefined*

Several years ago, the International Association of Orofacial Myology – then known as the International Association of Oral Myology – faced the identity crisis: What should we call ourselves? The members of our association were from divergent professional backgrounds, primarily speech pathology and dentistry. Our special training and expertise involved the positioning and movements of the oral-facial musculature at rest and during swallowing and sometimes during speech. There was no consensus as to a label for what we were treating. Historically, it had been called tongue thrust, deviate swallow, deviant swallow, reverse swallow, infantile swallow, or total oral facial muscle imbalance. The persons treating the disorder were referred to as tongue thrust therapists, myofunctional therapists, or by our primary designation as speech pathologists or dental hygienists. Our association chose to call ourselves ORAL MYOLOGISTS, and later amended the label to OROFACIAL MYOLOGISTS.

By definition, “oral” means “of the mouth”, “facial” means “of the face”, “myo” means “muscles”, “ology” means “the study or science of”. Therefore, orofacial myologist means “one who has studied the muscles of the mouth and face.” We assumed this terminology to describe our role in the treatment of patients whose oral-facial habits may have a detrimental effect on the dentition.

I feel that the services we can provide in conjunction with the dentist and orthodontist are valuable indeed. I feel that we have proven that time and again over the years – and that is why we have survived. There has been a tremendous amount of controversy, we have had to fight the battles of skepticism and criticism – and we have survived. We have been introspective. We have taken a hard look at ourselves, our diagnostic procedures, our treatment programs and we have continually upgraded our professionalism. We have built a good foundation and earned respect in our communities.

Now, it is time to look outward, to make some decisions about “Where do we go from here?” and “What can we do to expand our horizons?”

I think that there are some very logical next steps that we as orofacial myologists should consider. I personally enjoy very much the work I do with tongue thrust, but there is so much more to life than tongue thrust. With our special skills, our knowledge of the oral-facial musculature, our experience in designing and implementing specific exercises for specific oral-facial muscle movements and positions, and our understanding of the neurological and physiological aspects of the swallowing mechanism, it seems appropriate to me that we should become clinically involved with several other populations, specifically the neurologically involved. Those of us with speech pathology backgrounds would need very little additional training to become experts in working with neurologically handicapped individuals. For example, one of the primary concerns with regard to cerebral palsied children is feeding. We could utilize our skills to remediate the sucking, chewing, swallowing problems present in the cerebral palsy population. Consider how many of the exercises we use in tongue thrust therapy that could be used successfully with cerebral palsied patients to assist them in overcoming feeding difficulties.

Likewise, we are uniquely qualified to treat the motor speech problems of dysarthria and apraxia which result from cerebrovascular incidents and other neurological pathologies. We can become involved in the diagnosis and treatment of dysphagia, a disturbance in swallowing that is addressed in this series of articles. I find that many of the exercises which were developed for tongue thrust are quite appropriate and useful for apraxic, dysarthric, and dysphagic patients.

I believe that if we are going to call ourselves “orofacial myologists,” and truly by experts in the study of the oral-facial musculature, then it is incumbent on us to get whatever additional training we need, by attending workshops and reading the current information in journals and texts, to expand our expertise and equip us to work with the neurologically involved population. I would like to see the International Association of Orofacial Myology take an active role in this endeavor.

SECTION II.

State of the Art: Speech Pathology – Redefined

One of the first things I learned as a student of speech pathology was the dichotomy between “functional” and “organic” speech disorders. The organic disorders were those which were caused by a medically diagnosed malady, such as cleft palate, vocal nodules, restricted frenum, cerebrovascular accident, cerebral palsy, and similar conditions. All other speech and voice disorders were considered to be functional, or caused by faulty learning, misuse of the vocal mechanism, emotional problems, lack of stimulation and/or motivation and a number of other “non-medical” causes. At the time, I accepted the dichotomy because it seemed reasonable and because students learn what they are taught. I did not have the clinical expertise to question. Later, in my work with patients in a full-service rehabilitation center and in private practice, the “functional-organic” dichotomy bothered me more and more. Just because an organic etiology is not readily apparent does not mean that one does not exist.

At the 1981 IAOM Eastern Rap Session, Dr. James Dworkin shared with us some preliminary data from a study in Kentucky public schools. Students and faculty at the University of Kentucky did a survey of children enrolled in public school speech therapy. The public school therapists consistently reported that 90% of their caseload were “functional articulation cases.” Researchers from the university examined these children and found that a large number had neurologically based speech problems.

Many of the children labelled “functional” do indeed have subtle or not-so-subtle organic/neurological bases

for the speech disorder. We as clinicians do not have enough good examination procedures or test instruments to establish organicity; or we don't have sufficient skills or knowledge to use the procedures which are available to correctly identify and diagnose organicity. If there is not an apparent organic condition, such as cleft palate, cerebral palsy, or a syndrome, then the child is labelled “functional.”

