Lateral Medullary Syndrome (Wallenberg Syndrome) and Dysphagia:

An Analysis of the Literature and Case Studies

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Author's Note

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Contents

Abstract

To the knowledge of this author, this is the first major literature analysis and discussion of dysphagia in depth of its kind on lateral medullary syndrome. A lateral medullary infarction or Wallenberg syndrome is the most common type of a brainstem stroke, and it presents completely differently than typical cerebral vascular accidents. The effects of this infarction need not be devastating. With early identification and treatment, including early swallowing evaluations, the medical team can prevent aspiration pneumonia, intubation, and death from pneumonia. The goals of this paper are (a) to provide a review of the literature of LMS, (b) to discuss the symptoms and challenges of differential diagnosis, (c) to analyze the dysphagia research in LMS, especially how it relates to the central pattern generators (CPG) for swallowing, (d) to discuss the pharyngeal, laryngeal, and esophageal aspects of the dysphagia in LMS; and e) to review recovery, prognosis, and treatment options. Finally, the author presents two cases that illustrate points such as the younger age of onset of LMS in comparison with most stroke populations, symptomatology, early identification, the need for instrumental evaluations of swallowing, therapy techniques, and outcomes. LMS patients tend to show rapid and steady recovery of their swallowing function. The subjects of the two case studies at the end of this article made significant progress in swallowing based on a comparison between the evaluations at two days after the stroke and at just six days later.

Keywords: lateral medullary syndrome (LMS), lateral medullary infarction (LMI), Wallenberg syndrome, dysphagia, dysphonia, dysarthria, aspiration, modified barium swallow studies (MBSS), hyolaryngeal elevation, UES dysfunction, cricopharyngeal muscle.

(Note: The terms *LMS* and *LMI* will be used interchangeably throughout this article, depending on how the condition was referred to by the researcher being discussed.)

Introduction

Lateral medullary syndrome is a stroke in the lateral medulla and is also known as Wallenberg syndrome. This is the most common and classic brain stem vascular syndrome, involving the territory of the vertebral arteries (VA) and/or the posterior inferior cerebellar artery (PICA), usually affecting the dominant branch (Groher, 1992; Sacco et al., 1993)**.** Wallenberg syndrome was initially reported by Gaspard Vieusseux, MD of Geneva in 1810 at the Medical and Chirurgical Society of London. He described his own affliction as "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). Marcet (1811) published a highly detailed case study of Gaspard Vieusseux, in which he speculated that the peculiar sensations in Vieusseux's head and the derangement of equilibrium arose from a nervous state, rather than an "organic affection of the brain" (p. 233). However, Alex Marcet, MD perfectly related the sudden onset of LMS with vomiting, followed by a complete loss of voice (without affecting articulation), considerable difficulty in swallowing liquids, weakness of his left side, insensible right side to being scratched or pricked, and finally hiccups by the third day. Vieusseux also noted that an "etherized julep" (a cold cocktail) felt lukewarm when taken in the right hand but cold when held in the left hand (p. 221). Other than cures by cigarettes or sensory testing with cold cocktails, Vieusseux and Marcet hit upon the main cluster of symptoms that are consistent with the research of today. However, it was not until Adolf Wallenberg from 1895 through 1922 provided case reports and anatomical evidence of lesions in the lateral medulla that this syndrome got its name (Pearce, 2000).

Symptomatology and Anatomy

J.S. Kim (2003) performed the first large clinical-MRI correlation study, which followed 130 patients with pure LMI. He started with 222 consecutive patients who presented at the Asan Medical Center in Seoul, South Korea from 1994 to 2002 . He found that patients had a sudden onset of

symptoms 75% of the time. The non-sudden onset, 25% of the time, typically started with a headache, vertigo, dizziness, and gait ataxia. Later symptoms were the sensory signs, dysphagia, hoarseness, and hiccups. Kim found that sensory symptoms were the most frequent, as they were present in 96% of patients. The pattern of ipsilateral trigeminal/face-contralateral limb/body was the most common sensory sign, but other patients had a contralateral trigeminal-contralateral limb/body, a bilateral trigeminal-contralateral limb/body, or symptoms only in the face or only in the limb/body. The loss of the sensation of pinprick and temperature were the typical impairments, but a loss of vibration sense in the hypalgic areas was seen in 12% of patients. Other "very common symptoms/signs" were ataxia at 92%, dizziness 92%, and Horner's sign (i.e., constricted pupil, ptosis, decreased sweating on half of face) at 88%. The "moderately common symptoms/signs" were dysphagia at 65% (severe in 40%), hoarseness 63%, vertigo 57%, nystagmus 56%, limb ataxia 55%, nausea/vomiting 52%, and headache 52%. Headaches were most often in the ipsilateral occipital region, and a frontal headache was the second most common. "Less common symptoms/signs" were: skew deviation, diplopia, dysarthria, facial paresis, and gaze deviation.

Many of the older studies were performed with smaller numbers of participants and with concomitant features of previous strokes, transient ischemic attacks, or cerebellar involvement. In the Sacco et al. (1993) study, ataxia was the most common symptom in 23 out of 33 patients (70%), numbness in 21 out of 33 (64%) (with the contralateral limb 9 out of 21, ipsilateral face 8 out of 21, and both 4 out of 21), then vertigo 52%, dysphagia 53%, nausea-emesis 48%, headache 48%, dysarthria 39%, diplopia or blurred vision 33%, hoarseness 30%, hiccups 12 %, and facial pain 9%. However, percentages differ in the neurologists' initial findings which list symptoms as Horner's sign 91%, followed by contralateral decreased pain and temperature sensation (hypalgesia) at 85%, ataxia at 85%, and horizontal and rotary nystagmus, facial hypalgesia, palatal weakness, and facial weakness at 61%, 58%, 52%, and 42%, respectively.

J.S. Kim, J.H. Lee, Suh, and M.C. Lee (1994) also studied 33 patients and found that hemibody

sensory changes were the most common symptom reported in 94% of cases (facial sensory changes were 85%), followed by vertigo in 91%, ataxia 88%, nausea and vomiting 73%, Horner's syndrome 73%, nystagmus 67%, dysphagia 61%, and hoarseness 55%. They found that nausea and vomiting and Horner's syndrome were common regardless of the location of the lesion, whereas caudal lesions (lower medulla or both lower and middle) correlated more with nystagmus, vertigo, and gait ataxia. Mild central facial paresis on the ipsilateral side is not as common (36%), and most of the patients with facial paresis had rostral lesions (upper medulla or both upper and middle medulla) without pontine infarction. Headache was more common in patients with caudal lesions than in those with rostral lesions. The research of Bassetti, Bogousslavsky, Mattle, and Bernasconi (1997) also defined the upper (rostral), middle, and lower (caudal) portions of the medulla.

J.S. Kim, J.H. Lee, and Choi (1998) studied 34 patients and found similar numbers to those in the above Kim et al.'s 1994 study. In the 1998 research, the most common group of symptoms was vertigo/dizziness, gait ataxia, and Horner's sign - all at 88% - followed by nystagmus (71%), nausea/vomiting (65%), dysphagia (62%), hoarseness (41%), and sensory changes (11%).

Kameda et al. (2004) performed a large-scale study comparing lateral and medial medullary infarction in 214 patients who were treated between January 1996 and December 2000 in 35 major stroke centers in the Tohoku district of Japan. They found that the dorsolateral medullary infarct is the most common of the LMI at 36% of total, followed by the inferolateral at 27%. Sensory dysfunction was the most common at 89%, then dysarthria 75%, vertigo/dizziness 73%, Horner's 72%, ataxia 69%, diminished pharyngeal reflex 64%, and dysphagia 57%. Interestingly, the patients with medial medullary infarction (MMI) had lingual palsy 30% of the time, versus only 9% in LMI when lesions extended dorsally. The most common medial medullary symptoms were motor weakness (93%) and sensory disturbance of the extremities (68%). The researchers found that LMI were most frequently in the middle medulla, and MMI was most commonly located in the upper medulla. Overall, these percentages should be viewed with caution as there were concomitant cerebellar lesions in 34 of the

167 LMI cases. These cerebellar lesions were found mainly when the LMI was in the dorsal region (42%).

Martino, Terrault, Ezerzer, Mikulis, and Diamant (2001) presented a case report of a 65-year-old man with a right (R) superior or rostral lateral medullary infarction with the following neurological findings: R facial paresis, a crossed sensory deficit with the R face and left (L) arm, deviation of palate to L (ipsilateral paresis on R), R Horner's, R-sided ataxia, incoordination, hoarseness, and severe dysphagia. (See Table 1 below for these researchers' organized symptomatology chart.) The dysphagia was better defined in this study as the researchers performed esophageal motility studies and videofluoroscopy (also known as the modified barium swallow study or MBSS). The dysphagia was to both solids and liquids and was associated with gagging, coughing, and choking episodes. The patient localized his dysphagia to the "suprasternal region." His hospitalization was complicated by aspiration pneumonia, requiring a tracheostomy tube and a percutaneous gastrostomy tube (PEG). For further discussion on the specific characteristics of dysphagia according to these researchers and others, see the details under the heading "LMS research that has included instrumental testing."

Table 1

Features common with lateral medullary syndrome

Signs	Structures affected
Ipsilateral loss of pain and thermal sense over half of face	Spinothalamic tract and descending nucleus and tract of cranial nerve V
Ataxia, falling to side of lesion	Inferior cerebellar peduncle, spinocerebellar tract
Ipsilateral Horner's syndrome (constricted pupil, ptosis, decreased sweating)	Descending sympathetic tract
Dysphagia, dysarthria, ipsilateral paralysis of palate and vocal cord, diminished ipsilateral gag reflex	Efferent fibers of cranial nerves IX and X, and nucleus ambiguus (NA)
Contralateral impaired pain and thermal sense below the neck (UE and LE)	Spinothalamic tract and nucleus of cranial nerve V carrying pain and temperature sense to the opposite side of the body
Vertigo/dizziness	Vestibular nuclei and vestibulocerebellar pathway in the inferior cerebellar peduncle (J.S. Kim, 2000)
Nausea/vomiting, nystagmus, diplopia	Vestibular nuclei (J.S. Kim, 2000)

Note. Adapted from "Dysphagia in a Patient with Lateral Medullary Syndrome: Insight into the Central Control of Swallowing," by R. Martino, N. Terrault, F. Ezerzer, D. Mikulis, and N. Diamant, 2001, *Gastroenterology* 121, p. 421. Copyright 2001 by the American Gastroenterological Association.

Last two rows are additions made to the original chart in the publication.

Vertebral Artery (VA) and Posterior Inferior Cerebellar Artery (PICA)

The rostral dorsolateral medulla is supplied either by the posterior group branches from the medial PICA or by the lateral group branches from the VA (Tatu, Moulin, Bogousslavsky, and Duvernoy, 1996). Bassetti et al. (1997) reviewed the research on the vascular supply to the brainstem (citing research from 1916 to 1995) and created a summary of the four territories that supply the medulla. First, the *lateral medullary arteries* arise from the VA and the PICA and supply the lateral olivary nucleus, the spinal trigeminal tract and nucleus, the spinothalamic and spinocerebellar tracts, the lateral reticular formation, and the caudal cranial nerve nuclei of IX and X. Second, the *anteromedial medullary arteries* arise from the anterior spinal artery (ASA) to supply the lower medulla. However, they arise from the VA for the upper medulla and the dorsomedial medulla (which houses the hypoglossal nerve and central reticular formation). Third, the *anterolateral medullary arteries* arise from the anterior spinal artery and supply only a small territory in the ventral (anterior) aspect of the medulla which covers a portion of the pyramidal tract and the medial part of the inferior olive. The fourth and final territory is that of the *posterior medullary arteries*, arising from medial branches of the PICA (supplying the vestibular nuclei, the inferior cerebellar peduncle, the nuclei gracilis and cuneatus, and caudal cranial nerve nuclei IX and X). This posterior territory is much larger in the caudal dorsal (lower posterior) medulla than in the rostral dorsal (upper posterior) medulla. In contrast, the territory for the *lateral medullary arteries* is much larger in the rostral medulla than in the caudal medulla (See Figure 1, in Bassetti et al., 1997, p. 883). This difference between the rostral and caudal areas will be further discussed in the section titled “Dysphagia Research Related to LMS.”

Sacco et al. (1993) noted that 73% of patients with LMI had vertebral artery disease, based on combined results from angiography and vertebral duplex doppler studies. Five patients out of 33 (15%) had VA dissection, and they were all younger and had severe headache and neck pain (Sacco et al., 1993). Kameda et al. (2004) found VA dissection in 29% of the patients with LMI, naming this as an important cause of both LMI and medial medullary infarcts (MMI). According to Sacco et al. (1993),

cerebellar infarct is uncommon in patients with LMI despite VA occlusions. These researchers also noted that lateral medullary arteries that originate from the distal VA may become occluded and spare the arterial supply to the cerebellum (Sacco et al., 1993). However, Kameda et al. (2004) found that cerebellar infarcts were usually present with dorsal medullary infarcts in 42% of the total LMI cases.

The PICA is not a major supplier of the lateral medulla (Sacco et al., 1993). The majority of patients (38.2%) in the 1998 study by J.S. Kim et al. had distal VA disease (i.e., stenosis of >50% or occlusion). Only 23.5% had PICA stenosis or occlusion; 26.5% had both PICA and VA stenosis or occlusion, and 11.8% had normal angiograms. J.S. Kim's larger study in 2003 found that VA disease was present in 67% of patients, compared to PICA disease in 10%. Short-segment VA disease (<2cm) was most associated with the classic diagonal band-shaped lesions confined to the lateral medulla. These lesions present the classic symptomatology of lateral medullary syndrome. Long-segment VA disease (longer than 2 cm) caused larger lesions as noted on MRIs (J.S. Kim et al., 1998).

The magnetic resonance angiogram (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 may have been an occlusion that then re-canalized at the time of the angiography (J.S. Kim et al., 1998). Using conventional transfemoral angiography, the 1998 researchers found that lesions produced by VA disease did not appear to be significantly different from lesions due to PICA disease, stating that the territories supplied by the PICA and the VA frequently overlap (J.S. Kim, Lee, and Choi, 1998).

