Lateral Medullary Syndrome (LMS) and Dysphagia: Literature Analysis and Case Studies

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Other Terms in the Literature

- Wallenberg Syndrome
- Lateral Medullary Infarct (LMI)
- Most common form of a Brainstem Stroke
- Posterior Circulation Stroke
- Vertebrobasilar Stroke
Goals For Today

Attendees will be able to:

1. Identify the classic signs and symptoms of LMS during a Clinical Bedside Swallow Evaluation, especially how it relates to voice, swallowing and oral-motor function.
2. Discuss the aspects of the pharyngeal dysphagia, in addition to debating the cause of the cricopharyngeal/UES dysfunction.
Goals For Today

Attendees will be able to:

3. Identify methods that prevent aspiration and aide in the rapid recovery of swallowing function.
   - Specifically, we will discuss head turns, the Mendelsohn Maneuver, bolus volume, and the Shaker Exercise.
Effects of this stroke need not be devastating when the Speech-Language Pathologist is involved immediately post-onset.
Nucleus ambiguus – motor 9 + 10

Medullary syndrome:

| Lateral | Medial |


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Outline of the Presentation

- Historical Perspective
- Onset & Symptoms
- Case Example #1
- Prognosis
- Challenges in Early Detection & Differential Diagnosis
- Examples of ED presentations

- LMS Dysphagia Research with Instrumental Exams
- Case Example #2
- Case Example #3
- Oropharyngeal and Esophageal Dysphagia
- UES function and dysfunction
- Treatment Ideas
Historical Perspective
Wallenberg Syndrome

Initially described by Gaspard Vieusséux, MD, of Geneva in 1810

“Vertigo, unilateral facial numbness, loss of pain and temperature appreciation in the opposite limbs, dysphagia and hoarseness, minor tongue involvement, hiccups (cured by the taking of the habit of a morning cigarette) and a dropped eyelid”

(Pearce, 2000, p. 570)
Wallenberg Syndrome

Vieusseux also noted that a bottle of an “etherized julep” felt lukewarm when taken in the right hand but cold when held in the left hand.

(Marcet, 1811, p. 221)
Wallenberg Syndrome

In 1811, Dr Alex Marcet published a highly detailed case study of Gaspard Vieusseux.

Peculiar sensations and the derangement of equilibrium arise from a nervous state, rather than an “organic affection of the brain.”

(Marcet, 1811, p. 233)
Marcet did report the sudden onset of LMS:

- Vomiting, followed by complete loss of voice (without affecting articulation)
- Difficulty swallowing liquids
- Weakness of his left side
- Insensible right side to being scratched or pricked
- Hiccups by the third day
Adolf Wallenberg

From 1895 to 1922, he provided case reports and anatomical evidence of lesions

(Pearce, 2000)
Dysphagia to solids and liquids

Wallenberg described how the patient could not swallow solids at all.

Liquids went down in “minute amounts.”

(Robbins & Levine, 1993, p. 43)
Onset and Symptoms of Wallenberg Syndrome
J.S. Kim (2003) performed the first large clinical-MRI correlation study, which followed 130 patients with pure LMI.
Kim's Findings on Onset

• Sudden onset 75% of the time
• Non-sudden onset 25%
• Typically starts with: headache, vertigo, dizziness, and gait ataxia
• Later symptoms: dysphagia, hoarseness, hiccups, and sensory signs (Hypalgesia - loss of pain and thermoanesthesia – loss of temperature sensation)
Sensory Symptoms in 96% of Patients

- Ipsilateral trigeminal and face, Contralateral limb and body
- Contralateral trigeminal/face – Contralateral limb/body
- Bilateral trigeminal/face – Contralateral limb/body
- Or symptoms only in the face or only in the limb/body

(J.S. Kim, 2003)
Signs and Symptoms

- Ataxia in 92%. (Limb ataxia 55%)
- Dizziness in 92%
- Horner's sign in 88% (constricted pupil, ptosis, decreased sweating in half of the face)
- Dysphagia 65%
- Hoarseness 63%
- Vertigo 57%
- Nystagmus 56%
- Nausea and Vomiting 52% (J.S. Kim, 2003)
Headache in 52% of Patients

- Ipsilateral occipital region was the most common
- Frontal headache was second most common
Less Common Symptoms

- Skew deviation
- Diplopia
- Dysarthria
- Facial Paresis
- Gaze deviation
- Loss of vibration sense (12%)
Older Studies

Many were performed with smaller numbers of participants and with concomitant features of previous CVA’s, TIA’s, or cerebellar involvement.

