Any questions regarding the dysarthria's we've covered in class which includes:

Flaccid-

Flaccid dysarthria is a result of damage or impairment to the lower motor neurons of the cranial or spinal nerves (damage to PNS).
Flaccid dysarthria is characterized by:

• Paralysis, weakness, hypotonicity, atrophy, and hypoactive reflexes of the involved speech subsystem musculature

• Weakness of speech or respiratory musculature resulting in the presence of distinctive qualities

The distinctive qualities (depending on which nerve or combination of nerves is disrupted) of flaccid dysarthria are:

• Slow-labored articulation
• Distorted productions of lingual consonants
• Marked degrees of hypernasal resonance
• Hoarse-breathy phonation

• Caused by any disorder that disrupts flow of neural impulses along LMNs that innervate muscles of respiration, phonation, articulation, prosody, or resonance

Caused by any disorder or injury that disrupts the flow of motor impulses along cranial or spinal nerves, which innervate the muscles of speech production
• The most common causes are:
• Physical trauma
• Brain stem CVA
• Myasthenia gravis
• Guillain-Barré Syndrome
• Polio
• Tumors
• Muscular dystrophy
• Progressive bulbar palsy

Most Common Speech
Production Errors in Flaccid Dysarthria
Landmark study in 1969 by Darley, et al. ranked the nine most common speech production errors in flaccid dysarthria:
1. Hypernasality
2. Imprecise consonants
3. Breathiness
4. Monopitch
5. Nasal emission
6. Audible inspiration
7. Harsh vocal quality
8. Short phrases
9. Monoloudness

Treatments for Flaccid:
Treatments for flaccid dysarthria are grouped according to which cranial nerve or combination of nerves are damaged.
• Needs and abilities of patients vary greatly and may be impacted by comorbidities.
Note on nonspeech oral strengthening exercises
• Efficacy is open to question as nonspeech oral exercises require much greater force of movement than speech production.
• General rule: If improving speech production is the goal, treatment activities should concentrate directly on speech production.

Spastic –

Spastic dysarthria is caused by bilateral damage to upper motor neuron tracts.
• The articulators are often stiff and may move with reduced range of motion (especially within the vocal tract).
• Speech is often slowed, prolonged, and labored.
• Spastic paralysis or paresis is present in the involved muscles.
• Hyperreflexia may be present.

Significance of
Bilateral Damage (cont.)
• The resulting symptoms are marked by:
• Paresis or paralysis: particularly of the labial and lingual muscles
• Slowness in the execution of movement: particularly

of the labial and lingual muscles
• Spasticity: most noticeable in the laryngeal muscles
• Hyperadduction of the vocal folds is the most common spastic symptom noted
• Abnormal reflexes (gag, jaw, and bruxism

Etiology of Spastic Dysarthria
• Spastic dysarthria is caused by any injury that causes damage to the UMNs of the pyramidal and extrapyramidal systems, within both the left and right hemispheres
• The most common causes are:
• Stroke/cerebral vascular accidents (CVA)
• Degenerative diseases
• Traumatic brain injuries
• Anoxia
• Infections of the brain tissue
• Tumors

Landmark study in 1969 by Darley, et al. ranked the fourteen most common speech production errors in spastic dysarthria:
1. Imprecise consonants
2. Monopitch
3. Reduced stress
4. Harsh vocal quality
5. Monoloudness
6. Low pitch
7. Slow rate
8. Hypernasality
9. Strained-strangled quality
10. Short phrases
Speech Characteristics of SD, Part II
11. Distorted vowels
12. Pitch breaks
13. Breathy voice (continuous)
14. Excess and equal stress

• **The fourteen speech errors that are seen in spastic dysarthria are a result of**:
• Spasticity of the musculature
• Slowness of motor movements
• Weakness in the vocal tract muscles
• Components of speech
• Articulation, phonation, resonance, and prosody are typically impacted more than respiration

Origin of damage

• Spastic dysarthria results from bilateral damage to the UMNs of the pyramidal and extrapyramidal systems.
• Flaccid dysarthria results from damage to LMNs.

Evaluation Tasks for Spastic Dysarthria

Key tasks that assist with the diagnosis of spastic dysarthria
• Alternate motion rate (AMR)
• Highlights the slow rate of phoneme production
• Sustained vowel production
• Evokes phonatory deficits that are seen as a result of a spastic vocal tract
• Conversational speech and passage reading
• Assesses resonance, articulation, and prosody

Treatment of Spastic Dysarthria

• Goals should always be patient specific and target the impairments that when resolved will demonstrate the greatest functional improvement in speech intelligibility.
• Treatment goals for spastic dysarthria should target:
• Phonation
• Articulation
• Prosody
• Resonance
• Respiration is not typically impacted to a level that requires intervention; difficult to tease out and separate respiration from phonatory deficits.