The etiologies of these vascular diseases are as follows according to the J.S. Kim et al. study (1998): (a) probable or possible *dissection* (i.e., sudden head/neck trauma, caused by chiropractic manipulation, yoga, golf practice, etc); (b) *atherosclerosis* (with at least one conventional risk factor); (c) probable cardiogenic *embolism* (presence of emboligenic heart disease such as atrial fibrillation, prosthetic valve, sick sinus syndrome, valvular disease, cardiomyophathy, recent myocardial

infarction); and (d) *small vessel disease* (i.e., hypertension, >50 years of age, no emboligenic heart disease, and normal angiogram). In 1993, the Korean Neurological Association found that cardiogenic embolism was the cause of stroke in only 7% of the stroke population, and J.S. Kim's 2003 study showed cardiogenic embolism the cause in only 5%. Large vessel infarcts were at 50% and dissections were at 15% (J.S. Kim, 2003)**.**

Isa, Kimura, Yasaka, Minematsu, and Yamaguchi (1999) reported a case of a 55-year-old man with occlusions of the left VA immediately after the branching of the PICA. This patient was a smoker with diabetes. They noted that there was a failure of microcirculation in and around the lateral portion of the medulla, causing frequent TIAs before and after the onset of his LMI. The micro-embolism was from the proximal end of the occluded left VA.

Diagnostic Issues

Finding the Lesion

The CT is completely unable to detect brainstem infarcts due to bony artifacts that obscure the anatomic details (Sacco et al., 1993). In fact, out of 14 head CT's administered prior to the MRI, all 14 CT's failed to show the medullary lesions (J.S. Kim et al., 1994)**.** Sacco et al. (1993) had two patients who even had a normal MRI, but clearly showed LMS symptoms in a neurological exam. These researchers found that the presence of facial weakness or ocular symptoms increased the likelihood of finding the infarct on an MRI.

Age of Onset

Sacco et al. (1993) studied 33 consecutive patients with LMS at a stroke center in New York between 1983 and 1989, and he noted that their ages ranged from 24 to 82 years old, with a mean of 58 years old. Twenty-five out of the 33 patients were men. Aydogdu et al. (2001) studied 20 patients in Australia and reported a mean age of 58.2 (with the range from 37 to 75 years old). There were three women and 17 men in that study. Kameda et al. (2004) performed a comparative analysis of 214 patients in 35 stroke centers in Japan. The mean age of onset was 60.7 in LMI and 65.0 in MMI. J.S.

Kim (2003) recorded a mean age of 57 years old, +/- 11.9 years in his large study.

Differential Diagnosis Difficulties

The cases that will be presented at the end of this paper were not identified when the patients were first admitted to the hospital through the emergency department. Differential diagnosis of Wallenberg syndrome can be obscured by similar presentations of suspected drug overdose, suspected gastroenteritis, history of alcohol abuse, and other diagnoses. Patients frequently present with vertigo, nausea, and vomiting. However, these symptoms are among the most common in presentations to emergency departments. Wallenberg syndrome does have a typical *cluster* of expected symptoms, but some lateral medullary infarcts can cause only isolated symptoms.

J.S. Kim (2000) studied three patients with LMI that presented with isolated vertigo and ataxia without the usual signs of Wallenberg syndrome, because they had small infarcts with selective involvement of the central vestibular pathways. These small medullary infarcts were located dorsolaterally and rostrally. They could mimic labyrinthine disorders. Kim explained that in the rostral medulla near the junction with the pons, these central vestibular pathways are located far from the nucleus ambiguus and sensory tracts, therefore preserving swallowing and sensation. These central vestibular pathways are closer together in the caudal medulla.

H. Lee and Sohn (2002) studied axial lateropulsion, which is a compelling sensation of being pulled toward the side of the lesion. This is a well-known feature of Wallenberg syndrome due to the central vestibular pathways, but these authors presented a case of a 66-year-old woman who had this as the only feature of her LMI, other than a horizontal nystagmus on R gaze. Her MRI also showed a *rostral* dorsolateral medullary infarct. The diagnosis of LMI needs to be considered as a differential diagnosis of isolated axial lateropulsion. The researchers described that the axial lateropulsion is caused by the olivocerebellar projections or climbing fibers traveling through the rostral dorsolateral area. There was selective involvement of the *inferior cerebellar peduncle* that contained these fibers with inputs to the flocculus lobe of the cerebellum.

Sacco et al. (1993) described ocular movement symptoms other than the typical nystagmus. For example, he noted horizontal or vertical or oblique diplopia, blurry vision, vertical inversion of images, and oscillopsia (a sense of moving objects).

The differential diagnosis of posterior circulation strokes and LMI may include the presence of a coexisting cerebellar infarction. As with a LMI that is caused by vertebral artery dissection, a cerebellar infarction may be a result of vigorous chiropractic manipulation (Heros, 1992). Early symptoms of cerebellar infarct may be headache, dizziness, nausea, vomiting, and loss of balance, along with signs of ataxia, nystagmus, and dysarthria. However, if the cerebellar edema progresses, compression may occur initially on the posterior aspect of the pons. This leads to compression of the nucleus of VI and the lateral gaze center, and may lead to loss of ipsilateral gaze (Heros, 1992). Heros reinforced the frequent coexistence of a lateral medullar infarction and a cerebellar infarction. Heros referenced Sypert and Alvord (1975) when he stated: “this [coexistence] is not surprising because most symptomatic cerebellar infarctions are due to vertebral occlusion involving the origin of the posterior inferior cerebellar artery, which supplies the lateral medullary region” (Heros, 1992, p. 938). Heros also reinforced the fact that most LMI signs and symptoms are present at onset and are not accompanied by a later change in sensorium, unlike the signs and symptoms of a pontine compression from cerebellar edema that requires surgical intervention.

Risk Factors

Sacco et al. (1993) found that 50% of the patients in their study had hypertension, 33% had diabetes, 25% had coronary artery disease, and 24% had a previous TIA or CVA (the majority with vertebrobasilar circulation issues). J.S. Kim et al. (1994) noted risk factors in the 33 patients as 24 with hypertension, 9 with hyperlipidemia, 8 with diabetes, 8 with smoking, and 3 with coronary artery disease. Only three had no risk factors. J.S. Kim's 2003 research found hypertension in 64% of the patients, diabetes in 25%, smoking in 25%, and atrial fibrillation in 5%. Kameda et al. (2004) noted that the prevalence of diabetes is high with myocardial infarction, and that age and diabetes were risk

factors for medullary infarcts, specifically for MMI. Kameyama, Fushimi, and Vdaka (1994) studied autopsies, and results indicated that diabetic patients have an increased frequency of severe atherosclerosis of the intracranial VA.

Dysphagia Research Related to LMS

A major goal of this literature review is to thoroughly discuss the critical symptom of dysphagia in the LMS patients. It is this author's opinion that if the dysphagia is tested thoroughly enough, the frequency of dysphagia as a symptom may be much higher than the 53-65% as noted in the research listed in the symptomatology section above. Most studies have not evaluated the dysphagia in detail using typical systems of classification in the speech-language pathology literature (i.e., through use of videoflouroscopy/modified barium swallow studies, fiberoptic endoscopic evaluation of swallowing, or manometry).

Research by Logemann (1998) also points to the need for instrumentation to clarify definitions of dysphagia. Logemann comprehensively examined the pharyngeal swallow in medullary stroke patients at the Northwestern Memorial Hospital whose swallow post-stroke was “functional” (meaning the patients were eating normal diets with no aspiration and only small pyriform sinus residue). Despite the fact that those subjects had functional swallows, the measures of their pharyngeal swallow were still not within normal limits for their age and gender. What may appear functional to a bedside examiner, can be revealed as a dysphagia with further instrumental examination.

Sacco et al. (1993) reported that the dysphagia was “usually mild” and only two cases were “severe enough” to require nasogastric tube feedings. The patients in the Sacco study may have had less dysphagia due to smaller lesions, milder symptoms, and partial LMS only. The J.S. Kim et al. study in 1994 only classified dysphagia as – (absent), + (mildly present), and ++ (needs nasogastric tube)**.** Kim's 2003 study reported that only 5% of the patients developed aspiration pneumonia, but that is not an accurate indicator of the severity of the dysphagia. Not all prandial aspirators get sick with an aspiration pneumonia. Additionally, this later study still only classified the dysphagia as “severe”

(needing a nasogastric tube) versus “mild” (when the patients had difficulty feeding, but did not require a feeding tube). The presence or absence of a nasogastric tube should not be an indicator of dysphagia as its placement may be a subjective decision and a preventative measure introduced by the medical team.

Even the Aydogdu et al. study (2001), which tested swallowing with electromyography (EMG), only rated the “degree of dysphagia” in the following way: (I) for no clinical signs and symptoms, (II) as very mild suspected by clinical examination, although the patient never complained of dysphagia; (III) when patient complained of dysphagia, but non-oral feeding was not necessary; and (IV) when the patient had obvious clinical signs and symptoms, including aspiration, and needed non-oral feedings. Defining dysphagia by the patient's report is highly problematic as the patient’s sensation and awareness of aspiration and pharyngeal residue is likely to be impaired. Aydogdu used only the bedside “clinical signs and symptoms”, which is not as accurate as the use of an instrumental exam, i.e., the use of a gold standard such as the the modified barium swallow study (MBSS) (Logemann, 1993). The MBSS is the exam of choice for assessing swallowing physiology, which can vary based on the bolus consistency, volume, taste and texture; therefore, the MBSS tests standardized volumes to give an overall impression of the swallowing impairment (Martin-Harris, et al., 2008).

Additionally, much of the research on Lateral Medullary Syndrome has used ratings of “mild” versus “severe” as noted in the previous discussion. This may not be thorough enough to capture all patients who have dysphagia, and the ratings are not consistent with the classification systems used by speech-language pathologists. Many SLPs now use the MBS Imp (Martin-Harris, et al., 2008), which quantifies the swallowing impairment based on 17 physiologic swallowing components. These components cover oral, pharyngeal, and esophageal aspects of the swallow, and severity is rated with three-point to five-point rating scales. Another less in depth measure of the swallowing impairment is the Waxman Dysphagia Severity Rating Scale (Waxman, Durfee, Moore, Morantz, and Koller, 1990). (See Table 2 below for these definitions). Clinicians can also evaluate a patient's functional swallowing

status at evaluation, during therapy, and at discharge with the ASHA NOMS Functional Communication Measures, which includes a seven-point rating scale (please see www.asha.org/NOMS/ for more information).

Table 2

Dysphagia Severity Rating Scale

Rating	Explanation
0	Normal swallowing mechanism.
1	*Minimal dysphagia*—video swallow shows slight deviance from a normal swallow. Patient may report a change in sensation during swallow. No change in diet is required.
2	*Mild dysphagia*—oropharyngeal dysphagia present, which can be managed by specific swallow suggestions. Slight modification in consistency of diet may be indicated.
3	*Mild-moderate dysphagia*—potential for aspiration exists but is diminished by specific swallow techniques and a modified diet. Time for eating is significantly increased; thus supplemental nutrition may be indicated.
4	*Moderate dysphagia*—significant potential for aspiration exists. Trace aspiration of one or more consistencies may be seen under videofluoroscopy. Patient may eat certain consistencies by using specific techniques to minimize potential for aspiration and/or to facilitate swallowing. Supervision at mealtimes required. May require supplemental nutrition orally or via feeding tube.
5	*Moderately severe dysphagia*—patient aspirates 5% to 10% on one or more consistencies, with potential for aspiration on all consistencies. Potential for aspiration minimized by specific swallow instructions. Cough reflex absent or nonprotective. Alternative mode of feeding required to maintain patient's nutritional needs. If pulmonary status is compromised, "nothing by mouth" may be indicated.
6	*Severe dysphagia*—more than 10% aspiration for all consistencies. "Nothing by mouth" recommended.

Note. Adapted by Gramigna (2006) from Waxman, Durfee, Moore, Morantz, and Koller (1990). Table cited from "How to Perform Video-Fluoroscopic Swallowing Studies," by G. Gramigna, 2006, *GI Motility Online* (2006), doi:10.1038/gimo95.

LMS Research that Has Included Instrumental Testing (MBSS, Esophageal Motility, and EMG)

Kwon, Lae and J.S. Kim (2005) stated that dysphagia in medullary stroke has rarely been studied using videoflouroscopy (MBSS), and the definitions of dysphagia are frequently unclear. They studied LMI versus MMI (medial medullary infarction) in 46 patients from May 2003 to August 2004 at the Asan Medical Center in South Korea, correlating the MBSS with the MRI findings. Their report defined dysphagia as the presence of penetration or aspiration on videoflouroscopy. They classified dysphagia as (a) problems in the *timing* of the hyolaryngeal elevation or (b) problems in the *excursion of the hyolaryngeal elevation*. They found that 78% (7 out of 9) of the patients with MMI had dysphagia. The importance of an instrumental exam was reinforced with this study, as five out of the seven MMI aspirators had silent aspiration. According to these researchers' literature review, previous studies of MMI have found that dysphagia is uncommon in MMI (11-29%). For example, Bassetti et al. (1997) found that only one out of seven patients in their study had dysphagia, but the patients were not tested with an instrumental exam.

Kwon et al. (2005) noted that the primary deficit in MMI was a delay in the *timing* of the pharyngeal swallow response once the bolus hit the valleculae or pyriforms (>1 second initiation of the reflexive swallow). They speculated that problems in the timing of the hyolaryngeal excursion in MMI may indicate “damage in the corticobulbar tract innervating the nucleus ambiguus, rather than direct involvement of the structure” (p. 717). Their other explanation was an involvement of the swallowing premotor neurons (central pattern generator) in the reticular formation and surrounding nucleus ambiguus (NA), resulting in a delay in swallowing.

Kwon et al. (2005) found that dysphagia was only present in 35% (13 out of 37) of the LMI patients, but this may be due the Kwon et al. study having more patients with caudal infarcts and superficial lesions than the J.S. Kim's larger 2003 LMI study. The patients with LMI had an increased deficit in the *range of hyolaryngeal excursion*, usually caused by direct involvement of the NA. They proposed that treatments targeting an improved range of movement would be beneficial to the patients

with LMI, whereas the MMI patients may need a chin tuck or thickened liquids.

Vigderman, Chavin, Kososky and Tahmoush (1998) correlated the MRI findings with MBSS and manometric evidence. This was a case study of a 57-year-old male with diabetes myelitis whose onset symptoms were left-sided facial heaviness, unsteady gait, and aphagia (or loss of swallowing). The neurologist found mild horizontal gaze-evoked nystagmus, decreased pinprick appreciation on the left face and right extremities, partial left Horner's, left palatal paresis with absent gag, and decreased fine motor skills in the left upper extremity. The patient was hypernasal, hoarse, dysarthric, and had trouble managing secretions. He was tested at Day 9 with an MRI, which showed left rostral dorsolateral medullary infarct. The MBSS on Day 9 showed he was unable to initiate a swallow. Manometric findings on Day 11 were: (a) low pharyngeal resting pressures, (b) weak pharyngeal contractions (the peak pharyngeal contraction at 11.6 mm Hg, with normal being between 40-250 mm Hg), (c) “markedly reduced” upper esophageal sphincter (UES) resting pressures, and (d) normal UES relaxation. The patient received a percutaneous gastroscopy tube (PEG) at two weeks after the onset. The followup MBSS two months later, showed ability to take thick boluses, but the patient had significant pooling and required several swallows. His peak pharyngeal pressures were improved (38 mm Hg), but still lower than normal controls. The pharyngeal contraction was occurring before UES relaxation, showing a poorly coordinated swallowing pattern. Five months later, the patient's condition was improved enough for a pureed diet, with no penetration on the MBSS. At 20 months, he still had mild hoarseness and pooling in the valleculae and pyriforms on MBSS. Manometry still showed the peak pharyngeal contraction occurring before the full UES relaxation. However, the PEG was removed as the patient was then on a regular diet. The researchers stressed the fact that a small unilateral lesion can cause a severe bilateral pharyngeal paresis and aphagia.

