Contents

composed of smooth muscle. The esophageal phase of swallowing involves a sequential peristaltic wave that propels food and liquid into the stomach. Normal transit time varies between 8 to 20 seconds (Dodds, Hogan, Reid, Stewart, & Arndorfer, 1973).

Variability in Swallowing

Although specific events for normal swallowing are described above there is considerable variability in the system associated with intrinsic (e.g., aging) and extrinsic (e.g., bolus volume) factors. The influences of specific factors on normal swallowing are summarized in Table 3–1.

Table 3–1. The Influence of Intrinsic and Extrinsic Variables on Swallowing in Healthy Adults

Increased Age

- Increased oral transit time (Cook et al., 1994; Shaw et al., 1995)
- Increased stage transition duration (Logemann et al., 2000; Robbins et al., 1992; Tracy et al., 1989)
- Increased volume required to evoke a pharyngeal swallow (Shaker et al., 1994)
- Increased airway invasion (Daggett, Logemann, Rademaker, & Pauloski, 2006; Daniels et al., 2004)
- Increased pharyngeal residue (Cook et al., 1994)
- Decreased isometric tongue pressure (Robbins, Levine, Wood, Roecker, & Luschei, 1995)
- Decreased onset of submental contraction (Ding, Logemann, Larson, & Rademaker, 2003)
- Decreased extent of hyoid movement (Kern et al., 1999; Logemann et al., 2000)
- Decreased pharyngeal and laryngeal sensation (Aviv et al., 1994)
- Reduced pharyngeal contraction (Tracy et al., 1989)
- Delayed onset of UES relaxation (Shaw et al., 1995)

Table 3–1. *continued*

- Increased intrabolus pressure (Kern et al., 1999; Shaw et al., 1995)
- Increased duration of UES opening (Rademaker, Pauloski, Colangelo, & Logemann, 1998; Robbins et al., 1992)
- Decreased diameter of UES opening (Logemann et al., 2000; Shaw et al., 1995; Tracy et al., 1989)
- Increased duration of swallowing apnea (Hiss, Treole, & Stuart, 2001)
- Increased incidence of inhalation after swallowing (Martin-Harris et al., 2005)

Increased Volume

- Bolus held more posteriorly in the oral cavity (Tracy et al., 1989)
- Decreased oral transit time (Rademaker et al., 1998; Tracy et al., 1989)
- Decreased stage transition duration (Rademaker et al., 1998)
- Earlier onset of palatal elevation (Dantas et al., 1990)
- Increased duration of velopharyngeal closure (Rademaker et al., 1998)
- Earlier onset of anterior tongue base movement (Dantas et al., 1990)
- Earlier onset of laryngeal elevation (Dantas et al., 1990)
- Increased extent of hyoid movement (Dodds et al., 1988; Logemann et al., 2000)
- Increased duration of laryngeal closure (Logemann et al., 1992)
- Increased intrabolus pressure (Jacob et al., 1989; Kern et al., 1999)
- Earlier onset of UES opening (Cook et al., 1989; Dantas et al., 1990)
- Increased diameter of UES opening (Dantas et al., 1990)
- Increased duration of UES opening (Dantas et al., 1990; Rademaker et al., 1998; Tracy et al., 1989)
- Earlier onset of swallowing apnea (Hiss et al., 2004)
- Increased duration of swallowing apnea (Hiss et al., 2001)

Increased Consistency

- Increased oral transit time (Dantas et al., 1990)
- Increased stage transition duration (Robbins et al., 1992)
- Increased oral pressure (Pouderoux & Kahrilas, 1995; Shaker, Cook, Dodds, & Hogan, 1988)
- Increased amplitude and duration of contraction of the inferior orbicularis oris, submental, and infrahyoid muscles (Ding et al., 2003)

continues

Table 3–1. *continued*

- Increased duration of velar excursion (Robbins et al., 1992)
- Increased laryngeal elevation (Shaker et al., 1990)
- Increased duration pharyngeal contraction (Dantas et al., 1990)
- Increased duration of UES opening (Dantas et al., 1990)
- Later onset of swallowing apnea (Hiss et al., 2004)

Taste

- Increased tongue pressure (Pelletier & Dhanaraj, 2006)
- Earlier onset of submental and infrahyoid contraction (Ding et al., 2003)
- Increased submental amplitude (Ding et al., 2003)
- Increased pharyngeal pressure (Palmer, McCulloch, Jaffe, & Neel, 2005)
- Reduced swallowing speed (volume swallowed per second) (Chee, Arshad, Singh, Mistry, & Hamdy, 2005)
- Increased number of swallows to drink 50 ml (Chee et al., 2005)

Sequential Swallowing (as compared to single swallows)

- Decreased oral transit time (Chi-Fishman & Sonies, 2000)
- Increased stage transition duration (Chi-Fishman & Sonies, 2000)
- Decreased duration of UES opening (Chi-Fishman & Sonies, 2000)
- Repetitive activation and partial deactivation of surface electromyographic waveform patterns (Chi-Fishman & Sonies, 2000)
- Reduced amplitude and velocity of hyoid movement (Chi-Fishman & Sonies, 2002)
- Two patterns of HLC movement: lowering of the HLC with the epiglottis returning to upright between swallows; partial HLC elevation with continued epiglottic inversion between swallows (Daniels et al., 2004; Daniels & Foundas, 2001)

Cued Swallows (as compared to noncued)

- Bolus held more posteriorly in oral cavity (Daniels, Schroeder, DeGeorge, Corey, & Rosenbek, 2007)
- Decreased oral transit time (Daniels et al., 2007)
- Decreased stage transition duration (Daniels et al., 2007)

HLC = hyolaryngeal complex; UES = upper esophageal sphincter.

