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Management of Dysphagia in Acquired and Progressive Neurologic Conditions

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Abstract

As the act of deglutition involves much of the central and peripheral nervous systems, neurologic disease can affect swallowing behaviors ranging from mild to profound in severity. The key in working with neurogenic dysphagia is to have a solid foundation in normal swallowing processes, including neural control. Within this framework, then, understanding how the neurologic condition affects neural control will guide hypothesis-based assessment and evidence-based treatment. The purpose of this article is to provide an overview of evaluation and treatment of neurogenic dysphagia in adult populations as well to propose assessment of co-occurring speech, language, and airway compromise. Furthermore, it is vital to be familiar with ethical decision making and end-of-life issues. Continuing education in the form of research articles, conferences, and professional discussion boards is useful in maintaining a high level of service delivery. Whenever possible, an inter- or transdisciplinary approach is recommended.

Keywords

dysphagia; neurologic disease; parkinsonism; multiple sclerosis; motor neuron disease

Deglutition is defined as the entire act of eating, including food recognition and procurement into the mouth, oral preparation, oropharyngeal swallowing, and transit through the esophagus and into the stomach. This entire act is a series of interconnected and complex behaviors, requiring intact cognition, reward/drive processing, sensory processing, sensorimotor control, airway protection, and autonomic function.¹ In other words, the

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Conflict Of Interest

None declared.

complex act of deglutition uses much of the central and peripheral nervous system for safe and efficient ingestion of the bolus and saliva management. It is not a surprise, then, that conditions that are neurologic in nature can profoundly disrupt swallowing function. As such, the speech–language pathologist treating neurogenic dysphagia must have a robust understanding of the neural control of deglutition.² Furthermore, the competent clinician must understand the etiology of the neurogenic condition to form hypothesis-driven evaluation methods and a treatment plan based on underlying impairment.

Before delving into evaluation in treatment, we will provide a brief overview of different etiologies that can cause dysphagia. It is beyond the scope of this article to comprehensively cover every condition. For a deeper dive into the neural basis of deglutition and how neurologic disorders affect deglutition, please see studies of Miller and Mistry and Hamdy.^{3,4} Acquired neurologic conditions have many different etiologies, including, but not limited to, traumatic, autoimmune, neoplastic, toxic, metabolic, infectious, genetic, and idiopathic, although the majority are idiopathic. In addition to understanding the etiology, it is also important to consider the nature of the disorder in terms of onset, course (likelihood of spontaneous recovery or further degeneration), and site of lesion(s), keeping in mind that some diseases affect multiple levels of the central and peripheral nervous systems, as this will guide your approach to the patient. Further, most patients are on several medications related to their condition or other medical comorbidities. They may also have had functional neurosurgery. As such, it is vital to have a complete medical history on your patient.

Terminology within the field of neurology, and certain disease, can be, classified into several subcategories. For example, Parkinson’s disease (PD) is a gradual-onset neurodegenerative disease, but is also considered a movement disorder, whereas multiple sclerosis is a degenerative disease with a remitting/relapsing course that is also considered a demyelinating disease. The following are a series of questions to guide assessment of the condition, and common neurologic disorders in terms of these parameters are given in Table 1.

Onset: Is the onset acute or gradual?

Course: Is the course stable, relapsing/remitting, progressive, or is spontaneous or treatment-based recovery expected?

Site of lesion(s): Is this disorder/disease affecting muscle, neuromuscular junction, lower motoneuron, upper motoneuron, sensory end organs, primary/secondary/tertiary sensory pathways, brainstem nuclei, subcortical structures (basal ganglia, thalamus, cerebellum), cerebral pathways, or cortical areas?

A further consideration is that our understanding of neurologic disease is constantly evolving. We have advanced imaging capabilities as well as a deeper understanding of genetics, epigenetics, and molecular biology. This has caused a shift in not only our theoretical framework but our approach to treating these conditions.

