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Dysphagia after Stroke: an Overview

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Abstract

Dysphagia affects the vast majority of acute stroke patients. Although it improves within 2 weeks for most, some face longstanding swallowing problems that place them at risk for pneumonia, malnutrition, dehydration, and significantly affect quality of life. This paper discusses the scope, the disease burden, and the tools available for screening and formal evaluation of dysphagia. The most common and recently developed treatment interventions that might be useful in the treatment of this population are discussed.

Keywords

Dysphagia; Stroke; Swallowing; Deglutition

Introduction

For the vast majority of the 6.2 million stroke survivors [1] in the US one of the first hurdles on the path to recovery is swallowing dysfunction. Dysphagia not only increases morbidity and mortality after stroke but also significantly affects quality of life when it is not possible to share meals with family and friends. Prompt evaluation and treatment of swallowing disorders can mitigate the development of secondary complications and foster prompt reintegration into society.

Epidemiology

Dysphagia affects more than 50% of stroke survivors.[2] Fortunately, the majority of these patients recover swallowing function within 7 days, and only 11-13% remain dysphagic after 6 months. [3, 4] One study reported that 80% of patients with prolonged dysphagia required alternative means of enteral feeding.[5]

The most feared complication of dysphagia after stroke is aspiration pneumonia. Dysphagia identified during bedside clinical examination was associated with an increase of 17% in the incidence of pulmonary infection compared to those that were not dysphagic (33% vs. 16% respectively).[6] In the same study mortality was more than 30% in stroke survivors with dysphagia. Dehydration and malnutrition also are common in dysphagic patients especially

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those who receive thickened liquids or modified diets. One study reported that 49% of stroke survivors admitted to a rehabilitation unit were malnourished, and that malnutrition was associated with dysphagia.[7] Gordon et al. (1987) reported that approximately 58% of acute stroke survivors with dysphagia had signs of dehydration (urea concentration of 10 mmol/l or higher) compared to 32% of those that were not dysphagic. [8]

Dysphagia can adversely impact quality of life. Only 45% of patients with dysphagia find eating enjoyable, and 41% of patients with dysphagia experience anxiety or panic during mealtimes.[9] More than 1/3 of patients avoid eating with others because of dysphagia. [9]

Swallowing Physiology

Swallowing requires food passage from the mouth through the esophagus and into the stomach without compromising the adjacent structures: nasal passages, larynx and the lower respiratory tract. The process starts after food ingestion and can be divided into four stages defined by the location of the bolus: [13]

1. Oral preparatory stage: prepare bolus for propulsion into pharynx
2. Oral propulsive stage: tongue pushes bolus through the fauces into the pharynx
3. Pharyngeal phase: pharyngeal structures move bolus through the upper esophageal sphincter.
4. Esophageal phase: esophageal peristalsis and gravity move the bolus through the lower esophageal sphincter into the stomach.

More detailed perspectives on swallowing physiology can be found in reviews by Cecconi and Di Piero, Miller, and Matsuo and Palmer. [10-12]

Oral Stage

Once a liquid bolus is ingested it is sealed off between dorsal tongue and the soft palate to prevent leakage into the pharynx until ready for swallowing. During the propulsive stage the tip of the tongue contacts the hard palate behind the upper anterior teeth and the tongue surface moves upward. The tongue-palate contact area expands posteriorly and squeezes the liquid bolus into the oropharynx.

In contrast, when eating solids, the tongue shifts backwards and rotates its surface to one side pulling the food back to the molar region and placing it on the occlusal surfaces for mastication (also known as stage 1 transport). During mastication oral structures work to reduce bolus size and soften it in preparation for the pharyngeal stage. [14-16] Between mastication cycles particles that have reached the appropriate size and consistency are moved to the dorsal tongue surface and are transferred to the oropharynx (Stage 2 transport).[17] These particles may accumulate for several seconds until a critical mass is reached and the pharyngeal stage of the swallow takes place.

Pharyngeal Phase

Pharyngeal transport requires multiple coordinated and almost simultaneous events: [18-20]

1. The soft palate elevates and seals the nasopharynx.
2. Pharyngeal tongue surface pulls back while the pharyngeal wall contracts squeezing the bolus downward. The pharynx contracts sequentially from top to bottom,[21] and shortens vertically to reduce its volume.[22]
3. The hyoid and larynx move superiorly and anteriorly while the epiglottis folds backwards sealing the laryngeal vestibule. These motion helps protect the airway.[23]
4. The vocal folds close the glottis [24-26] and interrupt breathing for 0.4-1.0 seconds to prevent aspiration.[27, 28]
5. The upper esophageal sphincter (UES) opens by a combination of cricopharyngeus relaxation (UES is held closed by its active contraction), suprahyoid muscle contraction, and the force of gravity generated by the down-moving bolus. [23, 29]

Esophageal Phase

Once the bolus passes through the UES, peristalsis and gravity move it down to the lower esophageal sphincter through which food reaches the stomach.