15 Diagnosis of Dysphagia in Stroke

Establishing an accurate dysphagia diagnosis is a process of problem-solving through what is inferred from the clinical assessment and what is visualized on the instrumental evaluation. This information is then compared with normative data and the limited etiology-specific data available. The diagnosis of dysphagia frequently is initiated from a consideration of the symptoms presented, for example, postswallow residue. However, the thorough examination is incomplete if the clinician fails to identify the underlying physiologic basis of the dysfunction. Both components are of substantial importance. Symptoms of dysphagia more often are addressed through compensatory management, whereas the physiologic abnormality is targeted by direct rehabilitative exercise. Elucidation of one without the other leaves the dysphagic patient with an incomplete treatment plan and reduced potential for positive outcome. Table 15–1 describes dysphagic symptoms and their physiologic etiologies.

In this chapter, we present a format for diagnosis of oropharyngeal dysphagia by identifying symptoms and then determining the underlying basis of each symptom through an understanding of physiology. This methodical approach suits the nature of data available from many of our instrumental examinations. The signs or symptoms of dysphagia (e.g., preswallow pooling, postswallow residual) are more often static and thus, frequently easier to visualize. The clinician can observe these features over a longer period of time. In comparison, underlying physiologic abnormalities (e.g., hyoid movement, epiglottic deflection) are frequently dynamic and require a 'quick eye' to visualize. Thus, direct observation can be

Table 15–1. Differentiation Between Symptoms and Physiologic Abnormalities Underlying Dysphagia

Symptoms	***Physiologic Abnormalities***
• Inadequate bolus preparation • Anterior leakage • Postswallow oral residual • Premature spillage • Preswallow pharyngeal pooling to the level of the _______ • Inadequate epiglottic deflection* • Inadequate opening of the UES* • Postswallow vallecular residual • Postswallow piriform sinus residual • Aspiration • Penetration	• Oral motor impairment • Delayed pharyngeal swallow • Inadequate BOT to PPW approximation • Weakened pharyngeal contraction/poor stripping • Inadequate epiglottic to arytenoid deflection* • Inadequate hyolaryngeal excursion • Incomplete velopharyngeal closure • Impaired opening of the UES

*May be both symptom and physiologic abnormality; UES = upper esophageal sphincter; BOT = base of tongue; PPW = posterior pharyngeal wall.

more challenging. A structured approach to problem-solving will focus the clinician toward an accurate diagnosis without distraction from the dynamic array of diagnostic data. At the conclusion of the examination and based on the identified sign or symptom, the clinician should be able to present a diagnostic summary that reflects the sequence of problem-solving in the following format:

> "The patient presents *[which phase]* dysphagia characterized by *[symptoms]* secondary to *[physiologic abnormality]*."

Frequently a single physiologic abnormality can result in a number of observed signs and symptoms. Thus, written presentation of written data may be facilitated by structuring the diagnostic summary with the physiologic abnormality first.

"The patient presents *[which phase]* dysphagia characterized by *[physiologic abnormality]*, resulting in *[symptoms]*."

To facilitate understanding and clinical carryover, two table formats are presented. The first, Table 15–2 supports the approach that clinical problem-solving starts at the symptoms and works its way into the physiologic abnormality. The second, Table 15–3, may aid the clinician in clear documentation by presenting physiologic abnormalities followed by their consequent symptoms.

ORAL PHASE

Within the oral phase of swallowing, an array of symptoms can be visualized on both clinical and diagnostic examination. These are summarized in Table 15–3, and all are a consequence of the primary physiologic abnormality of **poor orolingual control**. Unfortunately, a more specific and objective definition of this physiologic abnormality is difficult with our current instrumentation and, thus, requires more subjective speculation. Poor orolingual control may feasibly be secondary to bilateral or hemiweakness, spasticity, or a discoordinated quality characteristic of apraxia. The term "apraxia of swallowing" has been applied to patients with "the inability to organize the front-to-back lingual and bolus movement normally characteristic of a swallow or . . . simply holding the bolus without initiating any oral activity" (Logemann, 1998, p. 83). Describing this disorder as "apraxia," however, implicates specific principles in the act of swallowing. It suggests that swallowing is learned, skilled movement and that the abnormal movement pattern observed is not attributable to sensory or elemental motor deficits. The similarities and differences of apraxia of swallowing with more traditional disturbances of the praxis system (limb apraxia, buccofacial apraxia, apraxia of speech) have previously been reviewed (Daniels, 2000).