Similar results in the following:

- Sacco et al. (1993) 33 patients
65 year old man evaluated 12 weeks post with a R rostral dorsal LMI:

- Ipsilateral facial paresis, ipsilateral palatal paresis
- Crossed sensory signs
- R-sided Ataxia, incoordination, R Horner's
- Hoarseness, profound dysphagia

Martino, Terrault, Ezerzer, Mikulis, & Diamant (2001)
<table>
<thead>
<tr>
<th>Signs Common with LMS</th>
<th>Structures Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral loss of pain and thermal sense over half of face</td>
<td>Spinothalamic tract and descending nucleus and tract of cranial nerve V</td>
</tr>
<tr>
<td>Ipsilateral Ataxia, falling to side of lesion</td>
<td>Inferior cerebellar peduncle, spinocerebellar tract</td>
</tr>
<tr>
<td>Ipsilateral Horner’s syndrome</td>
<td>Descending sympathetic tract</td>
</tr>
<tr>
<td>Dysphagia, dysarthria, ipsilateral paralysis of palate and vocal cord, diminished ipsilateral gag reflex</td>
<td>Efferent fibers of cranial nerves IX and X, and nucleus ambiguus (NA)</td>
</tr>
<tr>
<td>Contralateral impaired pain and thermal sense over half of limbs and trunk</td>
<td>Spinothalamic tract and nucleus of cranial nerve V carrying pain and temperature sense to the opposite side of the body</td>
</tr>
<tr>
<td>Vertigo/dizziness</td>
<td>Vestibular nuclei, vestibulo-cerebellar pathway in the inferior cerebellar peduncle (J.S. Kim, 2000)</td>
</tr>
<tr>
<td>Nausea/vomiting, nystagmus, diplopia</td>
<td>Vestibular nuclei (J.S. Kim, 2000)</td>
</tr>
</tbody>
</table>
Note on Chart:


- Last two rows are additions made to the original chart in the publication.
When we are familiar with the classic signs of Wallenberg Syndrome, we can assist the medical team with differential diagnosis.
CASE EXAMPLE #1

SLP ~ critical member in critical care
Vascular Supply
Rostral versus Caudal
Cerebellar Infarct with LMI

47 y/o Female

- MRI report initially only noted several acute infarcts in L cerebellum & cerebellar flocculus.

- However, Neurologist and SLP noted diplopia, horizontal nystagmus in extreme right gaze, and dysphagia to solids.
Beginning of Hospital Course

- **ONSET**: Sudden dizziness, Nausea/Vomiting, Left side weak & numb.
- **Bedside Swallow Evaluation**:
  - Pushed down solids with sips of liquids.
  - Needed moist ground.
  - Was using large bites and effortful swallows.
Finding the Lateral Medullary Lesion

- SLP reviewed MRI with radiologist due to suspicion of a LMI. There was a small infarct in the caudal medulla.
- Research shows that small superficial infarcts in the LOWER or CAUDAL medulla have less dysphagia.
Patient had MRA the Next Day Due to Small LMI

MRA Brain Findings:
• No flow in L Vertebral Artery which is likely occluded. Otherwise symmetric branching in intracranial circulation. Circle of Willis is patent.

MRA Neck Findings:
• Left VA is small in caliber ("string sign"). V3 & V4 segments are not visualized indicating slow flow or thrombosis. Suggestive of VA dissection.
No VA on Left

No PICA on Left
MRA – VA Dissection or Thrombosis on Left Side
MRA Neck: String Sign in VA

Distal thrombosis or slow flow
Started Heparin Drip

- Dysphagia to solids resolved by next day.
  - Upgraded to regular solids.
  - Denied any difficulty with bolus clearing on one swallow. No need to wash down with liquids. MBSS cancelled per ICU team.

- Minimal dizziness, no diplopia, no nystagmus, no further nausea.