The resulting large number of “functional” cases has made our work more within the realm of teaching in an educational sense than of treating in a medical sense. I feel that this has hurt our profession in terms of gaining respect as a medical ancillary. We can provide valuable information to help physicians diagnose problems they might have missed by working as a team and by providing input and expertise from our background.

We can also better serve our patients by doing a comprehensive diagnosis, discovering neurological or other organic causes if they exist, because this will assist us in planning more appropriate treatment. Identifying possible organicity will also, not incidentally, assist the patient in getting reimbursement from insurance companies for our services.

My personal bias is that there are very few functional articulation cases. The responsibility is ours to sharpen our skills and develop new tools to enable us to establish organicity where it exists. Any less would be a disservice to ourselves and our patients.

SECTION III.

Dysphagia

“Dysphagia” – derived from the Greek “dys” meaning difficult or disordered and “phagein” meaning to eat – is currently an area of interest to a number of physicians, nurses, speech pathologists, physical therapists, occupational therapists, and nutritionists. Since orofacial myologists are knowledgeable regarding the swallowing process and musculature, dysphagia might be an area for further study and program development by orofacial myologists.

Dysphagia is a term used to denote problems with swallowing. The currently accepted definition of dysphagia is the inability to consume enough liquid and nourishment orally to sustain life, without increasing the possibility of certain medical complications. The difficulty may be present at the site of any of the three

phases of the swallowing process: oral, pharyngeal, or esophageal. It may be psychogenic, pathologic, or neurogenic. These and other considerations of dysphagia will be discussed in this section.

Swallowing is an extremely complex sensory and motor act. The swallowing center is located in the medulla oblongata of the brainstem. This center receives and processes sensory input from the 5th, 9th, 10th, and 12th cranial nerves and the first three cervical nerve segments. When this center determines the need to swallow, it inhibits competing centers and assumes control of all motor neurons and related muscles necessary for swallowing. The swallowing center preempts higher brain functions such as speech and breathing. We cannot talk and swallow simultaneously. Likewise,

respiration is halted temporarily while swallowing is accomplished. The swallowing center coordinates the movements of the primary muscle groups involved: the 5th, 7th, 9th, 11th, and 12th cranial nerves and the first three cervical nerve segments. A number of pathological and neurological conditions can adversely affect the swallowing process, resulting in varying degrees of difficulty.

Classification of Dysphagic Conditions

1. Psychogenic swallowing disturbances. Psychogenic dysphagia is more literally a "behavioral disturbance" than a true swallowing disorder. The swallowing mechanism is quite functional; the patient is afraid to swallow. He exhibits a hysterical reaction to swallowing or simply refuses to eat. Almost without exception, there was a precipitating event or experience of choking which led the patient to believe he could not swallow. The patient must be medically evaluated very carefully to rule out any pathologic or neurogenic factors. If the dysphagia is truly psychogenic, the therapist should begin working with the patient to restore swallowing behavior. It has been my experience that these patients are usually handling saliva without any difficulty and fear only food and drink. By starting with something they can do, i.e. swallowing saliva, and working up a hierarchy of tiny, then progressively larger, sips of liquid (room temperature, not the ice water available in hospital rooms), the patient is able to regain his confidence in his ability to swallow and thus overcomes psychogenic dysphagia.

The fear of choking is great in some patients and cannot be overcome even by a confident, competent therapist or psychotherapist. A small percentage of psychogenic dysphagic patients will have to resort to an alternate method of feeding (I.V. solution or nasogastric tube). This author is presently working with a post-C.V.A. patient who uses a nasogastric tube. There is paresis of the tongue and lips, which classifies her as an oral phase neurogenic dysphagic. There is no drooling present and, about once a week, the patient eats a large serving of a favorite dessert with minimal oral problems, but the rest of the time, she gets five feedings a day of Ensure poured down her N-G tube. The neurogenic problems could be overcome but the psychogenic problems cannot.

2. Pathologic swallowing disturbances. Pathologic dysphagia is the result of a structural disturbance at some point along the swallowing pathway. The obstruction prevents the normal passage of the bolus from the oral cavity to the stomach. These patients often complain of painful swallowing and can usually identify fairly accurately where the food "gets stuck". Examples of pathologic dysphagic conditions are carcinoma, abscess, tumor, hernia, esophageal web and similar medical conditions, which usually are evident in x-rays, cine-radiography and/or fluoroscopy. The diagnosis and treatment of pathologic dysphagia are the responsibility of the radiologist and the surgeon. Surgery may be required to remove the obstruction; radiation, chemo-

therapy or medication may shrink the obstruction; or an alternate method of feeding (I.V., N-G tube, etc.) may circumvent the obstruction.