Neural Control of Swallowing

Normal control of swallowing requires appropriate function of the brain stem, the basal ganglia, the thalamus, the limbic system, the cerebellum, and the motor and sensory cortices (Table 1). These systems control afferent and efferent, anticipatory and preparatory, voluntary and automatic processes. Over 30 muscles are involved in swallowing and are coordinated by a complex neural network that is not completely understood.

The central pattern generator (CPG) for swallowing is located in the area of the nucleus tractus solitarius (NTS), the reticular formation, and nucleus ambiguus (NA) in the rostral and ventrolateral medulla.[30-32] Its interneuronal network controls timing of the deglutition phases and integrates sensory and supramedullary afferent with efferent processes.[30]

Sensory input from mechanoreceptors, chemoreceptors and thermoreceptors in the oral cavity, pharynx and larynx to the CPG has been shown to affect swallowing initiation, facilitation and airway protection.[31-34] The oral cavity sensory neurons synapse in the trigeminal sensory nuclei, while pharyngeal and laryngeal sensory neurons travel in branches of CN IX, X and XI to synapse in the NTS.[35]

The ventral CPG premotor neurons connect with CN V, VII and XII and CN IX and X in the NA.[31] Timing of motor outputs varies with bolus characteristics.[36] Functionally, these peripheral connections coordinate different phases of deglutition. For instance, peripheral feedback inhibits the esophageal swallow during the pharyngeal swallow. It is likely that the trigeminal nucleus and reticular formation control the oral phase. The NTS regulates the sensation and pattern-generation, with the NA and dorsal motor nucleus controlling motor efferents for pharyngeal and esophageal phases.[37] The ventromedial nucleus of the NTS may be responsible for coupling the pharyngeal and esophageal phases.[37] Since these areas are crucial for pattern generation a lateral medullary stroke (Wallenberg's Syndrome) can

result in dysphagia that is usually severe and results in aspiration.[34, 38] This lesion affects the CPG and the CNs involved in swallowing; paralyzes or weakens the ipsilateral pharynx, larynx and the soft palate; and initiates and coordinates the pharyngeal stage of deglutition. Notably, electrophysiological studies demonstrate that the acute disconnection of contralateral swallowing centers also takes place.[34]

Several supratentorial structures are associated with swallowing. In stroke, the size of the unaffected swallowing cortical area predicts dysphagia symptoms.[39]

Voluntary initiation of deglutition requires cortical motor inputs.[33, 40, 41] The motor and pre-motor cortices control deglutition bilaterally but asymmetrically[42] with no clear left-right laterality.[43] Recovery from supratentorial stroke induced dysphagia has been associated with compensatory cortical reorganization. [44]

Suprabulbar Palsy associated with dysphagia, dysarthria, dysphonia, loss of voluntary tongue and face movement and emotional lability, may be caused by bilateral lacunar infarcts or amyotrophic lateral sclerosis. Dysphagia in these patients has been correlated with lesions in the basal ganglia resulting in poor swallow triggering and upper esophageal sphincter control.[45]

Lesions in the left periventricular white matter may be more disruptive to swallowing behavior than those on the right.[46] Oral transfer can be significantly impaired in patients with purely subcortical strokes.[46] Positron emission topography (PET) has visualized asymmetric swallow-associated loci in the right orbitofrontal cortex, left mesial premotor cortex and cingulate, right caudolateral sensorimotor cortex, right anterior insula, bilateral medial cerebellum an bilateral temporopolar cortices with the strongest signals in the sensorimotor cortices, insula and cerebellum.[47]

Leopold and Daniels (2010) have documented the roles of brain locations and swallowing stages.[40]

