Differential Diagnosis of the Dysarthrias Using the ELMS Model

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Welcome!!
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Dysarthria

* impaired *speech production*
* disturbed *neuromuscular control* of speech mechanism
* **Any or all** of the speech production processes
Speech Production Processes

- Respiration
- Phonation
- Resonance
- Articulation
- Prosody
Dysarthria......

* results from damage to CNS or PNS
* strictly impaired speech production due to neuromuscular dysfunction
* different types (classified by DAB)
  * Very important study!!
Mayo Clinic study in the 1960s
- Study with 212 patients
- Seven categories of neurological diagnoses
  - e.g., parkinsonism, ALS, “palsy”
* “Grandfather” passage
* 30 s samples scored by the 3 authors
* Judged on 38 speech/voice dimensions…
  * High pitch
  * Short phrases
  * Overall intelligibility
  * “bizarreness”
* Created several “clusters” of symptoms
“Pure” Dysarthrias

1. Flaccid
2. Spastic
3. Ataxic
4. Hypokinetiic
5. Hyperkinetic
6. UUMN
Accurate diagnosis (ELMS)

Some things to consider…..

1. **Etiology**
2. **Lesion site**
3. **Motor signs**
4. **Speech symptoms**
Flaccid Dysarthria
Damage to **LMNs**
- motor neurons in PNS
- *final common pathway*

**flaccidity**

“flaccid paralysis”

hyporeflexia

**NOTE:** “flaccidity” and “weakness” are NOT the same!
Etiologies of FD

- **Physical trauma:**
  - surgeries
  - TBI
- **Brainstem stroke:**
  - near cranial nerve nuclei
Etiologies of FD

- **Myasthenia Gravis:**
  - affects neuromuscular junction
  - antibodies damage Ach receptors
  - rapid fatigue of muscle contractions
  - recovery after a rest
Etiologies of FD

* Tumors:
  * brainstem
  * along CN
Speech Characteristics

- **Resonance:**
  - hypernasality (vowels)
  - nasal emissions (consonants)
  - weak pressure consonants
  - shortened phrases
  - **CN X** (pharyngeal branch)
**Speech Characteristics**

* **Articulation:**
  * imprecise consonants
  * mild to unintelligible
  * CNs V, VII, & XII
Speech Characteristics

* **Phonation:**
  * breathiness *(continuous)*
  * inhalatory stridor
  * harsh quality
  * monopitch
  * monoloudness

* **CN X:**
  * external superior laryngeal n.
  * recurrent laryngeal n.
Speech Characteristics

- **Respiration:**
  - reduced loudness
  - monopitch
  - monoloudness
  - harsh voice quality
  - shortened phrase length
  - **C & T spinal nerves**
  - may affect prosody

- **Prosody:**
  - monopitch
  - monoloudness
Spastic Dysarthria
Neurological Basis of SD

- damage to **UMNs:**
  - motor neurons in **CNS**
  - **bilateral** damage
  - pyramidal & extrapyramidal tracts that serve speech muscles
Pyramidal System:
- originate in PMC
- synapse w/ LMNs @ brainstem
- direct activation pathway
- corticobulbar tract serves speech muscles
Extrapyramidal System:
* fibers originate in cortex & brainstem
* make numerous connections
* eventually synapse w/ LMNs
* indirect activation pathway
* maintains posture, regulates reflexes, monitors muscle tone
* works in parallel with pyramidal system
**Signs of UMN Lesions**

- **SD**: bilateral damage to both pyramidal and extrapyramidal fibers that innervate speech muscles
- **Combination** of symptoms:
  - weakness
  - slow movements
  - spasticity (hypertonicity)
  - hyperreflexia