3. Neurogenic swallowing disturbances. Neurological conditions which cause muscular weakness, slowness, incoordination, and/or paralysis frequently produce swallowing disorders. Examples of these conditions include muscular dystrophy, cerebrovascular accident, encephalitis, meningitis, Parkinson's, neurosurgery, brain tumor, cerebral palsy, polio, and Huntington's Chorea. The location and extent of the neurological damage determine the site and severity of the neurogenic dysphagia.

A. Oral Phase Neurogenic Dysphagia. Weakness, incoordination, or paralysis in the oral cavity disrupts the patient's ability to grind and shape the food in preparation of the bolus for entering the pharynx. Common problems observed include drooling, pocketing of food in the cheek or impacting it against the hard palate, inadequately chewing food, and choking or coughing. Almost without exception, dysarthria of speech is present to some extent. The patient may have reduced sensations of taste, temperature, touch and texture.

Therapeutic intervention is usually successful to some degree with these patients. They can benefit from exercises to increase muscle strength and control, particularly resistance exercises. Many of the tongue and lip exercises traditionally used in tongue thrust therapy, plus neuromuscular facilitation exercises, are helpful with oral phase neurogenic dysphagia. Mirror work is extremely important, both while doing tongue and lip exercises and while chewing and swallowing. Since the patient has decreased sensitivity in and around the oral cavity, he/she needs the visual feedback of looking in a mirror. If one side of the face is paralyzed, the patient may think he/she is sticking the tongue out straight when in fact it is deviating to one side. Likewise, the patient may be unaware of food pocketed in one cheek unless he uses his finger to retract the lips and sees the food in the mirror.

Assistive devices, such as a "Tommee Tippee cup" or a "push-off spoon", may be quite helpful to the patient who has difficulty forming an adequate lip seal. The occupational therapist has a wealth of information regarding assistive devices.

Food consistencies are also an important consideration. Liquids are difficult because they require considerable intraoral control to prevent them from drooling out the front of the mouth or draining back into the pharynx and larynx and precipitating coughing or choking. Textured foods such as meats are difficult to chew properly and to gather into a bolus. Semi-solid or pureed foods are the best consistency for the patient with oral phase neurogenic dysphagia.

B. Pharyngeal Phase Neurogenic Dysphagia. The most complex muscular movements of the swallowing act occur when the bolus enters the pharynx and is transported downward to the superior cricopharyngeal sphincter. During the pharyngeal phase of swallowing, the bolus must move rapidly and efficiently through the pharynx because respiration must be halted momentarily. The opening to the airway must be protected to prevent

aspiration into the lungs. The complexity and precision of the muscular movements involved render the pharyngeal phase particularly vulnerable to neurogenic impairments.

Pharyngeal phase neurogenic dysphagia is characterized primarily by coughing. Other problems often present include nasal regurgitation, aspiration, delayed initiation of swallow, and reduced or absent gag reflex.

Therapeutically, this type of problem is the most difficult to manage. The author has had some limited experience in working with patients in a hospital setting (where nurses are present or close by to suction the patient if necessary) but has not attempted treatment in a home or office setting without suctioning equipment or skilled nursing assistance at hand.

Patients with pharyngeal phase neurogenic dysphagia have the most difficulty with liquids and the least difficulty with semi-solid or pureed foods. Positioning is most helpful: the patient should be sitting up straight in a chair with the head bent slightly forward. The slight flexion at the neck assists the epiglottis and the base of the tongue in closing off the entrance to the larynx.

If the mechanism for protection of the airway is non-functioning and cannot be stimulated, the patient may have to use an alternate method of feeding. It is important that family members, hospital personnel, and therapists be trained in the Red Cross method of applying sharp blows to the back and in the Heimlich maneuver. Other than advising on food consistencies and positioning, there is little else to my knowledge, that the therapist can do for the pharyngeal neurogenic dysphagic patient.

C. Esophageal Phase Neurogenic Dysphagia. During the esophageal phase of swallowing, the bolus moves from the level of the superior cricopharyngeal sphincter to the stomach by peristaltic wave contraction. Esophageal phase neurogenic dysphagia is characterized by reversal or reflux of the food. The peristalsis is reversed, as by esophageal spasm, either during or after eating or drinking.

Clinically, these patients do better with liquids than with solids or semi-solids. Positioning the patient properly is of great importance. The patient should sit up as straight as possible in a chair during, and for an hour after, eating.

Summary

There are three classifications of dysphagic conditions: psychogenic, pathologic, and neurogenic.

In psychogenic dysphagia, the swallowing mechanism is intact but the patient has a fear of swallowing, usually precipitated by a choking experience. Treatment involves helping the patient to regain his confidence in his ability to swallow.

In pathologic dysphagia, there is an occlusion or obstruction at some point between the oral cavity and

the stomach. The patient feels that food or liquid "gets stuck". Pathologic conditions must be diagnosed and treated by the radiologist and the surgeon.