- Heparin drip increased blood flow in VA or increased collateral blood flow to medulla
Vascular Supply

- *Lateral medullary arteries* arise from the VA and the PICA
- 73% of patients with LMI had vertebral artery disease (Sacco et al., 1993)
- 29% of the patients with LMI had VA dissection (Kameda et al., 2004)
J.S. Kim's Research on VA vs PICA

Kim's large study in 2003 found:

- **VA** disease was present in 67% of patients
- **PICA** disease in 10%
- Short-segment VA disease (<2cm) was most associated with the classic diagonal band-shaped lesions confined to the lateral medulla.
Negative MRA Results

• MRA frequently fails to provide reliable and precise information on the PICA (J.S. Kim, 2000; J.S. Kim, 2003; J.S. Kim et al., 1998).

• Normal MRA findings may mean that the exact pathogenesis is unknown (J.S. Kim, 2000).

• Normal angiograms frequently had a cardiac source of embolism or an occlusion that then re-canalized at the time of the angiography (J.S. Kim et al., 1998).
Notes on Rostral vs Caudal LMI

• Rostrally located lesions: dysphagia, facial paresis, and dysarthria more often (J.S. Kim, 2003)

• Increased dysphagia in rostral lesions (Kameda et al., 2004)
Notes on Rostral vs Caudal LMI

- J.S. Kim et al. (1994) noted that all patients in the rostral group had more hoarseness, dysphagia & needed nasogastric tube insertion for feeding.
- Rostral lesions: tend to be thicker/deeper
- Caudal lesions: tend to be superficial
  Only 2/21 caudal-lesion patients had transient dysphagia.
Prognosis
Prognosis for LMI Patients

• Only 1 patient in 130 died during the hospitalization. Overall excellent prognosis. (Kim, J.S., 2003)

• Progress was “rather slow, although steady” (Aydogdu, et al., 2001, p. 2084)

• Dysphagia initially severe, but improves rapidly in 1-2 months. Due to early treatment, none of the patients in the treatment program developed pneumonia. (Kim, H., et al., 2000)
Challenges in Early Detection and Differential Diagnosis
Finding the Lesion

The CT is unable to detect brainstem infarcts due to bony artifacts that obscure the anatomic details

(Sacco et al., 1993; J.S. Kim et al., 1994)
Younger Mean Age of Onset

- Researchers have recorded mean ages from 57 to 60 years old, with as young as 24 years old in one study and 37 years old in another.

- More common in men than women.

(Sacco et al., 1993; Aydogdu et al., 2001; Kameda et al., 2004; J.S. Kim, 2003)
Presentations to ED

The patient may be misdiagnosed with:

• Gastrointestinal infection due to the nausea/vomiting
• Respiratory infection due to the hoarseness and cough from dysphagia
• Drug overdose or alcohol intoxication from “bizarre” behavior and ataxia
Mimics Other Disorders

• J.S. Kim (2000): 3 patients with LMI with isolated vertigo and ataxia
  Could mimic labyrinthine disorders

• H. Lee & Sohn (2002): patient had only horizontal nystagmus on R gaze & **axial lateropulsion** (compelling sensation of being pulled toward the side of the lesion)
Examples of ED Presentations and Clarification of Symptoms...
Presentation to ED

• Sudden onset of altered mental status, bizarre behavior, unsteady gait, change in speech & voice, inability to eat/drink.
• “Mumbled,” and “garbled speech” per notes.
• Blurred vision, dizziness, nausea/vomiting
• History of Schizophrenia. Pt stated: “I took too many of the wrong pills.”
• Given charcoal absorbital liquid (a liquid slurry) and went into respiratory distress
• Intubated/ventilated until day 2.
36 y/o Vietnamese Male
HTN, smoker, Schizophrenia

Clarification of Symptoms
• Ataxia, leaning right
• Weak right side on transfer per nurse
• Dysarthria per interpreter
• No facial asymmetry
• Doctor noted nystagmus
• Decreased gag bilaterally
• Hoarseness started one week PTA, sustained “ah” = 2 seconds
• Lingual deviation to right with moderate weakness
56 y/o Vietnamese Male
HTN, diabetes, smoker

Presentation to ED

- Nausea, vomited twice in the ED, 5x total
- Headache all day
- Left facial droop
- Dizziness, unsteady gait
- “Scratchy throat” per ED notes, cough
- Initial ED diagnosis: “dizziness and upper respiratory infection”
Clarification of Symptoms on day two

- Progressive headache that started 4 wks prior
- Diplopia started night of admission
- Numbness, decreased sensation on right upper and lower extremities
- Horner’s syndrome (L eyelid), nystagmus
- Absent gag bilaterally
- Dysphagia, choked on soup on Day 1
- Hoarse, “ah” < 2 seconds
- Lingual deviation to right
We are DETECTIVES!