H. Kim, Chung, K.H. Lee, and Robbins (2000) studied 23 consecutive patients with LMI from 1995 to 1997, testing them by MBSS with thin liquids in 3 ml and 9 ml amounts. They were asked to “hold the barium until you are told to swallow.” Only 44% aspirated (10 out of 23), but three patients

also penetrated the thin liquids. Patients with penetration into the laryngeal vestibule would likely become aspirators after the swallow due to poor sensation and poor vocal cord function in LMS patients. Adding the penetrators to the number of aspirators would increase the percentage to 56.5% (closer to previous studies). In addition, patients in this study had been told to hold the bolus in the mouth, thereby controlling the bolus and preventing premature spillage into the hypopharynx and/or larynx. This may have helped prevent aspirations before the swallow, acting as a safe-swallow strategy, especially in patients with a slow pharyngeal-phase swallow response. Daniels, Schroeder, Degeorge, Corey and Rosenbek (2007) found that verbal cues to hold the bolus caused patients to have a higher bolus position on the tongue base when the swallow was triggered, avoiding spillage of the bolus into the pharynx. Eleven out of the 23 patients had nasogastric tubes in place. 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 per H. Kim et al. (2000). However, the patients with combined lower and middle lesions were all aspirators. Non-aspirators were an average of 7.7 days post onset. Aspirators were an average of 4.9 days post onset; however, this difference was not statistically significant. Nevertheless, as shown in the case studies at the end of this paper, significant differences were seen on bedside and instrumental exams between two days and six days post onset.

Aydogdu et al.'s study (2001) showed that MRI findings and lesion location alone do not account for the variability of swallowing disorders. They studied 20 patients in Australia with LMS and compared them to 22 patients with cortical unilateral cerebral vascular accidents (CVAs), four patients with 9th and 10th cranial nerve palsy, and 30 age-matched healthy controls. Nine out of the 20 patients with LMS needed non-oral feeding. Ninety-five percent of the LMS patients had some dysphagia. Only one patient out of the 20 exhibited no clinical signs and symptoms of dysphagia. The patients were tested with EMG with laryngeal sensors to detect the onset and duration of the swallow response, the upward laryngeal movement, variations in the swallow response time, and pathological use of piecemeal swallows. In patients with LMS, the pharyngeal phase swallow reflex was extremely slow

and delayed, and the severity of the dysphagia was greater. (This is contrary to the finding discussed above in 2005 by Kwon et al., which found the MMI patients had greater swallow delays than the LMI patients.) There was greater variability in the duration of laryngeal movements, whereas the duration of laryngeal movements remained normal in unilateral hemispheric CVA. The hemispheric CVA patients had more oral deficits. According to the researchers, the extremely prolonged pharyngeal phase of the swallowing in LMS patients was the most significant finding of the study. Although a medullary lesion is unilateral, its effect on swallowing is bilateral. Aydogdu et al. confirmed that the pharyngeal and laryngeal deficits were bilateral with electrophysiological findings, and they used the term "laryngopharyngeal paresis" (p. 2085). This research also found the presence of a "dysphagia limit" (duplication of swallowing or piecemeal swallowing with <20 ml of water) in all patients with LMS (p. 2083). For example, 40% of LMS patients needed the volume to be 1 ml of water to clear the bolus in one swallow. The volumes of water that could be tolerated in one swallow were consistently lower for the LMS patients in contrast with the patients with hemispheric CVA or 9th and 10th cranial nerve palsy. The researchers also noted that the pharyngeal and laryngeal paresis in 9th and 10th cranial nerve palsy is unilateral and much more mild than that of LMS patients. The four patients with unilateral peripheral 9th and 10th cranial nerve palsies had a 10 ml dysphagia limit, but they solved the dysphagia problem with a head turn maneuver to the paretic side. This is a strategy that can also be used by LMS patients, but it was not mentioned in the Aydogdu et al. (2001) article. More on treatment strategies in the "Prognosis and Treatment" section.

Correlating Hoarseness and Dysarthria with Dysphagia

Many LMI studies mention the symptom of hoarseness without discussing the cause for this change in vocal quality. Ipsilateral vocal cord paralysis is common in LMI (Groher, 1992). This can lead not only to aspiration because of inadequate airway protection, but also to a non-productive cough. Aydogdu et al. (2001) noted that 85% of the 20 patients with Wallenberg syndrome in his study had hoarseness and 100% had vocal cord paresis, causing a weak cough 80% of the time. It is well

established in the literature that hoarseness (dysphonia) and dysarthria are correlated with dysphagia and aspiration risk. For example, Horner, Massey, Riski, Lathrop and Chase (1988) found that 91.3% of aspirating patients had dysphonia, versus 68.2% of non-aspirating patients. They suggested that the presence of dysphonia in neurogenic dysphagia should indicate that evaluation for aspiration is needed. Horner, Buoyer, Alberts and Helms (1991) studied 23 patients with brainstem strokes, and MBSS showed aspiration in 15 out of the 23 patients. All 15 aspirators had pontine or medullary infarcts. There was a statistical significance between aspiration and the following four factors (a) pharyngeal residue, (b) cranial nerve IX abnormality, (c) vocal fold weakness, and (d) severe dysarthria.

Daniels, McAdam, Brailey and Foundas (1997) and Daniels et al. (1998) created six clinical indicators correlated with aspiration during a videofluoscopic exam (MBSS) in stroke patients: (a) absent volitional cough, (b) dysphonia, (c) dysarthria, (d) cough after swallow, (e) voice change after swallow, and (f) absent gag. They found that the presence of two out of the six factors could distinguish patients with moderate to severe dysphagia from those with mild or no dysphagia. Furthermore, Daniels, Ballo, Mahoney and Foundas (2000) found that early identification, evaluation with MBSS, and treatment of acute stroke patients brought improved outcomes. Schroeder, Daniels, McClain, Corey and Foundas (2006) found poor acute and long-term outcomes when the patients experienced four or more of the six indicators above. They also confirmed that aphasia (deficit in language areas of comprehension, fluency, naming and/or repetition, according to the researchers) was not significantly associated with initial or final dysphagia outcomes.

H. Kim et al. (2000) stated that all their aspirating patients with pure medullary infarction had dysphonia. In that study, dysphonia was one of the three factors that predicted aspiration (the others being facial hypesthesia and soft palate motor dysfunction). The researchers summarized that dysphonia closely correlates with aspiration due to the nucleus ambiguus.

Patients with dysphagia had hoarseness and dysarthria more often than those without dysphagia (J.S. Kim, 2003). Dysphagia and dysarthria were more common in the “large type” lesions referred to

in J.S. Kim's horizontal classification system (J.S. Kim, 2003).

More on Symptoms and Lesion Location: Rostral versus Caudal Lesions

Patients with rostrally located lesions tend to have dysphagia, facial paresis, and dysarthria more often (J.S. Kim, 2003). Kameda et al. (2004) also found dysphagia more often in the rostral infarcts as opposed to those in the caudal region. In 1994, J.S. Kim et al. noted that all patients in the rostral group had more hoarseness and dysphagia and needed nasogastric tube insertion for feeding. However, the rostral lesions were generally thicker and deeper and the caudal lesions tended to be superficial lesions. Only two out of 21 caudal-lesion patients in the study had transient dysphagia, and they had concomitant medial medullary lesions.

LMI symptoms of palatal and laryngeal weakness, with hiccups at times, indicate an involvement in the nucleus ambiguus (Groher, 1992)**.** H. Kim et al. (2000) found that soft palate dysfunction is frequently compromised in aspirators, as the levator veli palatini is under control of the vagus and nucleus ambiguus. Accordingly, Bassetti et al. (1997) asserted that palatal and pharyngeal weaknesses are rare in medial medullary infarcts (MMI). Their patients with MMI had less dysphonia and dysphagia, which is consistent with the fact that the nucleus ambiguus is not within the medial medulla.

Frequently, the patients' tongue movements and muscle mass are not impaired in LMI, indicating that the hypoglossal nerve nucleus is not injured with lesions in the rostral dorsolateral medulla (Vigderman, Chavin, Kososky and Tahmoush, 1998). Note: The hypoglossal nerve nucleus is in the dorsal medial area of the middle medulla (H. Kim et al., 2000). Like Vigderman et al., Kwon et al. (2005) noted that dysarthria and tongue weakness were less common in LMI and more common in MMI. Interestingly, in Kwon et al.'s study, dysarthria (and not dysphonia) was the only neurological sign that correlated with dysphagia. This may be due to the fact that the vagal motor neurons innervating the larynx are located more caudally than those of the pharynx (Bassetti et al., 1997). Kwon et al. (2005) referred to a rat study by D. Bieger and D.A. Hopkins (1987) when they stated that the

esophageal and pharyngolaryngeal motor systems are regulated by the rostral to intermediate area of the nucleus ambiguus, whereas the intermediate-caudal aspects of the nucleus ambiguus regulate phonation through cricothyroid and other intrinsic laryngeal muscles.

Kwon et al. (2005) noted that dysphagia was not only more frequent in the rostral group (six out of seven patients with rostral lesions), but also more common with deep lesions, (i.e., those extending more dorsally versus superficial lesions). They noted that the nucleus ambiguus is located deep in the medulla and the structure is larger in the rostral portion of the medulla. J.S. Kim et al. (1994) explained that in the middle portion of the medulla the nucleus ambiguus looks like a vertical column. In his 2003 study, J.S. Kim explained that the nucleus ambiguus in the caudal area is not related to pharyngeal musculature, and this description is consistent with the rat study noted above. He also stated that caudal lesions do not extend deeply enough to involve the nucleus ambiguus. Kwon, et al. (2005) indeed found that the rostral lesions tended to be deeper and non-superficial and the caudal lesions were primarily superficial.

Lesions involving the nucleus of the solitary tract (also known as the nucleus tractus solitarius or NTS) cause decreased sensation in the pharynx, specifically the base of the tongue and epiglottis (H. Kim et al., 2000)**.** This decreased sensation may lead to premature spillage of the bolus into the pharynx before the swallow, causing an increased risk for penetration into the laryngeal vestibule. The decreased sensation may also decrease a patient's awareness of pharyngeal residue after the swallow. Lesions of the spinal trigeminal nucleus (which is located dorsolaterally in the middle to caudal medulla) will cause decreased sensation in mucosa of oral structures like the tongue, gums, floor of the mouth, and palate, which also may lead to decreased bolus control and reduced awareness of residue or pooling (H. Kim et al., 2000).

Finding the "Swallowing Center" or the Central Pattern Generator (CPG)

A great deal of research has attempted to locate the central control for swallowing. The analysis of the swallowing deficits in patients with LMS can provide keen insight into this investigation.

Swallowing is a patterned sequence of motor activity that results from the bilateral input from the swallowing centers in the rostral dorsolateral medulla (Vigderman et al., 1998). The major swallowing centers are the nucleus of the solitary tract (NTS), nucleus ambiguus (NA), and the reticular formation (Aydogdu et al., 2001). Vigderman et al. (1998) provided insight into human swallowing with the following statement: “The severe and bilateral pharyngeal paresis from a unilateral lesion shows that injury to the ipsilateral swallowing center in man is sufficient to result in complete loss of swallowing” (p. 210). These researchers also referred to Doty, Richmond and Storey's research in 1967 that showed the ablation of the rostral dorsolateral medulla in animals caused only an ipsilateral loss of swallowing. Humans have more integrated swallowing centers.

Aydogdu et al. (2001) described the dysphagia in patients with Wallenberg syndrome as an “acute disconnection syndrome,” due to the disruption of premotor neurons in and around the NA, NTS and reticular formation causing bilateral dysfunction (p. 2086). According to these researchers, this disturbs the sequential muscle activity. The severity and duration of the dysphagia depends on the extent of the lesion and the degree to which the dorsal medulla is affected around the NTS. They note that LMI primarily affects the NTS in the dorsal region, the NA and surrounding reticular formation in the more ventral region, as well as the extensive interconnections between these dual swallowing centers. Damage to these areas may cause the severe incoordination that prolongs the pharyngeal phase of the swallow. The pharyngeal and esophageal phases of swallowing are coordinated by the bilateral representation of these dual swallow centers (Aydogdu et al., 2001).

Vigderman et al. (1998) also discussed that the *dorsal* component of the medullary swallowing center is the NTS and the surrounding reticular formation; and the *ventral* component is the lateral reticular formation above the NA. Dorsal neurons integrate cortical input with cranial nerves V, IX, and X. Then they transmit to the ventral component, which coordinates the patterned sequence of motor activity through projections to the ipsilateral and contralateral cranial nerve motor nuclei that control the muscles involved in swallowing, namely V, VII, IX, X, XI, and XII.

Martino et al. (2001) posit that the central pattern generators regulate the central control of swallowing. It is a "serial network of linked neurons within the nucleus of the solitary tract (NTS) and neighboring reticular formation" (p. 423). They described a sequential excitation rostrally to caudally along the "deglutition pathway." It activates the cranial nerve motor neurons which include the nucleus ambiguus and the vagal dorsal motor nucleus. These cranial nerves in turn innervate the muscles of swallowing. Per these researchers, dysphagia results from lesion in the area that connects the central pattern generator (which is more dorsal and medial in the brainstem) to the nucleus ambiguus (which is deeper and more lateral in the brainstem).

Aydogdu et al. (2001) cited Haines, D.E. (1991) in *Neuroanatomy: An Atlas of Structures, Sections, Systems* when he described an important point: "A transverse section through the medulla corresponding approximately to the rostral third to fourth of the principal (inferior) olivary nucleus contains the site at which the NTS and the NA are almost equally affected by the occlusion of the posterior inferior cerebellar artery" (pp. 2085-2086).

Robbins et al. (2008) stated that the central pattern generators (CPG) of swallowing are neural networks that are flexibly organized and multifunctional. They describe how this flexible organization or neural plasticity coordinates the swallowing activity, rather than containing all the neural circuitry to initiate a pharyngeal swallow response. The CPG also provide cross-system coordination with the laryngeal and respiratory systems (Robbins et al., 2008). Chaudhuri et al. (2002) speculated on the proximity of the central respiratory, cardiac, and swallow centers when they found reports of tachycardia and arrhythmia in patients attempting swallowing maneuvers and exercises that have the patient perform a valsalva or an effortful breath hold. The researchers suggest that the vagus nerve nucleus may be a common pathway that mediates these functions in the body. Arai, Obuchi, Matsuhisa, Takahashi and Takatsu (2008) reviewed two cases of eighty year olds with unilateral medullary infarction associated with acute central respiratory failure a few days after onset. Both of these octogenarians had dysphagia and respiratory failure, and the researchers postulated that a lesion

causing dyspnea may be close to the lesion that causes dysphagia.

