



Post-stroke Dysphagia: Recent Insights and Unanswered Questions

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Abstract

Purpose of Review We explored themes in recent post-stroke dysphagia literature, focusing on the following questions: (1) What does post-stroke dysphagia look like?; (2) Who gets post-stroke dysphagia?; (3) What are the consequences of post-stroke dysphagia?; and (4) How can we improve treatment of post-stroke dysphagia?

Recent Findings There have been several improvements in quantitative descriptions of swallowing physiology using standard and new evaluation techniques. These descriptions have been correlated with lesion locations, and several factors can predict development of post-stroke dysphagia and its sequelae. Novel treatment paradigms have leveraged post-stroke neuroplastic improvements using neurostimulation and biofeedback techniques. Despite recent findings, the field is limited by lack of standardization and unanswered questions on rehabilitation variables.

Summary Our improved understanding of post-stroke dysphagia will enhance our ability to prevent, identify, and treat it. Future work should be grounded in swallowing physiology and continue refining treatments, particularly in the acute stage.

Keywords Stroke · Deglutition · Dysphagia · Rehabilitation

Introduction

Swallowing is a complex sensorimotor process involving the coordination of multiple muscle groups to propel a bolus of food or liquid from the mouth to the stomach while protecting the airway and minimizing residue. Swallowing pattern-generating neural circuitry is situated in the rostral medulla [1], with widespread cortical and subcortical activation associated with movement preparation and sensory processing [2••]. Thus, stroke-related lesions in either cortical hemisphere, subcortical control circuits, or in the brainstem can result in difficulty swallowing or *dysphagia* [2••, 3]. See Felix and colleagues' recent review for a tutorial on current

practice for screening, evaluation, and treatment of post-stroke dysphagia [4••].

Review of recent literature regarding post-stroke dysphagia underscores four themes of research questions: (1) What does post-stroke dysphagia look like?; (2) Who gets post-stroke dysphagia?; (3) What are the consequences of post-stroke dysphagia?; and (4) How can we improve treatment of post-stroke dysphagia? This review will focus on publications that explored these questions with discussions of potential for implementation into clinical practice and unanswered questions.

What Does Post-stroke Dysphagia Look like?

Swallowing function is typically first evaluated at bedside, either by a dysphagia screen or by thorough evaluation of orofacial structure and function by a speech-language pathologist. Although it is an integral part of swallowing evaluation, there is consensus among the dysphagia community that instrumented approaches are instead the only ways to assess certain aspects of swallowing physiology, such as airway protection, bolus propulsion, and pharyngeal residue, to name a few [5].

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There has been considerable effort to quantify aspects of swallowing physiology visualized on videofluoroscopy, an x-ray evaluation of swallowing physiology (Fig. 1a, b). The Modified Barium Swallow Impairment Profile (MBSImP) operationalizes 17 components of oral, pharyngeal, and esophageal swallowing into Likert-type scales [6]. MBSImP metrics involving bolus transport in the oral cavity, epiglottic movement, and upper esophageal sphincter opening have shown good relationship with improvement in diet levels in the acute stages post-stroke [7]. Swallowing function can also be assessed as coordination of biomechanical movements and bolus passage over time through frame-by-frame video analysis. This level of analysis has revealed significant swallowing discoordination in patients with stroke-related dysphagia compared with healthy controls [8, 9]. Furthermore, this level of analysis identified quantitative swallowing coordination differences in patients with stroke and no dysphagia, showing that stroke may impact swallowing physiology below the threshold of clinical significance [10]. Combined analysis of biomechanics and timing can be achieved through algorithms tracking oropharyngeal structures over time in Computational Analysis of Swallow Mechanics (CASM) [11]. Compared to healthy controls, persons with stroke show quantitative differences in hyoid excursion, laryngeal elevation, tongue base retraction, pharyngeal shortening, and timing of bolus movement [11, 12].