-Unilateral Upper Motor Neuron (Fairly New)

Unilateral UMN dysarthria is a fairly new diagnosis

• It was not specifically researched until the 1980s
• Defined as a motor speech disorder caused by damage to the UMNs on a single side of the brain that supply the cranial and spinal nerves involved in speech production
• Characterized by weakness of the lower face, lips, and tongue on the contralateral side of the lesion, resulting in motor speech deficits

Unilateral UMN Dysarthria Defined (cont.)

• Primary motor speech deficits observed are:
• Imprecise consonant production (most common)
• Slowed diadochokinetic rate
• Potential voice disorder due to vocal fold paresis/paralysis

Neurological Basis

• Most cranial nerves serving the speech muscles, except the lower face and tongue, receive bilateral innervation from UMNs.
• Thus, motor speech deficits after unilateral UMN damage are typically less severe than when bilateral damage occurs.
• The affected side will demonstrate:
• Paresis or paralysis with facial asymmetry/facial droop
• Slowed movements
• Reduced range of motion of lower face and tongue
• Tongue may deviate to the affected side

• UMNs bilaterally innervate velum, pharynx, and larynx. Therefore, in theory, they should not be affected by unilateral UMN damage, as they are sufficiently innervated from the unaffected side.
• In reality, unilateral UMN damage appears to impact the function of bilaterally innervated structures.

Causes of Unilateral UMN Dysarthria

• Unilateral UMN dysarthria is caused by any condition that damages the UMNs on a single side of the brain.
• Damage can occur in either the left or right hemisphere.
• Left hemisphere: Unilateral UMN dysarthria often cooccurs with aphasia or apraxia of speech.
• Right hemisphere: Unilateral UMN dysarthria often co-occurs with cognitive-linguistic deficits.
• Pathologies that cause focal lesions are the most common cause.

-Stroke
-Brain Tumors
-TBI

Speech Characteristics, Part III

• In 1986, Duffy and Folger ranked the 12 most common speech production errors seen in unilateral UMN dysarthria:

1. Imprecise consonants
2. Slow AMRs
3. Harsh vocal quality
4. Imprecise AMRs
5. Irregular AMRs
6. Slow rate

7. Irregular articulatory breakdowns
8. Mild hypernasality
9. Reduced loudness

Speech Characteristics, Part IV

10. Strained-strangled vocal quality
11. Increased rate of speech in segments
12. Excess and equal stress

Key Evaluation Tasks

- Detailed chart review of symptomology, site of lesion, NIHSS score, past medical history, etc.
- Oral mechanism exam
- AMR tasks
- Prolonged vowels
- Sentence repetition or reading
- Conversational speech
- Picture description
- Passage reading

Treatment of UUMN Dysarthria, Part I

- Often other coexisting deficits are allotted the bulk of treatment time if they are deemed greater priority.
- Articulation may not be treated until a later time, if still needed.
- Articulatory deficits may be so mild that if the patient is deemed to be a functional communicator, they may not be addressed.
- Patients may benefit from a brief education on intelligibility strategies, if they are not candidates for further skilled speech pathology intervention.

Treatment of UUMN Dysarthria, Part II

- Imprecise consonant production is the primary articulation error seen in unilateral UMN dysarthria
- Treatment interventions for articulation
- Traditional articulation drills
- Recommended for imprecise consonant productions
- Focus is on increasing the patient's awareness of their articulation errors and practicing best phoneme productions
- May include intelligibility drills, training of phonemic placement, exaggeration of consonants, and minimal contrast drills

Treatment of UUMN Dysarthria, Part III

- Treatments for phonation (requires ENT clearance for diagnosis)
- Weakness of the vocal folds
- Pushing and pulling exercises

• Holding breath to facilitate greater adduction
• Head turn to weak side
• Chunking utterances into syntactic units (breathe when normal pauses would occur)
• ENT referrals for vocal fold injections/augmentation

Treatment of UUMN Dysarthria, Part IV

• Spasticity of the vocal folds
• Musculoskeletal relaxation exercises
• Easy onset of phonation rather than hard glottal attack
• Yawn-sigh exercise

Ataxic:

Ataxia is a term that means widespread incoordination.