Caveats to the localization of one "swallowing center" are as follows. Ding, Larson, Logemann and Rademaker (2002) noted that if the swallow was solely controlled by the brainstem, one would expect little variability between subjects in regard to the amplitude and duration of the swallow. They found large variability despite restricted head movement and only delivering 5 ml bolus to all subjects. They tested five muscle groups on normal subjects drinking 5 ml of water, studying a normal swallow versus a swallow with the trained Mendelsohn maneuver. Ding and colleagues described that "swallowing is a highly complex adaptive motor activity, which probably utilizes more than the brainstem mechanisms" (p. 11). They listed the following: primary somatosensory cortex, premotor cortex, the primary motor cortex, and the anterior insula.

Dantas et al. (1990) clarified that it is not only the CPG that execute a stereotypic swallow sequence, but also modulations due to sensory feedback from different bolus variables, such as volume and viscosity. These can create significant physiological changes in the oral, pharyngeal and esophageal stages of the swallow. They explained that increased bolus volume increases (a) the distance of anterior hyolaryngeal excursion, (b) the pharyngeal expansion, and (c) the upper esophageal sphincter opening. The treatment section below will discuss use of bolus volume in evaluation and therapeutic feeding trials with LMS patients. A key question throughout the rest of this paper is: what part of the swallow is under a reflexive brainstem control by the CPG, and what is under a more volitional control to potentially modify and compensate for the dysphagia?

Dysphagia in LMS: Are the Upper Esophageal Sphincter (UES) and Cervical Esophagus Involved?

There is considerable evidence to suggest that the swallowing problem in LMS may be due to more than a pharyngeal dysphagia alone. Research discussed in this section, as well as the case studies at the end of this article, point out that the cause of UES dysfunction may not be solely due to impaired laryngeal elevation and bolus propulsion. The patients may also have esophageal dysphagia in the

striated portion of the esophagus and a hypertensive cricopharyngeal muscle. First, for a better understanding of normal UES function here is a review of some of the research on normal adults.

Background on the Normal Upper Esophageal Sphincter (UES) Function

UES opening is the "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). Jacob, Kahrilas, Logemann, Shah and Ha (1989) also described the UES sphincter opening as an active mechanical event rather than simply a consequence of CP relaxation. This is a five step sequence: (1) Relaxation, (2) Opening, (3) Distention, (4) Collapse, and (5) Closure with sphincter contraction.

The following is Jacob et al.'s description (1989) of these five steps:

Relaxation. The first step in the sequence is a cessation of tonic activity of the striated muscles of the sphincter, which occurs during laryngeal elevation. Relaxation precedes opening by only 0.1 second.

Sphincter opening. This occurs only after significant anterior and superior laryngeal excursion, causing a pulling force from muscular activity. There is a shortening of the "infrahyoid compartment" starting 0.2 seconds before the sphincter opening. The CP muscle is attached to the lateral aspects of the arch of the cricoid cartilage, so that the CP muscle and cricoid move in unison. Anterior laryngeal excursion is a function of contraction of the suprahyoid muscles (mylohyoid per the trigeminal nerve, geniohyoid per the hypoglossal nerve, and the anterior belly of the digastrics), plus the contraction of the thyrohyoid muscle (Mepani et al., 2009; Aydogdu et al., 2001; Shaker et al., 1997). There is an intraluminal negative UES pressure attributed to the suction created by the hyoid traction on the anterior wall of the sphincter, but then the pressure increases to 12 mm Hg after opening during the sphincter distention (Jacob et al., 1989).

Distention. Jacob et al. (1989) further described that distention of the UES by the bolus head is modulated by intrabolus pressures due to: (a) posterior tongue thrust, (b) pharyngeal wall contraction, and (c) initiation of pharyngeal peristalsis. These researchers found that maximum distention occurred

within 0.13 seconds of opening. Intrabolus pressures during the distention phase correlated with the increased anterior dimension of the UES opening, whereas the posterior dimension was correlated to the extent of hyolaryngeal elevation. Bolus acceleration and anterior UES distention closely correlated with posterior tongue-base movement. The researchers described this sequence as follows: (1) the bolus arrives on the tongue base and in the valleculae, (2) the tongue's piston-like action increases the propulsive forces on the bolus, and then (3) the tongue base contact with the pharyngeal wall marks the onset of pharyngeal peristalsis.

Collapse and contraction. Closure occurred with sphincter contraction when the tail of the bolus transversed the sphincter segment, and with the manometric arrival of pharyngeal peristaltic contraction.

Overall, this Jacobs et al. research (1989) suggests that three variables can be manipulated in the sequence of UES opening: diameter, duration, and velocity of the head of the bolus. (These issues will be discussed in the "Prognosis and Treatment" section of this paper, which proposes ways that clinicians can help patients actively compensate for pharyngeal dysphagia and UES dysfunction.)

How Does Upper Esophageal Sphincter (UES) Dysfunction Contribute to the Dysphagia in LMS?

Strokes of the brainstem usually affect the ability of the cricopharyngeal muscle to relax (Groher, 1992). Wilcox, Liss and Siegal (1996) studied inter-rater reliability among ten speech-language pathologists (SLP) who reviewed MBS studies on three patients. One patient was a 44-year-old male with LMS, and ten out of ten SLPs observed impaired cricopharyngeal opening on thick liquids and barium paste. Eight out of the ten agreed on the presence of pooling in the pyriforms. The patient had dysphagia to solids that was greater than his dysphagia to liquids, but his gag, cough, lingual and labial strength, and range of motion were intact.

Aydogdu et al. (2001) noted that patients with LMS abnormally needed piecemeal swallows with all volumes greater than 3 ml, and 40% of these patients had multiple swallows on <1 ml. This

may be due to the absence of cricopharyngeal muscle relaxation prior to UES opening, but it may also be caused by decreased hypopharyngeal intrabolus pressure due to weak pharyngeal muscles and decreased anterior hyolaryngeal elevation and excursion. The laryngeal elevators are the anterior digastrics and the mylohyoid (controlled by the trigeminal nerve) and the geniohyoid (controlled by the hypoglossal nerve).

Logemann (1987) reviewed two patients with LMS on an instructional video designed to train speech-language pathologists in the interpretation of modified barium swallow studies. She described that both patients had cricopharyngeal dysfunction. The first patient had only a mild reflex delay; however, due to his cricopharyngeal dysfunction, he was left with a moderate to severe residue after the swallow in the pyriform sinuses. He then aspirated after the swallow as he inhaled the residue and was unable to cough it out. The second patient had a timely pharyngeal swallow, but she had impaired laryngeal elevation and cricopharyngeal dysfunction, leaving two-thirds of the bolus in the pyriforms after the swallow. Repeated dry swallows only cleared a very small amount of the bolus through the UES. The second patient was able to clear the bolus with a head turn to the damaged-left side, as the cricopharyngeal dysfunction appeared to be worse on the left. Logemann noted that the cricopharyngeal dysfunction and reduced laryngeal elevation often co-occur, as the superior and anterior hyolaryngeal excursion are responsible for a large portion of cricopharyngeal opening. This video did not address whether or not there is a deficit in the relaxation phase of the cricopharyngeal muscle.

In Logemann's (1998) textbook, she described the following oropharyngeal deficits seen in patients with medullary lesions: patients have functional oral phases of the swallow, followed by an absent or very weak pharyngeal swallow response. They may have normal submandibular, tongue base, and hyoid bone movement, and their effort to propel the bolus posteriorly may be misleading and mistaken for an actual pharyngeal swallow. If the patient is able to trigger a pharyngeal swallow, he or she typically still shows reduced laryngeal elevation and anterior excursion, causing decreased

mechanical opening of the UES and therefore pooling in the pyriform sinuses, especially on the ipsilateral or weaker side.

According to Logemann (1998), these patients also have unilateral pharyngeal weakness, causing decreased bolus pressure. Bolus pressure is a major contributor to UES opening as well. Logemann speculated that the cricopharyngeal muscle is *not* actually spastic (not a deficit in relaxation); rather it is the decreased laryngeal elevation and anterior movement, as well as the lack of bolus pressure (due to pharyngeal weakness) that does not allow for adequate UES opening or distention. As Jacob et al. (1989) stated: "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" (p. 1478).

However, the Martino et al. (2001) study provides evidence contrary to Logemann's position that the cricopharyngeal muscle is not actually spastic. They found a lack of relaxation of the cricopharyngeal muscle, as well as no contractile activity in the striated portion of the esophagus. The study's background is as follows: Martino and her colleagues in Toronto studied a 65-year-old male with LMS who had an intact oral phase, but still had an absent pharyngeal swallow response at 12 weeks after the onset. He had a right superior dorsolateral medullary infarction of 5 mm in diameter when it was considered matured and edema decreased (as seen in an MRI at 7 months post onset). The infarct was centered in the dorsal medulla just anterior to the floor of the 4th ventricle and rostral to the obex. MBSS studies were performed at 12 weeks, 9 months, 14 months, and 27 months. At 9 months, the patient had evident and "only slightly reduced" anterior and vertical elevation of the larynx. He had decreased epiglottic deflection, but the major deficit was with the pharyngeal and the UES function. There was no contraction of the middle and inferior pharyngeal constrictors with an absent bolus drive. The UES failed to open adequately, and there was pooling in the hypopharynx and silent penetration. The patient was then trained to perform a Mendelsohn maneuver, which extends the elevation of the larynx in range and time, thereby allowing for small amounts of liquid boluses to pass through the

UES. He started with purees and liquids in therapeutic trials at 9 months and advanced to soft solids by the 14-month MBSS. He was observed to "advance the bolus through the UES" in a "piecemeal fashion." (This is consistent with Aydogdu et al's finding in the 2001 LMS study of the abnormal piecemeal swallows.) This patient continued to use the Mendelsohn maneuver as a compensatory strategy to volitionally elevate the larynx at the 27th-month MBSS.

Martino et al.'s (2001) motility studies at 8 months confirmed: (a) an absent pharyngeal contraction 1 cm above the UES, (b) no effective relaxation of the UES, and (c) no contractile activity in the proximal 6 cm of the upper esophagus (striated muscle region). Also the basal UES pressure was 44 mm Hg, which was described as a significant resting tone. However, the patient did have a discrete pharyngeal contraction at 2.5 cm above the UES, as well as normal contractile activity lower in the esophagus at 8-15 cm. The LES pressure was within normal limits at 36 mm Hg.

Martino et al. (2001) described how the vagal efferents, which originate in the nucleus ambiguus, are responsible for motor activity of the striated muscle of the pharynx, UES, and esophagus. Her article also referred to the Bieger and Hopkins (1987) study of the upper alimentary tract in the medulla oblongata in the rat. These researchers had found that the motor efferents located more rostrally in the nucleus ambiguus innervate the pharyngeal muscles, whereas more medial and caudal efferents innervate the striated muscle of the esophagus. Martino and her colleagues speculated that the UES fibers originate between these two zones (rostral versus the medial to caudal areas). However, the LES and the intact smooth muscle of the esophagus have efferents originating in the dorsal motor nucleus of the vagus. Vigderman et al. (1988) also concluded that normal pressures more distally in the esophagus suggest that the smooth muscle is innervated by the dorsal motor nucleus of the vagus, as this area was not damaged in their patient.

The pharyngo-esophageal motor system is regulated by the more rostral area of the nucleus ambiguus. The more caudal aspects of the nucleus ambiguus regulate phonation through the cricothyroid and intrinsic laryngeal muscles (Bassetti et al., 1997; Kwon et al., 2005). Kwon et al.

(2005) used this explanation when noting how dysarthria correlated significantly with dysphagia and not dysphonia.

H. Kim et al. (2000) reported that the pharyngeal constrictor muscle paresis was due to the damage of the nucleus ambiguus and the vagus nerve, which control the striated muscles of the palate, the base of tongue, the pharynx, and the larynx. However, most studies do not discuss the striated muscle of the cervical esophagus as did the Martino et al. (2001) study above.

The only other research article found in this literature review that addresses the LMS patient's UES dysfunction, and specifically the cricopharyngeal muscle, was one by Robbins and Levine (1993). They studied a 62 year old female with a left LMI secondary to a left VA occlusion, with the infarction extending into the left inferior olive and the left inferior cerebellar peduncle. They labeled this patient as having *lateral medullary syndrome-plus*, as the MRI and angiography showed pontine-level ischemic changes, as well as "probable bilateral brain stem ischemic potential secondary to markedly impaired (mid-basilar) arterial flow" (p. 50). Even though the MRI showed only a left LMI, the researchers questioned bilateral damage due to the bilaterally impaired swallowing. However, as Vigderman et al. (1998) and Aydogdu et al. (2001) reported a unilateral lesion in the lateral medulla is sufficient to cause an absent swallow and bilateral pharyngeal paresis.

This 62 year old female was referred to the dysphagia clinic at the University of Wisconsin at 19 months after her stroke. She had been receiving nutrition solely through a gastrostomy tube due to prolonged and profound dysphagia. She was still constantly expectorating her secretions. The MBSS study at 19 months found no epiglottal dorsoflexion, "hyolaryngeal bobbing," no pharyngeal muscular activity, and no UES opening (p. 47). There was still no UES opening despite the use of strategies during the MBSS, such as cues to increase the piston-like thrusting action on the bolus with the tongue, the Mendelsohn maneuver, and a head turn to the weaker side.

By the MBSS at 20 months, the patient was able to learn to voluntarily sustain maximum hyolaryngeal anterior and superior excursion by prolonging the swallow with the Mendelsohn, as well

as to increase the lingual force to increase the intrabolus pressure. This resulted in her first UES opening documented in 20 months. However, the bolus flow remained absent due to a "hypertonic cricopharyngeal muscle" (p. 53). The researchers described that the Mendelsohn could override the UES high pressure zone, but that it was still in the "presence of impaired cricopharyngeal relaxation" (p. 53). The MBSS at 21 months still showed minimal UES opening and an obstructing cricopharyngeal bar despite vigorous coaching. Manometry showed that the UES sphincter pressure was distinctly abnormal at 130-160 mmHg with incomplete relaxation. The ear, nose and throat (ENT) consultation at close to 24 months after her stroke recommended a cricopharyngeal myotomy with an approach from the left side of the neck as the left true vocal fold was found to be paralyzed. The MBSS eight days after the cricopharyngeal myotomy showed increased UES opening with the use of the strategies noted above. At 25 months, the MBSS showed increased duration and range of motion of the UES opening, allowing for 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.