• Use interpreters
• Gather background info and interview patient and family
• Perform thorough bedside exams that address: oral-motor status, voice, speech, language, cognition, and swallowing
• Add more sensory testing—specifically cold sensation
Literature shows Dysphagia Occurs with LMI at 53-65%

• However, much of the literature has poorly classified dysphagia
• Rarely using videofluoroscopy
• Definitions of dysphagia are unclear

Kwon, Lae and J.S. Kim (2005)
LMS Dysphagia Research with Instrumental Exams
Kwon, Lae and J.S. Kim (2005)

- Studied 46 patients total; 37 with LMI & 9 with MMI (Medial Medullary Infarction)
- Importance of instrumental exam due to silent aspiration. Used Videofluoroscopy.
- **MMI**: problems in the timing of the hyolaryngeal elevation
- **LMI**: problems in the excursion of the hyolaryngeal elevation

**Direct involvement of the Nucleus Ambiguus (NA)**
Martino et al. (2001)

MBSS at 3, 9, 14, 27 months post LMI:

• **3 MONTHS**: Normal A-P propulsion, but absent pharyngeal swallow

• **9 MONTHS**: Anterior and vertical elevation of larynx only slightly reduced, but epiglottic deflection absent. No contraction of middle and inferior pharyngeal constrictors. UES failed to open. Boluses pooled in hypopharynx. Penetration.
Martino et al. (2001)

• **14 MONTHS**: Implemented a volitional elevation of the larynx to clear bolus through UES. Piecemeal swallows effective with liquids and soft solids.

• **27 MONTHS**: More consistent epiglottic deflection for airway protection. More efficient pharyngeal phase, but still impaired. Still needed “volitional laryngeal elevation.”
Vigderman et al. (1998)

- Studied 57 year old:
- L rostral dorsolateral medullary infarct with aphagia, using MBSS and Manometry.
- Small unilateral lesion can cause a severe bilateral pharyngeal paresis and aphagia
• **DAY 11**: Weak pharyngeal contractions (peak 11.6 mm Hg, Normal is 40-250 mm Hg)

• **2 MONTHS**: Pharyngeal contraction was improved, but occurring before UES relaxation. Poorly coordinated swallowing pattern. Able to take only thick liquids.

• **20 MONTHS**: mild pooling on MBSS. Manometry still showed the peak pharyngeal contraction occurring before the full UES relaxation. PEG removed. Patient on a regular diet.

- Studied 23 LMI patients
- 44% had full aspiration, additional 3 patients penetrated. (Told to hold the bolus)
- No aspiration was noted in patients with lower level medullary lesions (inferior-dorsal lesion), as the dorsal region of the lower medulla does not contain the NA
- Combined lower and middle lesions were all aspirators
Aydogdu et al. (2001)

- Studied 20 patients with LMS
- 9 of the 20 patients needed non-oral feeding
- 95% had some dysphagia. Only 1 out of the 20 exhibited no clinical s/sx of dysphagia.
- EMG testing: laryngeal sensors detected
  - the onset and duration of the swallow response,
  - the upward laryngeal movement,
  - variations in the swallow response time, and
  - pathological use of piecemeal swallows
Aydogdu et al. (2001)

- 85% of the 20 patients had hoarseness
- 100% had vocal cord paresis
- Weak cough 80% of the time
“Dysphagia Limit”

- Duplication of swallowing or piecemeal swallowing with <20 ml of water in ALL patients with LMS
- 40% of LMS patients could handle only 1 ml of water on one swallow
- Dysphagia Limit was consistently lower for the LMS patients (in contrast with the patients with hemispheric CVA or 9\textsuperscript{th} and 10\textsuperscript{th} cranial nerve palsy).
Extremely Delayed Pharyngeal Swallow Response

- Severity of the dysphagia was greater in LMI, when compared to patients with unilateral CVA and 9th/10th cranial nerve palsy.
- Greater variation in duration of laryngeal movements.
- Although a medullary lesion is unilateral, its effect on swallowing is bilateral.
Unilateral LMI produces bilateral pharyngeal and laryngeal dysfunction per electrophysiological evidence, making the dysphagia severe.