**Table 5-1** Direct and indirect activation pathway signs of UMN lesions.

<table>
<thead>
<tr>
<th>Damage to</th>
<th>Direct activation pathway (pyramidal tracts)</th>
<th>Indirect activation pathway (extrapyramidal tracts)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of fine, skilled movement</td>
<td>Increased muscle tone</td>
<td></td>
</tr>
<tr>
<td>Hypotonia</td>
<td></td>
<td>Clonus</td>
</tr>
<tr>
<td>Weakness</td>
<td></td>
<td>Spasticity</td>
</tr>
<tr>
<td>(distal &gt; proximal)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent abdominal reflexes</td>
<td>Decorticate or decerebrate posture</td>
<td></td>
</tr>
<tr>
<td>Babinski sign</td>
<td>Hyperactive stretch reflexes</td>
<td></td>
</tr>
<tr>
<td>Hyporeflexia</td>
<td>Hyperactive gag reflex</td>
<td></td>
</tr>
</tbody>
</table>

*UMN*: upper motor neuron.
Etiologies of SD

* **Stroke:**
  * most common cause of SD
  * single stroke in **brainstem**
  * or, single stroke with or following one in the **other hemisphere**
  * or, **pre-existing condition** on other side
Etiologies of SD

- **TBI:**
  - cortical
  - subcortical
  - brainstem
  - herniation
  - hemorrhages
  - hematomas
Etiologies of SD

- Brainstem Tumors:
  - compress or destroy UMNs or CN nuclei
Etiologies of SD

- **Cerebral Anoxia:**
  - possible widespread damage to both sides of brain
Etiologies of SD

- Viral or Bacterial Infections:
  - e.g., meningitis
Articulation:

- imprecise consonants
- incomplete articulatory contacts
- incomplete clusters
* **Phonation:**
  * harsh voice quality
  * friction through partially open glottis
  * “strained-strangled” voice
  * low pitch
  * spasticity of TA muscle
Speech Characteristics

- Resonance:
  - hypernasality (vowels)
Speech Characteristics

**Prosody:**
- monopitch
- monoloudness
- short phrases
- slow speech rate
  - reduced speed of articulators
  - reduced ROM
  - weakness
  - short phrases
Additional Characteristics

* Pseudobulbar affect
  * “emotionally labile”
  * UMN damage
* Drooling
* Positive Babinski sign
  * bilateral in SD
Pathologic reflexes:

- suck
- snout
- jaw jerk
Unilateral Upper Motor Neuron Dysarthria
Neurological Basis of UUMN Dysarthria

- **damage to UMN**s: motor neurons in **CNS**
- **unilateral** damage
- mainly affects muscles of **contralateral lower face & tongue**
- most other head/neck muscles receive **bilateral innervation** from cortex
**Etiologies**

- **Stroke:**
  - most common cause of UUMN
  - *91% of cases* (Duffy & Folger, 1986)
Speech Characteristics

* **Articulation:**
  * imprecise consonants:
    * weakness
    * reduced ROM
    * decreased fine motor control of tongue & lips
  * **AMRs:**
    * slow
    * imprecise
Prosody:

- slow speech rate
- reduced speed of articulators
- reduced ROM
- weakness
Additional Characteristics of UUMN Dysarthria

- Pseudobulbar affect
  - “emotionally labile”
  - UMN damage
- Drooling (one side of mouth)
- Positive Babinski sign
  - contralateral to lesion
Ataxic Dysarthria
Cerebellar damage
- or its pathways
- **Ataxia** = incoordination
- Coordinates **timing & force** of muscular contractions
- Makes sure that **skilled, voluntary movements** are appropriate for intended task
Cerebellar Ataxia:
- disequilibrium
- nystagmus
- intention tremor
- hypotonia
- problems with motor learning
Etiologies of AD

* Degenerative Diseases:
  * Friedrich’s ataxia:
    * progressive
    * hereditary
    * symptoms appear in late 20s
Etiologies of AD

* Stroke:
  * occlusions
  * ruptured aneurysms
  * AVMs
  * cerebellar arteries
Etiologies of AD