Table 15–2. Swallowing Symptoms Associated with Specific Physiologic Abnormalities

The Symptoms of	***Occurring***	***Can be Secondary to***	***In Which Phase***
Anterior leakage	Preswallow	Poor orolingual control	Oral
Inadequate bolus preparation			
Inadequate bolus formation			
Oral residual	Postswallow		
Pharyngeal pooling to the level of ____	Preswallow	Delayed pharyngeal swallow	Pharyngeal
Nasal regurgitation	During the swallow	Poor pharyngeal motility	
Inadequate epiglottic deflection		Decreased anterior hyoid movement	
		Intrinsic structural changes in supportive tissue	
Vallecular residual	Postswallow	Decreased base of tongue to posterior pharyngeal wall approximation	
		Inadequate epiglottic deflection*	
Inadequate opening of the UES*	During the swallow	Decreased anterior hyoid movement*	
		Intrinsic structural functional changes in cricopharyngeus	

Piriform sinus residual	Postswallow	Inadequate opening of the UES*	Pharyngeal
Penetration	Preswallow	Pharyngeal pooling*	Oral
			Pharyngeal
	During the swallow		Oral
		Inadequate epiglottic deflection*	Pharyngeal
		Inadequate supraglottic shortening/laryngeal elevation*	
	Postswallow	Oral residual*	Oral
		Pharyngeal residual*	Pharyngeal
Aspiration	Preswallow	Pharyngeal pooling*	Oral
	During the swallow	Inadequate true vocal fold closure	Pharyngeal
	Postswallow	Oral residual*	Oral
		Pharyngeal residual*	Pharyngeal

*Occasionally a symptom will be caused by another symptom, which requires the clinician to problem-solve through to the initial presenting physiologic abnormality.

UES = upper esophageal sphincter.

Table 15–3. Physiologic Abnormalities with Their Consequent Symptoms

	The patient presents oral phase dysphagia characterized by poor oral lingual control resulting in:
Preswallow	• Anterior leakage • Inadequate bolus preparation • Premature spillage with pharyngeal pooling to the level of _____ • Inadequate mastication • Supraglottic penetration of preswallow pooling • Aspiration of preswallow pooling
During the swallow	• Supraglottic penetration of pooled material • Aspiration of pooled material
Postswallow	• Anterior leakage of postswallow oral residual • Postswallow oral residual • Postswallow pharyngeal pooling of oral residuals to the level of _____ • Supraglottic penetration of postswallow oral residual that pools into pharynx • Aspiration of postswallow oral residual that pools into pharynx
	The patient presents pharyngeal dysphagia characterized by delayed pharyngeal swallow resulting in:
Preswallow	• Pharyngeal pooling to the level of _____ • Supraglottic penetration of preswallow pooling • Aspiration of preswallow pooling
During the swallow	• Supraglottic penetration of pooled material • Aspiration of pooled material
Postswallow	• None
	The patient presents pharyngeal dysphagia characterized by inadequate anterior hyoid movement resulting in:
Preswallow	• None
During the swallow	• Decreased epiglottic deflection[1] • Decreased traction force for UES opening[2] • Supraglottic penetration
Postswallow	• Vallecular residual >piriform sinus as a secondary effect[1] • Piriform sinus residual >vallecular as a secondary effect[2]

Table 15–3. *continued*

Postswallow *continued*	• Supraglottic residual • Aspiration of supraglottic or pharyngeal residual

The patient presents pharyngeal dysphagia characterized by inadequate base of tongue to posterior pharyngeal wall resulting in:

Preswallow	• None
During the swallow	• Impaired bolus transport through proximal pharynx
Postswallow	• Postswallow vallecular residual (>piriform sinus residual) • Supraglottic penetration of residual • Aspiration of supraglottic or pharyngeal residual

The patient presents pharyngeal dysphagia characterized by impaired UES opening in the presence of substantial anterior hyoid movement resulting in (implies intrinsic cricopharyngeus abnormality or timing issue):

Preswallow	• None
During the swallow	• Impaired bolus transport through cricopharyngeus
Postswallow	• Postswallow piriform sinus residual (>vallecular residual) • Supraglottic penetration of residual • Aspiration of supraglottic or pharyngeal residual

The patient presents pharyngeal dysphagia characterized by poor pharyngeal motility resulting in:

Preswallow	• None
During the swallow	• Impaired bolus transport throughout the pharynx • Nasal redirection • Supraglottic penetration
Postswallow	• Diffuse (nonspecific) pharyngeal residual • Nasal residual • Supraglottic penetration of residual • Aspiration of supraglottic or pharyngeal residual

[1,2]The physiologic abnormality results in a symptom during the swallow that consequently results in another symptom postswallow.

UES = upper esophageal sphincter.

Regardless of the semantic or theoretical issues, however, there *is* an oral dysmotility disturbance that is characterized by repetitive, disorganized anterior-posterior bolus movement in the oral cavity, which prolongs oral transfer and is evident in stroke patients (Daniels et al., 1999; Robbins & Levine, 1988; Robbins et al., 1993).

Oral physiologic abnormality may also be disguised as, or exacerbated by, decreased attention. In these cases, the prolonged and inefficient oral phase of swallowing is not solely physiologically based but is complicated by cognitive factors. Augmentative instrumental assessments, such as oral manometry (e.g., Iowa Oral Pressure Instrument), or the more invasive intramuscular electromyography (EMG) may provide valuable information; however, these techniques are rarely incorporated into clinical practice. Normative data using these measures are not available, the availability is limited, and specific expertise may be required (in the case of intramuscular EMG), thus discouraging clinical application. Integration of cranial nerve findings will assist in differential diagnosis of oral inefficiency. A patient with no evidence of hypoglossal nerve damage on assessment but who demonstrates oral inefficiency during ingestion more likely may present with cognitive inattention as the primary etiology.