(Aydogdu et al., 2001)
“Acute disconnection syndrome”

- Between the 2 halves of swallowing centers.
- Between the NTS and NA
- Between the medullary pre-motor neurons (located in NTS, NA, surrounding reticular formation) and the synaptic connections with peripheral afferents through cranial nerves.
- Between the medulla and cortical swallowing-associated areas.

Aydogdu et al. (2001)
“Acute disconnection syndrome”

Disconnection and disruption results in:

- Central Pattern Generator (CPG) for swallowing to NOT perform it's sequential muscle activity
- Severely incoordinated muscle activity
- Prolonged pharyngeal phase
- Even caused bilateral dysfunction of the submental muscles (laryngeal elevators)
Central Pattern Generators (CPG)

- Swallowing is a patterned sequence of motor activity that results from the bilateral input from the swallowing centers in the rostral dorsolateral medulla.
- Swallowing centers: nucleus of the solitary tract (NTS), nucleus ambiguus (NA), and the reticular formation.

Vigderman et al. (1998); Aydogdu et al. (2001)
Martino et al. (2001) on the CPG

• “Serial network of linked neurons within the nucleus of the solitary tract (NTS) and neighboring reticular formation” (p. 423)

• Sequential excitation rostrally to caudally along the “deglutition pathway.” Activates the cranial nerve motor neurons (NA and the vagal dorsal motor nucleus), which in turn innervate the muscles of swallowing.

• **Dysphagia in LMS**: lesion in the area that connects the CPG (which is more dorsal and medial in the brainstem) to the NA (which is deeper and more lateral in the brainstem).
• Neural networks that are flexibly organized and multifunctional.
• This flexible organization coordinates the swallowing activity, rather than containing all the neural circuitry to initiate a pharyngeal swallow response.
CASE STUDY #2
Pure LMI
Presentation to ED

- Fatigue, Ataxia, R-Sided weakness x3 days
- Slurred speech
- Nausea/vomiting x5 days
- Symptoms started one week prior to admission, but progressed
- Head CT Negative
- Initial ED diagnosis: recurrent hyponatremia, daily ETOH
66 y/o Caucasian Male
HTN, COPD, ETOH

Clarification of Symptoms per SLP

- Hoarseness, unable to sustain phonation
- Not managing secretions, wet voice
- Mild ipsilateral palatal weakness, absent gag
- Overt s/sx aspiration with thin and nectar thick by cup
- Ipsilateral loss of thermal sense on R face and contralateral loss of thermal sense on L UE/LE
MRI Findings: R dorsal-lateral medullary infarct
MRA: Non-visualization of
R VA
consistent with occlusion
R PICA
MRA of Brain

- Maximum Image Projection (MIP).
- Absent Right Vertebral Artery
MBSS 8 Days Post-onset

- Moderate Dysphagia
- Pharyngeal swallow delay with spillage to pyriforms with thin and nectar thick
- Decreased pharyngeal peristalsis: moderate-severe residue after swallow. Residue bilateral, but R > L
- Moderately decreased laryngeal elevation with minimal anterior hyolaryngeal excursion
- UES Dysfunction
- Silent penetration with thin and nectar thick
$1^{st}$ bolus: Paste/Puree - with no strategies cued
Larger bolus of puree with strategies:
Thin liquid by cup with head turn and tuck to right
Meat vs. Cracker

Effortful Swallow
Ground Meat

Mendelsohn Added
Cracker
Bread - with strategies
MBSS Recommendations

- **Honey** thick liquid by cup: safer to use the larger bolus sizes for more effective UES distention.


- Medication: embedded in applesauce with liquid washes.

- Acute Rehab. Shaker Exercises.

- Repeat MBSS per SLP within 2 weeks.
CASE STUDY #3
56 y/o Vietnamese Male
HTN, diabetes, smoker

Presentation to ED

• Nausea, vomited twice in the ED (Total 5x)
• Headache all day
• Left facial droop
• Dizziness, unsteady gait
• “Scratchy throat” per ED notes, cough
• Initial ED diagnosis: “dizziness and upper respiratory infection”
Clarification of Symptoms

• Progressive headache that started 4 wks prior
• Diplopia started night of admission
• Numbness, decreased sensation on right upper and lower extremities
• Horner’s syndrome (L eyelid), nystagmus
• Absent gag bilaterally
• Dysphagia, choked on soup on Day 1
• Hoarse, “ah” < 2 seconds
• Lingual deviation to right
MRI Images:

- DWI Image
- ADC map
“Small area of restricted diffusion within the left dorsal mid/lower medulla, with corresponding T2 hyperintensity in the region” (left: T2 picture)
MRI showed a combined lower and middle lesion, and he had significant aspiration issues on his MBSS.