- **Toxic Conditions:**
  - lead, mercury, cyanide, etc.
  - h/o ETOH
  - phenytoin (Dilantin)
  - metabolic conditions:
    - vitamin deficiencies
    - hypothyroidism
Etiologies of AD

* TBI:
  * cerebellum
  * cerebellar control circuit
Etiologies of AD

- **Tumors:**
  - cerebellum
  - cerebellar control circuit
movements of speech mechanism are **poorly coordinated**

* **timing & force** of muscular contractions for clearly articulated speech
* “drunken speech”
* “inebriated speech”
* “scanning speech”
Speech Characteristics

* **Articulation:**
  * imprecise consonants
  * slurred
  * **irregular**
  * inconsistent errors
  * vowel distortions
Prosody:
- equal & excess stress
- prolonged phonemes
- prolonged interval between phonemes
- slow speech rate
  - increased PT and/or AT
- monopitch
- monoloudness
Hypokinetic Dysarthria
Hypokinetic Dysarthria

* Hypokinesis, but not hypotonia
  * less movement, but more muscle tone
* Vast majority of cases due to parkinsonism
Characteristics of Parkinsonism

* Collective term for a group of disorders with many of the same symptoms
* Neurologist’s diagnosis based on TRAP:
  * Tremor
  * Rigidity (e.g., rigid facies)
  * Akinesia
  * Postural Instability
Parkinsonism

- Damage to BG and/or its control circuit
- Reduction of dopamine in the striatum (i.e., caudate nucleus + putamen)
- Striatum depends on dopamine from the substantia nigra to function properly
Idiopathic form of parkinsonism
* Most common form of parkinsonism & of HD
* High prevalence of **speech deficits**; 60-80% (Adams, 1997)
Neurons in Substantia Nigra

The pars compacta region of the substantia nigra in the normal brain appears dark because dopamine-producing neurons are highly pigmented; as neurons die from Parkinson’s disease, the color fades.
## The Most Common Speech Production Errors in 32 Individuals With Hypokinetic Dysarthria (DAB)

<table>
<thead>
<tr>
<th>Rank</th>
<th>Speech Production Errors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Monopitch</td>
</tr>
<tr>
<td>2</td>
<td>Reduced stress</td>
</tr>
<tr>
<td>3</td>
<td>Monoloudness</td>
</tr>
<tr>
<td>4</td>
<td>Imprecise consonants</td>
</tr>
<tr>
<td>5</td>
<td>Inappropriate silences</td>
</tr>
<tr>
<td>6</td>
<td>Short rushes</td>
</tr>
<tr>
<td>7</td>
<td>Harsh voice quality</td>
</tr>
<tr>
<td>8</td>
<td>Breathy voice (continuous)</td>
</tr>
<tr>
<td>9</td>
<td>Pitch level</td>
</tr>
<tr>
<td>10</td>
<td>Variable rate</td>
</tr>
</tbody>
</table>
The Hyperkinetic Dysarthrias
Hyperkinetic Dysarthria

* **Hyperkinesis:**
  * too much movement
  * excessive **involuntary** movements
* HD is a **group** of MSDs, each associated w/ a **hyperkinesia**
* SLPs diagnose the dysarthria, **not** the movement disorder
Neurological Basis of HD

- Damage to basal ganglia
- Imbalance of dopamine & Ach
- PD patients given too much L-dopa often develop hyperkinetic movements

When dopamine-producing neurons die, loss of dopamine release in the striatum causes the acetylcholine producers there to overstimulate their target neurons, thereby triggering a chain reaction of abnormal signaling leading to impaired mobility.
Etiologies of Hyperkinetic Dysarthria
Chorea

- Rapid, involuntary movement
- limbs, trunk, head, neck
- “chorea” = dance
Chorea

- **Sydenham’s Chorea:**
  - “St. Vitus Dance”
  - rare, idiopathic
  - childhood
  - 40% have HD of chorea
Chorea