Dysphagia Screening

Dysphagia screening serves to determine the possibility of aspiration (overt or silent) before complications such as pneumonia, dehydration, malnutrition, or airway obstruction develop. Multiple clinical tools have varying sensitivity and specificity (Table 2). Several of these tests are used not only for screening, but also for bedside assessments. Most tools assess a few clinical features and/or a water swallowing trial. Daniels et al. (1997) proposed a screen that does not include a water swallowing trial, but has comparable sensitivity and specificity to other tests involving water swallowing trials.[48] The screening is considered positive if any two of the following are present: 1)dysphonia, 2)dysarthria, 3) abnormal gag, 4) abnormal volitional cough, 5) cough after swallowing, or 6)voice changes after swallow. The Toronto Bedside Swallowing Screening Test (TOR-BSST©) is the only screening tool to includes an assessment of pharyngeal sensation.[49]

In a systematic review of bedside screening tests, Bours et al proposed a water swallowing test combined with pulse oximetry with end points of coughing, dysphonia, and choking as a method to screen patients with dysphagia and aspiration.[50, 51]

In attempt to validate a physician-specific tool for screening dysphagia, Antonios et al. (2010) have proposed the use of The Modified Mann Assessment of Swallowing Ability(MMASA).[52] Their preliminary findings suggest that the MMASA is valid and reliable for screening stroke survivors with dysphagia.

Diagnostic Evaluation of Dysphagia: bedside and instrumental assessment

Many bedside and instrumental tools have been developed for the diagnosis and treatment of post-stroke dysphagia. These tools acquire data with regard to pressure, range, strength of structural movement, airway protection, sensation, bolus clearance and efficiency, and bolus flow patterns.[53]

Dysphagia evaluation tools can be grouped broadly as imaging (Ultrasound, Videofluroscopy, Fiberoptic endoscopic evaluation of swallowing, and Fiberoptic endoscopic evaluation of swallowing with sensory testing) and non-imaging(beside assessment tools, and pharyngeal manometry).

Clinical Bedside Assessments

Carnaby-Mann and Lenius (2008) defined a dysphagia clinical bedside assessment as encompassing clinical history, and thorough examinations of the oral, pharyngeal, and laryngeal anatomy.[54] In addition, a neurological examination focusing on sensory and motor function, cognitive, behavioral, language abilities, and a trial of feeding should be performed if clinical indicated.[54, 55] Clinical bedside assessments are inexpensive, non-invasive, and easy to perform by speech language pathologists. The initial evaluation provides the foundation on which a treatment plan can be synthesized.[56, 57]

Though clinical evaluation provides valuable information, sensitivity and specificity for identifying aspiration risk is generally low.[58-60] Many clinical assessment tools have been proposed for dysphagia [61-64]. A summary of the most common bedside swallowing evaluations, their features, and validation data can be found in Table 3.

Videofluorographic Swallowing Study(VFSS)

The VFSS, also known as Modified barium swallowing (MBS) study, is considered the gold standard for evaluation of oropharyngeal dysphagia.[65, 66] The VFSS usually is performed by a speech language pathologist and physician (Physiatrist or Radiologist), and allows direct visualization of bolus flow, swallowing physiology, and airway invasion in real time. The ability to observe the oropharyngeal phase of swallowing allows clinicians to characterize the mechanism and severity of impairment. The VFSS also allows the clinician to observe the important relationships between swallowing, food consistency, position, and ventilation.[53, 66] The protocol described by Logemann et al. in 1993 continues to be followed in most clinical settings.[67] The process involves anteroposterior and lateral view of the oral-pharyngeal phase, with slow motion features to allow characterization of the

swallow mechanism and severity of dysfunction. Lateral view allows assessment of oral-pharyngeal transit time, delay, and physiological problems. Anterior views delineate residue asymmetries in the valleculae and pyriform sinuses, and visualize adduction/abduction of the vocal folds. Specifically the study measures the speed and efficiency of swallow, and defines the movement patterns of the oral cavity, pharynx and larynx. By knowing where, when, and how much aspiration occurs during the study the clinician can evaluate effectiveness of planed rehabilitation strategies.

Most recently, a protocol has been developed for standardization of the VFSS.[66] The development of the MBSImp allows quantification of swallowing impairments identified during VFSS.