In neurogenic dysphagia, the problems in swallowing result from neurological damage such as that caused by stroke, cerebral palsy, and neuromuscular disease processes. Weakness, spasm, incoordination, and/or paralysis in the muscles of mastication and/or deglutition interfere with the oral, pharyngeal, or esophageal phases of swallowing, depending upon the location and extent of the neurological damage. Except in deteriorating disease processes, the patient can usually benefit from tongue and lip exercises, attention to food consistencies, and proper positioning.

Some dysphagic conditions can be treated successfully by a trained therapist; some are untreatable. Some must be medically or surgically corrected; some require mechanical feeding through a nasogastric tube or intravenous solutions. Correct diagnosis of the problem is a team effort.

References

Much of the information in this section is based on the booklet: *Dysphagia: An Assessment and Management Program for the Adult*, James R. Roueche, Sister Kenny Institute Rehabilitation Publication #706, 1980.

Other References

- Buckley, J.E., Addicks, C.L., and Maniglia, J., "Feeding Patients with Dysphagia", *Nursing Forum*, 15:69-85, 1976.
Dobie, R.A., "Rehabilitation of Swallowing Disorders," *Am. Family Physician*, 17:84-95, 1978.
Gallender, D., *Teaching Eating and Toileting Skills to the Multi-Handicapped in the School Setting*, Charles C. Thomas, Publisher.
Gaffney, T.W. and Campbell, R.P., "Feeding Techniques for Dysphagic Patients", *Am. J. Nursing*, 74:2194-2195, 1974.
Griffin, K.M., "Swallowing Training for Dysphagic Patients," *Arch. Phys. Med. Rehab.*, 55:467-470, 1974.
Hawkins, C.F., "Dysphagia," *Brit. Med. J.*, pp. 663-667, Dec., 1967.
Heimlich, H.J. and O'Connor, T.W., "Relearning the Swallowing Process," *Am. J. Oto. Rhino. Laryn.*, 88:795-797, 1979.
Hellemans, J., Pelemans, W., and Vantrapper, G., "Pharyngoesophageal Swallowing Disorders and the Pharyngoesophageal Sphincter," *Med. Clin. N. Am.*, 65:1149-1167, 1981.
Knott, M., "Neuromuscular Facilitation in the Child with Central Nervous System Deficit," *J. Am. Phys. Ther. A.*, 46:721-724, 1966.
Larsen, G.L., "Rehabilitating Dysphagia: Mechanical, Paralytic, Pseudo-bulbar," *J. Neurosurgical Nursing*, 8:14-17, 1976.
Larsen, G.L. "Rehabilitation for Dysphagia Paralytic," *J. Sp. Hear. Dis.*, 37:187-193, 1972.
Logemann, J.A., and Bytell, D.E., "Swallowing Disorders in Three Types of Head and Neck Surgical Patients," *Cancer*, 44: 1095-1105, 1979.
Meadows, J.C., "Dysphagia in Unilateral Cerebral Lesions," *J. Neuro. Neurosur. Psych.*, 36:853-860, 1973.
Mysak, E.D., "Dysarthria and Oropharyngeal Reflexology: A Review," *J. Sp. Hear. Dis.*, 28:252-260, 1963.
Nahum, A.M., Harris, J.P., and Davidson, T.M., "The Patient Who Aspirates - Diagnosis and Management," *J. Otolaryn.*, 10:10-16, 1981.
Pitcher, J.L., "Dysphagia in the Elderly: Causes and Diagnosis," *Geriatrics*, pp. 64-69, 1973.
Seaman, W.B., "Pharyngeal and Upper Esophageal Dysphagia," *J. Am. Med. A.*, 235:2643-2646, 1976.
Silverman, E.H., and Elfant, I.L., "Dysphagia: An Evaluation and Treatment Program for the Adult," *Am. J. Occ. Ther.*, 33:382-392, 1979.
Zimmerman, J.E., and Oder, L.A., "Swallowing Dysfunction in Acutely Ill Patients," *Am. J. Phys. Ther.*, 61:1755-1763, 1981.

SECTION IV.

Motor Speech Disorders (Apraxia and Dysarthria)

In the last ten years, there has been increased awareness among speech pathologists of the motor speech disorders, apraxia and dysarthria. This interest is evident in the publication of journal articles and text books and in presentations at ASHA conventions and state speech and hearing association meetings. The publications and presentations provide information both on research into a better understanding of these disorders and methods of providing therapy to alleviate the problems of apraxia and dysarthria. The purpose of this section is to provide a brief description of the motor speech disorders, to encourage the speech pathologists within our association to become active in the treatment of motor speech disorders, and to educate the non-speech pathologists in our association so that they may be better able to recognize these disorders and refer appropriately to speech pathologists for treatment.

Apraxia

Apraxia is a motor speech disorder. It is an impairment of the ability to program voluntarily the positioning of the speech musculature and the sequencing of muscle movements for speech.