EVALUATION

Depending on the disease, swallowing function will likely need to be evaluated more than once, and possibly at regular intervals. The onset and course of the disease will determine this, and it may change in progressive diseases as severity increases. Evaluation tools for swallowing include clinical evaluation, questionnaires and/or scales, imaging, and high-resolution manometry (HRM).

The components of a clinical evaluation are similar for patients with acquired neurologic disease as for other patient populations. Typically, this will include a cranial nerve exam, voluntary swallows, test trials of ice or water, and voluntary coughing. Outcomes of these tests include integrity of the oral motor mechanism, ability to volitionally swallow and cough, and/or throat clears, coughing, or change in vocal quality (to wet/gurgle) post swallow. However, there are some special considerations that should be included when performing the clinical or bedside evaluation in these patients. For example, patients with neurological disease may exhibit oral apraxia, and thus voluntary tasks may be impaired or the patient may be completely unable to complete them. However, reflexive behaviors may be intact.

The use of quantitative data regarding cough strength may also be useful as part of the clinical exam. Research on the relationship between cough and swallow has shown that reduced peak cough airflows may indicate increased risk for dysphagia in some patient groups. Peak cough airflows vary according to how many coughs are produced and the instructions provided. Two common methods to elicit voluntary cough are to ask the patient to “cough one time as hard as possible,” and to ask the patient to cough “as if something went down the wrong pipe, multiple times in a row.” The single, hard cough will have higher peak airflow rates than multiple coughs. Collectively, much of the research relating to cough and swallow has shown that single hard cough airflow rates less than approximately 4.5 to 6 L/s (270–360 L/min) are associated with reduced swallow safety and dysphagia.^{5–7} The research on sequential cough is less robust; however, Hegland and colleagues showed that, in PD, people without penetration or aspiration during swallowing had a more organized cough in terms of peak airflows and durations compared with those who did exhibit penetration or aspiration during swallowing.⁸ While the measurement of peak cough airflow for a single voluntary cough can be achieved with relatively inexpensive equipment, namely, any commercially available peak airflow meter, the measurement of sequential cough requires more expensive and cumbersome spirometry equipment that may not be readily available to most speech–language clinicians.

There is a growing body of literature examining the use of reflexive, or induced, cough to identify the presence of penetration, aspiration, and swallowing dysfunction. To assess induced cough, it is necessary to expose the patient to a tussigenic (cough-inducing) stimulus and record the response. There are several tussigenic stimuli that have been studied, including citric acid, L-tartaric acid, capsaicin, and distilled water. Outcome measures for induced cough are the same as voluntary cough in terms of airflow. Other metrics include whether cough was produced, number of coughs produced to the stimulus, and the perceived urge to cough (UtC) associated with exposure to the stimulus, regardless of whether cough

was actually produced. The European Respiratory Society (ERS) recommends the following convention in terms of coding patients as responders or nonresponders: produce at least two sequential coughs in two-thirds of trials of induced cough testing.⁹ If a patient does not produce this at a minimum, he/she would be considered a nonresponder to that level (concentration) of that particular stimulus. Hegland et al found that patients with PD who were nonresponders to aerosolized distilled water (FOG) were more likely to exhibit deep laryngeal penetration or aspiration during swallowing.¹⁰

The practicalities of induced cough testing limit its use to primarily research-based protocols to date; however, there are some ways speech–language clinicians can implement components of induced cough testing into their practice. One very simple and inexpensive tool is the modified Borg scale for measuring UtC (Table 2).¹¹ The UtC is a respiratory sensation associated with exposure to a tussigenic stimulus, whether that stimulus is one of the above-mentioned substances, or aspirate material. A median of 5 to 6 on the Borg scale is expected when a cough response is present to a given stimulus.¹² In our practice, we utilize the modified Borg scale during modified barium swallow (MBS) evaluations and query patients when penetration or aspiration is observed, regardless of whether they respond to the stimulus. Oftentimes when they do not respond they will report a slight UtC (2–3 on the Borg scale; typically described as a “tickle”) and this provides scaffolding with which to counsel the patient on the importance of cough and responding to that sensation to clear the material from the airway. We have learned that in many cases, patients still detect the aspirate material, but simply do not realize that it is something to which they should respond.