• Ataxia is the Greek word for "lack of order."
• Ataxic dysarthria (AD) is a motor speech disorder that results from damage to the cerebellum or the neural pathways that connect the cerebellum to other portions of the CNS.
• Resultant speech errors are primarily articulatory and prosodic, which gives speech and unsteady, slurred quality
• Often likened to "drunken speech"
• Timing and force of movement of the articulators is irregular and slowed

Neurologic Basis

Cerebellum

• Primary function is to coordinate the timing and force of muscular contractions to create voluntary movement
• Imposes precise muscular control over movement
• Processes sensory information from the entire body and integrates this information into the execution of movement
• Much greater ratio of neural fibers that relay information to the cerebellum than relay information from the cerebellum

Speech Characteristics of AD

• Motor speech movements appear poorly coordinated
• Patients display difficulties controlling the timing and force of movements needed for clear speech production
• Articulation is noted to be slurred, and prosody is monotonous: often described as "drunken"
• Ataxic dysarthria is primarily a disorder of articulation and prosody
• Scanning speech
• Term used to describe the slow, deliberate production of syllables, with each syllable in a word receiving equal stress

Speech Characteristics of AD (cont.)
• Landmark study in 1969 by Darley, et al. ranked the 10 most common speech production errors in ataxic dysarthria:
1. Imprecise consonants
2. Excess and equal stress
3. Irregular articulatory breakdown
4. Distorted vowels
5. Harsh vocal quality
6. Prolonged phonemes
7. Prolonged intervals
8. Monopitch
9. Monoloudness
10. Slow rate

Key Evaluation Tasks for AD
• Detailed chart review of symptomology, site of lesion, NIHSS score, past medical history, etc.
• AMR tasks (will likely be slower than normal; in severe cases may speed up abruptly then unexpectedly slow)
• Sentence repetition or reading
• Conversational speech
• Picture description
• Passage reading
• Each of these tasks contain numerous multisyllabic words and will allow for the symptomology associated with ataxic dysarthria to come to the forefront

Treatment for Respiration
• Strength training of the respiratory muscles should not be a focus of skilled intervention.
• Instead, the SLP should focus on controlling airflow more accurately during speech tasks, as uncoordinated/paradoxical movements of the respiratory muscles cause the patient to speak on residual air.
• Speaking on residual air leads to impairments in prosody and phonation.
Treatment for Respiration (cont.)
• Treatment interventions to improve breath control during speech
• Slow and controlled exhalation
• Speak at peak of inhalation/immediately on exhalation
• Cease phonation early to prevent speaking on residual air
• Optimizing the breath: teaching how many syllables or words can be said clearly on one full inhalation
Treatment for Prosody, Part I

• The prosodic errors present within ataxic dysarthria include those of:
• Rate
• Stress
• Intonation
• When rate of speech is slowed, intelligibility can be positively impacted
• By incorporating more typical stress and intonation patterns into utterances, a patient may exhibit a more natural quality to his/her speech

Treatment for Prosody, Part II

• Training rate control for spoken utterances
• Typically a slow, irregular rate is characteristic of ataxic dysarthria. However, a patient may attempt to speak too rapidly for their motor capabilities.
• The articulators are not given enough time to reach target positions for phoneme production.
• The listener is not provided enough time to assimilate the spoken message.

Treatment for Prosody, Part III

• Treatment interventions targeting rate control
• Reciting syllables to a metronome
• Finger/hand tapping
• Cued reading material (sentences or short passages)
• Clinician points to word/syllable at the targeted rate
• Slash marks or spaces to indicate necessary pauses when reading

Treatment for Prosody, Part IV

• Skilled intervention for stress and intonation should concentrate on developing more natural pitch and loudness variations within connected speech
• Treatment for stress and intonation
• Pitch range exercises
• Contrastive stress drills
• Chunking utterances into syntactic units (breathe when normal pauses would occur)

Treatment for Articulation

• May improve with slowed rate as additional time may allow for improved accuracy to hit phoneme targets
• Intervention should focus directly on improving phoneme production
• Treatment for articulation
• Traditional articulation/intelligibility drills
• Phonetic placement training
• Exaggeration of consonants (overarticulation)

• Minimal contrast drills

-Hypokinetic

-Chapter 4-8 (You should be able to answer questions regarding the neurology, symptomology, etiology, assessment, and treatment) (Also you should be able to answer any questions that might lend yourself to a discussion about these dysarthria's in Chapter 2 & 3 – neurology and assessment respectively

-Brain stem and brainstem strokes

Stroke/CVA

Occurs when the arterial blood flow is interrupted via a blockage or the breakage of a vessel

• The most common cause of spastic dysarthria is a CVA.
• However, CVAs will result in spastic dysarthria only when:
• Two or more CVAs occur in certain combinations in the cerebrum.
• A single CVA occurs in brain stem where the neural fibers of each pathway are extremely close in proximity.
• A single CVA occurs in one hemisphere, and a preexisting condition has previously damaged the pyramidal and extrapyramidal tracts of the opposite hemisphere.