Apraxia is caused by damage to the brain, usually the left cerebral hemisphere in the region known as Broca's area. Apraxia was first identified by Dr. Paul Broca, around the mid-1800's, and has been inaccurately termed Broca's aphasia. Apraxia often co-exists with aphasia but aphasia is a language disorder; apraxia is a motor speech disorder. The apraxic patient knows what he wants to say. His problem is not one of word-finding or retrieval as in aphasia. He cannot execute the proper movement sequence of the articulators to form the words.

The neurological (organic) damage in apraxia is often subtle and not easily identified. It may result from vascular problems, such as CVA, head injury, such as birth trauma or automobile accident, or disease, such as encephalitis. It can be congenital, in which case it is referred to as developmental apraxia, or acquired through neurological insult. Some of the etiological factors such as stroke and gunshot wound are evident on x-rays, C.A.T. scans, etc.; however, many apraxic conditions must be diagnosed solely on the speech symptomatology.

The single most distinguishing feature of apraxia is the inconsistency of the articulatory errors. An apraxic patient attempting to say "tornado" might say "danordo," "tunato," "tranado," or any other combination of consonant and vowel errors. Apraxic patients are usually aware that they are off-target and struggle to self-

correct. The attempts to self-correct are a groping "trial-and-error" behavior. The patient is unable to program the motor movements of the tongue, lips, palate, and mandible.

Speech is characterized by substitutions, distortions, additions, omissions, and repetitions of sounds. Sounds and syllables may be transposed. The degree of difficulty increases with polysyllabic words: the longer the word, the more articulatory movements that are required, the more problems the apraxic patient will have. Similarly, the more complex sounds such as affricates /tʃ, dʒ/ and consonant clusters (l - blends, r - blends, s - blends) are more often inaccurately produced. The less visible sounds /k, g/ and the glides /j, l, and r/ seem to be extremely difficult for apraxic children in my experience.

There is no generalized motor deficit. The apraxic patient has adequate strength and mobility of the speech apparatus. He is able to control the oral muscle movements for vegetative functions, such as chewing, gathering, and swallowing. There is sometimes an associated oral or non-speech apraxia. In oral apraxia, the patient also has no difficulty with vegetative functions but cannot program the oral musculature for non-speech functions imitatively: sticking out the tongue, whistling, puffing out the cheeks, kissing. These are movements which the oral apraxic can do automatically but not volitionally or "on command."

Apraxia is identified on the basis on speech symptoms. C.C. Publications, Inc. has published two very good diagnostic instruments: Screening Test for Developmental Apraxia of Speech, and Apraxia Battery for Adults. The Screening Test is designed for children age four through age twelve and was developed by Dr. Robert Blakely. The Apraxia Battery for Adults was developed by Dr. Barbara Dabul. It was developed to diagnose apraxia, to differentiate it from aphasia and dysarthria, and to estimate the severity of the disorder. Retesting at intervals provides an assessment of improvement in motor speech programming as therapy progresses.

Apraxia patients can be helped by a trained and qualified speech pathologist. The bibliography below lists several excellent references on treatment approaches. Many of the good therapy techniques are being developed at the Mayo Clinic and various Veterans Administration Hospitals around the country. In reference to the section "Speech Pathology - Redefined" published above, many of the "functional" articulation cases we see, the really stubborn ones who don't respond to traditional articulation therapy techniques, perhaps are undiagnosed apraxics.

Table 1. Motor Speech Disorder – Apraxia

LEVEL OF INJURY	SPEECH SYMPTOMATOLOGY	NON-SPEECH CLINICAL MANIFESTATIONS
Psychomotor	1. Definition: Inability to program elemental speech postures in absence of paralysis or paresis of speech musculature due to impairment of Broca's Area functioning	A. Difficulty in volitional oral non-speech tasks (Non-Speech Apraxia)
SITE OF LESION Left cortical hemisphere; probably Broca's area or association fiber disruption from Broca's area to other primary language processing areas	2. Characteristics: a. Inconsistent and variable articulation errors: substitution and omission types most common b. Serial motion rates irregular with intermittent phoneme (or word) transpositions c. Occasional prosodic variations: pauses, slowed rate, and equalization of stress	B. No noticeable muscular weakness, spasticity, flaccidity, or any other characteristics common to neuropathologies at different levels of the nervous system
ETIOLOGY Vacular/trauma/surgery/neoplasm/encephalitis/etc.		

Dysarthria

The other motor speech disorders which will be addressed in this article are actually a cluster of disorders which fall within the broad term of dysarthria.

Dysarthric conditions result from weakness or paralysis, incoordination, or sensory loss in one or more of the muscle groups responsible for respiration, phonation, articulation, resonance, or the prosody features of speech (rate, stress, inflection, rhythm).