The use of questionnaires or scales to evaluate dysphagia can be tricky in patients with acquired neurologic conditions. This is because, in many of these diseases, there is the potential impact to the sensory system and/or sensory perception, and consequently they may not self-report symptoms of disordered swallowing even when they exist. Another possible explanation is that, particularly in very slowly progressing diseases, changes in swallowing function may occur very gradually over time, and patients simply may not notice. For example, in PD there is a degradation to sensory perception of airway stimuli and slow progression of swallowing problems, leading many of these patients to under-report symptoms of dysphagia. The response to penetrant or aspirate material may be blunted or absent.¹³ Combined, lack of patient-reported signs of dysphagia and lack of response to aspirate material may lead the clinician to think swallowing function is not impaired where it actually is. Thus, imaging is critically important to evaluate swallowing in these patients.

The gold standard for evaluating swallowing in patients with acquired neurological diseases is the use of imaging. Because many patients will have oral phase deficits, the MBS evaluation may be preferable over flexible endoscopic evaluation of swallowing (FEES). However, both can yield useful information. Validated scales, such as the penetration-aspiration scale (PAS), the dynamic imaging grade of swallowing toxicity (DIGEST), and the Modified Barium Swallow Impairment Profile (MBSImP) can be used to characterize the evaluation.^{14–16} While the PAS can be used with FEES, to date the DIGEST and MBSImP have been validated only for MBS. The PAS is a functional measure of swallow safety, and provides information about whether bolus material entered the larynx or lower airway, and

also whether or not any residue was present in the larynx or lower airway, and whether it was ejected in the case of aspiration.¹⁴ The DIGEST also includes a swallow safety component using the PAS and adds swallow efficiency information by categorizing whether and how much pharyngeal residue was present for different bolus types. To date, the DIGEST has been validated in head and neck cancer patients; however, efforts are underway to validate the tool in neurological diseases, and clinically it is being used to characterize safety and efficiency of the swallow. The MBSImP is a 17-item tool that grades different facets of the swallow on Likert scales ranging from 0 to 2, 3, or 4. The summation of the 17 items yields an MBS composite score, and this can be segregated into oral, pharyngeal, and esophageal composites to understand where the patient's swallow is most impacted. In most settings, however, clinicians develop their own metrics tailored toward the patients they regularly evaluate. We acknowledge that validated scales can be time consuming but recommend their use whenever possible to standardize procedures.

HRM is the measurement of pressure changes associated with muscular force and geometric reconfiguration in the pharynx and esophagus. The procedure, minimally, involves transnasal passage of a catheter through the nose, past the upper esophageal sphincter and into the stomach, followed by measurement of timing and pressure events during saliva swallows, bolus trials, and compensatory techniques. The catheter contains sensors that are spaced at close intervals (2 cm or fewer), allowing for a high degree of spatial resolution. Because data are captured at a nearly continuous rate, simultaneously high temporal resolution is also possible. This is relevant to swallow function, as pressure changes in the pharynx and in the bolus are the primary drivers of bolus transit from the oropharynx to the esophagus. As a result, HRM has begun to be implemented by speech–language pathologists and dysphagia clinicians.¹⁷ The primary advantage of HRM over methods of swallow assessment that rely upon visual interpretation by the speech–language pathologist (i.e., FEES and MBS) is that it provides objective numeric data regarding spatiotemporal pressure differentials in the pharynx. HRM can also be combined with simultaneous impedance testing, allowing clinicians to determine bolus position relative to pressure changes and to subsequently calculate bolus-related measurements of pressure and time.^{18,19}