While the quantitative evaluation of videofluoroscopy is still largely subjective, a relatively new technique has been established to objectively and quantitatively measure pressures generated along the pharynx and esophagus during a swallow: high-resolution manometry [13•] (Fig. 1c). Patients with post-stroke dysphagia can have reduced pharyngeal propulsive pressures, abnormal upper esophageal sphincter relaxation pressures, or both [14, 15]. Pharyngeal manometry has revealed interesting compensations to swallowing function in those with brainstem stroke: (1) *pharyngeal mis-sequencing* [16], where pharyngeal pressures are generated simultaneously instead of in

a top-down pattern, and (2) *vacuum swallowing* [17, 18], where extreme sub-atmospheric pressure generated in the upper esophageal sphincter and esophagus will “suck” the bolus downwards in the setting of low pharyngeal pressures.

Improvements in evaluation techniques will lead to more precise dysphagia diagnoses and better assessment of functional improvement. While these new approaches provide us with enhanced ways of understanding post-stroke swallowing physiology, they do not give information on their own regarding how disruptions of certain neural pathways result in altered physiology or how to best treat these physiologic deficits.

Who Gets Post-stroke Dysphagia?

Stroke is one of the leading causes of dysphagia, with incidence rates up to 80% [19]. Incidence estimates depend on the definition of dysphagia, which can range from failing a dysphagia screen, to prescribed diet modifications, to measures of physiology on an instrumented swallowing study [20].

A common focus of post-stroke dysphagia research in the past 5 years has been to predict what patient characteristics are associated with a greater risk of dysphagia, particularly in the acute and subacute phases. Table 1 summarizes independent predictors of post-stroke dysphagia. Other predictors of post-stroke dysphagia not appearing in multiple sources include male sex [21], higher Glasgow Coma Scale on admission [22], hemorrhagic vs. ischemic stroke [37], anterior or middle cerebral artery involvement [22], large vs. small vessel occlusion [23], and facial palsy [31]. Hyperlipidemia and lesions isolated to the thalamus are reported to occur less frequently in those with post-stroke dysphagia [24, 25], but there is no apparent protective measure of either condition. Many studies excluded patients requiring intubation, but Dunn and colleagues [26] report intensive care unit stay, length of intubation, and need for tracheostomy to also be independent

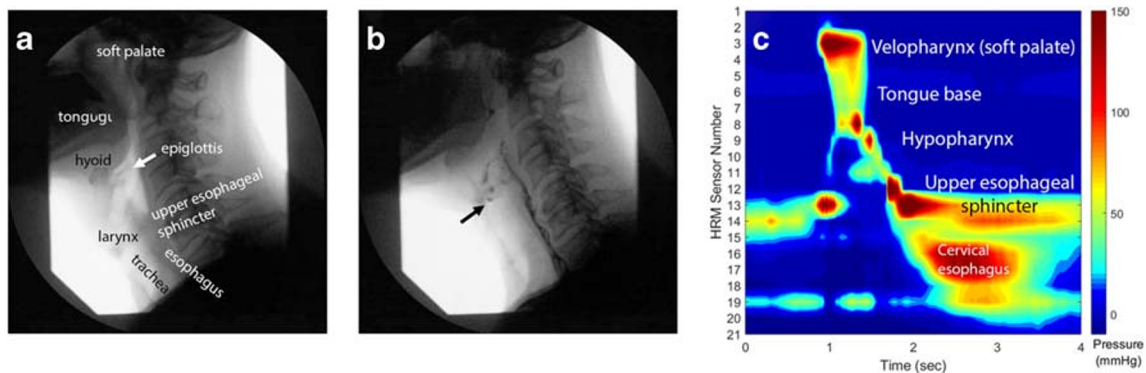


Fig. 1 Swallowing evaluations in a 58-year-old man in the chronic stage following stroke. (a) Videofluoroscopic swallow study frame in a rest position prior to swallowing with anatomic labels; (b) video frame mid-swallow; arrow identifies aspirated material; and (c) pharyngeal high-

resolution manometry spatiotemporal plot displaying pressure generated over the entire swallow; hypopharyngeal pressures are generated at the level of the larynx. Studies were performed on the same patient on the same day