The characteristic which most clearly differentiates dysarthria from apraxia is consistency. Neurologically, the muscles involved are weak or paralyzed, therefore, the patient consistently misarticulates the same sounds regardless of the context or speaking situation. Biological and vegetative processes are affected as well as speech. For example, the patient with weakness or paralysis of the lip muscles would not be able to use those muscles effectively for the formation of speech sounds/p,b,m,f,v/ but also could not control lip movements or posture during chewing, swallowing, or at rest.

Some of the medical conditions which result in dysarthria are the same as those which can result in apraxia: cerebrovascular accident, traumatic brain injury, cerebral palsy, Parkinson's disease, bulbar palsy, myasthenia gravis, encephalitis, and other neurological disorders. Some are congenital, others are acquired, still others are degenerative disease processes.

The site of the lesion (bulbar, cerebellar, etc.) and the extent of the damage will determine which speech processes and which speech valves are involved. The speech processes which might be affected are as follows:

1. Respiration – damage to the muscle control of the diaphragm, lungs, and/or intercostal muscles. This can result in weak voice production, audible inhalation or exhalation, bizarre stress patterns, inadequate or irregular loudness, and similar breath support problems.

2. Phonation – damage to the muscle control of the larynx. This can result in breathy, hoarse, harsh, or strident voice, problems with voiced/voiceless cognates, problems initiating phonation, bizarre pitch variations, or dysphonia/aphonia.

3. Articulation – damage to the muscle control of the tongue, lips, and mandible. This can result in sluggish or imprecise speech sound production.

4. Resonance – damage to the muscle control of the larynx, pharynx, and, most often, affecting the opening and closing of the velopharyngeal port mechanism. This can result in hypernasality or hyponasality.

5. Prosody features – damage to the muscle control of respiration, phonation, articulation, or resonance. This can result in bizarre patterns in the rate, stress, inflection, and rhythm of speech. There might be excessive pausing, prolongation of sounds or syllables, too slow or too rapid rate of speaking, explosive or absent stress patterns, or dysfluency similar to stuttering.

Figure 1.

Speech Valves System of Classification (from Leonard LaPointe, "Evaluation and Treatment of Motor Speech Disorders," ABESPA workshop, August, 1981).

1. Respiratory System
 2. Laryngeal System
 3. Velopharyngeal System
 4. Tongue blade
 5. Tongue tip
 6. Lips
 7. Mandible
- } Oropharyngeal System

Rosenbek and LaPointe originated the Speech Valves System of classifying dysarthric conditions, shown in Figure 1. The Speech Valves System is also known as the "Point-Place" system or as "Seven Stops Along the Nile." This system focuses attention on the underlying neuromuscular deficit, pinpointing the area or areas where the patient is having the most difficulty, and subsequently assists the clinician in planning the direction and focus of treatment.

It is not within the scope of this paper to outline treatment for the dysarthrias. As with apraxia, many excellent treatment techniques are being developed in V.A. Hospitals and universities throughout the United States. I would encourage my colleagues to read text books and journal articles, to attend workshops and presentations, and to become actively involved in the treatment of the motor speech disorders.

Table 2. Spastic Dysarthria

LEVEL OF INJURY	SPEECH SYMPTOMATOLOGY	NON-SPEECH CLINICAL MANIFESTATIONS
Pyramidal	1. Definition: Paresis and spasticity of speech musculature due to bilateral Corticobulbar Tract impairment	A. Bilateral symptomatology
SITE OF LESION	2. Characteristics:	B. Unaltered structural appearance of affected musculature
Upper motor neuron predominantly extrapyramidal (Spastic I) or predominantly pyramidal (Spastic II)	a. Slow and indistinct articulation, imprecise consonant and vowel production	C. Hyperactive orofacial reflexes of certain affected muscles
ETIOLOGY	b. Hypernasality	D. Spasticity
Trauma/C.P./	c. Hoarse, weak, monopitch, monoloudness, and strained-strangled phonation	
Vascular (Spastic I)	d. Reduced alternate motion rates of tongue and lips	
Encephalitis/Hemorrhage (Spastic II)		

Table 3. Hyperkinetic Dysarthria

LEVEL OF INJURY	SPEECH SYMPTOMATOLOGY	NON-SPEECH CLINICAL MANIFESTATIONS
Extrapyramidal	1. Definition: Irregular, unsustained, rapid, jerky, random, and unpatterned motor speech activities as are observed in chorea, athetosis, and dystonia due to Caudate and Putamen Nuclei impairment	A. Same as those listed for Spastic Dysarthria
SITE OF LESION		B. Neck, trunk, and limb hyperkinesias during walking and other forms of bodily movement
Basal ganglia		
ETIOLOGY	2. Characteristics:	
Focal dystonia/ tardive dyskinesia/ chorea	a. Imprecise consonant production	
	b. Harsh and monopitched voice	
	c. Prolongation of inter-syllabic intervals	
	d. Irregular lingual and labial alternate motion rates	

