Assessing and Managing Dysarthria

Background: Dysarthria is a motor speech impairment that affects speech execution and control. It results from an impairment in the motor neurons (upper and/or lower), neuromuscular junction, brain areas supporting speech feedback control (i.e., basal ganglia and cerebellum), or the speech musculature itself. Dysarthria is classified by the neuroanatomical structures affected (see Tables 1 and 2), and can affect any of the speech subsystems (respiration, phonation, articulation, resonance, and prosody). Dysarthria is one of the most common first signs of stroke (Table 1 details post-stroke dysarthria), but it can result from many different neurological impairments, including traumatic injuries, tumors, degenerative processes (e.g., Parkinson's disease, Huntington's Disease, multiple sclerosis), and transient impairments (e.g., Bell's palsy).

Table 2 presents a summary of each dysarthria type, neuroanatomy implicated, and common etiologies. Tables 1 and 2 also include details pertaining to AOS to illustrate how these motor speech impairments differ, and features that distinguish each disorder.

Assessment and Differential Diagnosis

Dysarthria assessment must include an oral mechanism/cranial nerve evaluation. ASHA's practice portal offers detailed procedures for assessing dysarthria (see link below). In addition, your evaluation report should consider how the dysarthria impacts intelligibility. Specifically, consider how reduced intelligibility affects your patient's ability to communicate during emergency situations, verbalize important information regarding his/her medical care, and engage in vocational and/or social activities. Considering these factors will also help when developing functional treatment goals.

Several assessment tools exist. These include:

- Frenchay Dysarthria Assessment-2nd Edition (FDA-2; Enderby & Palmer, 2008)
- Dysarthria Examination Battery (Drummond, 1993)
- Dysarthria Profile (Robertson, 1982)
- And tests of intelligibility

Assessment of Intelligibility of Dysarthric Speech (AIDS; Yorkston et al., 1984)
Speech Intelligibility Test (SIT; Beukelman et al., 2007)

- Mayo Clinic (informal) (Duffy, 2005).

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Treatment

The goal of treatment is to improve the domain of production that has the greatest impact on intelligibility. Dworkin (1991) suggests a treatment hierarchy based on speech subsystems, where resonance and respiration are "first order" targets, phonation is a "second order" target, and prosody and articulation are "third order" targets. The rationale here is a top-down approach, based on the interdependence of the speech subsystems. Targeting first order targets first is likely to have the greatest impact on intelligibility, and have "downstream" effects on the second and third order targets. Clark (2014) also discusses that training prosody, specifically phrasing of breath groups and lexical stress, may have a substantial impact on intelligibility early in the course of treatment. With this in mind, treatments may focus on the following:

- Improving **respiratory** support for speech (consider implementing diaphragmatic breathing, postural adjustments, training inhalation/exhalation coordination for speech, using expiratory muscle trainers (Laciuga et al., 2014), phrase grouping strategies)
- Consideration of prosthetic device to manage **resonance** if appropriate. Behavioral intervention for resonance may also include use of nasal mirrors or a See-Scape for biofeedback while training resonance. Yorkston et al. (2001) provide detailed EBP regarding the management of velopharyngeal insufficiency in dysarthria.
- **Phonation** may be addressed through vocal function exercises (Stemple et al., 2014), managing laryngeal strain (McCullough et al., 2012), or improving loudness with Lee Silverman Voice Treatment (LSVT, especially effective for hypokinetic dysarthria due to Parkinson's disease (Mahler et al., 2015)
- Training "over-**articulation**" to improve intelligibility (Park et al., 2016)
- Using pacing strategies/metronome to target **prosody** (Blanchet & Snyder, 2010)

This is not an exhaustive list of treatments, but a guide to get you started! In addition to the suggestions above, goals may need to include the use of augmentative/alternative strategies and environmental modification (e.g., reducing background noise, or communication partner training techniques). Early implementation of AAC in degenerative dysarthrias is especially important to give patients time to learn these strategies before degenerative processes progress. Refer to ASHA's practice portal and the references for greater details on each of these approaches.

As a reminder, treatment should **NOT** incorporate non-speech oral motor exercises for improving speech production. There are several reasons for this: First, very little research supports the use of non-speech oral motor exercises for improving speech intelligibility. Second, other studies suggest that parts of the brain that are active during non-speech oral movements are only partially overlapping with those that are active during speech motor movements (Basilakos et al., 2018). Therefore, the use of non-speech oral motor movements to improve speech violates the specificity principle of plasticity (Kleim & Jones, 2008).

Table 1. Differential diagnosis of MSDs caused by stroke/traumatic injury. Additional dysarthria characteristics are included in Table 3.