If the dysphagia of LMS was only caused by the oropharyngeal deficits alone, then the maneuvers and strategies listed above would have been sufficient to pass the bolus through the UES. Robbin and Levine (1993) described that the hypopharyngeal suction pump (HSP) needs the presence of 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.

A PubMed search did reveal another article that described cricopharyngeal dysphagia; however, this was in Japenese. The abstract revealed that Katoh, Hayakawa, Ishihara, and Kazumi (2000) studied a 65-year-old man with Wallenberg syndrome and severe dysphagia. The swallowing was assessed with the use of videofluorography or MBSS. The patient was treated with a balloon dilatation method for his cricopharyngeal dysphagia, and he showed less aspiration on the repeat MBSS three months later.

The case studies at the end of this article will discuss deficits in pharyngeal peristalsis, hyolaryngeal excursion, and UES dysfunction with a lack of cricopharyngeal relaxation. More research is needed in this area to delineate the level of impairment, and whether it is solely in the pharyngeal phase of the swallow or in both the pharyngeal and esophageal stages.

Prognosis and Treatment

Aydogdu et al. (2001) noted that the dysphagia after LMI can range from very mild to severe and prolonged. In their study, 13 of the 20 patients were re-evaluated. Only three patients out of the 13 still had severe dysphagia. The other 10 had significant recoveries, with three patients making a complete recovery. However, the timing of the re-examinations with EMG was not at consistent intervals. The three patients who still had severe dysphagia were at 47, 55, and 362 days after the first evaluation. However, as Aydogdu et al. Noted (2001), the recovery process is: “rather slow, although steady” (p. 2084). The remaining ipsilateral premotor neurons around the NTS, NA, as well as contralateral centers, eventually contribute to the improvements.

Recovery

Dysphagia can be severe enough to require intubation or tracheostomy, but recovery is expected (Groher, 1992). Such severe cases reinforce the need for intensive attention immediately after the stroke. Elderly patients suffering from unilateral LMI with dysphagia can present with respiratory failure (Arai et al., 2008). Severe dysphagia can lead to aspiration pneumonia and prolonged hospitalization, so extra care is necessary with the large rostral lesions (J.S. Kim, 2003). Out of the 130 patients with pure LMI in the J.S. Kim (2003) study, only one patient (0.8%) died during the hospitalization. The study found an excellent prognosis overall (J.S. Kim, 2003).

H. Kim et al. (2000) noted that dysphagia is initially quite severe and even requires non-oral feedings, but it improves rapidly in one or two months. Due to the researchers' early implementation of diet modifications along with postural adjustments while the patients were eating, none of the patients in the treatment program developed pneumonia. Most of the patients in the study with nasogastric tubes

initially were able to transition to full oral intake in two months. Systematic control of the initial swallowing evaluation, as well as re-evaluations at consistent intervals after the stroke, were critical to demonstrating quick recovery in patients with LMS who aspirate.

J.S. Kim (2000) gave recovery information regarding the three LMI patients with isolated vertigo and ataxia. Case One at two months after the stroke still had mild dizziness on walking, which was worse with rapid movements. Case Two at two months had slight instability on tandem gait. Case Three at four months walked normally but had intermittent dizziness on sudden positional changes.

Treatment

The head turn maneuver. The Aydogdu et al. (2001) research mentioned the head turn maneuver during a swallow. Logemann, Kahrilas, Kabara and Vakil (1989) observed benefits in a patient with LMS with turning the head to the paralyzed side to achieve closure of the hemipharynx on that side. Tsukamoto (2000) studied a 55-year-old male with LMS performing a head turn to the weaker side, and he found neck CT evidence of closure of the hypopharyngeal cavity above the level of the pyriform sinus due to a shift in soft tissue. Specifically, this closure was obtained at the level of the hyoid bone to just above the pyriforms. This maneuver acted to dilate the opposite hypopharyngeal wall, which can help the bolus move into the stronger pyriform.

Kwon et al. (2005) compared medial medullary lesions with lateral medullary lesions. They discussed strategies of thickening liquids and chin tuck for patients with delayed swallows due to medial medullary infarction; in contrast, treatments for increased range of hyolaryngeal movement and pharyngeal peristalsis would be more beneficial for patients with LMS. They also found that the head turn was more effective than the chin tuck strategy for patients with LMS.

Neural plasticity. Robbins et al. (2008) performed a review of the state of swallowing rehabilitation science and mentioned several treatments that may pertain to patients with LMS. The principles of neural plasticity tell us that we need to “Use it or lose it; use it and improve it, and repetition and intensity matter” (pp. S278-281). The authors speculated that prolonged periods of no

oral intake (NPO), such as after an acute lateral medullary infarction, may have a detrimental effect on the neural representation for swallowing.

Bolus volume. In this same literature review, Robbins et al. (2008) noted how all aspects of the oropharyngeal swallow have been found to be modifiable depending on the sensory input. For example, increasing the bolus volume allows for increases in: (a) pharyngeal wall movement, (b) hyolaryngeal excursion (vertical and anterior movement), (c) superior UES movement, (d) anterior-posterior dimension/width of UES opening, (e) intrabolus pressure, and (f) bolus velocity through the UES (Jacob et al., 1989). These researchers observed that the UES is actually open wider and for a longer time with large bolus sizes. The magnitude and duration of the intrabolus pressure also increased with bolus volume. In the same study, the velocity of the head of the bolus increased significantly (affecting UES distention), but the pharyngeal peristalsis did not vary significantly with bolus volume. On the other hand, Dantas et al. (1990) found the vertical and anterior hyolaryngeal excursion and width of UES opening is dependent on *viscosity* alone, independent of the volume. The Jacob et al. (1989) research found that the *duration* of the hyoid movement was influenced by bolus volume, but the extent of the movement was not significantly affected. A MBSS can more aggressively and safely test larger bolus volumes. It may be the larger bolus size that actually allows for improved UES opening and increases the functionality of the swallow in a patient with LMS.

Maneuvers and exercises. The factor of bolus volume alone may not increase the pharyngeal peristalsis or the extent of hyolaryngeal elevation. Clinicians have to look to swallowing maneuvers and exercises to address deficits in these areas. If pharyngeal peristalsis is a concern, the clinician may recommend a head turn as mentioned above, a dry swallow, a liquid wash, or a chin tuck and effortful swallow. The last two strategies may help to decrease at least the valleculae residue (Logemann, 1993). The Robbins et al. (2008) article reviewed the literature in motoric exercises with and without the swallow, which have demonstrated "behavioral plasiticity" but not neural plasticity. For example, tongue-base exercises can increase the strength of the piston action to increase the propulsive forces on

the bolus. Additionally, to increase the extent and duration of the hyolaryngeal movement and UES opening, the Mendelsohn maneuver or the Shaker exercise may be employed.

The Mendelsohn Maneuver. The purpose of this maneuver is to increase the extent and duration of laryngeal elevation to increase the width and duration of cricopharyngeal opening (Logemann, 1993). The patient is instructed to swallow and hold the larynx in the elevated position without letting it drop back down for several seconds. Kahrilas, Logemann, Krugler and Flanagan (1991) noted that the Mendelsohn volitionally increases the duration of UES opening by prolonging the superior and anterior excursion of the larynx. Boden, Hallgren and Will (2006) analyzed swallowing maneuvers in normal subjects with videomanometry and found that the Mendelsohn allowed for both longer durations of pharyngeal contraction and longer bolus transit time. This may allow for improved bolus propulsion through the UES.

Ding, Larson, Logemann and Rademaker (2002) studied the surface electromyographic and electroglottographic parameters in 20 normal subjects under the conditions of a normal swallow and under conditions of a Mendelsohn maneuver while swallowing 5 ml of water. The swallow sequence was the same in both conditions: muscle activity progressed from orbicularis oris inferior to orbicularis oris superior to masseter to *submental* (suprahyoid) muscle group to infrahyoid muscle group. The researchers defined the submental group as the laryngeal elevators, which are the digastrics, mylohyoid, and geniohyoid. The *infrahyoid* group was defined as the laryngeal depressors, and includes the sternohyoid, omohyoid, and thyrohyoid. As revealed by the surface EMG activity measured, the *submental* muscle group was the only one that demonstrated a difference between normal swallowing and the Mendelsohn swallow. This muscle group may be linked to the Mendelsohn because it is necessary in order to hold the larynx up.

The Shaker exercise. In an effort to strengthen the *infrahyoid* muscle group as well (specifically the thyrohyoid), Mepani et al. (2009) found that the Shaker exercise caused thyrohyoid muscle shortening, in addition to strengthening the suprahyoid (called submental above) muscles. Also,

Shaker stated in his 2002 study that the Mendelsohn increases the duration of the UES opening, but the Shaker exercise increases the magnitude of the UES opening (Shaker et al., 2002).

The Shaker Exercise is performed three times a day for at least six weeks. It includes three repetitive head raisings from a supine position sustained for up to one minute each (with a one minute rest in between), followed by 30 consecutive repetitions of head raisings. The clinician should make sure there is no movement in the shoulders (Shaker et al., 1997). This exercise is a physiological manipulation of the striated muscle group involved in swallowing, and its use is similar to that of the Mendelsohn in prolonging UES opening (Shaker et al., 1997). Shaker's 1997 description of why it works is as follows. There is an increased anterior excursion of the thyroid cartilage (with suprahyoid muscle group involvement, especially the mylohyoid, geniohyoid, and potentially the anterior segment of the diagastric). The result is a decrease in hypopharyngeal intrabolus pressure (measure of the decreased resistance to the transphincteric flow and the pharyngeal outflow) and an increase in the anterior-posterior diameter of the UES opening. Shaker's studies indicate that patients who were dependent on tube feedings were able to return to oral intake after completing the exercise program (Shaker et al., 1997; Shaker et al., 2002).

Specifically, in 2002, Shaker et al. studied the use of the Shaker exercise in 27 patients (7 of whom had a brainstem CVA) who were fed by gastrostomy or jejunostomy tubes while residing at home. The mean age was 72 years old, and the days post onset varied greatly from 9 days to 2880 days. The brainstem patients varied from 10 to 210 days post stroke. The results are summarized in six points. First, the researchers found an increased anteroposterior diameter of the UES opening, and second, they noted an increased extent of the maximum anterior laryngeal excursion. No change was noted in superior laryngeal excursion or in actual hyoid bone anterior or superior excursion. These results held true when the more acute stroke patients were removed from the data. The third outcome was an increase in the functional outcome assessment measure of swallowing (FOAMS score), where “1” is “profound” and “7” is “functional.” Five out of the seven brainstem patients returned to a “7.” A

fourth finding was a significant decrease in pyriform sinus residue. Fifth, all 27 patients had a complete resolution of post-deglutitive aspiration after six weeks of exercise. (However, it did not change the aspiration before the swallow.) The sixth and final result was that all the patients resumed oral intake and a discontinuation of the tube feedings. Twenty out of the 27 patients were on a regular diet. Only 2 out of 27 patients were still on nectar thick liquid due to aspiration before the swallow.

Mepani et al. (2009) studied the Shaker exercise in a randomized controlled trial starting with 19 patients. Only six of the patients completed a traditional therapy protocol and only five completed the Shaker protocol. These researchers review the inclusion and exclusion criteria for the use of the Shaker exercise (i.e., contraindications are pharyngeal surgery to strap muscles, cervical spine injury, lack of cognition, and inability to exercise independently or with a caregiver). The researchers stated, "contraction of the UES opening muscles is amenable to direct rehabilitation" (p. 30). After the six-week Shaker protocol, the maximum anterior-superior hyoid excursion increased from the baseline. There was statistical significance in comparing the after-Shaker group with the after-traditional-therapy group. This increased anterior excursion that results from the Shaker exercise allows for increased anteroposterior UES opening, and is due to contraction of the suprahyoid muscles and the thyrohyoid muscle. The effect of shortening and contraction of the thyrohyoid had not been studied prior to this research. The researchers also found this exercise to be effective in patients with post-deglutitive aspiration (aspiration after the swallow).

Summary

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. The patients may require an NPO status for several days due to an initially absent or non-functional swallow. They may not even be managing their secretions. Instrumental swallowing evaluations (i.e., MBSS) can help return the patient to safe oral intake as quickly as possible. The MBSS can be used in a therapeutic manner to improve swallow safety by

combining swallowing maneuvers (i.e., the Mendelsohn maneuver), swallowing strategies, a variety of food and liquid viscosities and bolus volumes under videoflouroscopy. Swallowing therapy should be initiated within the inpatient hospital stay and carried over aggressively through the patient's rehabilitation. For example, the teaching of the Shaker exercise can start while the patient is in the hospital. This approach accurately follows the neural plasticity principles of "Use it or lose it; use it and improve it, and repetition and intensity matter" (Robbins et al., 2008, pp. S278-281). Leaving a patient NPO for an unnecessarily prolonged period of time can be quite detrimental to the patient's outcomes.

Introduction to Case Studies

The following two cases have been presented in detail to reveal the following insights into diagnosis and treatment of lateral medullary infarction (LMI): challenges in the differential diagnosis of LMI; similar early neurological findings with the two patients; rapidly changing presentations; quick progress within the hospital stays, the importance of early and full swallowing evaluations to prevent aspiration, intubation, and pneumonia; and the fact that early initiation of exercises can safely return a patient to a regular diet within three months. See Table 3 and Table 4 for summaries of how these patients presented and how they progressed through their modified barium swallow studies (MBSS). All MBSSs were performed as described by Logemann (Logemann, 1993) and utilized EZEM Varibar barium sulfate suspensions for standardized liquid consistencies. Note: The rating system used for these MBSS was the Waxman Dysphagia Severity Rating Scale (1990) as noted in Table 2 on page 16 of this article. The testing of these patients preceded the MBS Imp measuring tool for swallow impairment.

Table 3

A Summary of the Two Cases of Young Vietnamese Males with LMI

Case 1: 36-year-old; hypertension, smoker	Case 2: 56-year-old; hypertension, diabetes, smoker
Presentation to the Emergency Department (ED):	Presentation to the Emergency Department (ED):
● Altered mental status, unsteady gait, change in speech and voice, inability to eat and drink, blurred vision, nausea and vomiting (all new per family) ● "Mumbled," and "garbled speech" ● History of Schizophrenia. Pt stated, "I took too many of the wrong pills." Was given charcoal absorbital liquid and went into respiratory distress. ● Intubated and ventilated until Day 2.	● Nausea, Vomited twice in the ED, 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 per Team and SLP Evaluation after Extubation (Day 3):	Clarification of Symptoms per MD (Intern) and SLP Evaluation (Day 2):
● Ataxia, leaning right ● Weak right side on transfer per nurse ● Dysarthria per interpreter ● No facial asymmetry ● Decreased gag bilaterally ● Hoarse, sustained "ah" = 2 seconds ● Lingual deviation to right with moderate weakness	● Progressive headache that started 4 weeks prior to admission ● 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, sustained "ah" < 2 seconds ● Lingual deviation to right

Case Study #1

This patient was a 36-year-old Vietnamese male with a history of hypertension, chronic paranoid schizophrenia, smoking, and asthma.