Fiberoptic Endoscopic Evaluation of Swallowing (FEES)

Fiberoptic endoscopic evaluation of swallowing (FEES) often complements the VFSS where limitations exist.[68-71] FEES is a safe and well tolerated procedure done by both the otolaryngologist and/or speech pathologist alone.[72, 73] FEES is as or more sensitive than VFSS in assessing delayed swallow initiation, pharyngeal residue, and aspiration.[74-76]

The FEES examination uses a flexible endoscopy for evaluation of static and dynamic pharyngeal anatomy, the presence and ability to manage oropharyngeal secretions, and swallowing different consistencies of solids and liquids.[77]

During swallowing, transition duration, evidence of penetration and aspiration, the number of swallows to clear the bolus, and the extent of airway closure is noted. The scope is advanced trans-nasally along the floor of the nose until the end of the scope is at the base of uvula or at the tip of the epiglottis. This allows visualization of tongue base, lateral and posterior pharyngeal walls, pyriform sinus, and endolarynx.

In the mid-1990's Aviv et al introduced fiberoptic endoscopic evaluation of swallowing with sensory testing (FEESST).[78] FEESST is similar to FEES, but includes controlled air pulses to allow objective determination of laryngopharyngeal sensory discrimination thresholds. The air pulses are increased in pressure until a laryngeal adductor reflex(LAR) is elicited. The normal LAR has been established as less than 4.0 mmHg.[79] Utility of LAR is questioned by some clinician due to intra-rater and inter-rater reliability. [80] Laryngeal sensation as measured during FEESST is not an important factor when evaluating swallowing of pureed foods. [81] A prospective, randomized comparison of FEESST and VFSS demonstrated similar abilities to prevent aspiration pneumonia.[82]

Some proponents of FEES and FEESST feel these tests have surpassed the gold standard of VFSS. However, it is important to note that while sensation and vocal cord pathology are best evaluated with FEES, the oral and esophageal segments are poorly assessed. VFSS remains the best way to visualize these areas. Thus, VFSS and FEES/FEESST have different clinical applications.[80]

Pharyngeal Manometry

Pharyngoesophageal manometry is used to investigate physiological functions of the upper esophageal sphincter, integrity of the pharyngeal peristalsis, and intrabolus pressures. Esophageal manometry is the gold standard for evaluation of esophageal motor function. However, It is not the primary diagnostic tool for dysphagia in stroke, and is performed only when history, videoflurography, or endoscopy has failed to provide diagnosis or point to a motor disorder. Manometry usually is performed by a gastroenterologist, and is indicated in dysphagia with solid food associated with weight loss and regurgitation. The American Gastroenterological Association has provided a technical review on the indication of manometry in relation to other diagnostic tools for assessment of dysphagia.[56, 83]

Treatment of dysphagia post stroke

Through interview, clinical swallow evaluations, and instrumental assessments, the clinician gathers information regarding the patient's cognition, physiological impairments, sensory impairments, and appropriateness for initiation of oral intake. During objective assessments, the effectiveness of compensatory strategies also is evaluated to provide the patient with the safest yet least restrictive diet. The information gathered from these assessments then is used to develop an appropriate and individualized rehabilitation program.

Dysphagia rehabilitation is comprised of both compensatory and rehabilitative approaches.[84] Compensatory strategies are used to reduce symptoms of dysphagia without altering the physiology, while rehabilitative approaches are designed to improve swallowing physiology and improve swallow safety and tolerance of the least restrictive diet.[85]

Some strategies are both compensatory and rehabilitative in nature, in that they may eliminate symptoms of dysphagia acutely, and when used over time, improve swallowing physiology (Table 4). Several techniques are commonly applied in dysphagia rehabilitation. Traditional treatment techniques include tongue strengthening exercises, thermal-tactile stimulation, tongue hold exercises, Mendelsohn maneuver, supraglottic and super-supraglottic swallow, effortful swallow, and the Shaker exercise.[86]

After an objective assessment, compensatory strategies and a combination of traditional therapy techniques are initiated. Traditional therapy techniques significantly improve swallowing physiology with each exercise having a different impact on the swallow.[87]

However, it can be difficult to attribute the improvement to any one technique, as they are often used in combination.[88] Table 5 describes traditional therapies and their expected effects in more detail.