PHARYNGEAL PHASE

Preswallow Pooling

A common symptom of dysphagia in stroke is the presentation of **preswallow pooling** in the pharynx. Table 15–3 presents this symptom, as well as aspiration and penetration, which are produced by the sensory deficit of **delayed pharyngeal swallow**. At first glance, this appears rather straightforward; however, working from the symptoms of preswallow pooling to delayed pharyngeal swallow, in practice, is quite complicated.

The conclusion of oral parameters of swallowing is marked by volitional transfer of the prepared bolus into the oropharynx and, thereby, outside the reach of voluntary control. The transition between oral and pharyngeal components of the swallowing process is heavily influenced by the integrity of neurosensory response systems and subsequent timing of onset of the pharyngeal swallow in relation to voluntary transfer. The videofluoroscopic swallow study (VFSS) can provide an image of bolus transfer and swallowing onset marked by hyoid movement. However, VFSS cannot provide specific measures of sensory thresholds; for diagnosis of pharyngeal onset disorders, the clinician must infer sensory deficit based on biomechanical data. This is a difficult and perhaps imprecise task. Augmentative instrumental assessments are emerging for evaluation of sensory systems. Fiberoptic endoscopic evaluation of swallowing with sensory testing and cough reflex testing are two of these (see Chapters 9 and 12 for review). However, these procedures are more heavily focused on laryngeal, rather than pharyngeal, sensitivity. The clinical technique of assessing gag reflex is considered to be a direct evaluation of glossopharyngeal sensory integrity; however, as previously discussed, it lacks diagnostic sensitivity.

One complication in diagnosing delayed pharyngeal swallow arises from the fact that the primary symptom of this disorder, that of preswallow pharyngeal pooling, is shared by the physiologic abnormality of poor orolingual control. Differential diagnosis of these two disorders is difficult based on VFSS and has substantive clinical consequences. A misdiagnosis may result in the clinician providing a sensory-based treatment for a motor-based disorder, or vice versa, with consequent treatment failure. This represents a waste of health care resources, and frustration for both patient and clinician. Several observations, summarized in Table 15–4, may guide the clinician toward a physiologic diagnosis based on the symptom of preswallow pooling; however, none of these in isolation can be considered an undisputed feature of either diagnosis. There exists no peer-reviewed research to document the sensitivity and specificity

Table 15–4. Differential Diagnosis of the Etiology of Preswallow Pooling: Delayed Pharyngeal Swallow Versus Premature Spillage Due to Poor Orolingual Control

Clinical Question	*Poor Orolingual Bolus Control*	*Delayed Pharyngeal Swallow*
As the bolus approaches the oral cavity (preoral), what does the base of tongue do?	Does not approximate soft palate for protective glossopalatal seal	Arches to approximate soft palate for protective glossopalatal seal
How does the bolus enter the pharynx?	In noncohesive, unformed bits as it falls off of base of tongue during bolus preparation	As a cohesive, single bolus unless the patient volitionally segments the transfer
Is there a pronounced drop of the base and push of the blade of the tongue to transfer the bolus?	No	Yes, although there will be a significant temporal delay between this movement and onset of pharyngeal swallow
On which consistency is the pooling most pronounced?	Heavier consistencies, solids	Liquids

of these radiographic features, in part, because we lack reliable sensory data on which to validate the observations.

The second major complication inhibiting accurate assignment of a diagnosis of delayed pharyngeal swallow is the innate variability in temporal relationships in nonimpaired individuals and the associated flexibility provoked by consistency adaptation. Specific measures of swallowing onset include the temporal measures of stage transit duration (STD), which is measured from the point where the bolus head reaches the ramus of the mandible to the onset of maximum hyolaryngeal elevation. Strict interpretation of STD is discouraged as this may lead to overdiagnosis. As discussed in Chapter 3, recent research has indicated that the bolus may be inferior to the ramus of the mandible at onset of maximum hyolaryngeal elevation during sequential swallowing and single swallows in healthy adults (Chi-Fishman & Sonies, 2000; Daniels et al., 2004; Daniels & Foundas, 2001; Martin-Harris et al., 2007; Stephen et al., 2005). This indicates that hypopharyngeal bolus location at onset of the pharyngeal swallow cannot be interpreted as abnormal if all other physiologic components of swallowing are intact. That is, clinicians must understand that although onset of the pharyngeal swallow may occur deep in the pharynx, for this to be classified as "normal" swallowing, airway protection must be maintained and risk of pulmonary invasion must be consistently low.