	AOS	Dysarthria
Rate of occurrence	No reliable estimate	Estimates up to 69.5% acutely[1]
Speech characteristics[2-5]	-Distorted sound substitutions/additions -Increased errors upon utterance length and complexity -atypical prosody (i.e., segmented syllables within words, across utterances)	Post-stroke dysarthria generally associated with imprecise production of consonants, reduced rate, dysprosody, strained-strangled vocal quality -Other characteristics may reflect specific neuroanatomical regions/ cranial nerves affected
Associated lesion [6-7]	Cortical motor areas (premotor, primary motor, and supplementary motor areas), as well as post-central somatosensory areas and possibly the inferior frontoinsular regions. Typically left hemisphere	Supratentorial regions associated with spastic dysarthria (bilateral damage); unilateral damage may result in UUMN dysarthria Damage to basal ganglia can result in hypo- or hyper-kinetic dysarthrias Brainstem stroke associated with flaccid dysarthria, with impairments corresponding to cranial nerve(s) affected Cerebellar stroke may result in ataxic dysarthria
Differential Diagnosis[2-4]	*Unique AOS symptoms:* -Distortion errors that increase with utterance length and/or complexity -Distorted sound substitutions/additions (not distorted sound production can occur in both AOS and dysarthria) -Shortened phrase lengths not explained by reduced respiratory capacity	*Symptoms that occur in dysarthria but not AOS or aphasia:* -Muscle paralysis or paresis -Altered vocal quality, deviant nasality -Decreased respiratory capacity -Pathological reflexes (spastic)
Other presenting impairments	**-Buccofacial apraxia** (evident in non-speech movements in the absence of muscular weakness and/or spasticity; 48-75% co-occurrence with AOS[2] **-Limb apraxia** **-Dysphagia** (impairment in oral, pharyngeal and/or esophageal phases of swallowing, 28% co-occurrence with dysarthria[8] -**Aphasia** (81% co-occurrence with AOS; 28-47% co-occurrence with dysarthria[1,2])	

Table 2. Characteristics and etiologies of AOS and the dysarthrias

	Distinguishing Characteristics	Common Etiologies	Neuroanatomy Implicated
Apraxia of Speech	Distorted substitution and addition errors, off-target, uncoordinated DDK rates, atypical prosody	Stroke Brain injury Degenerative process	Premotor, primary, supplementary, & sensorimotor cortices
Flaccid Dysarthria	Voice is breathy, may also be wet/hoarse, imprecise consonants; reduced speed and ROM in articulators; tongue fasciculation; fatigue upon DDK rates, DDKs may be slow and slurred	Stroke Poliomyelitis Myesthenia gravis Cranial nerve palsies	Motor units (lower motor neuron, myoneural junction, speech musculature)
Spastic Dysarthria	Strained/strangled vocal quality, "grunting" at end of phrases, slow DDK rates; pathological reflexes may be present	Stroke Brain injury Cerebral Palsy (spastic)	Upper motor neurons (bilaterally)
Unilateral Upper Motor Neuron Dysarthria	Unilateral facial weakness, flattening of nasolabial fold; articulatory imprecision; harsh voice	Unilateral stroke	Upper motor neurons (unilaterally)
Hypokinetic Dysarthria	Reduced loudness, complaints of not being heard; short rushes of speech, flat pitch, DDK rates may be fast and imprecise	Parkinson's Disease Medication use	Basal ganglia (dopamine depletion)
Hyperkinetic Dysarthria	Involuntary movements at rest/during speech, articulatory breakdowns, voice stoppages (involuntary vocal fold adduction/ abduction)	Huntington's Disease Psychotrophic medication use	Basal ganglia (excess dopamine)
Ataxic Dysarthria	Slurred speech with "scanning" quality (excess/ equal stress), Irregular articulatory breakdowns Harsh voice	Cerebellar stroke/ injury, atrophy of cerebellum and connections; congenital conditions (e.g., Freidrich's ataxia[9])	Cerebellum and associated pathways

Mixed dysarthrias are also possible in cases where pathology diffusely affects the brain. Some examples include mixed dysarthrias that result from: multiple sclerosis (spastic-ataxic), amyotrophic lateral sclerosis (spastic-flaccid), multiple systems atrophy (hypokinetic-spastic-ataxic), progressive supranuclear palsy (hypokinetic-spastic-ataxic), and corticobasal degeneration (hypokinetic-spastic; (Duffy, 2005).

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Additional Resources

ASHA's practice portal for dysarthria: https://www.asha.org/PRPSpecificTopic.aspx?folderid=8589943481&section=Overview

Dr. Heather Clark's e-Workshops, Assessing Dysarthria in Adults:
https://www.asha.org/eweb/OLSDynamicPage.aspx
Webcode=olsdetails&title=Assessing+Dysarthria+in+Adults

Treating Dysarthria in Adults:
https://www.asha.org/eweb/OLSDynamicPage.aspx?
Webcode=olsdetails&title=Treating+Dysarthria+in+Adults