Table 1 Independent predictors of post-stroke dysphagia across multiple studies

Older age [21–30]
Greater NIHSS [21, 23, 25, 27, 30–36]
Greater mRS [28, 32, 37]
Lower Barthel index [37, 38]
Malnutrition or lower BMI at admission [28, 37, 39]
Greater lesion volume [27, 31, 36, 40]
Subcortical vs. cortical involvement [24, 41, 42]
Brainstem involvement [3, 24, 25, 32, 34, 41, 43]
Corticobulbar tract involvement [38, 44]
White matter involvement [25, 38, 44]
Presence of dysarthria [31, 32]
Presence of dysphonia or reduced maximum pitch [31, 45]
Cognitive impairment or dementia [22, 29]

NIHSS, National Institutes of Health Stroke Scale; mRS, Modified Rankin Scale; BMI, body mass index

predictors for post-stroke dysphagia. Many publications on predicting dysphagia post-stroke rely on failing a dysphagia screen—or even participating in a screen to begin with—to identify dysphagia. Dysphagia screening is an important part of the clinical process, can reduce rates of aspiration pneumonia [46], and is part of the guidelines set by the American Heart Association and American Stroke Association [47], but its use to identify persons with dysphagia in research is problematic. As with any screen, those for dysphagia consist of observing a patient perform a simple task to identify those who would benefit from further evaluation. Screening tests inherently have high specificity, but many suffer from low sensitivity [48•, 49]. Although the aforementioned guidelines mention the utility of dysphagia, they do not specify types of dysphagia screens or optimal times when dysphagia should be evaluated.

With refinements on quantitative analysis of video swallowing evaluations, research has begun to investigate infarct locations that are associated with particular physiologic swallowing deficits and their sequelae. While still in early stages, a summary of findings of region of interest-based lesion mapping associated with dysphagia signs is outlined in Table 2. Notably, neither Daniels et al. [3] nor Wilmskoetter et al. [53] reported lesion location relationships when dysphagia physiology metrics were collapsed into a *total impairment* score. These complex findings highlight the multivariate nature of swallowing and the heterogeneity that can be seen in post-stroke dysphagia. An important limitation of these studies was the exclusion or underrepresentation of patients with brainstem lesions; its exclusion from Table 2 should not be a sign of irrelevance of brainstem lesions. This line of work is also an important step in understanding neural control of separate aspects of swallowing and how it may be disrupted by neurologic damage.

What Are the Consequences of Post-stroke Dysphagia?

Dysphagia is a significant predictor of negative health and psychosocial outcomes. Consequences associated with post-stroke dysphagia mirror those of other dysphagia etiologies and include malnutrition, dehydration, reduced quality of life, and aspiration pneumonia [54]. Furthermore, all-cause dysphagia increases length of inpatient stays by 2.8 days, costs by 34%, and more than doubles in-hospital mortality rate [55].

Recent work in stroke has attempted to predict the influence of post-stroke dysphagia and other patient factors on unfavorable outcomes. There is an association of post-stroke dysphagia with increased mortality [22, 28, 30, 34, 37, 56–58], overall unfavorable outcome [34, 35, 58], greater dependency [30, 36, 57], longer length of stay [33, 36], less likely to discharge home [35, 36, 57, 58], and modified diet or reliance on tube feeding [22, 35, 43, 57]. Longer length of stay due to dysphagia may occur in patients whose medical evaluation is completed, but the medical team is waiting to see if the dysphagia will resolve or a percutaneous gastrostomy tube is needed, as many skilled nursing facilities will not admit patients reliant to tube feeding, but without a permanent feeding tube.

Possibly the most cited sequela of post-stroke dysphagia is an increased risk of pneumonia [21, 30, 34, 36, 56, 57, 59–62]. Development of post-stroke pneumonia is multifactorial [60–62], and dysphagia should thus only be considered a risk factor. Furthermore, patients placed *nil per os* (NPO) due to severe dysphagia will continue to aspirate saliva and refluxate. NPO status and tube feeding dependence have long been understood as independent predictors of aspiration pneumonia [63•]. Implementing oral care protocols to decrease oral bacteria may help with reducing risk of pneumonia [47], but more studies are needed to assess causation.

Work investigating post-stroke dysphagia outcomes also suffers from the limitation that dysphagia screens are used to identify patients with dysphagia. However, the only study to investigate a particular dysphagia sign and outcomes was with spontaneous saliva swallow rate [35]. More precise understanding of swallowing physiology allows not only for more precise approaches but for better understanding of prognoses.