Postswallow Residual

The pharyngeal swallow is signaled by the onset of hyolaryngeal excursion, particularly anterior movement of the hyoid, which plays an important role in pharyngeal dynamics as discussed in Chapter 3. As such, **inadequate anterior hyoid movement** is a common symptom of dysphagia in stroke and can lead to a cascade of pharyngeal events and symptoms, as outlined in Table 15–3. Of note is that the impaired anterior hyoid movement is the etiology of other impaired biomechanical events, which subsequently cause other observable symptoms. Working backward from the symptoms (see Table 15–2), the presentation of postswallow **vallecular residual** is a consequence of (1) **decreased epiglottic deflection**, thus "trapping" the bolus in the superior pharynx, or (2) **decreased base of tongue to posterior pharyngeal wall approximation** with resulting inadequate positive pressure to drive the bolus into the hypopharynx. VFSS is not the appropriate instrument to comment directly and objectively on base of tongue to posterior pharyngeal wall pressure generation; pharyngeal manometry would be the technique of choice. Thus, observation of epiglottic deflection and a diagnosis by exclusion is the more usual course of clinical problem-solving for determining the source of vallecular residual. If epiglottic deflection has failed, then the etiology of this biomechanical movement must consequently be questioned. Failure to deflect the epiglottis may be a consequence of either (1) intrinsic **tissue changes** in the cartilaginous tissue of the epiglottis as in irradiated patients or those with connective tissue disease, or (2) inadequate anterior hyoid movement, which fails to pull the base of the epiglottis anteriorly and shift the apex over the airway. Tissue characteristics cannot be directly evaluated with our clinical tools and in uncomplicated stroke are unlikely. Therefore, observation of hyoid movement is imperative for understanding the underlying basis of epiglottic deflection.

Decreased anterior hyoid movement also contributes indirectly to the symptom of postswallow **piriform sinus residual**. Again working backward from the symptom, if a patient presents with pyriform sinus residual greater than vallecular residual, this would typically signal an isolated impairment of upper esophageal sphincter (UES) opening. **Impaired UES opening** can logically be a consequence of (1) decreased anterior hyoid movement, (2) intrinsic **structural functional changes of cricopharyngeus muscle**, or (3) a **mistiming of biomechanical events** with neurophysiologic relaxation of the muscle. Of these three potential etiologies, VFSS is the technique of choice to visualize hyoid movement. Pharyngeal manometry with or without intramuscular EMG may be required to optimally evaluate the other two possible sources of piriform sinus residual. Manometry will aid in documentation of the relationships between pressure in the pharynx and cricopharyngeus and the amplitude of pressure drop in the cricopharyngeus. EMG will provide specific objective information about cricopharyngeal activation and deactivation.

Nasal Redirection

The presenting symptom of **nasal redirection** of the bolus is one of controversy as we lack substantive data to guide our practice. Invasion of the bolus into the nasal cavity is not simply an issue of impairment of velopharyngeal closure, but this symptom, more importantly, requires impairment of pressure systems that provide the driving force behind the bolus. Therefore, this is likely to be presented in cases of pharyngeal dysmotility, where pressure systems are disrupted or mistimed. Although VFSS reveals bolus flow patterns, more specific information about pharyngeal pressure systems would best be obtained through pharyngeal manometry. Using this instrumentation, the clinician may obtain objective measures of dysmotility patterns that underlie the symptom of nasal redirection.

Reduced Pharyngeal Motility

In the prior section, specific biomechanical characteristics that are subject to impairment in stroke and a method for problem-solving from symptom to specific physiologic etiology were discussed. In many individuals with stroke, the dysphagic presentation is much more extensive with multiple components of impairment with diffuse postswallow residual. The categorical term of **poor pharyngeal motility** may be applied when either all components of the process are collectively impaired or a specific etiology is not able to be identified. Poor motility may be a result of any number of neuromuscular or temporal deficits, characterized with terms such as weakness, spasticity, slowness, reduced pharyngeal shortening, or discoordination. Again, VFSS reveals bolus flow patterns and allows for assessment of timing measures; pharyngeal manometry would be required to provide specific objective measures of pressure systems or very detailed pressure sequences. Neuromuscular substrates such as weakness and spasticity are only presumed in our current practice due to inadequately developed clinical instrumentation.

Airway Invasion

Supraglottic penetration can be a symptom of any number of physiologic abnormalities and can occur preswallow from pooled material, postswallow from oral or pharyngeal residual, or during the swallow secondary to either impaired epiglottic deflection and pharyngeal/supraglottic shortening or overflow of pooled material that enters the airway as the larynx elevates. Aspiration, as well, can occur before or after the pharyngeal swallow, but only occurs during the swallow in the case of specific impairment of either the degree or timing of vocal fold closure. It is not an uncommon finding for stroke patients to present with supraglottic penetration during pharyngeal swallowing and then proceed to aspirate on postprandial glottic opening. VFSS and videoendoscopy will allow for detection of supraglottic penetration and aspiration. Videoendoscopy may more optimally visualize vocal fold closure and identify impairments of adduction. Multimodality assessment using more standard techniques paired with respiratory airflow will be required to evaluate swallowing respiratory coordination.

ORAL AND PHARYNGEAL DYSMOTILITY IN STROKE

Given this overview of diagnosing dysphagia, what can research and clinical observation teach us about swallowing following stroke? Any clinician who has worked with stroke patients knows there is no "prototypical" swallowing pattern in this population, aside perhaps from patients with lateral medullary syndrome (LMS). Research findings (Table 15–5) when integrated with clinical observations can, however, help focus clinicians on particular patterns of pathophysiology that may be evident following stroke.