Biofeedback methods, such as surface electromyography (sEMG) have been used in conjunction with traditional therapy approaches to "increase awareness of swallowing patterns and to help the patient modify, monitor, and challenge performance while executing swallowing maneuvers". [89] sEMG has been reported to increase the rate of progress seen in traditional therapy approaches for patients with chronic dysphagia.[90]. In a retrospective study of stroke survivors or head and neck cancer patients, the use of biofeedback with

traditional therapy approaches significantly improved swallowing function and oral diet tolerance, with the stroke survivors benefitting more than the cancer patients.[90]

Neuromuscular electrical stimulation (NMES) has gained increased attention due to the controversy surrounding its effectiveness as a treatment approach for dysphagia. NMES has been used to retrain pharyngeal musculature, improve swallow function, and promote reorganization of the motor cortex. [91] Permsirivanich and colleagues (2009) conducted a randomized controlled trial comparing NMES to traditional therapy techniques. Both treatment approaches were found to positively impact swallow function, with NMES having slightly better outcomes.[92] Conversely, in Bulow's et al 2008 randomized study comparing NMES and traditional treatment, all subjects had significant improvements in swallow function, but the differences between the two treatment groups was not significant.[91] A study by Ludlow et al. (2006) found that the surface electrical stimulation used was either too weak or did not penetrate the mucosa deep enough to stimulate the muscles responsible for hyolaryngeal elevation. [93] Their findings suggest electrical stimulation could be used for patients with weakened musculature who are able to elevate the larynx during the swallow. However, for patients who were unable to elevate the larynx during the swallow, electrical stimulation was found to negatively impact laryngeal elevation, resulting in increased difficulty for airway protection and increased risk of aspiration. Leelamanit et al.(2002) found that surface electrical stimulation resulted in improved laryngeal elevation that may positively affect cricopharyngeal opening.[94] Given the inconsistencies in the literature, it is unclear whether neuromuscular electrical stimulation for the treatment of dysphagia is effective.

For stroke survivors, a reduction in lingual strength may have serious implications on swallowing function. According to Hewitt and colleagues (2008), decreased lingual muscle mass can negatively impact bolus propulsion into the pharynx.[95] As there is an increased emphasis on the diagnosis and treatment of lingual dysfunction, several devices are being used to evaluate tongue force and pressure output. These include the Iowa Oral Performance Instrument (IOPI), Tongue Force Measurement System (TOMS), Kay Elemetrics Swallowing Workstation Lingual Force Transducer, and the Madison Oral Strengthening Therapeutic device (MOST). A prospective cohort intervention study in acute stroke survivors, found that an isometric lingual exercise program utilizing the Iowa Oral Performance Instrument (IOPI) was effective in increasing isometric pressures for the anterior and posterior tongue, increasing maximum swallowing pressures, significantly decreasing overall residue, and improving penetration-aspiration ratings.[96]

Although positive results have been found in the use of sEMG, NMES, and isometric lingual exercise programs, continued research is needed to determine their effectiveness in stroke survivors. However, the positive results found in sEMG and isometric lingual exercise programs are promising. These interventions are likely to play a role in dysphagia therapy in combination with traditional therapy approaches for the rehabilitation of dysphagia.

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Table 1

Neural regions associated with swallowing function

Region	Hypothesized role	References
Primary Somatosensory, motor and Motor Supplementary cortices (BA 1, 2, 3, 4, and 6)	Cortical processing of swallowing, including motor regulation and execution and motor control.	(Hamdy *et al*. 1999a; Hamdy *et al*. 1999b) (Mosier & Bereznaya 2001) (Martin *et al*. 2001)
Anterior cingulate (BA 24 and 32)	Higher order motor processing: swallowing movement planning and execution.	(Hamdy *et al*. 1999a; Hamdy *et al*. 1999b)
	Cognitive perceptual processes such as attention and response selection.	(Martin *et al*. 2001) (Martin *et al*. 2004)
Orbitofrontal cortex (BA 10, 11, 12, 44, 45, and 47)	Unclear	(Mosier *et al*. 1999b)
Parieto-occipital cortex (BA 7, 17, 18, 40)	Sensory processing of swallowing.	(Hamdy *et al*. 1999a) (Kern *et al*. 2001)
	Task-cue processing, not swallowing per se.	(Toogood *et al*. 2005)
	Movement planning and execution.	(Mosier & Bereznaya 2001)
Temporopolar cortex (BA 22 and 38)	Unclear	(Mosier *et al*. 1999b)
Insular cortex	Processing of gustatory input. Intraoral sensory modulation.	(Daniels *et al*. 2006) (Daniels & Foundas 1997) (Mosier *et al*. 1999a; Mosier *et al*. 1999b)
Internal capsule	Functional connection of the cortical and brain stem nuclei via the corticobulbar tracts.	(Mosier *et al*. 1999a; Mosier *et al*. 1999b) Gonzalez-Fernandez, et.al, 2008)
Thalamus	Sensory and motor input processing via thalamocortical and thalamostriatal pathways.	(Daniels *et al*. 1998) (Mosier *et al*. 1999b)
Basal Ganglia (caudate and/or putamen)	Gating of Sensory Output.	(Mosier & Bereznaya 2001) (Daniels *et al*. 1998)(Suzuki *et al*. 2003)
Cerebral Peduncle	Descending pathways from the cortex.	Miller(Miller, 1982)
Brain Stem	Central pattern generator, swallowing regulation.	(Jean, 1972; Jean, 1984; Jean, 2001)
Cerebellum	Regulation of adaptive coordination, sequencing, timing, learning and memory of motion.	(Zald & Pardo 1999) (Mosier & Bereznaya 2001) (Suzuki *et al*. 2003)