Table 4. Hypokinetic Dysarthria

LEVEL OF INJURY	SPEECH SYMPTOMATOLOGY	NON-SPEECH CLINICAL MANIFESTATIONS
Extrapyramidal	1. Definition: Rigidity and tremor of orofacial musculature accompanied by slowness and limited range and force of motor speech activity as are observed in Parkinsonism due to Substantia Nigra impairment	A. Same as those listed for Spastic Dysarthria
SITE OF LESION		B. Masklike facies
Basal ganglia (substantia nigra)		C. Slow, shuffling gait
ETIOLOGY	2. Characteristics:	D. Flexed posture of neck, trunk, and limbs
Parkinsonism/ Phenothiazine toxicity	a. Harsh, breathy, monopitched, and monoloud voice of reduced intensity	
	b. Durational variations – excessive pausing, prolongation of syllables, intermittent periods of slow and rapid speaking rates	
	c. Imprecise consonant production.	
	d. Variable lingual and labial alternate motion rates – sometimes rapid, other times slow	

Table 5. Ataxic Dysarthria

LEVEL OF INJURY	SPEECH SYMPTOMATOLOGY	NON-SPEECH CLINICAL MANIFESTATIONS
Cerebellar	1. Definition: Errors in timing, speed, range, force, and direction of whole and individual parts of movements of the speech mechanism due to cerebellar impairment	A. Bilateral symptomatology
SITE OF LESION		B. Flabby and hypotonic muscle appearance
Cerebellum		C. Ataxic trunk and limb activities
ETIOLOGY	2. Characteristics:	
Toxicity/trauma/ neoplasm/vascular, etc. (idiopathic)	a. Imprecise consonant and vowel production	
	b. Scanning speech	
	c. Occasional periods of explosive stress patterns	
	d. Hoarse, breathy, monopitched, monoloud voice	
	e. Dysrhythmic respiratory cycling	
	f. Slow and irregular lingual and labial alternate motion rates	

Table 6. Flaccid Dysarthria

LEVEL OF INJURY	SPEECH SYMPTOMATOLOGY	NON-SPEECH CLINICAL MANIFESTATIONS
Final Common Pathway	1. Definition: Paralysis and flaccidity of speech musculature due to unilateral or bilateral motor unit impairment	A. Unilateral or bilateral symptomatology
SITE OF LESION		B. Weakness, hypotonicity, atrophy, corrugation, fasciculations, and occasionally discoloration of affected musculature
Lower Motor Neuron	2. Characteristics:	C. Hypoactive orofacial reflexes of certain affected muscles
1. brain stem (site dependent)	a. Impairment of all types of motor activities of speech mechanism including voluntary, automatic and reflexive	D. Rapid and progressive muscular fatigue as observed in Myasthenia Gravis
2. myoneural junction	b. Hypernasality	
ETIOLOGY	c. Imprecise consonant production	
1. Bulbar palsy, etc.	d. Reduced lingual and labial alternate motion rates	
2. myasthenia gravis	e. Breathless – hoarse voice	
	f. Audible inspiration	

Table 7. Diagnostic Differences

	APRAXIA	DYSARTHRIA	FUNCTIONAL ARTICULATION
Movements of tongue, lips, and palate	Normal movements, except for speech; if nonverbal apraxia is also present, only volitional oral movements will be affected; vegetative function remains normal	Movement of one or more muscle groups obviously affected; difficulty with vegetative functions as chewing and swallowing	Normal
Neurological signs	"Soft" neurological signs; difficulty with fine motor coordination, gait, and alternate movements of the extremities	Usually part of a more generalized neurological impairment, such as cerebral palsy or other neuromuscular difficulties	Normal
Speech development	Sometimes retarded, but usually within normal range	Usually some delay	Normal
Phonation	Normal	May be incoordination of phonation and articulation, or of both with respiration	Normal
Ability to imitate speech sounds	Auditory stimulus is usually insufficient; imitation should be assisted by visual stimulus and may be normal when the child is watching therapist's movements; seeing the word in print helps the older child	Normal on auditory stimulus within the limits of muscular movements	Normal
Diadochokinetic rates	Slower than normal; /p-t-k/ may not be accomplished in correct order	Usually slower than normal but accurate within limits of muscular movement; correct syllable order preserved	Normal
Articulation	Inconsistent errors: distortion, additions, and substitutions are variable; imitation and use of single sounds, syllables, or words is better than their use in long sequences; transpositions of sounds, vowels affected in more severe impairments	Consistent errors; consonants often omitted; substitutions determined by the group of muscles chiefly affected; vowels usually normal	Consonant substitutions usually consistent; omissions and additions are rare; vowels are normal
Error patterns	Difference in error pattern between performance in spontaneous speech and repeated speech tasks	Consistent from task to task	Consistent from task to task
Prosody	Intentional slow rate, even stress to compensate for difficulties	Rate is slow according to degree of incoordination	Normal
Groping "trial-and-error" behavior	Present in older children or in those who have received speech therapy	Absent	Absent