To facilitate discussion, stroke is discussed in terms of supratentorial (cortical, subcortical) and brainstem lesions. Research has suggested that dysphagia following stroke primarily is secondary to large cortical lesions, for example, middle cerebral artery territory infarcts (Alberts et al., 1992; Robbins et al., 1993). Other studies, however, have demonstrated that changes in swallowing can occur with small subcortical lesions (Daniels & Foundas, 1999; Logemann et al., 1993) with some proposing that swallowing generally is functional with small subcortical lesions, albeit different from age-matched controls (Logemann et al., 1993). As no study has identified specific dysmotility patterns distinguishing swallowing between cortical and subcortical lesions, cortical and subcortical lesions are discussed under the umbrella term, supratentorial.

Dysphagia in Supratentorial Stroke

Oral dysmotility is a common problem following supratentorial stroke characterized by longer transfer and possibly discoordination in oral transfer. Stroke research has focused on defining oral stage impairment primarily by measuring oral transit time (OTT). This is defined as the time from onset of bolus movement to the point where the bolus head reaches the ramus of the mandible. OTT is increased for stroke patients as compared to healthy controls (Robbins & Levine, 1988; Robbins et al., 1993). "Apraxia of swallowing" also has been described in a subset of patients with left hemisphere damage (LHD) and has been characterized by a "lack of labial, lingual, and mandibular coordination" with OTT of over 10 seconds (Robbins et al., 1993, p. 1298).

Table 15–5. Research Detailing Dysphagia in Stroke Patients Using O

Authors	***Subjects***	***Time Post-Onset***	***Trials/Stimuli***	***Technique***
Butler et al. (2007)	26 stroke with dysphagia (11 aspirators, 15 nonaspirators) 20 healthy adults	N/A	Two trials: 5, 10 15, 20 ml thin and thick liquid	
Chen, Ott, Peele, & Gelfand (1990)	46 stroke	1 month	3 and 5 ml thin and thick liquid; 3 ml paste; ¼ cookie	
Daniels et al. (1999)	59 stroke	5 days	Two trials: 3, 5, 10, 20 ml; 1 tsp paste; ½ cookie	
Daniels & Foundas (1999)	54 stroke	5 days	Two trials: 3, 5, 10, 20 ml; 1 tsp paste; ½ cookie	
Daniels et al. (2006)	13 healthy adults 9 stroke	2 days 33 days	Two trials: 5 ml liquid	

muli	Techniques	Measures	Results
	Simultaneous VFSS and respiratory measure	Objective	– ↑ SAD and variability of duration in stroke patients as compared to controls – ↑ SAD in aspirators as compared to non-aspirators – ↑ in I-I respiratory pattern in aspirators and greater dysphagia severity
	VFSS	Subjective	– 39 oral and pharyngeal dysmotility – 5 isolated pharyngeal dysmotility – 2 isolated oral dysmotility – 18 mild dysphagia – 23 moderate dysphagia – 5 severe dysphagia – Dysmotility pattern not associated with hemisphere
	VFSS	Subjective and Objective	– Equal incidence of lingual discoordination in RHD and LHD
		Subjective and Objective	– Equal incidence of dysmotility patterns (subjectively measured) and aspiration between in LHD and RHD – 19 oral and pharyngeal dysmotility – 20 isolated pharyngeal dysmotility – 3 isolated oral dysmotility
	VFSS	Objective	– Dysphagia defined as dysfunction on 2 of 6 swallowing measures: OTT, STD, PTT, P-A Scale, vallecular residual, piriform sinus residual – 2 SD above normal means to determine dysfunction – 5 stroke patients presented with dysphagia acutely – 2 presented with continued dysphagia at 1 month.

continues

Table 15–5. *continued*

Authors	*Subjects*	*Time Post-Onset*	*Trials/Stimuli*	*Technique*
Irie & Lu (1995)	74 stroke	2 to 59 days	3 ml liquid and paste; mouthful liquid	
Leslie et al. (2002)	18 stroke patients with clinically determined dysphagia 50 healthy adults	4 to 28 days	5, 20 ml liquid; 5 ml pudding	
Logemann et al. (1993)	8 LHD (basal ganglia/internal capsule) 8 healthy adults	21 to 28 days	Two trials; 1, 3, 5, 10 ml liquid; 1 ml paste; ½ cookie	
Mann et al. (2000)	128 stroke	10 days	5, 10 ml thin liquid, thick liquid, paste; 20 ml thin liquid	
Nilsson, Ekberg, Bulow, & Hindfelt (1997)	33 neurologically impaired (including stroke) patients with clinically determined dysphagia	N/A	Mouthful thick liquid barium	

muli	Techniques	Measures	Results
	VFSS	Objective	– 33 oral and pharyngeal dysmotility – 8 isolated pharyngeal dysmotility – 24 isolated oral dysmotility – stroke isolated oral dysmotility in LHD – ↑ in both oral and pharyngeal dysfunction in RHD
	Simultaneous VFSS and respiratory measure	Objective	– ↑ inspiration after swallow in stroke group
	VFSS	Subjective and Objective	– ↑ OTT – ↓ OPSE* – ↓ PRT
	VFSS	Subjective and Objective, ordinal scale for severity; weighted median score determined dysphagia and severity	– 36 oral and pharyngeal dysmotility – 22 isolated pharyngeal dysmotility – 3 isolated oral dysmotility – 37 mild dysphagia – 39 moderate dysphagia – 6 severe dysphagia
	Simultaneous VFSS and respiratory measure	Objective	– Airway invasion associated with lower SSI** – Postswallow respiratory phase not associated with airway invasion