HRM has been shown to be useful in identification and characterization of dysphagia of multiple etiologies, including neurogenic processes.^{20–27} Jones et al demonstrated that several measures obtained from HRM were capable of distinguishing between individuals with mid-stage PD and healthy age- and sex-matched controls.²¹ Use of HRM in patients with varying sites of stroke has demonstrated a high degree of correlation between swallowing kinematic measures obtained via MBS and outcomes related to the presence of pharyngeal bolus residue and tracheal aspiration.^{23,24} In the light of the significant motor variability present in many populations with neurologic diagnoses, the large volume of finely detailed data obtained from HRM represents a method for detecting and describing even subtle aspects of pathologic change to swallow function, which may go unnoticed by other instrumented assessments early in the disease process.²²

Changes to swallow function will vary according to disease but broadly may reflect weakness, spasticity, rigidity, hypo/hyper-kinesias, and/or ataxia/discoordination. If apraxia is present, this would affect the voluntary oral phase of swallow, but once initiated the

reflexive pharyngeal phase will likely be relatively preserved. To date, dysphagia in neurological patients is not conventionally described as “spastic,” “flaccid,” “hypokinetic,” etc., as is the case with motor speech deficits present in many of the neurologic diseases; however, the prudent clinician should be aware of the underlying pathology as it relates to the swallowing problems identified. Table 2 identifies the underlying motor pathology in each of the disease/conditions. This knowledge should help shape the treatment plan.

TREATMENT

As we have outlined previously, treatment is based on findings from the comprehensive evaluation. This includes findings from the history and physical examination, clinical swallowing examination, and instrumented evaluations, including airway assessment. Treatment is then based on the underlying impairment to physiology. The goal of treatment is to provide the most safe and effective oral intake while maintaining nutrition and hydration needs and maximizing quality of life. There is no “one-size-fits-all” treatment for any of these neurologic conditions that we have described as patients have individual needs. Thus, the patient’s age, physical status, cognitive status, social status including living situation, vocation, support system, and of course wishes must be considered in the treatment plan.

Treatment is designed to rehabilitate or compensate for the physiologic deficits. This can be done with several approaches. Compensatory maneuvers are designed to compensate for impairments in physiology. These maneuvers should be chosen based on the specific impairment in physiology and should be verified with instrumented evaluation whenever possible. Diet changes are frequently recommended but must be undertaken only when indicated by clinical/instrumented evaluation and the consequences on nutrition, hydration, and quality of life have been addressed. Common approaches are as follows:

- Chin down position—This places the larynx in a more anterior and thus “protected” position and shortens the pharynx.
- Head turn—Closes off the lateral channel on the weakened side and directs the bolus.
- Mendelsohn maneuver—Holds the larynx in a more elevated position during the pharyngeal swallow.
- Supraglottic swallow/Intentional breath hold—Closes the airway before and immediately after the swallow with an opportunity to clear residue.
- Exercise—Strengthening, coordination, biofeedback, expiratory muscle strength training, neuromuscular electrical stimulation.
- Diet modification—Thickening liquids, soft solid foods, pureed foods, etc. Long-term diet modification (in particular, thickening liquids) should be used sparingly and the effectiveness of the recommendation should be verified on imaging. Please refer to the new International Dysphagia Diet Standardisation Initiative: <https://iddsi.org/>.

- Surgical—In specific cases, surgical intervention may alleviate signs of dysphagia. In patients with acquired neurological condition, often injection of botulinum toxin (Botox) or cricopharyngeal myotomy is performed to facilitate compliance of the upper esophageal sphincter.

Drooling management.

Drooling can occur because of sensory or motor compromise or as a side effect of medications. Behavior management is indicated in some conditions. Botox of salivary glands can also be indicated in some circumstances. In certain populations, medications such as atropine and scopolamine are prescribed to reduce saliva production. These medications often have at least some degree of neuromodulatory effect, and thus the swallow clinician must be aware of the side-effects of such medications, and how they might interact with the neurodegenerative disease process.²⁸ Others, such as glycopyrrrolate seem to have a more limited impact on cognitive function.²⁹

For patients with a grossly stable neurogenic dysphagia, or for those who are expected to have a return to normal or near-normal function, follow-up assessment is determined primarily by goal status. For example, an individual who is 1 year post-stroke might be safe and efficient in taking a mechanical soft diet with thin liquids and use of compensatory strategies. While further liberalization of diet might be possible, patients may state that they are comfortable with their current status and have no wish to proceed with further intervention. Conversely, the patient may have a different perspective at some point in the future, at which point a new evaluation would occur and new goals jointly developed.