According to the study by H. Kim et al. (2000), patients with this type of large LMI were all aspirators.
Day 2 Clinical Bedside Swallowing Evaluation

• Appeared to initially tolerate puree. Then overt s/sx of aspiration with prolonged distress with honey thick by spoon, and delayed sensation of residue.

• Expectoration of all puree and honey trials bedside after 10 min, showing that it must have pooled in pharynx.

• NPO that weekend
Day 6 MBSS

• Moderate oropharyngeal dysphagia
• Delayed pharyngeal swallow
• UES dysfunction, decreased UES opening, proximal esophageal stasis
• Aspiration with thin and nectar
• Tolerated puree and honey thick liquid with head turn and tuck to L (weaker side). Effortful swallow. Double swallow to clear residue above UES.
MBSS at 3 months post-stroke

- Minimal oropharyngeal dysphagia.
- Mildly delayed pharyngeal swallow, mildly reduced laryngeal elevation.
- Penetration above the VC with thin liquids, but ejected. No aspiration, even with large sips.
- Improved anterior excursion of larynx and no residue above the UES with solids.
- Minimal residue in the proximal esophagus.
Good anterior hyolaryngeal excursion. Good UES opening.
Piecemeal swallow, but good UES opening and distention.
Thin liquid

No penetration
Oropharyngeal Dysphagia, or Oropharyngeal PLUS Esophageal Dysphagia?
Debate: Is this just a pharyngeal dysphagia, or a combination of pharyngeal and esophageal? What is causing the UES dysfunction?
The Debate

**OPINION A:**
- Reduced laryngeal elevation and anterior excursion
- Pharyngeal weakness and decreased bolus pressure
- All deficits reduce mechanical UES opening

**OPINION B:**
- Weakness and mechanical issues are present, **PLUS**
- Cricopharyngeal muscle is actually spastic,
- Dysfunction in striated portion of proximal esophagus
Opinion A:
Decreased laryngeal elevation and bolus pressure

• Logemann (1998): CP muscle is not actually spastic. Decreased laryngeal movement & lack of bolus pressure does not allow for adequate UES opening or distention.

• Jacob et al. (1989): “UES opening seems to be an active mechanical event dependent on muscular traction to the anterior sphincter wall rather than simply a consequence of cricopharyngeal relaxation”
Logemann et. al. (1989)

- Studied head rotation in 9 normal and 5 LMS
- Abnormal swallows with LMS characterized:
  1. Barium residue unilaterally due to pharyngeal weakness
  2. Decreased laryngeal elevation during swallow
  3. Barium residue in pyriform on weaker side
- Logemann discussed Passive (CP relaxation) and Active opening forces (superior and anterior excursion of the larynx to mechanically open the sphincter segment)
Logemann's conclusion (1989)

“Although head turning was shown to diminish UES pressure in healthy subjects, and this could be of therapeutic importance in the subset of patients with defective UES relaxation or diminished compliance of the sphincter as a pathophysiologic process, the improvement observed in our (LMS) patients seems to have been more dependent on maximizing the impact of opening forces brought to bear on the sphincter.”

(p. 771)
Recall the Vigderman et al.'s (1998) Case Study

- Patient unable to initiate swallow.
- Pharyngeal manometry showed low resting pressures and weak pharyngeal contractions.
- Severe bilateral pharyngeal paresis.
- UES relaxation was normal per manometry.
- But the UES resting pressures were reduced.
- However, they found incoordinated swallowing: Pharyngeal contractions occurring BEFORE UES relaxation (similar to Aydogdu).
Opinion B:

Opinion A is true, AND the cricopharyngeal muscle and striated portion of esophagus are spastic.
Martino et al. (2001)

Motility Studies at 8 months post LMI found:

1. Absent pharyngeal contraction 1 cm above the UES,
2. No effective relaxation of the cricopharyngeal muscle,
3. No swallow activity at the level of the UES,
4. No definite contractile activity in the proximal 6 cm of the upper esophagus (striated region).
Martino et al. (2001)