Day one. The patient was admitted to the emergency department with "bizarre behavior," change in mental status, unsteady gait, change in speech and voice, inability to eat and drink, blurred vision, nausea, and vomiting (all reported by the family as new that morning). The emergency department flow sheets at 1:30 P.M. hours report "dizzy since last night," nausea, questionable speech problems, unsteady gait, difficulty ambulating since last night, and left extremity weakness. Several notes described "mumbled" and "garbled speech." The patient was a poor historian, but via the interpreter he stated that "he took too many of the wrong pills." He also said "360 pills and 10 people." His head CT was negative and the initial chest x-ray was clear. The patient received a charcoal absorbital slurry (a thin liquid viscosity) and went into acute respiratory distress at 5:25 P.M. The repeat chest x-ray then showed left lower lobe consolidation secondary to aspiration and his white blood cell count increased to 12.2. His oxygen saturation dropped to 70% and emergent-non-traumatic intubation was done at 6:02 P.M. with ventilation. Charcoal was suctioned via the endotracheal tube. The blood toxicology screen was negative, but the urine screen positive for opiods. The differential diagnosis was possible opiod induced pulmonary edema and adult respiratory distress syndrome. However, the notes stated that the degree of opiod overdose was not known, and "urine opiods could be due to medications rather than drug use."

Day two. The nursing staff reported that the patient was sedated while intubated but still agitated and restless. An oral-gastric tube was in place and draining small amount of charcoal. Nursing notes indicated he was "suctioned orally for large amount bright red blood and large clots from oral cavity." He was extubated by 3:00 P.M. on day two. A nursing note reported that nystagmus was seen by a doctor. His white blood cell count was up to 20.9.

Day three. He was seen by Psychiatry, who noted the patient had no history of suicide attempts

and was in outpatient psychiatric treatment with only one hospitalization. The patient denied suicidal ideation. His speech was described as "coherent and somewhat pressured." The psychiatry report stated, "there is a question of an overdose" and "patient may be a candidate for involuntary inpatient psychiatric treatment" after medical clearance. The white blood cell count was down to 12.0.

Clinical bedside swallow evaluation (CBSE). The patient was seen by a speech-language pathologist (SLP) in the afternoon of Day Three. He was sitting up in chair with oxygen via face mask, maintaining an oxygen saturation of 97-99%. The interpreter reported that his speech was dysarthric but improved from the day of admission. The patient's voice was hoarse with decreased loudness, which he reported started suddenly one week prior to admission. His maximum phonation duration on the sound "ah" was two seconds. He had lingual deviation to the right on protrusion with moderately decreased strength.

The oral-motor examination also noted good lingual range of motion and lateralization, no facial asymmetry, good palatal elevation bilaterally, but a decreased gag bilaterally. His swallow function was tested with thin liquid, nectar thick liquid, honey thick liquid, and puree (baby food applesauce). He appeared to initially tolerate honey thick by spoon and 4 ounces of puree without overt signs or symptoms of aspiration. However, he had immediate coughing and overt signs or symptoms of aspiration with nectar thick liquid by spoon and cup and with thin liquid. The nursing note reported the patient was leaning to the right and had right-sided weakness on a transfer. The first SLP recommendation was for the Neurology evaluation to rule-out a brainstem cerebral vascular accident due to lingual deviation to the right and right-sided weakness, as well as sudden onset of ataxia, dysphonia, dysarthria, and dysphagia. A modified barium swallow study (MBSS) was ordered for the next morning, and the patient was kept NPO overnight.

Day four. The patient's oral motor status changed slightly: his lingual protrusion was within functional limits for strength, but still with mild deviation to the right. There was a positive gag bilaterally. Labial retraction was still symmetrical.

First MBSS. An MBSS classified the swallowing impairment as "severe pharyngeal and upper esophageal dysphagia." The report noted minimal laryngeal elevation or anterior hyoid excursion, incomplete laryngeal vestibule closure, and very poor upper esophageal sphincter (UES) opening. A minimal amount of the liquid bolus passed through the UES despite the use of head turns, effortful swallows, and the Mendelsohn maneuver. Silent penetration occurred during the swallow when nectar thick liquid and honey thick liquid were given via spoon in attempts to wash down the bolus of puree. The patient was suctioned to prevent aspiration, and the study was discontinued to prevent gross aspiration.

Nursing reported that the patient was taking large bolus sizes of applesauce on the ICU without significant noted distress. Large bolus sizes may have increased his UES opening with increased distention. However, this strategy was not deemed safe to attempt on this day.

The patient was made NPO and recommendations made to "consider MRI to rule-out brainstem infarct (due to) UES opening dysfunction." ENT was also recommended for laryngoscopy to view vocal cords. Therapy recommendations were for initiating the Shaker exercises for increased UES opening. The MBSS video was shown to the medical team on ICU to discuss a suspicion of lateral medullary syndrome due to classic symptoms bedside and on the MBSS. His poor UES opening seen on the MBSS was suspected to be due to a lack of relaxation of the CP muscle. The patient was started on a program of oral motor exercises and swallowing exercises that included the Shaker exercise. An MRI was ordered for that afternoon. His white blood cell count ranged from 10.3 to 14.4 on this day.

MRI/MRA of brain. (Imaging was performed utilizing A GE 1.5T LX MRI system. Sagital T1-weighted, axial T-2 weighted, axial flair fluid attenuated inversion recovery, axial iron T2 gradient echo susceptibility, axial and coronal diffusion weighted imaging.) The MRI showed a "fairly discrete left medullary focus of increased T2 signal, which shows a restricted diffusion consistent with an acute infarct." This was caudal and medial to the inferior cerebellar peduncle. The conclusion was a "discrete infarct of the Left Lateral Medulla. Clinical correlation for Wallenberg syndrome findings

warranted."

Day five. The Neurologist's findings were Left lateral medullary infarction due to a left vertebral artery occlusion. She noted left lateral rotating nystagmus, left Horner's, decreased gag bilaterally, and ataxia. The patient was started on Coumadin for 6-8 weeks.

Day six. An SLP followup visit in the morning noted only mild lingual weakness, no deviation of the tongue on protrusion, increased gait, and increased phonation duration on "ah." The patient still had decreased gag bilaterally, and he reported a decreased sensation of residue with bedside trials. A second MBSS was requested by the ICU team to re-start oral intake and facilitate discharge planning, as the patient had been wanting to leave against medical advice.

Second MBSS. The patient had a moderate pharyngeal and esophageal dysphagia with improved UES opening, suggesting improved cricopharyngeal functioning. The results showed improved, but still decreased, hyolaryngeal excursion. Large silent penetration occurred during the swallow with honey thick liquid, but this cleared after the swallow and did not lead to aspiration. Nectar thick liquid penetrated during the swallow with immediate aspiration during the swallow, showing potentially impaired vocal cord adduction. He had no spontaneous cough response, and his cued cough was non-productive. A head turn and tuck down to the left was more effective than to the right. (The residue was bilateral, but the left had more residue than the right.) This allowed for increased clearance of the bolus and decreased residue post-swallow in the valleculae, pyriform sinus, and posterior pharyngeal wall. Larger bolus sizes were attempted this time with increased bolus clearance with increased UES opening and distention. The patient began oral intake of puree and honey thick liquid with close supervision. Recommendations were for allowing large bolus sizes with the use of a head turn and tuck down to the left side and multiple swallows per bite and sip. Medications were crushed in applesauce. The patient was on strict aspiration precautions. The prognosis was deemed to be good due to the current rate of progress. However, the patient refused to go to a rehabilitation facility and was discharged to his home with his parents.

Day 12 and third MBSS. The patient had another MBSS as an outpatient six days later at the request of his primary care physician and due to the patient's complaints of coughing and choking on thin liquid and bread the night before. He had been non-compliant with the diet of puree and honey thick liquids at home. The third MBSS showed a continued moderate dysphagia, but he was able to be tested with more advanced solids (cracker and bread) and thin liquid. Only half of the pureed bolus and cracker bolus cleared through the UES on the first swallow. The ground meat bolus did not clear through the UES at all on the first swallow. He needed multiple swallows and liquid washes with nectar thick liquids. He only penetrated during the swallow with nectar thick liquid. There was no aspiration of the nectar thick liquid, as had occurred on the inpatient MBSS study six days earlier. He had improved hyolaryngeal excursion and laryngeal vestibule closure. However, he still aspirated on the thin liquid and on a mixed consistency (testing mixed consistencies simulates foods like soup with broth and cold cereals with milk). This aspiration was still during the swallow, but it was not silent (as in the previous study). The patient was educated by watching the video and being given a written swallowing instructions guide. His diet recommendations were for nectar thick liquids and a dysphagia ground diet (the safest range for solids was from pureed foods to very soft-moist solids and moist ground meat). He was advised not to eat any whole meats or challenging mixed consistencies. He was trained to use multiple swallows and liquid wash, only using nectar thick liquids. Outpatient speech therapy services were recommended.

Multiple re-evaluations with modified barium swallow studies. One month after his acute stroke, he was admitted with "flu-like symptoms," with two weeks of cough, fever, chills, and a five-pound weight loss. His chest x-ray showed right middle and lower lobe and left lower lobe patchy infiltrates and an "ill defined RUL density, may represent sub-acute resolving pneumonia." The admission note questioned a visual field deficit, noted continued tobacco use, and questioned the patient's compliance with medications. His white blood cell count was 15.7 on admission but decreased to 13.4 on the next day. The same speech-language pathologist (SLP) performed a clinical bedside

swallowing evaluation. The patient reported that his mother had been blending rice and solids at home. He stated that he used the thickener at home, but the SLP questioned his compliance as the patient was observed taking large sips of thin liquids during the hospitalization. An oral motor exam revealed that the patient was within normal limits for all testing, with no lingual deviation. His voice was perceptually noted still to be hoarse, but within functional limits for loudness. The bedside swallowing evaluation noted immediate coughing with trials of thin liquid. The cough was present even with nectar thick liquid due to impulsivity and large sips. Less coughing was noted with honey thick, but he was less likely to be compliant with this level of thickness. Puree was tolerated without overt signs or symptoms of aspiration. The patient was placed on a puree and nectar thick liquid diet with one-on-one supervision, small sips, and alternating liquids and solids during the hospital stay. Again, the inpatient SLP recommended outpatient therapy or an SLP from the visiting nursing association. He was advised to continue the Shaker exercises. His white blood cell count decreased to 8.8 on discharge after a few days of compliance with the diet. One month later, the patient was called by the inpatient SLP via an interpreter, and he stated that he was still being followed by the visiting nursing association.

Fourth MBSS. Two months later, an outpatient MBSS was performed as requested by the patient's outpatient therapist. The patient complained that his voice hoarseness was worse this week than during the last several weeks. Mild oropharyngeal and esophageal dysphagia was observed. He demonstrated the following: decreased mastication with whole cookie and whole meat, no clearance of these boluses through the UES on the first swallow, only one-third of the bolus cleared with three dry swallows, and severe residue in the pyriforms after a challenging solid bolus. He needed liquid wash to clear residue. Large sips of thin liquid caused silent penetration during the swallow, and he had mild pyriform pooling after the swallow with liquids. No frank aspiration was noted.

Delayed esophageal emptying was noted on a screening of the esophagus during this MBSS. A follow-up barium swallow revealed “spasticity of the esophagus at the gastroesophageal junction, particularly with the patient in the upright position so that the esophagus emptied rather slowly.” No

difficulty was noted in the prone and oblique position and there was no reflux or stricture.

A diet upgrade was recommended to a regular diet with cut-up meats and thin liquid. Cues were given to slow down his rate of intake, chew well, and alternate liquids and solids to clear the solid residue through the UES. Slow intake would allow for esophageal emptying. The patient was encouraged to continue the Shaker exercises. He was trained by watching the video; the therapist reviewed aspiration precautions, and encouraged him to increase his physical activity to allow for improved pulmonary clearance if aspirations occur.

Readmission. Two years later, when the patient was 39 years old, he was admitted with a change in mental status. He was diagnosed with aspiration pneumonia and hyponatremia. The patient had reported nasal regurgitation of liquids and solids and was seen by Neurology to rule out Wallenberg syndrome. The findings were as follows: intact cranial nerves, no nystagmus, intact gag bilaterally, motor 5/5, no drift, no cerebellar signs, sensory intact, and mildly wide-based gait. Neurology summarized that there was no recurrence of Wallenberg syndrome symptoms.

A repeat MRI/MRA showed only the previous left medulla infarction. MRA neck angiography was unremarkable, showing a dominant right vertebral artery. MRA brain angiography used axial 3D-time of flight MRA, and the findings were as follows: “the rostral left vertebral artery is seen from the level of the anterior inferior cerebellar artery (AICA) to the basilar. The **proximal left vertebral artery is likely occluded**. There is probable retrograde filling of the most rostral segment of vertebral artery. The right vertebral artery appears unremarkable. The basilar artery is unremarkable. The internal carotid arteries, anterior cerebral and middle cerebral arteries are unremarkable. There is no evidence of intracranial aneurysm or AVM.” Conclusion: “Non-visualization of the left vertebral except for probable retrograde filling from the level of AICA to the basilar.”

Fifth MBSS. During this inpatient stay, another MBSS was done to determine if there was an increased dysphagia. The MBSS found only mild pharyngeal dysphagia, which was similar to those of the fourth exam two years earlier. His pharyngeal swallow was only slightly delayed . He had mildly

weak closure of the laryngeal vestibule, allowing for penetrations to occur above the level of the vocal cords during the swallow with large sips of thin liquid from a cup. The penetration cleared after the swallow. The patient was independently using dry swallows, which cleared the mild amount of pyriform sinus pooling and residue. There was no cricopharyngeal dysfunction, with large liquid and solid boluses easily traversing the UES. The study also confirmed adequate velopharyngeal closure without penetration into the nasopharynx. The patient was placed on a regular diet and thin liquids with small sips and cues to decrease impulsivity. The SLP and the Neurologist agreed that the patient was back to his baseline, and the exacerbation of the dysphagia, causing the aspiration pneumonia, was likely due to hyponatremia. ***More admissions.*** The patient was repeatedly admitted between two years after the stroke to three-and-a-half years afterward for hyponatremia, change in mental status, and pulmonary infiltrates. However, the patient frequently signed out against medical advice (AMA).