-Pyramid and Extrapyramidal tracts

Pyramidal tract

• Originates in the primary motor cortex and courses to the LMN (corticobulbar and corticospinal tracts)
• Damage can result in weak, slow skilled movements of the tongue, lips, velum, and other articulators

Extrapyramidal tract

• Originates primarily in the brain stem and incorporates critical cortical information as well
• An indirect pathway between the cortex and the LMN
• Transmits signals that regulate reflexes, maintain

posture, and monitor muscle tone
• Damage can result in weakness, increased muscle tone (spasticity), and abnormal reflexes

-MS

Multiple Sclerosis (MS)
• Multiple sclerosis is a suspected immunologic disorder resulting in the inflammation of or the complete destruction of the myelin sheath that covers the axons.
• When the UMNs of the pyramidal and extrapyramidal tracts in the bilateral hemispheres are involved, spastic dysarthria may result.
• Due to fact that MS may impact numerous other areas of the CNS, other dysarthrias may also result. MS does not necessarily have a direct correlation to spastic dysarthria.

-LSVT Loud and it's requirement for certification

- Cerebellum and how it communicates with the CNS

The cerebellum is attached to the posterior portion of the brain stem
• **Communicates with the CNS through three bundles of neural tracts called cerebellar peduncles:**
1. Inferior peduncle
2. Middle peduncle
3. Superior peduncle
Pathways to/From Cerebellum, Part II
• **The inferior peduncle allows the cerebellum to**:
• Receive sensory information from the entire body about position of its body parts in space
• Recognize what the body is doing during movement, and whether a motor impulse to the muscles is accomplishing the intended result
• Monitor timing and force of movements while being performed
Pathways to/From Cerebellum, Part III
• **The middle peduncle is the largest of the peduncles. It allows the cerebellum to:**
• Receive preliminary information from the cortex regarding planned movements (a rough approximation)
• Coordinate planned movements by integrating

sensory information from the body with an individual's experience of what appropriate movement should be, smoothing and refining according to current conditions

Pathways to/From Cerebellum, Part IV

• **The superior peduncle allows cerebellum to:**

• Provide output through one main channel to the CNS

• Send its processed motor impulses to motor areas of cortex, completing corticocerebellar control circuit

- Vocal Fold Pathology (within scope of practice)

-Hyperreflexia and what its commonly associated with

- Know about cerebellum and anatomical structures and what those parts do

The cerebellum influences motor speech movements in two main ways:

1. The corticocerebellar control circuit

• Planned motor speech movements are sent from the cortex to cerebellum.

• The cerebellum then coordinates and refines preliminary movements based on sensory information received from the body and prior knowledge.

• Coordinated motor impulses are then sent to the thalamus to be refined further before being sent to motor the cortex and the muscles.

The Cerebellum and Speech (cont.)

2. Cerebellar connections to the extrapyramidal system

• Makes rapid adjustments in the timing and force of movements to compensate for unexpected changes in circumstances of movement

• Example: speaking with a dental retainer

-LSVT – what improvements you might see

Causes of Ataxic Dysarthria

• Damage to cerebellum or its control circuits can result in a variety of movement disorders that are accompanied by dysarthria.

• The coordination of speech movement is highly dependent on the midpoint of the cerebellum between the two hemispheres, known as the

vermis.
• Ataxic dysarthria is most often a result of generalized damage to the cerebellum, though some studies have suggested that focal damage may also cause the motor speech disorder.
- You should know what the frequent cause of the dysarthrias are

- Cranial Nerves (which nerves do what) – Look at ONENOTE

-How many pairs do we have (in those pairs, how many are important for speech)

Six pairs of cranial nerves play vital roles in speech production. Referred to as "cranial nerves of speech production."