How Can We Improve Treatment of Post-stroke Dysphagia?

Traditional dysphagia therapy following stroke focuses on compensations and behavioral rehabilitation strategies. Treatment is patient-specific, with successful dysphagia therapies implemented in one patient population not necessarily producing the same results in another population [64]. The understanding of spontaneous swallowing recovery following

Table 2 Lesion locations associated with specific signs of dysphagia

Deficit/sequela	Brain region
Prolonged oral bolus transit time	L inferior frontal lobe [50] L precentral gyrus [50]
Prolonged pharyngeal bolus transit time	Insula [51] R basal ganglia [50] R corona radiata [50] Corticobulbar tract white matter [44]
Delayed onset of pharyngeal swallow trigger	R middle temporal gyrus [52] R superior temporal gyrus [52] R temporal pole [52] R lateral occipital cortex [52] R angular gyrus [52] R supramarginal gyrus [52] R planum temporale [52] R caudate [52] R superior longitudinal fasciculus [52] R parietal operculum cortex [52] Corticobulbar tract white matter [44]
Reduced laryngeal elevation	R precentral gyrus [53] R anterior & posterior insula [53] R external capsule [53] R superior corona radiata [53] R superior longitudinal fasciculus [53] Corticobulbar tract white matter [44]
Reduced anterior hyoid excursion	L amygdala [53] L ansa lenticularis [53] L lenticular fasciculus [53]
Reduced laryngeal vestibule closure	L postcentral gyrus [53] L supramarginal gyrus [53] R anterior & posterior insula R superior corona radiata [53] R external capsule [53]
Pharyngeal residue	R postcentral gyrus [53] R supramarginal gyrus [52, 53] R angular gyrus [52, 53] R superior & posterior corona radiata [53] R tapetum [53] R internal capsule [53] R superior longitudinal fasciculus [53] R posterior superior temporal gyrus [52, 53] R middle temporal gyrus [52] R planum temporale [52] R frontal orbital cortex [52] R parietal operculum [52]
Penetration/aspiration	R precentral gyrus [53] R postcentral gyrus [53] R superior longitudinal fasciculus [53] R supramarginal gyrus [40, 53] R superior temporal gyrus [40] R planum temporale [40] Corticobulbar tract white matter [44]
Impaired cough response	R paracingulate gyrus [52] R accumbens [52] R cingulum [52] R amygdala [52] R pallidum [52]

Table 2 (continued)

Deficit/sequela	Brain region
Impaired oral intake	R superior longitudinal fasciculus [52] R inferior frontal gyrus [52] L middle temporal gyrus [52] L postcentral gyrus [52] L supramarginal gyrus [52] Insula [36] Rolandic operculum [36] Superior corona radiata [36] Putamen [36] External capsule [36] Superior longitudinal fasciculus [36]
Tube feeding dependence	Insula [36] Rolandic operculum [36]
Pneumonia	R precentral gyrus [52] R postcentral gyrus [52] R paracingulate gyrus [52] R anterior cingulate gyrus [52] R supplementary motor cortex [52] R superior frontal gyrus [52] R middle frontal gyrus [52] R inferior frontal gyrus [52] R cingulum [52] R amygdala [52] R & L thalamus [52] L anterior thalamic radiation [52]

R, right; L, left; if there is no demarcation of R or L, there was no significant association of hemisphere

stroke is constantly evolving, and novel treatment methods are being developed and implemented to better manage and enhance swallowing recovery. Two major themes emerged in reviewing recent post-stroke dysphagia rehabilitation literature: neurostimulation and biofeedback.

Neurostimulation

With evidence of neural repair mechanisms and increased cortical activity playing a significant role in the swallowing recovery process following stroke, noninvasive neurostimulation therapies are of particular interest in treatment in post-stroke dysphagia. Neurostimulation can promote cortical reorganization to accelerate the natural process of stroke recovery and is characterized as peripheral or central stimulation [65]. Stimulation techniques hold promise for implementation in the early stages post-stroke, when patients may not have the language or cognitive barriers that may preclude following complex therapeutic instructions.