Almost four years after the initial infarct, he had two admissions for "odd" or "bizarre" behavior. This was at one and two months before he died. He had increased falls, lethargy, headaches, an unsteady gait, confusion, seizure activity, and "eyes shifting to the left." Bilateral patchy infiltrates were found on both admissions. Head CTs were negative; and the patient was adamant about leaving AMA.

On his last admission, he complained of "not feeling well yesterday." The family thought he was intoxicated, but he had no history of alcohol use or abuse. The patient fell asleep in the bathtub. He was seen shaking at times. He had said to his family, "Someone is trying to kill me." He was unresponsive in the emergency department, pupils dilated, and notes reported "patient seizing," "foaming from mouth." His four extremities were shaking for 10-15 seconds, and then he went into respiratory and cardiac arrest. CPR was started, and he was intubated. The patient was transferred to another hospital for further cardiac care, and there he died. The conclusion was seizure induced rhabdomyolysis in combination with opioid overdose (as positive opiates were found).

Case one conclusion. This patient clearly showed rapid progress initially and was able to return

to a dysphagia ground (mechanically altered) diet and nectar thick liquids within 12 days. After three months he was upgraded to a dysphagia advanced (regular soft) diet with thin liquids. However, barriers to treatment were refusals of early rehabilitation, non-compliance with diet restrictions, and impulsivity with rapid intake due to his mental illness issues at baseline. He did perform the Shaker exercises (almost compulsively), as reported by family and outpatient therapists. The later issues in this case point to questions of whether the initial lateral medulary infarct was producing TIA's or seizure activity that was exacerbating his symptoms. In the months leading up to his death, he had three recurrences of the "bizzare behavior," similar to that which was actually seen on the initial stroke.

This literature review has revealed one study of a case of frequent TIA's before and after the onset of the lateral medullary infarction (Isa, Kimurea, Yasaka, Minematsu and Yamaguchi, 1999). He had new onset of seizure activity and may have been having cerebellar involvement as well. Heros (1982) stated that "many patients with cerebellar infarction run a subacute course, and some of these patients have premonitory signs or TIAs before the onset of their stroke" (p. 106).

Case Study #2

This patient was a 56-year-old Vietnamese male with a past medical history of hypertension, diabetes, hypercholesterolemia, insomnia, anxiety, and smoking.

Day one. The patient presented to the emergency department at 7:45 P.M. He was alert and oriented, but he complained of nausea, vomiting, and headache all day. The notes reported that the cranial nerves of 2-12 were intact "except the smile with left facial droop." The staff was told by the wife that the facial droop was old. The patient was in his "usual state of health until yesterday." He complained of dizziness that started in the emergency department. His blood pressure in ambulance was 162/98, but the emergency department reported 172/77, 184/87, and 197/91. His blood sugar was 203 when tested by EMS. Staff notes reported no diarrhea, a constant lightheadedness not related to movement and without a sensation of the room spinning, 5/5 strength in all four extremities, and

"unsteady gait." The patient complained of a "scratchy" throat and cough. He vomited twice in the emergency department by 10:05 P.M. (five times total). His troponins were negative. The Head CT was negative (old lacunar infarct, L basal ganglia, no intracranial hemorrhage or mass effect). The initial diagnosis was dizziness and an upper respiratory infection.

Day two. The House officer's assessment and progress note at 6:00 A.M. clarified the history of the illness further with the fact that the onset of the headache had been four weeks earlier, but it had been getting progressively worse. The headache was the worst on the day of admission with associated nausea, vomiting, and myalgia. The attending physician also clarified that double vision and dysphagia had started one day prior to admission. The fatigue, aches, "not feeling well" and dizziness had started a week before admission. In addition to the sore throat and body aches, the patient complained of numbness on the right side of his body. The house officer's 6:00 A.M. note reported the following additional findings: decreased sensation on right, hoarseness, diplopia, left eyelid droop, disjoint gaze (dysconjugate gaze), weak right gaze, dysphagia, neck pain, nystagmus, left facial droop, and subtle tongue deviation to the right. The patient was made NPO (nothing by mouth) as the findings indicated a possible stroke. A request for consultation was placed to Neurology, and the house officer called the speech-language pathologist to discuss the case.

Clarification of onset of symptoms. The SLP performed a clinical bedside swallowing evaluation (CBSE) with an interpreter present. The patient clarified that his symptoms, other than the headache, started suddenly at noon on the day of admission. These were gait instability, dizziness, and numbness on the right upper and lower extremity. He came into the hospital because of these symptoms. The diplopia, hoarseness and dysphagia to all food and liquid started the suddenly on that first night in the hospital. He was choking with soup and liquid and coughed it right back up. The family reported that he was "vomiting" up the food that he tried.

Oral-motor status evaluation. This revealed a left facial droop, decreased left nasolabial fold, decreased labial retraction on the left, adequate lip seal bilaterally, mild lingual weakness with slight

tongue deviation to the right on protrusion, adequate lingual range of motion, uvula deviation to the left, absent gag reflex, weak cough, effortful volitional swallow, and mild difficulty managing secretions with a wet-gurgly voice.

Speech and voice evaluation. The patient's articulation was deemed to be within functional limits in conversation, with the patient, interpreter and daughter denying any imprecise consonants or decreased intelligibility. Rapid repetition of the Vietnamese sound for “d” was slow, weak, but adequate in placement. His voice was judged to be moderately hoarse, weak, decreased loudness, and changed from baseline. During an attempt at maximum phonation duration of the “ah” sound, he was unable to sustain the sound longer than two seconds.

Food presentations. The patient was given ice chips, then puree (applesauce) in ¼ teaspoon to full teaspoon size bites, and honey thick liquid by spoon. He appeared to tolerate ice chips over two trials. Puree showed a prompt swallow, but with decreased laryngeal elevation to palpation. He denied a sensation of residue post-swallow. Honey thick liquid by spoon caused overt and immediate signs of aspiration with prolonged distress, coughing, and wet voice. Suctioning was performed orally with a yankauer suction. Testing returned to a ½ teaspoon size bolus of applesauce to determine if the patient could at least safely take his medications in applesauce. However, he then started to feel the residue and attempted to perform multiple dry swallows to clear. An ice chip was given to assist in triggering a dry swallow to clear the thick bolus residue; however, this increased the distress. These trials were discontinued. After 15 minutes, the daughter reported that he had just spit out a large amount of applesauce and cranberry juice (about the size of the palm of the hand and equal to the amount of oral intake given).

Impressions. The SLP reported that the patient was not safe for any oral intake. Severe pharyngeal dysphagia was expected. He was able to trigger a prompt pharyngeal swallow, but the motility was potentially non-functional. The SLP suspected a lack of UES relaxation and opening as the entire bolus was expectorated after the swallow. (Findings with the food presentation pointed to the fact

that the bolus was probably not entering the UES, and food and liquids were being retained in the pharynx). The patient initially had poor sensation of the bolus collecting in the hypopharynx, until the residue increased. His strong cough and expectoration showed that he could continue to manage secretions adequately. The SLP discussed her findings with house officer and the neurologist , and the team had concerns for Wallenberg syndrome (i.e., sensory changes, hoarseness, left Horner's, nystagmus, and sudden onset of dysphagia). ***Recommendations for the weekend.*** These included the NPO status and medications to be administered non-orally. If possible, a nasogastric tube should be placed. Nursing should repeat the nursing swallow screen with small amounts of puree only, as no thin liquid should be tested until the modified barium swallow study (MBSS) in the following week. The SLP expected rapid improvements in the swallowing function in 2-3 days so that the patient would achieve some functional modified diet for oral intake instead of the need for a percutaneous gastronomy tube. The house officer ordered an immediate MRI/MRA of the head and neck.

Neurologist evaluation later that day. The neurologist saw patient after the CBSE and prior to the MRI. Remarkable findings were as follows: eyes showing mild asymmetry of pupil size with left at 1.5 mm and right at 2.5 mm, left eyelid droop, mild nystagmus on extreme gaze with corrective component to the right, mild left central facial, decreased left nasolabial fold, and tongue deviation mildly to the right. No pronator drift was noted, although the patient was slow on the right finger tap. He had a very subtle dysmetria on the right hand with the finger-to-nose test. He was able to raise both legs against gravity and showed bilateral plantar reflexes, but the neurologist did note “some ataxia.” Recommendations were for a workup for stroke with brain MRI/MRA, neck MRI/MRA and an echocardiogram. The patient was started on Lovenox.

The carotids were unremarkable. The echocardiogram was unremarkable with an EF of 50-75%. The lipid panel showed elevated cholesterol. Troponins were negative over three tests. The attending physician suggested that the patient's “tachycardia may be due to anxiety.”

MRI and MRA. The MRI of the brain was performed without contrast (sagital T1 and axial T2,

flair, gradient echo, and diffusion-weighted images) and showed: “small area of restricted diffusion within the left dorsal mid/lower medulla, with corresponding T2 hyperintensity in the region.” The patient also had chronic lacunar infarcts with old hemorrhagic products in the posterior aspect of the left external capsule. There was an additional chronic lacunar infarct within the posterior limb of the internal capsule and tiny chronic lacunar infarcts within the basal ganglia bilaterally. The MRA of the neck was unremarkable. An MRA of the head without contrast showed a “hypoplastic left A1 segment in an otherwise unremarkable examination.”

Image 1 on MRI for Case Study #2

*Restricted diffusion in the left lateral medulla shows up as a bright signal (white in the first image) on diffusion weighted imaging (DWI), with corresponding low signal (dark on image 2) on apparent diffusion coefficient (ADC) sequences. (***this comment describes both images)**

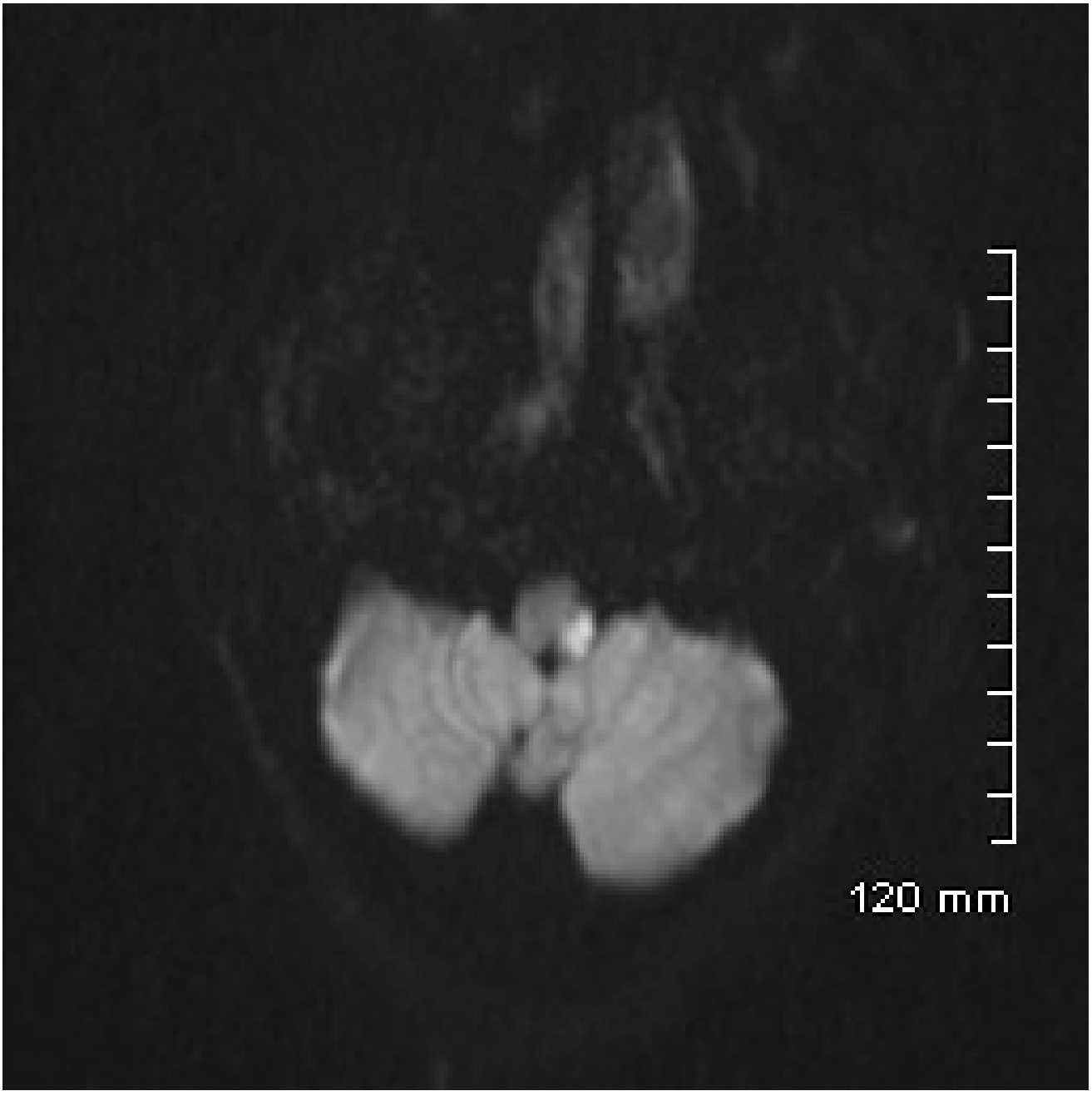
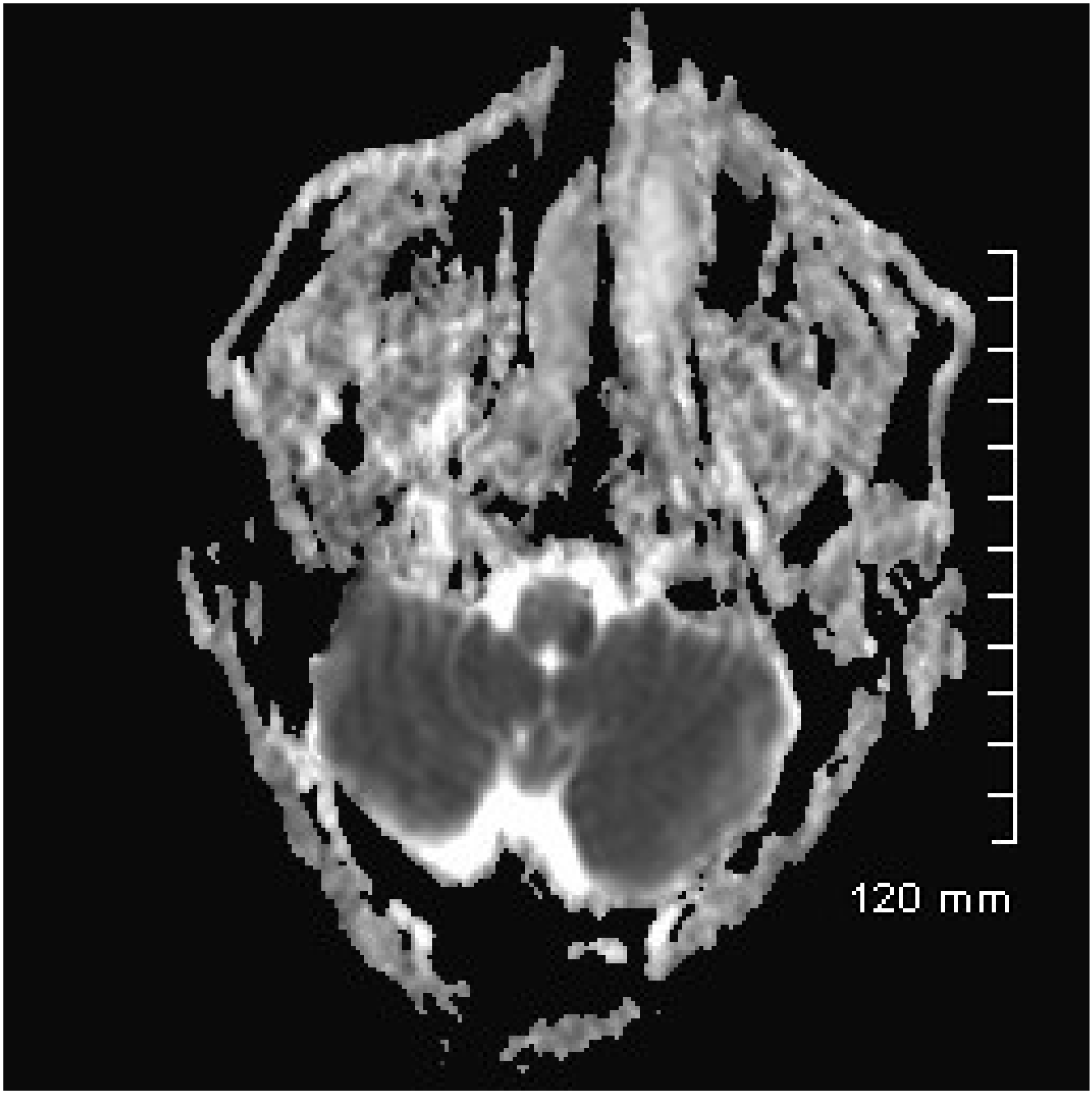