continues

Table 15–5. *continued*

Authors	*Subjects*	*Time Post-Onset*	*Trials/Stimuli*	*Technique*
Perlman et al. (1994)	330 (101 stroke)	N/A	N/A	
Robbins & Levine (1988)	8 LHD 8 RHD 8 healthy adults	3 wks	Two trials: 2 ml liquid and paste	
Robbins et al. (1993)	20 LHD 20 RHD 20 healthy adults	3 wks	Two trials: 2 ml liquid and paste	
Robbins et al. (1999)	15 multi-infarct 98 healthy adults	mean—146 days	Two trials: 3 ml liquid	
Selley et al. (1989b)	21 neurologically impaired patients with complaints of dysphagia (11 stroke)	N/A	5 ml liquid	
Smithard et al. (1997)	121 stroke (only 95 had VFSS	3 days 29 days	Thin and thick liquid	
Teasell et al. (2002)	20 medullary stroke (only 9 had VFSS) 8 healthy adults	4 to 77 days	Thin and thick liquid, pudding, solids	
Veis & Logemann (1985)	38	<1–4 months	Two trials: 1/3 tsp liquid and paste	

↑ = increase; ↓ = decrease; I-I = inspiration-inspiration, LHD = left hemisphere damage, N/A = not available, OPSE = oropharyngeal swallowing efficiency, OTT = oral transit time, P-A = penetration-aspiration, PRT = pharyngeal response time, PTT = pharyngeal transit time, RHD = right hemisphere damage, SAD = swallowing apnea duration, SSI = swallowing severity index, STD = stage transit duration, VFSS = videofluoroscopic swallow study.

muli	*Techniques*	*Measures*	*Results*
	VFSS	Subjective and Objective	– Deviant epiglottic inversion, delayed pharyngeal swallow, vallecular residue, hypopharyngeal residue, decreased hyoid elevation; Linear trend between incidence of aspiration and severity of postswallow residual and delayed pharyngeal swallow
	VFSS	Objective	– ↑ OTT and “apraxia of swallowing” in LHD – ↑ PTT and aspiration in RHD
	VFSS	Objective	– ↑ OTT and “apraxia of swallowing” in LHD – ↑ STD, PTT and aspiration in RHD
	VFSS	Objective	– ↑ in P-A scale scores – ↑ silent aspiration – ↑ within subject variability
	Nasal airflow	Subjective and Objective	– ↑ inspiration after swallow
	VFSS	Objective	– ↑ aspiration acute LHD/RHD – ↑ aspiration RHD at 1 month
	VFSS	Subjective and Objective	– Postswallow residual – Delayed pharyngeal swallow – Aspiration – Reduced epiglottic deflection – Reduced hyoid movement
	VFSS	Subjective and Objective	– Delayed onset of the pharyngeal swallow – Reduced pharyngeal peristalsis – Reduced lingual control

amage, N/A
time, P-A =
ransit time,
swallowing
study.

*OPSE—calculated by dividing the percentage of the bolus swallowed (minus percentage of oral residue, pharyngeal residue, and aspiration) by oral plus pharyngeal transit times.

**SSI—calculated by dividing SAD by PTT.

Daniels and colleagues (1999) describe lingual discoordination in patients with LHD as well as right hemisphere damage (RHD) ranging from durations of 1 to 3 seconds (mild), 4 to 10 seconds (moderate), or greater than 10 seconds (severe). Verbal cue to swallow has been reported to exacerbate this oral dysmotility pattern (Logemann, 1998; Robbins & Levine, 1988; Robbins et al., 1993) with resolution of oral dysfunction during the normal mealtime environment. Conversely, others note persistent oral dysmotility in the natural environment in patients with LHD as well as RHD (Daniels et al., 1999).

Although preswallow pooling is common following supratentorial stroke, research has not attempted to identify if the etiology of the pooling is more related to oral dysmotility or delayed evocation of the pharyngeal swallow. As noted previously, oral dysmotility yielding preswallow pooling is prominent following stroke. Stroke patients, however, frequently demonstrate increased STD (Daniels et al., 1996; Robbins & Levine, 1988; Robbins et al., 1993), which yields pharyngeal pooling. Increased STD has been identified as an independent predictor of aspiration, with increased delay associated with an increasing likelihood of aspiration (Perlman, Booth, & Grayhack, 1994). In this study, STD was rated on a Likert scale from 1 (STD between 1 and 2 seconds) to 3 (STD >5 seconds). Although this study consisted of a heterogeneous population, one-third of the patients had a stroke etiology.

The study of bolus flow has been the primary focus in stroke research; however, pharyngeal biomechanics can be impaired. No study has compared objective temporal and spatial structural measures in patients with supratentorial strokes and age-matched healthy participants. Only one study has detailed pharyngeal biomechanical events in a homogeneous cohort which included stroke patients (Perlman et al., 1994); however, the measure of these events was qualitative more than quantitative. This study, however, focused on the relationship between swallowing biomechanics and aspiration.