Peripheral Stimulation

Pharyngeal Electrical Stimulation

Pharyngeal electrical stimulation (PES) passively stimulates the pharynx with low-amplitude electrical impulses. Using

transnasal catheters with electrodes in the pharyngeal region, PES is thought to increase brain activity in areas that control swallowing and promote cortical reorganization of the swallowing motor cortex [66].

Early research in PES established effective stimulation parameters of 5 Hz, 10 min/day for 3 consecutive days at an intensity determined by the patient's perceptual threshold plus 75% the difference between the patient's maximum tolerated threshold and perceptual threshold [67]. These parameters have been implemented in recent studies to evaluate PES treatment in hemispheric stroke with dysphagia complications in the early subacute phase. Initially, these studies showed promising results in improving swallowing outcome; however, more recent research has been unable to reproduce such findings, showing no significant improvement in aspiration and clinical dysphagia at 2 weeks and 3 months posttreatment [68, 69].

Despite inconclusive results of the effectiveness of PES, other studies focused on administering PES to tracheostomized stroke patients with dysphagia yielded positive results. PES improved dysphagia enough to enable decannulation in the majority of patients [70, 71, 72]. The effectiveness of PES may be related to stroke severity, with more severe stroke patients exhibiting better therapeutic response than patients with mild stroke [68, 70]. However, long-term effects of PES on swallowing outcome, relative effectiveness of active vs. passive PES, optimal timing of PES therapeutic intervention, and the optimal stimulation parameters remain unclear.

Neuromuscular Electrical Stimulation

Another method of peripheral stimulation under exploration is neuromuscular electrical stimulation (NMES). Transcutaneous electrodes pass electrical current to generate muscle contractions in suprahyoid or infrahyoid muscles [65, 73]. Suprahyoid stimulation will activate the *geniohyoid*, *mylohyoid*, and *anterior belly of the digastric* muscles, resulting in elevation of the hyoid bone and larynx, while infrahyoid stimulation will activate the *omohyoid*, *sternohyoid*, and *sternothyroid*, depressing the hyoid bone and larynx [74]. Suprahyoid stimulation is thought to enhance the strength of weak muscles in order to elevate the hyolaryngeal complex to facilitate airway protection for swallowing, whereas infrahyoid stimulation is thought to depress the hyolaryngeal complex as a mechanism to swallow against resistance [73]. A meta-analysis concluded that treatment with NMES was more effective in improving swallowing function in the short-term than treatment without NMES in both acute/subacute and chronic stroke patients [73]. However, there was significant heterogeneity across the studies, which was attributed to variability in stroke type and duration, frequency and intensity of stimulation, and sample size and blinding [73].

NMES for dysphagia treatment is controversial and is not covered by some health insurers [75]. While NMES treatment

has generated some positive results in treating dysphagia when coupled with traditional dysphagia therapy in stroke patients, the exact mechanism of NMES is unclear, and there has been little consensus on optimal electrode placement as well as proper frequency and intensity for stimulation. Use of NMES in therapy requires careful consideration of the individual patient's specific swallowing deficits and clear understanding of which muscles are being targeted.

Central Stimulation

Repetitive Transcranial Magnetic Stimulation

Repetitive transcranial magnetic stimulation (rTMS) uses a copper coil placed on the scalp to deliver magnetic stimulation, resulting in a change to electrical activity on neocortex directly under the coil [76]. Pulses are delivered at a low frequency of ~1 Hz, and rTMS slows neuronal excitability and produces an inhibitory effect, whereas pulses delivered at ≥ 3 Hz increase neuronal excitability [76, 77]. Both high-frequency and low-frequency rTMS have been evaluated as treatment methods for post-stroke dysphagia.

Some randomized, controlled trials have explored the effectiveness of rTMS in dysphagia rehabilitation in the acute, subacute, and chronic stages. Meta-analyses by Pisegna et al. and Liao et al. evaluated a total of 11 studies and concluded that rTMS over the pharyngeal motor cortex paired with traditional dysphagia therapy was an effective method to improve swallowing function in stroke patients with dysphagia [76, 77]. rTMS showed therapeutic benefit at 4 weeks post-treatment and was more effective at high-frequency rather than low-frequency [77]. Bilateral or contralesional stimulation may also have a positive effect on dysphagia after stroke, while ipsilesional stimulation showed no effect [77]. There is an ongoing trial evaluating effectiveness of rTMS applied to the cerebellum in post-stroke dysphagia [78]. Consensus has not yet been reached on specific stimulation parameters, number of pulses per session, and length of treatment.