- Basal UES pressure = 44 mm Hg (significant resting tone)
- However, the patient did have a discrete proximal pharyngeal contraction at 2.5 cm above the UES, as well as normal contractile activity lower in the esophagus at 8-15 cm (smooth muscle).
- LES pressure was WNL at 36 mm Hg.
10 Grain Barium Pill
Robbins & Levine (1993)

- 62 y/o female with a left LMI secondary to a left VA occlusion
- Bolus flow remained absent due to a “hypertonic cricopharyngeal muscle”
- Mendelsohn and lingual piston-thrust-swallow could override the UES high pressure zone to an extent, but it was still in the “presence of impaired cricopharyngeal relaxation”
Robbins & Levine (1993)

- **Manometry**: UES sphincter pressure was distinctly abnormal at 130-160 mmHg with incomplete relaxation

- Needed a **Cricopharyngeal Myotomy**: approach from the left side of the neck as the left true vocal fold was found to be paralyzed
Robbins & Levine (1993)

- Repeat MBSS after Myotomy:
  - Increased duration & range of motion of the UES opening
  - 90% of the small liquid bolus to pass into the UES
  - However, when the patient was coached to perform a head turn to the left, use the piston swallow, and perform the Mendelsohn maneuver, she swallowed the bolus without any post-swallow residue in the pharynx.
Hypopharyngeal suction pump (HSP) needs cricopharyngeal relaxation as a prerequisite.

Then the hyolaryngeal anterior and superior excursion can provide the traction to mechanically open the UES, resulting in a negative pressure or suction that pulls the bolus into the esophagus.

(Robbins & Levine, 1993)
If the dysphagia of LMS was caused by the oropharyngeal deficits alone, then the extensive therapy with maneuvers and strategies should have been sufficient to pass the bolus through the UES.
More Research Needed

• Pharyngeal constrictor muscle paresis: damage of the nucleus ambiguus and vagus nerve, controlling the striated muscles of the palate, the base of tongue, the pharynx, and the larynx (H. Kim, 2000)

• However, most studies do not discuss the striated muscles of the cervical esophagus as did the two studies above.
Treatment Ideas
Larger Bolus Volume

Larger boluses found to increase

- Pharyngeal wall movement
- Duration of hyolaryngeal excursion
- Superior UES movement
- A-P dimension/width of UES opening
- Magnitude and duration of intrabolus pressure
- Velocity through UES
- Duration of UES opening

(Jacob et al., 1989)
Larger Bolus Volume

- Stroke patients had decreased pharyngeal delay, increased duration of laryngeal closure, longer CP opening. Lazarus et al. (1993)
- Prolonged UES opening seen in 8 normals Kahrilas & Logemann (1992)
- Decreased pharyngeal delay time in neurologically impaired groups with increased bolus volume and viscosity. Bisch et al. (1994)
- Increased median intrabolus pressure during UES relaxation (using HRM) Ghosh, et al. (2006)
- Increase in UES opening duration (HRM) Hoffman et al. (2010)
Head Turn to Weaker Side

• Neck CT evidence of closure above the level of the pyriform sinus to the level of the hyoid (in a LMS patient). (Tsukamoto, 2000)

• Head turn more effective than chin tuck strategy for LMS:
  *Chin tuck and thickening liquids were more effective in patients with Medial Medullary Infarction, in contrast to treatment for increased range of hyolaryngeal movement and pharyngeal peristalsis for LMS. (Kwon et al., 2005)
Mendelsohn Maneuver in Normals

- Longer durations of pharyngeal contraction & longer bolus transit time. (Boden et al., 2006)
- Only the submental group (suprahyoid/laryngeal elevators = digastrics, mylohyoid, geniohyoid) demonstrated a difference with Mendelsohn per EMG findings. (Ding et al., 2002)
- Mendelsohn increases duration of UES opening, but Shaker exercise increases magnitude or diameter of UES opening. (Shaker et al., 2002)
Shaker Exercise

• Increased A-P diameter of UES opening
• Increased max anterior laryngeal excursion
• No change in:
  o hyoid bone anterior/superior excursion,
  o superior laryngeal elevation, or
  o lateral laryngeal excursion. (Shaker et al., 2002)
• Most effective in patients with aspiration after the swallow (Mepani et al., 2009; Shaker et al., 2002; Logemann et al., 2009)
Shaker Exercise