(From: *Terminology of Communication Disorders*, Nicolosi, Harryman, Kresheck, pp. 272-273)

References on Apraxia

- Aten, J., Johns, D. and Darley, F., "Auditory Perception of Sequenced Words in Apraxia of Speech," *J. Sp. Hear. Res.*, 14:131-143, 1971.
- Chappell, G., "Childhood Verbal Apraxia and its Treatment," *J. Sp. Hear. Dis.*, 38:362-368, 1973.
- Dabul, B., and Bollier, B., "Therapeutic Approaches to Apraxia," *J. Sp. Hear. Dis.*, 41:268-276, 1976.
- Darley, F., Aronson, A.E., and Brown, J.R., *Motor Speech Disorders*, W.B. Saunders, 1975.
- Deal, J., and Darley, F., "The Influence of Linguistic and Situational Variables on Phonemic Accuracy in Apraxia of Speech," *J. Sp. Hear. Res.*, 15:639-653, 1972.
- Halpern, H., Darley, F., and Brown, J., "Differential Language and Neurologic Characteristics in Cerebral Involvement," *J. Sp. Hear. Dis.*, 38:162-273, 1973.
- Johns, D., and Darley, F., "Phonemic Variability in Apraxia of Speech," *J. Sp. Hear. Res.*, 13:556-583, 1970.
- LaPointe, L. and Wertz, R., "Oral-Movement Abilities and Articulatory Characteristics of Brain-Injured Adults," *Perceptual Motor Skills*, 39:39-46, 1974.
- Rosenbek, J.C., "Treating Apraxia of Speech" in *Clinical Management of Neurogenic Communicative Disorders*, (D. Johns, Ed.) Boston: Little, Brown and Co., 1979.
- Rosenbek, J., Lemme, M., Ahern, M., Harris, E., and Wertz, R., "A Treatment for Apraxia of Speech in Adults," *J. Sp. Hear. Dis.*, 38:462-472, 1973.
- Skelly, M., Schinsky, L., Smith, R., and Fust, R., "American Indian Sign (Amerind) as a Facilitator of Verbalization for the Oral Verbal Apraxic," *J. Sp. Hear. Dis.*, 39:445-456, 1974.

References on Dysarthria

- Darley, F.L., Aronson, A.E. and Brown, J.R., *Motor Speech Disorders*, W.B. Saunders, 1975.
- Darley, F.L., Aronson, A.E. and Brown, J.R., "Differential Diagnostic Patterns of Dysarthria," *J. Sp. Hear. Res.*, 12:246-269, 1969.
- Darley, F.L., Aronson, A.E. and Brown, J.R., "Clusters of Deviant Speech Dimensions in the Dysarthrias," *J. Sp. Hear. Res.*, 12:462-496, 1969.
- Johns, Donnell F. (ed.), *Clinical Management of Neurogenic Communicative Disorders*, Little, Brown and Co., 1978.
- Kent, R., Netsell, R. and Abbs, J., "Acoustic Characteristics of Dysarthria Associated with Cerebellar Disease," *J. Sp. Hear. Res.*, 22:627-648, 1979.
- Kent, R.D., Netsell, R. and Bauer, L.O., "Cineradiographic Assessment of Articulatory Mobility in the Dysarthrias," *J. Sp. Hear. Res.*, 40:467-479, 1975.
- LaPointe, L.L., and Wertz, R.T., "Oral-movement Abilities and Articulatory Characteristics of Brain-injured Adults," *Perceptual Motor Skills*, 39:19-46, 1974.
- Mason, R.M., and Simon, C., "An Orofacial Examination Checklist," *J. Lang. Sp. Hear. Serv.*, 8:155-164, 1977.
- Netsell, R., "Evaluation of Velopharyngeal Function in Dysarthria," *J. Sp. Hear. Dis.*, 34:113-122, 1969.
- Netsell, R., and Cleeland, C.S., "Modification of Lip Hypertonia in Dysarthria Using EMG Feedback," *J. Sp. Hear. Dis.*, 38:131-140, 1973.
- Peachar, W.G., "The Etiology and Differential Diagnosis of Dysarthria," *J. Sp. Hear. Dis.*, 15: 252-265, 1950.
- Tikofsky, R.S., "A Revised List for the Estimation of Dysarthric Single Word Intelligibility," *J. Sp. Hear. Res.*, 12:59-64, 1970.
- Yorkston, K., and Beukelman, D., "A Comparison of Techniques for Measuring Intelligibility in Dysarthric Speech," *J. Comm. Dis.*, 11:499, 1978.
- Yorkston, K., and Beukelman, D., "A Clinician-judged Technique for Qualifying Dysarthria Speech Based on Single-word Intelligibility," *J. Comm. Dis.*, 13:15, 1980.