Table 2

Dysphagia screening tests

Test	Gold standard for validation	Includes Clinical Evaluation	Includes Sensory testing	Water Trial protocol		Sensitivity	Specificity
Burke Dysphagia Screening Test	VFSS	Yes	No	3oz Water swallow		88	22
Standardized Swallowing Assessment	VFSS	Yes	No	**1**	5ml × 3	68	86
				2	Cup drinking		
Timed tests of Hinds and Wiles	Symptom Quest.	Yes	No	**1**	5-10 ml	73	67
				2	100-150 ml		
Bedside swallow assessment	VFSS/CE	Yes	No	**1**	5 ml × 3	70	66
				2	60 ml		
Toronto Bedside Swallowing Screening Test TOR-BSST©	VFSS	Yes	Yes	**1**	5ml swallo w × 10	91.3	66.7
				2	Cup sip		
Clinical examination	VFSS	Yes	No	N/A		92	67
Modified Mann Assessment of Swallowing Ability MMASA	MASA	Yes	No	N/A		93	86

Table 3

Strengths and limitations of VFSS and FEES

	Strengths	Limitations
Videofluoroscopy (VFSS)	• Direct visualization of oral pharungeal and esophageal structures in real time. • Structural movements and bolus flow can be directly evaluated. • Conpensatory stretegies can be evaluated and their effectiveness immediately determined. • Cricopharyngeal function (opening) can be directly visualized. • Anatomical structures can be directly evaluated.	• Radiation exposure • Limited space for patient positioning may be problematic for obese patients or those with contractures. • Required the use of oral contrast for visualization.
Videoendoscopy (FEES)	• Can be performed at the bedside with portable equipment • Does not require the use of contrast. Regular food can be used during the evaluation. • The larynx is directly visualized. • The presence, quantity and management of secretions can be directly evaluated.	• The oral and esophageal stages are not visualized • Bounce back of the light during the swallow (white out) impedes direct visualization of structures during swallowing limiting evaluation to immediately before and after. • Evaluation of bolus flow is limited by the lack of oral and esophageal information.

Table 4

Behavioral Treatment Approaches

Compensatory	Both	Rehabilitative
• Postural adjustments • Altering bolus characteristics (consistency, viscosity, volume of bolus, temperature, taste) • Increase volitional control	• Effortful swallow • Mendelsohn maneuver • Supraglottic Safety Swallow & Super Supraglottic Safety Swallow (breath hold) • Increase sensory input	• Tongue hold exercise • Shaker exercise (head-raise) • Lingual exercise/ resistance training • LSVT • EMST • NMES

LSVT, Lee Silverman Voice Therapy; EMST, Expiratory Muscle Strength Training; NMES, Neuromuscular Electrical Stimulation.

* From Gonzalez-Fernandez and Daniels Phys Med Rehabil Clin N Am19 (2008) 867–888

Table 5

Dysphagia Therapeutic Techniques

Treatment Technique	Effect on Swallow Function
Effortful Swallow	Improves base of tongue retraction during the swallow and improves clearance of the bolus from the vallecula (Speyer, 2012)
Mendelsohn Maneuver	Increases extent and duration of laryngeal elevation and thereby enhancing during and width of cricopharyngeal opening (Logemann, 2010)
Shaker Exercise (Head lift exercise)	Indicated for patients with reduced extent or duration of cricopharyngeal opening resulting in pyriform sinus residue (Speyer, 2012).
Supraglottic Safety Swallow	Breath hold closes vocal folds prior to swallow initiation and clears possible residue from the laryngeal vestibule (Speyer, 2012)
Super-Supraglottic Safety Swallow	Increase chance of true vocal fold and false vocal fold closure by bearing down (Speyer, 2012)
Tongue Hold Exercise	Improves contact between the base of tongue and posterior pharyngeal wall (Speyer, 2012)