• Mepani et al. (2009): small numbers
  6 patients completed traditional therapy
  5 completed Shaker protocol
• “Contraction of the UES opening muscles is amenable to direct rehabilitation.” (p. 30)
• Increased anterior-superior hyoid excursion, allowing for increased A-P UES opening.
• **Thyrohyoid** muscle shortening + strengthening the suprhyoid muscles. The effect of shortening and contraction of the thyrohyoid had not been studied prior.
Transcranial magnetic stimulation (TMS)

- 11 LMI and 11 Brainstem acute stroke patients (pontomedullary dysfunction)
- Randomly selected to receive active vs sham
- Active rTMS: 10 trains of 3 Hz stimulation, each lasting 10 seconds. Repeated every minute for 10 minutes. 5 consecutive days.
- Positioned over “oesophageal cortical area of both hemispheres.” Intensity of 130% of the resting motor threshold (RMT), to spread as much as 2-3 cm from the coil.

(Khedr & Abo-Elfetoh, 2009)
Transcranial magnetic stimulation (TMS)

- No instrumental pre or post testing
- Patients scored with swallowing questionnaire and bedside examination
- Given a degree of dysphagia (DD) rating:
  - DD-I: no clinical signs or symptoms
  - DD-II: Very mild dysphagia without patient complaint of symptoms
  - DD-III: Patient complaint of dysphagia and clinical signs, but still taking PO intake.
  - DD-IV: Obvious signs and symptoms of dysphagia, including aspiration, and severe enough for non-oral feeding.
Transcranial magnetic stimulation (TMS)

• Tested prior to, after the 5th session, and then at one and two months post treatment.
• Five daily sessions of rTMS produced a more substantial improvement in dysphagia rating versus the sham TMS group.
• Beneficial effects lasted up to two months.
• Researchers speculated: could rTMS have sped up the natural spontaneous recovery?
• However, a LMS patient can improve in the matter of days without intervention.
Further Discussion

- Cases
- Treatment ideas
- Research ideas
Public Health Reminder

• Posterior circulation strokes present differently. Look for dizziness, nausea, vomiting. May have low NHI stroke score.
• LMS patients need early identification within the emergency department to avoid any oral intake prior to a formal swallowing evaluation.
• This can prevent aspiration, intubation and life-threatening aspiration pneumonia.
Special Thanks

• Alissa Saunders, MD – Radiologist
• John Mahoney, MD – Neurologist
• Jeanne Goodman – Software Specialist
Thank you for your attention and participation!

Questions?
UES Relaxation vs Opening

- Need to separate the Passive relaxation from the Active opening.
- Need manometry to test relaxation.
- We perceive “opening” on our MBSS.
- These are two different stages of UES functioning.
- Need further discussion of the UES before discussing treatment strategies.

(supplemental info on UES)
UES Opening

“Sum effect of the relaxation of the cricopharyngeal (CP) muscle, its pliability, and the distraction forces imparted on the sphincter.”

(Shaker et al., 1997, p. G1518)
Normal UES function per Jacob et al. (1989)

1. **Relaxation**: cessation of tonic activity of striated muscles. During laryngeal elevation. 0.1 seconds before opening.

2. **Opening**: Anterior and superior laryngeal excursion. Infrahyoid compartment shortening 0.2 seconds before the opening. Suprahyoid (also called submental) and thyrohyoid contraction. Hyoid traction on the anterior wall of the sphincter, as CP muscle is attached to the lateral aspect of cricoid cartilage. Intraluminal negative pressure → creates a suction. (Hypopharyngeal Suction Pump)
3. **Distention**: modulated by intrabolus pressures due to posterior tongue thrust, pharyngeal wall contraction, initiation of pharyngeal peristalsis. Max distention occurred within 0.13 seconds of opening.

4. **Collapse**

5. **Contraction**: closure occurs when the tail of the bolus transverses the sphincter
Background on UES - Crucial to plan treatment

- CP Myotomy for chronic impairments in relaxation. But we can affect OPENING and DISTENTION

- Jacob et al. (1989) suggested that 3 variables can be manipulated in the sequence:
  1. Diameter,
  2. Duration, and
  3. Velocity of the head of the bolus

- Dantas et al. (1990) found increased bolus volume increases anterior hyolaryngeal excursion, pharyngeal expansion, and UES opening and distention.
References


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