Dichotomous yes/no scores were used to define abnormal structural movement, whereas depth of postswallow residual was measured on a scale of 1 (mild) to 3 (severe). Abnormal epiglottic inversion and reduced hyoid elevation as well as bolus flow measures of vallecular residual and diffuse hypopharyngeal residual were strongly related to aspiration. As with STD, as severity of the residue increased, the number of patients who aspirated increased. As discussed earlier, reduced extent of structural movement can lead to postswallow residual. Patients with supratentorial stroke may present with unilateral pharyngeal hemiparesis, which yields postswallow residual on the contralesional side of the pharynx.

Increased airway invasion (laryngeal penetration and aspiration) has been documented in patients with supratentorial stroke (Alberts et al., 1992; Mann et al., 2000; Robbins et al., 1993). When using the Penetration-Aspiration Scale to rate airway invasion, stroke patients generally have higher scores as compared to healthy controls (Robbins et al., 1999). Aspiration in stroke patients, particularly those with supratentorial stroke, may not be hallmarked by a cough or voice change and is frequently termed as "silent." In a study of consecutive acute stroke patients, aspiration was identified in 38% of the patients, with 33% of these patients aspirating overtly, that is, coughing, and 67% aspirating silently (Daniels et al., 1998). Although lesion location (supratentorial or brainstem) was not specified, the increased incidence of inspiration after swallowing in stroke patients (Leslie et al., 2002; Selley et al., 1989b) has been shown to be associated with increased aspiration in this population (Butler et al., 2007).

Although stroke can impact all phases of swallowing, the clinician must also consider the impact of reduced cognition on swallowing. Cognitive deficits, particularly neglect, have been correlated with dysphagia. Hemispatial inattention has been associated with increased nonoral intake in acute stroke patients (Schroeder et al., 2006). Reduced awareness of dysphagia results in lack of self-modification of swallowing behavior and increased medical complications as compared to patients who are aware of dysphagia symptoms (Parker et al., 2004). Moreover, rehabilitation of swallowing is longer in stroke patients with neglect (Neumann, 1993).

Although hemispatial inattention and other cognitive disorders are not totally lateralized, they generally occur more frequently in patients with RHD as compared to patients with LHD (Heilman et al., 2003). These cognitive deficits may yield greater functional impairment in patients with RHD even though swallowing pathophysiology may be similar in patients with RHD and LHD. Greater functional impairment may lead the clinician to impose greater restrictions on oral intake for patients with RHD. Thus, in addition to rehabilitating swallowing, it is critical that clinicians also address cognitive deficits in treatment.

Dysphagia in Brainstem Stroke

Research is limited concerning swallowing in brainstem stroke. Studies are generally limited to single case reports that have outlined the progression of swallowing recovery in patients with brainstem stroke (Logemann & Kahrilas, 1990; Martino, Terrault, Ezerzer, Mikulis, & Diamant, 2001; Robbins & Levine, 1993) or focused on aspiration in case series (Kim, Chung, Lee, & Robbins, 2000; Teasell et al., 2002). Clinicians, however, are probably aware of the patients with LMS presenting with classic features. These are the patients who in the acute stage are expectorating saliva into a container due to inability to swallow. The clinical swallowing examination generally is characteristic of intact cognition and language and the presence of dysphonia and dysarthria. Unilateral true vocal fold paresis is not uncommon in patients with LMS. Patients are fully aware of swallowing deficits with intact sensation and immediate coughing with attempts to swallow the smallest of volume.

The preoral and oral stages of swallowing generally are intact. Although attempts at evocation of the pharyngeal swallow are present (on the clinical swallowing evaluation, the clinician may palpate multiple lingual hyolaryngeal gestures), the pharyngeal swallow is frequently never evoked or, if evoked, it is significantly delayed with limited extent of superior and anterior hyolaryngeal movement and UES opening (Logemann, Kahrilas, Kobara, & Vakil, 1989; Martino et al., 2001; Teasell et al., 2002). Unilateral pharyngeal hemiparesis is not uncommon in patients with LMS and is characterized by post-swallow residual on one side of the pharynx (Logemann & Kahrilas, 1990; Logemann et al., 1989). Airway invasion may be evident before, during, or after the pharyngeal swallow. Although swallowing is severely impaired in patients with LMS and recovery frequently is slow, they make the ideal client for swallowing rehabilitation due to intact sensation, cognition, and motivation.

Patients with brainstem stroke not involving the lateral medulla or with pontine stroke also may present with dysphagia, but characteristics are not as circumscribed as those with LMS. Given the close proximity to the medullary swallowing center and the multiple neural networks involved with swallowing, these patients warrant swallowing evaluation.

SUMMARY

The analysis of swallowing biomechanics and physiology is a complex process of integrating what is known of normal swallowing processes, paired with amalgamation of both subjective and objective evaluation of instrumental and clinical data. Given the complexity of this task and the substantive consequences of inaccurate or incomplete diagnosis, the astute clinician will develop a methodical approach for problem-solving relying on the easily observable symptoms to lead to the physiologic source of the impairment.