Transcranial Direct Current Stimulation

Transcranial direct current stimulation (tDCS) involves a low-intensity, electrical current (1–2 mA) applied between a cathode and anode placed on the scalp [76, 79]. Anodal tDCS depolarizes the resting membrane potential and thereby increases neuronal excitability, whereas cathodal tDCS hyperpolarizes the resting membrane potential, decreasing neuronal excitability [79]. Anodal tDCS has been explored as a method to enhance swallowing recovery in dysphagic stroke patients. Anodal tDCS can be unilateral or bilateral. *Unilateral anodal tDCS* involves placing the anode over the affected pharyngeal motor cortex and the cathode over the contralateral supra-orbital region, whereas *bilateral tDCS* places an anode over the

pharyngeal motor cortex of both hemispheres and a cathode over each corresponding contralesional supraorbital region [79]. *Dual stimulation* utilizes both anodal and cathodal tDCS, implementing anodal tDCS over the damaged hemisphere and cathodal tDCS over the contralesional one [80].

Current research on tDCS as a treatment method for dysphagic stroke patients is highly variable. Information regarding the site of electrode placement (ipsilateral vs. contralateral), stimulation parameters (intensity and duration), method of stimulation (unilateral vs. bilateral), time post-stroke, and the type of stroke patient that would most benefit is lacking. A meta-analysis evaluated three small-scale studies that explored the effectiveness of unilateral tDCS on swallowing function in stroke patients and found tDCS treatment to have a moderate but not significant pooled effect size when compared to treatment without tDCS [76]. However, the studies varied in the hemisphere stimulated, treatment protocol, and the outcome measurements obtained [76].

More recent studies exploring bilateral tDCS have resulted in positive findings. Ahn et al. [79] found significant improvement in swallowing function in chronic stroke patients. Additionally, Li et al. found that unilateral and bilateral tDCS of the affected hemisphere combined with traditional dysphagia therapy were both effective in improving swallowing function in chronic stroke patients, with bilateral tDCS resulting in a more substantial improvement [81]. Further studies are needed to compare the effectiveness of unilateral and bilateral stimulation as well as ipsilateral and contralateral stimulation in order to gain a greater insight into the most effective method for tDCS in stroke patients with dysphagia.

Paired Associative Stimulation

Paired associative stimulation (PAS) is a neurostimulation technique that simultaneously implements peripheral and central stimulation methods to induce excitation of the pharyngeal motor cortex [65]. The rationale behind PAS is further built on Hebbian neuroplastic principles through the delivery of stimulation at multiple sites. Hamdy et al. were the first to explore PAS as a treatment method for dysphagic stroke patients. Pairing PES with TMS, they found that PAS led to short-term improvement in swallowing function, increasing excitability of the unaffected pharyngeal cortex and reducing penetration-aspiration scores in chronic stroke patients [82, 83]. Additionally, they determined that repeated stimulation could induce excitability in patients that did not respond to PAS initially and that administering PAS for a shorter time duration (~10 min) resulted in more substantial changes in cortical excitability than administering PAS for a longer time duration (~30 min) [82, 84].

Recently, little research has been conducted to further explore the effectiveness of PAS as a treatment method for post-

stroke dysphagia. In 2019, Zhang et al. reported that TMS paired with NMES was more effective in treating post-stroke dysphagia than NMES alone and that bilateral TMS paired with NMES was more effective than unilateral stimulation [85]. However, this study had a small sample size, and these results would need to be validated by a larger study.

Biofeedback

As swallowing is a complex behavior with many small muscle groups working deep in the oral cavity and pharynx, the patient's ability to assess their own performance is limited. Biofeedback is an adjunct to swallowing therapy, wherein the patient is provided visual or auditory signals that enable them to alter conscious or subconscious mechanisms involved in the swallowing process and improve their swallowing performance [86, 87].