Image 2

MRI on Case Study #2



Weekend events. The house officer noted that the patient slowly recovered some sensation in the right upper and lower extremities. He still presented with a left eyelid droop and left nasolabial fold droop, but the raising of the eyebrows was intact. Still no gag was noted. The uvula deviated to the right, and tongue deviation persisted. The nasogastric tube was not placed. The patient's strength was still adequate in all four extremities. Notes reported that this was "likely a hypertensive CVA."

Day Six. The notes stated that the patient had double vision in left eye. The doctor's impressions were that arrhythmia was the cause of embolic event for CVA. The SLP reported that the patient denied a headache, but recalled that he had one the night before. He complained of dizziness and diplopia, with second image above and to the right of the first image. He also reported increased sensation on the right upper extremity. His lingual deviation was only slightly to the right with moderately weak lingual strength on protrusion and lateralization. He still showed a slightly weak left nasolabial fold. Secretions pooled posteriorly, and the patient reported secretions getting stuck in his throat and causing a blockage of the airway at times and difficulty breathing. His voice was not wet-sounding at the time, but it had decreased loudness and became more hoarse after a sustained phonation of 3-4 seconds; however, this phonation duration was longer than on the initial evaluation.

First modified barium swallow study (MBSS). An MBSS was performed later that morning with an interpreter present. The consistencies tested proceeded in this order: ½ teaspoon of nectar thick liquid, full teaspoon of nectar thick liquid over two trials, ½ teaspoon of applesauce, full teaspoon of applesauce, applesauce with a head turn to the left, teaspoon of pudding with head straight ahead, ½ teaspoon of thin liquid, full teaspoon of thin liquid, cup sip of thin liquid, cup sip with chin tuck, cup sip with turn and tuck to the left over two trials, nectar thick by cup, honey thick by cup, and thin liquid by cup. A head turn to the left was deemed to be most effective, and this was likely due to ipsilateral paresis of left true vocal cord and left-sided pharyngeal weakness with his left LMS. He was cued to perform effortful swallows with all presentations.

MBSS Impressions: The patient presented with moderate oropharyngeal dysphagia due to an

acute left lateral medullary infarct. Initially, the patient had moderate residue with larger bites of applesauce from the tongue base through to the pyriforms. Pooling with liquids increased as the viscosity increased. There was mild to moderate pooling in the left pyriform with thin liquids by cup, but moderate pooling after honey thick liquids. Residue and pooling decreased with a head turn and tuck to the left for the subsequent dry swallows.

The patient's pharyngeal swallow response was mildly delayed. He had slow and moderately decreased hyolaryngeal excursion, epiglottic inversion, and UES opening. The patient was found to have a cricopharyngeal dysfunction (decreased sphincter relaxation) with proximal esophageal stasis, especially with honey thick liquids. Larger boluses were effective in improving the UES opening, as the large boluses aided in improved distention.

He had delayed initiation of the pharyngeal swallow with the head of the bolus in the pyriforms with thin and nectar thick liquids before the swallow. He tolerated the spoon sips with thin and nectar thick liquids. Very small sips of thin liquid by cup caused penetration above the level of the vocal cords during the swallow. Penetration and aspiration were not prevented by using a chin tuck or chin turn/tuck down to the left for thin liquid by cup. He had one gross aspiration out of three trials, even with the head turned and tucked down to the left. The aspiration was during and after the swallow. He responded with a spontaneous cough, but it did not clear the aspiration. Nectar thick liquids by cup without the use of strategies caused a penetration during and after the swallow to the level of the vocal cords, which lead to aspiration after the swallow due to non-productive cough. He also had increased pooling in the left pyriform sinus with the nectar thick liquid, which he did not detect; therefore, it would further increase his risk for aspiration after the swallow. Although the patient tolerated honey thick liquid by cup without any penetration, he was advised to perform a double swallow using the chin turn and tuck to the left to clear the pooling in the left pyriform. Pooling in the pyriforms and aspiration after the swallow are indications of UES dysfunction and for the use of the Shaker exercise program.

Recommendations. The safest diet to start oral intake was deemed to be pureed solids, honey

thick liquids by cup, and medications crushed in applesauce. The important swallow strategies were for small sips, double swallow with liquids with chin turn and tuck down to the left, slow rate, small bites, and frequent coughs with an immediate dry swallow throughout the meal. The patient's prognosis was deemed to be good, but acute rehab was recommended. A repeat MBSS was recommended in two weeks at acute rehab as progress is typically fast. Therapy plan suggestions were to start the Shaker exercises to improve the extrinsic muscles of the larynx (laryngeal strap muscles) for better laryngeal elevation and UES opening. Oral-motor exercises and the Mendelsohn maneuver were recommended as a focus of therapy as well.

Day Seven Through Nine. The patient did not qualify for acute rehab because he had no insurance. The primary care physician wanted him to stay through the end of the week to obtain as much therapy at the hospital as possible before being discharged to his home without services. He did receive some free home-care services. He had daily physical therapy through that week in the hospital, and notes reveal that he was very unsteady. Lovenox was discontinued, and the patient started on Plavix. The notes stated to continue ACE and statins. His headache pain level was down to Level 1 out of 10 daily with pain medication, but he still complained of dizziness, blurry vision, and double vision.

Each day the speech-language pathologist trained the patient and his family members extensively through interpreters to ensure that they could continue the therapy at home. The training included a Shaker exercise program, and the patient was doing these two times a day. The patient and his family demonstrated competency in thickening liquids with a thickener powder for safety at home. All the safe-swallowing strategies were reviewed with the patient and his family. He was independent in using these strategies during meal observations and showed no distress at meals. His intake of meals was 100%. Oral-motor progress was as follows: increased lip seal on left, increased retraction of the nasolabial fold on left, tongue fully at midline by Day Eight on protrusion exercises, and only mild weakness noted in lingual exercises. The patient's voice improved in loudness and duration with the patient producing an “ah” while pushing or pulling with the left hand. He was given vocal function

exercises, including maximum phonation durations with “ah” and falsetto “ee.” These were effortful and his voice quality was still hoarse, but he could sustain his vocalization for three seconds. The patient was discharged home with a plan to return for a repeat MBSS within two or three weeks for further diet upgrades.

Second modified barium swallow study. This study was done 22 days after the CVA by a different therapist as an outpatient. The patient had mild-moderate oropharyngeal dysphagia with reduced lingual strength, delayed bolus formation, and decreased anterior-posterior propulsion of the bolus. He continued to experience a mildly delayed initiation of the pharyngeal swallow. Reduced pharyngeal peristalsis (pharyngeal stripping wave) was noted with slow pharyngeal transit of the soft and regular solids. Reduced epiglottic deflection caused mild amounts of solid residue to remain in the valleculae. Moderately decreased hyolaryngeal excursion and UES opening caused moderate amounts of residue and pooling in the pyriform sinuses with both liquids and solids. Multiple swallows and alternation of liquids and solids were effective to reduce this residue to trace amounts. The patient denied experiencing a sensation of this residue. No penetration or aspiration was seen with thin liquids by spoon, cup, or straw. Impulsivity was noted at times as he consumed solids in large bites.

Recommendations. A dysphagia ground diet and thin liquids were recommended. The patient still required one-on-one supervision for meals. Strategies were for medications to be crushed in applesauce, double swallows with liquids and solids, alternating liquids and solids, no straws, small bites and sips, slow rate, and checking for pocketing on the left side of the mouth. The plan was to continue the swallowing exercises including oral-motor strengthening, laryngeal elevation exercises, and the use of safe swallowing strategies. The MBSS was recommended to be repeated in four weeks to determine the patient's readiness for diet upgrades.

Final outpatient modified barium swallow study. This outpatient study was done almost exactly three months after the CVA by the original clinician who evaluated him as an inpatient.

Interview. The patient was interviewed through an interpreter at this return visit, and the

following information was gathered. He had been performing oral motor exercises daily, especially the lingual strengthening exercises. He performed the Shaker exercise "many times" every day, and he clarified this to say at least 30 times per day. His said that his weakness "comes back" if he stopped exercising, and he felt this weakness bilaterally. He denied that he felt numbness in the face and extremities, but stated it is "uncomfortable." He denied that he had double vision, but if he looked down for a long time, he still felt some dizziness. He described the dizziness as "often" during the day, but better on his medications. His walking around the radiology department was steady but slow. Overall, his swallowing was "better;" however, his family was still worried about his coughing with rapid intake. He was eating a regular consistency, but the wife cut the food into small pieces. He complained about breads and dry foods. The solids were easier than liquids, as liquids still made him cough with rapid intake. He denied that he had difficulty with talking medications whole with thin liquids.

Oral-motor examination. There was mild weakness on lingual protrusion, but the tongue protruded to midline. Lateralization of the tongue was mildly weaker to the right side. Labial retraction and palatal elevation were within normal limits bilaterally. His voice was reduced in loudness, but adequate for conversation. Maximum phonation duration was 10 seconds with "ah."

Findings of the MBSS. The patient presented with only minimal pharyngeal dysphagia. The patient had a mildly slow pharyngeal swallow initiation with mildly reduced hyolaryngeal excursion at times, especially with smaller boluses. There were slight penetrations above the level of the vocal cords with thin liquids just before achieving full epiglottic inversion, potentially due to the mildly delayed swallow. The penetrations were ejected out of the laryngeal vestibule with the completion of the swallow, and no aspirations were noted. He tolerated large consecutive sips of thin liquids without penetration to the vocal cords or aspiration. He achieved full laryngeal elevation and improved anterior excursion with **large bolus sizes** of ground meat, cracker, bread, and whole meat. No residue after the swallow was noted. He only had minimal residue in the proximal esophagus after the swallow.

Recommendations. The SLP recommended a regular diet and a return to normal-size bites. The only strategies that were suggested were chewing well and eating at a slow rate to allow for his slightly delayed pharyngeal swallow initiation.

Case two conclusion. This final MBSS showed a good return to a fairly normal swallowing function three months after his large lateral medullary infarct. This patient's recovery is consistent with research findings of a quick recovery in young patients with this type of stroke. This patient complied with instructions well and was able to perform daily oral motor and swallowing exercises at home, including the Shaker exercise. These measures contributed to his good outcomes and to the spontaneous recovery. He had no aspiration pneumonia re-admissions as occurred with case one above.

This case study reinforces J.S. Kim's (2003) point about how 25% of patients may present with headache, vertigo, dizziness, and ataxia first. Then the later symptoms can be the hoarseness and dysphagia. This patient's ipsilateral facial weakness and contralateral lingual weakness were interesting findings. He also had the less common sensory pattern in just the contralateral limb and body versus the typical loss of pain and temperature sensation to the ipsilateral trigeminal/face and the contralateral limb and body (J.S. Kim, 2003). The patient's 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. On the other hand, J.S. Kim (2003) demonstrated that rostral lesions tend to have more aspiration.

The important message is that with any suspected brainstem infarction, the patient should remain NPO until the swallowing is fully evaluated. In this case, early identification of the infarction by the team prevented major aspiration and intubation.

Table 4

Comparison of Progress Documented with Modified Barium Swallow Studies (MBSS) in Two Young Vietnamese LMS Patients

Case 1	Case 2
Day 4 MBSS Severe pharyngeal and esophageal dysphagia. No UES opening. Minimal amount of liquids passed through UES. Penetration and suctioning needed. Remained NPO. **Day 6 MBSS** Now moderate dysphagia. Some UES opening. Larger boluses partially cleared through UES, using head turn and tuck to L (weaker side). Silent aspiration with nectar thick liquid. Tolerated puree and honey thick liquid with head turn and tuck to L and double swallow.	**Day 2 Clinical Bedside Swallowing Evaluation** Was not ready yet for MBSS due to overt residue, aspiration, and expectoration of all oral trials bedside. NPO through that weekend. **Day 6 MBSS** Moderate oropharyngeal dysphagia. Delayed pharyngeal swallow, decreased UES opening, aspiration with thin and nectar. Tolerated puree and honey thick liquid with head turn and tuck to L (weaker side). Double swallow to clear residue above UES.
MBSS at 2 ½ months post Mild oropharyngeal and esophageal dysphagia. Still no clearance of the bolus through the UES on the first swallow. Severe residue in pyriforms. Needed double swallows and liquid wash. Silent penetration only with large sips of thin liquid. Regular soft diet with strategies and supervision.	**MBSS at 3 months post** Minimal oropharyngeal dysphagia. Mildly delayed and slow swallow, mildly reduced laryngeal elevation. Penetrations with thin liquids, but above level of vocal folds and ejected. Improved anterior excursion of larynx and no residue above the UES with solids. Minimal residue in the proximal esophagus.