In cases of most progressive neurologic conditions, regularly scheduled multidisciplinary follow-up visits are the standard of care.^{30,31} Speech–language pathologists are often integral members of such multidisciplinary clinic teams and through such settings can monitor patient progress. In both multidisciplinary clinics and in medically segregated clinical settings, it is essential that the speech–language pathologist maintain open lines of communication with the coordinating members of the medical team. In doing so, they will provide insight into expected changes in dysphagia status relative to primary disease progression and to the specific needs of individual patients, regardless of whether the patient is seeing the speech–language pathologist in a formal capacity.

As discussed earlier, treatment of dysphagia must be tailored to each individual, particularly in light of the fact that dysphagia in patients with neurologic conditions has a significant negative impact on quality of life.^{32,33} Further, this tailoring will likely need to be modified over the course of months and years as disease processes progress. For example, goals may shift from increasing safety of PO intake with solids to maintain adequate nutrition to caregiver training to provide the patient with safest-possible liquid boluses as a comfort-care measure. In each situation, it is of paramount importance that the clinician considers the underlying pathophysiology of the disease process/progression and its dysphagic manifestation, current best evidence for intervention, and the individual needs and goals of the patient and their caregivers.

OTHER CONSIDERATIONS

Speech, Language, Cognition

Speech, language, cognition, and swallowing share many neural substrates impacted by neurological disease. Further, speech and swallow share peripheral anatomic substrates as well. This means that speech, language, and/or cognitive problems have a high likelihood of coexisting with dysphagia in patients with neurological disease. A comprehensive evaluation should include at a minimum a screen of these behaviors and referral to a clinician who will also manage these deficits. This may be you, as the primary swallowing clinician, or another SLP. Table 2 summarizes the presence and type of speech, language, and/or cognitive changes in the disease.

Speech.—Patients with acquired neurologic disease are likely to exhibit dysarthria at some point in their disease process. Dysarthria can be categorized as flaccid (lower motor neuron involvement), spastic (bilateral upper motor neuron involvement), hyperkinetic or hyperkinetic (basal ganglia involvement), ataxic (cerebellar involvement), or mixed. As well, apraxia of speech may be appreciated. Evaluation of motor speech function should yield a characterization of the motor speech disorder (i.e., type of dysarthria, or apraxia of speech) and severity. While delving into the various tools available to assess motor speech disorders is beyond the scope of this article, we direct the reader to the study of Duffy for comprehensive review of motor speech disorders and their evaluation and treatment.³⁴ Detailed information regarding the hierarchy of neural players involved in motor speech disorders can be gleaned from van der Merwe.³⁵

Language and cognition.—Due to wide-spread involvement of various neural structure, patients with acquired neurologic diseases will often exhibit language or cognitive deficits, particularly later in the disease process. When present, cognitive and/or language disorders can have an effect on the patient's ability to verbally communicate and/or understand spoken or written language. This is an important consideration during evaluation in terms of knowing whether your patient can understand instructions or participate in various types of assessment and/or treatment. If cognitive-linguistic disorders are present, the patient's primary caregiver may need to facilitate any home-based treatment recommendations if the patient is not able to understand or remember the treatment tasks.

Ethical Considerations

Due to the progressive nature of most neurological diseases, it is important to communicate with patients and family members about the expected course of their disease, and how swallowing function may decline over time. Early conversations about nonoral feeding methods will provide the patient and family/caregiver necessary education about the prospect of tube-feeding and allow them to make informed decisions about what they want as their disease progresses. In some cases, the decision to place a feeding tube surgically will be influenced by other factors, notably the patient's respiratory status. In ALS, for example, the surgical placement of a feeding tube incurs additional safety risk as the patient's respiratory status declines. Thus, it may be prudent to place the tube before it is necessary

from a swallow perspective to do it at a time when the patient's respiratory function is still relatively preserved.