The most common biofeedback technique used in post-stroke dysphagia therapy is surface electromyography (sEMG). sEMG biofeedback uses two small electrodes placed on the submental muscles to measure the timing and force of the muscle contraction, which is displayed graphically on a screen [88, 89]. A recent meta-analysis found that sEMG biofeedback coupled with swallowing maneuvers increased hyoid displacement in post-stroke dysphagia patients when compared with controls [88]. sEMG biofeedback has the potential for short-term benefits to dysphagia rehabilitation; however, it has been hypothesized that once patients learn swallowing therapies, biofeedback yields no further benefits. Careful training must be undertaken to ensure that patients can generalize behaviors so they can be replicated without the feedback.

Another novel dysphagia treatment approach using sEMG biofeedback is Biofeedback in Strength and Skill Training (BiSSkiT). BiSSkiT is a training protocol focused on improving the precision of swallowing muscle contraction, whereas typical sEMG biofeedback typically focuses on amplitude of muscle contraction. One of the BiSSkiT treatment methods involves the participants swallowing such that their peak waveform fell within a square target displayed on the computer monitor. This target moves randomly and requires the participants to practice precision and control of their swallowing movements in order to successfully fall within the target. Initial work in neurodegenerative conditions suggests that skill training improved coordination and timing of swallowing and could be effective in dysphagia rehabilitation [90, 91]. While no trials have been published in post-stroke dysphagia, the positive findings of early studies suggest that skill training has potential to be effective in dysphagia rehabilitation, particularly in cases of swallowing dyscoordination.

A Cochrane review in 2018 revealed no subgroup effects for acute/subacute therapy approach (traditional therapy, NMES, PES, tDCS, TMS, acupuncture, drug therapy) on outcomes such as death/dependency/disability, length of

inpatient stay, swallowing ability, or aspiration pneumonia [20]. However, this review also identified very low quality evidence for the ability of swallowing treatment to improve swallowing ability or prevent aspiration pneumonia [20]. There is much more work needed to optimize post-stroke dysphagia rehabilitation.

Clinical Implementation

How can post-stroke dysphagia clinical practice be impacted by recent research findings? The field is continually evolving, but a lack of standardized evaluation and treatments and challenges to rapid translation of research findings to clinic can impede practice improvements.

Improvements in our clinical evaluation methods have the potential to revolutionize our understanding of dysphagia pathophysiology. However, implementation of videofluoroscopic quantification methods requires intensive initial and reliability training and can take more time out of a busy clinic day to implement. Further, recent findings of coordination deficits are informative, but the bulk of the traditional dysphagia therapies are built on a foundation of strengthening swallowing musculature, not improving coordination. Acquiring high-resolution manometry equipment is costly and has other institutional barriers, such as a lack of a reimbursable Medicare code that is billable by speech-language pathologists [92]. Clinicians are the drivers of change and need to be persistent and persuasive with hospital and clinic leadership to move the field forward and ensure patient care happens at the leading edge of the evidence base.

Predictive modeling—both for determining risk for dysphagia and consequences thereof—is helpful to know for clinical practice, particularly for triaging patients who may require more urgent, swallowing evaluation. However, a takeaway should not be that a patient with none of the risk factors will not develop dysphagia. The impetus remains on clinicians to use critical thinking and sound judgment to examine each patient as a case study and work to treat their specific deficits.

In the current climate, another barrier to implementing ideal clinical practices is the very real threats associated with COVID-19. Telehealth for swallowing therapy has been deemed “safe and effective” [93]. However, this does not take into account the need for imaging assessments, particularly in the acute phases of stroke, where a patient’s status is in a constant state of flux. See the recent taskforce report put forth by the Dysphagia Research Society and an international effort derived from physical therapy for best practice recommendations in the setting of COVID-19 [94, 95].

Overall, there are barriers to adoption of new practices hospital-wide, including obtaining needed equipment, supplies, and training, limiting the ability of the US medical system to reimburse novel treatment paradigms. These

limitations make it difficult for clinicians to practice at the cutting edge of the evidence base [96•].