- Huntington’s Disease:
  - progressive
  - degeneration of neurons in BG & cerebral cortex
  - onset in middle age
  - intellectual deficits
  - dementia
  - chorea
  - dysphagia
  - hyperkinetic dysarthria
Speech Characteristics of HD of Chorea

- **Prosody:**
  - prolonged intervals
  - variable speech rate
  - monopitch
  - inappropriate silences
  - monoloudness

- **Articulation:**
  - imprecise consonants:
  - distorted vowels
  - prolonged phonemes
Phonation

- harsh voice quality
- excess loudness variations
- strained-struggle voice
- breathy voice (intermittent)
- voice stoppages
Essential (Organic) Tremor

* idiopathic
* tremulous movements
* faster than tremor in PD
* action (or intention) tremor
* no other neurological signs

**Essential Voice Tremor:**
* tremulous, quavering voice
* rhythmic, involuntary contractions of laryngeal muscles
Dystonia: Spasmodic Dysphonia

- many features of a **focal dystonia**
- involuntary VF movements **during phonation**
- contractions are **vigorous** and **active**
- **AD SD** > strained-strangled
- **AB SD** > breathy or aphonic
- **Mixed SD**
- **spontaneous, involuntary** vocalizations are usually free of contractions
- **may** be related to a BG disorder
Case Analysis Time!

1. Etiology
2. Lesion site
3. Motor signs
4. Speech symptoms
A 29-year-old woman presented to a rehabilitation unit 14 months after cerebral anoxia that developed secondary to cardiac arrest during a tubal ligation. Neurologic exam revealed neck and left upper extremity rigidity, and weakness in all extremities. Gait was slow with short steps. She had difficulty with chewing and swallowing and frequently choked on solid foods. Speech evaluation revealed reduced loudness, imprecise articulation, accelerated speech rate, little variation in pitch, loudness and syllable duration, and reduced range of articulatory movement. Speech AMRs were “super fast and blurred.”
ELMS Analysis

* **E:** Cerebral anoxia; r/o FD and AD, and possibly UUMN (anoxia usually affects both hemispheres). No other types r/o yet.

* **L:** Cerebrum, although a more specific site of lesion is not given. SD, hypo, and/or hyper are all possibilities at this point.

* **M:** Neck and L UE rigidity, weakness in all extremities; gait slow w/ short steps. Symptoms of hypokinesia; suspect hypokinetic.

* **S:** Reduced loudness, imprecise artic, accelerated speech rate, little prosodic variation, reduced range of articulatory mvt; speech AMRs “super fast and blurred.” Reduced ROM and excessive speech rate c/w hypo. Other deficits are also c/w it, though they occur w/ other types as well.
Diagnosis...

- Hypokinetic dysarthria
A 63-year-old woman was hospitalized for evaluation and treatment of cardiovascular problems. She had a history of myocardial infarction and had coronary bypass surgery 6 mo. previously. Three weeks before admission, she developed sudden onset of speech difficulty and problems with gait. She had no difficulties with language, chewing, or swallowing. OME was normal. Speech was characterized by irregular articulatory breakdowns, irregular speech AMRs, and unsteady vowel prolongation; intelligibility was normal.
ELMS Analysis....

* E: Unknown, but h/o cardiovascular issues suggests a CVA. No types of dysarthria r/o yet.

* L: Unknown

* M: Problems w/ gait; no difficulties chewing or swallowing; normal OME. Suggestive of cerebellar ataxia, as a normal OME would be highly unlikely in cases of spasticity, flaccidity, rigidity, or hyperkinesias. Also, cerebellar ataxia is often characterized by gait difficulties; AD suspected.

* S: Irregular articulatory breakdowns, irregular speech AMRs, and unsteady vowel prolongation; normal intelligibility. Highly suggestive of AD, as irregularity of speech characteristics c/w incoordination of speech musculature.
Diagnosis...

- Ataxic dysarthria
THANK YOU