• Trigeminal (V)
• Facial (VII)
• Glossopharyngeal (IX)
• Vagus (X)
• Accessory (XI)
• Hypoglossal (XII)
Damage to the cranial nerves of speech production may decrease as a result of:
• Brain stem CVA
• Tumor
• Viral or bacterial infections
• Physical trauma
• Surgical accident

-Diseases: Polio, Huntington's chorea, myasthenia gravis

Myasthenia Gravis
• Disease process that affects the neuromuscular junction (point where the nerves meet the muscle tissue)
• Caused by antibodies that block/damage muscle tissue
• Characteristic symptom is rapid fatigue of muscular contractions over short period of time with recovery after rest
• Key task within a motor speech assessment:
• Stress testing involving asking the patient to count from 1 to 100, or to read a lengthy paragraph

Polio
• An infectious viral disease that attacks cell bodies of lower motor neurons
• Vaccines have reduced the incidence of this greatly
• Most frequently affects cervical and thoracic spinal nerves, causing:
• Labored inhalation during speech
• Shortened speech phrases

• Speaking on residual air
• Decreased vocal loudness
• Can also impact cranial nerves in 10–15% of the cases

- Speech systems of different dysarthria's – consonant impression, ect.

- What damage to the part of the brain causes parkinsonism

- Friedrich Ataxia and MS.

Friedreich's ataxia
• Progressive hereditary disease affecting the spinal cord as well as the cerebellum
• Dysarthria may be mixed in origin (ataxic-flaccid) as this disease impacts the LMNs as well
- Spastic dysarthria (what's common in it)

Multiple Sclerosis (MS)
• Multiple sclerosis is a suspected immunologic disorder resulting in the inflammation of or the complete destruction of the myelin sheath that covers the axons.
• When the UMNs of the pyramidal and extrapyramidal tracts in the bilateral hemispheres are involved, spastic dysarthria may result.
• Due to fact that MS may impact numerous other areas of the CNS, other dysarthrias may also result. MS does not necessarily have a direct correlation to spastic dysarthria.

-Name main branches of trigeminal nerve

Trigeminal nerve (V)
• Attached to brain stem at level of pons
• Divided into three main branches:
1. Ophthalmic
2. Maxillary
3. Mandibular
• Mandibular branch is most important for speech and for innervating the muscles in the lower jaw and velum
• Damage to the trigeminal nerve can be unilateral or Bilateral. Damage to trigeminal nerve may cause difficulty elevating the jaw sufficiently to bring the articulators into contact with each other

- May need to elevate jaw by hand, Kinesio Tape, or splinting (OT)

• Bilateral damage has a severe impact on speech intelligibility

- Differentiate between pseudobulbar palsy and pseudobulbar affect

Symptoms of Spastic Dysarthria:
Pseudobulbar affect
• Marked by uncontrollable crying or laughing that can accompany damage to the UMNs of the brain stem
• Appears to be caused by damage to the part of the brain that is important in inhibiting emotions
• May not necessarily reflect a patient's true emotions
Pseudobulbar palsy (clinical syndrome that incorporates spastic dysarthria)
• Weakness, spasticity, and slowness in muscles innervated through medulla, including tongue, velum, larynx, and pharynx
• Caused by damage to UMNs

-Talk about whether the arteries that serve cerebellum

- Answer difference between superior laryngeal nerve and recurrent laryngeal nerve (what do they serve and what can damage cause)

- Know the common etiologies of dysarthrias

- What should the focus be on traditional articulation drills

- What is dopamine in the brain

- What s a goal in forcilogic treatment in parkinsons

-Whats the focus of LSVT loud

- Know what the phrenic nerve

The phrenic nerve is one of most important nerves of respiration:
provides the motor innervation of the diaphragm

Injury to phrenic nerve will paralyze the diaphragm (Week 5)

-What is the EMST 150

The EMST150 is a calibrated expiratory muscle strength trainer designed specifically for individuals who want to enhance their breathing and swallow function.

-What is hyperrelexia

-Whats the newest dysarthria classification or diagnosis

-What is a stroke

Occurs when the arterial blood flow is interrupted via a blockage or the breakage of

a vessel

Stroke/CVA
• The most common cause of spastic dysarthria is a CVA.
• However, CVAs will result in spastic dysarthria only when:
• Two or more CVAs occur in certain combinations in the cerebrum.
• A single CVA occurs in brain stem where the neural fibers of each pathway are extremely close in proximity.
• A single CVA occurs in one hemisphere, and a preexisting condition has previously damaged the pyramidal and extrapyramidal tracts of the opposite hemisphere.

-Define ataxia in its general terms

- What are some surgical treatments for parkinsons

- what are those couple of neurotransmitters in the basal ganglia