Other ethical considerations relate to the patient and caregiver perspectives on intervention and diet modification. Because most acquired neurologic diseases affect all body systems, they may be engaged in multiple types of therapy, doctor visits, support groups, etc. As well, because the demographic of patients with acquired neurologic diseases tends to be older, they may have an aging spouse or partner who also has a condition or conditions that they are managing. A team approach to management allows for communication across professions and for the selection of treatment targets based on the relative severity of functional deficits, along with patient and caregiver priorities. With progressive diseases, the goals of treatment may relate more to maintenance or preservation of function rather than improvement and, therefore, may be initiated before moderate or severe functional deficits are appreciated. Clear communication with the patient and caregiver about this approach is important for “buyin” and adherent participation in treatment.

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Learning Outcomes:

As a result of this activity, the reader will be able to (1) select appropriate evaluation tools for dysphagia in patients with acquired neurologic disease; (2) identify the onset, course, and site of lesion for discussed neurologic diseases; and (3) discuss how the course of the disease will impact evaluation and management of dysphagia in acquired neurologic disease.

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Table 1
A Summary of Common Neurologic Conditions that may Affect Swallow Function

Disease/Condition	Onset	Course	Site of lesion	Other considerations
Muscular dystrophy	Varied	Progressive	Muscle	Multiple forms
Myasthenia gravis	Often sudden	Varied; can sometimes resolve	Neuromuscular junction	Exercise generally contraindicated
Guillain-Barre	Sudden	Resolving over weeks to years	Lower motoneuron	Often worsens for days to weeks after onset prior to improving
Primary lateral sclerosis	Gradual	Progressive	Upper motoneuron	Bulbar involvement rare until later stages of disease process
Multiple sclerosis	Varied	Relapsing/Remitting; often progressive	CNS, PNS, mixed upper and lower motoneuron	Demyelinating-widespread depending on subtype
PD	Gradual	Progressive	CNS: widespread pathology	Changes in cognitive status, disease progression, and medication dosage can have a significant impact on swallow function
Atypical parkinsonism (corticobasal degeneration, multiple systems atrophy, progressive supranuclear palsy)	Gradual	Progressive	CNS: widespread pathology	More rapidly progressive than PD, swallowing may become impaired earlier in the disease process than PD
ALS	Gradual	Progressive	CNS/PNS; motor neurons; widespread pathology	Bulbar onset often rapidly progressive

Abbreviations: ALS, amyotrophic lateral sclerosis; CNS, central nervous system; PD, Parkinson's disease; PNS, peripheral nervous system.

Summary of Underlying Motor Pathology, Speech, Cognitive, and/or Language Involvement in Selected Acquired Neurological Diseases

Table 2

Disease/Condition	Primary motor problem	Presence of motor speech disorder	Presence of cognitive or language disorder
Muscular dystrophy	Weakness	Yes—flaccid dysarthria	No
Myasthenia gravis	Weakness	Yes—flaccid dysarthria	No
Guillain-Barre	Weakness	Yes—flaccid dysarthria	No
Primary lateral sclerosis	Spasticity and weakness	Yes—spastic dysarthria	No
Multiple sclerosis	Spasticity, weakness, or ataxia	Yes—spastic, flaccid, ataxic, or mixed dysarthria	Possible cognitive involvement late in disease process
Parkinson's disease	Hypokinesia	Yes—hyperkinetic dysarthria	Possible cognitive involvement late in disease process
Atypical parkinsonism (corticobasal degeneration, multiple systems atrophy, progressive supranuclear palsy) Amyotrophic lateral sclerosis	Hypokinesia, spasticity, ataxia, or apraxia	Yes—spastic, flaccid, ataxic, or mixed dysarthria; apraxia of speech	Likely cognitive and/or language involvement
	Spasticity and weakness	Yes—flaccid and/or spastic dysarthria	Possible cognitive involvement late in the disease process