Unanswered Questions

There are still many unanswered questions that limit the practicing speech-language pathologist and intraprofessional team from answering: *What do we need to do for the patient now? In the upcoming weeks/months? Long-term?*

Due to the enhanced neuroplasticity that occurs early after stroke onset, it is highly beneficial to implement treatments in acute and subacute phases of stroke [97]. From a practical perspective, interventions intended to decrease the length of hospital stay or occurrence of dysphagia-related aspiration pneumonia would likely need to be initiated early following stroke. However, the neurophysiology of dysphagia recovery following stroke is complex and incompletely understood, and inpatient speech-language pathologists often have very little time or resources to implement dysphagia treatment protocols [96•]. This presents challenges to managing dysphagia, particularly in the acute and subacute phases, especially since many of these studies are focused on the chronic period of recovery and there is not a universally accepted method for evaluating post-stroke dysphagia in the inpatient setting. Determining the optimal treatment methods to implement in individual patients and understanding the neurophysiological effects of more novel methods remain unclear. Studies have widely varying parameters and intervention timings, and their results are confounded by many factors including mixed types of stroke, stages of stroke, and types and severity of dysphagia.

While the novel methods reviewed in this paper present a growing consensus for the effectiveness of neurostimulation and biofeedback in enhancing dysphagia rehabilitation, there are many questions and uncertainties that need to be explored. By nature, stroke severity is influenced by multiple factors, and due to the complexity of the treatment interventions, it is difficult to determine what exactly led to recovery [98]. Studies reviewed here consisted of stroke patients with a mix of subacute and chronic stroke who will naturally recover at different rates making it difficult to determine the effectiveness of novel therapies. Additionally, stimulation parameters and treatment times greatly varied in the studies with suboptimal parameters or late intervention yielding neutral results for promising neurostimulation methods. Lastly, the majority of literature reviewed were small-scale studies or eliminated patients who had multiple strokes or comorbidities that could result in dysphagia; thus, they were not fully representative of stroke populations, and their results could fail to accurately represent their effectiveness.

With the exception of PES, a particular gap in the rehabilitation literature is on rehabilitation commencing in the acute phases, when the patient may not be able to participate in active rehabilitation or take an oral diet. There are many unknowns about the degree of decompensation that can happen in the absence of eating and drinking, or if there is a critical window for rehabilitation in patients who go on to develop chronic and treatment-refractory dysphagia. Questions on timing of dysphagia intervention might be better explored with animal models of stroke. Cullins and Connor [99] used a middle cerebral artery occlusion rat model to specifically investigate post-infarct dysphagia with validated, species-specific swallowing physiology measures. This and other animal models will improve our understanding of the pathophysiology of post-stroke dysphagia and explore questions regarding timing, dose, and other unknown aspects of post-stroke swallowing rehabilitation. With dysphagia being associated with increased hospital stay and the development of aspiration pneumonia, treatments that could be effective in the acute period of recovery may be of particular interest. Interventions that can potentially decrease the length of hospital stay or prevent aspiration pneumonia, particularly during the COVID-19 pandemic, would likely be especially relevant to the clinical community.

Systematic research programs are needed with rigorous, prospective, well-designed studies to answer these questions. Clinicians need to take an active role in staying up to date with the literature and being proactive with implementing change in a standardized way.

Conclusions

Stroke can have significant negative consequences on swallowing physiology. Great strides have been taken in the past few years in an effort to better characterize post-stroke dysphagia, to predict which stroke variables and patient factors lead to dysphagia and negative sequelae, and to address rehabilitation from a neuroplastic perspective. Several challenges to implementation and questions regarding post-stroke dysphagia evaluation and treatment remain and will be aided with continued focus on defining dysphagia by swallowing physiology rather than screening failure or diet level. Creative and interdisciplinary work will continue to reduce stroke risk, prevent negative consequences of post-stroke dysphagia, and help rehabilitate patients back to functional swallowing.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

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- [org/10.1016/S1474-4422\(18\)30255-2](https://doi.org/10.1016/S1474-4422(18)30255-2) **Strong randomized control trial investigating the effects of PES on tracheostomized stroke patients with dysphagia. Trial ended early due to positive results.**
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